

Toxicants and Environmental Health: A Psychological Issue

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It is widely accepted that physical and psychological health are interconnected and interdependent. Although human well-being and survival depends on the natural environment, environmental health is often treated as separate and distinct. A review of the literature reveals significant associations between various industrial, consumer, and household chemicals and psychological and physiological ailments such as developmental disabilities, mental health issues, neurological impairments, reproductive abnormalities, and cancer. Given that the health of humans is inextricably connected with the integrity of planetary ecosystems, psychologists and other health professionals play a critical role in addressing the risks associated with toxic exposures.

Keywords: Mental Health, Developmental Disabilities, Neurological Disorders, Pesticides, Heavy Metals, Environmental Toxicants

LIST OF ABBREVIATIONS

ADHD	Attention deficit/hyperactivity disorder
ASDs	Autism Spectrum Disorders
BPA	Bisphenol A
CD	Conduct Disorder
CDC	Centers for Disease Control and Prevention
DDT	Dichlorodiphenyltrichloroethane
DEHP	Di(2-ethylhexyl)phthalate
EPA	Environmental Protection Agency
HAP	Hazardous Air Pollutants
MR	Mental Retardation
PBB	Polybrominated biphenyls
PBTs	Persistent bioaccumulative and toxic pollutants
PCBs	Polychlorinated biphenyls

It is clear that human health – broadly defined – is inextricably connected to the health and integrity of the planet's ecosystems (Nadakavukaren, 2000). The contemporary state of environmental degradation is thus not surprisingly correlated with unprecedented increases in the prevalence of many psychiatric, neurological, and developmental disorders. Nearly one-half of U.S. citizens experience some form of mental illness during their life-span (Kessler, et al., 2005), and the prevalence of developmental disabilities has increased 17% over the last decade (Centers for Disease Control and Prevention, 2011). Although the specific etiology varies between disorders and reflects complex interactions among factors including genetics and social factors, the evidence is building for a significant role of environmental toxicants. The following review focuses on the myriad psychological and physiological disabilities associated with exposure to various toxic chemicals, and we argue that psychologists can play a critical role in addressing these risks.

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Environmental Toxicants

Various chemicals released in the production, use, and disposal of consumer products, alone or in combination, can be detrimental to human and ecosystem health. Many substances act as persistent bioaccumulative and toxic pollutants (PBTs; U.S. EPA, 2002); that is, they have the capacity to persist in the environment while retaining their potency, making them potentially harmful even years after release. Similarly, *bioaccumulation* refers to their becoming more and more concentrated as they move up the food chain.

Some of the most commonly noted PBTs in state-issued advisories are the heavy metals mercury and lead, the industrial chemicals polychlorinated biphenyls (PCBs), industrial byproducts known as dioxins, and certain pesticides (i.e., chemicals that are designed to be toxic in order to kill insects (insecticides), weeds (herbicides), rodents, etc.). Humans are exposed to PBTs via a number of pathways: occupational exposures to industrial chemicals or pollutants, breathing in contaminated air, eating contaminated animal products, using certain plastics, electronics, cosmetics and personal care items, or coming in contact with furniture, clothing, electric appliances, or household products treated with flame retardant chemicals (Charboneau & Koger, 2008; Costa & Giordano, 2007; McGinn, 2002; Muir & Zegarac, 2001).

Acute exposure to various PBTs can directly cause symptoms ranging from hyperventilation, nausea, and vomiting to severe poisoning, involving convulsions, unconsciousness, and death (Nadakavukaren, 2000; Weiss, 1997). The phrase *environmental illness* refers to various allergies, chemical sensitivities, and “sick building syndromes” associated with air pollution (e.g., Arnetz, 1998). Environmental illness tends to co-occur with psychiatric conditions including mood and anxiety disorders, as well as somatoform complaints; that is, physical symptoms with no apparent physical cause, including nausea, dizziness, and pain (Bornschein, Hausteiner, Konrad, Forstl, & Zilker, 2006). Depression is directly associated with exposures to chemicals such as pesticides (Weiss, 1998) and indirectly associated with feeling pessimistic or stressed about the quality of the environment and one's ability to affect change (i.e., *learned helplessness*; e.g., Peterson, Maier, & Seligman, 1993). Chronic exposure to PBTs, alone or in combination, can result in

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neurological, hormonal, immunological, reproductive, and carcinogenic effects that manifest in sometimes debilitating problems in both children and adults. Table 1 lists many of the most common PBTs and their associated psychological and physiological effects at different approximate exposure levels.

Table 1. Common PBTs and Their Health Effects at Corresponding Exposure Levels

PBT Type	Effects	Exposure
Lead (blood concentrations)	<ul style="list-style-type: none"> Increased symptoms and risk of ADHD; increased hyperactivity and impulsivity (children) Decreased IQ; decreased cognitive performance; decreased reaction times (children and adults); decreased cognitive development (prenatal exposure)^a Increased risk of meeting criteria for CD; increased symptoms of CD Increased risk of schizophrenia (prenatal exposure)^a Impaired learning and social function (monkeys); impaired gross motor development; increased adult mortality in later life; increased risk of cancer Increased prevalence of autism 	<ul style="list-style-type: none"> 2-5 µg/dL 5-10 µg/dL 1.5-10 µg/dL 15 µg/dL 20-40+ µg/dL
(HAP concentrations)		<ul style="list-style-type: none"> 0.0093 µg/m³
Mercury (blood concentrations)	<ul style="list-style-type: none"> Increased risk of ADHD Increased prevalence of MR Increased depression, anxiety Decreased psychomotor performance Increased neurodegenerative effects^c Increased prevalence of autism 	<ul style="list-style-type: none"> > 3.5 µg/L ≥ 5 µg/L ≤ 40 µg/L; 3-9 µg/day^b 500+ µg/L < 1 µg/kg^d/day
(HAP concentrations)		<ul style="list-style-type: none"> 0.0008 µg/m³
Pesticides (urinary concentrations)	<ul style="list-style-type: none"> Increased risk of ADHD Decreased reflex function Decreased stamina; decreased gross and fine motor function Increased risk of depression 	<ul style="list-style-type: none"> 11-97 nmol*g creatinine/L (organophosphates) 130 nmol/L (organophosphates)^e 0.03-0.1 ppm
(cord blood concentrations)	<ul style="list-style-type: none"> Increased risk or depression and suicide Increased risk of Parkinson's; increased risk of cancer 	<ul style="list-style-type: none"> > 750 days; with incidence of pesticide poisoning^f Acute and chronic exposure^g Chronic exposure
PCBs (plasma concentrations) (blood concentrations)	<ul style="list-style-type: none"> Increased risk of cancer Increased risk and severityⁱ of endometriosis 	<ul style="list-style-type: none"> Chronic low dose^h 0.15-0.4 µg/mL^j

	<ul style="list-style-type: none"> • Inhibition of Purkinje cell development^k (mice) • Neurobehavioral effects such as decreased cognitive function, decreased memory, decreased inhibition (monkeys) • Reproductive failure; decreased conception (female monkeys) • Decreased sperm potency (male monkeys) 	<ul style="list-style-type: none"> • 5×10^{-12}-5×10^{-11} mol/L • 2-3 µg/L • 0.02 mg/kg/day • 0.1 mg/kg/day
Phthalates (plasma concentrations)	<ul style="list-style-type: none"> • Increased risk of endometriosis • Testicular damage (rats) • Infertility (female mice) 	<ul style="list-style-type: none"> • 0.5 µg/mL • 40 mg/kg/day • 400 mg/kg/day
BPA (blood concentrations)	<ul style="list-style-type: none"> • Early onset of puberty in female offspring^a (mice) • Abnormal development of oocyte (mice) • Abnormal pubertal development (female mice)^m • Increased risk of cancer • Increased prevalence of recurrent miscarriage 	<ul style="list-style-type: none"> • 2-20 µg/kg/day • 25-250 ng/kg/day^l • Environmentally relevant levels • 50 µg/kg/dayⁿ • 2.5 ng/mL^o
<p><i>Notes.</i> PBT effects and approximate exposure levels are compiled from the findings of the studies discussed in this review. Effects refer to effects on humans unless otherwise noted. All exposure levels presented are approximate.</p> <p>^aexposure levels of mother during pregnancy.</p> <p>^baverage exposure from dental amalgams.</p> <p>^cassociated with neurodegenerative diseases such as Alzheimer’s and Parkinson’s disease.</p> <p>^dindicates per kg of body weight.</p> <p>^eaverage exposure level was approximately 130 nmol/L but effects were observed at exposure as low as 12 nmol/L.</p> <p>^fpesticide poisoning occurs when there is approximately 20% inhibition of baseline/normal red blood cell/plasma cholinesterase levels. (Baseline levels differ for individuals and the normal range varies between testing labs.)</p> <p>^gEPA’s definitions based off animal studies of “acute” exposure is approximately 0.0005 mg/kg/day, and “chronic” exposure is approximately 0.00003 mg/kg/day.</p> <p>^hlow/average dose plasma concentrations are approximately 0.2-0.5 µg/g lipid.</p> <p>ⁱseverity of endometriosis, indicated by stage, increased with an increase in concentration.</p> <p>^jexposure levels resulting in observed effects vary depending on PCB compound.</p> <p>^kassociated with ADHD and autism.</p> <p>^lequivalent to environmentally relevant levels in humans.</p> <p>^mimplicative of increased breast cancer risk.</p> <p>ⁿenvironmentally relevant levels (range of typical human exposure).</p> <p>^oconsidered “high” exposure.</p>		

Developmental Disabilities in Children

Pre- or postnatal exposure to toxins can result in developmental abnormalities in children because the central nervous system is extremely vulnerable in its developing stages (e.g., Goldman, 1995; Rodier, 1995; Weiss, 2000). 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). Underdeveloped immune systems and detoxification processes do not adequately protect the fetus from chemicals passed from mother to child via the placenta, or postnatally by infant consumption of breast milk. Infants and toddlers crawling on the floor stir up dust and potentially toxic residues that are then

breathed in or ingested by way of infants' natural method of exploration, putting objects in their mouths (Weiss, 2000). Likewise, because children consume more fruit, juice, and water than adults, they are more likely to be exposed to pesticide residues and contaminants in these substances (Environmental Working Group, 2008). Children may also be exposed to harmful chemicals through plastic baby bottles and toys, as well as oral consumption of household cleaning agents that are not properly kept out of reach (Landrigan et al., 2002).

Because normal neurological development is based on exquisitely choreographed sequences of cellular events, such early chemical exposures may result in impaired cognition, attention, behavior, social functions and emotional regulation (Koger, Schettler & Weiss, 2005; Koger & Winter, 2010; Schettler, Stein, Reich, & Valenti, 2000; Tyler, White-Scott, Ekvall, & Abulafia, 2008). Various chemicals can have significant effects on brain development and function via direct neurotoxicity, interference with normal neurotransmission, endocrine function, and cell signaling, and neurodevelopmental processes such as cell replication, and cell differentiation (e.g., Koger, et al., 2005; Porterfield, 2000; Rice & Barone, 2000; Rodier, 1995; Schettler, et al., 2000).

Environmental toxins' role in the etiology of developmental disabilities may also be an issue of social justice. It is known that low-income, often times ethnic and racial minority communities are disproportionately affected by environmental hazards and burdens (Brulle & Pellow, 2005; Metzger, Delgado, & Herrell, 1995; Mott, 1995). A recent study examining National Health Interview Surveys from the years 1997 through 2008 revealed higher prevalence of developmental disabilities including Attention Deficit/Hyperactivity Disorder, autism, cerebral palsy, blindness, and hearing loss, as well as learning disabilities, intellectual disabilities, seizures, and stuttering or stammering in children of families with income levels below the federal poverty line or insured by Medicaid versus private insurance (Boyle et al., 2008). These findings indicate that discussions about the psychological and physiological effects of environmental toxins are also appropriate, and necessary, in the arenas of social and economic justice.

Attention Deficit/Hyperactivity Disorder

Attention Deficit/Hyperactivity Disorder (ADHD) is defined by inattention, hyperactivity, and/or impulsivity that interferes with social, academic, or occupational functioning (American Psychiatric Association, 2000). Although genetic inheritance may confer a predisposition (Mill & Petronis, 2008), ADHD has also been linked to a number of external factors such as diet and television watching (Christakis, Zimmerman, DiGiuseppe, & McCarty, 2004; Richardson & Puri, 2002; Schab & Trinh, 2004), and evidence supporting the connection between ADHD and chemical exposures is accumulating. Childhood exposure to lead (in paint, commercial products, contaminated soil and water from foundries and smelters, or corroded lead pipes) appears to be most strongly related to the development of attentional deficits (e.g., Needleman, et al., 1979; Needleman, 2004). It is estimated that about 290,000 (roughly 5% of) ADHD cases in the U.S. could be accounted for by childhood lead exposure (Braun, Kahn, Froehlich, Auinger, & Lanphear, 2006). Studies conducted years after the U.S. prohibition of lead-based paint and leaded gasoline continue to reveal elevated blood lead levels in many children and a strong positive correlation between childhood lead exposure and

ADHD symptoms and diagnoses (Bellinger, 2008; Braun et al., 2006; Chiodo et al., 2007). The U.S. Centers for Disease Control and Prevention (CDC) currently defines blood lead levels over 10 µg/dL as of concern; blood lead levels of over 20 µg/dL are generally considered high and levels below 10 µg/dL as low, although recommendations have been made to lower the "elevated" definition to levels above 5 µg/dL (Center for Disease Control and Prevention, 2012). Significantly higher blood lead levels have been found in children manifesting both inattention and hyperactive-impulsive symptoms (i.e., ADHD-Combined type) relative to children without ADHD or those with predominantly symptoms of inattention, suggesting a connection between levels of lead exposure and ADHD symptom severity (Braun, et al., 2006). These associations between blood lead levels and ADHD occur even when lead levels are consistent with or below the U.S. and Western Europe population exposure averages (Nigg, Knottnerus, Martel, Nikolas, Cavanagh, Karmaus, & Rappley, 2008; Nigg, Nikolas, Knottnerus, Cavanagh, & Friderici, 2009). Recent research conducted in China and Korea also revealed significant positive correlations between low level childhood exposure to and blood concentrations of lead, and the presence of ADHD and ADHD-like symptoms in pre-adolescent children (Ha et al., 2009; Wang et al., 2008).

Although the majority of research on the link between environmental toxins and ADHD has focused on lead, exposure to mercury or pesticides may also play a role in the development of ADHD. For instance, a cohort of Chinese children with ADHD was found to have significantly higher blood mercury levels than their developmentally normal counterparts (Cheuk & Wong, 2006). Among these, the children with the highest blood mercury levels had over nine and a half times the risk of having ADHD. The CDC and EPA define mercury exposure over 0.1 µg/kg body weight/day (or blood levels of 5.8 µg/L) as elevated and cause for concern; exposure levels below this are generally regarded as low (U.S. EPA, 2012c).

Certain pesticides, such as the organophosphate insecticides, have neurotoxic effects and can thus adversely impact neurodevelopment (e.g., Schettler et al., 2000). One study utilized data from the National Health and Nutrition Examination Survey (2000-2004) and found that pre-adolescent and adolescent children with higher concentrations of the urinary metabolites indicative of average exposures to organophosphates (e.g., in blueberries and strawberries) were significantly more likely to receive a diagnosis of ADHD than those with lower concentrations of these same metabolites (Bouchard, Bellinger, Wright, & Weisskopf, 2010).

Autism Spectrum Disorders

Autism Spectrum Disorders (ASDs) are defined by impairments in social interaction and communication, as well as the exhibition of stereotypical repetitive behaviors or interests (American Psychiatric Association, 2000), and are estimated to affect about one in every 110 children (Centers for Disease Control and Prevention, 2006). Prevalence is apparently increasing (National Institute of Mental Health, 2009), with one estimate showing a 289.5% increase between 1997-2008 (Centers for Disease Control, 2011).

ASDs have been associated with exposure to hazardous air pollutants such as methyl mercury (resulting from burning coal and other fossil fuels, as well as mining processes), lead, solvents, and diesel particulate matter (Windham, Zhang,

Gunier, Croen, & Grether, 2006). ASD prevalence apparently increases with closer proximity to National Priority List Superfund sites that contain many of these same pollutants (DeSoto, 2009). Specifically, the risk for autism, the most severe form of ASD, was significantly positively correlated with the amount of mercury released from industrial sites, and negatively correlated with the distance from these mercury release sites (Palmer, Blanchard, Stein, Mandell, & Miller, 2006; Palmer, Blanchard, & Wood, 2009). As argued elsewhere, oxidative stress likely mediates the relationship between methyl mercury pollution and autism (Leslie & Koger, 2011).

Although the majority of research regarding ASDs and environmental insult has focused on mercury, research suggests that industrial PCBs may also play a role in brain disorders such as ADHD and autism by disrupting hormone systems and normal neuronal development (Kimura-Kuroda, Nagata, & Kuroda, 2007). The EPA currently considers exposures greater than 20 ng/kg body weight/day of PCBs as “high” and warranting of concern (U.S. EPA, 2012e).

Mental Retardation and Motor Impairments

In addition to ADHD and ASDs, various PBTs have been linked to mental retardation, decreased IQ, and impaired motor skills. Early postnatal lead exposure and higher blood lead levels have consistently been significantly associated with decreased IQ and poor cognitive development in infants and children (Chen, Dietrich, Ware, Radcliffe, & Rogan, 2005; Jusko et al., 2009; Needleman & Bellinger, 2001; Schnaas et al., 2006), although social factors including SES and parental care reflect critical moderating variables (Koller, Brown, Spurgeon & Levy, 2004). Even children with relatively low blood lead levels have registered impairments in cognitive function (e.g., Lanphear, Dietrich, Auinger, & Cox, 2000; Nation & Gleaves, 2001).

Prenatal methyl mercury exposure may account for more than 1500 cases of mental retardation in the U.S. each year (Trasande, Schechter, Haynes, & Landrigan, 2006). In addition to cognitive impairments, mercury and lead exposure at higher doses can adversely affect motor-coordination, and can result in seizures (Mendola, Selevan, Gutter, & Rice, 2002; Schettler, 2001). Similarly, pre and postnatal pesticide exposure was linked to impaired motor coordination, balance, and reflexes (Bellinger & Adams, 2001; Grandjean & Landrigan, 2006; Guillette, Meza, Aquilar, Soto, & Garcia, 1998; Young et al., 2005).

Psychosocial Impairments

Some children exposed to an agricultural insecticide exhibited greater impulsiveness, anger, and other interpersonal problems (Ruckart, Kakolewski, Bove, & Kaye, 2004). Psychosocial symptoms of lead exposures include antisocial, aggressive, and delinquent behavior (Needleman et al., 1996, 2002), violence (Masters, 1997), criminality (Nevin, 2000), and overall conduct disorder (Braun et al., 2008).

To summarize, various psychological processes involving cognitive, attentional, behavioral, social, or emotional functions can be adversely impacted by neurotoxic and endocrine disruptive chemicals released intentionally or unintentionally into the environment. It is highly likely that toxins combine with other factors to impact the developing brain (Bellinger & Adams, 2001; Weiss, 2000). As Weiss (2000) observed, even though a relatively small number of children experience clinical poisoning severe enough to attract medical intervention, a much

larger population is affected by subclinical poisoning, detectable by neuropsychological testing. Latent, or “silent” toxicity only emerges with additional risks, such as interactions with other toxins or other health issues. The effects may not become evident until the child enters school or faces other intellectually and socially challenging situations (Weiss, 2000, p. 377).

Adult Disorders

In addition to their effects on neurodevelopment and contributions to childhood disabilities, PBT exposures have also been associated with various disorders affecting adults. In fact, early brain damage (i.e., fetal or neonatal) may not be observable until much later in life when the brain is less adaptable (Weiss, 1998). Thus, adult mental illness and neurological diseases including schizophrenia, Alzheimer’s, and Parkinson’s may actually originate in toxic exposures occurring during neurodevelopment.

Again, the social justice issue of socioeconomic status (SES) in conjunction with toxic exposures is present in the realm of adult psychological and physiological disorders. Recent studies have found an increased risk of depression for low-income women (Block Joy & Hudes, 2010), higher prevalence rates of schizophrenia (Saraceno, Levav, & Kohn, 2005) and Parkinson’s disease (Lix et al., 2010) among low SES groups, and higher rates of mortality from cancer in poor versus affluent counties in the U.S. (Ward et al., 2004).

Mental Health Issues

Exposure to pesticides at levels that result in poisoning is significantly correlated with depression and depressive symptoms in adult men and women (Beseler et al., 2006; Stallones & Beseler, 2002; Weiss, 1998). Reference doses vary depending on the type of pesticide, but for many of the most common pesticides used in the U.S., exposure levels are considered high if they are greater than approximately 0.0005 mg/kg body weight/day for acute exposure and 0.00003 mg/kg/day for chronic exposure (U.S. EPA, 2006; U.S. EPA, 2012d). Specific exposure to organophosphate insecticides has been linked to depression, and may play a causal role in the prevalence of suicide in pesticide-exposed populations (London, Flisher, Wesseling, Mergler, & Kromhout, 2005). Adults with chronic exposure to pesticides or acute exposure to mercury also manifested depressive symptoms as well as symptoms of distress and anxiety (Bluhm et al., 1992; Keifer & Mahurin, 1997). In particular, areas polluted with methyl mercury were associated with high occurrences of depression and anxiety (Ushijima, Shono, Kitano, & Futatsuka, 2005). Comparably, dental staff experience chronic low level mercury exposure from dental amalgams (Sellars, Sellars, Liang, & Hefley, 1996), which was significantly associated with increased depression, anxiety, and psychoticism (Aydin et al., 2003; Heyer et al., 2004).

Exposure to lead, mercury, pesticides, and solvents have all been linked to depression, anxiety, and insomnia, but recent studies also reveal an association between these PBTs and psychotic symptoms such as hallucinations and paranoia (Collaborative on Health and the Environment, 2008; Rhodes, Spiro, Aro, & Hu, 2003). Lead exposure in childhood may also represent a significant risk factor for schizophrenia in adulthood (Opler et al., 2004; Opler & Susser, 2005). Several of the known effects of lead such as reduced academic abilities and psychosocial impairments resemble early antecedents of schizophrenia (Opler & Susser, 2005).

Neurological Impairments in Adults

PBT exposures have been linked to neurological impairments in adults including an accelerated decline in cognitive function (Stein, Schettler, Rohrer, & Valenti, 2008; Stewart & Schwartz, 2007; Weiss, Clarkson, & Simon, 2002; Weiss & Simon, 1975). For instance, adults exposed to high doses of lead exhibited impaired verbal learning ability, memory, dexterity, and executive function years after the exposure occurred (Schwartz et al., 2000).

PBTs can disrupt various endocrine processes and may thus increase risk for Alzheimer's Disease (Stein et al., 2008; Weiss, 2007). Alzheimer's, the most common cause of dementia (National Institute on Aging, 2008), is a degenerative and fatal form of dementia that involves the progressive loss of memory, language and interpersonal skills, and eventually, bodily functions (American Psychiatric Association, 2000).

Comparably, Parkinson's disease, a degenerative disorder that generally affects adults, is linked to certain PBTs such as pesticides (Landrigan et al., 2005; Le Couteur, McLean, Taylor, Woodham, & Board, 1999; Stein et al., 2008). Over time, chronic low level methyl mercury exposure may also result in the development of Parkinson's and other neurodegenerative diseases (Weiss, Clarkson, & Simon, 2002).

Reproductive Abnormalities

It is likely that the various neurological (including developmental and psychiatric) disorders associated with PBT exposures are at least partly attributable to endocrine disruption (e.g., Colborn, 2004; Collaborative on Health and the Environment, 2008; Stein, et al., 2008; Weiss, 2006, 2007). Certainly, this is an important mechanism by which various reproductive abnormalities occur in both human and non-human animals (Swan et al., 2005), along with consequent oxidative stress (Wong & Cheng, 2011). Phthalates and bisphenol A (BPA) are among the most well-researched endocrine disrupting chemicals; these are ubiquitous in everyday consumer products including baby bottles, plastic water bottles and foodware, cosmetics, personal care items, flame retardants, detergents, and even baby toys (Sathyanarayana et al., 2008; Weiss, 2007). Exposure to the most common phthalate, Di(2-ethylhexyl)phthalate (DEHP), is considered high and of concern above levels of 0.02 mg/kg body weight/day (U.S. EPA, 2012b). Similarly, levels of BPA exposure are regarded as "high" if they exceed 0.05 mg/kg/day (U.S. EPA, 2012a).

Non-human animal models have revealed that PBT exposure leads to decreased sperm count and potency and infertility in males (Anway, Cupp, Uzumcu, & Skinner, 2005). Studies in humans have also associated greater exposure to PBTs including glues, solvents, silicone, lead, mercury, cadmium, BPA, and pesticides with decreased fertility and sperm quality in men (Benoff, Jacob, & Hurley, 2000; Mendiola et al., 2008; Strohmmer, Bolizsar, Plöckinger, Feldner-Busztin, & Feichtinger, 1993; Wong & Cheng, 2011). Similarly, strong significant associations have been observed between phthalates and PCBs and the development of endometriosis in human women, a gynecological disorder that often leads to infertility (Cobellis et al., 2003; Reddy et al., 2006; Reddy, Rozati, Reddy, & Raman, 2006). Formaldehyde and organic solvent exposures are also significantly correlated with slowed conception and increased risk of spontaneous abortion in adult women (Taskinen et al., 1999).

PBTs that mimic estrogen hormones and alter the activities of natural estrogen in young girls may have adverse effects on pubertal development. BPA, dichlorodiphenyltrichloroethane (DDT), polybrominated biphenyls (PBB), and phthalates have all been linked to earlier onset of puberty in young girls (Roy, Chakraborty, & Chakraborty, 2009). Moreover, this early pubertal development may also result in negative psychological effects in girls, such as depression, anxiety, psychosomatic symptoms, eating disorders, decreased academic success, substance use, earlier sexual behavior, and delinquency (Mendle, Turkheimer, & Emery, 2006).

Cancers

A number of PBTs are linked to the development of cancer. PBTs such as poly-aromatic hydrocarbons (from the burning of fossil fuels), heavy metal contaminants in fruits and vegetables, pesticides, and PCBs have been suggested to play a role in the development of breast cancer, gastrointestinal cancer, pancreatic cancer, lung, and stomach cancer (Bu-Tian et al., 2001; Gammon et al., 2002; Lucena, Allam, Costabeber, Villarejo, & Navajas, 2001; Pavuk et al., 2004; Türkdöğän, Kilicel, Kara, Tuncer, & Uygün, 2003). Various PBTs have also been directly linked to brain, cervical, ovarian, prostate, and kidney cancer, as well as leukemia and non-Hodgkin's lymphoma (Bassil et al., 2007; Clapp, Jacobs, & Loechler, 2008; vom Saal et al., 2007).

Conclusions

The ubiquity of chemicals with known or suspected neurological, psychiatric, developmental, reproductive, and carcinogenic effects is a compelling reason for cautionary use and efforts to prevent exposures. However, there are potentially even more detrimental effects to human health that result from the combination of chemicals or chronic low-level exposures to toxins that cannot be simulated under laboratory conditions and in experiments utilizing non-human animals. Likewise, subtle disturbances in behavior (e.g., language) or cognition may not be detectable in non-human subjects. The delay between exposures and evidence of disability or disease, the role of critical periods of development, and interactions between genetic factors and toxic exposures all make it very difficult to accurately estimate the direct effects of environmental toxins on humans, and likely result in a gross under-estimation of the detrimental health effects of toxic exposures (e.g., Koger, et al., 2005; Schettler et al., 2000).

There are important social justice issues related to toxicant exposures, as well. Low income and minority populations are exposed to more environmental pollution including toxic waste (Nadakavukaren, 2000; Evans, 2004) 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. Given sufficient political and public will, the hazards of environmental pollutants are inherently preventable. Unfortunately, prevention is not typically assigned a high priority, particularly when it is the less fortunate members of our society who are most at risk (Albee, 1998; Needleman, 1998).

Overall, the issue of public health is often absent from social and political debates where topics of economic costs and benefits dominate. Ironically, there are significant adverse economic consequences associated with disruptions to

psychological and physical health, including direct health care costs and decreased worker productivity in the disabled and diseased. A decade ago it was estimated that between \$81.5 and \$167 billion was 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); these costs are likely much higher today. Conservatively, even if only 10% of the incidence of cerebral palsy, mental retardation, and autism are attributable to exposure to environmental toxins, the annual cost exceeds \$9.2 billion (Landrigan, et al., 2002). A recent study of the annual “costs of environmentally attributable disease and disability” just in the state of Oregon reaches \$1.57 to 2 billion; childhood lead exposure alone in Oregon is estimated at \$878 million including lost earning potential from diminished IQ; neurobehavioral disorders reach \$187 million each year (Hackenmiller-Paradis, 2008). The costs of caring for people with Alzheimer’s disease are almost \$150 billion per year while Parkinson’s disease is estimated to cost between \$13 to \$28.5 billion per year in the United States (Stein et al., 2008).

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 any decrease of average population IQ not only decreases “the number of people with IQs above 130, but concurrently increases the number of those with IQs below 70, thereby increasing demand for remedial education and other services” (p. 380). One analysis concluded that the reduction in lead exposures since regulations were increased in the 1970s could increase each year’s newborns’ earning power as much as \$110 to \$318 billion, relative to that of the previous generation (Grosse, Matte, Schwartz, & Jackson, 2002). Yet we wonder if monetary cost-benefit analyses are even appropriate to the question of ensuring the health of future generations.

Recognizing that the health of humans and the natural world are inextricably intertwined, it is critical that members of the psychological community become a more active and influential part of the environmental health debate (Koger, et al., 2005; Weiss, 1983). Psychologists in various applied fields have a clear stake in this issue: for instance, educational psychologists and teachers increasingly encounter special needs children. Clinical and neuropsychologists as well as psychiatrists see many affected individuals and are regularly consulted in toxic tort cases. Clinical, health, and biological psychologists can contribute to research on the stress associated with toxic environments and inform coping strategies (e.g., behaviors that minimize exposures).

Fortunately, if people perceive a situation or product to be harmful to their own or their family’s health, they are generally more motivated to engage in problem solving and specific behaviors that act to reduce the risks (Homburg & Stolberg, 2006). In that regard, there are various approaches that can help address people’s dependence on and exposures to potentially toxic substances (Koger, 2009):

- Highlighting the health incentives to behavioral and legislative change;
- Paying particular attention to those chemicals (e.g., pesticides; other consumer products) or activities

(lobbying, voting, community action) within one’s immediate control;

- Obtaining clear and detailed information about non-toxic or less toxic alternatives to various products, available on-line from several organizations (e.g., see the Northwest Coalition for Alternatives to Pesticides, <http://www.pesticide.org>; the Oregon Toxics Alliance, http://www.oregontoxics.org/safer_alternatives.html; The Environmental Working Group, <http://ewg.org/>).
- Reducing consumption of animal products, and purchasing organic produce whenever possible.
- Making a public commitment to changing a relevant behavior, such as pledging to refrain from idling one’s car to help improve local air quality, and reduce adverse impacts on lung and heart health. (See the no-idling campaign initiated by the Spokane Regional Clean Air Agency, available at http://www.spokaneleanair.org/no_idle_zone.asp)
- Initiating community efforts to reduce local and household hazardous wastes (Werner, 2003; Werner & Adams, 2001).

Our collective health and that of future generations depends on such applications of psychological theory and research.

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