

Chapter 7

Chemicals, Regulations & the Environment



Scope of the Chemical Problem

Scarce information about the health effects of the approximately 80,000 chemicals in commercial use, pervasive human exposures to many toxic chemicals, and seriously inadequate regulatory oversight combine to create a global environmental threat to our children.

“Children today live in a very different environment from years ago,” said pediatrician Philip Landrigan, MD, MSc, Chair of the Department of Community and Preventative Medicine at the Mt. Sinai Medical Center. “There are new patterns of illness emerging, and many more chemicals to which children are exposed. More than 10 million products contain chemicals. Toxicity testing has not even begun to keep pace with disease. We are conducting a vast toxicological experiment on our children which will affect generations to come,” said Landrigan.¹

Toxics Release Inventory Reveals Over a Billion Pounds of Neurotoxicants Released Into Environment

The Toxics Release Inventory (TRI) reporting for the year 1997 reveals that a total of 2.58 billion pounds of toxic chemicals were released nationwide in

the United States by the large, industrial facilities required to report under TRI.²

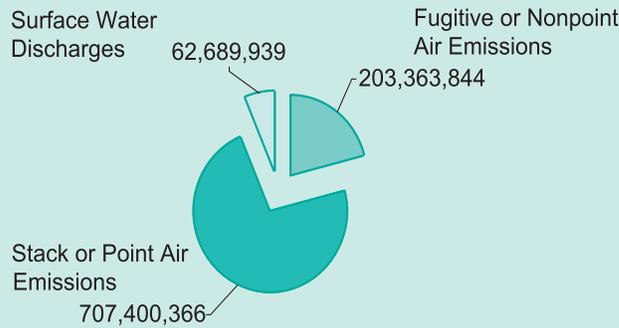
Of the 20 TRI chemicals on the list with the largest total releases, nearly three-quarters are known or suspected neurotoxicants.³

Nearly a billion pounds of these neurotoxicants were emitted by facilities on-site directly into just the air and surface water, with the potential to be inhaled, absorbed or otherwise ingested through our food and water supplies. Additional amounts were released on and off-site into underground wells, landfills and other disposal facilities, bringing the total releases to over 1.2 billion pounds.

In order of total releases, the top chemicals that are known or suspected neurotoxicants include methanol, ammonia, manganese compounds, toluene, phosphoric acid, xylene, n-hexane, chlorine, methyl ethyl ketone, carbon disulfide, dichloromethane, styrene, lead compounds, and glycol ethers. The 1997 TRI also reports metals released to the environment. Again, over half of those listed are known or

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**Toxics Release Inventory (TRI)
1997 Top 20 Chemicals Released
On-Site Releases to Air and Surface Water of
Known or Suspected Neurotoxic Chemicals**



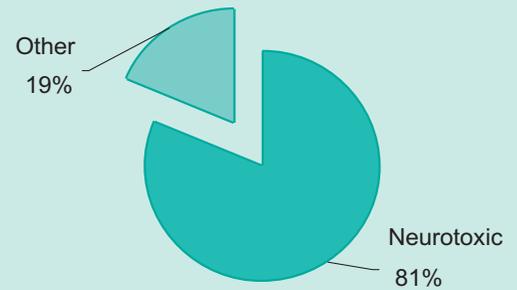
suspected to be toxic to the central nervous system.⁴ These include antimony, arsenic, barium, cadmium, lead, manganese, nickel, selenium, thallium, cobalt and mercury.⁵

Known or suspected neurotoxicants represent 81 percent of the total top 20 chemicals released to just air and surface water. They comprise 71 percent of total on-site releases to air, water and land of the top 20 chemicals.

The 1997 TRI release data include information on only about 650 chemicals, or less than 1% of the 80,000 chemicals in commercial use.⁶ They also do not provide chemical release information on all industries, including small-quantity generators

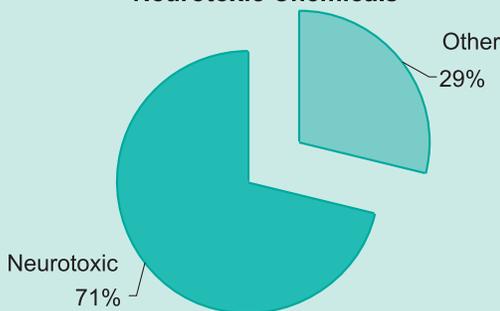
and certain industry sectors. For example, the major sources of mercury, including coal-fired power plants and incinerators, were not required to report to TRI in 1997. Electric utilities and six other industry sectors will be required to report emissions beginning with 1998 data, but incinerators and other facilities will still escape right-to-know reporting requirements. Other exemptions include sources that use less than 25,000 pounds

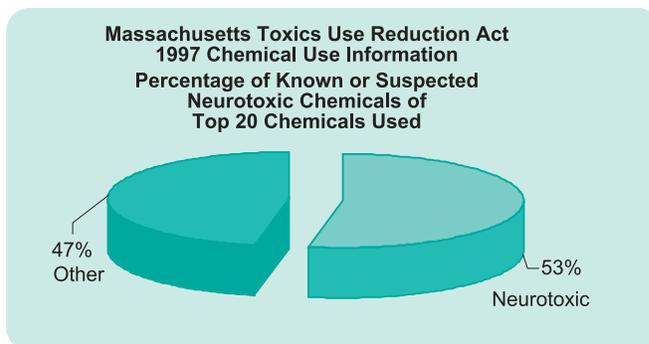
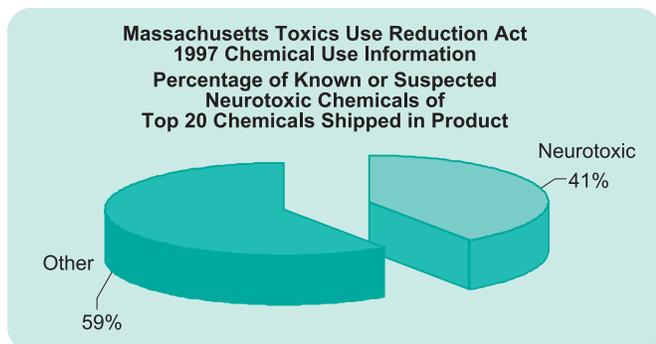
**Toxics Release Inventory (TRI)
1997 Top 20 Chemicals Released
On-Site Releases to Air and Surface Water
Percentage of Known or Suspected
Neurotoxic Chemicals**



of chemicals. This has important implications for chemicals shown in this report to exert adverse effects at extremely low levels. Due to concern about these low-level effects, EPA recently lowered the reporting thresholds for a number of persistent, bioaccumulative toxic chemicals (PBTs) including mercury, dioxins, PCBs and some pesticides.⁷ Unfortunately, lead was not included in this list. A separate rule to ensure the reporting of lead emissions has been opposed by the lead industry and delayed by Congress.⁸

**Toxics Release Inventory (TRI)
1997 Top 20 Chemicals Released
On-Site Releases to Air, Water and Land
Percentage of Known or Suspected
Neurotoxic Chemicals**





Use of Neurotoxicants High

TRI does not account for toxic chemicals incorporated into products, which may be a source of significant fetal or childhood exposures. However, toxics use information, which is only available in a few states, provides important additional information regarding potential human exposures to neurotoxicants.

An analysis of 1997 data stemming from the Massachusetts Toxics Use Reduction Act (TURA) reveals that over half of the top 20 chemicals “shipped in product,” and half of the top 20 chemicals used by industrial facilities required to report in Massachusetts, are known or suspected neurotoxicants.⁹ (“Shipped in product” includes chemicals that are incorporated into final products, such as styrene monomer into polystyrene, and also distribution of chemicals that are themselves the end product, such as solvents.) These neurotoxicants used by industrial facilities total over 500 million pounds and represent over 50 percent by weight of the top 20 chemicals used, and over 40 percent of the top 20 shipped in product in Massachusetts for the latest reporting year.¹⁰ Chemicals bound up in products may not represent a toxic threat during use, but may be a very real threat during shipping or handling, or when they are released during disposal, including incineration. Chemicals used in facilities provide opportunities for

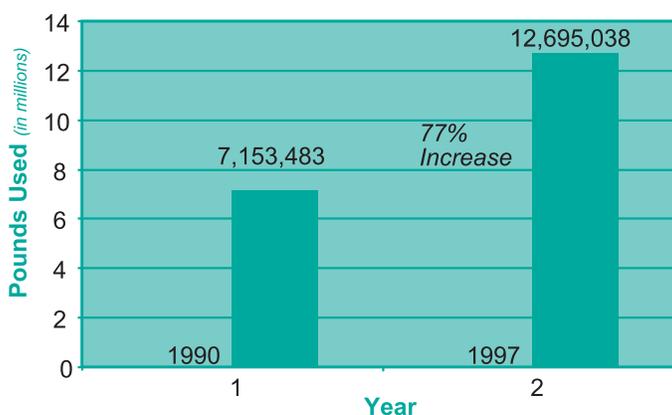
occupational exposures, sometimes at high levels, and pose additional risks to people in surrounding communities.

One of the most disturbing observations in the 1997 TURA data is that the use of lead and lead compounds has risen a dramatic 77 percent from 1990-1997 (lead use alone rose 83 percent, lead compounds 75 percent.)¹¹ Lead compounds appear in the top 20 TURA list for both chemicals shipped in product and used.¹² Products that account for some of the increase include use of lead in polyvinyl chloride (PVC) and coated wire products.

Exposures to Pesticides Pervasive

Although some pesticides were added to TRI in 1995, requiring manufacturers to report releases of listed chemicals, agricultural and other commercial users of pesticides are not required to report releases under TRI. The U.S. used approximately 1.23 billion

Massachusetts Toxics Use Reduction Act, 1997 Data
Lead Use in Massachusetts 1990-1997





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pounds of “conventional” pesticides in 1997 and a total of about 4.5 billion pounds when all types of pesticides are included, such as wood preservatives and chlorine/hypochlorites. Home pesticide use accounted for about 76 million pounds in 1997.¹³

The EPA estimates that about 23 percent of the total U.S. use of pesticides occurs in nonagricultural areas.¹⁴

The failure to include these intentional environmental pesticide releases in TRI reporting requirements impedes exposure assessment and prevention efforts. This is troublesome since children are among the most vulnerable to adverse health effects from pesticides. The 1993 National Academy of Sciences report, *Pesticides in the Diets of Infants and Children*, emphasized that children are not little adults and that, pound for pound, their chemical exposures are often greater than adults. Children are also frequently less able to detoxify substances such as pesticides, and their developing organs, including the brain, are more vulnerable. Enhanced susceptibility to adverse effects combines with relatively larger exposures to create substantially increased risks.

Children eat more fruits and vegetables than adults, on a weight-adjusted basis. Twenty million American children five and under eat an average of eight pesticides every day through food consumption.¹⁵ Thirty-seven pesticides registered for use on foods are neurotoxic

organophosphate insecticides, chemically related to more toxic nerve warfare agents developed earlier this century. One such pesticide, chlorpyrifos (commonly sold as Dursban), is among the most widely-used insecticides in homes. A national health exposure study detected chlorpyrifos residues (as the metabolite TCP) in the urine of 82% of a representative sample of American adults. A more recent study in Minnesota revealed that an even higher 92% of children had detectable levels of this metabolite in their urine.¹⁶ TCP is also a metabolite of chlorpyrifos methyl, used extensively in grain storage, so it is not possible to fully determine the source of exposure.

Seventeen organophosphates (including chlorpyrifos) are registered by EPA for “residential” uses, including in homes, on lawns, in schools, and on playgrounds. Children play in the grass where pesticides have been used and on carpets, which are toxic reservoirs for garden pesticides, lead dust, and other toxic substances.¹⁷ In a 1999 study on the distribution of pesticides and polycyclic aromatic hydrocarbons (PAHs—found in cigarette smoke and the products of fuel combustion) in house dust, 14 pesticides and 10 PAHs were detected in residential house dust collected from a typical North Carolina suburb.¹⁸ Chemicals that might biodegrade quickly outside when exposed to sunlight, water, and microorganisms remain for much longer periods of time in carpets.

Schools are another source of pesticide exposure for children. Surveys in Massachusetts and Connecticut have shown that more than 80% of schools

routinely spray pesticides. A New York study found that at least 50 active pesticide ingredients are regularly applied in the buildings and on the grounds of schools in that state. These applications expose our children to hundreds of active pesticide ingredients as well as an array of solvents and other chemicals misleadingly labeled “inert” ingredients.^{19 20} The trend is toward increasingly common exposures to organophosphates. For example, chlorpyrifos detections in urine increased more than tenfold from 1980 to 1990.²¹

Regulatory Requirements

Limited Toxicity Data

Lack of even the most basic information about the health effects of thousands of chemicals being made, sold, and emitted has serious implications for our most vulnerable population.

Information that might be used to regulate exposure to chemicals comes largely from results of toxicity testing in whole animals, cell cultures, or epidemiological studies of exposed people. However, even for those chemicals that have undergone some degree of examination, studies in both animals and humans have deficiencies.

Because of obvious ethical concerns associated with toxicity testing in humans, our regulatory system for chemicals has historically been based on toxicity testing in animals, with extrapolation of these results to estimate risks for average adult humans. (However, animal studies commonly fail to predict the particular sensitivity of the

developing human brain.) Implicit in this approach is the assumption that animal studies are relevant to humans. This assumption is widely accepted because, with some notable exceptions, test animals and humans absorb, metabolize, respond to, and excrete chemicals in substantially similar ways.

Despite validated and standardized testing protocols, toxicity testing data for individual chemicals from animal studies are woefully inadequate. For example, nearly 3,000 “high production volume” (HPV) chemicals are produced at greater than one million pounds per year. Yet, for 75 percent of these top-volume chemicals, even the most basic toxicity testing data are lacking.²² For about three-quarters of these commercial chemicals, the public record holds no data reporting the results of toxicity testing in developing animals.²³

Among the approximately 890 registered pesticidal active ingredients, EPA considers about 140 to be neurotoxic.²⁴ Between 3 and 5% of non-pesticidal chemicals have been estimated to be toxic to the nervous system.²⁵ Yet the Environmental Protection Agency (EPA) asserts that an overwhelming majority of the materials in commercial use have not been tested specifically for neurotoxic potential, making this estimate highly speculative.²⁶ Since 1991, for example, EPA has had a validated, accepted guideline for assessing a chemical’s toxicity to the nervous system in immature or developing animals. By December 1998, however, manufacturers had submitted results from this

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developmental neurotoxicity (DNT) testing for only 12 chemicals — nine pesticides and just three non-pesticide commercial chemicals.²⁷

Why Data are Lacking

Laboratory animal tests of single chemicals do not reflect the real world of mixed exposures, and commonly fail to predict the sensitivity of the developing human brain.

Animal toxicity testing data are inadequate for a number of reasons. First, the “core” or basic toxicity testing requirements necessary for registering chemicals are often inadequate. Second, “triggered” or conditional testing requirements may be incompletely or ineffectually enforced. Third, when additional tests are triggered, the testing guidelines or protocols themselves may be deficient. Finally, laboratory animal tests of single chemicals do not reflect the real world of mixed exposures, and commonly fail to predict the sensitivity of the developing human brain.

1. Inadequate “Core” or Basic Testing Requirements

The lack of toxicity data for chemicals currently on the market stems directly from the lack of requirements for testing prior to registration or manufacture of these chemicals. For most non-pesticidal and non-pharmaceutical chemicals that are regulated under the Toxic Substances Control Act (TSCA), manufacturers are required to notify the EPA of their intent to manufacture a new chemical or use an existing chemical in substantially new ways. Yet, there are no requirements for performing developmental neurotoxicity (DNT) testing of the proposed chemical. In fact, *no* pre-manufacturing toxicity testing of

any type is required under TSCA.²⁸ Instead, toxicity testing of these chemicals has largely occurred at the manufacturer’s discretion, or after manufacture and use of the chemical has already raised questions about its impact on the health of exposed persons. Though Section 6 of TSCA authorizes the EPA Administrator to take action to control risks from toxic chemicals, in its 20-year history EPA has taken Section 6 regulatory actions against only five chemicals or chemical classes.²⁹

In contrast, pesticides must undergo a battery of required toxicity tests prior to their registration, manufacture and use. Of more than 890 pesticide “active ingredients” registered with EPA, 523 are registered for use on food or feeds.³⁰ Regulation of food-use pesticides takes place under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), the Federal Food, Drug and Cosmetic Act (FFDCA), and since 1996, the Food Quality Protection Act (FQPA). The battery of required toxicology tests for registering or re-registering a pesticide used in or on food are found in the Code of Federal Regulations, last revised in 1984. Even the core requirements are alarming in their omissions. They fail to include, for example, specific tests of a chemical’s toxicity to the function of the nervous system, the immune system, or the endocrine system. EPA has repeatedly acknowledged the deficiency of these testing requirements for pesticides, particularly in terms of testing their potential effects on the nervous system, and the agency has signaled its intent to revise them. *See sidebar page 110.*

2. Inadequate “Triggered” or Conditional Testing

Registration of a new pesticide does not require pre-market testing for effects on the developing or adult brain or nervous system. EPA only recommends this kind of testing after certain conditions have been met—in other words, they are “conditionally required.” For example, EPA’s recommendation for DNT testing is contingent upon the fulfillment of certain criteria, or “triggers,” that were decided upon at a decade-old workshop sponsored by both EPA and the National Institute for Drug Abuse. From highest to lowest priority, these triggers include chemicals that are: CNS/behavioral teratogens (and structural analogues), adult neuropathic agents, adult neuroactive agents, hormonally-active compounds, and developmental toxicants that do not necessarily produce CNS effects.^{40 41} After nearly a decade, EPA’s tiered or triggered system for making recommendations for developmental neurotoxicity testing has prompted manufacturers to submit just nine complete DNT tests out of 890 registered pesticides. The explanation for this record is multifaceted.

It is not that there is a lack of accepted methods for testing developmental neurotoxicity. EPA has had validated, “accepted” guidelines for performing DNT tests since 1991. Moreover, tests using these validated DNT guidelines appear to be somewhat sensitive at detecting neurotoxicity in developing animals. Of the small number

of pesticides tested with it, seventy-eight percent (7 of 9) were found to have an effect on the developing nervous system.⁴² Rather, the triggers themselves are inadequate or not enforced. It would not be unreasonable to expect more than nine complete DNT studies over the last decade inasmuch as EPA has already identified 140 or more of existing pesticides to be neurotoxic.

In 1998 a working group of EPA scientists looked at the agency’s track record on DNT testing and concluded:

“In the past, developmental neurotoxicity study was based on criteria or triggers from both adult and developmental toxicity data and a weight-of-the-evidence review of all available data for each chemical. Such triggers were probably a reasonable place to start; however, they were based on experience with a limited number of agents. More recent information suggests that these triggers may not be inclusive enough to signal all chemicals that have the potential to produce developmental neurotoxicity. Based on the data currently available, it is impossible to predict how many neurotoxic agents will show developmental neurotoxicity, nor do we currently have sufficient information to predict how many agents that are not neurotoxic or that do not show CNS malformations will cause developmental neurotoxicity.”⁴³

It is impossible to predict how many neurotoxic agents will show developmental neurotoxicity.

More generally, the concept of tiered or “triggered” toxicity testing itself is probably flawed, at least with respect to the nervous system. Under existing EPA regulations the trigger for a “conditional” requirement that a chemical undergo

The data call-in fails to answer the need for neurotoxicity testing for new pesticides being registered.

DEVELOPMENTAL NEUROTOXICITY AS A CORE TESTING REQUIREMENT

As early as 1994, EPA recognized that its toxicology testing requirements for registering new pesticides were inadequate, particularly with respect to testing for toxicity to the nervous system. In that year, EPA finished, and asked its FIFRA Scientific Advisory Panel (SAP) to review proposed revisions to these requirements, found in part 158, subpart F, of section 40 of the Code of Federal Regulations. EPA's 1994 proposed revisions would have made it a core requirement for newly registered pesticides to be screened for neurotoxicity, including acute and subchronic testing in adult animals.³¹ The SAP generally endorsed the proposal.³² But EPA has failed repeatedly to issue a proposed rule and finalize these revisions, even after repeated public announcements of its intention to do so.^{33, 34}

In the intervening years, however, EPA's proposed revisions have expanded. In March 1998, for example, the entire FIFRA SAP recommended to EPA that it consider requiring developmental neurotoxicity (DNT) testing for *all* neurotoxic insecticides, with a portion of the panel urging a developmental neurotoxicity testing requirement for *all* pesticides, period.³⁵ An internal EPA working group then reexamined the agency's core testing requirements for pesticides, and concluded "40 CFR Part 158.340 (Subpart F) should be updated as soon as possible to include the adult and developmental neurotoxicity guidelines and to refer to the newly revised two-generation reproduction and prenatal developmental toxicity testing guidelines."³⁶ This recommendation differs from EPA's 1994 proposed revisions with the addition of DNT as a basic core requirement. An October 1998

memorandum—jointly signed by the heads of EPA's Office of Prevention, Pesticides and Toxic Substances (OPPTS), the Office of Research and Development, and the Office of Children's Health Protection—affirmed the agency's intention to accept this recommendation and expand the core requirements for all new pesticides to include DNT testing.³⁷ The memo referred to the long-delayed revisions which "are expected to go to OMB (Office of Management and Budget) in November (1998), and which are scheduled for public notice and comment in Spring 1999."³⁸ Neither step occurred.

One thing that did happen in August 1999 is that EPA announced an imminent "data call-in," or DCI, for about 140 already registered pesticides considered to be neurotoxic.



The DCI's first phase—initiated September 10, 1999—focuses on just 34 cholinesterase-inhibiting organophosphate insecticides. It requires manufacturers of these chemicals to conduct and submit tests of acute, subchronic and developmental neurotoxicity to EPA within two years.³⁹ EPA has not estimated how long it will take to complete the entire DCI for all 140 pesticides.

Although this DCI begins the process of collecting neurotoxicity data, it 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 new pesticides being 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.

basic screening for nervous system toxicity hinges on results from other, less specific, toxicological testing that generally does not involve the nervous system. Yet, as has been pointed out by Dr. Deborah Rice, an EPA neurotoxicologist, the triggers for recommending a DNT study in some cases depend on information best obtained from the DNT study itself.⁴⁴

Finally, it is critical to note that even when prior testing triggers a recommendation for DNT testing, a chemical manufacturer is under no obligation to perform such testing.⁴⁵ Thus, while 12 complete DNT studies had been submitted to EPA by December 1998, various agency scientific review committees had recommended DNT testing of an additional 26 chemicals. Though some of these recommendations date back more than six years, none of the recommended testing has ever been completed. A complete DNT study can be planned and completed in less than 2 years.

3. Deficient Guidelines for Performing Toxicity Testing

Another problem with some of EPA's current guidelines for performing toxicity testing in animals is that they omit key measures of toxicity. For example, manufacturers of organophosphate and carbamate insecticides, specifically designed to inhibit acetylcholinesterase, a key enzyme for the development and function of the nervous system, are not currently required to submit studies that will quantify the level of cholinesterase inhibition stemming from exposure to their product.

Similarly, the current DNT guideline eventually must be revised to better assess



the risks of chemical exposure to the nervous systems in children. For example, a March, 1998 panel of the EPA's Scientific Advisory Panel (SAP) reviewed the DNT guideline. It unanimously agreed that this guideline "must be further refined to develop more sensitive endpoints which are relevant to significant outcomes in humans such as learning disabilities and behavioral issues."⁴⁶ In addition, Tilson and others have identified the exposure period in the current DNT guideline as being far too short to reflect the entire vulnerable period of brain development in children.⁴⁷ The current DNT guideline requires that test animals be dosed with a chemical through the 10th postnatal day. Yet the critical period of rapid growth in the human brain, extending from the 3rd trimester through the second year of life, corresponds to the first 21-28 days of life in rats or mice—not ten days.⁴⁸

Omissions like these led the National Academy of Science to conclude in 1993 that EPA's "current testing protocols do not, for the most part, adequately address the toxicity and metabolism of pesticides

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in neonates and adolescent animals or the effects of exposure during early developmental stages and their sequelae in later life”.⁴⁹ In the first phase of its data call in for 34 registered organophosphate insecticides, EPA has taken steps to ensure that more useful neurotoxicity information is collected. For example, the agency’s recent DCI specifically requires that a comparative evaluation of cholinesterase inhibition in both adult and young animals be included. It further requires that animals in the DNT study

There is as yet no requirement that new pesticides be routinely tested for any neurotoxicity, including developmental neurotoxicity.

be dosed from day 6 of gestation through postnatal day 21, significantly beyond the 10th postnatal day required under the current guideline.⁵⁰ However, the DCI thus far applies to relatively few pesticides, many of which have already been on the market for two or three decades or more. As noted above, however, there is as yet no requirement that new pesticides be routinely tested for any neurotoxicity, including developmental neurotoxicity.

The flaws in the current DNT guideline do not make it worthless. On the contrary, the 1998 EPA review of 12 DNT studies revealed that “The developmental neurotoxicity study protocol (OPPTS 870.6300) includes unique endpoints which are not examined in any other standard toxicity testing protocol, enabling the detection of effects on nervous system development of the offspring following pre- and/or postnatal exposure.”⁵¹ And a March 1998

Scientific Advisory Panel concluded that “any pesticide that works by poisoning the nervous system” should be considered for developmental neurotoxicity testing “by the most sensitive validated methods available.”⁵² The current DNT guideline is EPA’s most sensitive validated means of doing so.

4. Laboratory Conditions Do Not Reflect the Real World, Animal Studies May Underestimate Sensitivity of Human Brain

Animal testing typically assesses the toxic effects from exposure to only one chemical at a time. This fails to provide information about the cumulative and interactive effects from exposure to multiple chemicals that often occur in real life settings.⁵³ For example, a five-year study led by Dr. Warren Porter at the University of Wisconsin, identified significant shortcomings in toxicological testing requirements currently used to register pesticides in the United States. The study suggests that combinations of commonly used agricultural chemicals, in levels typically found in groundwater, can significantly influence immune and endocrine systems as well as neurological health. Tests in laboratory animals showed that combinations of the pesticides aldicarb and atrazine, along with nitrates, each widespread contaminants of groundwater in the U.S., resulted in altered immune, endocrine, and nervous system function.⁵⁴ The study identified additional deficiencies in EPA’s core requirements for registering pesticides, including the lack of testing for low dose exposures, no testing for endocrine and

Autism Cluster Sparks Study of Environment

At the National Institutes of Health state of the science meeting on autism held in 1995, the phrase “environmental cause” was never mentioned.¹ Yet only three years later the Centers for Disease Control (CDC) and the Agency for Toxic Substances and Disease Registry began compiling information on potential environmental pollution contributors to a purported autism cluster in Brick Township, New Jersey. According to the New Jersey Bergen Record, this is “uncharted territory” for the CDC.² At the same time the CDC began studying autism and its potential environmental connections in a region around Atlanta. Why the dramatic turnaround?

One catalyst was surely Bobbie Gallagher, Brick Township resident and mother of two autistic children. Frustrated by a lack of information about the cause of her children’s disabilities, she began to look for causes in the environment. What she found were plasticizers in the water supply and a nearby Superfund site at the local landfill oozing a toxic soup of chemicals. She also discovered about 30 other children in the area who had been diagnosed with autism. It was small comfort to know that she was not alone.³ (At least 42 children have subsequently been identified with autism in Brick Township, population 76,000.)

Gallagher teamed up with the National Alliance for Autism Research (NAAR) in Princeton, New Jersey, which proposed to the CDC that five new Centers for Research in Autism Epidemiology be established. As a result of this proposal, studies in Brick Township looking at drinking water and also the Metedeconk River are now underway, as is a study in five counties around Atlanta, Georgia.

According to Dr. Eric London, medical director of NAAR, epidemiologic studies from around the world have shown a steady increase in the prevalence of autism, from around 4/10,000 in the early Eighties to about 12/10,000 in the Nineties. (The CDC fact sheet on autism spectrum disorders estimates prevalence may be as high as 20/10,000 children). Other evidence suggesting that autism may be increasing dramatically includes a recent study done by the California Department of Developmental Services released in March 1999. The study looked at pervasive developmental disorders (PDDs) from 1987 through 1998 and showed a 210 percent increase in cases entered into the autism registry during those years.⁴ ⁵ If the incidence of autism is increasing, and or clusters of autism are being discovered, an environmental influence is likely.⁶

Evidence indicating the environment as a contributing factor to autism is mounting. Studies suggest there are both genetic and environmental components to the disorder.⁷ However, definitive causes of autism remain elusive. Brick Township, New Jersey may provide some important missing pieces to the puzzle.

¹ London E. Looking at Hard Topics in Autism. Environmental Influences on Children conference, NY Academy of Medicine, May 24-25 1999.

² McEnery R. CDC Studying link between environment and autism. Bergen Country Record, November 8 1999.

³ Gallagher B. Looking at Hard Topics in Autism. Environmental Influences on Children conference, NY Academy of Medicine, May 24-25 1999.

⁴ Boyle C. Epidemiologic Perspective on Autism. Environmental Influences on Children conference, NY Academy of Medicine, May 24-25 1999.

⁵ California Department of Developmental Services. Changes in the Population of Persons with Autism and Pervasive Developmental Disorders in California's Developmental Services System: 1987 through 1998. A Report to the legislature, Mar 1999.

⁶ London E. Looking at Hard Topics in Autism. Environmental Influences on Children conference, NY Academy of Medicine, May 24-25 1999.

⁷ Hollander E. Environmental Factors in the Development of Autism. Environmental Influences on Children conference, NY Academy of Medicine, May 24-25 1999.

immune functions, and no tests of commonly found mixtures of substances that represent real-world exposures.

Neurotoxicity studies submitted to EPA, typically in adult rodents, often vastly underestimate the sensitivity of the developing human brain. For example, based on comparisons of animal and human data, animal studies of lead, mercury and PCBs predict a “safe” exposure level in humans that is 2-4 orders of magnitude (100-10,000 fold) higher than levels that actually cause effects in humans.⁵⁵ These limitations must be kept in mind as we use the results of animal testing to estimate “safe” human exposure levels.

Costs incurred by industry, as a result of proposed regulation, must be factored into decision-making.

Additional Regulatory Authority and Weaknesses

Besides TSCA and FIFRA, other major federal laws with regulatory authority over chemicals include the Clean Air Act (CAA), the Clean Water Act (CWA), and the Safe Drinking Water Act (SDWA). Each has weaknesses that

allow neurotoxic and other toxic substances to be emitted into air, drinking water, food, and onto land. For example, although the SDWA requires the EPA to set Maximum Contaminant Levels (MCLs) for certain listed chemicals, the level that is actually set to protect health is based on considerations that include costs of water treatment and also best available water treatment technology. Some standards are obsolete due to a decline in the toxic threshold for a previously recognized effect. Others are obsolete because recent evidence has revealed altogether new effects such as endocrine disruption that occur at lower levels of exposure than previously noted.⁵⁶

Except for food-use pesticides, FIFRA and TSCA require cost-benefit analyses of the impact of proposed standards, in addition to health evaluations. This means that costs incurred by industry, as a result of proposed regulation, must be factored into decision-making.⁵⁷

1 Dr. Philip Landrigan quoting Dr. Herbert Needleman. Toxics Release Inventory and Right-to-Know Conference, Washington, DC. September 9, 1997.

2 U.S. EPA, 1997 Toxics Release Inventory Public Data Release. May 13, 1999.

3 Environmental Defense Fund “Scorecard” (www.scorecard.org) health effects of chemicals - neurotoxicity-compiled from 21 databases or references including EPA, National Institute for Occupational Safety and Health’s Registry of Toxic Effects of Chemical Substances, NJ Dept. of Health Services TRI Fact Sheets and Casarett and Doull’s Toxicology, the Basic Science of Poisons, edited by C. Klaassen, M. Amdur and J. Doull, 5th Ed. Pergamon Press, NY 1996.

4 Ibid.

5 U.S. EPA. 1997 Toxics Release Inventory Public Data Release. May 13, 1999.

6 U.S. EPA, Office of Prevention, Pesticides and Toxic Substances. Endocrine Disruptor Screening and Testing Advisory Committee. Final report. Washington DC, 1998.

7 Federal Register October 29, 1999, Vol 64:209, Rules and Regulations, Page 58665-58753.

8 Personal communication with Paul Orum, Right-to-Know Network. January 31, 2000.

9 Environmental Defense Fund “Scorecard” (www.scorecard.org) health effects of chemicals - Ibid.

10 Massachusetts Department of Environmental Protection, Bureau of Waste Prevention, in conjunction with the Office of Technical Assistance for Toxics Use Reduction, Toxics Use Reduction Institute, Massachusetts Executive Office of Environmental Affairs. 1997 Toxics Use Reduction Information Release, March 23, 1999.

11 Personal communication with Liz Harriman, Massachusetts Toxics Use Reduction Institute. 12/29/99.

12 Massachusetts Department of Environmental Protection, Bureau of Waste Prevention, in conjunction with the Office of Technical Assistance for Toxics Use Reduction, Toxics Use Reduction Institute, Massachusetts Executive Office of Environmental Affairs. 1997 Toxics Use Reduction Information Release, March 23, 1999.

- 13 U.S. EPA. OPPTS. Pesticides Industry Sales and Usage, 1996 and 1997 Estimates. 733-R-99-001, November 1999.
- 14 U.S. EPA, OPPTS, Pesticides and National Strategies for Health Providers, Workshop Proceedings, April 23-24 1998. EPA 735-R-98-001, July 1998
- 15 Environmental Working Group. How Bout Them Apples? Pesticides in Children's Food 10 Years After Alar., Washington DC. 2/25/99.
- 16 U.S.EPA, Chlorpyrifos:HED Preliminary Risk Assessment for the Reregistration Eligibility Decision Document. 10/18/99.
- 17 Raloff J. Home carpets: Shoeing in toxic pollution. *Science News*, 138:86, 1990.
- 18 Lewis R, Fortune C et al. Distribution of pesticides and polycyclic aromatic hydrocarbons in house dust as a function of particle size. *Enviro Health Perspect*, 107:9, 1999.
- 19 Johnston L. Primary Exposure: Pesticides in MA Schools. MASSPIRG Education Fund, 1996.
- 20 Dr. John Wargo. New Directions for Children's Environmental Health Law and Policy. Environmental Influences on Children, Brain, Development & Behavior Conference, New York Academy of Medicine, NY, May 1999.
- 21 Schettler T, Solomon G. et al. Generations at Risk: Reproductive Health and the Environment. MIT Press, Cambridge, MA. July 1999. Pg. 225.
- 22 Roe D, Pease W., Florini K, Silbergeld E. Toxic Ignorance: The Continuing Absence of Basic Health Testing for Top-Selling Chemicals in the United States, Environmental Defense Fund, 1997.
- 23 Ibid.
- 24 Federal Register. 64(151):2945-42947, 1999.
- 25 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.
- 26 U.S. EPA, Guidelines for Neurotoxicity Risk Assessment, April 30 1998, EPA/630/R-95/001Fa.
- 27 Makris S, Raffaele K, Sette W, Seed J. A retrospective analysis of twelve developmental neurotoxicity studies submitted to the U.S. EPA Office of Prevention, Pesticides, and Toxic Substances (OPPTS), draft, 11/12/98.
- 28 Schettler T. et al. Generations at Risk: Reproductive Health and the Environment. MIT Press, Cambridge, MA. July 1999. Pg. 240.
- 29 Roe D, Pease W., Florini K, Silbergeld E. Toxic Ignorance: The Continuing Absence of Basic Health Testing for Top-Selling Chemicals in the United States, Environmental Defense Fund, 1997. Pg. 28.
- 30 U.S. EPA. OPPTS. Pesticides Industry Sales and Usage, 1996 and 1997 Estimates. 733-R-99-001, November 1999.
- 31 U.S. EPA, Changes to 40 CFR. Part 158, September 14, 1994, Presented to the FIFRA Scientific Advisory Panel (SAP), November 29-30, 1994.
- 32 U.S. EPA, Final Report from the FIFRA Scientific Advisory Panel, A Set of Scientific Issues Being Considered by the Agency in Connection with the 40 CFR Part 158 Proposed Rule: Pesticide Registration Data Requirements, December 19, 1994.
- 33 U.S. EPA, Office of Pesticide Programs Annual Report for 1994, EPA 735-R-95-001, <http://www.epa.gov/oppfead1/annual/1994>, January 1995.
- 34 U.S. EPA, Memorandum to the Administrator from Lynn Goldman, Henry Longest II, and Ramona Trovato, Update and Implementation Plans for the Child Safety Factor of the Food Quality Protection Act, October 14, 1998.
- 35 U.S. EPA, Office of Science Coordination and Policy, Final Report: Scientific Advisory Panel (SAP) March 1998 Meeting, at http://www.epa.gov/scipoly/sap/1998/march/fqpa_10x.html, page 5.
- 36 U.S. EPA, Toxicology Data Requirements for Assessing Risks of Pesticide Exposure to Children's Health, Draft Report of the Toxicology Workgroup of the EPA 10X Task Force, November 30, 1998, p. 29.
- 37 U.S. EPA, Memorandum to the Administrator from Lynn Goldman, Henry Longest II, and Ramona Trovato, Update and Implementation Plans for the Child Safety Factor of the Food Quality Protection Act, October 14, 1998.
- 38 Ibid., p. 2.
- 39 Federal Register. 64(151):42945-42947, 1999.
- 40 Claudio L, Kwa WC, Russell AL, Wallinga D. Testing methods for developmental neurotoxicity of environmental chemicals, *Toxicol. Appl. Pharmacol.*, 164. (accepted January 4, 2000).
- 41 Francis, EZ, Kimmel, CA, Rees, DC, Workshop on the qualitative and quantitative comparability of human and animal developmental neurotoxicity: Summary and Implications. *Neurotoxicol and Teratol.* 12:285-292 (1990).
- 42 U.S. EPA, Dr. Hugh Tilson, Environmental Influences on Children, Brain Development & Behavior Conference, NY Academy of Medicine, May 1999.
- 43 U.S. EPA, Draft Report of the Toxicology Workgroup of the EPA 10X Task Force, Toxicology Data Requirements for Assessing Risks of Pesticide Exposure to Children's Health, April 28, 1998, p. 12.



- 44 Statement by Dr. Deborah Rice, Seventeenth International Neurotoxicology Conference: Roundtable Discussion — Do the EPA Developmental Neurotoxicity Guidelines Detect Human Developmental Neurotoxicity?, Little Rock, AK, October 20, 1999.
- 45 Ibid., presentation by David Wallinga, M.D.
- 46 U.S. EPA Report of the FIFRA Scientific Advisory Panel Meeting, December 8, 1998, held at the Sheraton Crystal Hotel, Arlington, VA, SAP Report No. 99-01B, January 22, 1999, p. 18.
- 47 Tilson, HA, The concern for developmental neurotoxicology: Is it justified and what is being done about it? *Environ. Health Perspect.* 103(6):147-151.
- 48 Claudio L, Kwa WC, Russell AL, Wallinga D. Testing methods for developmental neurotoxicity of environmental chemicals, *Toxicol. Appl. Pharmacol.*, 164. (accepted January 4, 2000).
- 49 National Research Council, Pesticides in the Diets of Infants and Children, National Academy Press: Washington, D.C., 1993.
- 50 U.S. EPA, Letter to Registrants from Lois Rossi, Director, Special Review and Reregistration Division with multiple attachments, OPPTS, September 10, 1999.
- 51 Makris S, Raffaele K, Sette W, Seed J. A retrospective analysis of twelve developmental neurotoxicity studies submitted to the U.S. EPA Office of Prevention, Pesticides, and Toxic Substances (OPPTS), draft, 11/12/98.
- 52 U.S. EPA, Office of Science Coordination and Policy, Final Report: Scientific Advisory Panel (SAP) March 1998 Meeting, at http://www.epa.gov/scipoly/sap/1998/march/fqpa_10x.html.
- 53 Schettler T, Solomon G et al. Generations at Risk: Reproductive Health and the Environment. MIT Press, Cambridge, MA. 1999. Pgs. 21-48.
- 54 Porter WP, Jaeger JW, Carlson IH. Endocrine, immune and behavioral effects of aldicarb (carbamate), atrazine (triazine) and nitrate (fertilizer) mixtures at groundwater concentrations. *Journal of Toxicology and Industrial Health* 15:133-150, 1999.
- 55 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.
- 56 Olsen E. Think Before You Drink: The Failure of the Nation's Drinking Water System to Protect Public Health. NRDC, 1993. pg. V.
- 57 Schettler T, Solomon G et al. Generations at Risk: Reproductive Health and the Environment. MIT Press, Cambridge, MA. 1999. Pg. 236.