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Protecting Children from Exposure to Lead Old Problem, New Data, and New Policy Needs

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Abstract

The detrimental effects of lead exposure in children have been known for over 100 years. Although a few initial measures implemented about 30 years ago were effective in somewhat reducing levels of lead exposure in children, relatively little has been done recently from a policy perspective to protect children from lead. We now know from recent research that much more work is needed. Recent events highlighted in the media show that several urban communities still have unacceptable levels of lead in water systems. Early research identified high levels of lead as being particularly detrimental to children's intellectual and behavioral development. However, new studies have discovered that lower levels of lead, levels once thought safe, also cause considerable damage to children's developmental outcomes. This social policy report summarizes new data on the intellectual, academic, and behavioral deficits seen in children exposed to both low and high levels of lead, discusses the biological and neurological mechanisms of lead poisoning, explores sources of environmental lead exposure and lead abatement practices, shows that current federal and state-level child screening and lead level reporting practices are inadequate, and makes policy recommendations centered on increasing education, intensifying abatement efforts, strengthening and regulating mandatory screening practices, and reducing the federal threshold of allowable levels of lead.

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From the Editor

With Volume 24, Issue 1, a team from Frank Porter Graham Child Development Institute at The University of North Carolina at Chapel Hill will begin the editorship of the SRCD *Social Policy Report (SPR)*. Reiterating our statement in the last issue, we appreciate the great expertise and leadership that Lonnie Sherrod and Jeanne Brooks-Gunn provided for *SPR*. We hope to extend and elaborate on the momentum they created for the quarterly report. *SPR* stands as the preeminent policy publication addressing developmental science topics for policymakers and broader consumer audiences. The report's translational function is complemented with concise and attractive *SPR Briefs* produced by Marty Zaslow, Sarah Hutcheon, and Sarah Mandell in the SRCD Office for Policy and Communications, and Anne Bridgman, the *Brief* science writer. SRCD strives to inform policy through scientific evidence, and we will continue to make the *SPR* the premier report for lawmakers, policy experts, and researchers involved in developmental science issues.

This issue also inaugurates some important changes. The *SPR* has a new look and feel but maintains the essential informational elements of the old format. This new format will be disseminated only electronically but conforms to the word length requirements of the previous print issues. However, if you print out the issue, it will take more pages because of the changes in design, which we hope will enhance readability. Also, the electronic format includes links to available citations and abstracts, allowing our readers to go directly to the information source. In the future, we hope to use other forms of technology to add convenience and convey the most essential information covered in the *SPRs*. We invite readers to share their observations or comments about the new format. Please send any questions or comments to Anne Hainsworth at anne.hainsworth@unc.edu.

When we sat down to discuss topics for the first report of 2010, we debated the merits of focusing on the ongoing issue of childhood lead exposure. The topic did not seem to be very cutting edge. Did we as a society not address this problem years ago, and is not the situation better now? Cole and Winsler's review of newer (as well as older) data on the detrimental effects of low-level lead exposure made us sit up and pay attention. Nigg highlights in his commentary the research linking lead to ADHD and raises the question about other future potential neurotoxins. Lanphear's commentary reminds us of the world's long struggle to reduce lead levels in children and recommends increased efforts to eliminate lead from consumer products. A separate commentary by Gould and Hertel-Fernandez, though, raises the important issue of considering cost-benefit ratios of various lead reduction strategies before recommending or implementing policies. Although some progress has been made over the past 20 years, it is very clear that more can be done to reduce and prevent lead exposure among our nation's children. We hope this issue of *SPR* brings renewed interest in this old, but nevertheless dangerous, problem.

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Protecting Children from Exposure to Lead

Old Problem, New Data, and New Policy Needs

In 2000, the District of Columbia Water and Sewer Authority (WASA) instituted a new method of water disinfection, changing from the use of free chlorine to chloramines. The addition of chloramines to the water system had several unintended side effects, primarily increased corrosion of the city's water pipes, many containing lead. Such corrosion led to dangerous increases in lead levels in the city's drinking water (Edwards, Triantafyllidou, & Best, 2009; Environmental Protection Agency [EPA], 2007; Guidotti, Moses, Goldsmith, & Ragain, 2008). The current allowable amount of lead in drinking water is set by the EPA at 15µg/L (EPA, 2004a; EPA, 2006). A water lead level above this amount exposes the public to unsafe amounts of lead. By late 2001, tests of the D.C. drinking water showed lead water level readings in excess of the allowable level set forth by the EPA. In 2004, a *Washington Post* article exposed the elevated water lead levels, instigating widespread concern among community members worried about the effects of elevated levels of lead. WASA serves over 500,000 D.C. residents, and many parents were appropriately concerned with the effect of lead on their children. Many parents and community members searched for information on the effects of increases in lead exposure that remained below the federal threshold, but earlier reports were not conclusive as to the effects of low levels of lead on children.

D.C. is not alone in its trouble with lead-contaminated drinking water. Articles in the *Seattle Post* (Bach, 2004) and *Seattle Times* (Bhatt, 2005) reported that in 2004, tests of Seattle's public schools indicated that 70 of 88 schools had at least one water fountain test above the EPA recommended lead level. Nineteen of the schools tested had over half of their drinking fountains exceed the limit. One fountain tested at 1,600µg/L, an amount more than 80 times greater than the EPA's allowable lead level. Tests conducted back in 1990 and 1992 also indicated elevated lead levels within the Seattle school system,

indicating an ongoing problem.

In a follow-up to the initial D.C. article, Leonning, Becker, and Nakamura (2004) of the *Washington Post* examined past records of lead water level tests. Their results indicated that large municipalities including Boston, Philadelphia, and New York had avoided testing homes that were likely to show high water lead levels, and had dismissed tests that indicated unsafe levels of lead within their water systems. Additionally, some states chose not to report federally required water lead level violations to the EPA, as federally required, providing even more uncertainty about the safety of our nation's water. These reports indicate that high lead levels in drinking water may be more common than generally thought and that we have not done enough yet to prevent the exposure of children to lead. In addition, water is just one of many potential sources of lead exposure, which include lead-based paints, house dust, soil, and consumer products.

Thus, it is critical for us to understand the effect of lead on child outcomes.

In 1994, the Society for Research in Child Development (SRCD) published a *Social Policy Report* describing the most recent research concerning the effects of lead on children, and suggested possible ways of protecting children from exposure (Tesman & Hills, 1994). In 1991, the EPA safety threshold for Blood Lead Level (BLL) was set at 10µg/dL, and any BLL greater than this was considered unsafe.

The prior *SPR* detailed the effects of large amounts of lead on children's development (BLL > 10µg/dL), and only hinted at possible effects of low-level exposure (BLL < 10µg/dL). In the last 15 years, many new studies have focused on the effects of lead on children's development when exposure is well below the current 10µg/dL threshold. The results of these studies are quite disturbing. The goal of the present report is to review and update our knowledge base on the negative developmental effects of even low levels of lead exposure on children and to make an urgent call for policy action to reduce and eliminate the harmful

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effects from this fully preventable risk factor for negative child outcomes.

This report is divided into three main sections. First, we will discuss the research on the effects of lead exposure in children after providing a brief history of research conducted in this area. The biological mechanisms of human lead exposure will also be discussed, as will children's particular neurodevelopmental sensitivity to lead. The second section will describe environmental sources of lead and ways in which children's exposure to lead can be reduced via parent education, lead abatement, and child screening practices. The report will conclude by showing the inadequacies of the current reporting and screening systems and suggesting policy recommendations aimed at the long-term reduction of lead exposure in children.

History of Childhood Lead Research

Concerns about potential negative effects of lead on children began to emerge in the 1890s, with reports from Australia documenting various unusual illnesses. Children were found to have symptoms such as headache, nausea, and motor problems, and in 1904, these symptoms were traced to high lead levels in both home water tanks and paint dust (Gibson, 1904; Needleman, 2004; Tesman & Hills, 1994). Initially, there was widespread skepticism as to the negative effects of lead on children, but in the 1930s and 1940s, societal views began to change. Several outbreaks of acute lead poisoning in the United States gave researchers an opportunity to observe the effects of large levels of lead exposure first-hand. In 1943, a study of 20 children who had suffered acute lead poisoning found that 19 of them had long-term deficits in behavior, learning, and school performance (Byers & Lord, 1943, as cited in Needleman, 2004). These early studies promoted the general understanding that toxic lead poisoning causes long-term developmental deficits in children.

Prior to the mid-1970s, the Centers for Disease Control and Prevention (CDC) had mandated that a BLL above 60µg/dL be deemed toxic to children (CDC, 1991). In the early 1970s, the federal government enacted guidelines for lead screening in children (Tesman & Hills, 1994). Data from these screenings provided new information indicating that children who had high (>10µg/

dL) but not toxic (>60µg/dL) BLLs also showed deficits in behavior, learning, and intelligence. Based on this research, the CDC revised its standards for blood levels in children and reduced the acceptable amount to 30µg/dL. Research conducted in the mid-1970s and 1980s focused on the effects of high BLLs on children. In response to this research, the CDC again reduced acceptable BLLs in children to 25µg/dL and eventually to 10µg/dL. Researchers wondered, however, if even lower levels of lead negatively affected children (CDC, 1991; Tesman & Hills, 1994).

Mechanisms of Lead Neurotoxicity

Although researchers have long known that lead negatively affects child outcomes, it is only recently that the biological mechanisms of lead exposure have been discovered. Although there are many different mechanisms by which lead affects development, there seem to be several broad categories of function. First, lead seems to promote abnormal cell apoptosis (programmed cell death); second, it seems to perturb normal protein function within the brain; and third, it seems to alter neuro-

chemical functioning within the brain. Many of lead's varying mechanisms of action are driven by its ability to bind to calcium receptors within the body. Lead passes through the body's blood-brain barrier in part because of its ability to "substitute" for calcium. In a normal brain, neurons employ calcium channel pumps to regulate their electrical gradient, allowing for the production of action potentials and electrical impulses. These electrical impulses serve as one of the main modes of communication within the brain. Lead has the ability to be taken

in by calcium channel pumps and enter neurons in this manner (Kerper & Hinkle, 1997). Once lead enters the neuron, it disrupts normal cell functioning which causes apoptosis. The intake of lead into the neuron disrupts the calcium gradient within the cell, damaging neuronal mitochondria which often results in cell death. In addition, when present in large amounts, lead is absorbed by the mitochondria, damaging the organelle and preventing proper neuronal energy production. Mitochondrial damage prevents normal cell functioning and results in abnormal neuronal signal transmission (Lidsky & Schneider, 2003). Mitochondrial apoptosis has been observed in

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cultures and in the retina at levels of 10nm to 1um (He, Poblentz, Medrano, & Fox, 2000).

Lead also affects neuronal development by disrupting normal protein function. In rats, lead has been found to alter lipid peroxidation, which causes damage to neuronal cell membranes. Lead also affects Protein Kinase C (PKC), which plays an important role in neuronal potentiation. In a normal cell, PKC is regulated by nanomolar concentrations of calcium, but when large amounts of lead are present, PKC expression is reduced. Reduced levels of PKC affect neuronal potentiation and differentiation, which may have long-term effects on the development of learning and memory (Nihei, McGlothlan, Toscano, & Guilarte, 2001). Studies using rat models have shown that small concentrations of lead can perturb normal PKC function (Markovac & Goldstein, 1988).

Lead also can affect neurotransmission through perturbation of neurochemical functioning. The presence of lead causes an abnormal inhibition of delta aminolevulinic acid dehydratase. Inhibition of this enzyme results in increased levels of aminolevulinic acid (ALA) within the brain. ALA is a gamma-aminobutyric acid (GABA) agonist and therefore reduces GABA release through pre-synaptic inhibition. This perturbation in GABA release is thought to be responsible for many of the behavior changes associated with lead exposure (Needleman, 2004). Campagna, Huel, Girard, Sahuquillo, and Blot (1999) discovered that perturbation in delta aminolevulinic acid dehydratase functioning can be seen when BLLs are above 3.2µg/dL, but not below, suggesting a possible threshold effect. In addition, lead seems to target mesencephalic dopamine cells, causing apoptosis. This destruction of dopamine-specific cells results in abnormal changes in dopamine levels and transmission throughout the brain, and has been seen at lead concentrations as low as .3 um (Scortegagna & Hanbauer, 1997). Lead's effects on brain function are severe and wide reaching, because lead's ability to substitute for calcium presents many possible mechanisms of action within the brain. Additionally, observed effects of lead seem to occur at relatively low levels of exposure and affect not only the development of the overall structure of the brain, but also communication between neurons, as well as the internal working of the neurons themselves.

Children in low-income families are more likely to be exposed to lead.

Children's Sensitivity to Lead

Although lead exposure is not beneficial at any age, children are particularly sensitive to its negative effects, arising from both their early development stage as well as their biologically driven sensitivity to lead. Exposure can begin prenatally, since lead easily crosses the placental barrier, and research indicates that mother and placental lead levels are very similar. The presence of lead in the womb is extremely troublesome as it can disrupt normal developmental processes (Goyer, 1990). BLLs in children have generally been found to peak around the age of 2 and decline in the following years (Brody et al., 1994). This peak in lead levels around the age of 2 is due to children's crawling and walking behaviors coupled with their desire to mouth objects. Lead contaminated house

dust is one of the most common sources of lead exposure and is often found on the floor and in windows of older homes. Young children are particularly vulnerable to lead dust, as their early crawling and walking behav-

iors position them near the floor. When young children come in contact with lead, they are likely to ingest it via hand/object-to-mouth exposure. In addition, the gastrointestinal tract absorbs lead more efficiently than the lungs or the skin, which can lead to increased lead intake for this young population (Leggett, 1993).

Another factor that makes children specifically vulnerable to lead is that children generally absorb lead more efficiently as it mimics calcium within the body. Young children's rapid growth, and their resulting need for calcium, often results in greater absorption of lead by the gastrointestinal tract than typically would be seen in adults. Increased lead absorption by the gastrointestinal tract results in larger lead levels in the blood, bone, and teeth of this age group, and therefore, larger lead-related effects (Cory-Schlecta & Schaumburg, 2000). In children, bone is constantly being built and re-absorbed by the body (Matkovic, 1991). This means that lead stored in bone can leach into children's blood over time, and thereby access the brain. In addition, children's blood-brain barriers are less efficient at filtering out lead, which means more is allowed into the brain. Increased lead levels in the brain result in further damage to brain function.

Children in low-income families are more likely to be exposed to lead (Brody et al., 1994; Lin-Fu, 1992; Rutter, 1983). Brody et al. (1994) found that as income

increased, lead levels tended to decrease in children. In this study, 16.3% of children categorized as low income were found to have elevated lead levels ($>10\mu\text{g}/\text{dL}$) as compared to 5.4% and 4.0% for children categorized as medium or high-income. In addition, children from low-income households appear to be more sensitive to the effects of lead and show deficits at lower BLLs than their high-income counterparts (Bellinger, Leviton, & Solman, 1990). This may be due to the fact that children in poverty are likely to have other risk factors, such as low birthweight, school absences, less education, more stress, more punitive parents, and lower levels of self-esteem (Aber, Bennett, Conley, & Li, 1997).

The half-life of lead is 35 days when located in the bloodstream, 2 years when located in the brain, and decades when located in the bone (Lidsky & Schneider, 2003). The inefficiency of children's blood-brain barrier, coupled with their rapid growth, makes it more likely that lead will be stored in their bone and brain tissue. This storage causes lead to persist longer in children than in adults, which in turn increases the duration of time that lead can perturb child functioning. Finally, due to the developing nature of the child's brain, children are more sensitive to the changes in protein and neurochemical regulation that lead produces. Lead exposure in children, therefore, has the potential for longer and more widespread effects on development and later performance than is seen in adult exposure (Lidsky & Schneider, 2003).

Methods of Lead Detection

The main ways of detecting lead in children are through tests of the blood, teeth, and bones, although urine, feces, nail, hair and saliva samples have been used in the past. We will briefly discuss each measurement technique. BLLs are detected through capillary or venous puncture samples and are generally reported in micrograms per deciliter ($\mu\text{g}/\text{dL}$). These samples reflect the amount of lead currently in a person's system, as blood does not store lead the way bones and teeth do. BLL may be assessed by looking at whole blood or blood plasma. Plasma lead level is thought to provide a more useful representation of exposure to lead. Tooth, or dentine, lead levels represent a person's lifetime exposure to lead, since lead is stored in the teeth as we grow. Different teeth emerge at different points during childhood, and different tissues of the teeth form and absorb lead at different time points. This allows for a history of lead exposure to be assessed. For dentine lead collection, families usually submit a baby tooth for examination. Dentine

lead levels are typically reported in micrograms ($\mu\text{g}/\text{g}$) or parts per million (ppm). Studies that have compared dentine lead levels to BLLs indicate that these measures roughly relate in a 1:2 ratio, that is, finding a $1\mu\text{g}/\text{g}$ level of dentine lead generally relates to a finding of $2\mu\text{g}/\text{dL}$ of lead in that child's blood (Rabinowitz, 1995).

Bone lead levels also reflect a person's lifetime exposure to lead as it is also stored in the bones as we grow. Lead in bone can be detected through post mortem collection or through a type of low energy x-ray called an XRF (in vivo X-ray fluorescence). XRF tends to become unreliable as the amount of tissue covering the bone increases; therefore, this technique would be more accurate for certain bones. Additionally, different bones may absorb lead at different rates, depending on the amount of blood flow to that bone and the type of bone tissue. Therefore, appropriate bone samples must be carefully chosen for these types of analyses. Generally, blood or bone lead detection methods are preferred (Barbosa, Tanus-Santos, Gerlach, Parsons, 2005; Lanphear et al., 2008; Tesman & Hills, 1994).

Lead sampling from urine represents current lead levels and is the most useful in long-term lead tracking studies (mostly longitudinal occupational lead exposure studies) since single-sample measures have produced inconsistent results. Fecal samples tend to reflect current lead levels as undigested lead or lead that has been processed through endogenous fecal routes. Although this technique is generally non-invasive, differences in day-to-day biological processing result in variations in fecal lead levels that could be wrongfully attributed to changes in lead exposure. Nail sampling represents long-term lead exposure. Clippings are generally taken from the toes as they are less contaminated than fingernails by external lead exposure. A drawback of this method is that there is variation in lead levels between individual fingers and toes from the same subject. Hair sampling techniques are non-invasive, but lead absorption by the hair seems to differ based on age, gender, ethnicity, and hair color. Additionally, it is difficult to distinguish internal lead absorption from external environmental presence on top of the hair. Saliva samples are easily collected but lead measurements across time points are not consistent. Lead readings change depending on the time of day the sample is collected, whether it is collected before or after a meal, or whether the sample is stimulated or naturally occurring. Given that urine, fecal, nail, hair, and saliva samples often produce inconsistent results, they are rarely found within the scientific literature (Barbosa, Tanus-Santos, Gerlach, &

Parsons, 2005; Tesman & Hills, 1994).

Developmental Effects of Lead Exposure

The effects of lead exposure on children are seen in many domains of development, but most prominently in intelligence/cognitive functioning and behavior. We will briefly review findings concerning large levels of lead exposure on neurodevelopmental functioning (studies focusing on lead levels above 10µg/dL) and will then present new evidence concerning the effects of lower levels of lead exposure (studies focusing on lead levels below 10µg/dL) in these same areas.

Intelligence/Cognition

High lead levels. Several studies have documented the effects of high levels of lead on children's intelligence. In 1979, Needleman and colleagues studied children with dentine lead levels between 12 and 54µg/dL. Researchers split the children into high- (m=35.5µg/dL) and low- (m=23.8µg/dL) lead groups. Children's intelligence and school experiences were assessed at age 6-7, and again when they reached 5th grade. Additionally, 39 control variables that might account for IQ performance were recorded, such as parents' IQ, child and parent SES, parental occupation, home environment, and parenting practices. Results indicated that dentine lead levels significantly related to performance. Children with high levels of dentine lead scored about a third of a standard deviation lower (a non-trivial difference) on the Full WISC-R (Wechsler, 1974) than those with low lead levels. Children with high lead levels also showed worse verbal processing.

Another study tested primary-school-aged children of skilled manual workers living in London (Yule, Lansdowne, Millar, & Urbanowicz, 1981). Children had BLLs between 7 and 32µg/dL with mean BLL being 13.5µg/dL. Results indicated a relation between BLL and IQ and verbal skills, statistically significant even after controlling for children's SES. These two studies are representative of much research examining IQ and lead exposure. It is now well established that lead levels greater than 10µg/dL negatively affect IQ, particularly reading and verbal skills.

Low lead levels. Research focusing on the effects

of low levels of lead exposure presents a more nuanced picture of the effect of lead on intelligence. Several methodologically rigorous, prospective longitudinal studies have examined the effects of lead on children's cognitive performance. Canfield and colleagues (2003) followed children with both low BLLs (<10µg/dL) and children with high BLLs (>10µg/dL) from age 6 months until age 60 months. Child intelligence was assessed using the Stanford-Binet Intelligence Scale-IV (Thorndike, Hagen, & Sattler, 1986). Several covariates were included in the analysis—child sex, birthweight, race, mother's IQ and years of education, tobacco use during pregnancy, and SES. Results indicated that BLL was significantly related to differences in IQ performance. Specifically, as BLL concentrations increased from 1 to 10µg/dL, IQ decreased an average of 7.4 points. This trend was seen in children with BLLs above 10µg/dL as well, but decreases in IQ score were less pronounced (a 2.5 point IQ drop when BLL rose from 10 to 30µg/dL).

Lanphear and colleagues (2005) prospectively followed children with a wide range of BLL until the age of 10. Information on children's sex, birth order, and their mother's age and marital status were included as covariates. Exposure to lead had a statistically significant effect on IQ as measured by the WISC-III. Specifically, 3.9 IQ points were lost when BLL rose from 2.4-10µg/dL while only a 1.9 point IQ drop was associated with a BLL rise from 10-20µg/dL, and a 1.1 point drop with a BLL rise from 20-30µg/dL. This study indicates that increases in BLL from 0-10µg/dL have a greater effect on IQ

than increases in BLL above 10µg/dL, and that even at low levels of exposure, increasing lead level is related to decreases in intelligence and performance.

Several cross-sectional studies have also shown effects of low-level lead exposure on children's cognitive performance, confirming the results of the longitudinal research reported above. Lanphear, Dietrich, Auinger, and Cox (2000) examined data from the Third National Health and Nutrition Examination Survey (NHANES III). Children in the study had BLLs of between 2.5 and 10µg/dL. Researchers evaluated children's performance on assessments of arithmetic skills, reading skills, nonverbal reasoning skills, and short-term memory using the WISC-R (Wechsler, 1974) and the Wide Range Achievement Test

Several studies have documented the effects of high levels of lead on children's intelligence.

(Jastak, 1984). Covariates included gender, race/ethnicity, poverty, region of the country, parent educational level, marital status, the child's serum ferritin level (blood iron level), and the child's serum cotinine level (measure of exposure to smoking). For every 1µg/dL increase in BLL (up to 10µg/dL), there was a .7 point decrease in arithmetic score, a 1 point decrease in reading score, a .1 point decrease in non-verbal reasoning tasks, and a .5 decrease in short term memory. Given that the standard deviation on these measures is 15, and the point decreases reported have to do with just a 1µg/dL increase in BLL, the difference in cognition for children with, say, 10µg/dL compared to 2µg/dL is clinically important.

In a similar study, Kordas and colleagues (2006) examined children with BLLs between 0 and 45µg/dL. Children between the ages of 6 and 8 years old were assessed using 14 different measures of cognitive achievement. Covariates included in this study were age, gender, SES, maternal education, parental involvement in schooling, family structure, birth order, and arsenic level. Researchers found statistically significant decreases in cognitive functioning associated with lead exposure. Specifically, an increase in BLL from 0-14µg/dL was associated with greater cognitive losses than BLL increases above 14µg/dL.

In 2007, Surkan and colleagues (2007) conducted a study on the effects of low levels of lead on children's intelligence. Children ages 6 to 10 with BLLs between 1-2µg/dL, 3-4µg/dL, and 5-10µg/dL were compared on the WISC-III (Wechsler, 1991). Intelligence was significantly related to age, race, SES, birthweight, parent IQ, and marital status so the researchers adjusted scores to account for these covariates. IQ was found to be significantly different between the 1-2µg/dL and the 5-10µg/dL groups, but not between the 1-2µg/dL and the 3-4µg/dL groups. On average, children with BLLs between 3 and 4µg/dL scored .12 points lower on the WISC-III compared to children with BLLs of 1-2µg/dL. However, children with BLLs of 5-10µg/dL were found to score 5-6 points lower on the WISC-III compared to children with BLLs of 1-2µg/dL.

A recent study by Hornung, Lanphear, and Dietrich

... the effect size for lead's influence on cognitive outcomes is similar in magnitude or greater than other well-known risk factors, such as poverty and maternal education.

(2009) examined children between the ages of 2 and 6. Researchers were interested in determining both the effects of lead on intelligence, and the age when lead exposure has the greatest effect on IQ. Children's BLL was collected at ages 2 and 6. At age 6, children were assessed using the WISC-R (Wechsler, 1974). Researchers used a multiple regression model to determine the effect of children's past and current BLL on IQ. Researchers controlled for home environment, birthweight, maternal IQ, and maternal education. After accounting for lifetime lead exposure, results indicated that having a higher BLL at age 6 as compared with age 2 was associated with lower IQ scores at age 6. In fact, children who had greater BLL levels at age 6 had an estimated 5.3 point loss in IQ compared to children whose BLL had peaked at age 2. This re-

gression model predicted even greater proportional losses in IQ when analysis was restricted to children with BLLs of ≤10µg/dL. This study indicates that current, rather than past, BLL is a better predictor of intellectual outcomes, which highlights the importance of reducing and treating lead exposure when found in later childhood. Importantly, these authors also show that the effect size for lead's influence on cognitive outcomes is similar in magnitude or greater than other well-known risk factors, such as poverty and maternal education.

Although we have only highlighted a few recent studies, it is important to note that the evidence is quite robust, with many other investigations also finding negative effects of low levels of lead on children's cognitive skills (Al-Saleh et al., 2004; Bellinger et al., 1991; Bellinger, Stiles, & Needleman, 1992; Emory, Ansari, Pattillo, Archibold, & Chevalier, 2003; Jusko et al., 2008; Needleman & Gatsonis, 1990). Also worth noting is that these studies typically control for a whole host of other family and environmental factors known to correlate with intelligence. So the sizable effects observed here are net of other important factors associated with negative child outcomes showing that lead exposure, specifically, is indeed harmful to children's development.

Behavior

Similar to IQ, behavioral deficits have been seen in children exposed to both high and low levels of lead. Research on the behavioral effects of lead most often focuses on aggression, hyperactivity, and attention problems. As before, we will first discuss research focusing on high levels of lead exposure and will then discuss studies on low levels of lead exposure.

High lead levels. Many studies (Factor-Litvak, Wasserman, Kline, & Graziano, 1999; David, 1974; Ris, Dietrich, Succop, Berger, & Bornschien, 2004; Roy et al., 2009) have detailed the effects of high levels of lead on children's behavior. A study conducted in 1992 by Sciarillo, Alexander, and Farrell compared children with high BLLs (27.8 $\mu\text{g}/\text{dL}$) to children with low BLLs (9.2 $\mu\text{g}/\text{dL}$). Child behavior was measured using the *Child Behavior Checklist* (CBCL; Achenbach & Edelbrock, 1983). Researchers controlled for age of mother, maternal education and depression, parental employment, parental marriage status, and number of children currently in the household. Children with higher levels of lead were found to score higher on the Total Behavior Problem scale that includes both internalizing and externalizing behavior problems. Specifically, children in the high-lead group were 2.7 times more likely to score in the clinical range for behavior problems.

In 1994, Bellinger, Leviton, Allred, and Rabinowitz studied children with dentine lead levels between 0.1 $\mu\text{g}/\text{g}$ and 35 $\mu\text{g}/\text{g}$. This study was specifically interested in how behavior changed as dentine lead level increased. Children's behavior was rated by their teachers using the Teacher Rating Scale of the *Child Behavior Profile* (Conners, 1969). Researchers controlled for SES and maternal characteristics. Results indicated that increases in tooth lead levels were associated with more internalizing and externalizing behavior problems. In addition, extreme behavior profiles were disproportionately identified in children with the highest tooth lead levels.

In a similar study (Needleman, Reiss, Tobin, Biesecker, & Greenhouse, 1996), the behavior of children with low (<15 $\mu\text{g}/\text{dL}$) and high (>15 $\mu\text{g}/\text{dL}$) bone and blood lead levels was examined. The researchers were interested in how behavior changed as bone lead level increased. Children's behavior was assessed at 7 and 11 years using the CBCL. In addition, every 6 months, children completed the *Self-Reported Delinquency Scale* (Elliott, Huizinga, & Ageton, 1985) and the *Self-Reported Antisocial Behavior Scale* (Loeber, 1989). The researchers accounted for the effects of maternal intelligence, SES, and quality of child

rearing. Children in the high-lead group were more likely to be rated by their parents and teachers as aggressive, more delinquent, and to report more somatic complaints compared to their low-lead peers.

In yet another study conducted by Mendelsohn and colleagues (1998), the behavior of children with BLLs between 10 and 29 $\mu\text{g}/\text{dL}$ was examined using the Behavior Rating Scale of the *Bayley Scales of Infant Development* (Bayley, 1969). Researchers identified six variables that were related to behavior (child's age and gender, mother's age, verbal IQ, depression, and provision of cognitive stimulation) and included these variables in their regression models to account for their effects on child behavior. Results indicated that greater BLLs were associated with increased ratings of hyperactivity, distractibility, and frustration. The studies above indicate that high levels of lead are associated with increases in aggressive and destructive behavior and inattention, and that behavior problems increase as child lead levels increase.

Low levels of lead. Although it is well established that high levels of lead contribute to behavior problems in children, studies that include children with low levels of lead are less numerous. In a study of infants with a wide range of BLLs (between .52 and 25 $\mu\text{g}/\text{dL}$), Plusquellec and colleagues (2007) examined infant behavior using the *Bayley Scales of Infant Development* and observer ratings of child behavior. Several factors were controlled for including parental education, maternal distress, maternal intelligence, home violence, SES, pre-natal exposure to drugs, birth complications, and child characteristics (age, gender, etc.). Infants with BLLs as low as 4.5 $\mu\text{g}/\text{dL}$ showed a statistically significant increase in hyperactive behaviors and decreased attention spans. This study indicates that BLLs below 10 $\mu\text{g}/\text{dL}$ can affect child behavior.

Braun, Kahn, Froehlich, Auinger, and Lanphear (2006) used nationally representative data collected during the 1999-2002 National Health and Nutrition Examination Study (NHANES) to examine the relationship between BLL and ADHD diagnosis in children between the ages of 4 and 15. Researchers established ADHD status through parental report of child diagnosis and report of doctor prescription for ADHD medications. Researchers examined several covariates including child age, race, gender, SES, health insurance coverage, pre-school attendance, birth weight and complications, and blood iron levels. Logistic regression analysis indicated that BLL was a statistically significant indicator of ADHD diagnosis in children. This relationship was found even after researchers restricted

their analysis to children with BLLs of $\leq 5\mu\text{g}/\text{dL}$. These researchers estimated that 21.1% of ADHD cases nationally, in children between the ages of 4 and 15, were attributable to having a BLL of $>2\mu\text{g}/\text{dL}$.

Chiodo, Jacobson, and Jacobson (2004) examined children with diagnoses of ADHD. After adjusting for 19 control variables (e.g., SES, age, parental marital status, parental education, gender, parenting quality, alcohol and drug use, and the home environment), higher lead levels were associated with greater ratings of ADHD behaviors, and significantly higher inattention scores on the *Barkley-DuPaul Attention Deficit Hyperactivity Scale* (Barkley, 1990). Children with higher BLLs were also rated by teachers as having poorer attention. Regression analysis in this study indicated that attention problems could be seen in children with BLLs greater than $3\mu\text{g}/\text{dL}$, suggesting a possible threshold value for lead exposure.

Similarly, in 2008, Wang and colleagues studied children with BLLs between 5 and $10\mu\text{g}/\text{dL}$. Researchers used a pair-match design to control for effects of age, gender, and SES. Results indicated that children with BLLs between 5- $10\mu\text{g}/\text{dL}$ were found to be significantly more likely (3.5 to 7 times) to be diagnosed with ADHD than children with BLL less than $5\mu\text{g}/\text{dL}$. This study complements research conducted by Nigg and colleagues (2008) where children already diagnosed with ADHD were assessed for levels of lead exposure. The sample had very low exposure levels (average BLL for ADHD-combined type = $1.26\mu\text{g}/\text{dL}$), consistent with national averages, but results indicated that as lead levels increased from 0 to $3.4\mu\text{g}/\text{dL}$, levels of hyperactivity and impulsivity in those with ADHD-combined type increased significantly. The results of this study have recently been replicated (Nigg, Nikolas, Knottnerus, Cavanagh, & Friderici, in press).

The levels of lead examined in these studies are commonly found in children in the U.S. and thus provide evidence of the possible effects of lead on a large proportion of American children. These studies provide further clear support that levels of lead below $10\mu\text{g}/\text{dL}$ increase a child's risk for attention and behavioral problems. In addition, they provide evidence that lead exposure is related to increased risk of developing clinically significant attention and behavior problems.

Other Child Outcomes

We have seen that children exposed to both high and low levels of lead show cognitive deficits and disturbed behavior. Two long-term outcomes associated with these deficits can be seen in school performance and criminal

behavior. Several studies have found that lead exposure has a negative impact on behavior and school performance, and in this section we will describe just a few. In 1984, Bellinger, Needleman, Bromfield, and Mintz studied the school performance of 141 elementary school children who were classified as having either elevated ($>20\text{ppm}$), mid-range ($10\text{-}19.9\text{ppm}$), or low ($<10\text{ppm}$) dentine lead levels. Their study indicated that increases in dentine lead levels were associated with worse school performance. Additionally, students with higher dentine lead levels were more likely than their peers to repeat a grade. A longitudinal study of children exposed to lead was conducted by Needleman, Schell, Bellinger, Leviton, and Allred (1990). Children were assessed at 7 years of age and again at 18 years. Researchers considered maternal age and IQ, SES, family functioning, number of siblings, race, and past medical history as covariates. As BLL in children increased, so did their likelihood of not graduating from high school. This effect on drop-out was also seen in children with BLLs below $10\mu\text{g}/\text{dL}$, but was more pronounced at higher levels of lead exposure.

Wang and colleagues (2002) found that elevated lead levels were negatively associated with student achievement. After controlling for possible confounds due to SES and maternal education level, children's academic performances in the areas of math, science, history, and language were all significantly negatively associated with BLL. In a final study, Fergusson, Horwood, and Lynskey (1997), collected dentine lead levels of children at 8 years of age and assessed their academic and intellectual performance at age 18. Measures of mother's education, responsiveness and punitiveness, and father's occupation/SES were collected and incorporated into the analysis. As dentine lead increased, so did the likelihood that children would fail to complete high school. The amount of dentine lead present in children was also negatively related to the number of educational certificates the students completed.

A second outcome often seen in children exposed to lead is criminal activity (Nevin, 2007; Stretesky & Lynch, 2004). In a 2001 study, Dietrich, Ris, Succop, Berger, and Bornschein examined the relationship between lead exposure and later criminal activity, analyzing data from the Cincinnati Lead Study. The researchers specifically assessed 30 possible covariates including SES, gender, maternal IQ, attendance in preschool, etc. The researchers found that lead levels as low as $2.5\mu\text{g}/\text{dL}$ were associated with significantly greater amounts of parent- and self-reported criminal activity and higher rates of police intervention.

Similarly, in 2008, Wright and colleagues measured the association between children's BLL from birth to 6 years of age and later criminal arrests. The subjects were contacted at 19-24 years of age, and past criminal activities were documented. Covariates included gender, birth weight, the quality of their early care giving, maternal drug use, maternal IQ, the total number of prior maternal arrests, and SES. Results indicated that increased lead levels were significantly associated with increases in the number of total arrests and violent crimes committed by the participants in adulthood. Specifically, every 5µg/dL increase in BLL during early childhood was associated with a 1.07 increase in the number of total crimes the subjects had committed, and a 1.3 increase in the number of violent crimes for which the subjects had been arrested.

Societal Costs

From the previous discussion, we have seen that lead negatively impacts children's intellectual and behavioral development and that the long-term consequences of this exposure result in lower school performance and greater instances of criminality in adulthood. Studies have indicated that the implications of lead exposure are not just intellectual, behavioral, or social, but monetary as well. The general medical treatment of a child with lead exposure is between \$100 and \$5,200 (CDC Cost of Illness Handbook, n.d.), but the long-term losses in relation to economic earning, tax contributions, and educational assistance can be much greater. In 1994, Schwartz conducted a cost analysis associated with children's lead exposure. He concluded that a reduction of 1µg/dL nationwide would result in a total benefit of \$5.06 billion per year in earnings per annual birth cohort. In 1995, Schwartz reconsidered his estimates based on data from the National Longitudinal Study of Youth and recommended a 50% increase in his benefit estimates. This would bring the estimated economic benefit associated with a 1µg/dL drop in children's BLL to \$7.56 billion per annual birth cohort. In an additional study, Landrigan, Schechter, Lipton, Fahs, and Schwartz (2002) explored the economic impact of lead exposure and showed that in 1997, children 5 years of age would lose \$43.4 billion in future earnings due to IQ loss associated with lead exposure. Similarly, in 1991, the CDC estimated each 1µg/dL drop in a child's BLL was associated with an increase of \$1,147 in later lifetime earnings (CDC, 1991).

Gould (2009) also demonstrated the economic costs associated with lead exposure and the economic benefits of lead reduction. Gould estimated that \$11-53

billion are spent on health care costs associated with lead exposure treatment. Additionally, she estimated that the lowering of IQ due to lead exposure results in \$190-268 billion in lost earnings and lost tax revenue. The costs associated with increases in special education needs and ADHD were estimated at an additional \$297-413 million, and increased associated crimes cost society \$1.7 billion. In sum, the overall cost of lead exposure can be estimated at \$192-270 billion. Gould estimates that lead hazard control practices would likely cost under \$11 billion. Therefore, for each dollar invested in lead hazard control, \$17-221 would be returned through increased income and tax contributions, and health, crime, and special education savings.

The monetary benefit of reducing children's exposure to lead is greater than the monetary benefits seen for vaccinating children against common diseases (\$5.30-16.50 saved for dollar spent on vaccinations). Vaccination programs are widespread and generally accepted as worthwhile by society. Based on this information, lead reductions should also be a socially promoted priority. Thus, lead exposure affects not only personal achievement, intelligence, and behavior, but impacts society as a whole. The personal and economic implications of exposure are great. It is for this reason that it is imperative that we increase efforts to reduce children's exposure to lead. We now turn our attention to the prevention of lead exposure in children. Sources of environmental lead will be identified and methods of lead abatement discussed.

Lead Abatement

Childhood exposure to lead remains a problem, but before we can address ways to prevent this exposure, we must understand the sources of lead in children's environment. Historically, one of the most recognized sources of childhood lead exposure is leaded gasoline. In 1973, the EPA began to reduce the amount of lead used in gasoline fuel, and by 1996, the sale of all leaded gasoline in the U.S. was banned. In 1999, Thomas, Socolow, Fanelli, and Spiro conducted a review of 19 studies describing the effects of leaded gasoline on lead exposure. The studies discovered that the elimination of lead in gasoline in the United States was associated with .8µg/dL drop in citizen BLLs per year. The elimination of lead in gasoline greatly reduced air-related lead exposure in children. Although lead in automobile gasoline has been banned, no such regulations exist for jet fuel. The presence of lead in jet fuel, and the presence of aerosolized lead from industrial sources are likely the reason why studies find that air-

related lead is still a significant source of lead contamination for children (Pirkle et al., 1998).

Children are particularly susceptible to contamination from lead-based paint and paint dust. In 1977, the U.S. Consumer Product Safety Commission banned the sale of leaded paint within the United States (Chisolm, 1986). This ban, however, did not affect houses built and/or painted before 1977. Lead dust and paint chips settle onto the floors of homes. Young children crawl on floors and mouth objects that have been on floors, activities that enable consumption of the lead (Lidsky & Schneider, 2003).

Children's exposure to lead through contact with contaminated soil is also a common occurrence. Lead levels in soil are highly correlated with lead levels found in air, dust, and paint. Air lead eventually settles on the ground and contaminates the soil. Similarly, lead-based paint chips and dust from the exteriors of older houses fall to the ground and mix with the soil. Unlike air-based or paint-based lead, soil lead is long lasting and can persist for months after the reduction of air levels or the removal of leaded paint (Weitzman et al., 1993).

Another important source of lead exposure in children comes from tap water. In 1986 and 1996, new amendments to the Safe Drinking Water Act required public water distribution systems to consistently check their drinking water for lead and to enact abatement services if lead levels were found in excess of the allowable action level (currently set at 15µg/L for public drinking water, and 20µg/L for water fountains in schools and childcare centers) (EPA Press Release, 1986, 1996, EPA 2004a, EPA 2006). Lead leeches into tap water through contact with lead-based piping or through the corrosion of pipes in water treatment systems and in household plumbing. PH imbalances in water may promote corrosion of pipes (CDC, 2002). This corrosion is particularly destructive as it can be hard to predict, and replacement of pipes can be costly.

An emerging area of concern is the presence of lead contaminated toys. The CDC identified leaded paint and leaded plastic as two potential sources of lead for children (CDC, 2009). Although lead paint was banned from houses, food containers, and children's products in 1978, it is still widely used in other countries. Therefore, imported toys may still contain amounts of lead. Additionally, lead is often used to soften plastics,

making them more flexible and resistant to heat. When these plastics are exposed to sunlight, air, or detergents, the chemical bond between the lead and the plastic can break, creating leaded dust. In rare cases, lead may be used as part of a base for metallic toys. Exposure to lead from toys occurs when children mouth, chew, or swallow the toys.

Having identified the five main sources of lead exposure to children (air, paint/paint dust, soil, water, and toys), we will examine the ways that lead can be removed from children's environments. Since the elimination of lead from gasoline in 1997, the most prominent contributors to air lead levels are found in the industrial sector.

The EPA estimates that 42.8% of lead in air comes from industrial processes (EPA, 2002). Many commercial enterprises, from food processors to plastics manufacturers, put off potentially harmful lev-

els of lead during production. Three main procedures are recommended for the reduction of lead air levels from industrial sources. Most industrial lead abatement procedures can be accomplished through the use of dry systems, wet scrubbers, and electrostatic precipitators. Dry systems use gravity, filters, or centrifugal forces to trap lead in air, while wet scrubbers use water streams to increase the efficiency of lead collection. Electrostatic filters work by creating an electrostatic attraction that traps pollutants before they reach the atmosphere. The use of these three methods has been shown to significantly reduce the amount of lead released into the atmosphere (Hartman, Wheeler, & Singh, 1994).

Lead abatement of contaminated paint and paint dust centers around removal, replacement, or encapsulation of the original lead paint. Paint removal is also paired with a concerted cleaning effort to reduce the amount of loose lead dust found in homes. Removal practices center around the complete removal of structures within the home that have been contaminated with lead paint. Replacement procedures seek to replace lead-contaminated materials with appropriate non-lead painted products, while encapsulation methods seek to seal lead paint behind a barrier (such as varnish) so that it can no longer chip or create lead paint dust (Virginia Department of Historic Resources, 1993). Studies indicate that traditional methods of removal and encapsulation do result in significant decreases in household lead, although

42.8% of lead in air comes from industrial processes.

maintenance is needed to provide optimal reduction (Farfel, Chisolm, & Rhode, 1994). About 80% of houses built before 1950 are estimated to contain lead paint (Needleman, 2004). In 1991, abatement for paint in these houses (over a 30-year period) was estimated at \$33.1 billion. While this seems expensive, the benefits from lead paint abatement were estimated conservatively at \$61.7 billion (Needleman, 2004). Therefore, abatement of lead paint would provide an overall \$28.6 billion in savings.

Cleaning procedures designed to remove lead dust from the home often include vacuuming and wet dusting of household surfaces, especially those that may be a source of lead contamination. In addition to cleaning these surfaces, residents are encouraged to wash their hands regularly. These are done in an attempt to reduce the amount of lead dust that settles from unabated structures and to prevent accidental ingestion of the lead dust. Studies have indicated that while dusting will remove lead-based paint in the short term, long-term BLL is dependent on permanent removal or encapsulation of the lead paint (Lanphear, Eberly, & Howard, 2000). Additionally, studies have found that while vacuuming reduces 95% of lead dust on hard floors, it is not an effective method for removing lead dust from carpets (Ewers, Clark, Menrath, Succop, & Bornschein, 1994).

Hand washing regimens are another method often implemented in an effort to reduce child lead intake within the home. Children and adults are encouraged to wash their hands before meals and after playing outside (if they have known soil contamination). A study conducted by Lanphear and Roghmann (1997) sought to determine the pathway of lead into children bodies. The researchers measured child lead levels and several factors that might contribute to elevations or reductions in child lead levels. Behaviors such as eating dirt, sucking thumbs, hand washing, mouthing, and vacuuming were investigated. Results indicated that hand washing before eating and hand washing after playing outside were not significantly related to child BLL, although these easy preventative steps (along with cleaning nails and frequent nail clipping) are still typically safely recommended for families as easily performed acts that might help.

Soil abatement practices include removal of contaminated soil and replacement with clean soil. Generally 15-20cm of topsoil is removed and replaced during the abatement process. Farrell, Brophy, Chisolm, Rohde, and Strauss (1998) found that soil replacement did not significantly lower children's lead levels. In a similar study,

Aschengrau, Beiser, Bellinger, Copenhafer, and Weitzman (1994) found that soil abatement was effective but only for higher income persons who washed their hands before meals, had low initial lead levels, and who were away from home often. Children living in apartments where dust was present derived no benefit from the soil abatement. Studies seem to indicate that soil replacement is effective only if re-contamination of the soil does not occur. Soil replacement must be done in conjunction with exterior lead removal to ensure that soil is not re-contaminated (Weitzman et al., 1993).

Abatement of lead in water systems occurs mainly through the replacement of older pipes found to contain lead or treating the water so it is less corrosive. Additional methods of abatement include flushing of water systems before use, usually for 10 minutes. A study examining flushing practices in water systems of schools indicated that lead levels do reduce immediately after flushing but rapidly rebound, and seem to increase with frequent tap use (Murphy, 1993). If flushing is to be a useful way to reduce lead in water it must be done frequently throughout the day to prevent reestablishment of lead within the drinking water.

When considering removal of lead pipes as a method of abatement, it is important to remember that water service providers are only responsible for replacing pipes directly connected to their systems, so any internal piping within the home must be replaced by the homeowner. The D.C. water system has a program to replace lead pipes that are part of the public water system. WASA will replace lead pipes between the main line and homeowner's property line if the homeowner agrees to replace lead water lines on their private property (Quander-Collins, 2008). The District of Columbia replaces private lead pipes for the cost of \$100 per foot (plus a \$500 fee to extend the pipe into the home), and provides loans and grants to qualifying homeowners for the purpose of replacing their lead pipes (D.C. WASA, 2007; D.C. WASA, 2009). For homeowners who do not qualify for grants, or who live in a city without such a program, pipe replacement can be very costly, causing some homeowners to leave old water pipes in place even after the threat of lead is known (CDC, 2002).

Current State of Lead Control Policy

Two of the main agencies working to prevent the public's exposure to lead are the EPA and the CDC. The EPA's main goal is the creation and enforcement of environmental regulations and the protection of natural resources. The

EPA's role in lead exposure mainly concerns the promulgation and enforcement of regulations concerning lead levels in water, air, soil, paint, and drinking water (EPA, n.d.). The EPA sets action levels or levels of concern for lead in water, soil, and air. An action level is a threshold level, over which certain treatment requirements must be enacted. A level of concern relates to an amount of a substance that can cause harm to general populations. According to the Agency for Toxic Substances and Disease Registry (ATSDR, 2007), the current EPA action level for lead in drinking water is 15ppb. For soil, the EPA has set the level of concern (for federally funded projects) at 400ppm by weight in child play areas and 1200ppm by weight in non-play areas. The EPA's level of concern for ambient air is currently set at $.15\mu\text{g}/\text{m}^3$. In addition to setting the allowable environmental lead limits, the EPA also sets lead testing requirements for both public and private service providers. The CDC's primary goal is to develop and then apply disease prevention and control practices with the aim of improving public health (CDC, 2009a). As such, the CDC plays a major role in the establishment of allowable BLLs and in screening and reporting practices. Currently the CDC has set the allowable BLL limit at $10\mu\text{g}/\text{dL}$ (ATSDR, 2007).

Although both entities strive to prevent lead exposure, their regulations are not typically followed well. In a 2004 report, the EPA revealed that only 23% of water systems had reported their lead testing results as required (EPA, 2004b). Analysis of these water testing reports indicated that between 2000 and 2004, 29 states and D.C. had water systems test above the EPA allowable $15\mu\text{g}/\text{L}$ water lead level (EPA, 2005). Indeed, 4.2% of all water systems sampled had at least one test above the allowable lead water limit.

The CDC also is challenged in that they are not an enforcement entity and, therefore, have almost no way to require states to comply with their recommendations, however beneficial they might be. This leads to state and nationwide inconsistency in lead exposure practices. Currently, lead screening practices are created at the state level, with each state identifying and agreeing on its own lead screening guidelines (CDC, 2005). States vary widely in their approach to lead screening. Most states have a plan targeting children under the age of 6, but these plans vary greatly. Some state action plans are over fifty pages long while others are only three pages. Some states advocate universal screening (ex. Tennessee, Connecticut) while some advocate risk-based screening (ex. Illinois, Florida). Risk-based screening is usually ac-

complished through a parent questionnaire that identifies children who may be at higher risk for lead exposure and then only testing those at-risk. In addition, some states test children of certain SES designations, or who live in lower income areas or in older housing.

The CDC's 2006 national survey was answered by only 36 states and D.C. The map in Figure 1 shows data from this survey: each state's percentage of children screened for lead levels, the percentage of children tested found to exceed the minimum allowable lead level threshold, and the scope of the state's advocated screening approach (universal or risk targeted). Although states may create lead screening plans, they do not always follow their own guidelines. For example, Tennessee and Connecticut advocate universal lead testing, but had only tested 14% and 26% (respectively) of children less than 72 months of age. The average screening rate for states advocating universal testing was only 21.3% with Massachusetts (47%) testing the highest number of children and Kentucky (5%) the lowest. Among states that advocate risk-based testing, the numbers were even lower, with the average risk-based testing rate being 13.4%, with Minnesota as the highest test rate (22%) and Nebraska the lowest (.03%) (CDC, 2006).

Recommendations for Action

Researchers have known since the early 1900s that lead is harmful to children's development. When the CDC set the current BLL to $10\mu\text{g}/\text{dL}$ in 1991, reports were already beginning to appear that even lower levels of lead are detrimental to children's health. For the past 10 years, study after study has indicated that children are being exposed to unacceptable levels of lead in their daily lives, and that even a low level of lead exposure harms children. The U.S. is negligent in its testing, reporting, prevention, and treatment practices for lead exposure. Lead exposure in children is fully preventable, yet the U.S. government has failed to commit fully to the resolution of the problem. Cost-benefit analyses show that it is a relatively inexpensive problem to solve, and its resolution would lead to great economic returns. Research reported in this paper clearly shows that stricter regulations work and lead to less exposure. The new data summarized here suggest that new policy action is needed for this well-known problem. In order to prevent further exposure of children to lead, we suggest four main types of action be taken, specifically: 1) primary prevention in the form of regulations limiting lead exposure, 2) second-

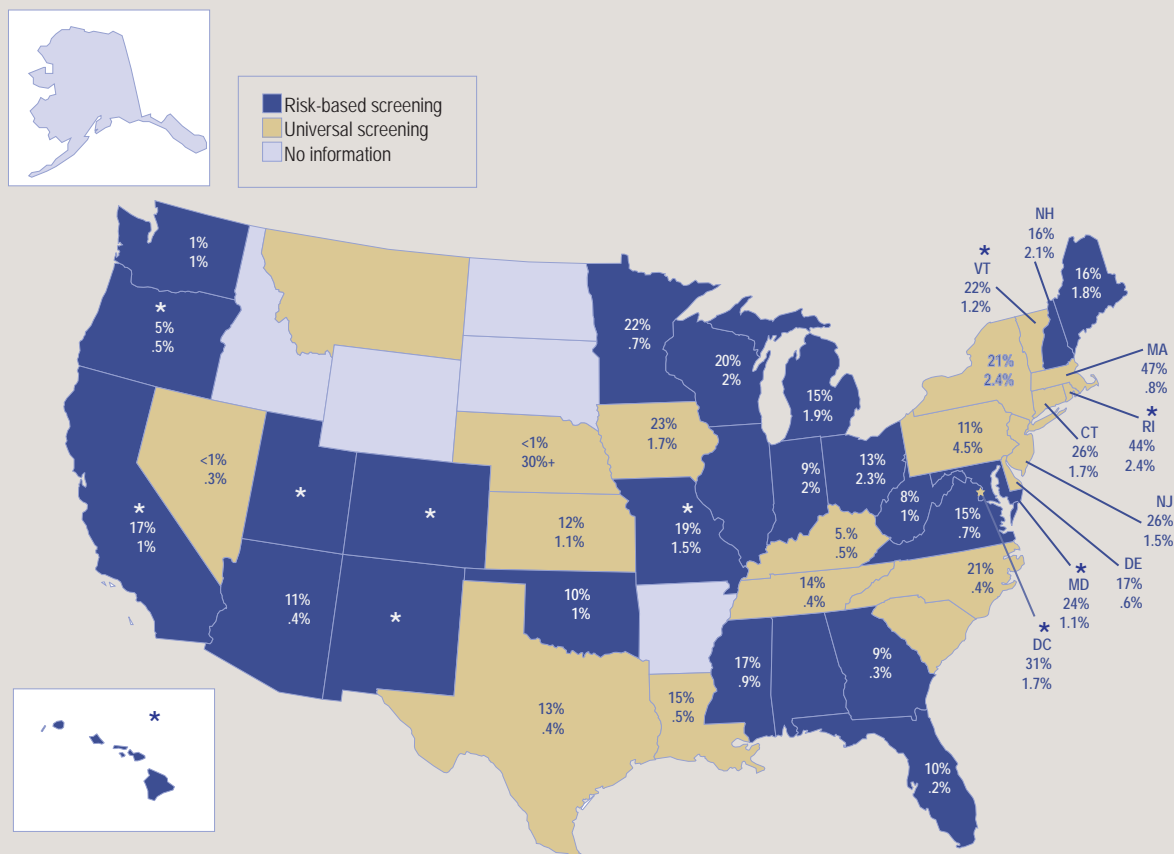
ary prevention via increases in education, guidance, and screening practices, 3) tertiary follow-up support and treatment for children with known lead exposure, and 4) greater organizational cooperation (see Table 1).

Our first main recommendation is to prevent the exposure of children to lead through increases in environmental lead regulations, enforcement, and abatement practices. The detrimental effects of lead on children could be completely eliminated if children were not exposed to lead in the first place. In the previous sections, we mentioned several environmental sources of lead and the many ways that lead from environmental sources may be eliminated.

The most important recommendation may be simply

to lower the acceptable BLL for children. Currently, the CDC sets the allowable BLL for children at 10µg/dL (CDC, 2005). In the past, when new research has shown that current lead levels are unsafe, the CDC has lowered its acceptable lead threshold (CDC, 1991). It is clear that levels of lead far below the 10µg/dL threshold have noticeable negative effects on children’s intellectual and behavioral development (Lanphear, Dietrich et al., 2000; Surkan et al., 2007; Wasserman et al., 1998). Although current research indicates that there is no safe level of lead, we recommend setting the allowable limit of lead at least to 5µg/dL if not lower. Setting the threshold lower would allow assistance to be available for children with low-lead level contamination. Lowering the ‘ac-

Figure 1. Lead Screening Plans and Statistics by State



Notes

- The top number for each state is the percentage of children less than 72 months old who were screened for lead.
- The bottom number for each state is the percentage of screened children less than 72 months old with lead levels >10ug/dL.
- Risk based screening varies from state to state, with some states basing screening on completion of a risk questionnaire, on the SES of the child, on the area where the child lives, or on the age of their domicile. Nationwide, all children receiving Medicare benefits are required to be screened for lead.
- Testing guidelines for states marked with an * were retrieved from the individual states Government and Department of Health webpages. All other guidelines were retrieved from the CDC (2009b).

- State and local testing programs. Retrieved on April 25, 2009, from <http://www.cdc.gov/nceh/lead/programs.htm>. Many states did not report.
- The percent of children screened for lead and the percent of children testing >10ug/dL were retrieved from the CDC (2006). 2006 Case and Screening Rate Maps by County for Selected States. Retrieved April 25, 2009 from <http://www.cdc.gov/nceh/lead/data/national.htm>.
- + The abnormally high figure for Nebraska may be due to the fact that incomplete data from this state were reported to the EPA and only a small % of children were tested.

Table 1. Recommendations for Action

1. Increase Abatement Practices
 - ☒ Lower the Lead Action Level
 - ☒ Lower the CDC's allowable lead level from 10ug/dL to 5ug/dL
 - ☒ Encourage the development and use of lead-free jet fuels
 - ☒ Provide stricter enforcement of industry pollution practices
 - ☒ Require companies to publicly disclose factory lead emission levels
 - ☒ Provide incentives and assistance to aid homeowners with replacement of lead pipes
 - ☒ Require homeowners to test their houses for lead paint before they rent or lease their property
 - ☒ Mandate abatement if lead is found in a home
 - ☒ Provide monetary assistance for testing and abatement in low-income populations
 - ☒ Increase federal regulation to insure that imported toys and child-related products are lead-free
2. Increase Education and Screening
 - ☒ Increase education concerning lead exposure for the general population
 - ☒ Pay increased attention to education of at-risk groups
 - Those in poverty
 - Those living in older or low-income housing
 - Pregnant women
 - Families with children under 6 years of age
 - ☒ Increase screening
 - ☒ Create federally-mandated screening guidelines
 - ☒ Require universal testing of children under age 6
 - Annual screening should occur at yearly well-child visits
 - ☒ Require lead screening for pregnant women
 - ☒ Establish a national testing compliance system to track state progress
3. Increase Follow-Up
 - ☒ Provide immediate lead abatement assistance when an elevated BLL is detected
 - ☒ Provide subsidized consultation and abatement services for low-income families
 - ☒ Provide immediate psycho-educational evaluation to lead-exposed children
4. Increase Collaboration between the EPA and the CDC
 - ☒ Encourage further use of the National Lead Information Center
 - ☒ Make the NLIC the central point for compiling information on lead from both the EPA and the CDC
 - ☒ Provide internet access to NLIC information
 - ☒ Encourage collaboration between the government agencies and the research community
 - ☒ Encourage partnerships between various disciplines interested in studying lead exposure

ceptable' level of lead is a critical first step to implementing the additional regulations proposed below to reduce exposure.

Although lead in automobile gasoline has been banned, no such regulations exist for jet fuel. Jet fuel may enter into the environment during the burning of the fuel, through evaporation during transportation, or through spills (Faroon, Mandell, & Navarro, 1995). We recommend the similar removal of lead from jet fuel and/or the development and use of alternative fuel compositions that don't include lead. The airline industry should encourage the development of these fuels and the use of them once they become available. Further, since industrial sources contribute 42.81% of the lead in the air (EPA, 2002), we recommend stricter regulations and better enforcement of industrial pollution practices. Currently it is extremely difficult to find information on lead output from factories. Lead measurements from individual facilities should be publicly disclosed so companies can be held accountable for their compliance (or lack thereof) with clean air practices.

The presence of lead in water is also an area where more could be done to eliminate exposure. States should subsidize programs that offer low-cost identification and replacement of lead piping. Cities that are in the process of replacing lead pipes located on public property should offer greater incentives and assistance to homeowners in replacing lead pipes on their private property. The replacement of lead piping can be costly, and many home-owners would benefit greatly from increased assistance in removing this health hazard. Removal of public lead pipes will not appreciably reduce lead

exposure unless lead-based pipes on private property are removed.

Lead paint and lead paint dust are a third main contributor to environmental lead. Currently owners of dwellings built before 1978 are required to provide a statement to renters or prospective buyers that lead may be a problem in a home. Additionally they are required to disclose any information they might have regarding the presence of lead paint in a home (U.S. Department of Housing and Urban Development, 2008). We recommend that all homeowners in possession of a dwelling built before 1978 be required to test their home for lead before that home is sold or rented. If lead is found in a home, that homeowner should be required to take appropriate abatement steps. Additionally, testing and abatement of lead in homes should be subsidized for those of low SES, especially given that these populations tend to live in poorer quality and older housing that is more likely to have lead problems (Evans & Kantrowitz, 2002).

We also recommend increased federal regulation to ensure that toys and other child-related products manufactured in other countries and imported to the U.S. are lead-free. The most basic way we can protect children from lead exposure is to remove lead from their environment. Encouraging detection and abatement of current air-, water- and paint-based lead sources will go a long way toward protecting our children.

Our second group of recommendations concerns increased education and screening practices. Among the general population, we must increase emphasis on the negative effects of lead exposure on children. Special attention should be paid to at-risk groups, such as pregnant women or low-income families with young children, groups who are more likely to live in older, inner-city, and/or low-quality housing with a greater risk of lead exposure. Outreach to both low SES groups and expectant mothers or mothers of small children could be accomplished in several ways. Pamphlets in the offices of pediatricians and obstetricians would assist with specifically targeting parents. Special consideration should be taken to target doctors serving lower income or Medicaid patients, and pamphlets should be written in a variety of languages to help target non-English speaking populations. Pamphlets should direct patients to additional resources, and it should be obvious who to call for screening, environmental testing, or help with abatement. Television, internet, and radio announcements would also be effective in reaching a large percentage of the population. Announcements could be

performed in numerous languages on different stations based on the targeted population.

Screening practices at the state and national level should also be increased. Several states have no identifiable lead screening plan and are not reporting screening information to the CDC. Additionally, we know that many states only screen based on risk level and that no state (even those who advocate universal screening) tests all children. We therefore recommend creating federally-mandated screening practices for children under the age of 6 and for pregnant women. A federally-mandated screening requirement would allow for more uniform lead screening practices to take place. In addition to a federal mandate, a verification system should be put in place to ensure that states are complying with federal law. We should universally test children for lead. The first step in tackling this problem is obtaining good data on the magnitude of toxic lead exposure in our children and the amounts of lead present in our environment.

Screening of blood lead levels should begin prenatally. Lead passes through the placental barrier; therefore, pregnant women's exposure to lead can harm the fetus. If our goal is to protect children from lead exposure, then it is only natural that we begin with this group. After birth, children should be screened annually, with low-income children's testing being covered by Medicaid insurance. Annual screening will allow children's lead exposure to be tracked, and early detection will allow abatement procedures to remove the source of lead from the child's environment. Early screening will not only provide swift identification and intervention opportunities for children, but also save parents, schools, and society money in the long run.

Our third main recommendation involves follow-up practices for children found to have elevated BLLs. If a pregnant woman or child is found to have an elevated BLL during a pre-natal checkup or during annual screening, assistance with lead abatement should be immediately offered. The reduction of environmental lead is not effective unless the source of the lead is removed (Lanphear, Winter, Apetz, Eberly, & Weitzman, 1996); therefore, assistance should be offered in the identification and removal of sources of lead from the environment. Additionally, children who are found to have elevated levels of lead should be tested for learning and behavior problems. The effects of lead on cognition and behavior are well known (Lanphear, Dietrich et al., 2000; Surkan et al., 2007; Wasserman, Staghezza-Jaramillo, Shrout, Popovac, & Graziano, 1998). Early identification of these

problem areas in children could help reduce the long-term effect of lead exposure on their future achievement and functioning. The neurodevelopmental effects of lead are far reaching, and reduction of lead exposure will produce better outcomes for children in the long run. Better childhood outcomes will reduce the amount of future resources the state has to spend on special education and criminal justice programs.

One final recommendation is to promote better communication between the EPA, the CDC, researchers, and the public on the issue of lead exposure. Information concerning lead is scattered throughout several government agencies and websites. The information is often difficult to find and contradictory. The EPA and the CDC jointly contribute to the National Lead Information Center (NLIC) (<http://www.epa.gov/lead/pubs/nlic.htm>), a place where homeowners and interested persons can find information about lead on a variety of topics. The information may be requested over the phone or on the internet, and the information is either faxed or mailed to the requestor. Although this collaborative center is a valuable resource, we believe its utilization could be improved. This center could become a central location for the collection and publication of new information and rules and regulations concerning lead. Collecting and organizing information in one location would provide a better organized and inclusive view of the many facets of lead exposure. Information concerning environmental lead levels could be more easily coupled with blood screening information, resulting in a better understanding of the causes and effects of lead exposure. In addition, the multitude of information provided through this center could be uploaded to the NLIC website to allow for faster and easier access to its information.

In addition to cooperation between the EPA and the CDC, there should be more interaction between these government organizations and the research community. Researchers interested in studying the effects of lead come from many different disciplines, including persons from the fields of environmental science, toxicology, medicine, psychology, and education. Lead prevention activities and lead research could be greatly increased should communication between and within these groups and the government be encouraged. At present, it would

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appear there is little communication between these disciplines and the agencies that create and enforce lead policies and regulations. The CDC creates recommendations that guide acceptable exposure levels and testing practices for lead in humans, while the EPA creates policies, regulations, and testing practices concerning lead in the environment. But the link, for example, between how much lead is found in water systems and what that means for blood lead levels in children is not at all clear. The EPA, concerned with amounts of lead in the environment, needs to better communicate with the CDC, which is responsible for information concerning lead in children. These two agencies then need to address the scientific community and foster more cooperation between themselves and interested researchers.

Finally, applied developmental psychologists and interventionists working with children and families need to be cognizant

of the possibility that lead exposure may be present for families and may be a significant contributor to the child behavior and cognitive problems observed. We know that lead affects children's behavior, intelligence, and attention. As such, lead exposure reduction needs to become a more central component of home-visiting, early education, and early intervention programs that are currently underway. Only with this combined cooperation can the issue of lead exposure in our children be fully addressed.

Although it is probably not possible to eliminate lead entirely from all children, a lowered lead exposure threshold would help reduce most of the negative effects seen at higher levels of exposure. Lead is detrimental to children's development, biologically, intellectually, and behaviorally. If we are to give our children the best opportunity to succeed, we must tackle the preventable and addressable problem of children's exposure to lead.

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If Ever A Time for Precaution

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Coole and Winsler rightly refocus our attention on the once-forgotten story of lead exposure and child health. The story of lead provides an object lesson for policymakers. Decades after lead came into routine consumer and industrial use, scientists are still grappling with its subtle yet extraordinarily costly effects on children's development. It has been horrifying to discover that much of the deleterious effect of lead on cognition and behavior occurs at the beginning of exposure—equivalent to exposures still commonplace in America. The unusual consistency of findings showing that lead is correlated, even at levels still typical in the U.S. population, with lower IQ and attention-deficit/hyperactivity disorder (ADHD) is sobering for a field accustomed to conflicting and ambiguous scientific reports.

The increasingly well-documented effects on ADHD are important because ADHD develops very early and is a precursor to conduct disorder, delinquency, substance use disorders (Mannuzza et al., 2008; Martel et al., 2008), and other outcomes of major concern to society. Attention problems predict academic failure over and above externalizing problems (Breslau et al., 2009). In our data, decrements in attention problems due to lead

exposure fully account for decrements in IQ, but not the reverse (Nigg et al., 2008), suggesting that lead damage to regulatory systems in the brain may also account for the well documented impacts on IQ. In short, the cascade of developmental effects beginning early in life that may be related to insults, like seemingly modest lead exposure, is of major concern to society.

Now policymakers, who many believed had dealt with lead a generation ago, have to grapple with two issues. The first is determining whether further reductions in societal lead burden are needed. The even more momentous issue is what to do about future potential neurotoxins. The regulatory policies of the past century have amounted in one sense to a colossal experiment on America's children, not only with lead but hundreds of other substances. What happens to children when exposed to lead? To the hundreds of new chemical compounds permitted in the past decade? To the dozens of new nanotechnologies now coming to market? Policymakers should learn from the lead experience that it may take decades for science to find the unfortunate answers, at enormous economic cost to society. Moreover, medical study of the health effects will never catch up with the pace of compounds being developed. Such an approach wastes scientific time and resources, diverting those efforts from finding cures to other serious disease.

These observations raise serious ethical and policy problems for domestic industry and government. Policymakers and industry need to grapple more honestly with applying a well-defined precautionary principle to potential neurotoxins—both chemical and nano—as is now required prior to the release of pharmaceuticals. Such an approach shifts the burden of proof for a potentially dangerous action from acting until proven dangerous, to waiting until proven safe. Extreme application of the principle can be rightly criticized, but reasonable and effective definitions, justifications, and applications are readily available (Fisher, Jones, & von Schomberg, 2006; Petrenko & McArthur, 2009) and have already been applied in international law and treaty (Fisher et al., 2006). Identifying the appropriate role of a precautionary principle in protecting children's health from potential neurotoxins is a policymaking discussion that is urgently overdue. This should be policymakers' take home realization from the present report.

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Commentary

Childhood Lead Poisoning: Designing Effective Public Policy

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As Congress and the Obama administration debate an overhaul of our nation's health care system, the emphasis of the national discourse is predominantly on medical care financing and insurance coverage. Discussions of public health, on the other hand, are notably absent. Although universal access to affordable medical care is necessary for a healthy nation, there is ample evidence that policymakers must look beyond the system of direct care to broader population-based initiatives. In no area is this more apparent than childhood lead poisoning, as Cole and Winsler describe in the present issue. Research has shown that at least 7 million children under the

age of six (or about 25% of children that age) could have lead levels high enough to induce developmental damage. Cole and Winsler's piece provides an extensive review of the biological and neurological effects of lead poisoning on these children and analyzes possible interventions. The authors conclude by offering an extensive set of policy recommendations for reducing children's exposure to lead.

While we concur with many of the recommendations forwarded by Cole and Winsler, we would have liked to see more discussion of the costs of each measure relative to their risk reduction and net benefits. In a world of fixed government resources, policymakers must ultimately choose a limited set of actions. We thus encourage a more complete cost-benefit analysis

of which recommendations would produce the largest gains in terms of population health. In particular we are wary of an increased focus on universal screening and medical intervention that could shift limited public health resources and medical attention away from at-risk populations (especially low-income and minority children) that are currently targeted for primary prevention.

Instead of increased laboratory screening for lead poisoning, some have called (see e.g. Brown and Meehan, 2004) for resources to be directed towards universal education for parents of lead hazards, better follow-up screening for infants that have elevated blood lead levels, improved coordination between state and federal governments, better risk factor screening, and household lead abatement (indeed, all suggestions

later offered by Cole and Winsler). Our own research has highlighted the cost-effectiveness of household lead abatement (Gould, 2009). This is especially true if household interventions are targeted towards historically at-risk neighborhoods and geographic areas. The emphasis of childhood lead policy thus ought to move towards more primary prevention of poisoning at their source.

Eliminating childhood lead poisoning is an economic and moral imperative, and ought to be pursued aggressively as part of a broader public health agenda. What form these policies should take, however, deserves careful attention to benefits (in the form of risk reduction) and costs, as well as unintended

consequences for other at-risk populations.

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Commentary

The Saturnian Predicament

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Lead is an ancient poison. Dioscorides, a Greek physician who lived in the first century A.D., wrote that lead makes the mind “give way” (Needleman, 2009). Lead poisoning, or Saturnism, was associated with Saturn by the alchemists because it was thought to be the most ancient of metals.

Saturn, the son of Earth (Tellus) and Sky (Caelus), was the supreme god or titan on earth. It was prophesized that he would be dethroned by one of his children. To retain his throne, Saturn devoured each of his children at birth. Saturn's predicament—losing his power or devouring his children—reflects our own predicament of losing a profitable poison or sacrificing our children to the toxic effects of lead.

Cole and Winsler have written a comprehensive review of toxicity and prevention of childhood lead exposure. There are a few points one might quibble about (e.g., it is unclear to what extent prenatal exposure elicits persistent effects on children). But more importantly, these two investigators have concluded what most objective scientists would if they took the time to study it; we have, for far too long, failed to

protect children from exposure to a substantial and preventable poison.

Their article is only the most recent in a series of pleas over the past century to prevent childhood lead poisoning. The first plea was published 100 years ago (Turner, 1908). After a decade of research and failed attempts to prevent lead poisoning by educating mothers, Turner concluded that “legislative interference” was necessary to protect children. In the 1920s, Alice Hamilton and Yandell Henderson argued—unsuccessfully—that the addition of tetra-ethyl lead to gasoline by the Ethyl Corporation would lead to cases of lead poisoning (Rosner & Markowitz, 1985; Rabin 1985). In the 1970s, research and legislation led to a reduction in allowable levels of lead in air, paint and water (Landrigan, Whitworth, Baloh, Staehling, Barthel, & Rosenblum, 1975; Needleman, Gunnoe, Leviton, Reed, Peresie, Maher, C., et al., 1979; Mahaffey, Annest, Roberts, & Murphy, 1882; Lanphear, Dietrich, & Berger, 2003). In the 1980s and 1990s, a series of studies implicating even lower levels of lead exposure with adverse effects on children’s intellectual abilities were published (Lanphear, Dietrich, & Berger, 2003; Needleman, Schell, Bellinger, Leviton, & Allred, 1990; Burns, Baghurst, Sawyer, McMichael, & Tong, 1999; CDC, 1991). In the 1990s, the Centers for Disease Control (CDC) and the World Health Organization lowered the acceptable level of lead in blood to 10 µg/dL for children (CDC, 1991; Tong, von Schirnding, & Prapamontol, 2000). Finally, in the first decade of the 21st century, another wave of research implicated lead as a risk factor for cognitive deficits and psychopathology at blood levels considerably lower than

10 µg/dL, prompting calls for the global elimination of all non-essential uses of lead (Wright, Dietrick, Ris, Hornung, Wessel, Lanphear, et al., 2008; Lanphear, Hornung, Khoury, Yolton, Baghurst, Bellinger, et al., 2005; Froehlich, Lanphear, Auinger, Hornung, Epstein, Braun, et al., 2009; Nigg, Knottnerus, Martel, Nikolas, Cavanagh, Karmaus, et al. 2008).

At each wave of research or advocacy, a handful of physicians, policymakers, scientists or community leaders were utterly convinced that there was sufficient evidence to protect children against lead poisoning through legislation. Unfortunately, despite some success in banning or reducing lead in gasoline, paint, industrial emissions, solder used in food cans and other consumer products, we continued to use it (Lanphear, et al., 2003; Tong, et al., 2000). It was simply too profitable to ban lead and too easy to dismiss any long-term consequences on children’s health.

Despite reductions in children’s blood lead levels (Jones, Homa, Meyer, Brody, Caldwell, Pirkle, et al., 2009), too many children still have blood lead levels indicative of lead toxicity. Moreover, while there has been a dramatic decline in lead toxicity among children in developed countries, the prevalence of lead toxicity in many developing or industrializing countries is epidemic (Tong et al., 2000).

There is both renewed optimism and urgency about eliminating childhood lead exposure (Lanphear, 2007; Ramazzini Collegium, 2009). In many countries, childhood lead exposure is considerably lower today than at anytime in the past 50 years, and fewer than twenty countries continue to use leaded gasoline

(OECD, 1999). It is feasible to eliminate lead from paint and many other consumer products worldwide. The elimination of lead won’t be easy, but with concerted effort it could be the environmental equivalent of smallpox eradication. The myth of Saturn also offers some hope; Saturn’s son, Jupiter, ultimately deposed his father after his mother, Rhea, kept him from being devoured.

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