

REPORT 7 OF THE COUNCIL ON SCIENCE AND PUBLIC HEALTH (A-10)
Lead Levels in Children
(Resolution 401, A-09)
(Reference Committee D)

EXECUTIVE SUMMARY

Objective. To update the Council's 1994 report on lead in children by briefly reviewing the current epidemiology of lead exposure, current lead screening guidelines, relevant federal regulations, and key studies that have emerged on the effects of blood lead concentrations on cognition and behavior.

Data Sources. English-language reports on studies using human subjects were selected from a Pub Med search of the literature from 2000 to March, 2010 using the MeSH terms "lead," "child," and "environmental exposure" in combination with "lead poisoning" or "cost," and "cost benefit." Additional text terms included "pediatric," "lead levels," and "lead standards." Additional articles were identified by manual review of the references cited in these publications. Web sites of the Centers for Disease Control and Prevention (CDC), Agency for Toxic Substances and Disease Registry, Environmental Protection Agency (EPA), United States Consumer Product Safety Commission (USCPSC), were searched for relevant resources.

Results. Lead is a harmful neurotoxicant present in air, soil, water, and commercial products. Lead impacts the developing brain; in addition children have increased exposure. Studies have reported that peak blood lead concentrations generally occur around the age of two. Data since 1994 have pointed to impaired cognition, lowered IQ, and behavioral problems for children exposed to lead at blood concentrations below the CDC's current level of concern of 10 µg/dL. There is increasing evidence that the harmful effects of lead are significant at low levels of exposure. Some studies have identified long-term effects of lead exposure on cognition and behavior into early adulthood. These effects remain even after controlling for a variety of confounders, including but not limited to race, age, sex, education status of parents or caregivers, and geographic location. Removal of lead from gasoline and decreasing the allowable limit of lead-based paint eliminated the majority of lead from commercial sources; however, airborne lead, soil-based lead and lead in the water, as well as lead in imported foods, jewelry, toys, and other commercial products remain. Lead continues to pose a disproportionate burden for specific populations, such as racial and ethnic minority groups. Racial and ethnic gaps in both exposure and screening remain, despite screening and testing recommendations.

Conclusions. Prevention remains the key to reducing lead exposure in children, and every effort must be taken to reduce the amount of lead to which children are exposed. This includes decreasing children's exposure to lead-contaminated dust, enforcement and monitoring of the current clean air standards, and removal of lead from consumer products. Federal recommendations should reflect the current body of scientific evidence.

REPORT OF THE COUNCIL ON SCIENCE AND PUBLIC HEALTH

CSAPH Report 7-A-10

Subject: Lead Levels in Children
(Resolution 401, A-09)

Presented by: C. Alvin Head, MD, Chair

Referred to: Reference Committee D
(Diana E. Ramos, MD, Chair)

1 Resolution 401 (A-09), "Lead Levels in Children," introduced by the American Academy of Child
2 and Adolescent Psychiatry, American Psychiatric Association, and the American Academy of
3 Psychiatry and Law at the 2009 Annual Meeting and referred to the Board of Trustees, asks:

4
5 That our American Medical Association (AMA) Council on Science and Public Health
6 (CSAPH) review and update its 1994 report on children and lead;

7
8 That our CSAPH generate appropriate evidence-based recommendations regarding screening
9 for lead; and

10
11 That our AMA advocate for updated policies, procedures, and standards with respect to lead
12 screening programs, consistent with current research and scientific knowledge.

13
14 **BACKGROUND**

15
16 The Council previously examined this topic in 1994 in a report that: (1) described the historical
17 sources of lead exposure in the United States; (2) reviewed the current epidemiology of lead
18 toxicity in children; and, (3) reviewed the most recent guidelines for screening and treatment of
19 lead toxicity. Current AMA Policies on screening children for blood lead concentrations and
20 reporting such results reflect the guidelines in use at that time (Policies H-60.956 and H-60.977,
21 AMA Policy Database). AMA policy also supports community awareness about the hazard of
22 lead-based paint and its removal (Policy H-440.943), and to reduce lead exposure emanating from
23 household or workplace products (Policy H-135.959).

24
25 Currently, the Centers for Disease Control and Prevention (CDC) maintains an advisory group (the
26 Advisory Committee of Childhood Lead Poisoning Prevention or ACCLP) that meets annually to
27 evaluate clinical and policy information related to the prevention of lead poisoning. In 1991, this
28 group established the blood lead "level of concern" (the level at which public health actions are
29 initiated) at 10 µg/dL.¹ In 2005, the Committee issued a summary report and updated guidelines on
30 the effects of low blood lead concentrations in children.¹ After reviewing studies that examined
31 adverse cognitive effects in children with blood lead concentrations less than 10 µg/dL, the CDC
32 concluded that although no accepted "safe" level for lead in children can be established, it was not
33 advisable to reduce the "level of concern" because the feasibility and effectiveness of individual

Action of the AMA House of Delegates 2010 Annual Meeting: Council on Science and Public Health Report 7 Recommendations Adopted, and Remainder of Report Filed.

1 interventions to further reduce blood lead concentrations below 10 µg/dL had not been
2 demonstrated.²

3
4 The American Academy of Pediatrics (AAP) through its Environmental Working Group recently
5 reaffirmed its 2005 policy statement, “Lead Exposure in Children: Prevention, Detection and
6 Management,” in May 2009.³ The policy statement reviewed the scientific literature on lead
7 exposure, clinical effects, and data on screening rates and also issued recommendations for clinical
8 guidance and government action. The full set of recommendations is available online
9 [<http://aappolicy.aappublications.org/cgi/content/full/pediatrics;116/4/1036>]. One recommendation
10 urges the government to fund studies to either confirm or refute findings that blood lead
11 concentrations <10 µg/dL are associated with lower IQ, and to conduct studies that can adequately
12 account for confounding of this issue by socioeconomic factors. This recommendation alludes to
13 the fact that several studies have concluded that harmful effects are associated with blood lead
14 concentrations <10 µg/dL in infants and children.

15
16 Accordingly, this report briefly reviews the current epidemiology of lead exposure, lead screening
17 guidelines, relevant federal regulations, and some key studies that have emerged on the effects of
18 lead on cognition and behavior including several that have focused on lower blood lead
19 concentrations.

20 21 METHODS

22
23 English-language reports on studies using human subjects were selected from a Pub Med search of
24 the literature from 2000 to March, 2010 using the MeSH terms “lead,” “child,” and “environmental
25 exposure” in combination with “lead poisoning” or “cost,” and “cost benefit.” Additional text
26 terms included “pediatric,” “lead levels,” and “lead standards.” Additional articles were identified
27 by manual review of the references cited in these publications. Web sites of the CDC, Agency for
28 Toxic Substances and Disease Registry (ASTDR), Environmental Protection Agency (EPA), and
29 United States Consumer Product Safety Commission (USCPSC) were searched for relevant
30 resources.

31 32 CURRENT EPIDEMIOLOGY

33
34 The National Health and Nutrition Examination Survey (NHANES) is the primary source for
35 information on blood lead concentrations in U.S. children. NHANES is a set of surveys conducted
36 by the CDC’s National Center for Health Statistics. Information is collected by a phone interview,
37 and physiologic data are collected by a mobile van.^{4,5} CDC surveillance data indicate that
38 approximately 1% of children under age 5 who are tested have elevated blood lead concentrations,
39 and approximately 310,000 children aged 1 to 5 years exceed the CDC’s “level of concern” for
40 lead (a blood lead concentration of more than 10 µg/dL) according to the 1999-2002 NHANES
41 survey period.⁴ Nationwide, nearly 7 million children have blood lead concentrations between 2
42 and 10 µg/dL.⁶

43
44 Table 1 summarizes elevated blood lead concentrations levels by race for three NHANES survey
45 periods through 2004. These NHANES data indicate that substantial progress has been made in
46 reducing the total pool of children with blood lead concentrations exceeding 10 µg/dL; however,
47 compared with Hispanic and non-Hispanic white children, non-Hispanic black children have a
48 higher percentage of elevated blood lead concentrations (see Table 1).⁷⁻⁹ The geometric mean
49 blood lead concentrations also remained higher for non-Hispanic black children compared with
50 Hispanic and non-Hispanic white children in the 1999-2004 NHANES (2.8 vs. 1.9 and 1.7 µg/dL,
51 respectively).⁷

52

Lead Exposure Pathways

Children and adults are exposed to lead from several environmental (e.g., paint, dust, renovation, carpet), occupational, and household sources; the primary exposure pathway of concern continues to be lead paint for most children. Less important household sources are various folk remedies, cosmetics, and contaminated dietary supplements. Water, air, and soil all have the potential to be contaminated with lead from consumer products, waste disposal, or pollution.¹¹ Some consumer products, including various imported toys, food, and jewelry also are potential sources of contamination.^{12,13} Lead also enters the water supply through soldered pipes that leach lead.^{3,14} Lead also is an occupational hazard in many manufacturing processes, and thus worker exposure levels are regulated by the Occupational Safety and Health Administration (ASTDR Web site). Because of the pervasive and toxic nature of lead, its presence is regulated by several federal agencies to limit exposure (See Appendix I).

OTHER VARIABLES INFLUENCING PEDIATRIC RISK

Lead Toxicity

High blood lead concentrations ($> 60\mu\text{g/dL}$) in children can produce a constellation of symptoms, including headache, abdominal pain, constipation, loss of appetite, motor impairment, agitation, and/or somnolence, although many children even at these high levels may not seem overtly ill.³ Current patterns of lead exposure result in mostly subclinical effects associated with lower blood lead concentrations (see below).

Lead is a known neurotoxin with complex effects on neurodevelopment, including neurotransmitter function, mitochondrial functions, intraneuronal regulatory mechanisms, and the development and function of oligodendroglia and astroglia. Lead also exacerbates certain neuronal pathways (e.g., excitotoxicity) that lead to cell death and directly induces apoptosis.^{15,16} Studies of lead exposure in humans have used deciduous teeth or skeletal x-rays, although most studies rely on blood lead concentrations. The latter offer the advantage of providing a temporal “snapshot” of the lead burden and the opportunity to correlate this value with cognitive function or other dependent variables.

Lead Disposition

The distribution of lead in the body is an important consideration in understanding the impact of lead exposure. In the short term, lead circulates in the blood stream with an elimination half-life of approximately 30 days. Lead accumulates in soft tissues such as the liver, kidneys, or brain, but the majority of lead eventually is stored in calcified tissues, such as bone and teeth.¹⁷⁻¹⁹ These lead deposits may be mobilized during periods of increased calcium need relative to intake (e.g., pregnancy, lactation, menopause, chronic disease, advanced age). Because it is a heavy metal, lead disposition also may be influenced by factors that govern the disposition of iron or calcium in the body, so that individuals who are iron- or calcium-deficient are more likely to absorb ingested lead and mobilize bone-based lead, respectively, into the blood stream.¹⁵

The absorption of lead also is enhanced in children compared with adults. Because the blood/brain barrier is not yet fully formed in infants, lead may more easily penetrate the central nervous system. Combined with the fact that children are more prone to ingest lead than adults, these physiologic and anatomical factors place exposed children at increased risk of lead toxicity.²⁰

1 Genetic factors also may influence lead disposition. One genetic polymorphism that modifies the
2 pharmacokinetic distribution of lead (and therefore its toxicity) concerns the alleles that code for
3 the production of [delta]-aminolevulinic acid dehydratase (ALAD), which is involved with heme
4 biosynthesis. Individuals with certain polymorphisms in the gene encoding this enzyme (ALAD2
5 isoform) have higher blood lead concentrations than those with the normal enzyme subunit
6 (ALAD1). Lead also can interfere with the vitamin D receptor gene because of its ability to
7 simulate calcium ion function, and also may interfere with the function of human hemochromatosis
8 protein, which is involved in regulating iron absorption and storage.¹⁵
9

10 SCREENING GUIDELINES

11
12 Lead screening guidelines have evolved over the past 30 years to accommodate the reduction in
13 environmental lead burden, the demographics of children with elevated blood lead concentrations,
14 and the availability of data to accurately assess both the number of children with elevated
15 concentrations as well as established relationships between lead concentrations and toxicity.
16

17 From 1985 to 1991, the CDC recommended that all children be tested for blood lead concentrations
18 unless it could be documented that the community in which they lived did not put them at risk for
19 lead contamination; the corresponding “level of concern” was set at 25 µg/dL.²¹ This policy
20 effectively meant that most children were screened for elevated lead levels to either comply with
21 the CDC recommendation, or alternatively to prove that there was no community risk of lead
22 contamination by demonstrating that the overwhelming majority of children in a community were
23 lead-free. In 1991, the CDC revised its guidelines to lower the level of concern from 25 µg/dL to
24 10 µg/dL, but maintained the universal screening recommendation.²¹ In 1997, the CDC
25 recommended targeted screening based on the prevalence of elevated blood lead concentrations and
26 the age of homes, with continued universal screening of Medicaid patients as a high risk group
27 [<http://www.cdc.gov/nceh/lead/publications/screening.htm>]. In late 2009, the CDC revised its
28 guidelines on screening for Medicaid patients, again based on newly available state data. The CDC
29 now recommends “risk-based” lead testing for those children thought to be at the greatest risk of
30 lead exposure.²²
31

32 The AAP currently recommends that pediatricians screen Medicaid-eligible children and conform
33 with other city or state health department guidance on screening children not eligible for Medicaid.
34 In the absence of such guidance, pediatricians should consider screening all children at least once
35 when they are 2 years of age and ideally twice at ages 1 and 2 years recognizing that measuring
36 blood lead concentration only at 2 years of age, when blood lead concentration usually peaks, may
37 be too late to prevent peak exposure.³
38

39 EFFECTS OF LEAD BELOW 10 µG/DL

40 41 *Effects on Cognition and Measures of Intelligence*

42
43 Although the current level of concern for lead is 10 µg/dL, several studies have been conducted to
44 evaluate the effects of lower blood lead concentrations.²³ As early as 1972, studies of low-level
45 lead exposure in children showed an association with diminished cognitive function, and others
46 followed.²⁴⁻²⁶ Prior to development of the 1991 CDC Guidance, most studies focused on the effects
47 of lead concentrations between 10 and 30 µg/dL. Because these lead concentrations are
48 consistently associated with adverse cognitive effects in children, research in the past 20 years has
49 focused on the effects of lead at lower exposure levels. The overwhelming majority of such studies
50 show a significant inverse association between blood lead concentrations and neurodevelopmental
51 measures.²⁷⁻²⁹ The magnitude of the association differs based on research methodology, but studies

1 remain consistent in their findings of adverse effects of lead on cognition. Representative studies
2 and their findings are identified below:

- 3
- 4 • In a prospective cohort study of 172 children followed for more than 5 years, a significant
5 inverse relationship between blood lead concentrations lower than 10 µg/dL and a composite
6 score on the Stanford Binet Intelligence Scale was identified in children aged 3 or 5 years.
7 Using a linear model, an increase of 10 µg/dL in lifetime blood lead levels indicated a 4.6 point
8 IQ loss. Using nonlinear models, a 7.4 point IQ loss was indicated. Thus, the dose-response
9 relationship between lead exposure and decline in cognitive function was nonlinear, with more
10 marked effects for lead concentrations below the current 10 µg/dL benchmark.³⁰
11
 - 12 • In a prospective study of 294 children in Mexico City, researchers found that both mental
13 development and psychomotor skills were affected in children with elevated lead
14 concentrations (those greater than 10 µg/dL) at 24 months of age. Maternal or cord blood
15 concentrations did not affect the lead-cognition relationship.³¹
16
 - 17 • A meta-analysis of seven international studies measuring full-scale IQ score and its
18 relationship to blood lead concentrations was completed by Lanphear in 2005. Using a log-
19 linear model, he identified a 6.9 IQ point decrement associated with an increase blood lead
20 levels from 2.4 to 30 µg/dL. In addition, for a given increase in blood lead, the lead-associated
21 intellectual decrement for children with a maximal blood lead level < 7.5 µg/dL was
22 significantly greater than that observed for those with a maximal blood lead level ≥ 7.5
23 µg/dL.³²
24
 - 25 • One analysis of NHANES data from 1988-2004 showed performance on cognitive tests was
26 diminished in children with blood lead concentrations as low as 2.5 µg/dL. Similar deficits
27 were found independently for reading and math scores at lead concentrations between 2.5 and
28 10 µg/dL.³³
29

30 Although the majority of studies on lead are performed in young children below the age of 5, some
31 studies have estimated the long-term impact of lead exposure in older children, adolescents, and
32 young adults. Lanphear, et al. evaluated the association between mean blood lead concentrations
33 below 10 µg/dL and academic performance in children aged 6 to 16 years. Adjusted rates show a
34 0.7 and 0.99 decrease in scores on arithmetic and reading for each 1 µg/dL increase in blood lead
35 concentration, respectively.³³ Other data suggests that the blood concentration of lead at age 6 is
36 more predictive of such decrements than concentrations at age 2.³⁴
37

38 Similarly, in a follow-up study of 45 young adults with previously documented elevated blood lead
39 levels, long term effects were identified. Two distinct findings emerged: First, participants with
40 higher blood lead concentrations as children had diminished activity in brain regions associated
41 with language activity. Second, these individuals had higher activity in the right hemisphere
42 homologs of Wernicke's area (an area associated with understanding of written and spoken
43 language), suggesting that compensatory mechanisms may have been triggered in the contralateral
44 areas of the brain in response to diminished function. It is not evident, however, that these
45 alternate mechanisms are sufficient or provide equivalent function compared with children who did
46 not suffer increased lead exposure. Socioeconomic status, IQ, birth weight, gender, drug use, and
47 gestational age all were evaluated as potential confounders in this study.³⁵
48

49 Relying solely on IQ scores as an outcome of the effects of lead exposure in children can be
50 problematic. Because the IQ score is a cumulative measure based on a series of tests that assess

1 specific problem solving capabilities, a total IQ score can mask a very low score on a particular
 2 cognitive ability or test and therefore may be a deceptive measure of the impact of lead exposure.¹⁵
 3 Therefore, although many studies of pediatric blood lead concentrations and IQ exist, some have
 4 used a specific instrument to measure one area of cognitive or developmental function. In some
 5 cases, these may be a proxy for total IQ score. It is also thought that IQ can not be measured
 6 reliably in children until they are between the ages of 5 and 6, so testing IQ and lead levels below
 7 this age may be inaccurate.

8 9 *Relationship of Lead Exposure to Mental Disorders*

10
 11 The effects of lead are not limited to cognition. Over the last 15 years, attention has been devoted
 12 to studying the association between mental disorders and blood lead concentrations, including
 13 Attention Deficit Hyperactivity Disorder (ADHD), conduct disorder, and delinquency. A meta-
 14 analysis of 19 studies concluded that a significant relationship exists between lead and certain
 15 conduct disorders.³⁶ Included in this analysis was a case-control study of incarcerated delinquent
 16 adolescents (cases) and high school adolescents (controls) that found incarcerated delinquents had
 17 significantly higher bone lead concentrations.^{37,38} Similarly, using a cohort of children from the
 18 Cincinnati Lead Study, a significant relationship was detected between self- and parent-reported
 19 delinquent behavior and blood lead concentrations.³⁹

20
 21 Additionally, a 2006 analysis of NHANES data specifically evaluated lower blood concentrations
 22 as a risk factor for ADHD. A “significant dose-response relationship” was found between blood
 23 lead concentrations and ADHD. Children with blood lead concentrations of 2 µg/dL had a four-
 24 fold higher risk for ADHD than children with blood lead concentrations <0.7 µg/dL.^{40,41}

25
 26 Decreased IQ or behavioral outcomes have several potential causal pathways that complicate
 27 analysis of the specific contribution of lead exposure. Gender, race, ethnicity, socioeconomic
 28 status, educational level, and marital status of parents and caregivers, geographic region, body iron
 29 status, and tobacco use are some of the recognized confounding variables that have been addressed
 30 in several trials. The presence of numerous confounders not only complicates the evaluation of the
 31 role of lead exposure but also provides an avenue for criticism of the validity of specific studies. In
 32 a reaction to one of these critiques, Lanphear, et al. devoted an article to the issue of confounding
 33 in lead studies, in which data sets were reanalyzed to address several questions, including the
 34 appropriateness of using “mouthing behaviors” (i.e., a measure of an infant or toddler’s tendency to
 35 chew on objects or ingest/place various materials in the mouth) as a confounder in studies of lead
 36 and intellectual impairment.⁴²

37 38 LEAD EXPOSURE PREVENTION AND POLICY FOR CONCENTRATIONS BELOW 10 39 µG/DL

40
 41 Neither the CDC nor the AAP have advocated lowering the level of concern for lead to less than
 42 10 µg/dL. Both groups remain committed to lead abatement guidelines and support the
 43 enforcement of current lead regulations and compliance with their recommended screening
 44 strategies to reduce childhood lead exposure. In addition, they also advocate and support continued
 45 research into childhood lead exposure prevention strategies.^{3,43}

46
 47 It may seem contradictory that the “level of concern” for children’s blood lead concentrations
 48 remains at 10 µg/dL, despite the apparent acknowledgement that cognitive deficits can be detected
 49 at lead exposures below this level, and that no “safe” threshold for lead exposure exists. The
 50 explanation for maintaining a standard of 10 µg/dL is based on the following reasoning:

- 1 • No effective clinical interventions are known to lower blood lead in children with
2 concentrations less than 10 µg/dL or to reduce the risk for adverse developmental effects;
3
- 4 • Children cannot be accurately classified as having blood lead concentrations below 10 µg/dL
5 because of the sensitivity and variance inherent in current laboratory testing; and
6
- 7 • No evidence exists for a threshold below which adverse effects are prevented.
8

9 Thus, any decision to establish a new level of concern may be considered arbitrary and would
10 provide uncertain benefits.⁴³

11
12 In response, some have argued that despite the lack of medical interventions for children with lead
13 concentrations less than 10 µg/dL beneficial environmental interventions do exist. If the “level of
14 concern” were lowered, current lead regulations and abatement policies could be more strictly
15 enforced. Finally, the lack of an absolute threshold for safety does not obviate the need for action
16 when it is clear that a public health problem exists.^{44,45} Therefore, blood lead concentrations in the
17 range of 1 to 5 µg/dL have been suggested as a more appropriate level of concern.^{44,45} However,
18 currently there is a lack of studies demonstrating that home repair is safe and effective in reducing
19 lead in children with blood concentrations <10 µg/dL. Studies that reveal the sources of lead in
20 such children also are lacking.

21 22 SUMMARY AND CONCLUSION

23
24 Lead is a harmful neurotoxicant present in air, soil, water, and commercial products, and is
25 regulated by several federal agencies. Children are more susceptible to the effects of lead because
26 they experience increased exposure by ingestion and absorb more lead when ingested. Lead
27 continues to be a burden for specific populations, including racial and ethnic minority groups.
28 Racial and ethnic gaps remain in both exposure and appropriate screening, despite ongoing
29 recommendations.

30
31 Established by the CDC, the current blood lead level of concern is 10 µg/dL. Data obtained since
32 1994 have pointed to impaired cognition, lowered IQ, and behavioral problems in children whose
33 blood concentrations are below this concentration. There is increasing evidence that the dose-
34 response effects of lead exposure are incrementally magnified at low levels of exposure. Some
35 studies have confirmed that residual effects of lead exposure on cognition and behavior persist into
36 early adulthood. These effects are evident even after controlling for a variety of confounders,
37 including but not limited to race, age, sex, education status of parents or caregivers, and geographic
38 location.

39
40 Removal of leaded gasoline and lead-based paint from the market has eliminated the majority of
41 lead from commercial sources; however, airborne lead, soil-based lead, and lead in the water as
42 well as lead in imported foods, jewelry, toys and other commercial products remain. Prevention
43 remains the key to reducing lead exposure in children, and every effort must be taken to reduce the
44 amount of lead that children are exposed to. This includes enforcement and monitoring of the
45 current clean air standards, and removal of lead from consumer products.

46
47 Review of the scientific literature on the effects of lead below the CDC’s level of concern should
48 be continued. Federal recommendations should reflect the current body of scientific evidence.
49 Additionally, a need exists to review effective strategies to lower lead concentrations in children
50 whose values are <10 µg/dL. Also there is a lack of studies describing the exposure pathways for
51 children with blood lead concentrations <10 µg/dL and consequently ways to prevent lead

1 exposures in such individuals; additional research on strategies to further reduce childhood
2 exposure to lead remains necessary. Thus, the Council is not in a position to establish science-
3 based guidelines on this topic that would complement or serve as a replacement for current
4 guideline-development processes.

5
6 RECOMMENDATIONS

7
8 The Council on Science and Public Health recommends that the following statements be adopted in
9 lieu of Resolution 401 (A-09) and the remainder of the report be filed.

- 10
11 1. That Policy H-60.956 be amended by insertion and deletion to read as follows:

12
13 H-60.956 Lead Poisoning Among Children

14
15 The AMA: (1) encourages physicians and public health departments to ~~regularly-screen all~~
16 ~~children under the age of six for lead exposure through history taking and when appropriate by~~
17 ~~blood lead testing. The decision to employ blood testing should be made based on prevalence~~
18 ~~studies of blood lead levels in the local pediatric population. Findings from these studies will~~
19 ~~determine whether universal or targeted screening should be employed; and based on current~~
20 recommendations and guidelines (2) encourages the reporting of all children with elevated
21 blood levels to the appropriate health department in their state or community in order to fully
22 assess the burden of lead exposure in children. In some cases this will be done by the
23 physician, and in other communities by the laboratories. (CSA Rep. 6 - I-94; Reaffirmed: CSA
24 Rep. 6, A-04) (Modify Current HOD Policy)

- 25
26 2. That Policy H-60.977 be amended by insertion and deletion to read as follows:

27
28 H-60.977 Lead Poisoning Threat to Children

29
30 Our AMA supports ~~evaluating the adequacy of existing and proposed guidelines concerning~~
31 ~~environmental lead exposure (including the CDC's Strategic Plan for the Elimination of~~
32 ~~Childhood Lead Poisoning), and supports appropriate initiatives- regulations and policies~~
33 ~~designed to more effectively protect young children from exposure to lead. (Sub. Res. 60, A-~~
34 ~~91; Reaffirmed: Sunset Report, I-01) (Modify Current HOD Policy)~~

- 35
36 3. That Policy H-135.959 be amended by insertion to read as follows:

37
38 H-135.959 Eliminating Lead, Mercury and Benzene from Common Household Products

39
40 Our AMA: (1) supports the development of standards to achieve non-hazardous levels of
41 exposure to lead, mercury, or benzene arising from common household or workplace products;
42 (2) encourages efforts to minimize or eliminate mercury use in hospitals and other health care
43 facilities; and (3) will work in coalitions with appropriate federal agencies and health care
44 organizations to educate physicians and other health care professionals about suitable
45 alternatives to the use of mercury and mercury-containing devices and the appropriate disposal
46 of mercury and mercury-containing devices; (4) encourages efforts to minimize or eliminate
47 lead in all commercial and household products (Sub. Res. 418, I-92; Appended: Sub. Res. 410,
48 A-00; Reaffirmation I-00; Reaffirmed A-03) (Modify Current HOD Policy)

- 49
50 4. That Policy H-440.943, "Lead-Based Paints," be re-affirmed. (Reaffirm HOD Policy)

- 1 5. That Policy D-135.988, “Protective NAAQS Standard for Airborne Lead,” be rescinded.
2 (Rescind HOD Policy)
3
- 4 6. That our AMA urge the Centers for Disease Control and Prevention to give priority to
5 examining the current weight of scientific evidence regarding the range of adverse health
6 effects associated with exposure to blood lead concentrations below the current “level of
7 concern” in order to provide appropriate guidance for physicians and public health policy, and
8 encourage the identification of exposure pathways for children who have low blood lead
9 concentrations, as well as effective and innovative strategies to reduce overall childhood lead
10 exposure. (Directive to Take Action)

Fiscal Note: Less than \$500

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Table 1. NHANES Data on Pediatric Lead Testing Children Aged 1-5

Category	NHANES 1988-1991	NHANES 1991-1994	NHANES 1999-2004
	Percent of tested children with blood lead concentrations > 10 $\mu\text{g}/\text{dL}$		
Geometric mean blood lead of all children tested, $\mu\text{g}/\text{dL}$	3.6	2.7	1.9
Overall % of tested children with concentrations > 10 $\mu\text{g}/\text{dL}$	8.6	4.4	1.4
% of Non-Hispanic black children with concentrations > 10 $\mu\text{g}/\text{dL}$	18.6	11.2	3.4
% of Hispanic children with concentrations > 10 $\mu\text{g}/\text{dL}$	7.2	4.0	1.2
% of Caucasian children with concentrations > 10 $\mu\text{g}/\text{dL}$	5.5	2.3	1.2

Data adapted from Jones⁴

APPENDIX I
Standards and Regulations for Lead Agency Media Level Comments¹⁹

Regulating Agency	Media	Concentration	Comments
OSHA	Air (workplace)	50 µg/m ³	Regulation. Permissible exposure limit (8 hr avg; general industry)
		30 µg/m ³	Action level if more than 30 days of exposure
OSHA	Blood	40 µg/dL	Regulation. Cause for written notification and medical exam
		60 µg/dL	Regulation. Cause for medical removal from exposure
CDC	Blood	10 µg/dL	Advisory. Level for individual management
CDC/NIOSH	Air (workplace)	100 µg/m ³	Recommended exposure level; (non-enforceable)
EPA	Air (ambient)	0.15 µg/m ³	Regulation. NAAQS 3-month average
EPA	Soil (residential)	400 ppm (play area) 1200 ppm (other)	Soil screening guidance; requirement for federally funded projects only
EPA	Water (drinking)	15 µg/dL	Action level for public supplies
FDA	Food	Various	Action levels for various food; lead-soldered food can banned
CPSC	Paint	600 ppm (0.06%)	Regulation. Concentration by dry weight. A new standard exists for lead in children's jewelry.

Adapted from Reference 19.

OSHA (Occupational Safety & Health Administration); CDC (Centers for Disease Control and Prevention); NIOSH (The National Institute for Occupational Safety and Health); EPA (Environmental Protection Agency); FDA (Food and Drug Administration); CPSC (Consumer Products Safety Commission).