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A rationale for lowering the blood lead action level from 10 to 2 μg/dL☆

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Abstract

Fifteen years ago, in 1991, the U.S. Centers for Disease Control and Prevention (CDC) established 10 μg/dL as the lowest level of concern for children's blood lead levels. This value is extremely important because, historically, policy makers and public health officials generally have acted to remove sources of lead exposure only after the CDC's level of concern had been exceeded. A growing body of evidence, however, reveals that blood lead levels below 10 μg/dL may impair neurobehavioral development. There is now sufficient and compelling scientific evidence for the CDC to lower the blood lead action level in children. This review argues that a level of 2 μg/dL is a useful and feasible replacement. Although it can be argued, in turn, that no threshold for the health effects of lead is demonstrable, analytically a blood level of 2 μg/dL is readily and accurately measured and provides a benchmark for successful prevention. Lowering the level of concern would encourage and accelerate the investments needed to ensure that children are protected from lead exposure in their homes, schools, and play settings. Such a program would also offer economic advantages because of the coupling between lead, educational attainment, earnings and anti-social conduct. By lowering the blood action level, CDC will promote policies and initiatives designed to further reduce children's exposure to this potent developmental neurotoxicant.

Keywords: Lead; Children; Ethics; CDC; Regulation; Earnings; Assays

1. Introduction

"If we were to judge of the interest excited by any medical subject by the number of writings to which it has given birth, we could not but regard the poisoning by lead as the most important to be known of all those that have been treated of, up to the present time" (Orfila, 1817).

The human health consequences of lead exposure were recognized over 2000 years ago ("lead makes the mind give way", second century BCE) but, until about 30 years ago, these consequences were framed from the standpoint of clinical lead poisoning. Contemporary views of lead toxicity, rather than addressing traditional poisoning, emphasize the sensitivity of the developing nervous system to remarkably low environmental levels of lead exposure. The developmental hazards of lead are no longer disputed. The question confronting us is how to translate this information into health policy to provide public health professionals, as well as the general public, with guidance necessary to protect child development. It is a question that bestrides the intersections of science, public health, and regulatory policy.

In 1991 the U.S. Centers for Disease Control and Prevention (CDC) established a blood lead intervention level or acceptable blood lead level (BLL) of 10 μg/dL for children (CDC, 1991) along with specific intervention recommendations (Table 1). Fifteen years have passed since a parallel and failed commitment to prevent childhood lead exposure (Needleman, 1998). This commitment was preceded by a gradual lowering of what was considered to be an acceptable BLL in children, starting in 1960 with a CDC value of 60 μg/dL (Fig. 1). This gradual reduction in what was considered a "safe" or "acceptable" blood lead level tracked the evidence from research in both laboratory animals and humans that even lower levels of lead exposure induced harmful consequences. During

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Table 1
Current CDC management recommendations

Blood lead level (μg/dL)	Actions	Time frame for beginning intervention
<10	None	
10–14	Provide caregiver lead education. Provide follow-up testing. Refer the child for social services if necessary	Within 30 days
15–19	Above actions, plus: if BLLs persist (i.e., two venous BLLs in this range at least 3 months apart) or increase, proceed according to actions for BLLs 20–44	Within 2 weeks
20–44	Above actions, plus: provide coordination of care (case management). Provide clinical evaluation and care. Provide environmental investigation and control current lead hazards	Within 1 week
45–70	Above actions	Within 48 h
70 or higher	Above actions, plus hospitalize child for chelation therapy immediately	Within 24 h

Adapted from Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention (CDC, 2002). Online: http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm

the last 15 years, this trend has continued. Numerous studies have repeatedly demonstrated adverse neurodevelopmental effects, such as lowered IQ, at BLLs below 10 μg/dL (Canfield et al., 2003; Fulton et al., 1987; Lanphear et al., 2000, 2005; Walkowiak et al., 1998).

Despite the accumulating data, CDC and some policy makers believe that CDC should retain the current 10 μg/dL blood lead standard (Bernard, 2003). They argue that a program targeted at levels below 5 μg/dL offers little benefit for the increased costs that would be incurred. They also claim that there are ''no effective clinical interventions known to lower children's blood lead levels''.

One consequence of the current CDC action level, however, is that public health officials define ''lead poisoning,'' an ambiguous term to begin with, as an entity that occurs at BLLs above 10 μg/dL (Dugbatey et al., 2005). In this article, we address some of the scientific, ethical, social, economic, and public health considerations that support lowering of the acceptable BLL for children, and propose a modification of the CDC intervention recommendations (Table 2). We propose a criterion of 2 μg/dL because it represents a reasonable blend of scientific information, feasible policy alternatives, and analytical reliability.

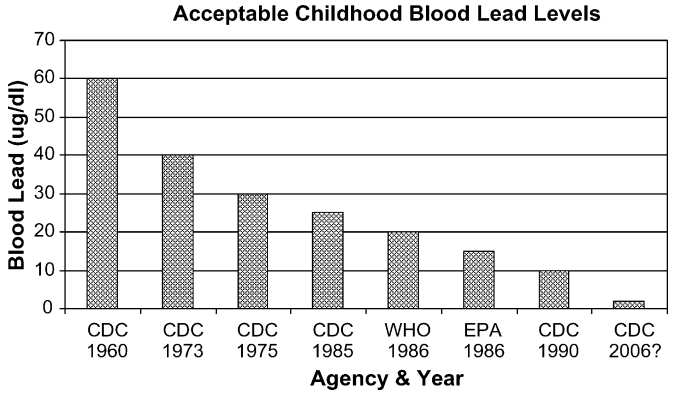

Fig. 1. The gradual decline in acceptable blood lead levels in children. The 2006 number is the recommend value based on current scientific knowledge.

2. Historical setting

Although the audience for this journal is generally aware of the history of lead toxicity, it bears repeating from the perspective of the current debate about lead exposure standards and, indeed, exposure standards for other neurotoxicants. Lead appears to have been first discovered and mined in Turkey in 6500 BCE. Its low melting point and malleability earned it recognition for its utility even at this early stage in human history. Those properties are also the reasons for its extensive exploitation and resulting deposition in the environment. The Romans widely mined and smelted lead from 500 BCE to 300 CE, which resulted in a spike in atmospheric lead release that was not eclipsed until the industrial revolution. Greek physicians provided the first clinical description of the health effects of lead in 100 BCE.

Widespread commercial use of lead soared with the recognition that lead-based paint was both highly protective and durable. The hazardous properties of lead pigments did not go unrecognized, however (Table 3). In 1887 a U.S. medical report documented childhood lead poisoning that, in 1904, was linked to lead-based paint. European governments moved to ban lead-based paints in the early 1900s, culminating in a ban by the League of Nations in 1922. Despite reports of childhood deaths related to consumption of leaded paint on cribs, the U.S. did not begin officially to phase out lead-based paint until 1971, with the passage of the Lead-Based Paint Poisoning Prevention Act. The gradual elimination of lead-based paint inventories meant that houses painted before 1978 may contain lead-based paint. Confronted by the publicity about the health hazards of lead, the paint industry aggressively promoted lead-based paint products including using children in their advertisements (Markowitz and Rosner, 2000a,b). Dismayingly, lead-based paint continues to be a major source of lead exposure in children.

The history of the development and use of leaded gasoline is equally provocative and disturbing (Needleman, 2000) (Table 4). Some observers consider the addition of lead to gasoline to be one of the greatest public health disasters of the 20th century (Lin-Fu, 1991). Tetraethyl lead was discovered by a German chemist in 1854. Its potential to curb engine knock

Table 2
Recommended revisions to the CDC action levels

Blood lead level (μg/dL)	Actions	Time frame for beginning intervention
<2	No action	
2–5	Provide caregiver lead education. Provide follow-up testing. Refer the child for social services to investigate possible sources of lead exposure	Within 30 days
5–10	Above actions, plus: if BLLs persist (i.e., two venous BLLs in this range at least 3 months apart) or increase, proceed according to actions for BLLs 10–20	Within 2 weeks
10–20	Above actions, plus: provide coordination of care (case management). Provide clinical evaluation and care. Provide environmental investigation and control current lead hazards	Within 1 week
20–70	Above actions	Within 24 h
70 or higher	Above actions, plus hospitalize child for chelation therapy immediately	Within 24 h

Adapted from Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention (CDC, 2002). Online: http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm

was recognized in 1921. By 1923, leaded gasoline was on sale in selected markets and by 1936, 90% of the gasoline sold in the U.S. contained lead. Its dominance continued until 1972, when the U.S. Environmental Protection Agency (EPA) proposed to phase out leaded gasoline based on its interference with catalytic converter operation, but growing recognition of the health effects of lead may have played a role as well. In the U.S. the primary phase-out was completed in 1986, but leaded gasoline remained available in selected markets until the early 1990s. The addition of lead to gasoline occurred despite warnings from scientists such as Alice Hamilton, a pioneer in occupational medicine, and the U.S. Public Health Service. With the removal of lead from gasoline, average childhood blood lead levels in the U.S. plummeted from approximately 16 μg/dL in 1976 to 3.2 μg/dL in 1994. Although its removal was properly viewed as a public health triumph, lead nevertheless continues to inflict harm on many children in the U.S. and throughout the world. The global phase-out of leaded gasoline has taken even longer. The tragic history of lead, despite the early knowledge of its adverse health effects, is a grave comment on societal values (Bellinger and Bellinger, 2006; Gilbert, 2005).

Table 3
History of lead-based paint

Year	Event
1887	U.S. medical authorities diagnose childhood lead poisoning
1904	Child lead poisoning linked to lead-based paints
1909	France, Belgium and Austria ban white-lead interior paint
1914	Pediatric lead-paint poisoning death from eating crib paint is described
1921	National Lead Company admits lead is a poison
1922	League of Nations bans white-lead interior paint; U.S. declines to adopt
1943	Report concludes eating lead paint chips causes physical and neurological disorders, behavior, learning and intelligence problems in children
1971	Lead-Based Paint Poisoning Prevention Act passed
1978	Lead-based house paint banned

Table 4
History of leaded gasoline

Year	Event
1854	Tetraethyl lead discovered by German chemist
1921	Midgley discovers that tetraethyl lead curbs engine knock
1922	Public Health Service warns of dangers of lead production, leaded fuel
1923	Leaded gasoline goes on sale in selected markets
1936	Ninety percent of gasoline sold in U.S. contains ethyl
1972	EPA gives notice of proposed phase out of lead in gasoline
1986	Primary phase out of leaded gas in U.S. completed
1994	Study shows that U.S. blood-lead levels declined by 78% from 1978 to 1991
2000	European Union bans leaded gasoline

3. Health effects of lead below 10 μg/dL

The definition of "low level" lead exposure has been revised progressively downward as our tools and study designs for evaluating neurodevelopment have evolved. Hints of health effects and intellectual impairment in children with BLLs below 10 μg/dL had already emerged by 1991, when CDC established 10 μg/dL as a level of concern (Fulton et al., 1987; Needleman and Bellinger, 1991). Now, not only is there overwhelming evidence of effects at low levels, but it is increasingly apparent that the rate of decline in intellectual impairment is greater at BLLs below 10 μg/dL than above (Canfield et al., 2003). Overall, every 1 μg/dL increase in blood lead results in a decrease of 0.87 IQ points. For BLLs below 10 μg/dL, a 1 μg/dL increase results in a 1.37 IQ decrease (Canfield et al., 2003). Such a fall in average IQ is consistent with several meta-analyses and reviews of childhood lead studies (Fulton et al., 1987; Lanphear et al., 2005; Needleman, 1990; Pocock et al., 1994; Schwartz, 1994). Several independent investigators also have concluded that BLLs below 10 μg/dL are harmful (Chiodo et al., 2004; Fulton et al., 1987; Landrigan, 2000; Schwartz, 1994; Schwartz and Otto, 1991; Selevan et al., 2003; Walkowiak et al., 1998; Wasserman et al., 2000). These studies, as well as the meta-analyses

confirm that a threshold for the adverse health effects of lead exposure cannot be calculated. Appendix A lists multiple statements supporting the absence of a threshold for the health effects of lead exposure.

Policy questions arising from this conclusion seem to have frozen Federal initiatives. The Agency for Toxic Substances and Disease Registry (ATSDR) has refused to set a minimum risk level (MRL) and the Environmental Protection Agency has refused to establish a reference dose (RfD) because some of the "health effects associated with exposure to lead occur at blood levels so low as to be essentially without a threshold" (IRIS, 2004). The Centers for Disease Control and Prevention (CDC) states: "Because no threshold for adverse health effects in young children has been demonstrated, public health interventions should focus on eliminating all lead exposures in children. Lead concentrations in drinking water should be below the EPA action level of 15 ppb" (MMWR, 2004).

4. Costs and consequences of lead exposure

Despite the dramatic fall in BLLs following the removal of lead from gasoline, elevated childhood lead levels persist as a source of public health concerns. Since 1991, CDC has maintained 10 μg/dL as a guide to excessive exposure. According to the CDC, in 1999 and 2000 2.2% of children in the 1–5-year age group exhibited lead levels above 10 μg/dL (http://www.cdc.gov/nceh/lead/faq/about.htm). Approximately 20 million children are under 5 years of age, which means that about 440,000 children in the U.S. exceed BLLs of 10 μg/dL. Currently, the CDC states that, "Approximately 310,000 U.S. children aged 1–5 years have blood lead levels greater than the CDC recommended level of 10 μg of lead per deciliter of blood" (CDC, 2005). But it has yet to establish a strategy for interventions.

No recent data allow us to specify the prevalence of children with BLLs greater than 5 μg/dL. Analysis of the NHANES III data (1988–1994) indicated that 25.6% of 1–5-year olds had BLLs at or above 5 μg/dL (Bernard and McGeehin, 2003). African American and Mexican American children are more likely to exhibit elevated BLLs than non-Hispanic white children (Table 5). Children in homes built before 1946 exhibit a greater likelihood of elevated BLLs (Table 5). These data indicate that demographic and socioeconomic variables are important determinants of elevated BLLs. It is an inescapable conclusion that environmental justice questions are a significant issue for lead exposure.

Table 5
Distribution of children (%) with blood lead levels greater or equal to 5 μg/dL

Characteristic	BLLs ≥ 5 μg/dL
Non-Hispanic black children	46.8
Mexican American children	27.9
Non-Hispanic white children	18.7
Housing built before 1946	42.5
Housing built between 1946 and 1973	38.9
Housing built after 1973	14.1

Adapted from Bernard and McGeehin (2003).

Beyond costs to the individual, elevated BLLs represent an economic drain on society as well. The direct and indirect costs to society of elevated BLLs were estimated to be $43.4 billion for one age group (Landrigan et al., 2002). This calculation was based on an average BLL of 2.7 μg/dL for a cohort of children at 5 years of age, a loss of 0.25 IQ point for each 1 μg/dL of blood lead, and the relationship between IQ and lifetime earnings. Over a 20-year period (one generation), the loss amounts to $868 billion. In 1990, the U.S. EPA was asked by Congress to estimate the benefits of the Clean Air Act. Fig. 2, based on the Agency's response, plots the number of IQ points that would have been lost from 1970 to 1990 had lead remained in gasoline. From this estimate, U.S. EPA calculated that the benefits of lead removal, based on IQ alone, translated into one trillion dollars.

Direct costs for children with BLLs greater than 10 μg/dL were estimated in Mahoning County, Ohio (Stefanak et al., 2005). They estimated "that lead poisoning costs local governments on the order of $0.5 million each year". These calculations did not include the indirect costs to society of lowered IQ. They concluded that it was cost effective to invest in the reduction of childhood lead exposure. A study in Washington State estimated the total cost of lead exposure as $1.5 billion for 5-year old children in that state for one cohort (Davies, 2005). Other investigators have found similar costs to society as well as the possible contributions to accelerated neurodegeneration associated with aging (Rice, 1998). A more profound issue, discussed later, that is not monetized is society's ethical responsibility for each individual's loss of potential.

Elevated lead body burdens are also associated with anti-social behaviors such as an elevated risk for adjudicated delinquency (Needleman et al., 2002), an endpoint not included in analyses focused on IQ scores. But the two criteria are intertwined, and raising IQ offers a number of documented benefits to society and the individual beyond earnings potential. Herrnstein and Murray, in their contentious book THE BELL CURVE: Intelligence and Class Structure in American Life (1996) calculated that a three-point rise in IQ (3%) results in a

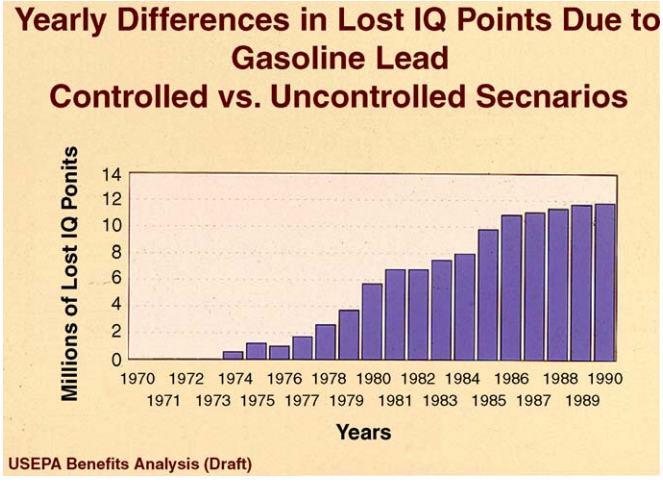

Fig. 2. Estimated losses in IQ if lead had remained in gasoline 1970–1990.

reduction of the following social indices amounting, on average, to about 20% each:

- males incarcerated in jail;
- reduced poverty rate;
- high school dropouts;
- children not living with parents;
- welfare recipient;
- out-of-wedlock children;
- low birth weight babies;
- bottom decile HOME scores;
- poverty during first 3 years.

Herrnstein and Murray never addressed the question of neurotoxic exposures in disadvantaged populations, the degree to which they contribute to social pathologies, or how their removal or reduction could elevate IQ scores. The list indicates that the calculations by Landrigan et al. (2002) and U.S. EPA, because they fail to include the costs of social disruption as well as loss of earning power, are gross underestimates of how much wealth lead drains from the economy.

These calculations also underscore the principle that the societal effects of low-level lead exposure on IQ only become apparent when viewed from the standpoint of population-level effects (Weiss, 1988). For an individual child, the consequences are difficult to discern given that small changes in IQ score occur from one test occasion to the next. Even a five-point IQ drop exerts a significant impact, however, when viewed from the perspective of a population. Assuming a mean IQ of 100 for a large population and a normal distribution, the tails of the curve represent those with superior IQ (greater than 130) and those with lower IQ (less than 70). IQs below 70 require significant societal support such as remedial education. A five-point drop in IQ would significantly change the number of people in the tails of this distribution. For example, in a population of 100 million with a mean IQ of 100 there would be 6 million people with IQs above 130 and an equivalent number with IQs below 70. A shift in the mean of 5 IQ points (5%) would result in only 2.4 million gifted people with IQs above 130 and 9.4 million people with IQs less than 70 who also require remedial assistance. The consequences to society will clearly be enormous (Fig. 3).

Furthermore, all populations are not equal. Disadvantaged populations begin with a handicap. A population with a mean IQ of 85, common among such communities, rather than 100, will suffer disproportionately when exposed to an agent that lowers IQ. Fig. 4 (Weiss, 2000) demonstrates the dramatic increase in the number of children with an IQ below 70 in a community with a mean IQ of 85 compared to a community with a mean IQ of 100 when the mean is reduced by as little as 1%. It is also instructive to consider the loss in high IQ children for a one to five point loss in IQ (Fig. 5). The number of children with an IQ less than 130 increases rapidly with a lowered mean IQ.

Disadvantaged populations also suffer from diminished educational opportunities, inflicted by their inability to support

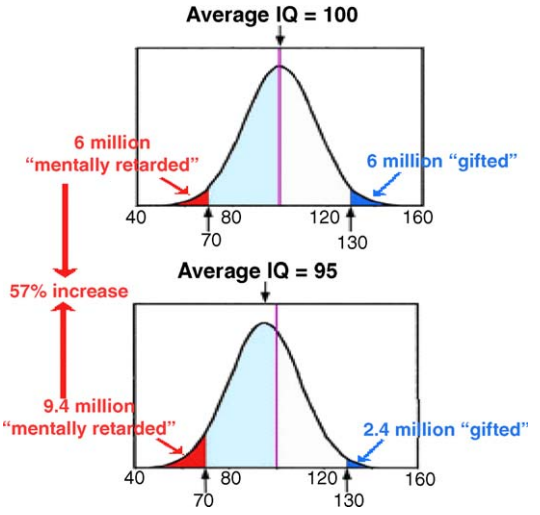

Fig. 3. Losses associated with five-point drop in IQ on a population of 100 million. Based on Weiss (1988) and modified by http://www.ourstolenfuture.org/NewScience/behavior/iqshift.htm.

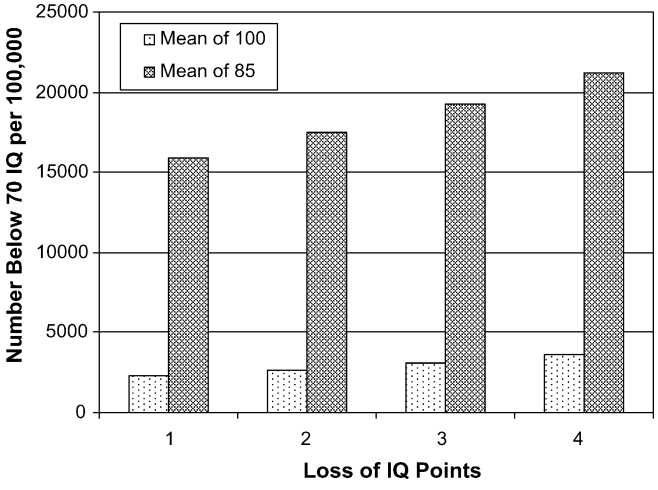

Fig. 4. The consequences of a one point (1%) drop in IQ depending on the mean population IQ (Weiss, 2000).

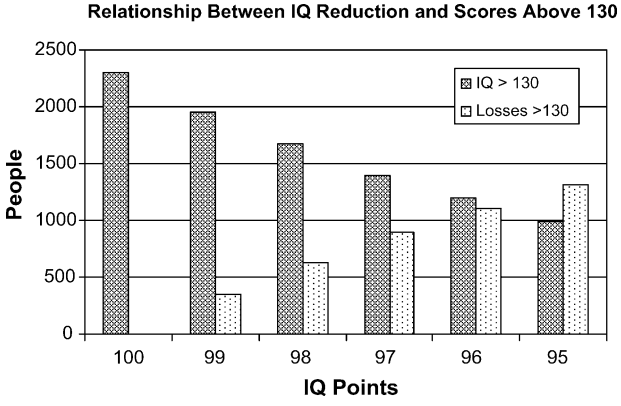

Fig. 5. Effect of reductions in mean IQ on the proportion of scores in the superior range.

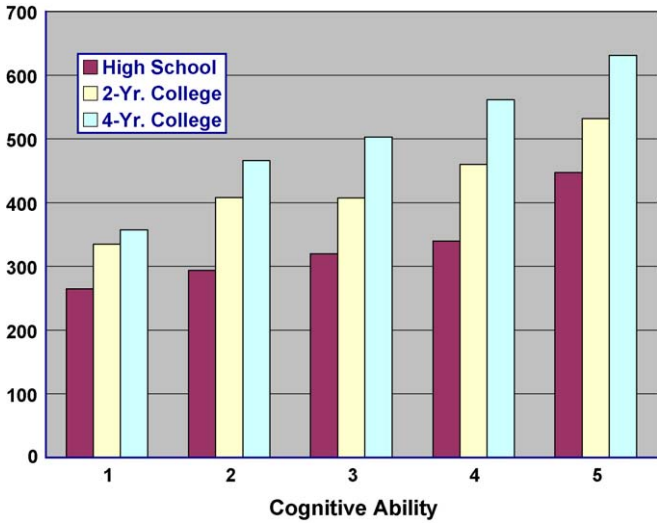

Fig. 6. Combined effects on weekly earnings (based on 1997 dollars) of cognitive ability (based on IQ) and educational attainment. Based on Ceci et al. (1997).

the costs of advanced schooling as well as by the reduced educational resources available to them as a result of skewed allocations. Ceci et al. (1997) argued that cognitive ability (measured by IQ scores) and years of education should be seen as joint determinants of earning potential rather than in isolation. Fig. 6, based on Ceci et al. (1997), charts this interaction and offers an additional perspective on the combined influence of elevated lead exposures and educational deficits. Put another way, the adverse effects of lead are multiplied by the adverse effects of curtailed educational opportunities.

Finally, humans are not exposed to lead in isolation from environmental factors such as stress or from other developmental neurotoxicants. The social ecology governing a child's environment can induce permanent changes in brain structure and function that almost certainly modify its vulnerability to toxic exposures (Weiss and Bellinger, 2006). Indeed, animal studies indicate that maternal stress is one determinant of the effects of lead (Cory-Slechta et al., 2004; Virgolini et al., 2005). And, further, the infant and the fetus are exposed to a broth of chemicals from their in utero environment and their mothers' breast milk that is poorly accounted for when assessing the effects of lead (Cory-Slechta, 2005).

5. Ethical considerations

Recognition that children deserve a supportive environment provides the foundation of ethical decisions bearing on children's health (Gilbert, 2005; Weiss, 2001). Even what are still deemed low levels of lead exposure diminish the chances that children will attain their full potential. Accepting childhood exposure to lead violates the basic tenets of established bioethical principles based on the Belmont Report (NIH, 1979): justice, beneficence, and respect for person (Weiss, 2001). Although the Belmont Report was undertaken to provide ethical guidance for clinical trials, it is no less applicable to exposures occurring in the environment. The principle of justice requires that benefit be in balance with harm. For clinical trials, it means that subjects take risks in accordance with presumed benefits. It can also be interpreted to mean a balance among communities. Numerous studies have documented that lead exposure is greatest for minority and lower socioeconomic status communities (Bernard and McGeehin, 2003). Beneficence requires that we maximize the benefits and minimize the harm. But no benefits accrue either to the individual or to society from lead exposure, and given the consensus among investigators that a threshold for lead neurotoxicity cannot be determined, the cost:benefit ratio is effectively infinity. Respect for persons requires that those exposed have a right to know and give informed consent. No child, of course, has ever given informed consent to lead exposure, nor have its surrogates—its adult caretakers.

6. The rationale for a 2 μg/dL action level

The following list summarizes why reducing the CDC action level from 10 to 2 μg/dL is desirable and achievable:

- There is sufficient scientific evidence that children suffer from cognitive and behavioral deficits even at BLLs less than 10 μg/dL.
- Lead toxicity is irreversible and its effects persist for a lifetime.
- Successful programs developed to reduce lead exposure have been established; they can be refined and extended.
- The CDC level of 10 μg/dL arms public agencies and commercial interests with the ability to argue against taking appropriate measures to reduce childhood lead exposure (for example, eliminating lead in the drinking water of schools throughout the country).
- A level of 2 μg/dL provides a tangible goal; a goal of zero, while defensible scientifically, does not and, as a result, would tend to be ignored. 2 μg/dL, so to speak, is psychologically as well as technically attainable.
- Appropriate analytical methodology is well developed, available, and comes at reasonable cost.

The CDC provides the following rationale for not changing from 10 to 5 μg/dL (http://www.cdc.gov/nceh/lead/spotLights/changeBLL.htm). Counterarguments follow each CDC assertion:

- No effective clinical interventions are known to lower the blood lead levels for children with levels less than 10 μg/dL or to reduce the risk for adverse developmental effects.

  Response: CDC is confusing medical interventions with environmental interventions when the appropriate goal should be to eliminate the need for medical interventions. Reducing lead exposure reduces blood lead levels. We know how to safely abate lead in or around homes. Families can be advised to perform simple procedures around the home to reduce childhood lead exposure; for example, remove shoes, dust and vacuum frequently, remove carpets, wash hands. This is a

sibling and multigenerational issue—protect the current child and others are also protected. Lowering the blood lead action level to 2 μg/dL would encourage all of the above.

- Children cannot be accurately classified as having blood lead levels above or below a value less than 10 μg/dL because of the inaccuracy inherent in laboratory testing.

  Response: There are adequate analytical procedures for a detection limit of 2 μg/dL and follow-up testing is readily available to confirm initial blood lead levels. Blood lead levels, although not the ideal biomarker for lead exposure because of the relatively short half-life of lead in blood (approximately 30 days), are the accepted measure for accessing current lead exposure (Barbosa et al., 2005). Historically, BLL analysis has been variable. This prompted government agencies to require laboratories to analyze standard samples, some of which are routinely below 2 μg/dL (OSHA, 2006). Although routine and accurate analysis of BLLs below 2 μg/dL may challenge some laboratories, the technology and methodology have been readily available for over 10 years (Schutz et al., 1996) and instrumentation companies offer equipment for low level lead analysis by techniques such as anodic stripping voltammetry (e.g., ESA Biosciences, Chelmsford, MA). No technical argument can be enlisted for not establishing routine assessment and monitoring of very low BLLs.
- Finally, there is no evidence of a threshold below which adverse effects are not experienced. Thus, any decision to establish a new level of concern would be arbitrary and provide uncertain benefits.

  Response: This is correct, but the conclusion does not follow. The conclusion should be that any detectable level of lead is unacceptable. The current level gives false and inaccurate guidance to health care professionals. Furthermore, it fails to provide recommendations to assist pediatricians, health care providers and parents to protect children until they exceed the current action level of 10 μg/dL. If lead caused cancer in children or adults we would not be having this discussion.

7. Conclusions

The 15 years since the CDC committed to preventing lead exposure in our children has confirmed that no level of lead exposure is safe. The discussion above demonstrates that it is reasonable, rational, and responsible for CDC to lower the blood lead action level from 10 to 2 μg/dL. It is reasonable because the most effective, practicable way to eliminate the neurobehavioral consequences of lead exposure is to take incremental steps to reduce exposure. It is rational because we have overwhelming evidence that health effects of lead occur at very low levels of exposure and there appears to be no threshold for these effects. It is responsible because the CDC has a scientific and ethical obligation to reflect our current knowledge in the guidance it provides to the nation's parents, school boards and administrators, and public health officials. Local initiatives to reduce lead exposure are unlikely to be undertaken until CDC itself moves in that direction. At present, such initiatives are constrained by the 10 μg/dL "level of concern," which, rather than acting as a catalyst for preventive action essentially becomes a surrogate for inaction. The American Academy of Pediatrics stated that, "The focus on childhood lead-poisoning policy, however, should shift from case identification and management to primary prevention, with a goal of safe housing for all children" (AAP, 2005). We recognize the current political hurdles that CDC must overcome to comply with our recommendations, which is why we have voiced them in this forum.

Acknowledgement

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Appendix A. Statements that support no threshold level for health effects of lead

"ATSDR has not derived MRLs (minimum risk level) for lead. The EPA has not developed a reference concentration (Rfc) for lead. EPA has also decided that it would be inappropriate to develop a reference dose (RfD) for inorganic lead (and lead compounds) because some of the health effects associated with exposure to lead occur at blood levels as low as to be essential without a threshold (IRIS, 2004)" (ATSDR Toxicology Profile for Lead—http://www.atsdr.cdc.gov/toxprofiles/tp13.html).

"Because no threshold for adverse health effects in young children has been demonstrated (Schwartz, 1994), public health interventions should focus on eliminating all lead exposures in children (Rogan and Ware, 2003). Lead concentrations in drinking water should be below the EPA action level of 15 ppb" (MMWR, 2004) (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm53d330a1.htm).

"Recent studies suggest that adverse health effects exist in children at blood lead levels less than 10 μg/dL. In the past the Centers for Disease Control and Prevention (CDC) has lowered the level considered elevated in response to similar reports. However, at this time the reasons not to lower the level of concern are as follows (see above for rational)" http://www.cdc.gov/nceh/lead/spotLights/changeBLL.htm.

"Lead poisoning is entirely preventable. However, nearly half a million children living in the United States have lead levels in their blood that are high enough to cause irreversible damage to their health" http://www.cdc.gov/nceh/lead/factsheets/leadfcts.htm.

"Even low levels of lead are harmful and are associated with decreased intelligence, impaired neurobehavioral development, decreased stature and growth, and impaired hearing acuity" http://www.cdc.gov/nceh/lead/factsheets/leadfcts.htm.

"Because there is no apparent threshold below which adverse effects of lead do not occur, "EBLL" must be defined arbitrarily" http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_chap1.htm.

"The WG identified and considered several issues that bear on drawing causal inference from the observed associations among children with blood led levels <10 μg/dL. After

considering these issues, the work group concluded that, while available evidence does not permit a definitive causal interpretation of the observed associations between higher BLLs in the range <10 μg/dL and adverse health indicators, the weight of available evidence favors, and does not refute, the interpretation that these associations are, at least in part causal. However, the WG also concluded that the possibility of residual confounding and other factors leaves considerable uncertainty as to the absolute size of the effect and shape of the dose response relationship at blood lead levels <10 μg/dL". A review of evidence of health effects of blood lead level <10 μg in children. Draft February 2004...Reported by Advisory Committee on Childhood Lead Poisoning Prevention to the CDC. http://www.cdc.gov/nceh/lead/ACCLPP/meeting-Minutes/lessThan10MtgMAR04.pdf

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