

# Should the Centers for Disease Control and Prevention's Childhood Lead Poisoning Intervention Level Be Lowered?

Susan M. Bernard, JD, DrPH, MPH

The US Centers for Disease Control and Prevention (CDC) in 1991 chose 10 µg/dL as an initial screening level for lead in children's blood.

Current data on health risks and intervention options do not support generally lowering that level, but federal lead poisoning prevention efforts can be improved by revising the follow-up testing schedule for infants aged 1 year or less with blood lead levels of 5 µg/dL or higher; universal education about lead exposure risks; universal administration of improved, locally validated risk-screening questionnaires; enhanced compliance with targeted screening recommendations and federal health program requirements; and development by regulatory agencies of primary prevention criteria that do not use the CDC's intervention level as a target "safe" lead exposure. (*Am J Public Health*. 2003; 93:1253–1260)

The US Centers for Disease Control and Prevention (CDC) has since 1970 set tiered screening and intervention levels for childhood lead poisoning. The purpose of these levels is to guide federal, state, and local health departments and individual pediatricians in identifying and responding appropriately to lead-exposed children.<sup>1</sup> No law requires development of the intervention levels, and criteria for setting and changing them are not well defined. They are set forth in CDC guidance documents that are implemented through conditions on funding to government and individual providers. The initial, or threshold, intervention level (referred to here as "the intervention level"), which was originally set at 40 µg/dL, was most recently lowered from 25 µg/dL to 10 µg/dL in 1991.<sup>1</sup>

Some researchers (e.g., Landrigan<sup>2</sup> and Lanphear and colleagues<sup>3</sup>) have suggested that the intervention level should be reexamined and possibly further lowered, and this issue is currently under consideration by the CDC's Advisory Committee on Childhood Lead Poisoning Prevention (M.A. McGeehin, oral communication, August 27, 2002). In other work, I conducted a statistical analysis of data from the Third National Health and Nutrition Examination Survey (NHANES III) to identify the prevalence of childhood blood lead levels (BLLs) of 5 µg/dL or higher and the socioeconomic and demographic charac-

teristics of 1- to 5-year-old children with BLLs of at least 5 µg/dL but less than 10 µg/dL.<sup>4</sup> In this article, I investigate whether data or policy considerations support lowering the childhood blood lead screening level.

## BACKGROUND

There is extensive literature on the health impacts of lead exposure in early childhood.<sup>5–7</sup> At high doses, these impacts can include damage to the nervous, hematopoietic, endocrine, and renal systems. At lower exposures, lead has been associated with adverse cognitive and neurobehavioral impacts. Epidemiological data on the adverse health outcomes of lead exposure are supported by research on mechanisms of lead toxicity and by animal studies, reviewed by the Agency for Toxic Substances and Disease Registry.<sup>7</sup>

Children in the United States have been exposed to lead from many sources, in particular lead used as an additive to gasoline<sup>8</sup> and as a component of paint.<sup>9,10</sup> Although each of these uses is now banned in the United States, children continue to be exposed to lead, primarily as a result of the presence in housing of lead-contaminated paint and resulting dust, soil, and chips.<sup>11–14</sup>

The high prevalence over the 20th century of clinical and subclinical lead intoxication among US children is well documented.<sup>5,15</sup> As exposures have been reduced, the levels and

prevalence of childhood lead intoxication have also declined.<sup>16–20</sup> Over the 6-year period of NHANES III (1988–1994), there was a 48.4% decline in the percentage of children with BLLs defined as elevated: during phase 1 (1988–1991), 8.9% of 1- to 5-year-olds had BLLs of 10 µg/dL or higher, while during phase 2 (1991–1994), 4.4% of 1- to 5-year-olds (890 000) had BLLs of 10 µg/dL or higher.<sup>18</sup> Much higher levels of lead poisoning have consistently been found among urban, lower-income, and African American children living in older housing in the Northeast and Midwest.<sup>16,17</sup> Mean BLLs in these higher-risk populations have declined over time<sup>21</sup> but remain elevated in some locations and among some populations.<sup>14</sup>

Medicaid eligibility is a strong predictor of lead poisoning risk. NHANES III phase 2 data (1991–1994) showed that the prevalence of BLLs of 10 µg/dL or higher among 1- to 5-year-olds whose families participated in Medicaid was, at 9%, 3 times higher than the prevalence among non-Medicaid-enrolled children.<sup>22</sup> Sixty percent of 1- to 5-year-olds with BLLs of 10 µg/dL or higher were Medicaid participants, and 83% of 1- to 5-year-olds with BLLs of 20 µg/dL or higher were Medicaid participants.<sup>22,23</sup>

Housing age, condition, and location are also important risk factors.<sup>23</sup> The risk posed by older housing (predating 1946) is higher for lower-income children.<sup>18</sup> In some older communities, lead poisoning is endemic. For example, a cross-sectional analysis of children in Illinois aged birth to 6 years for the years 1993 to 1997 showed no decline over that time in the number of children with BLLs of either 15 µg/dL or higher or 45 µg/dL or higher, in either the city of Chicago or the state as a whole.<sup>24</sup> While only 43% of the Chicago children lived within zip codes identified as being at high risk for lead, 99% of the hospitalizations for lead poisoning occurred among those children.<sup>24</sup>

**TABLE 1—Federal Lead Poisoning Prevention Programs**

Agency	Program and Duties
Department of Housing and Urban Development (HUD)	Lead Hazard Control Grant Program, enforcement of Federal Lead Paint Disclosure Rule (with EPA and DOJ) and Federally-Assisted Housing Lead Paint Regulations, National Survey of Lead Paint in Housing, Lead Hotline (with EPA), Internet listing of lead paint professionals, public education and training of housing professionals and providers and others, technical assistance, research.
Department of Health and Human Services	
Centers for Disease Control and Prevention	Blood Lead Screening Grant program, public education to medical and public health professionals and others, National Health and Nutrition Examination Survey, quality control for laboratories analyzing blood lead specimens, research.
Health Care Financing Administration <sup>a</sup>	Covers and reimburses for lead screening and diagnosis, lead poisoning treatment, and follow-up services for Medicaid-eligible children.
National Institute of Child Health and Human Development	Conducts and supports laboratory, clinical, and epidemiological research on the reproductive, neurobiological, developmental, and behavioral processes including lead poisoning-related research.
Health Resources and Services Administration	Directs national health programs to assure quality health care to under-served, vulnerable, and special need populations including children with lead poisoning.
Agency for Toxic Substances and Disease Registry	Undertakes the study of blood lead in populations near Superfund sites and funds state health agencies to undertake this type of work.
Food and Drug Administration	Enforces standards for lead in ceramic dinnerware; monitors lead in food.
National Institutes of Health	Basic research on lead toxicity.
Environmental Protection Agency (EPA)	Authorizes states to license lead paint professionals; environmental laboratory accreditation; enforcement of Disclosure Rule (with HUD and DOJ) and Pre-Renovation Notification Rule; Hazardous Waste Regulation; public education to parents, environmental professionals, and others; training curriculum design; Lead Hotline (with HUD); research; addresses lead contamination at industrial waste sites including drinking water and industrial air emissions.
Department of Justice (DOJ)	Enforces Disclosure Rule (with HUD and EPA), defends federal lead paint regulations, enforces pollution statutes including hazardous waste laws.
Consumer Product Safety Commission	Enforces ban of lead paint; investigates and prevents the use of lead paint in consumer products; initiates recalls of products containing lead that present a hazard; conducts dockside surveillance and intercepts imported products that present a risk of lead poisoning; recommends elimination of lead from consumer products through guidance policy on lead.
Occupational Safety and Health Administration	Worker protection regulations.
Department of the Treasury	Evaluates financial incentives (such as tax credits) for lead hazard control.
Department of Energy	Conducts weatherization activities in a lead-safe manner.
Department of Defense	Administers lead-based paint/lead hazard management programs in 250 000 family housing and child-occupied facilities worldwide, administers childhood lead poisoning prevention programs on installations worldwide, administers research and development programs to develop new cost-effective technologies for lead paint management and abatement, partner with other federal agencies to develop policies and guidance for lead hazard management on a national level.

Source. Reference 14.

<sup>a</sup>Health Care Financing Administration is now Medicare and Medicaid Services.

**Overview of Federal Lead Poisoning Prevention Efforts**

Federal lead poisoning prevention policy encompasses both primary and secondary prevention (Table 1). Exposure reduction (primary prevention) is the responsibility of the US Department of Housing and Urban Development, the US Environmental Protection Agency (EPA), other federal agencies such as the Consumer Product Safety Commission and the Food and Drug Administration, and states with funding from the Department of Health and Human Services (HHS).<sup>25</sup> Secondary prevention, in the form of surveillance and intervention in cases of clinical and subclinical lead poisoning, falls under HHS jurisdiction. Since the 1970s, federal support for childhood lead screening has been incorporated into funds allocated to states and into federal child health programs,<sup>5,25,26</sup> including the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC),<sup>23</sup> the Head Start program,<sup>14</sup> the federally subsidized Community Health Centers,<sup>14</sup> and Medicaid, which serves about a third of 1- to 5-year-olds nationwide.<sup>14</sup>

The CDC's lowering of the intervention level to 10 µg/dL in 1991 was part of a major shift in HHS policy<sup>1,26</sup> that called for "virtually universal" blood lead screening among 1- to 6-year-olds (i.e., universal except in "communities where large numbers or percentages of children have been screened and found not to have lead poisoning").<sup>1(p2)</sup> Pediatricians were to use a screening questionnaire to evaluate a child's exposure risk and to determine the frequency of blood lead screening for that child.<sup>1</sup> The HHS moved away from universal screening in a 1997 guidance document (the most recent) in light of widespread lack of screening and a decline in geometric mean BLL in 1- to 5-year-olds, from 15 µg/dL in 1976 to 1980 (NHANES II) to 2.7 µg/dL in 1991 to 1994.<sup>25</sup> A cost-benefit analysis done in support of the 1997 guidance found that where the prevalence of 1-year-old children in the United States with BLLs of 10 µg/dL or higher was less than 14% (range = 11%–17%), the costs of universal screening exceeded the monetized health benefits.<sup>27</sup>

The 1997 guidance document recommends targeting blood lead screening and interventions to high-risk areas.<sup>25</sup> State public health officials receiving lead poisoning prevention grants must develop statewide plans for either performing universal screening or requiring screening for (1) higher-risk areas within the state identified through housing stock age or a prevalence of BLLs of 10 µg/dL or higher; (2) children who receive services from public assistance programs such as Medicaid; and (3) children whose parents or guardians provide responses to a personal risk questionnaire that indicate elevated risk of lead exposure, or who lack sufficient knowledge to answer a personal risk questionnaire.<sup>25</sup> To develop such a plan, the guidance document recommends that states set up advisory committees, assess lead exposure and screening capacity, determine boundaries of targeted areas, decide on appropriate screening requirements, and write and implement plans with respect to areas with universal screening and with targeted screening.<sup>25</sup>

Investigations by the US General Accounting Office and the CDC have reported that throughout federally funded health programs, lead screening requirements are not satisfied.<sup>23,28</sup> For example, in 1996, 65% of Medicaid-enrolled 1- to 5-year-olds did not receive a blood lead test and, as a result, did not receive appropriate follow-up care and environmental services to reduce exposure.<sup>28</sup> Nearly half of state Medicaid programs (24 of 51) were not as rigorous with respect to lead screening policies as required by federal law.<sup>23</sup> A study by the CDC's Advisory Committee on Childhood Lead Poisoning Prevention of 42 state contracts with Medicaid managed care organizations showed that only 20 discussed lead-related services and only 15 discussed blood lead screening.<sup>28</sup> Other researchers have also found low rates of screening among children with clearly identified risk due to location of residence.<sup>24</sup>

### Prevalence and Risk Factors for BLLs of 5 µg/dL or Higher

NHANES III data indicated that more than a quarter (25.6%) of 1- to 5-year-olds had

BLLs of 5 µg/dL or higher.<sup>4</sup> While the proportion has almost certainly declined since 1994, it probably remains high, particularly among African American children and the urban poor. During the NHANES III survey period, the population of 1- to 5-year-olds with BLLs of 5 µg/dL or higher included 46.8% of non-Hispanic Black children, 27.9% of Mexican American children, and 18.7% of non-Hispanic White children.<sup>4</sup> Almost half (42.6%) of children in the Northeast, 21% in the Midwest, 18% in the South, and 12% in the West had BLLs of at least 5 µg/dL. Among children participating in Medicaid, 42.3% had BLLs of 5 µg/dL or higher. The majority of children overall and within higher-risk subpopulations had BLLs of less than 10 µg/dL.<sup>4</sup>

These data suggest that demographic and socioeconomic factors that characterize children with the highest levels of lead intoxication are also associated with children with lower levels of measurable blood lead. They also suggest that many children, even those considered to be in the lowest-risk groups, are exposed to some amounts of environmental lead. Sources of lead exposure other than those associated with residential paint may include drinking water (contaminated during delivery)<sup>13</sup>; glazing on certain imported pottery and ceramics<sup>29</sup>; certain imported foods<sup>30</sup>; exposure to aging buildings (especially schools) that are not the primary residence of the child but within which the child spends significant amounts of time; soil contamination not attributed to lead-based paint<sup>17</sup>; and pre- or perinatal exposure to maternal lead stores from past and current exposures.<sup>31–33</sup>

The risk of lead poisoning from many such exposures can be expected to diminish over time as a result of intentional and unintentional measures such as enforcement of lead prohibitions in consumer protection and public housing programs, voluntary lead abatement by private homeowners, replacement of water distribution lines, and replacement or renovation of housing and public buildings. Further research into individual children's cumulative exposure would be useful in explaining the continued prevalence of measurable levels of lead in children's blood.

### Health Outcomes at BLLs of Less Than 10 µg/dL

Research on the adverse neurocognitive and other health impacts of childhood lead poisoning published since the CDC set the intervention level at 10 µg/dL in 1991 has included (1) follow-up analyses of cohort studies begun in the early 1980s in the United States and internationally (e.g., Tong et al.<sup>34,35</sup> and Wasserman et al.<sup>36</sup>) and more recent cohort studies conducted in Mexico<sup>37</sup> and Costa Rica<sup>38</sup>; (2) cross-sectional studies seeking to find within older data sets an association between lead exposure and adverse health outcomes (e.g., Lanphear et al.<sup>3</sup> and Ballew et al.<sup>39</sup>); (3) research conducted in non-US populations in which exposure from airborne and other lead sources remains high<sup>40–43</sup>; and (4) meta-analyses undertaken to resolve inter-study differences.<sup>44–47</sup> These investigations generally support the CDC's previous determination that adverse cognitive development outcomes are associated with lead exposure "at least as low as 10 µg/dL."<sup>1</sup>

However, there is still substantial uncertainty with respect to health outcomes of childhood lead exposure resulting in BLLs below 10 µg/dL. There has been little research on health outcomes within populations of young children with BLLs below 10 µg/dL, and some investigators have questioned whether discerning neurocognitive impacts at such BLLs is feasible with current epidemiological and statistical methods.<sup>48</sup> Individual studies associating BLLs below 10 µg/dL with adverse cognitive impacts must be interpreted carefully in light of what is known about the significance of the timing within the lives of the study populations of exposure and outcome measurements; the importance of controlling for confounding and effect-modifying variables such as socioeconomic status, maternal education, and the quality of a child's home environment; uncertainties associated with various exposure and outcome measurements; and methodological limitations.<sup>34,49–51</sup> As methods of measuring both lead exposure and cognitive development become more sensitive, subtle adverse impacts of very low levels of lead exposure may become better quantifiable, and it is important to continue this research. Continued in vivo and in vitro research will also be critical.

## IMPACT OF LOWERING THE CDC'S INTERVENTION LEVEL

Given that there is no naturally occurring level of lead in the human body,<sup>5,52</sup> precautionary concerns might support lowering the intervention level pending further research if it could be demonstrated that lowering the level would benefit the target population at an acceptable cost or burden, factoring in the invasiveness of the screening methodology, the risk and discomfort to the individual patient, and the precision and validity of the test.<sup>53</sup> While capillary and venous blood lead measurements can produce satisfactorily precise and reliable data on a child's blood lead at the time of measurement,<sup>54</sup> most laboratories operate at a level in which samples in the 10- to 19- $\mu\text{g}/\text{dL}$  range produce results within 4  $\mu\text{g}/\text{dL}$  (95% confidence interval) of the

true BLL.<sup>55</sup> Validity and precision decrease as the lead concentration in the blood decreases.

Even if the test were shown to present minimal risk and discomfort at sufficient validity and precision, there is no clear benefit to most children of screening to detect BLLs of less than 10  $\mu\text{g}/\text{dL}$  (an exception, discussed below, is children aged 12 months or younger, who should be identified for short-term follow-up screening if their BLL is 5  $\mu\text{g}/\text{dL}$  or higher). Protecting children with BLLs of at least 5  $\mu\text{g}/\text{dL}$  but less than 10  $\mu\text{g}/\text{dL}$  would be the primary aim of lowering the intervention level from 10  $\mu\text{g}/\text{dL}$  to 5  $\mu\text{g}/\text{dL}$ , but for the most part screening would not benefit such children.

Table 2 sets out the current intervention guidelines tied to children's BLLs. If the intervention level were lowered, the most likely change to these guidelines would be

that interventions recommended for children with BLLs of 10  $\mu\text{g}/\text{dL}$  to 14  $\mu\text{g}/\text{dL}$  would now be recommended for children with BLLs of 5  $\mu\text{g}/\text{dL}$  to 14  $\mu\text{g}/\text{dL}$ . Thus, children with screening BLLs in this range would have diagnostic venipuncture within 3 months as well as family lead education, follow-up testing, and possible referral for social services. With respect to family lead education, providing basic information to all parents or guardians of pediatric patients about childhood lead poisoning exposure risk was suggested when the 1997 guidance was written,<sup>27</sup> is supported by the American Academy of Pediatrics (AAP),<sup>56</sup> and should not be dependent on the results of a screening blood test.

Intervention beyond education, such as follow-up social service referral, is very unlikely for children with BLLs between 5  $\mu\text{g}/\text{dL}$  and 10  $\mu\text{g}/\text{dL}$ . There are no studies supporting the use of screening for BLLs of less than 10  $\mu\text{g}/\text{dL}$  as a trigger for environmental interventions, which have been generally shown not to reduce BLLs of less than 25  $\mu\text{g}/\text{dL}$ .<sup>27,54,55,57-59</sup> Even if there were effective interventions at these lower levels of exposure, many health departments currently intervene only when children have BLLs of 20  $\mu\text{g}/\text{dL}$  to 25  $\mu\text{g}/\text{dL}$ , owing to limited resources.<sup>2</sup>

Because of the relatively high proportion of children with BLLs of 5  $\mu\text{g}/\text{dL}$  or higher, lowering the intervention level would likely result in a return to universal screening requirements. As a result, the change would result in the administration of blood tests to all 1- to 5-year-olds, even though at least 75% of them have BLLs of less than 5  $\mu\text{g}/\text{dL}$  and thus would not benefit from the screening. National, universal screening would substantially raise the cost of case identification of children with elevated BLLs. On a much smaller scale, for example, the cost per case identification via universal screening among children aged 6 months to 6 years in a low-prevalence community in Denver, Colo (2.9% with BLLs  $\geq 10$   $\mu\text{g}/\text{dL}$ , 0.3% with BLLs  $\geq 20$   $\mu\text{g}/\text{dL}$ ) was \$463 for children with BLLs of 10  $\mu\text{g}/\text{dL}$  or higher, \$1713 for children with BLLs of 15  $\mu\text{g}/\text{dL}$  or higher, and \$4925 for children with BLLs of 20  $\mu\text{g}/\text{dL}$  or higher, without factoring in staff time for phlebotomy

**TABLE 2—1991 and 1997 Guidelines for Follow-Up to Screening for Blood Lead Levels (BLLs) (1991 Recommendations Only Are in Italics)**

Screening BLL ( $\mu\text{g}/\text{dL}$ )	Follow-Up Diagnostic Testing	Follow-Up Interventions <sup>a</sup>
<10	None	Reassess or rescreen in 1 year. No further action unless exposure changes.
10-14	3 mo	Provide family lead education. Provide follow-up testing. Refer for social services, if necessary. <i>Many children (or a large proportion of children) with BLLs in this range should trigger communitywide childhood lead poisoning prevention activities.</i>
15-19	3 mo	Provide family lead education. Provide follow-up testing. Refer for social services, if necessary. If BLLs persist (i.e., 2 venous BLLs in this range at least 3 months apart) or worse, proceed according to actions for BLLs of 20 to 44 $\mu\text{g}/\text{dL}$ .
20-44	1 mo-1 wk	Provide coordination of care (case management). Provide clinical management. Provide environmental investigation. Provide lead hazard control.
45-59	48 h	Within 48 hours, begin coordination of care (case management), clinical management, environmental investigation, and lead hazard control.
60-69	24 h	Within 48 hours, begin coordination of care (case management), clinical management, environmental investigation, and lead hazard control.
$\geq 70$	Immediately as an emergency lab test	Hospitalize child and begin medical treatment immediately. Begin coordination of care (case management), clinical management, environmental investigation, and lead hazard control immediately.

Source. Adapted from reference 25.

<sup>a</sup>Interventions are triggered by diagnostic, not screening, BLLs, defined as the first venous BLL obtained within 6 months of an elevated screening BLL.

or time for administration and review of the screening risk questionnaire.<sup>60</sup>

Lowering the intervention level is likely to prove disadvantageous to the children with BLLs of 10 µg/dL or higher. First, while a return to universal screening, if fully implemented, would result in the discovery of children with elevated BLLs who would be missed by targeted screening, a risk-screening questionnaire, or both, it seems unlikely that there would be better compliance with the universal screening requirement than was prevalent between 1991 and 1997. Reasons given by physicians for failure to screen in the past included cost, futility where no interventions were available, lack of certainty in the health risks, and low population prevalence of elevated BLLs.<sup>2,23,28,52,54,58,61–65</sup>

Second, although lowering the intervention level and consequently identifying roughly a fourth of US children as at risk could lead to an increase in public awareness of the problem of lead poisoning and possibly more funding, most of the available funds would probably be spent to screen the blood of millions of children with minimal or no exposure to lead and diverted from the predominantly poor and African American children who are most at risk. Such an allocation of resources would be contrary to federal priorities with respect to children's health, disproportionate health burdens, environmental justice, and lead poisoning prevention.<sup>14,25,66–68</sup>

## RECOMMENDATIONS

Although an across-the-board lowering of the intervention level is not warranted at this time, a number of changes in the approach and implementation of the 1997 guidance are recommended. These changes are intended to prioritize eliminating childhood BLLs of 10 µg/dL and higher, in keeping with federal policy.<sup>14,68</sup> However, it is likely that improved implementation of the 1997 guidance would also protect children with BLLs of less than 10 µg/dL. For example, universal education about lead hazards might stimulate some prevention activities on the part of parents and guardians. In addition, abatement actions in response to community-wide or targeted interventions and incentives, or to the discovery of 1 or more children with

BLLs of 10 µg/dL or higher, could reduce the exposure of other children living in that same environment currently and in the future.

Brown and colleagues found that effective enforcement of state lead poisoning prevention laws significantly lowered the risk of a BLL of 10 µg/dL or higher for a child living in housing in which a child previously was found with a BLL of 25 µg/dL or higher.<sup>69</sup>

### Revise Follow-Up Testing Schedule for Infants Aged 1 Year or Younger With BLLs of 5 µg/dL or Higher

The AAP, in its 1998 policy statement on screening for elevated BLLs, recommends that pediatricians begin lead screening infants at 9 to 12 months and that screening “be considered again at ~24 months of age when BLLs peak.”<sup>56</sup> The 1997 guidance likewise recommends targeted screening at ages 1 and 2.<sup>25</sup> The CDC's recommendation should be revised to require that children aged 1 year or younger who are found to have BLLs of 5 µg/dL or higher be rescreened within 3 to 6 months. BLLs have been shown to rise from between birth and 2 years and peak between 18 and 24 months<sup>70,71</sup> as children become more mobile and eat more with their hands. The likelihood that a 1-year-old with a BLL of 5 µg/dL or higher will become a 2-year-old with a BLL of 10 µg/dL or higher will depend on individual risk factors, but the probability is sufficiently high that a full year should not pass before retesting, as recommended by the 1997 guidance and by the AAP. Data reported by the CDC indicated that the prevalence of BLLs of 10 µg/dL or higher among 1-year-olds in high-risk areas of Chicago in 1997 was 17%, while among 2-year-olds it was 29%.<sup>28</sup> Thirty-nine percent of the children with BLLs of less than 10 µg/dL at the age of 1 year during 1995 and 1996 were retested 1 year later; of those, 21% had developed BLLs of 10 µg/dL or higher.<sup>28</sup>

### Make Parent/Guardian Education Universal

Studies do not provide strong support for the usefulness of education interventions alone in preventing or reducing elevated BLLs.<sup>27,54,72,73</sup> However, because parents and guardians need to be educated about

exposure risks in order to give informed consent for a blood test or to complete a risk-screening questionnaire, and because such education would enhance the value of the risk-screening questionnaire, it should not depend on the outcome of a screening blood test. The AAP recommends that pediatricians provide guidance to parents of *all* infants and toddlers on risk factors for lead exposure and specific prevention strategies tailored to the family and community<sup>56</sup>; the 1997 guidance document is less explicit on the need for exposure risk education in advance of completing the basic personal risk questionnaire, but that is the focus of the questionnaire.<sup>25</sup>

In keeping with the AAP recommendations and the 1997 guidance document,<sup>25,56</sup> pediatricians and public health workers should provide more detailed, case-specific assistance in identifying and reducing actual exposures of children discovered to have elevated BLLs.

### Improve the Risk-Screening Questionnaire

The risk-screening questionnaire is critical to finding children who are not subject to targeted screening owing to risk factors such as Medicaid eligibility, but who nonetheless are at risk—for example, owing to the age or condition of their child care provider's facilities or because they live in older housing undergoing renovation.<sup>11</sup> Analyses of the questionnaire in use from 1991 through 1997 (when universal screening was required and the questionnaire was geared toward frequency of testing rather than necessity for testing) indicated that the questionnaire was insufficient to identify children with elevated BLLs.<sup>60,74–76</sup>

Suggestions for improving the questionnaire include adding questions that will identify children who have either emigrated to the United States with their families or been adopted and who may be at increased risk owing to either pre- or postimmigration exposure<sup>56,77–79</sup> and targeting children who may be exposed to lead-containing folk remedies. Other at-risk children who could be identified through a well-developed questionnaire include those whose parents are exposed to lead through occupation or hobby,<sup>77</sup> those whose developmental delay and associated

oral behaviors place them at significant risk for lead exposure, and victims of abuse or neglect.<sup>56</sup>

Identification of “locally important risk factors”<sup>75</sup> is important to the questionnaire’s effectiveness.<sup>80</sup> For example, in developing childhood blood lead screening guidelines, the state of Florida recognized that, although the 1997 guidance document recommends universal screening where there is a high prevalence of housing predating 1950, “dangerous amounts of lead were present in paint until the mid-1970s,” a 20-year period during which Florida’s population grew by more than 4 million.<sup>81</sup> As a result, the state developed screening guidelines that targeted children in pre-1970 housing. The use of geographic information system technology and other tools will be helpful to state and local governments in identifying neighborhoods in which children should be targeted for screening because of the age of the residential building stock<sup>82,83</sup> or owing to exposure to multiple sources of lead, including industrial emissions.<sup>84</sup>

### Track and Improve Compliance With Federal Screening and Intervention Requirements and Recommendations

Because children in federal health programs make up a disproportionate proportion (83%) of the group with BLLs of 20 µg/dL or higher,<sup>22,23</sup> they should be a priority for targeted screening. Since 1989, federal law has required that children enrolled in Medicaid be screened for blood lead as part of prevention services provided through the Early and Periodic Screening, Diagnosis, and Treatment program.<sup>28</sup> In 1998, Medicaid regulations were revised to impose a nonwaivable requirement that all children be screened for blood lead at 12 and 24 months of age (or between 36 and 72 months if they are enrolled later).<sup>14</sup> The Advisory Committee on Childhood Lead Poisoning Prevention issued a set of recommendations in December 2000 concerning implementation of these and other federal requirements for lead screening and follow-up in state Medicaid policies and managed-care contracts.<sup>28</sup> These recommendations, and federal support to ensure the delivery of such services through environmental and medical follow-up, must be fully imple-

mented and the success of such implementation tracked and reviewed periodically to ensure continued improvement.

### Stop Use of the CDC Intervention Level in Establishing Primary Prevention Goals

Although the CDC’s intervention level is not a statement concerning the level of childhood blood lead considered “safe” or “acceptable,” it has been interpreted as such by the general public (e.g., see Lambrecht<sup>85</sup>) and by federal regulatory agencies. For example, the goal of the EPA’s National Ambient Air Quality Standard for lead, which was set in 1976, was to lower the BLL of 95% of the population to less than 30 µg/dL, the then-applicable CDC intervention level.<sup>86,87</sup> More recently, standards for cleanup of lead-based paint hazards under section 403 of the Toxic Substances Control Act were set to achieve the current intervention level of 10 µg/dL.<sup>88</sup> Setting lead cleanup and abatement targets to achieve postabatement exposures of no more than 10 µg/dL does not adequately protect children’s health and may in some cases be contrary to federal environmental health laws and policies. In setting enforceable air quality standards under the Clean Air Act (National Ambient Air Quality Standard), for example, the EPA must identify the standards regarding the maximum level of the contaminant “which in the judgment of the Administrator [of EPA], based on criteria and allowing an adequate margin of safety, are requisite to protect the public health,” without regard to cost or technological feasibility, and must review the standards with the aid of an independent scientific review committee every 5 years.<sup>89</sup>

The EPA has not developed a reference dose for inorganic lead, as it has for other neurotoxins about which much less information is available. A reference dose is defined as “an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.”<sup>90</sup> The EPA has attributed this inaction to lack of evidence of a threshold for noncarcinogenic toxic effects, particularly neurobehavioral effects in children,<sup>7,90</sup> and it has not attempted to develop a refer-

ence dose using alternative methods, such as the benchmark dose approach<sup>91</sup> used in setting a reference dose for mercury,<sup>92</sup> that do not require identification of a lowest adverse effect level. Paradoxically, the use of the CDC’s intervention level as a de facto reference dose results in the use of an exposure target for lead that is degrees of magnitude higher than it would be if set by such risk assessment methodologies.

As a practical matter, developing a reference dose could result in setting lead exposure targets at—and, for some vulnerable populations, below—actual current exposures, and meeting such targets may prove difficult or even, in some situations, impossible. Yet simply using the screening and intervention level as a default exposure goal is not the answer to these complex questions. The federal government should reexamine its lead exposure reduction targets and redefine them as necessary, within the parameters specified by the relevant governing statutes and regulations, to fully protect children’s health. In setting such standards, the adverse health impacts of lead other than neurocognitive outcomes must also be considered, including impacts on physical growth (stature and head circumference<sup>93,43,39</sup>); impacts on hearing,<sup>94,95</sup> behavior and delinquency,<sup>96</sup> and heme biosynthesis<sup>42</sup>; and outcomes in adult populations, which may include adverse cognitive impacts.<sup>97</sup> The separate or combined effects of other environmental exposures on neurocognitive development are also an important area of investigation.<sup>98</sup> ■

### About the Author

*At the time of this study, the author was with the Johns Hopkins Bloomberg School of Public Health, Baltimore, Md.*

*Requests for reprints should be sent to Susan M. Bernard, JD, DrPH, MPH, Johns Hopkins Bloomberg School of Public Health, 615 N Wolfe St, Suite 7041, Baltimore, MD 21205 (e-mail: sbernard@jhsph.edu).*

*This article was accepted February 22, 2003.*

### Acknowledgments

I thank Michael A. McGeekin, US Centers for Disease Control and Prevention, and Thomas A. Burke, Johns Hopkins Bloomberg School of Public Health, for comments on drafts of this article.

### References

1. US Centers for Disease Control and Prevention. *Preventing Lead Poisoning in Young Children*. Atlanta, Ga: US Dept of Health and Human Services; 1991.

2. Landrigan PJ. Pediatric lead poisoning: is there a threshold? *Public Health Rep.* 2000;115:530–531.
3. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. *Public Health Rep.* 2000;115:521–529.
4. Bernard SM, McGeehin MA. Prevalence of blood lead levels  $\geq 5$   $\mu\text{g}/\text{dL}$  among US children 1–5 years of age, Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. *Pediatrics.* In press 2003.
5. National Research Council. *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations.* Washington, DC: National Academy Press; 1993.
6. *Analytic and Reporting Guidelines: The Third National Health and Nutrition Examination Survey, NHANES III (1988–94).* Hyattsville, Md: National Center for Health Statistics; 1996.
7. US Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Lead (Update).* Atlanta, Ga: US Dept of Health and Human Services; 1999.
8. US Environmental Protection Agency. Prohibition on Gasoline Containing Lead or Lead Additives for Highway Use. Final rule. 61 *Federal Register* 3832-38 (1996) (codified at 40 CFR §80).
9. Lead-Based Paint Poisoning Prevention Act of 1971, as amended by the National Consumer Information and Health Promotion Act of 1976. 42 USCA §4821 et seq.
10. Residential Lead-Based Paint Hazard Reduction Act, 42 USCA chap 63A (1992).
11. Children with elevated blood lead levels attributed to home renovation and remodeling activities—New York, 1993–1994. *MMWR Morb Mortal Wkly Rep.* 1997;45:1120–1123.
12. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: a pooled analysis of 12 epidemiologic studies. *Environ Res.* 1998;79: 51–68.
13. Manton WI, Angle CR, Stanek KL, Reese YR, Kuehnemann TJ. Acquisition and retention of lead by young children. *Environ Res.* 2000;82:60–80.
14. *Eliminating Childhood Lead Poisoning: A Federal Strategy Targeting Lead Paint Hazards.* Washington, DC: President's Task Force on Environmental Health Risks and Safety Risks to Children; 2000.
15. Lin-Fu JS. Modern history of lead poisoning: a century of discovery and rediscovery. In: Needleman H, ed. *Human Lead Exposure.* Boca Raton, Fla: CRC Press; 1991:23–43.
16. Pirkle JL, Brody DJ, Gunter EW, et al. The decline in blood lead levels in the United States: The National Health and Nutrition Examination Surveys (NHANES) [see comments]. *JAMA.* 1994;272: 284–291.
17. Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population: Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991) [see comments] [published erratum appears in *JAMA.* 1995;274:130]. *JAMA.* 1994;272:277–283.
18. Update: blood lead levels—United States, 1991–1994 [published erratum appears in *MMWR Morb Mortal Wkly Rep.* 1997;46:607]. *MMWR Morb Mortal Wkly Rep.* 1997;46:141–146.
19. Blood lead levels in young children—United States and selected states, 1996–1999. *MMWR Morb Mortal Wkly Rep.* 2000;49:1133–1137.
20. Centers for Disease Control and Prevention. Elevated blood lead levels among internationally adopted children—United States, 1998. *JAMA.* 2000;283: 1416–1418.
21. Centers for Disease Control and Prevention. Update: blood lead levels—United States, 1991–1994. *JAMA.* 1997;277:1031–1032.
22. *Medicaid: Elevated Blood Lead Levels in Children.* Washington, DC: US General Accounting Office; 1998. Publication GAO-HEHS-98-78.
23. *Lead Poisoning: Federal Health Care Programs Are Not Effectively Reaching At-Risk Children.* Washington, DC: US General Accounting Office; 1999. Publication GAO-HEHS-99-18.
24. Brown MJ, Shenassa E, Matte TD, Catlin SN. Children in Illinois with elevated blood lead levels, 1993–1998, and lead-related pediatric hospital admissions in Illinois, 1993–1997. *Public Health Rep.* 2000; 115:532–536.
25. *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials.* Atlanta, Ga: Centers for Disease Control and Prevention; 1997.
26. *Strategic Plan for the Elimination of Childhood Lead Poisoning.* Atlanta, Ga: Centers for Disease Control and Prevention; 1991.
27. Briss PA, Matte TD, Schwartz J, Rosenblum LS, Binder S. Costs and benefits of a universal screening program for elevated blood lead levels in 1-year-old children. In: *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials.* Atlanta, Ga: Centers for Disease Control and Prevention; 1997:appendix B.4.
28. Recommendations for blood lead screening of young children enrolled in Medicaid: targeting a group at high risk. *MMWR Morb Mortal Wkly Rep.* 2000; 49(RR-14):1–13.
29. Peraza MA, Ayala-Fierro F, Barber DA, Casarez E, Hatlelid K. Effects of micronutrients on metal toxicity. *Environ Health Perspect.* 1998;106(suppl 1):203–216.
30. Lynch RA, Boatright T, Moss SK. Lead-contaminated imported tamarind candy and children's blood lead levels. *Public Health Rep.* 2000; 115:537–543.
31. Lagerkvist BJ, Ekesrydh S, Englyst V, Nordberg GF, Soderberg HA, Kiklund DE. Increased blood lead and decreased calcium levels during pregnancy: a prospective study of Swedish women living near a smelter. *Am J Public Health.* 1996;86:1247–1252.
32. Gulson BL, Mahaffey KR, Jameson CW, et al. Impact of diet on lead in blood and urine in female adults and relevance to mobilization of lead from bone stores. *Environ Health Perspect.* 1999;107:257–263.
33. Markowitz ME, Shen XM. Assessment of bone lead during pregnancy: a pilot study. *Environ Res.* 2001;85:83–89.
34. Tong S, Baghurst PA, McMichael A, Sawyer M, Mudge J. Lifetime exposure to environmental lead and children's intelligence at 11–13 years: the Port Pirie Cohort Study [see comments] [published erratum ap-  
pears in *BMJ.* 1996;313:198]. *BMJ.* 1996;312: 1569–1575.
35. Tong S, Baghurst PA, Sawyer MG, Burns J, McMichael AJ. Declining blood lead levels and changes in cognitive function during childhood: the Port Pirie Cohort Study. *JAMA.* 1998;280:1915–1919.
36. Wasserman GA, Staghezza-Jaramillo B, Shrout P, Popovac D, Graziano J. The effect of lead exposure on behavior problems in preschool children. *Am J Public Health.* 1998;88:481–486.
37. Schnaas L, Rothenberg SJ, Perroni E, Martinez S, Hernandez C, Hernandez R. Temporal pattern in the effect of postnatal blood lead level on intellectual development of young children. *Neurotoxicol Teratol.* 2000;22:805–810.
38. Wolf AW, Jimenez E, Lozoff B. No evidence of developmental ill effects of low-level lead exposure in a developing country. *J Dev Behav Pediatr.* 1994;15: 224–231.
39. Ballew C, Khan LK, Kaufmann R, Mokdad A, Miller DT, Gunter EW. Blood lead concentration and children's anthropometric dimensions in the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. *J Pediatr.* 1999;134: 623–630.
40. Azcona-Cruz MI, Rothenberg SJ, Schnaas L, Zamora-Munoz JS, Romero-Placeres M. Lead-glazed ceramic ware and blood lead levels of children in the city of Oaxaca, Mexico. *Arch Environ Health.* 2000;55: 217–222.
41. Rothenberg SJ, Schnaas L, Perroni E, Hernandez RM, Ortega JF. Blood lead secular trend in a cohort of children in Mexico City, II: 1990–1995. *Arch Environ Health.* 2000;55:245–249.
42. Jacob B, Ritz B, Heinrick J, Hoelscher B, Wichmann H-E. The effect of low-level blood lead on hematologic parameters in children. *Environ Res.* 2000;82: 150–159.
43. Kafourou A, Touloumi G, Makropoulos V, Loutradi A, Panagiotou A, Hatzakis A. Effects of lead on the somatic growth of children. *Arch Environ Health.* 1997;52:377–383.
44. International Programme on Chemical Safety. *Environmental Health Criteria 165: Inorganic Lead.* Geneva, Switzerland: World Health Organization; 1995.
45. Needleman HL, Gatonis CA. Low-level lead exposure and the IQ of children: a meta-analysis of modern studies. *JAMA.* 1990;263:673–678.
46. Pocock S, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. *BMJ.* 1994;309: 1189–1197.
47. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environ Res.* 1994;65:42–55.
48. Bellinger D. Response to commentaries. *Neurotoxicol Teratol.* 1995;17:249–251.
49. Tong S, Lu Y. Identification of confounders in the assessment of the relationship between lead exposure and child development. *Ann Epidemiol.* 2001;11: 38–45.
50. Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study [see comments]. *Pediatrics.* 1992;90:855–861.

51. Baghurst PA, McMichael AJ, Tong S, Wigg NR, Vimpani GV, Robertson EF. Exposure to environmental lead and visual-motor integration at age 7 years: the Port Pirie Cohort Study [see comments]. *Epidemiology*. 1995;6:104–109.
52. Bellinger DC, Matthews JA. Social and economic dimensions of environmental policy: lead poisoning as a case study. *Perspect Biol Med*. 1998;41:307–326.
53. Last JM. *A Dictionary of Epidemiology*. 3rd ed. New York, NY: Oxford University Press; 1995.
54. Campbell C, Osterhoudt KC. Prevention of childhood lead poisoning. *Curr Opin Pediatr*. 2000;12:428–437.
55. Sargent JD, Dalton M, Klein RZ. Diagnostic testing unwarranted for children with blood lead 10 to 14 microg/dL. *Pediatrics*. 1999;103:e51.
56. American Academy of Pediatrics Committee on Environmental Health. Screening for elevated blood lead levels. *Pediatrics*. 1998;101:1072–1078.
57. Haynes E, Lanphear BP, Tohn E, Farr N, Rhoads GG. The effect of interior lead hazard controls on children's blood lead concentrations: a systematic evaluation. *Environ Health Perspect*. 2002;110:103–107.
58. Harvey B. Should blood lead screening recommendations be revised? *Pediatrics*. 1994;93:201–204.
59. Lanphear BP. The paradox of lead poisoning prevention [published erratum appears in *Science*. 1998; 282:51] [see comments]. *Science*. 1998;281:1617–1618.
60. France EK, Gitterman BA, Melinkovich P, Wright RA. The accuracy of a lead questionnaire in predicting elevated pediatric blood lead levels. *Arch Pediatr Adolesc Med*. 1996;150:958–963.
61. Gellert GA, Wagner GA, Maxwell RM, Moore D, Foster L. Lead poisoning among low-income children in Orange County, California: a need for regionally differentiated policy. *JAMA*. 1993;270:69–71.
62. Ferguson SC, Lieu TA. Blood lead testing by pediatricians: practice, attitudes, and demographics. *Am J Public Health*. 1997;87:1349–1351.
63. Campbell JR, Schaffer SJ, Szilagyi PG, O'Connor KG, Briss P, Weitzman M. Blood lead screening practices among US pediatricians. *Pediatrics*. 1996; 98(3 Pt 1):372–377.
64. Binder S, Matte TD, Kresnow M, Houston B, Sacks JJ. Lead testing of children and homes: results of a national telephone survey. *Public Health Rep*. 1996; 111:342–346.
65. Benjamin JT, Platt C. Is universal screening for lead in children indicated? An analysis of lead results in Augusta, Georgia in 1997. *J Med Assoc Ga*. 1999; 88:24–26.
66. Clinton W. Executive Order 12898: Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations. 59 *Federal Register* 7629, February 16, 1994 (amended by: Executive Order 12948, 60 *Federal Register* 6381, February 1, 1995).
67. Clinton W. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks. 62 *Federal Register* 19885, April 23, 1997 (amended by: Executive Order 13229, 66 *Federal Register* 52013, October 11, 2001; Executive Order 13296, 68 *Federal Register* 19931, April 23, 2003).
68. *Healthy People 2010: Understanding and Improving Health*. Washington, DC: US Dept of Health and Human Services; 2000.
69. Brown MJ, Gardner J, Sargent JD, Swartz K, Hu H, Timperi R. The effectiveness of housing policies in reducing children's lead exposure. *Am J Public Health*. 2001;91:621–624.
70. Dietrich KN, Berger OG, Succop PA, Hammond PB, Bornshein RL. The developmental consequences of low to moderate prenatal and postnatal lead exposure: intellectual attainment in the Cincinnati Lead Study Cohort following school entry. *Neurotoxicol Teratol*. 1993;15:37–44.
71. Bellinger D, Dietrich KN. Low-level lead exposure and cognitive function in children. *Pediatr Ann*. 1994; 23:600–605.
72. Manheimer EW, Silbergeld EK. Critique of CDC's retreat from recommending universal lead screening for children [see comments]. *Public Health Rep*. 1998; 113:38–46.
73. Roberts JR, Reigart JR, Ebeling M, Hulsey TC. Time required for blood lead levels to decline in nonchelated children. *J Toxicol Clin Toxicol*. 2001;39: 153–160.
74. Casey R, Wiley C, Rutstein R, Pinto-Martin J. Prevalence of lead poisoning in an urban cohort of infants with high socioeconomic status. *Clin Pediatr (Phila)*. 1994;33:480–484.
75. Blood lead levels among children in a managed-care organization—California, October 1992–March 1993. *MMWR Morb Mortal Wkly Rep*. 1995;44: 627–629, 635.
76. Kazal LA Jr. The failure of CDC screening questionnaire to efficiently detect elevated lead levels in a rural population of children. *J Fam Pract*. 1997;45: 515–518.
77. Fatal pediatric lead poisoning—New Hampshire, 2000. *MMWR Morb Mortal Wkly Rep*. 2001;50: 457–459.
78. Elevated blood lead levels among internationally adopted children—United States, 1998. *MMWR Morb Mortal Wkly Rep*. 2000;49:97–100.
79. Binns HJ, Kim D, Campbell C. Targeted screening for elevated blood lead levels: populations at high risk. *Pediatrics*. 2001;108:1364–1366.
80. Rolnick SJ, Nordin JJ, Cherney LM. A comparison of costs of universal versus targeted lead screening for young children. *Environ Res*. 1999;80:84–91.
81. Duclos C, Johnson T, Thompson T. Development of childhood blood lead screening guidelines, Duval County, Florida, 1998. *J Public Health Manag Pract*. 1999;5:9–10.
82. Reissman DB, Staley F, Curtis GB, Kaufmann RB. Use of geographic information system technology to aid Health Department decision making about childhood lead poisoning prevention activities. *Environ Health Perspect*. 2001;109:89–94.
83. Vine MF, Degnan D, Hanchette C. Geographic information systems: their use in environmental epidemiologic research. *Environ Health Perspect*. 1997;105: 598–605.
84. Gonzalez EJ, Pham PG, Ericson JE, Baker DB. Tijuana childhood lead risk assessment revisited: validating a GIS model with environmental data. *Environ Manage*. 2002;29:559–565.
85. Lambrecht B. US weighs lowering acceptable lead level in children. *St. Louis Post-Dispatch*. July 8, 2002:A1.
86. *Air Quality Criteria for Lead*. Research Triangle Park, NC: US Environmental Protection Agency, Office of Health and Environmental Assessment; 1986. Publication EPA/600/8-83/028F.
87. *Air Quality Criteria for Lead: Supplement to the 1986 Addendum*. Research Triangle Park, NC: US Environmental Protection Agency, Office of Research and Development; 1990. Publication EPA/600/8-89/049F.
88. Requirements for Notification, Evaluation, and Reduction of Lead-Based Paint Hazards in Federally Owned Residential Property and Housing Receiving Federal Assistance. Final rule. 64 *Federal Register* 50139-50231 (1999).
89. Air Pollution Prevention and Control Act. 42 USCA §7401 et seq. (1970).
90. US Environmental Protection Agency. Integrated Risk Information System (IRIS). Last updated February 1, 1997. Available at: <http://www.epa.gov/iris>. Accessed February 18, 2002.
91. Crump K, Allen B, Faustman E. *The Use of the Benchmark Dose Approach in Health Risk Assessment*. Washington, DC: US Environmental Protection Agency; February 1995. Publication EPA/630/R-94/007.
92. *Toxicological Effects of Methylmercury*. Washington, DC: National Academy of Sciences; 2001.
93. Schwartz J, Angle C, Pitcher H. Relationship between childhood blood lead levels and stature. *Pediatrics*. 1986;77:281–288.
94. Schwartz J, Otto D. Blood lead, hearing thresholds, and neurobehavioral development in children and youth. *Arch Environ Health*. 1987;42:153–160.
95. Schwartz J, Otto D. Lead and minor hearing impairment. *Arch Environ Health*. 1991;46:300–305.
96. Needleman HL, Riess JA, Tobin MJ, Biesecker GE, Greenhouse JB. Bone lead levels and delinquent behavior [see comments]. *JAMA*. 1996;275:363–369.
97. Schwartz BS, Stewart WF, Simon PD, et al. Past adult lead exposure is associated with longitudinal decline in cognitive function. *Neurology*. 2000;55: 1144–1150.
98. Perera FP, Illman SM, Kinney PL, et al. The challenge of preventing environmentally related disease in young children: community-based research in New York City. *Environ Health Perspect*. 2002;110:197–204.