Lead Poisoning: CDC’s New Target for Prevention

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Environmental Health Sciences & Epidemiology
Outline

- Lead exposure: trends and current sources
- Health effects: children and adults
- Prevention strategies
  - CDC’s new target for prevention
Acknowledgements

Pat McLain
Eliseo Guallar
Ellen Silbergeld
Virginia Weaver
Esther Garcia
Andria Apostolou
Jeffrey Fadrowski
Stephen Rothenberg
Brian Schwartz
Lead

• Toxic metal – used since Ancient times
• Ubiquitous in the environment – human activities
  - Mining, smelting
  - Production of batteries, ammunition, metal products, medical, research and military equipment, ceramic glazes, paint
  - Gasoline additives – phased out in 1970s in US
• Lead is in air, food, drinking water, rivers, lakes, oceans, dust and soil
Fig. 2. Lead concentration, profile in snow strata of Northern Greenland (EPA, 1986).
The Decline in Blood Lead Levels in the United States

The National Health and Nutrition Examination Surveys (NHANES)

James L. Pirkle, MD, PhD; Debra J. Brody, MPH; Elaine W. Gunter; Rachel A. Kramer, ScD; Daniel C. Paschal, PhD; Katherine M. Flegal, PhD, MPH; Thomas D. Matte, MD, MPH

NHANES II (1976 to 1980)

78% drop

NHANES III Phase 1 (1988 to 1991)

Fig 1.—Blood lead levels for persons aged 1 to 74 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).
Children: reasons for increased susceptibility

- Disproportionately heavier exposures than adults:
  - Drink more water, eat more food and breathe more air per weight unit
  - Hand-to-mouth behavior
  - Play close to the ground

- Metabolic pathways, especially in fetal life and first months after birth, are immature
  - Metabolic, detoxification and excretion processes different from adults
  - Blood-brain barrier not fully developed

Landrigan PJ. EHP 2004;112:257-265
Lead poisoning by age in US

FIGURE 3. Number of children with confirmed blood lead levels (BLLs) $\geq 10 \, \mu g/dL$ by program-relevant age group and BLL group — selected U.S. states, 2001

http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5210a1.htm
# Lead biomarkers – Blood vs. bone lead

<table>
<thead>
<tr>
<th>Bone (cortical, trabecular)</th>
<th>Whole blood</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Half-life</strong></td>
<td>Decades</td>
</tr>
<tr>
<td><strong>Reflect</strong></td>
<td>Cumulative exposure</td>
</tr>
<tr>
<td><strong>Analysis</strong></td>
<td>K X-ray fluorescence</td>
</tr>
<tr>
<td><strong>Monitoring</strong></td>
<td>No</td>
</tr>
<tr>
<td><strong>Criterion</strong></td>
<td>No</td>
</tr>
</tbody>
</table>

AAS: atomic absorption spectrometry / ICPMS: inductively coupled plasma mass spectrometry

Other lead biomarkers not usually used (hair, toenails, urine, plasma, serum)

Barbosa et al. EHP 2005;113:1669-74  
Hu et al. EHP 2007;115:455-63
Lead related health effects

- Neurotoxic (children <5 µg/dL, 2 µg/dL adults)
- Nephrotoxic (<5 µg/dL)
- Immunotoxic (<10 µg/dL)
- Alters heme synthesis
- Alters bone and teeth metabolism
- Probable carcinogen (IARC, NTP)
- Cardiovascular outcomes:
  - Increased blood pressure and incidence of hypertension
  - Potential association with cardiovascular mortality and morbidity

EPA 2006 – Air Quality Criteria for Lead
http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=158823
Blood lead and IQ in children – International pooled analysis

- Estimated IQ decrements estimated with increases in blood lead from:
  - 2.4 to 10 µg/dL: 3.9
  - 10 to 20 µg/dL: 1.9
  - 20 to 30 µg/dL: 1.1

→ Steepest declines were at blood lead levels <10 µg/dL
The weight of lead – Effects add up in adults

- Neurotoxic
- Nephrotoxic
- Immunotoxic
- Alters heme synthesis
- Alters bone and teeth metabolism
- Probable carcinogen (IARC, NTP)
- Cardiovascular outcomes

US EPA 2006 – Air Quality Criteria for Lead
http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=158823

Source: Environ Health Perspect 2007;115:A30-36

**Figure 1.** Age-adjusted death rates* for total cardiovascular disease, diseases of the heart, coronary heart disease, and stroke,† by year — United States, 1900–1996

*Per 100,000 population, standardized to the 1940 U.S. population.
†Diseases are classified according to International Classification of Diseases (ICD) codes in use when the deaths were reported. ICD classification revisions occurred in 1910, 1921, 1930, 1939, 1949, 1958, 1968, and 1979. Death rates before 1933 do not include all states. Comparability ratios were applied to rates for 1970 and 1975.
Conclusions

- Sufficient epidemiologic and mechanistic evidence to infer a causal effect of lead on blood pressure – no evidence of a threshold

- Suggestive but not sufficient epidemiologic evidence for clinical cardiovascular endpoints at blood lead < 5 µg/dL

- Suggestive but not sufficient epidemiologic evidence for cardiac function abnormalities: left ventricular hypertrophy and cardiac rhythm
Combined data from >30 original studies and ~60,000 participants consistently concluded that there is positive association between blood lead levels and blood pressure endpoints.


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Table 1. Reviews of the association between blood lead levels and blood pressure.

<table>
<thead>
<tr>
<th>First author, year</th>
<th>No. of studies included</th>
<th>Year of publication of studies (range)</th>
<th>Language of literature search</th>
<th>Total no. of participants</th>
<th>Age range of participants (years)</th>
<th>Comparison</th>
<th>Outcome</th>
<th>Median of estimates [change in mmHg (range)]</th>
<th>Conclusions as reported by authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sharp et al. 1987</td>
<td>Review 4</td>
<td>1982–1986</td>
<td>English, French</td>
<td>8,406</td>
<td>24–59</td>
<td>Per 2-fold ↑</td>
<td>SBP</td>
<td>1.9 (0.7 to 2.3)</td>
<td>Evidence consistent with causation</td>
</tr>
<tr>
<td>Hertz-Picciotto and Croft 1993</td>
<td>Review 13</td>
<td>1990–1992</td>
<td>English</td>
<td>22,923</td>
<td>12–80</td>
<td>Per 2-fold ↑</td>
<td>SBP</td>
<td>2.0 (−5.9 to 8.0)</td>
<td>Evidence strongly supports causal association</td>
</tr>
<tr>
<td>Staessen et al. 1994, 1995</td>
<td>SR, MA 23</td>
<td>1990–1993</td>
<td>English, French, German</td>
<td>33,141</td>
<td>10–88</td>
<td>Per 2-fold ↑</td>
<td>SBP</td>
<td>1.0 (−0.4 to 1.6)</td>
<td>MA suggests a weak association</td>
</tr>
<tr>
<td>Schwartz 1995</td>
<td>SR, MA 15</td>
<td>1985–1993</td>
<td>English, Men only</td>
<td>NR</td>
<td>18–76</td>
<td>Per 2-fold ↑</td>
<td>SBP</td>
<td>1.25 (0.87–1.63)</td>
<td>MA consistent with causal association</td>
</tr>
<tr>
<td>ATSDR 1999</td>
<td>SR 24</td>
<td>1980–1996</td>
<td>No language restriction</td>
<td>NR</td>
<td>All ages</td>
<td>Per 2-fold ↑</td>
<td>NR</td>
<td>NR</td>
<td>Suggestion of ↑ blood pressure, but evidence is inconclusive</td>
</tr>
<tr>
<td>Nawrot et al. 2002</td>
<td>SR, MA 31</td>
<td>1990–2001</td>
<td>English, French, German</td>
<td>58,518</td>
<td>10–90</td>
<td>Per 2-fold ↑</td>
<td>SBP</td>
<td>1.0 (0.5–1.4)</td>
<td>MA suggests a weak association</td>
</tr>
<tr>
<td>U.S. EPA 2006</td>
<td>SR, MA 9</td>
<td>1990–2003</td>
<td>English</td>
<td>27,424, 34,740</td>
<td>14–93</td>
<td>Per 2-fold ↑</td>
<td>DBP</td>
<td>0.61 (0.46–1.16)</td>
<td>MA suggests an effect of blood lead on SBP</td>
</tr>
</tbody>
</table>

Abbreviations: ‡, different; ↑, increase; CI, confidence interval; DBP, diastolic blood pressure; MA, meta-analysis; NHANES, National Health and Nutrition Examination Survey; NR, not reported; RR, relative risk; SBP, systolic blood pressure; SR, systematic review; U.S. DHHS, U.S. Department of Health and Human Services; U.S. EPA, U.S. Environmental Protection Agency.

*Systematic review: a search strategy and criteria for manuscript selection are specified. Meta-analysis: a pooled analysis using meta-analysis techniques are presented. ‡In the study by Sharp et al. (1987), we divided by 3 the change per 15 μg/dl (equivalent to comparing 10 μg/dl vs. 5 μg/dl). The study by Schwartz et al. (1995) reports the change in mmHg comparing 10 μg/dl vs. 5 μg/dl. ‡Pooled estimate using an inverse variance weighted random-effects model (Egger et al. 2001) of two pooled estimates for linear and log-linear estimates, respectively.
Lead and mortality – NHANES III
Mortality Follow-up

Figure 1. Multivariate adjusted relative hazard (left axis) of mortality associated with blood lead levels between 0.05 µmol/L (1 µg/dL) and 0.48 µmol/L (10 µg/dL). Histogram of blood lead levels is superimposed in the background and displayed on the right axis.

N = 13,964
Follow-up 12 years

### Bone vs. blood lead and myocardial infarction – VA Normative Aging Study

**Baseline 1991**
- \( N_{CVD-free} = 837 \)
- Men 21-80 y, Boston

**Follow-up Dec 2001**
- 83 fatal and nonfatal CHD events

**Blood lead (AAS)**
- Clinical exams every 3 to 5 years

**Patella, tibia lead (KXRF)**

**Questionnaire**

**Clinical exam**

**Laboratory**

<table>
<thead>
<tr>
<th></th>
<th>( \text{HR}_{\text{crude}} )</th>
<th>95% CI</th>
<th>( \text{HR}_{\text{adj}} )</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood lead (per log ( \mu g/dL ))</td>
<td>1.40</td>
<td>0.99 – 1.98</td>
<td>1.45</td>
<td>1.01 – 2.06</td>
</tr>
<tr>
<td>Patella lead (per log ( \mu g/g ))</td>
<td>3.27</td>
<td>1.41 – 7.58</td>
<td>2.64</td>
<td>1.09 – 6.37</td>
</tr>
<tr>
<td>Tibia lead (per log ( \mu g/g ))</td>
<td>2.76</td>
<td>0.94 – 8.12</td>
<td>1.84</td>
<td>0.57 – 5.90</td>
</tr>
</tbody>
</table>

Adjusted for age, race, HDL-cholesterol
No change in estimates when smoking, BMI, alcohol, blood pressure, family history of hypertension and total serum cholesterol were added to the model

**AAS:** atomic absorption spectrometry
**KXRF:** K X-ray fluorescence

Jain et al. Environ Health Perspect. Epub Feb 6 2007

Adjusted for age, sex, race, education, body mass index, alcohol intake, hypertension, diabetes, hypercholesterolemia, glomerular filtration rate and C-reactive protein

Further adjusted for smoking status (never/former/current) and serum cotinine

Public health implications of lead related cardiovascular disease

Sufficient evidence to infer a causal association with elevated blood pressure with no evidence of a threshold

Suggestive evidence for other cardiovascular endpoints at blood lead levels < 5 µg/dL

- Lower the current OSHA / WHO safety standards for blood lead in workers (40 µg/dL)
- Establishment of a criterion for elevated blood lead levels in adults
- Include hypertensive and cardiovascular effects of lead in risk assessment and in economic analyses of the impact of lead exposure
- Develop regulations and public health interventions to prevent and reduce lead exposure in adults needed
Adult Lead Exposure: Time for Change

*Brian S. Schwartz*¹,² and *Howard Hu*³

¹Departments of Environmental Health Sciences and Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland, USA; ²Department of Medicine, Johns Hopkins School of Medicine, Baltimore, Maryland, USA; ³Department of Environmental Health Sciences, University of Michigan School of Public Health, Ann Arbor, Michigan, USA

Blood lead levels defined as lead poisoning (mg/dL)

- 60 µg/dL
- 40 µg/dL
- 30 µg/dL
- 25 µg/dL
- 10 µg/dL
- 5 µg/dL

- 1976-1980 geo. mean = 15 µg/dL (children)
- 2002 geo. mean = 2.2 µg/dL (children)

OSHA criteria for workers

CDC criteria for children
Lead poisoning prevention programs

- Surveillance
- Secondary prevention
  - Case management children blood lead levels over a certain level
  - Identify most important sources related to this level of exposure: Housing age, lead paint-hazards, drinking water sources, industrial sources, other
- Primary prevention
  - Identification of most common sources, plot distribution
  - Target prevention strategies to reduce exposure of at-risk populations
  - Enact laws to require actions to protect children from exposure to hazards (housing standards)
- Secondhand smoke not considered by most programs
CDC’s new lead poisoning recommendations

1. Eliminates “level of concern”

2. Establishes a childhood BLL reference value based on 97.5\textsuperscript{th} percentile of the population BLL in US children ages 1-5 [now 5µg/dL] to:
   a. Identify children
   b. Identify environments with lead hazards

3. To develop and implement a national primary prevention strategy to ensure no US children live in or spend significant time in homes, buildings, other environments exposed to lead hazards
CDC’s new lead poisoning recommendations

4. **Clinicians** serve as a reliable source of information on lead hazards, taking primary role in educating families about prevention
   a. Environmental assessments prior to BLL screening

5. **Clinicians** notify family and monitor health status of children with confirmed BLL $\geq 5\mu g/dL$
   a. until environmental investigation/remediation complete

6. Where no mandatory reporting, **clinicians** to
   a. ensure reporting of all BLLs at or above reference value to local/state health and housing agencies
   b. collaborate with agencies to provide appropriate services and resources to children/families
CDC’s new lead poisoning recommendations

7. **Education** on **primary prevention** in homes and child-occupied facilities to eliminate hazards before children are exposed.
   
a. Targets families, providers, advocates, public officials

8. Develop **primary prevention infrastructure**:
   
a. Encourage data sharing between health and housing
   b. Develop and enforce preventive lead-safe housing standards for rental and owner occupied properties
   c. ID funding for lead hazard remediation
   d. Provide families with information so they can protect their children from home environment hazards
9. Work with elected officials, health, housing and code enforcement agencies to ensure adoption of a suite of **primary prevention policies** to protect children from lead exposure in their homes.

10. Adopt **primary prevention strategies** to reduce environmental exposures in soil, dust, paint and water before children are exposed

   a. Emphasize environmental assessment to ID and remediate lead hazards before children’s BLLs are at/above reference value
CDC’s new lead poisoning recommendations

11. **Multi-family housing**: if lead hazards trigger actions in any unit, apply the same actions to all similar but untested units in the complex unless risk assessment shows no hazards are present.

12. Encourage **health outcomes research** focused on interventions that can maintain child BLLs below reference value.

13. **Research priorities**:
   a. improve use of screening data,
   b. develop point-of-care analyzers,
   c. improve knowledge of epigenetic mechanisms of lead action.
Recommendations - Summary

Major shift to primary prevention
   Federal
   State
   Local
   Private providers
   Families

No level that can be thought to be “safe”

Unacceptable to wait until children reach a specific BLL to “qualify” for lead-safe housing
Secondhand tobacco smoke (SHS) remains a major source of indoor air pollution worldwide, causing major health effects in children, including sudden infant death syndrome, lower respiratory tract infections, reduced lung growth, and behavioral problems. In the United States, around 1 in 5 children aged 3 to 11 years live with at least 1 individual who smokes. Globally, the burden of SHS exposure during childhood is even higher. Lead, a major neurocognitive and kidney toxicant for children at relatively low levels, is a tobacco constituent that is measured in mainstream smoke (exhaled by the smoker) and sidestream smoke (from the burning cigarette), including the gas phase. During the period 1988 to 1994, US children exposed to SHS showed increased blood lead levels.

National and local childhood lead poisoning prevention programs identify and follow children.

**Objectives.** We evaluated the relationship between secondhand tobacco smoke (SHS) exposure and blood lead levels in US children and adolescents.

**Methods.** We analyzed data from 6830 participants aged 3–19 years in the National Health and Nutrition Examination Survey (1999–2004) who were not active smokers and for whom SHS exposure information and blood lead measurements were available.

**Results.** After multivariable adjustment, participants in the highest quartile of serum cotinine ($\geq 0.44$ µg/L) had 28% (95% confidence interval $= 21\%$, 36%) higher blood lead levels than had those in the lowest quartile ($<0.03$ µg/L). Similarly, blood lead levels were 14% and 24% higher in children who lived with 1 or with 2 or more smokers, respectively, than they were in children living with no smokers. Among participants for whom lead dust information was available, the associations between SHS and blood lead levels were similar before and after adjustment for lead dust concentrations.

SHS and Lead in NHANES III

Points: geometric mean; vertical bars: 95% CI

Mannino et al. Epidemiology 2003;14:719-727
## Ratio (95% CI) of geometric mean of blood lead by SHS exposure in NHANES 1999-2004

<table>
<thead>
<tr>
<th>Cotinine (µg/L)</th>
<th>N (%)</th>
<th>+Age, sex, race, country born, BMI, survey yr</th>
<th>+Household education, income</th>
<th>+Year home construction</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 0.03</td>
<td>1,538 (25%)</td>
<td>1.00 (ref)</td>
<td>1.00 (ref)</td>
<td>1.00 (ref)</td>
</tr>
<tr>
<td>0.03–0.07</td>
<td>1,876 (25%)</td>
<td>1.10 (1.04-1.16)</td>
<td>1.09 (1.03-1.15)</td>
<td>1.08 (1.02-1.15)</td>
</tr>
<tr>
<td>0.08–0.44</td>
<td>1,804 (25%)</td>
<td>1.26 (1.21-1.32)</td>
<td>1.19 (1.14-1.24)</td>
<td>1.17 (1.12-1.23)</td>
</tr>
<tr>
<td>≥ 0.44</td>
<td>1,612 (25%)</td>
<td>1.47 (1.40-1.55)</td>
<td>1.30 (1.23-1.37)</td>
<td>1.28 (1.21-1.35)</td>
</tr>
<tr>
<td>p-value for trend</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

### Smokers at home

<table>
<thead>
<tr>
<th></th>
<th>N (%)</th>
<th>+Age, sex, race, country born, BMI, survey yr</th>
<th>+Household education, income</th>
<th>+Year home construction</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>5,484 (78%)</td>
<td>1.00 (ref)</td>
<td>1.00 (ref)</td>
<td>1.00 (ref)</td>
</tr>
<tr>
<td>1</td>
<td>929 (14%)</td>
<td>1.26 (1.18-1.33)</td>
<td>1.16 (1.08-1.23)</td>
<td>1.14 (1.07-1.22)</td>
</tr>
<tr>
<td>≥ 2</td>
<td>417 (8%)</td>
<td>1.39 (1.32-1.47)</td>
<td>1.25 (1.17-1.33)</td>
<td>1.24 (1.16-1.33)</td>
</tr>
<tr>
<td>p-value for trend</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>
Ratio of blood lead levels: Smokers at home vs. no smokers

<table>
<thead>
<tr>
<th>Sex</th>
<th>Male</th>
<th>1.20 (1.25-1.45)</th>
<th>p = 0.85</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>1.16 (1.07-1.24)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>3-5</td>
<td>1.25 (1.12-1.37)</td>
<td>p = 0.87</td>
</tr>
<tr>
<td></td>
<td>6-11</td>
<td>1.16 (1.07-1.24)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12-14</td>
<td>1.22 (1.12-1.31)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>15-19</td>
<td>1.13 (1.02-1.24)</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>White</td>
<td>1.17 (1.08-1.26)</td>
<td>p = 0.49</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>1.16 (1.07-1.24)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mex/Am</td>
<td>1.07 (0.96-1.18)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>1.23 (1.06-1.40)</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>&lt;85</td>
<td>1.21 (1.14-1.29)</td>
<td>p = 0.01</td>
</tr>
<tr>
<td></td>
<td>85-95</td>
<td>1.18 (1.08-1.29)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;95</td>
<td>1.05 (0.95-1.15)</td>
<td></td>
</tr>
<tr>
<td>Educ</td>
<td>&lt;High School</td>
<td>1.21 (1.11-1.32)</td>
<td>p = 0.46</td>
</tr>
<tr>
<td></td>
<td>High School</td>
<td>1.16 (1.05-1.27)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;High School</td>
<td>1.16 (1.16-1.09)</td>
<td></td>
</tr>
<tr>
<td>PIR</td>
<td>&lt;1.3</td>
<td>1.20 (1.11-1.23)</td>
<td>p = 0.12</td>
</tr>
<tr>
<td></td>
<td>1.3-3.5</td>
<td>1.19 (1.08-1.30)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;3.5</td>
<td>1.05 (0.95-1.15)</td>
<td></td>
</tr>
<tr>
<td>House</td>
<td>Before 1950</td>
<td>1.19 (1.02-1.37)</td>
<td>p = 0.60</td>
</tr>
<tr>
<td></td>
<td>1950-1978</td>
<td>1.15 (1.06-1.25)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>After 1978</td>
<td>1.13 (1.05-1.21)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>1.17 (1.04-1.30)</td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>1.20 (1.11-1.25)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Sub-analysis: Children 3-5 years of age with house dust data

<table>
<thead>
<tr>
<th>Cotinine (µg/dL)</th>
<th>N (%)</th>
<th>All adjustment variables</th>
<th>+ Window lead dust</th>
<th>+ Floor lead dust</th>
<th>+ Window and floor lead dust</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 0.03</td>
<td>133 (17)</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
</tr>
<tr>
<td>0.03–0.07</td>
<td>189 (24)</td>
<td>1.01 (0.88-1.14)</td>
<td>1.00 (0.87-1.13)</td>
<td>1.00 (0.87-1.14)</td>
<td>1.00 (0.87-1.13)</td>
</tr>
<tr>
<td>0.08–0.44</td>
<td>236 (30)</td>
<td>1.14 (1.02-1.25)</td>
<td>1.13 (1.01-1.24)</td>
<td>1.13 (1.02-1.25)</td>
<td>1.12 (1.01-1.24)</td>
</tr>
<tr>
<td>≥ 0.44</td>
<td>233 (29)</td>
<td>1.31 (1.21-1.42)</td>
<td>1.31 (1.20-1.41)</td>
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<td>p-value for trend</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

### Nº smokers at home

<table>
<thead>
<tr>
<th>Nº smokers at home</th>
<th>N (%)</th>
<th>All adjustment variables</th>
<th>+ Window lead dust</th>
<th>+ Floor lead dust</th>
<th>+ Window and floor lead dust</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>623 (78)</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
<td>1.00 (ref.)</td>
</tr>
<tr>
<td>≥1</td>
<td>168 (22)</td>
<td>1.17 (1.04-1.30)</td>
<td>1.17 (1.04-1.30)</td>
<td>1.17 (1.04-1.30)</td>
<td>1.17 (1.04-1.30)</td>
</tr>
</tbody>
</table>
TABLE 3—Lead Dust in the Homes of Children Aged 3–5 Years: National Health and Nutrition Examination Survey, United States, 1999–2004

<table>
<thead>
<tr>
<th></th>
<th>No. (Weighted %)</th>
<th>Lead Dust on Window, μg/sq ft, Median (IQR)</th>
<th>Lead Dust on Floor, μg/sq ft, Median (IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sample</td>
<td>791 (100)</td>
<td>4.9 (2.2–18.7)</td>
<td>0.46 (0.24–0.93)</td>
</tr>
<tr>
<td>Child’s blood lead level, μg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤0.8</td>
<td>72 (13)</td>
<td>2.8 (1.6–4.8)</td>
<td>0.28 (0.17–0.46)</td>
</tr>
<tr>
<td>0.9–1.1</td>
<td>98 (15)</td>
<td>2.4 (1.6–5.9)</td>
<td>0.35 (0.19–0.53)</td>
</tr>
<tr>
<td>1.2–1.7</td>
<td>184 (27)</td>
<td>4.6 (2.4–12.2)</td>
<td>0.39 (0.23–0.67)</td>
</tr>
<tr>
<td>≥1.8</td>
<td>437 (45)</td>
<td>8.9 (2.8–37.7)</td>
<td>0.70 (0.36–1.56)</td>
</tr>
<tr>
<td>Child’s serum cotinine level, μg/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤0.03</td>
<td>133 (20)</td>
<td>2.9 (1.8–6.3)</td>
<td>0.33 (0.16–0.52)</td>
</tr>
<tr>
<td>0.031–0.074</td>
<td>189 (21)</td>
<td>3.3 (2.0–15.8)</td>
<td>0.50 (0.26–0.82)</td>
</tr>
<tr>
<td>0.075–0.44</td>
<td>236 (31)</td>
<td>5.4 (2.1–22.3)</td>
<td>0.45 (0.24–0.90)</td>
</tr>
<tr>
<td>≥0.441</td>
<td>233 (28)</td>
<td>7.9 (3.0–29.2)</td>
<td>0.66 (0.27–1.23)</td>
</tr>
<tr>
<td>Smoking at home</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>623 (79)</td>
<td>4.3 (2.0–13.1)</td>
<td>0.41 (0.23–0.80)</td>
</tr>
<tr>
<td>Yes</td>
<td>168 (21)</td>
<td>12.3 (3.0–34.9)</td>
<td>0.67 (0.28–1.24)</td>
</tr>
</tbody>
</table>
Implications for Public Health

- Eliminating SHS exposure in children could lower lead exposure and reduce adverse lead-related health effects.
- Lead poisoning prevention programs should systematically evaluate smoking at home (no. smokers, smoking bans).
- Lead poisoning prevention programs can borrow strategies from SHS prevention programs (explaining benefits of smoke-free homes and cessation counseling).
- Smoke-free programs can incorporate lead prevention as an argument to implement tobacco control initiatives, particularly in disadvantaged communities at increased risk of both lead and SHS exposure.