
The Emperor's Clothes: Why Aren't Chemicals Tested for Their Impacts on the Developing Brain...Why Is This Important?

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Background

More than 80,000 commercial chemicals now are registered with the Environmental Protection Agency and another 2-3,000 new chemicals are registered each year. These agents are used in industrial processes, put into consumer products, and intentionally added to the food chain. The U.S. Congress' Office of Technology Assessment (OTA) has estimated between 3 and 5 percent of non-pesticide chemicals—or 2,400 to 4,000 in total—to be nervous system toxins (OTA 1990). In 1997, known or suspected neurotoxins comprised nearly three-quarters of the top 20 toxic chemicals released by large U.S. industrial facilities, as reported by the Toxics Release Inventory (TRI)—1.2 billion pounds worth in total (Schettler et al. 2001). It has been conservatively estimated that 68 million children have had toxic exposures just to lead in gasoline.

As with the intentional introduction of lead—a known neurotoxin—to gasoline in the 1920s, the public has often been reassured with claims that widespread commercial use of chemicals will not expose children to risks. In recent years this notion has been repeatedly dispelled, as techniques for detecting chemical traces have improved and as the Centers for Disease Control and Prevention (CDC) began its initiative to monitor the blood and urine of adults and children for traces of a wide range of environmental pollutants.

We therefore now know that young children routinely carry detectable levels of lead, mercury, PCBs, dioxins, flame retardants, and certain pesticides, among other neurotoxins. Several known or suspected neurotoxins are known to travel across the placenta, while others have been found in meconium (a baby's first stool), both indicating *in utero* exposure. Breast milk contamination with many such compounds adds to health concerns. In several instances, it also has been discovered, industry has made claims that children's exposure to neurotoxins was benign or nonexistent while suppressing direct evidence to the contrary. In other cases, industry assurances have been based more on wishful thinking than on the results of any scientific testing.

False Assumptions Underlying Chemical Regulation

Now that scientific evidence makes it impossible to deny that children are routinely exposed to multiple neurotoxins on a daily basis, the public is receiving reassurances of a different sort. These include the following sorts of claims:

- FALSE CLAIM #1:** Children’s exposure to neurotoxic chemicals generally are of insufficient magnitude to pose risks to children (i.e. the dose makes the poison).
- FALSE CLAIM #2** If any of these chemicals constituted a true hazard to children, we (society) would have taken note by now (i.e. injured or dead children could be counted).
- FALSE CLAIM #3** Before being put on store shelves or the commercial market, federal laws require that neurotoxic chemicals be extensively tested for safety, including their safety to children and fetuses.

Collectively, these claims closely are more or less the cornerstone assumptions underlying the entire U.S. system or “regime” for regulating commercial chemicals. Individual chemical risk assessments largely take these assumptions as a given. However, firm science suggests they often are wrong. Each is worthy of longer discussion, but this essay deals solely with the third such assumption.

Extensive Testing to Ensure Safety Is NOT Required

Polling suggests that many Americans discount health concerns about exposures to environmental chemicals due to their very strong belief that any product allowed by the government to be marketed must have already been thoroughly tested for safety. One wants to believe it. And the EPA and regulated industries often claim it. But it’s simply not the case, and least not from the perspective of parents or children.

As described in *In Harm’s Way: Toxic Threats to Child Development*, (Chapter 7), the existing requirements and regime for testing commercial chemicals—especially in terms of their potential for inducing neurotoxicity to the developing fetus or child— is a case of the *emperor having no clothes* (Schettler et al., 2001). The illusion of comprehensive safety testing probably persists, at least in part, because regulated industries have a strong incentive to perpetuate it, but also because of the technical nature of the issues involved, and not least because the statutory requirements for such testing are quite opaque to public scrutiny.

Testing of Organophosphate Pesticides

Some public health failures of the current U.S. regulatory “regime” for chemical safety or toxicity testing can be illustrated by looking at the body of testing typically performed for

pesticides used on food crops, especially those from the organophosphate (OP) class. Codified testing requirements for the more than 520 pesticides approved for use on food crops are much more rigorous than those for other classes of commercial chemicals. If safety testing for these food-use pesticides is inadequate, therefore, one can safely assume the gaps are worse for non-pesticide chemicals. OPs are worth singling out because there's no dispute they were specifically designed to be toxic to the brain and nervous system; EPA in fact considers the OPs as a class to be the most toxic of all registered pesticides. Additionally, most OPs have been marketed and sold for three or four decades or more, plenty of time for manufacturers to have carried out testing.

The public health failures of the U.S. chemical testing regime fall into several categories, as discussed below:

1. THE REQUIREMENTS FOR BASIC TOXICITY TESTING ARE INADEQUATE.

Any government effort to limit children's exposure to environmental chemicals, especially pesticides, is likely to stem largely from the results of toxicity testing in laboratory animals. Ideally, one would like to have human data to assess risks to humans. But funding is scarce for conducting controlled human studies with any degree of statistical power, which are large and expensive to conduct. And besides, in the case of intentional poisons like pesticides, studies that would dose humans to ascertain a "safe" level of exposure would be considered unethical. Regulations typically rely on extrapolation from the results of animal studies to estimate the risks for the average adult human. The use of animal testing to help assess the risks to humans from exposure to neurotoxins and other chemicals is therefore essential.

Under U.S. laws governing chemical regulation, the point at which regulators have leverage to demand toxicity testing is when proponents of a particular chemical product are first seeking to register it or, periodically, re-register it with the EPA. The basic or "core" battery of toxicity testing required for registering a chemical therefore is particularly important in determining if there will be adequate information to assess whether a child's exposure to that chemical is safe. For pesticides used in or on food, these core requirements are codified in section 40 of the Code of Federal Regulations (CFR), part 158, subpart F. They were last revised 20 years ago, in 1984, when much less was known about pesticides and children's vulnerability to them.

EPA has repeatedly acknowledged the deficiency of these requirements, particularly for determining the potential impact of pesticide exposure on the nervous system (See Letter to EPA Administrator Carol Browner, attached). In 1994, to address these deficiencies EPA proposed revisions to the Part 158 requirements, and had them reviewed by its own pesticide Scientific Advisory Panel. Again, in late-1998, EPA officials announced their intent to revise the Part 158 requirements. Despite repeated promises—the last in the waning weeks of Carol Browner's administration—EPA has failed to ever bring its revisions to the Office of Management of Budget, a prerequisite for issuing a proposed rule change.

Therefore, the still-deficient 1984 testing requirements remain in place. As of 2000, the toxicity of many pesticides registered prior to 1984 (including many OPs) still had not been reviewed under the relatively newer 1984 requirements. This resulted in a situation where some decades-old neurotoxic OP insecticides remained in widespread use even after EPA acknowledged the manufacturers had failed to submit the all the (conditional) tests for neurotoxicity required after 1984.

2. CORE REQUIREMENTS FOR FOOD-USE PESTICIDES INCLUDE NO SPECIFIC TESTING FOR POTENTIAL TOXICITY TO THE BRAIN OR NERVOUS SYSTEM IN EITHER ADULT OR YOUNG ANIMALS.

Deficiencies in the 1984 requirements are many. Among the most basic, however, is that there is no core requirement that pesticides used on foods be tested for their possible toxicity to the brain or nervous system. Several protocols exist for doing such testing and are validated, but they are considered “conditional” under EPA’s tiered testing regime. That means EPA would require a study of acute delayed neurotoxicity (in hens), for example, only on the condition that the pesticide in question was an organophosphate or related compound. Similarly, EPA could “conditionally require” an acute or subchronic (90-day) neurotoxicity study in rats, but codified requirements indicate this would only happen if other “core” tests had already signified neurotoxicity (or if other conditional tests had done so).

The faulty logic at work is that the core tests do not specifically assess for neurotoxicity, nor do they include any evaluation for changes in neurobehavioral function. Thus, “conditional” or second-tier testing is contingent on a positive neurotoxicity finding from a first tier test not designed for that purpose.

EPA recognized the problem. When EPA began revising Part 158 requirements in 1994, it proposed (to its SAP) that these revisions include a core requirement to do neurotoxicity testing for all food-use pesticides. By 1998, in fact, EPA had fashioned an entirely new neurotoxicity screening battery for this purpose (U.S. EPA, Office of Prevention, Pesticides and Toxic Substances, Health Effects Test Guidelines: OPPTS 870.6200 Neurotoxicity Screening Battery, August 1998. Again, these EPA revisions never went forward.

3. INDIVIDUAL TESTS OF TOXICITY ARE INADEQUATE FOR ASSESSING POTENTIAL HARM TO CHILDREN AND FETUSES.

From a child’s perspective, it is not sufficient simply to test a chemical for its potential toxicity. The test itself has to be sensitive for evaluating the kinds of health impacts of concern, in this case impacts on the developing brain and nervous system. Core testing is performed according to established protocols, essentially the blueprints for how to do the tests. In 1993, the National Research Council (NRC) determined the protocols for doing the toxicity testing included in EPA’s core battery to be lacking for drawing conclusions about the safety of exposed children:

“In general, the committee found that current and past studies conducted by pesticide manufacturers are designed primarily to assess pesticide

toxicity in sexually mature animals. Only a minority of testing protocols have supported extrapolation to infant and adolescent animals. Current protocols, do not, for the most part, adequately address the toxicity and metabolism of pesticides in neonates and adolescent animals or the effects of exposure during early developmental stages and their sequelae in later life.” (NRC 1993)

More specifically, all but two of the EPA-required core tests are performed in adult animals. Two *do* include testing of immature animals: the developmental toxicity study, and the two-generation reproductive study. The former fails to dose or assess dosed animals post-nataly (they are sacrificed while in the womb of the dosed dam animal), making it inadequate for assessing potential effects on brain or other organ function in infants and toddlers. In other words, impacts on sensitive traits such as behavior, learning or memory are not tested. Neither of the two studies includes much more than crude measures of chemical insult to the developing brain and nervous system. The neonatal brain is not weighed in either test, for example.

Nor are EPA’s “conditional” tests of neurotoxicity particularly sensitive for endpoints of concern. For example, they lack any quantitative assessment of behavior, or measure of effects on learning and memory (Foss, 1994). So, these second-tier studies are also insufficient for assessing safety vis-à-vis an exposed child or fetus.

4. CORE REQUIREMENTS FOR FOOD-USE PESTICIDES INCLUDE NO TESTING FOR POTENTIAL EFFECTS ON THE DEVELOPING BRAIN.

Since 1991, EPA has had a validated protocol for how to specifically test a chemical for its potential toxicity to the developing brain and nervous system. In fact, EPA officials announced in 1998 their intention to propose revisions that would mandate developmental neurotoxicity (DNT) testing under 40 CFR Part 158 requirements. In January 1999, the EPA’s panel of scientific advisors agreed with this step, pointing out that even when DNT testing wasn’t quantitatively more sensitive than other required studies, “it provides important qualitative data not obtained in other types of testing.” By May 2000, pesticide manufacturers had submitted DNT studies for just 9 pesticides of nearly 900 registered, 140 of which EPA acknowledges to be neurotoxic. EPA scientists, in scientific review committees, had previously recommended DNT testing for an additional 26 chemicals. Unfortunately, these recommendations impose no obligation on the manufacturers. Additionally, in 1991 EPA obtained the authority from the Office of Management and Budget (OMB) to “call-in” data from DNT testing for certain chemicals when specific “triggers” or criteria had been met. In September 1999, EPA exercised this authority by beginning a data call-in for manufacturers of the 140 pesticides known to be neurotoxins. The first phase, which called in DNT studies for more than 30 organophosphate insecticides, required the results of such studies to be submitted to EPA within 2 years. By Fall 2001, however, only a third of the studies had even been started, with EPA and industry still discussing *how* testing for the majority should be performed.

It's worth noting that core testing requirements also fail to address other important endpoints of concern to children's health, like the potential for hormone disruption or for toxicity to the immune system. Unlike development neurotoxicity, however, EPA has yet to formally validate a protocol for *how to do* this type of testing. Even if comprehensive testing for these endpoints were to make sense from the standpoint of protecting children, a validated protocol would be necessary to ensure that any such testing submitted to EPA would be useful, in both a scientific and regulatory sense.

While animal testing has been essential for assessing chemical risks, the individual animal tests currently used are not without their limitations. One can imagine that testing animals for neurodevelopmental endpoints of concern to children, such as learning, attention, and memory, is difficult and inexact. But how sensitive are these animal studies? For very few agents do toxicity data from both animal and human studies exist of sufficient quality to allow for direct comparison as to their sensitivity. Looking at such data for methylmercury, PCBs, and lead, Deborah Rice concludes that the animal tests typically relied upon by EPA for regulatory purposes were 100 to 10,000-fold less sensitive than the human studies at determining levels of exposure to these neurotoxins that were "safe" (Rice).

EPA does recognize that animal and human data aren't equivalent. That's why in its regulatory decisions, it will often use a tenfold "uncertainty factor" to modify its animal data as it extrapolates to a regulatory level for human exposures. Rice's study shows that when regulators only have animal data for known and suspected neurotoxins, (which is most of the time), its use of this 10X uncertainty factor can result in legal exposure levels that are far too high to protect women and children. This accords with observation that what we formally considered to be "safe" exposure levels for lead, for mercury, for PCBs, now far exceed our notion of what is toxic.

Conclusions and Recommendations

As Dr. Schettler has noted, a large percentage of cases of mental retardation likely have more than a single cause; in many individual cases, multiple causative factors probably interact, among them genetics, infectious agents, birth and other trauma, hormonal factors (hypothyroidism) and chemical agents. Many studies demonstrate that fetal exposure to alcohol and other solvents, PCBs, and heavy metals (mercury and lead) can contribute to neurodevelopmental disorders including mental retardation, although the exposures responsible for the latter generally have been large. Lower level exposures to these agents can cause declines in IQ, in the case of lead even at levels below those considered to warrant action under current CDC guidelines. Evidence of this has been gathered for the relatively few neurotoxicants for which large epidemiological studies have been conducted.

In contrast, study of the contribution of environmental chemicals to mental retardation is limited. Regulation for the vast majority of the 80,000 commercial chemicals in commerce depends on testing in laboratory animals. And, as Schettler points out, there is

no good animal model for studying mental retardation as an endpoint of concern. Traits of neurodevelopment which can be measured with existing animal studies, however, including learning, attention, activity level and memory. Much evidence exists to link impairments of these traits with relatively lower level exposures to environmental neurotoxicants mentioned above. However, as noted previously in this discussion, the protocols for doing such testing are not required of chemical registrants, nor are they generally performed. The lack of requirements for systematically evaluating chemicals for their neurodevelopmental impact, generally, may well contribute to the lack of demand for EPA and other regulatory agencies to develop assessment of these same chemicals for their potential contribution to mental retardation.

The implications for anyone concerned about potential chemical contributors to developmental disabilities, including mental retardation, are fairly profound and include the following:

RECOMMENDATION #1: Advocates for preventing neurodevelopmental disorders should become more familiar with the flaws of the current regime for testing chemicals. More specifically, to address the inadequacy of “core” testing of pesticides—at least 140 of which are known neurotoxins—children’s advocates ought to push for the EPA to send its long overdue revisions to the testing requirements (found in Part 158 of the Code of Federal Regulations) to the Office of Management and Budget (OMB), and to continue to pressure OMB to approve EPA’s issuance of a proposed rule. This might coincide with the next AAMR lobbying day, for example.

Recommendation #2: Being long overdue, organophosphate insecticide manufacturers subject to the 1999 “call-in” of DNT data should face immediate sanctions and/or withdrawal of their products from the market pending the overdue completion of these studies.

Recommendation #3: EPA issuance of the remaining phases of the DNT data call-in, covering the rest of the 140 neurotoxic pesticides known to EPA, should proceed on a strict timeline, with similar sanctions facing those failing to comply with deadlines.

Recommendation #4: Given the lack of mandated DNT testing, and the snail’s pace of any additional DNT testing under the existing “tiered” testing regime (including from data call-ins), and finally the backlog of up to 4,000 neurotoxic chemicals already being legally emitted into the environment without DNT testing, there can be little expectation of expeditious testing of already-approved chemicals. Therefore, the AAMR and other children’s advocates should consider sponsoring amendments to the Toxic Substances Control Act (TSCA) as well as to the provisions of the Federal Insecticides Fungicides Registration Action (FIFRA) that would institute restrictions on environmental releases of existing chemicals pending the submission of DNT testing that can provide assurance with reasonable certainty that pregnant women and children exposed to such chemicals will suffer no harm. This amendment essentially would shift the onus onto chemical registrants to prove the safety of their products in exchange for the public license to sell them.

Recommendation #5: Similar amendments to the same laws could require that the 2-3,000 new chemicals registered each year must undergo neurodevelopmental testing according to the most up-to-date techniques prior to receiving approval for sale and use.

Recommendation #6: Current CDC national biomonitoring programs, as well as the National Children's Health Study, should include biomonitoring of children for as many of the 140 known neurotoxic pesticides as possible, in addition to heavy metals, PCBs and dioxins, and other known neurotoxins in the environment.

Recommendation #7: Comprehensive surveillance for neurodevelopmental disorders should occur at the national level. Specifically, we recommend CDC, the National Center on Birth Defects and Developmental Disabilities (NCBDDD) should implement a national surveillance system specific to developmental disabilities, including the mentally retarded as one of the included populations.

References:

- Claudio L., Kwa W.C., Russell A.L., Wallinga D. Testing methods for developmental neurotoxicity of environmental chemicals. *Toxicol Appl Pharmacol.* 2000 Apr 1;164(1):1-14. Review.
- Claudio L., Bearer C.F., Wallinga D. Assessment of the U.S. Environmental Protection Agency methods for identification of hazards to developing organisms, Part I: The reproduction and fertility testing guidelines, *American Journal of Industrial Medicine* 1999; 35(6):543-553
- Claudio L., Bearer C.F., Wallinga D., Assessment of the U.S. Environmental Protection Agency methods for identification of hazards to developing organisms, Part II: The developmental toxicity testing guideline. *American Journal of Industrial Medicine* 1999; 35(6):543-553.
- Foss, J.A., "The application of a functional observational battery and motor activity test in safety assessment studies," *Neurobehavioral Toxicity: Analysis and Interpretation*, B. Weiss and J. O'Donoghue, eds., New York: Raven Press, 1994.
- Kitman J.L., The Secret History of Lead, *The Nation*, March 2000.
- National Research Council, *Pesticides in the Diets of Infants and Children*, National Academy Press, Washington, D.C., 1993. Schettler T., Stein J., Reich F., et. al. In Harm's Way: Toxic threats to Child Development. Greater Boston Physicians for Social Responsibility: Boston, MA, January 2001.
- Rice D., Evangelista de Duffard A., Duffard R., et al. Lessons for neurotoxicology from selected model compounds: SGOMSEC joint report. *Environ Health Perspect* 104(suppl 2):205-215, 1996.

U.S. Congress, Office of Technology Assessment, *Neurotoxicity: Identifying and Controlling Poisons of the Nervous System*, OTA-BA-436 (Washington, DC: U.S. Government Printing Office, April 1990), p. 3.

USEPA Memorandum, Dichlorvos (DDVP) - Report of the FQPA Safety Factor Committee, June 2, 1998.

Wallinga D.B., Pesticides and Developmental Neurotoxicity: Assuring Children's Safety in the Context of the Food Quality Protection Act, Published Abstract in *Neurotoxicology* 1999; 20(1):125.

Wallinga, David, Putting Children First: Making Pesticide Levels in Food Safer for Infants and Children, Natural Resources Defense Council, Washington, D.C., April 1998.

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May 11, 2000

The Honorable Carol Browner
Administrator, Environmental Protection Agency
Mail Code 1101A
Ariel Rios Building
1200 Pennsylvania Ave., NW
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Re: Finalization of Revisions to EPA Pesticide Toxicity Testing Requirements

Dear Administrator Browner:

As organizations devoted to public health, the health and safety of children and the environment, we urge you to promptly finalize and sign the long-delayed revisions to requirements for testing pesticides for toxicity prior to their registration or re-registration.

EPA has been revising these requirements (in 40 CFR part 158, subpart F) since before March 1991, when it vowed to issue a formal proposal within six months. In November 1994, the FIFRA Scientific Advisory Panel (SAP) generally endorsed these revisions. Yet they were never signed or finalized.

If the revised requirements had been finalized in 1994, new pesticides for the first time would have been tested as a core requirement for their toxicity to the brain and nervous system, using the EPA's acute and 90-day neurotoxicity screening batteries in adult animals. Note that the State of New York, as well as various public health and public interest groups, had petitioned EPA in 1987 to routinely test pesticides for their neurotoxicity and neurobehavioral toxicity.

Since 1998, EPA again has been signaling its intent to revise the pesticide toxicity testing requirements, promising at one point to do so by Spring 1999. To its credit, EPA intends to make the latest revisions reflective of new science. For example, EPA now vows to add a requirement that all new pesticides be tested for toxicity to the developing nervous system as well.

We strongly support this addition, which follows directly from the National Academy of Science's 1993 finding that exposure to neurotoxic compounds at levels believed to be safe for adults, nevertheless can result in permanent loss of brain function if occurring during the prenatal and early childhood periods of brain development. In addition, it has been well established in the laboratory that developing animals are more sensitive than

adults to acute neurotoxicity from cholinesterase-inhibiting chemicals, like the organophosphate and carbamate insecticides – widely used in schools, around homes and on agricultural crops. Recent studies have only added to the substantial body of evidence that cholinesterase-inhibiting pesticides may adversely affect brain development through not just one, but multiple pathways.

In 1998, EPA scientists reviewed the results of all developmental neurotoxicity (DNT) testing submitted to EPA to date, including that for nine pesticides. The review suggested that for many chemicals a revised battery of core testing, including DNT testing, would be more sensitive than the current core battery in assessing many adverse effects, including effects on the developing brain and nervous system. It is apparent therefore that EPA's current requirements are insufficient to protect against a pesticide's possible effects on brain development in people.

Your own internal Toxicology Working Group concluded in November 1998 that “40 CFR Part 158.340 (Subpart F) should be updated as soon as possible to include the adult and developmental neurotoxicity guidelines...” In March 1998, the FIFRA Scientific Advisory panel unanimously recommended that EPA consider requiring DNT testing for all neurotoxic insecticides, with a portion of the panel urging a DNT testing requirement for all new pesticides. This same panel stressed: “One point of consensus is that the developing human, especially its nervous system, is vulnerable to a variety of toxicants, both pesticides and non-pesticides, and is certainly deserving of our best efforts to afford it protection with the intent of the 1996 FQPA.” In 1993, the National Research Council also recommended that developmental neurotoxicity data be included in pesticide evaluations.

One year ago, on May 12, 1999, the Learning Disabilities Association, National PTA, Physicians for Social Responsibility, American Public Health Association and other public health and children's groups wrote you urging EPA to move directly to include developmental neurotoxicity as part of revisions to 40 CFR Part 158 requirements. Today, amid mounting evidence that exposure to chemical neurotoxins may be linked to rising problems with developmental and learning disabilities, we reiterate that plea.

Although we understand that EPA has authority to go beyond the specific testing required by Part 158 without updating these requirements, information vital to the protection of children often remains absent from EPA's process for making decisions. For example, in September 1999 EPA started a “data call-in” to collect neurotoxicity data for 34 of the 891 registered pesticides. But this DCI is limited to pesticides already identified as neurotoxic. More importantly, the DCI only applies to chemicals already on the market; it fails to answer the need for neurotoxicity testing for pesticides being newly registered. Until this need is met, most new pesticides and other chemicals will continue to enter the market before any testing is done to predict toxicity to the brain and nervous system.

In summary, we urge EPA to finalize its revisions to 40 CFR Part 158 testing requirements immediately, and promptly forward them to OMB for approval. To wait for

the former until December 2000, as we understand to be the current EPA schedule, is fraught with problems.

Nearly 12 million children under age 18 suffer from a learning, developmental or behavioral disability. Many chemicals in a child's environment, including pesticides, can contribute to these problems. We remain in the dark about the vast majority of the 891 registered pesticides, in large part because they have never been tested for these effects. This ignorance has lasted long enough.

We look forward to your reply. Please direct any inquiries to Ted Schettler, M.D. at 617-536-7033.

Sincerely,

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