Environmental Tobacco Smoke Exposure in Inner-City Children

Virginia M. Weaver, Cecilia T. Davoli, Sharon E. Murphy, Jordi Sunyer, Patrick J. Heller, Stephen G. Colosimo, and John D. Groopman


Abstract

Exposure to environmental tobacco smoke (ETS) was assessed as part of a pilot study aimed at determining the extent of multiple toxicant exposures in children from inner-city areas of Baltimore, MD. Questionnaire data on sources of ETS and urinary cotinine were obtained in children considered at high risk for urban exposures because of previous or current overexposure to one inner-city environmental hazard, lead. Fifty-three (67.1%) of the 79 participants were exposed to ETS in the preceding 48 h as assessed by questionnaire. Cotinine was present in 77 (98.7%) of the 78 samples assayed with a mean of 79.2 ng/mg creatinine (54.7 ng/ml). Eighty % of children had cotinine values ≥30 ng/mg creatinine, a level commonly associated with household ETS exposure. Levels in children without reported ETS exposure in their homes were also elevated (mean = 45.0 ng/mg creatinine). As expected, blood lead levels were elevated with a mean of 23.6 µg/dl. We conclude that these inner-city children have substantial exposures to both ETS and lead. Furthermore, the presence of elevated cotinine levels in children without known household exposure suggests that ETS should be considered an urban toxicant as well as an individual residential exposure.

Introduction

The disparity in exposure to environmental toxicants between minority, low-income communities and more affluent areas is an issue receiving increasing attention by civil rights and public interest groups, the government, and environmental health researchers (1). The contribution to disease burden made by such exposures is unknown. However, it is a concern because residents of disadvantaged communities experience increased morbidity and mortality from a number of chronic diseases, including cancer (2). Scientific investigation of the extent of exposure to various pollutants found in high-risk areas is essential in understanding these exposure/disease outcome relationships and devising appropriate prevention strategies.

In an effort to provide such data, we performed a pilot study to evaluate the extent of exposure to several urban toxicants, including ETS, in children residing in Baltimore, MD. Smoking prevalence is increased in lower socioeconomic groups; this is reflected by higher levels of the nicotine metabolite, cotinine, in children from such groups (3). The United States Environmental Protection Agency has concluded that ETS is a human lung carcinogen in adults and is causally associated with an increased risk of lower respiratory tract infections and other adverse respiratory effects in children (4). Therefore, it seems likely that inner-city children would comprise a high-risk group with respect to ETS exposure and adverse health outcomes.

Materials and Methods

Study Population and Design. All children who were seen at the Kennedy Krieger Institute’s Lead Poisoning Prevention Clinic (Baltimore, MD) during a 4-week study period in September and October 1994 were eligible for enrollment. These children were originally referred to the clinic for evaluation of elevated blood lead levels; some receive ongoing follow-up due to persistently elevated blood leads or lead-related learning problems. This population was selected because children already exposed to one urban toxicant are likely to represent a high-risk group for other exposures. Parent/guardians and children were invited to participate during the clinic visit. Explanations to the adults and children (appropriately age-modified) were provided and informed consent was obtained from all participants. A questionnaire, administered to the parents, elicited basic information on the child, consisting of medical and environmental histories with data on sources of ETS exposure in the preceding 48 h. A urine sample was obtained and stored at –20°C until analyzed. Venous blood lead levels were obtained as part of routine clinical care. The parent/guardians of 117 children were approached regarding the study. Thirty-one of the children were excluded for one of the following reasons: not toilet trained, could not produce a specimen during the clinic visit, or the guardian was not present. The parents of 79 (91.9%) of the remaining 86 children agreed to participate.

Urinary Cotinine Assay. Cotinine values were measured by RIA at the American Health Foundation (Valhalla, NY). The assay, based on the Langone et al. (5) method, uses rabbit antiserum produced through injection of trans-4-carboxycotinine bound to albumin. The antibody also cross-reacts with 3-hydroxycotinine. The inter- and intraassay variations are <6% (6). The limit of detection is 2 ng/ml.

The abbreviation used is: ETS, environmental tobacco smoke.
Short Communication: ETS Exposure in Inner-City Children

One-half of the limit of detection.

Table 1. Urinary cotinine results

<table>
<thead>
<tr>
<th>Cotinine (ng/ml)</th>
<th>Cotinine:creatinine ratio (ng/mg creatinine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD</td>
<td>54.7 ± 45.6</td>
</tr>
<tr>
<td>Range</td>
<td>1&quot;-244</td>
</tr>
<tr>
<td>Geometric mean ± SD</td>
<td>37.5 ± 2.8</td>
</tr>
<tr>
<td>Median</td>
<td>44.5</td>
</tr>
<tr>
<td>≥30 ng/mg creatinine</td>
<td>62 (79.5%)</td>
</tr>
<tr>
<td>% detectable</td>
<td>77 (98.7%)</td>
</tr>
</tbody>
</table>

* One-half of the limit of detection.

Urinary Creatinine Assay. Creatinine was measured using the Sigma kit (Sigma Chemical Co., St. Louis, MO). This colorimetric assay is based on the difference of absorbance at 500 nm of the creatinine-picrate chromogen before and after acidification. The assay described in the kit was followed using a 10-fold urine dilution and one-third of the suggested sample acidification. The assay described in the kit was followed using a 10-fold urine dilution and one-third of the suggested sample acidification. Thus, 1 ml of alkaline picrate solution was added to 100 μl of diluted urine and 33 μl of acid reagent were added subsequently.

Blood Lead Analysis. Anodic stripping voltammetry was used to measure blood lead levels. An Environmental Science Associates 3010A Trace Metals analyzer with mercury-coated graphite electrode, Ag/AgCl reference electrode, and platinum counter electrode was used (Environmental Science Associates, Bedford, MA). The limit of detection is approximately 2 μg/dl.

Statistical Analysis. Because the cotinine distributions were skewed rightward, the natural logarithmic transformations (In) were used in analyses. The one value below the limit of detection was set at one-half of its level for data analysis. Initial comparisons included Pearson correlation coefficients for continuous variables and t-tests for dichotomous variables. Multiple linear regression models were fitted to assess independent associations while adjusting for other possibly confounding variables. All analyses were performed in BMDP (Los Angeles, CA).

Results

Seventy-nine children participated in the study. The mean age was 4.3 years with a median of 3.8 years; only five of the children were over 6 years old. The population was 96.2% African-American and 51.9% female. As expected, given the selection criteria of the study, the mean blood lead level was elevated at 23.6 μg/dl with a range of 5–45 μg/dl. Only two children had blood lead levels below the 10 μg/dl action level recommended by the Centers for Disease Control and Prevention (7).

Table 1 shows cotinine levels with and without adjustment for creatinine on 78 children (one specimen was too small to analyze). Almost 80% of the creatinine adjusted values were above 30 ng/mg creatinine, a level commonly thought to indicate household ETS exposure (8). More importantly, despite a limit of detection of 2 ng/ml, cotinine was measurable in the urine of all but one child.

Questionnaire data with accompanying cotinine levels are depicted in Table 2. Sixty-seven % of the subjects had been exposed to ETS in their home in the past 48 h (including 3 children whose parents smoked only outside the house). The difference in the mean ln cotinine:creatinine ratio between exposed and unexposed subjects is statistically significant (P < 0.0001). Levels in the unexposed children were also quite elevated. Exposure to parental smoke was not excessive, with 29.1% of children exposed to maternal ETS and 22.7% exposed to paternal ETS in the preceding 48 h. However, living arrangements provided the potential for several smoking adults to visit or live in the same residence. This resulted in a median of two smokers in the household of each child in the exposed group. For example, a grandmother who smoked was present in the homes of 16 (20.3%) of the children. Maternal smoking, when present, made a large contribution to ETS exposure, although this is not apparent in the “maternal smoker only” category because the mother of two of the six children smoked only outside the home.

Multiple linear regression analysis revealed associations noted previously by other researchers. The ln cotinine:creatinine ratio was strongly associated with total number of nonmaternal smokers in the household, maternal smoking, and age in years (Table 3). Sex made a borderline contribution with increased values in females (P = 0.07). Cotinine measures were not correlated with any lead exposure results (r = −0.051 for the correlation between blood lead and the ln cotinine:creatinine ratio).

Discussion

This study demonstrates that inner-city children who are overexposed to one urban toxicant, lead, also show evidence of increased ETS exposure. When compared with studies measuring urinary cotinine in children with similar mean ages (cotinine levels vary by age of the child, specimen source, and assay method), these cotinine results are strikingly elevated. For example, Chilmonczyk et al. found a range of median cotinine values from 5.6 ng/ml in unexposed children to 55.8 ng/ml in children exposed to ETS in homes with both maternal and other smokers (9). Erlich et al. (8) reported mean cotinine:creatinine ratios of 25.8 and 43.6 ng/mg creatinine in controls and asthmatics, respectively, with 25 and 38% of values >30 ng/mg creatinine (8). The presence of cotinine in all but one sample is

Table 2. Questionnaire data with associated cotinine values

<table>
<thead>
<tr>
<th>Questionnaire exposure status</th>
<th>n (%)</th>
<th>Mean</th>
<th>Median</th>
<th>Mean</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unexposed</td>
<td>26 (32.9)</td>
<td>33.5</td>
<td>28.5</td>
<td>45.0</td>
<td>35.8</td>
</tr>
<tr>
<td>Exposed (any household smoker)</td>
<td>53 (67.1)</td>
<td>65.3</td>
<td>53.0</td>
<td>96.3</td>
<td>80.0</td>
</tr>
<tr>
<td>Maternal and other smokers</td>
<td>17 (21.5)</td>
<td>91.6</td>
<td>79.0</td>
<td>127.5</td>
<td>95.4</td>
</tr>
<tr>
<td>Maternal smoker only</td>
<td>6 (7.6)</td>
<td>44.3</td>
<td>40.5</td>
<td>88.6</td>
<td>99.8</td>
</tr>
<tr>
<td>Nonmaternal smokers only*</td>
<td>30 (38.0)</td>
<td>54.2</td>
<td>46.0</td>
<td>79.7</td>
<td>78.6</td>
</tr>
</tbody>
</table>

* Cotinine values for 52 subjects.

* Cotinine values for 29 subjects.

Downloaded from cebp.aacrjournals.org on October 20, 2017. © 1996 American Association for Cancer Research.
also unusual unless the assay used has an extremely low detection limit.

We identified two reports with cotinine results similar to ours. One study focused on an asthmatic pediatric clinic population that was also drawn from urban Baltimore (10). These researchers found detectable cotinine in 100% of subjects; maternal smoking rate (63%) was much higher than in our population and the mean cotinine:creatinine ratio was slightly higher. The other report, in Australian children, found detectable cotinine in 94% (11). Median cotinine ranged from 31 ng/mg creatinine in children who were unexposed in their homes to 74 ng/mg creatinine in the highest group (both parents smoked).

Several reasons for our elevated cotinine values are possible. Most importantly, ETS exposure was high, as evidenced by the fact that 67.1% of the children had household exposure. This is consistent with the low socioeconomic status of the majority of these children. Although not specifically addressed in this study, the clinic population as a whole has 55.2% Medicaid patients and 35.4% health maintenance organization patients. Ethnic metabolic differences may also play a role.

Several studies have noted higher cotinine levels in similarly exposed African-American subjects compared to Caucasian or Hispanic subjects (12-14). In addition, this select group of children may have metabolic differences due to enzyme induction from exposure to multiple toxins.

The adverse health implications of this ETS exposure are significant. These children are at an increased risk for respiratory infections such as bronchitis and pneumonia, upper respiratory tract irritation, reduced lung function, and asthma (4). Recently, tobacco smoke-related carcinogens have been found in biological specimens from adults exposed to sidestream smoke and children passively exposed to smoke, thus providing additional evidence of the carcinogenic potential of ETS (12, 15).

The presence of cotinine in subjects who have no home ETS exposure has led other authors to conclude that this exposure should be considered as a community concern (16, 17). Cook et al. (16) have shown that mean cotinine levels in children without household ETS exposure are correlated with maternal smoking prevalence in the community. Jarvis et al. (3) have shown that, in addition to household exposure, indicators of poverty, such as low socioeconomic status and renting one’s home, are risk factors for higher cotinine values in children. Even if some parents underreported ETS exposure, our data provide further confirmation that it is a community environmental problem in low-income urban areas.

Clearly, efforts to reduce smoking and passive exposure are essential. Cotinine-assisted programs may be beneficial in encouraging parents to quit smoking or to smoke outside the home (18). If ETS is considered an urban toxicant and not just an individual habit, public policy options could include restriction of smoking in public places and efforts to reduce advertiser targeting of inner-city areas.

Acknowledgments

We would like to thank the children and parents who participated in this study. We are also grateful to the staff of the Kennedy Krieger Institute Lead Poisoning Prevention Clinic for their assistance in this project and to Jonathan Samet, MD (Johns Hopkins University School of Hygiene and Public Health), for his thoughtful review of the manuscript.

References

Environmental tobacco smoke exposure in inner-city children.
V M Weaver, C T Davoli, S E Murphy, et al.

Updated version
Access the most recent version of this article at:
http://cebp.aacrjournals.org/content/5/2/135

E-mail alerts
Sign up to receive free email-alerts related to this article or journal.

Reprints and Subscriptions
To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.

Permissions
To request permission to re-use all or part of this article, contact the AACR Publications Department at permissions@aacr.org.