Environmental Toxicants and Developmental Disabilities

A Challenge for Psychologists

Susan M. Koger — *Willamette University*
Ted Schettler — *Boston Medical Center*
Bernard Weiss — *University of Rochester Medical Center*

Developmental, learning, and behavioral disabilities are a significant public health problem. Environmental chemicals can interfere with brain development during critical periods, thereby impacting sensory, motor, and cognitive function. Because regulation in the United States is based on limited testing protocols and essentially requires proof of harm rather than proof of lack of harm, some undefined fraction of these disabilities may reflect adverse impacts of this "vast toxicological experiment" (H. L. Needleman, as quoted in B. Weiss & P. J. Landrigan, 2000, p. 373). Yet the hazards of environmental pollutants are inherently preventable. Psychologists can help prevent developmental disabilities by mobilizing and affecting public policy, educating and informing consumers, contributing to interdisciplinary research efforts, and taking action within their own homes and communities to reduce the toxic threat to children.

Developmental, learning, and behavioral disabilities[1] are a significant public health problem and appear to be increasing in prevalence (Goldman & Koduru, 2000; Schettler, Stein, Reich, & Valenti, 2000). According to the Centers for Disease Control and Prevention (2004), approximately 17% of U.S. children (i.e., 12 million individuals) under the age of 18 are affected by one or more developmental disabilities that have an impact on cognitive function, language or learning ability, emotional state, sensory and motor function, a variety of behaviors, or physical growth. By themselves, learning disabilities expressed as reading and writing difficulties and other disorders of academic skills affect nearly 4 million school-age children, and at least 20% of these children suffer from attentional problems (National Institute of Mental Health, 1999). Serious disorders like mental retardation or cerebral palsy affect 2% of children in the United States (Centers for Disease Control and Prevention, 2004).

Developmental disabilities are of particular concern to the psychological community not only because of the associated educational and therapeutic challenges but also because of their indirect effects. Although learning disabilities are generally thought to affect children and adolescents, they pose lifelong and widespread difficulties. It is more difficult for individuals with learning disabilities to hold employment, learn new skills, and work with others (Alexander, 1996). The learning disabled are often socially alienated and may be at a greater risk for suicide than others (McBride & Siegel, 1997). They may be more likely to enter the criminal justice system for delinquency and adult criminal behavior (Dickman, 1996; Eggleston, 1996), possibly because of academic difficulties that lead many to drop out of school (McGee, 1996). Similar consequences including vocational difficulties and mood and anxiety disorders are associated with attention-deficit/hyperactivity disorder (American Psychiatric Association, 2000; Fletcher & Shaywitz, 1996). In general, developmental disabilities frequently co-occur with anxiety disorders and a variety of psychiatric conditions affecting emotion, mood, and behavior (e.g., Arthur, 2003; Dekker & Koot, 2003; Ollendick, Oswald, & Ollendick, 1993). The developmentally disabled may also manifest greater medical morbidity and be more likely to manifest mental or neurodegenerative disorders as adults or in old age (Davidson et al., 2003).

In addition, the families of children with disabilities face serious emotional and financial costs (Wilton & Renaut, 1986). For example, having a disabled child can seriously strain family dynamics (Dyson, 1996), motivating the family to seek professional counseling; parents, especially mothers, often present with depression (Smith, Innocenti, Boyce, & Smith, 1993); and utilizing special educational and therapeutic services (e.g., psychological,

Susan M. Koger, Department of Psychology, Willamette University; Ted Schettler, Science and Environmental Health Network, Boston Medical Center; Bernard Weiss, Department of Environmental Medicine, University of Rochester Medical Center.

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Correspondence concerning this article should be addressed to Susan M. Koger, Department of Psychology, Willamette University, 900 State Street, Salem, OR 97301. E-mail: skoger@willamette.edu

[1] Throughout this article, the generic term *developmental disabilities* refers collectively to these three categories of disabilities.

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speech, language, motor) can require parents to miss work, exacerbating their emotional and financial stress.

Although the origin and manifestation of developmental disabilities reflect complex interactions among a variety of factors, including genetics, nutrition, and social influences, the case is building for a significant role of environmental toxicants (Bellinger & Adams, 2001; Costa, Aschner, Vitalone, Syversen, & Soldin, 2004; Masters, 2001; Needleman & Landrigan, 1994; Schettler et al., 2000; Weiss, 2000; Weiss & Landrigan, 2000). It is estimated that anywhere between 3% and 25% of developmental defects result from neurotoxic environmental exposures, either singly or in combination with other environmental factors such as maternal lifestyle choices (diet, tobacco, alcohol; Landrigan, Kimmel, Correa, & Eskenazi, 2004), occupation (a source of workplace neurotoxicants such as solvents), and economic circumstances. As one of us noted more than 20 years ago (Weiss, 1983), the impact of neurotoxicant exposure requires the attention of the psychological community, as psychologists are particularly well-trained to investigate the psychological and neuropsychological effects of pesticides and other toxicants (see also Fein, Schwartz, Jacobson, & Jacobson, 1983; Mearns, Dunn, & Lees-Haley, 1994).

Environmental Toxicants

It is well established that the developing nervous system is particularly vulnerable to environmental insults (e.g., Goldman, 1995; Rodier, 1995; Spyker, 1975; Weiss, 2000). Brain development begins early in the fetus, as cell division, migration, differentiation, and synapse formation proceed in well-ordered sequences with variable timing in different portions of the brain. Neural migration and myelination continue through infancy and the second year of life, and overall brain development continues into adolescence (Gogtay et al., 2004). Apoptosis (programmed cell death) and pruning of synapses further refine brain architecture. Many substances easily penetrate the placenta during prenatal development, and because the fetal blood–brain barrier is not fully formed, toxicants can enter and impact brain development through direct toxicity or through interference with a variety of cell-signaling mechanisms, including the endocrine system (Colborn, 2004; Weiss, Amler, & Amler, 2004).

Young children often encounter higher levels of toxicants than adults because of what Weiss (2000) called the "spatial ecology" of childhood. Because young children spend considerable time on floors, they stir up and breathe dust and residues, and their contact with dust may be 10 times greater than that of adults. Children naturally explore their environments by putting contaminated items into their mouths. In addition, the fact that children ingest relatively more juice, fruit, and water than adults gives them increased exposure to pesticide residues and contaminants in those substances (National Research Council, 1993).

Significant effects of environmental chemicals on various components of brain development and function are well documented in both humans and nonhumans (see Schettler et al., 2000, for a thorough review; see Table 1 for a synopsis of some of the health effects of toxicants). Heavy metals such as mercury, lead, and manganese, commercial chemicals such as some pesticides and PCBs, and inadvertent contaminants like dioxins are all of concern because of their direct neurotoxic potential as well as their ability to interfere with biochemical mechanisms. Neurotoxicants also target developmental processes, including cell replication, cell differentiation, and neural communication (Porterfield, 2000; Rice & Barone, 2000; Rodier, 1995). Because normal neurologic development is based on exquisitely choreographed sequences of cellular events, such chemical disruption has been related to disabilities, including mental retardation (Myers & Davidson, 2000; Schroeder, 2000), cerebral palsy, epilepsy, and deficits of learning, memory, and attention (Myers & Davidson, 2000; Porterfield, 2000), attention-deficit/hyperactivity disorder (Rice, 2000), and possibly even autism (London & Etzel, 2000).

Lead

Lead provides an important case study, as it represents the best-studied neurodevelopmental toxicant, and regulatory decisions have dramatically reduced exposures. Most people are at least somewhat aware of the risks associated with lead exposure as a result of intensive public awareness campaigns (e.g., see Figure 1) and governmental interventions to remove lead from gasoline and paint in the 1970s. Yet despite this legislation, exposures continue: Lead is still used in many industrial, commercial, and military products and applications; deterioration or renovation of homes and complexes constructed prior to 1980 can result in exposure to contaminated paint chips or dust; and lead-tainted soils can persist as long as 2,000 years (Nadakavukaren, 2000). Contaminated house dust is a significant source of lead exposure, particularly for children in urban communities (Haynes, Lanphear, Tohn, Farr, & Wioads, 2002).

Regulations governing lead exposure were largely based on an understanding of the dangers of acute poisonings, which have been recognized as a public health issue since antiquity (Needleman, 1980). Chronic or low-dose exposure was considered benign until the landmark observation of an association between higher lead levels in teeth and difficulty following classroom instruction, unruly behavior, greater distractibility, and reduced auditory and verbal processing, attention, and IQ scores, particularly on verbal components (Needleman et al., 1979). These results demonstrated that intellectual impairments and problematic behaviors could result from lead levels that were "regarded as normal" at the time (Rice et al., 1996, p. 205).

Needleman (1980) presaged that "it seems likely that the standard for acceptable exposure to lead, given the history of scientific progress, will require further downward revision as new information is gathered and evaluated" (p. 45). In fact, it is now generally held that there is no "safe" level of lead exposure. A recent study demonstrated that concentrations falling below levels of concern set by the Centers for Disease Control and the World Health Organization were found to inversely correlate with

Table 1
Toxicants and Their Health Effects

Toxicant	Study	Health effects/characteristics
Metals		
Cadmium	H, A	Learning disabilities Decreased IQ Motor dysfunction Hyperactivity Hypoactivity
Lead	H, A	Learning disabilities IQ deficit Attention deficit Impulsivity Violence Hyperactivity Aggression
Manganese	H, A	Brain damage Motor dysfunction Compulsive behavior Memory impairment Hyperactivity Learning disabilities Attention deficit
Mercury	H, A	Visual impairment Learning disabilities Attention deficit Motor dysfunction Memory impairment (minimal) *At higher levels:* Smaller brain size; cellular distortions in brain, mental retardation
Solvents		
Ethanol (alcohol)	H, A	Learning disabilities Attention deficits Memory impairment Behavioral disorders Eating and sleeping disorders Lower brain weight Craniofacial, limb and cardiovascular abnormalities associated with various growth and developmental delays Mental retardation
Styrene	A	Decreased activity Decreased avoidance behavior *In conjunction with dietary protein deficiency:* Lower brain weight-hyperactivity
Toluene	H, A	Learning disabilities Speech deficits Motor dysfunction Craniofacial abnormalities
Trichloroethylene	A	Increased exploratory behavior Hyperactivity
Xylene	A	Motor dysfunction Learning disabilities Memory impairment Decreased brain weight
Pesticides		
Organochlorines		
DDT	A	Hyperactivity
Mixture	H	Decreased stamina Decreased coordination Decreased memory Decreased ability to draw familiar objects
Organophosphates (including DFP, chlorpyrifos [Dursban] diazinon)	A	Developmental delays Hyperactivity Behavioral disorders Motor dysfunction
Pyrethroids (including bioallethrin, deltamethrin, cypermethrin)	A	Hyperactivity
Other		
Nicotine	H, A	Hyperactivity Learning disabilities Developmental delays in cognitive functions
Dioxins	H, A	Learning disabilities
PCBs	H, A	Learning disabilities Attention deficit Memory impairment Hyperactivity Psychomotor dysfunction
Fluoride	A H	Hyperactivity Decreased IQ (ecologic studies)

Note. Learning disabilities include dysfunctions in listening, speaking, reading, writing, spelling, or calculations. Only neurodevelopmental, learning, or behavioral effects of toxicants, or physicol impairments that lead to them, are listed. H = human studies; A = animal studies; DFP = diisopropylfluorophosphate. From *In Harm's Way: Toxic Threats to Child Development* [Report, p. 94], by T. Schettler, J. Stein, F. Reich, and M. Valenti, 2000, Cambridge, MA: Greater Boston Physicians for Social Responsibility. Copyright 2000 by Greater Boston Physicians for Social Responsibility. Adapted with permission.

Figure 1
Campaigns Such as This Poster From the U.S. Environmental Protection Agency Have Increased Public Awareness of the Developmental Hazards of Lead

intellectual function (Canfield et al., 2003). Lead exposure is associated with a variety of behavioral and cognitive problems in children, including antisocial and delinquent behavior (Needleman, McFarland, Ness, Fienberg, & Tobin, 2002; Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996), violence (Masters, 1997), criminality and unwed pregnancy (Nevin, 2000) and is known to produce deficits in motor coordination, intelligence, language function, learning, memory, attention, and executive function, as well as mental retardation at higher doses (Bellinger & Adams, 2001; Bellinger, Stiles, & Needleman, 1992; Campbell, Needleman, Riess, & Tobin, 2000; Lanphear, Dietrich, Auinger, & Cox, 2000; Needleman, 2004). One study suggests that lead exposure may even be a risk factor for schizophrenia spectrum disorder (Opler et al., 2004).

Mercury

Methylmercury (one of the organic forms of mercury) can influence both pre- and postnatal brain development depending on the timing and level of exposure. Power plants that burn fossil fuels, medical waste incinerators, other industrial processes, and volcanic activity emit elemental mercury vapor into the atmosphere, where it becomes ionized and returned to earth in rainfall and subsequently deposited in oceans, lakes, and streams (Clarkson, Magos, & Myers, 2003; Mason, Fitzgerald, & Morel, 1994). Microorganisms in aquatic sediments convert the inorganic mercury into the methyl form, which then ascends the food chain and reaches its highest levels in predatory fish and birds and mammals (sea and land) that eat the contaminated fish. Thus, seafood constitutes the primary source of methylmercury exposure in humans. At high doses in adults, methylmercury acts directly in the central nervous system by destroying or damaging nerve cells. In the developing brain, it interferes with cell proliferation and migration (Myers & Davidson, 2000). In severe poisoning cases in children, mental retardation or cerebral palsy may result (Bellinger & Adams, 2001; Myers & Davidson, 2000). Its less obvious effects include disordered cognitive development as reflected by lowered IQ scores, impairments of memory and attention, and coordination deficits.

Recent studies have assessed the degree of risk to brain development posed by methylmercury exposure in communities that consume large quantities of seafood. Two of these examined large cohorts in different locations. Both attempted to determine the relationship between maternal exposure to methylmercury on the basis of hair or cord blood levels and offspring development. Subtle adverse effects including performance on the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1976) were reported by Grandjean and colleagues (1997), who studied a population in the Faroe Islands. The other study, based on a population in the Seychelle Islands of the Indian Ocean, has observed no significant adverse effects in children up to 107 months

of age as related to maternal hair levels of mercury (Myers et al., 2003). Because the cohort studied by Grandjean et al. was exposed to PCBs and other halogenated organic contaminants in addition to methylmercury because of its consumption of whale meat and blubber, the results may partly reflect effects arising from combinations or interactions of these contaminants.

Regardless of the source of the different findings, the National Academy of Sciences used the results obtained by Grandjean et al. (1997) to recommend protective exposure levels. These were translated by the Environmental Protection Agency (EPA) into a maternal reference dose of 0.1 μg/kg body weight per day, an exposure level below which no increased risk of fetal effects is likely. To meet this criterion, The Food and Drug Administration and the EPA are currently "advising women who may become pregnant, pregnant women, nursing mothers, and young children to avoid some types of fish and eat fish and shellfish that are lower in mercury," but acknowledge that "nearly all fish and shellfish contain traces of mercury" (U.S. EPA, 2004, pp. 1, 2).

As Davidson, Myers, and Weiss (2004) have pointed out, policy issues regarding mercury are complex because there are contradictions in the scientific literature concerning the exposure level at which effects can be detected and concerns regarding balancing the relative risks of mercury exposure with the nutritional benefits of fish consumption. What is important for psychologists, however, is not which mercury exposure standard is promulgated but the fact that, as with lead, the criteria for such standards derive from neurobehavioral measures. Accordingly, psychologists have the specialized knowledge and opportunity to influence public policy on these issues.

Similarly, exposure standards for commercial chemicals such as pesticides and plastics and contaminants like dioxins are subject to methodological difficulties, incomplete data, and regulatory and legislative inertia. However, it is clear that these substances alter hormone or neurotransmitter function and may interfere with sexual differentiation of the brain (Hojo et al., 2002; Zareba et al., 2002), as well as affect cognitive abilities such as memory (Guillette, Meza, Aquilar, Soto, & Garcia, 1998).

Pesticides

Although they play an important role in food production and the control of vector-borne diseases, pesticides are toxic by design and are produced and released intentionally into the environment (Colborn, 1995; Weiss et al., 2004). Whether pesticides are meant to kill weeds (herbicides), insects (insecticides), rodents (rodenticides), or any other organism, humans are not impervious to the properties that render them toxic. In fact, many insecticides act as direct neurotoxicants, as they are designed to disable the nervous systems of insects through mechanisms directly relevant to human physiology (U.S. Congress, Office of Technology Assessment, 1990). Many insecticides function by affecting the metabolism of neurotransmitters, particularly acetylcholine, or by interfering with endocrine function (Mearns et al., 1994; Needleman, 1995; Weiss et al., 2004). Because of the ubiquity of such chemicals, people are exposed to them through contamination of drinking water, air, and house dust, residues on fruits and vegetables, and consumption of fatty tissue of exposed animals and their byproducts (i.e., meat, fish, eggs, and dairy products). One study reported that 98% of families used pesticides at least once per year, with two thirds using them more than five times per year. Eighty percent used pesticides during pregnancy, and 70% used them during the first six months of a child's life (Davis, Brownson, & Garcia, 1992).

Several recent studies have demonstrated learning and behavioral changes in rodents exposed to organophosphate pesticides (e.g., Icenogle et al., 2004; Lazarini, Lima, Guedes, & Bernardi, 2004; Ricceri et al., 2003; Sanchez-Santed, Canadas, Flores, Lopez-Grancha, & Cardona, 2004). This type of pesticide is commonly used for home, lawn, and garden pest control as well as on the commercial food supply. Schettler et al. (2000) reviewed the evidence concerning several different classes of pesticides (i.e., organophosphate, organochlorine, pyrethroid, pyrethrin) that produce neurodevelopmental toxicity in animals exposed during vulnerable neonatal periods (e.g., Day 10 of life). They concluded that "small exposures during those periods of susceptibility permanently alter brain neuroreceptor levels and cause hyperactivity in the animals as adults" (Schettler et al., 2000, p. 85; see also Ahlbom, Fredriksson, & Eriksson, 1995). Because of the parallels in brain development between rodents at postnatal Day 10 and humans during the last trimester of pregnancy, there is the potential for similar effects in the offspring of women exposed during pregnancy.

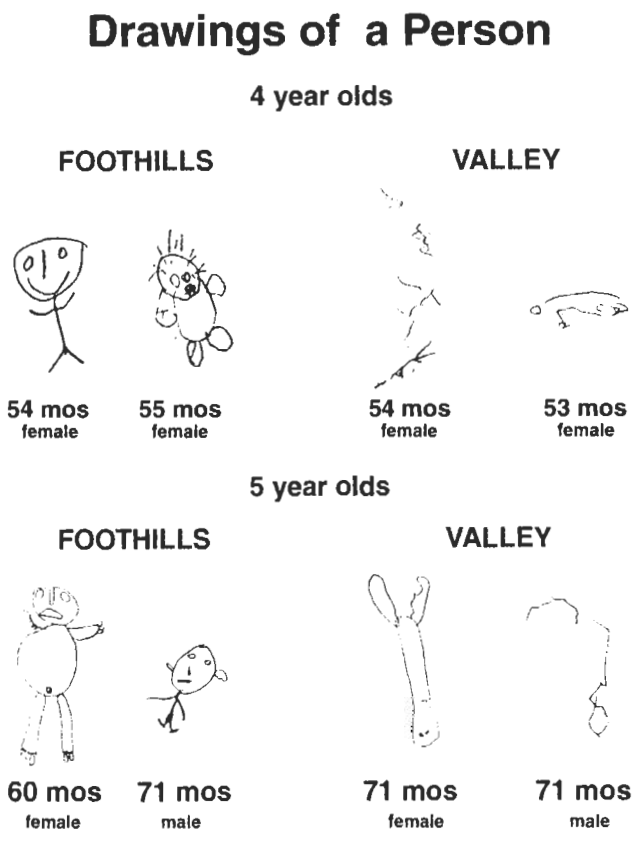
Very little research has directly assessed the developmental neurotoxicity of pesticides in humans. However, in one arresting example, Guillette and colleagues (1998) observed impairments in memory, social interaction, creativity, and motor skills in a population of Mexican children exposed to pesticides relative to a comparable group who lived in an untreated area. Most dramatically, the exposed children exhibited marked impairments in the "draw a person" task, which is generally considered a nonverbal measure of cognitive ability but may also indicate visuomotor incoordination (see Figure 2).

Regulatory Issues

Unfortunately, regulatory efforts to reduce exposure to toxicants are often reactionary rather than preventive and frequently lag behind scientific evidence for possible or probable harm. For example, it is ironic that although the toxicity of lead has been recognized for millennia, it remains "one of the most important pediatric diseases and is a major cause of behavior disorders and school failure" (Needleman & Landrigan, 1994, p. 79).

Twenty years ago the National Academy of Sciences recognized that only a small minority of the chemicals registered with the EPA received even minimal toxicity evaluation (Denison & Florini, 2003; National Academy of Sciences, 1984); that situation remains largely unchanged today. Approximately 85,000 chemicals are currently registered with the EPA; of those, more than 2,800 chemicals

Figure 2
Drawings of a Person by Four- and Five-Year-Old Mexican Yaqui Indian Children in a Study of the Effects of Pesticide Exposure on Neurologic Development



Note. Children in the agricultural regions of the valley were routinely exposed to pesticides, whereas pesticide use is avoided in the foothills. From "An Anthropological Approach to the Evaluation of Preschool Children Exposed to Pesticides in Mexico," by E. A. Guillette, M. M. Meza, M. G. Aquilar, A. D. Soto, and I. E. Garcia, 1998, *Environmental Health Perspectives, 106*, p. 351. Reproduced with permission from *Environmental Health Perspectives*.

are produced or imported in the United States at greater than 1 million pounds per year. For 43% of those high-production-volume chemicals, "no basic human health nor environmental toxicity [information] is publicly available. [Further], a full set of basic toxicity information [i.e., including evaluation of possible developmental effects] is available for only 7% of these chemicals" (U.S. EPA, 1998, p. 2). For example, of the 140 currently registered pesticides that are known neurotoxicants, only 12 have been specifically tested for potential impacts on children's development as evaluated by studies in laboratory animals.

In general, U.S. regulatory guidelines continue to be based on the premise of "innocent until proven guilty" when it comes to overseeing the production, use, and disposal of industrial and household chemicals. Yet an emphasis on scientific proof of harm may skew the balance between safety and economic concerns (Durnil, 1999; Schettler, 2001):

> Whether or not unassailable scientific proof of acute or chronic damage is universally accepted . . . the focus must be on preventing the generation of persistent toxic substances [e.g., dioxin, chlorinated industrial chemicals, and certain pesticides] in the first place, rather than trying to control their use, release, and disposal after they are produced. (International Joint Commission, 1992, pp. 5, 25)

Sweden has taken a leadership role in the precautionary regulation of chemicals, basing rules on suspected rather than proven human health and environmental impacts (Swedish Government Chemicals Policy Committee, 1997; see also Grandjean, 2004, for a discussion of the precautionary principle). The Swedish plan states that new products should be largely free from

- persistent and bioaccumulative substances;
- polyvinyl chloride (PVC) plastics and substances that are endocrine disruptive;
- mercury, cadmium, lead, and their compounds, and these and other metals should be used in such a way that they are not released into the environment to a degree that causes harm to the environment or human health.

The United States has signed an international agreement (the Stockholm Convention on Persistent Organic Pollutants) regarding regulation of 12 of the most troublesome persistent, bioaccumulative toxicants but has yet to ratify it.

Weiss (2001) has argued that the widespread production, use, and disposal of untested or inadequately tested chemicals essentially constitute large-scale human experimentation, but the ethical guidelines that generally mitigate potential adverse impacts to participants in clinical trials are not being implemented. For example, the Belmont Report (National Commission for the Protection of Human Subjects, 1979) requires (a) an acknowledgment of who will receive the benefits and who bears the burdens of the research (justice), (b) that the research maximize the potential benefits to the participants and minimize risk of harm (beneficence), and (c) that participants are involved on a voluntary basis and with adequate information to provide informed consent (respect for persons). Moreover, pharmaceuticals are required to demonstrate evidence of safety (rather than the absence of proof of harm) prior to marketing. These principles are not applied to chemicals dispersed into the environment.

Establishing Cause and Effect: A Research Nightmare

Some of the problems in prescribing exposure standards legitimately follow from methodological limitations. Establishing causal relationships between exposure to environmental chemicals and manifestations of disabilities in humans is a challenging task, particularly when the exposures occur at relatively low doses (Nadakavukaren, 2000; Schettler et al., 2000; Weiss, 1992, 1997).

First, humans are exposed to a plethora of substances, many of which have known toxic and neurotoxic or endocrine-disrupting properties; these include lead and other heavy metals; PCBs, dioxins, and other polycyclic aromatic hydrocarbons; pesticides; brominated flame retardants; and surfactants, cleansers, solvents, plasticizers, dyes, food additives, water purification byproducts, industrial process chemicals, pharmaceuticals, and contaminants from human waste (e.g., Muir & Zegarac, 2001). The ideal of an unexposed control group with which to compare exposed populations cannot be achieved.

Second, because humans are routinely exposed to a "toxic soup" of chemicals rather than to individual compounds that may not reveal hazards when tested independently, interactive effects of toxicants may remain unidentified. For example, the combination of two widely used pesticides, maneb and paraquat, induced effects on the nigrostriatal dopaminergic system in mice corresponding to the pathology associated with Parkinson's disease; these effects were not observed when the substances were administered individually (Thiruchelvam, Brockel, Richfield, Baggs, & Cory-Slechta, 2000; Thiruchelvam, Richfield, Baggs, Tank, & Cory-Slechta, 2000).

Third, human behavioral and cognitive effects from environmental exposures are generally subtle and difficult to diagnose, so they often go unnoticed. In the words of a former director of the National Institute of Environmental Health Sciences, "suppose that thalidomide, instead of causing the birth of children with missing limbs, had instead reduced their intellectual potential by 10%. Would we be aware, even today, of its toxic potency?" (Rall, as cited in Weiss, 1998, p. 37).

A fourth, related issue is that the majority of research on chemical toxicity is conducted on laboratory animals. The investigator's relative expertise with behavioral testing and interpretation, the choice of animal model, and the choice of functional system (e.g., specific sensory, motor, cognitive, or social) can all affect and potentially undermine the utility of test results as applied to human development (Cory-Slechta et al., 2001). In general, findings from nonhuman studies often significantly underestimate the effects on human populations because subtle cognitive impairments are often difficult to detect in nonhuman populations (Cory-Slechta et al., 2001; Rice et al., 1996).

Fifth, there is frequently a long latency period between the exposure (prenatal or during early postnatal development) and when any effects are observable or measurable. For instance, most intellectual deficits are not apparent until the individual encounters academic settings in later childhood or adolescence. "Delayed neurotoxicity" may even contribute to an acceleration of the aging process, thus remaining unobservable until one reaches middle or even old age (Rice & Barone, 2000; Weiss, Clarkson, & Simon, 2002; Weiss & Simon, 1975).

Sixth, because the developing brain is particularly vulnerable during critical periods of time, exposure to a chemical outside that window may appear to have no effect even if the same exposure would seriously damage brain development during another time period (Rice & Barone, 2000; Rodier, 1995). Thus, the precise timing, nature, and magnitude of the exposures must also be factored into the research design. This information may be very difficult if not impossible to attain for exposed human populations.

Seventh, genetic factors interact with environmental exposures (McClearn, Vogler, & Plomin, 1996), as genes regulate the metabolism and excretion of chemicals (Costa et al., 1999; Furlong et al., 1998). Environmental chemicals may also alter gene expression. Finally, low-income and minority populations are exposed to more environmental pollution including toxic waste (Evans, 2004; Nadakavukaren, 2000) and lead (McGinn, 2002); environmental and public health laws are often inadequately enforced in their communities (Bullard & Johnson, 2000); and environmental toxicants can interact with the poor nutritional status often seen in these individuals. For example, lead is absorbed more readily from the gastrointestinal tract when the person's diet is deficient in calcium or iron (Peraza, Ayala-Fierro, Barber, Casarez, & Rael, 1998). Roughly one third of urban African American children exhibit elevated levels of lead in their blood (McGinn, 2002). Thus, certain genotypes or socially vulnerable individuals represent susceptible subpopulations that may be lost in the general study population, and disproportionate effects on those individuals may go unrecognized. This kind of effect modification has been shown for lead (Bellinger, 2004) and PCBs (Walkowiak et al., 2001) and should be of special interest to psychologists, including those who study enriched and stressful environments in nonhuman animal models.

In sum, epidemiological research, particularly in developmental neurotoxicology, is pervaded by challenges. Further, these complications may lead some to deduce that researchers will never be able to determine conclusively the extent to which environmental toxicants account for disabilities. Science is largely based on avoiding false-positive statements (stating that an association exists when it truly does not; i.e., Type I errors) rather than avoiding false-negative conclusions (failing to recognize associations that do exist; i.e., Type II errors). Although this statistical strategy is accepted as properly conservative in the sciences, it is not the optimal strategy for protecting human health.

An advancement of society's understanding of these issues is also limited by the lack of communication between relevant disciplines. Developmental neurotoxicology bridges clinical and research domains within psychology, biology, public health, environmental sciences, and other disciplines. Because each field uses a specialized vocabulary, its findings are often inaccessible to the broader community studying child development. There is also a tendency to focus on confirming or clarifying effects seen with particular compounds rather than investigating new ones.

Due to the inherent complexity of environmental neurotoxicology, which requires multiple sources of data (exposure assessment, exposure timing, endpoint criteria, appropriate biomarkers, and so forth), interdisciplinary scientific exploration is essential to address this significant public and mental health threat. Given sufficient political

and public will, however, the hazards of environmental pollutants are preventable, more so than most other risks to healthy development (Schettler et al., 2000). Unfortunately, prevention is not typically assigned a high priority, particularly when it is the less fortunate members of society who are most at risk (Albee, 1998; Needleman, 1998).

The Costs of Developmental Disabilities

Despite the methodological limitations outlined previously, "what we do know about [various toxic agents'] effects on the fetal brain is convincing enough to demand caution in their distribution" (Needleman, 1995, p. 77). Even if the effects of toxic substances on cognitive ability are subtle, the economic and social effects can be profound. As Weiss (2000) noted, "if environmental contamination diminishes IQ in the U.S. population by an average of 1%, the annual cost would come to $50 billion and the lifetime costs to trillions" (p. 380). This sum is most likely an underestimate because "small decrements in population IQ will not only decrease the number of people with IQs above 130, but will concurrently increase the number of those with IQs below 70, thereby increasing demand for remedial education and other services" (p. 380; see also Schwartz, 1994; Rice et al., 1996).

Some industry representatives and legislators argue that it is too costly and impractical to increase regulation, especially if it involves requiring evidence that products are safe before allowing them on the consumer market or to be emitted from industrial processes. For example, the recent REACH proposal (Registration, Evaluation and Authorization of Chemicals) in Europe, which would require manufacturers to assess the safety of industrial chemicals before marketing, has been vigorously opposed in the United States by the current administration and the chemical industry. "The administration said publicly that the proposal would threaten the $20 billion in chemicals that the U.S. exports to Europe each year because the cost of testing would be prohibitive. . . . [Secretary of State Colin] Powell warned that $8.8 billion in products were at risk of being banned or severely restricted under Europe's proposed system" (Becker, 2004). Consequently, improperly evaluated exposures continue in the United States.

This outcome is ironic given the vast amounts of money spent on treatment and services for the disabled and diseased. Between $81.5 and $167 billion is spent each year in the United States on neurodevelopmental deficits, hypothyroidism, and related disorders, depending on costs of special education and whether loss of earning potential is included (Muir & Zegarac, 2001). Conservatively, even if only 10% of the incidence of cerebral palsy, mental retardation, and autism are attributable to exposure to environmental toxins, the cost is $9.2 billion annually (Landrigan, Schechter, Lipton, Fahs, & Schwartz, 2002).

These figures lead to the question of calculating the "true value" of avoiding or preventing developmental disabilities:

> All knowledge, all technology and manufactured capital, all forms of social and productive organization, all sustainable environments and natural capital stocks, and all true economic innovation and progress—all growth and development—are tributary products of human creativity and praxis. Surely, the ultimate pollution is the chemical contamination of the brain, mind, and intelligence that form the source of our good fortune. This pollution not only affects the educational attainment, economic performance, and income of the individual, but it also has an impact on the dynamic performance of the economy as a whole through its effect on the quality of the human capital stock, and in turn, on society's potential production possibilities, the rate of technical progress, and the overall productivity. (Muir & Zegarac, 2001, p. 892)

In order to determine the real costs of exposure to pollution and various toxic chemicals, we need to determine a means to measure reduced or lost cognitive and behavioral function. "Only when we attach all of the costs of production and consumption of commercial products will we be in a position to make informed judgments about their use and control" (Needleman, 1995, p. 78).

In fact, prevention of deficits associated with environmental toxicants has been shown to be cost effective. One analysis of the economic benefits related to reduced lead exposure since regulations were increased in the 1970s concluded that "the gain in earning power that each year's newborns experience as a result of not being exposed to the same level of lead as their counterparts were a generation earlier" could range from $110 to $318 billion (Grosse, Matte, Schwartz, & Jackson, 2002, p. 568). Yet one wonders if monetary cost–benefit analyses are even relevant to the question of ensuring healthy cognitive and behavioral development in children. As Needleman and Landrigan (2004) wrote, "only health-based criteria are acceptable for setting a health standard. Cost benefit analyses and policy issues are peripheral and subordinate to the central question: What level of lead in blood is toxic for a child?" (p. 8). That is, what exposure levels and associated disabilities is society willing to tolerate?

The Potential for Psychologists

Recent special issues of *American Psychologist* have provided reviews of the role of psychology in helping to create a sustainable future (e.g., Oskamp, 2000) and in addressing disability and rehabilitation issues (e.g., Pledger, 2003). It is time to merge these concerns regarding sustainability and disabilities in order to address the role of psychologists in the analysis of environmental toxicants and their effects on children's development and ways to mitigate or eradicate those effects. Virtually all areas of psychology are impacted in some way by the relationship between toxicants and child development or could contribute to ameliorating the problem. For example, educational psychologists are increasingly confronted with special needs children. Clinical and neuropsychologists as well as psychiatrists are increasingly called to testify in toxic tort cases. Cognitive psychologists interested in risk assessment have investigated the factors that have an impact on decision making (e.g., Slovic, 2000), and they could contribute to the discussion of conceptualizing risk differently as applied to

impacts on child development (Goldman & Koduru, 2000; Landrigan et al., 2004; Weiss, 1992).

Neuropsychologists can and have developed measures of the sometimes subtle deficits associated with chemical exposures, and psychologists in general are particularly well trained for developing tools to assess impaired behavioral and cognitive function. The science of psychology can illuminate the empirical dimensions of behaviors that contribute to and result from environmental threats including toxicants. Health, biological, clinical, and social psychologists can help examine the stress response associated with living in polluted environments and inform strategies to more effectively cope with such challenges (behaviors that minimize exposure, engaging in community activism, and so forth). McKenzie-Mohr (e.g., 2000a, 2000b) has developed a framework called community-based social marketing (CBSM), which uses principles of social psychology to promote community-based efforts toward sustainable practices. CBSM projects that have been implemented include efforts to reduce pollution and hazardous waste and to protect watersheds. The Web site (see www.cbsm.com) is a valuable resource to academics as well as concerned citizens, offering research articles, case studies, and a how-to manual. Such efforts are critical, as behavioral and social psychology has shown that succeeding and thus feeling empowered on one level often inspires people to work at a more global level (for further discussion of the intersection of psychology and environmental issues, see Winter & Koger, 2004).

It is also critical for people to educate and work for change directly within their spheres of influence—their offices, workplaces, homes, and communities—and to communicate with their local and federal legislative representatives. As a demonstrative case study, one of us (SMK) has been actively involved in a grass-roots community effort to promote the use of integrated pest management (IPM) in her city's parks and other local properties. These techniques rely primarily on nonchemical means such as fertilizing properly, weeding manually, and mowing. Thus, IPM reduces the direct impacts of pesticides to human and nonhuman animal health, as well as the contamination of soils and water supplies, the creation of resistant strains, and the destruction of beneficial species. The author's background as a physiological psychologist, combined with other citizens' expertise in medicine, pediatrics, education, and environmental issues (e.g., salmon conservation, watershed protection, and organic farming) were influential in the adoption of IPM by a "conservative" city council. The citizen group is also developing community outreach efforts to minimize use of pesticides in residents' homes and gardens. Because less toxic or nontoxic alternatives exist for many pesticides (see www.pesticide.org for more information), such efforts have the potential to dramatically reduce the use of toxicants in communities. The input of psychologists to this process is critical.

Psychologists also play an important role in interdisciplinary approaches to the problem of toxic effects on child development. One promising avenue for such collaboration exists in the recent creation of the Cornell Institute for Research on Children (Ceci & Williams, 2003; see also www.human.cornell.edu/units/circ/). The Institute's goal is to address questions of social importance by contributing to core knowledge through interdisciplinary research efforts. One of the first projects chosen by the Institute's board for inquiry was the effects of neurotoxicity on children's cognitive functioning, involving specialists from developmental psychology, animal learning, pediatrics, and neuroscience.

Conclusions

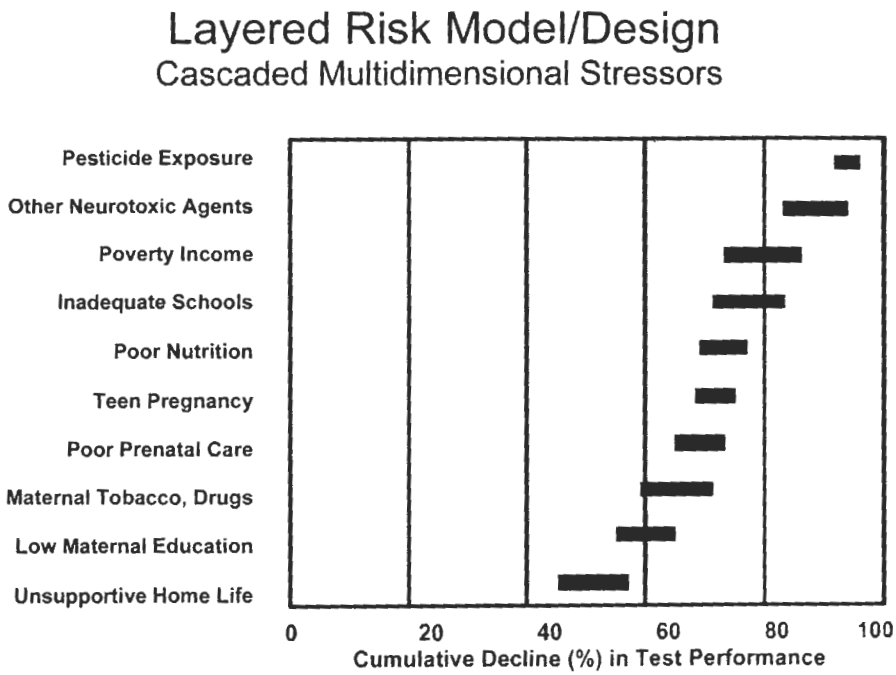
Environmental factors (e.g., enrichment vs. impoverishment) in brain development and subsequent cognitive function have long been recognized (Bennett, Diamond, Krech, & Rosenzweig, 1964; Hubel & Wiesel, 1962; Volkmar & Greenough, 1972; see also Greenough, Black, Klintsova, Bates, & Weiler, 1999, and Rosenzweig, 2002, for a discussion of the implications of this research for rehabilitation of human disability). Educational campaigns have publicized the fact that the developing nervous system is particularly vulnerable to environmental insults in attempts to curb maternal tobacco, alcohol, and other drug use. It is time to expand these campaigns to include use of and exposure to pesticides and other toxic substances.

In addition to the direct effects that toxicants have on neurologic development, stress—such as that associated with living in polluted environments or near a hazardous waste site—is well known to produce deficient cognitive performance, depression, and anxiety disorders (e.g., Bell, Greene, Fisher, & Baum, 2001; Evans & Cohen, 1987; Lundberg, 1998) and can increase the propensity for aggression and violence (Kuo & Sullivan, 2001). It is also clear that exposures to environmental toxicants interact with social factors. As discussed previously, minority and low-income populations are exposed to more environmental pollution, and they often do not have access to the same legal protections afforded the more affluent (e.g., Bullard & Johnson, 2000). Weiss (2000) advanced a conceptual model of how social and environmental factors could cumulate to produce a deleterious effect on cognitive development, even when each individual impact is subtle (see Figure 3).

Albee and Fryer's (2003) call for a critical public health psychology to prevent ill health and promote well-being would certainly include addressing the plethora of toxic chemicals to which all of us are regularly exposed. Yet, as they pointed out, an insidious form of societal neglect can explain why prevention of mental disorders and illnesses has not historically been a priority. The groups who are at greatest risk for exposure to toxic chemicals are also at higher risk for mental retardation or mental illness in general and include those marginalized portions of the population (i.e., minority, immigrant, or low-income; Albee, 1998). Paradoxically, those who become disabled as a result of exposure to toxicants are often subsequently marginalized because of their disability.

Psychologists have an important role to play in countering these disturbing trends. As clinicians, scientists, educators, and citizens, psychologists can mobilize to reduce

Figure 3
Schematic Model Demonstrating How Individual Components of a Stressful Environment Might Cumulate to Reduce Performance on IQ and Other Tests



Note. The individual stressors are shown as overlapping to suggest a lack of independence, and their length is meant to indicate that no single component is overwhelming in isolation. From "Vulnerability of Children and the Developing Brain to Neurotoxic Hazards," by B. Weiss, 2000, *Environmental Health Perspectives, 108*(Suppl. 3), p. 379. Reproduced with permission from *Environmental Health Perspectives.*

the toxic burden shared by all. A healthy and sustainable future depends on it.

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