Environmental Toxicants and Developmental Disabilities

A Challenge for Psychologists

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Developmental, learning, and behavioral disabilities are significant public health problems. 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 unidentified fraction of these disabilities may reflect adverse impacts of this "vast toxicological experiment" (H. L. Needler, as quoted in B. Weiss & P. J. Landrigan, 2000, p. 375). Yet the hazards of environmental pollutants are inherently preventable. Psychologists can help prevent developmental disabilities by monitoring 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 are significant public health problems and appear to be increasing in prevalence (Goldman & Kodu, 2000; Schechter, Stein, Nech, & 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; Eggelston, 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; Olendick, Oswald, & Olendick, 1993). The developmentally disabiled 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 & Beauden, 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, Innominati, Boyce, & Smith, 1993); and utilizing special educational and therapeutic services (e.g., psychological, occupational, and physical).

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Throughout this article, the generic term "developmental disability" often collectively to these three categories of disabilities.
speech, language, mood) 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 toxins (Bellinger & Adams, 2001; Costa, Aschner, Vitalone, Syverson, & 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 developmentally disabled children are the result of environmental exposure to a wide variety of substances and processes. Of these, many are neurotoxicants (Blaine, 1995; Dodson, 1983; Fein, Schwartz, Jacobson, & Jacobson, 1983; Mccann, Dunn, & Lesie-Haley, 1994).

Environmental Toxicants

It is well established that the developing nervous system is particularly vulnerable to environmental insults (e.g., Goldman, 1995; Kodner, 1995; Snyder, 1975; Weiss, 2000). Brain development begins early in the fetus, as cell division, migration, differentiation, and synapse formation proceed in well-ordered waves 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 later in the child's development during childhood and adolescence. Because of the neural blood-brain barrier that is not fully formed, toxicants can enter and interact in developing brain tissue through direct or indirect exposure through the variability of age-related 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 surroundings 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 toxicants 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 chlorine 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 autism (Sax & 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 & Enel, 2000).

Lead

Lead provides an important case study, as it represents the first-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 or dust; and lead-contaminated soils can persist as long as 2,000 years (Nadasen, 2000). Contaminated house dust is a significant source of lead exposure, particularly for children in urban communities (Hayes, Lamphere, Tobin, & Woods, 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. Needham (1996) and Chronic (1996) noted that lead exposure was considered benign until the landmark observation of an association between higher lead levels in teeth and difficulty following classroom instruction, shy or 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 in new information is gathered and evaluated" (p. 45). In fact, it is now generally held that three is no "safe" level of lead exposure. A recent study determined 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

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intellectual function (Cantifield 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, Bisceglier, & Greenhouse, 1996), violence (Masters, 1997), criminality and unwed pregnancy (Neve, 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; Lansheur, Dietrich, Ainger, & 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 disorders of cognitive development as reflected by lowered IQ scores, impairments of memory and attention, and cognitive 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 Seychelles 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 (Plots et al., 2002; Zarebsa et al., 2002), as well as affect cognitive abilities such as memory (Guillellet, Meza, Aguilar, Sato, & 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 (rodenicidites); 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 (Mears 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 fish and shellfish that contain traces of mercury (U.S. EPA, 2004, pp. 1, 2).

Several recent studies have demonstrated learning and behavioral changes in rodents exposed to organophosphate pesticides (e.g., Icenogle et al., 2004; Lautrin, Lima, Guoed, & Bernardi, 2004; Ricci et al., 2000; Sanchez-Santocj, Canadas, Flores, Lopez-Grencha, & Cardona, 2004). This type of pesticide is commonly used for home, lawn, and garden pest control as well as on 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 neurotransmitter level and cause hyperactivity in the animals as adults" (Schettler et al., 2000, p. 85; see also Albright, 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, Guillellet 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 neurocognitive measure of cognitive ability but may also indicate visuo-motor 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 & Landirgan, 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 & Florins, 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
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 or environmental toxicity [information] is publicly available. [Further,] a full set of basic toxicity information [i.e., including evaluator 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 neurotoxins, 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 unavailable 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. (National Joint Commission, 1992, pp. 5, 25)

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

- persistent and bioaccumulative substances;
- polychlorinated biphenyls (PCBs) 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 (Nadasdyvarkaren, 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 endo-
ctrine-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 ad-
ditives, water purification byproducts, industrial process chemicals, pharmaceuticals, and contaminants from human
waste (e.g., Muir & Zegarac, 2001). The ideal of an unex-
posed control group with which to compare exposed pop-
ulations cannot be achieved.

Second, because humans are routinely exposed to a "toxic soup" of chemicals rather than to individual com-
ponents that may not reveal hazards when tested indepen-
dently, interactive effects of toxins may remain uniden-
tified. For example, the combination of two widely used pesticides, malathion and parathion, 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 ad-

Third, human behavioral and cognitive effects from environmental exposures are generally subtle and difficult
to diagnose, or 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 in-
stead reduced their intellectual potential by 10%. Would we be
aware, even today, of its toxic potency?" (Rall, as cited in

A fourth, related issue is that the majority of research on
toxic chemistry 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 said potential under-
mine the utility of test results as applied to human de-
velopment (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 pop-
ulations (Cory-Slechta et al., 2001; Rice et al., 1996).

Fifth, there is frequently a long latency period be-
 tween the exposure (prenatal or during early postnatal
development) and when any effects are observable or mea-
surable. 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 pro-
cess, thus remaining unobservable until one reaches, middle
or even old age (Rice & Barone, 2000; Weiss, Carlson, &

Sixth, because the developing brain is particularly vulner-
able 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, 1998). 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 obtain for exposed human populations.

Seventh, genetic factors interact with environmental
exposures (McClan, 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; Nadaka-
vukuren, 2000) and lead (McGinn, 2002); environmental
and public health laws are often inadequately enforced in
their communities (Bullard & Johnson, 2000); and envi-
ronmental toxins 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, & Raq, 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 un-
acceptable subpopulations that may be lost in the general
study population, and disproportionate effects on those
individuals may go unrecognized. This kind of effect mod-
fication has been shown for lead (Bellinger, 2004) and
PCBs (Walkowicki et al., 2001) and should be of special
interest to psychologists, including those who study en-
riched and stressful environments in nonhuman animal
models.

In sum, epidemiological research, particularly in de-
velopmental 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 toxins account for dis-
abilities. 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 sci-
ence, 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 be-
tween relevant disciplines. Developmental neurotoxicology
bridges clinical and research domains within psychology,
biology, public health, environmental science, and other
disciplines. Because each field uses a specialized vocabu-
larly, its findings are often inaccessible to the broader com-
munity studying child development. There is also a ten-
dency to focus on confirming or clarifying effects seen with
particular compounds rather than investigating new ones.

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

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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. As emissions are being released during environmental processes, it 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 $92.1 billion annually (Landrigan, Schechter, Lipton, Fabs, & Schwartz, 2002).

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

All knowledge, all technology and manufactured corn, all forms of social and productive organization, all sustainable environments and natural capital stocks, and all true economic innovation and progress—growth and development—are tributary products of human creativity and praxis. Surely, the ultimate pollution is the chemical contami- nation of the brain, mind, and intelligence that form the source of our good fortune. This pollution not only afflicts 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 its 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 func- tion. "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, preventing of deficits associated with environ- mental 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 in the same level of lead as their counterparts were a generation earlier" could range from $110 to $318 billion (Gross, 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 ques- tion: What level of lead is 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., Orkamp, 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 im- pacted in some way by the relationship between toxicants and child development and 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 investi- gated the factors that have an impact on decision making (e.g., Slovic, 2000), and they could contribute to the dis- cussion of conceptualizing risk differently as applied to
impacts on child development (Goldman & Koduru, 2000; Landrigan et al., 2004; Weiss, 1992).

Neuroscientists 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 behavior 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 stress, minimize exposure, engaging in community activism, and so forth. McKenzie-Mohr (e.g., 2000a, 2000b) has developed a framework to provide community-based social marketing to improve community well-being, and this framework can be used to assess the effectiveness of interventions. CBM projects that have been implemented include efforts to reduce pollution and hazardous waste and to protect watersheds. The Web site (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 also 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 & Roger, 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 (SMM) 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, including landscape diversity, methods to encourage pollinators, reducing pesticide use, and habitat creation, have dramatically reduced the use of pesticides in communities, and the destruction of beneficial species. The author’s back yard is now a vibrant, healthy, and diverse habitat, combined with other citizens’ expertise in medicine, pediatricians, 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, 2000; see also www.human.cornell.edu/units/circ/c). 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 underway by the Institute’s board for inquiry was the effects of neurotoxicity on children’s cognitive functioning, in collaboration with investigators 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, Diamonf, Krechevsky, & Rosenzweig, 1964; Hubel & Wiesel, 1962; Volkmann & 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; Landsberg, 1998) and can increase the propensity for aggression and violence (Kao & 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 perspective, which would give more scientific attention to the link between social and physical environments, 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 incountering these disturbing trends. As clinicians, scientists, educators, and citizens, psychologists can mobilize to reduce

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

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