The National Learning and Developmental Disabilities Initiative: An Analysis of the Third CDC Biomonitoring Study

Background

The *Third National Report on Human Exposure to Environmental Chemicals (Third Report)* was released in July 2005 by the Centers for Disease Control and Prevention (CDC) of the Department of Health and Human Services. Exposures in a sample of the United States (U.S.) population for 148 environmental chemicals were determined from blood and urine specimens. This includes an additional 38 chemicals that were not measured in the *Second Report* in 2003. Measurements were taken from a random, representative sample of civilian, noninstitutionalized U.S. residents from 2001 to 2002. Not all chemicals were measured in all age categories.

This document summarizes and analyzes findings from this report on the measured chemicals or groups of chemicals that are known or suspected to cause learning or developmental disabilities. For the full CDC report, please see www.cdc.gov/exposurereport.

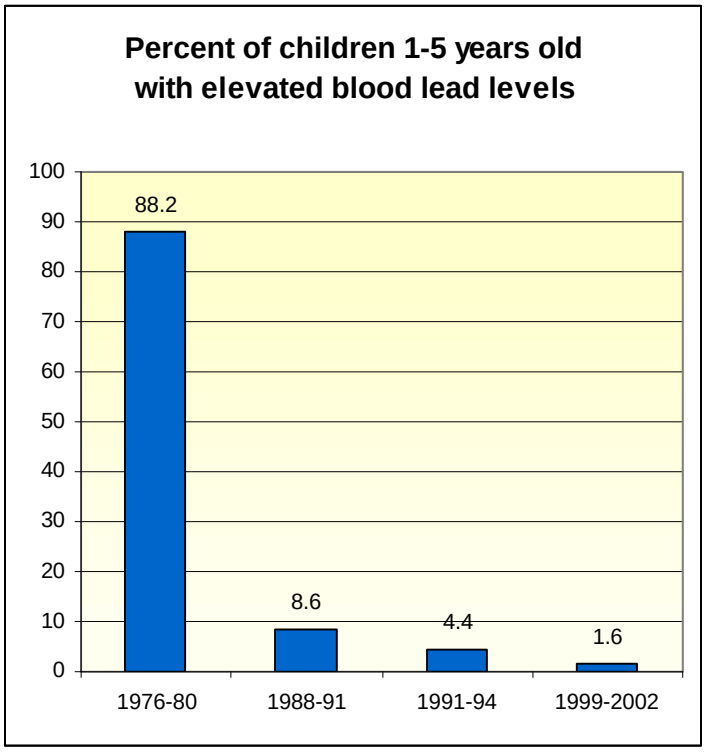
This summary was prepared by the Institute for Children's Environmental Health, the national coordinator for the Collaborative on Health and the Environment's Learning and Developmental Disabilities Initiative.

Findings

Lead

Low-level environmental lead exposure can contribute to impaired neurocognitive functioning in young children. For children, the major source of exposure is deteriorating lead-based paint in housing built before 1978 (and the contaminated dust and soil around that housing). Lead-soldered joints or lead pipes in plumbing, pewter dishes, some imported candy and some hobbies and folk remedies are other sources of exposure. Federal Environmental Protection Agency regulation of lead in drinking water, in industrial air pollution and in consumer products including paint and gasoline has dramatically decreased children's exposure, as shown in the chart below from the *Third Report*.

Even though exposures are down, elevated blood lead levels in the *Third Report*, currently defined as 10 µg/deciliter and above, were found more frequently in children who are non-Hispanic black and Mexican American (see the chart on page 5), who live in urban settings, who are from lower socioeconomic groups, who are immigrants, or who reside in housing built before 1950.



Mercury

High-level prenatal exposure to mercury may lead to a range of developmental deficits including mental retardation, limb deformities, and cerebral palsy. Lower levels of prenatal

exposure have been associated with neurocognitive abnormalities. For most people, their largest mercury exposure comes from eating fish and other seafood contaminated with methyl mercury. Environmental contamination originates in large part from the combustion of fossil fuels, primarily coal, when mercury is released and then falls into waterways and their sediments. Other sources of exposure may come from dental amalgams and vaccines that have a mercury-containing preservative known as thimerisol.

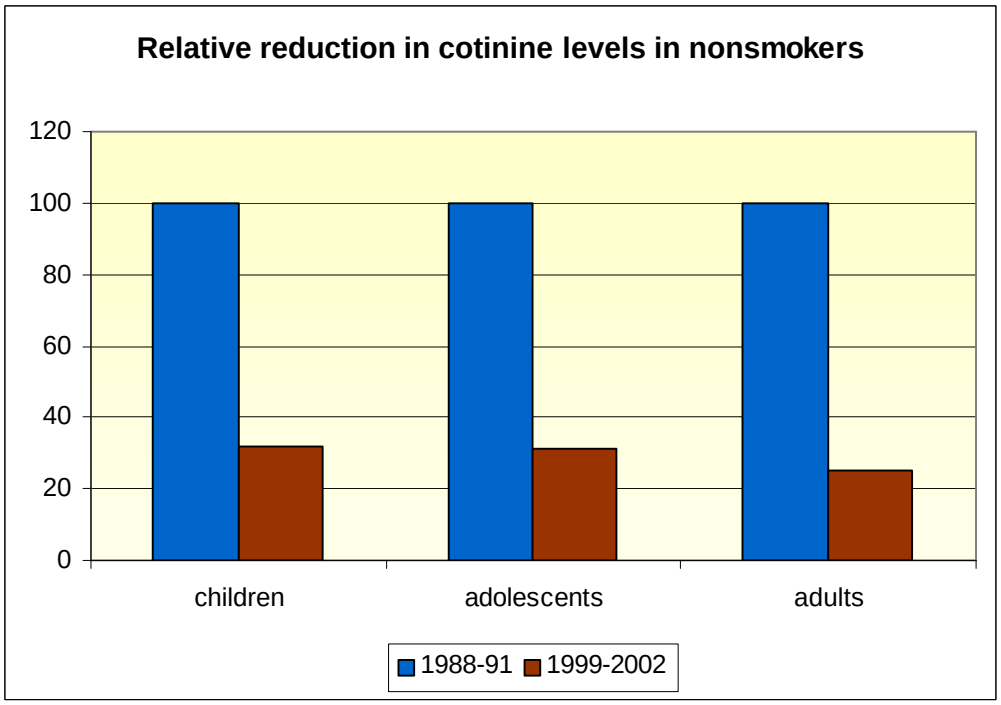
Blood mercury levels in the *Third Report* are below levels considered associated with known health effects, although 5.7% of women of childbearing age had levels within a factor of 10 of the level associated with neurological effects in children after exposure *in utero* (before birth). Non-Hispanic black women of childbearing age showed higher blood levels than non-Hispanic white or Mexican-American women (see the chart on page 5).

Tobacco Smoke (Cotinine)

While most adults have at least some control over whether they are exposed to tobacco smoke, children of smoking parents often do not. Tobacco smoking by pregnant women is associated with developmental delays in their children. Cotinine, a metabolite of nicotine, is regarded as the best biomarker of exposure to tobacco smoke in both smokers and nonsmokers. Since 1988-91, median levels of cotinine in nonsmokers have decreased significantly, as shown in the chart below. Non-Hispanic blacks show substantially higher levels of cotinine among nonsmokers than either Mexican Americans or non-Hispanic whites, as shown in the chart on page 5.

Polychlorinated dioxins, polychlorinated dibenzofurans, and dioxin-like polychlorinated biphenyls (PCBs)

Most of these chemicals are produced as unwanted by-products when waste is incinerated, when paper is bleached with chlorine, and when certain other chemicals are manufactured. Some PCBs were intentionally produced for various industrial uses until 1977 when their manufacture was banned. But because they are persistent and do not break down easily, PCBs continue to contaminate the environment and general food supply, although industrial releases of these chemicals are down 80% since the 1980s. Many soil and water sediment samples in the U.S. still



show measurable amounts of PCBs. Congenital abnormalities, prenatal growth retardation and impaired neurological development have been associated with PCBs, with more severe effects associated with higher exposures. These chemicals accumulate in foods, especially high-fat foods such as dairy products, eggs and animal fats. Breast milk is a substantial source for infants. Levels in the *Third Report* are generally below levels associated with exposures that produce health effects. Human serum levels of these chemicals have decreased by more than 80 percent since the 1980s.

Non-dioxin-like PCBs

These PCBs were widely used for fire prevention and as insulators in the manufacture of electrical transformers and capacitors, although production was banned in 1979. PCBs can contaminate animal feed, accumulate in animal fats, and migrate into food from packaging materials. PCBs are passed from mother to infant across the placenta and through breastmilk. Some PCBs can persist in the body for years after exposure. Babies born to women exposed to PCBs from contaminated fish or other sources have shown neurological abnormalities including behavioral changes, problems with motor skills, decrease in short-term memory, and cognitive impariment that may last for years. Many of the non-dioxin-like PCB congeners (chemically related PCBs) tested were below the level of detection, although improved detection limits compared to earlier reports permit comparison among demographic groups. Males tend to have slightly higher levels than females, and Mexican Americans had two-fold lower levels than either non-Hispanic blacks or non-Hispanic whites for a predominant PCB congener.

Phthalates

Phthalates are chemicals that are added to polyvinyl chloride plastics, adhesives, personal-care products and some pharmaceuticals. People are exposed through contact with items that contain phthalates or by eating food that is contaminated from phthalates in plastic packaging. Phthalates generally do not stay in the body long and do not accumulate. However, high-level exposure to phthalates is associated with fetal deaths and birth defects, including reproductive abnormalities, in lab animals.

The *Third Report*, like the *Second Report* in 2003, shows that children aged 6-11 excreted higher concentrations of most phthalate metabolites than older age groups. The reason for this finding is not known.

Organochlorine Pesticides

The following information is provided for some specific organochlorine pesticides:

Hexachlorobenzene (HCB)

HCB is an organochlorine pesticide that was used as a fungicide to pretreat grain, although its use was discontinued in 1984. HCB is also produced as a byproduct during the manufacture of certain chemicals. HCB is relatively persistent in the environment, and the main source of exposure is through foods high in fat, with small amounts of HCB also detected in outdoor air. HCB exposure is associated with damage to the liver, thyroid, nervous system, bones, kidneys, blood and immune and endocrine systems. HCB serum levels in the *Third Report* were below the level of detection.

Hexachlorocychlohexane (HCH)

HCH is another organochlorine pesticide, with lindane the common name of one HCH isomer. An isomer is a slightly different arrangement of the atoms in a substance). While lindane was discontinued for many agricultural uses in 1985, it is still approved for use in products to treat human scabies and lice. Seizures and death have resulted in children from excess application of lindane lotions and from accidental ingestion of lindane vaporizer tablets. Levels of lindane were generally below the level of detection in the *Third Repor*t, with the exception of workers involved in the manufacture, processing, application or formulation of HCH. Levels of Beta-HCH, another HCH isomer, have been declining since the 1970s.

Dichlorodiphenyltrichloroethane (DDT)

DDT was banned in the U.S. in 1973, but it is still being used in other countries. DDT is converted in the environment to other, more

stable chemical forms, including DDE and DDD. All three of these chemicals are highly persistent and can be found in soil, air and water sediments. DDE concentrates in fatty tissues and can cause a range of toxic effects, including neurologic developmental disorders, fetal death and prenatal growth retardation. Food is the primary source of exposure for people. While the estimated food intake of DDT in the U.S. has decreased since the 1950s, food imported to the U.S. from other countries may contain DDT or DDE residues.

Serum levels of DDT and DDE in the *Third Report* appear to be substantially lower than in a report from 1976-80, although direct comparisons are not possible between the two groups. Serum levels in Mexican Americans were approximately three times as high as in non-Hispanic whites, while levels in non-Hispanic blacks were about twice that of non-Hispanic whites (see the chart on page 5).

Chlordane and Heptachlor
These organochlorine chemicals were once used as pesticides in the U.S. Chlordane use was used to kill insects in crops, lawn, and buildings (termites), but its use was discontinued in 1988. Heptachlor is restricted to limited applications for controlling fire ants. Both chemicals are highly persistent in the environment and the body. Fatty foods are the most likely source of exposure, but indoor air is also a source, even 10 years after a termite treatment with chlordane. Animals exposed to these chemicals before birth or while nursing have shown behavioral (motor activity) or reproductive effects. In the *Third Report*, levels of a heptachlor metabolite were tenfold lower at the 90th percentile than in a cohort of women studied in 1963-67.

Mirex
Mirex was formerly used in the southern U.S. to control fire ants and as a flame-retardant additive. Although it has not been produced or used since 1977, it is environmentally persistent. It accumulates in fatty tissues in the body and is not metabolized, and the most likely source of exposure is food, especially from contaminated lakes and rivers. Mirex is associated with harmful effects on the nervous system, skin, liver and male reproductive system. Levels of mirex in serum from participants 12 years and older were generally below the level of detection in the *Third Report*. Younger subjects were not tested for mirex.

Endrin
Production and use of endrin, an organochlorine pesticide, was discontinued in 1986. The main sources of exposure are foods produced in countries where endrin is still used and living or working near hazardous waste sites where endrin was produced. Endrin is persistent in the environment, but it does not accumulate in body tissues. Instead it is metabolized and excreted relatively quickly. Exposure to endrin can cause harmful effects including death and severe central nervous system injury with high exposures. Birth defects have been reported in some animal studies. In the *Third Report*, serum levels were generally below the level of detection.

Organophosphate Pesticides

Organophosphate pesticides account for about half of all insecticides used in the United States. This group includes malathion, chlorpyrifos, parathion and diazinon, among others. Although organophosphates are still used for insect control on many food crops, most residential uses are being phased out. Exposure may be through ingestion, inhalation, or absorption through skin. Farmworkers, pesticide manufacturers and children of these workers are at higher risk of exposure. Recent research has linked some of these pesticides to a small but significant reduction in head circumference in infants, an indication of smaller brain size. Animal studies have found adverse effects on neurodevelopment from exposure *in utero* and in the neonatal period. The levels in subjects six to 11 years of age (the youngest group tested) were higher than in older groups in the *Second Report* in 2003, and this finding seems to be repeated in the *Third Report*.

Atrazine Mercapturate

Atrazine is one of the most widely used herbicides in the U.S., mostly on corn, although

it is also used on other crops and on golf courses and lawns in some regions. It has been associated with adverse developmental and reproductive outcomes in animals. Exposure occurs from consuming food or water containing atrazine or from applying it to lawns and crops. Atrazine is rapidly eliminated from the body. In the *Third Report*, urinary levels of the atrazine metabolite atrazine mercapturate were below levels of detection.

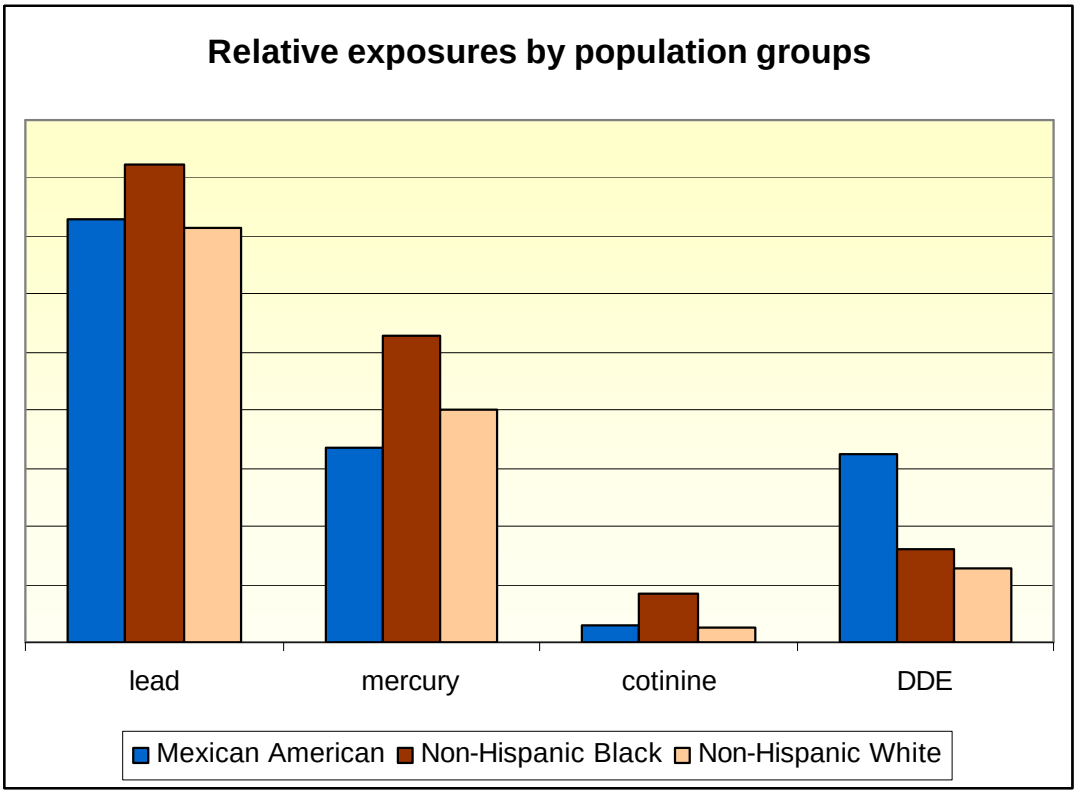
Conclusions

Regulation of chemicals such as lead, DDT and heptachlor has produced dramatic reductions in exposures, although further work is needed to eliminate the remaining preventable exposures among pregnant women and young children. A similar drop in exposure of nonsmokers to tobacco smoke will lead to progress in reducing the harmful effects of tobacco in young children.

The continued exposure to several banned chemicals, such as PCBs and DDT, indicates how dangerous such highly persistent chemicals can be. While stopping production is one part of the solution to exposure, removing existing toxic chemicals from the environment where they exist in hazardous waste sites or highly contaminated sediments in water bodies is also imperative.

The inability to detect exposures to the organochlorine pesticides HCB, HCH, mirex and endrin as well as atrazine in the *Third Report* is good news, although because endrin and atrazine are metabolized quickly, lack of detectable levels does not necessarily indicate a lack of exposure or that they are not potentially associated with health effects.

Disparities among racial/ethnic groups are a concern (see the chart below). The reasons for disparities in exposures to lead, mercury, cotinine (from tobacco smoke) and DDE (a DDT metabolite) need to be determined and addressed. Likewise, the reasons for higher phthalate and organophosphate pesticide detection in children than in adults should be investigated. Given how ubiquitous these chemicals are and that children are more biologically vulnerable to toxic



exposures, evidence of higher concentrations of these chemical metabolites also suggests that we need to develop and use less toxic alternatives as a preventive approach.

Overall, much more effort needs to go toward studying and preventing these exposures. While action by parents or individuals can prevent some of these exposures, further public policy shifts are needed to protect children from many of these chemicals.

Please see www.iceh.org/LDDI.html for more information about the Learning and Developmental Disabilities Initiative.