

A community-based study of tobacco smoke exposure among inner-city children with asthma in Chicago

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Background: Little is known about the level of tobacco exposure and the factors that influence exposure in children with persistent asthma.

Objective: We sought to measure tobacco smoke exposure and determine factors associated with exposure in a large urban sample of asthmatic children.

Methods: This cross-sectional study is based on a community-based cohort of 482 children (8-14 years old) with persistent asthma. Caregiver and household tobacco use were reported by the caregiver. Child tobacco smoke exposure was assessed by using salivary cotinine level. Multivariate linear regression of log-transformed salivary cotinine levels were used to characterize the relationship between smoke exposure and caregiver, household, and demographic characteristics. We used a multivariate logistic model to characterize associations with caregiver smoking.

Results: Overall, 68.5% of children had tobacco smoke exposure. Compared with nonexposed children, those exposed to smoking by a caregiver or another household member had cotinine levels that were 1.68 (95% CI, 1.45-1.94) or 1.40 (95% CI, 1.22-1.62) times higher, respectively. Compared with Hispanic children, African American and white/other children had 1.55 (95% CI, 1.16-2.06) and 1.59 (95% CI, 1.18-2.14) times higher cotinine levels, respectively. Child exposure was also associated with caregiver depression symptoms (odds ratio, 1.01; 95% CI, 1.01-1.02), and higher household income was protective (odds ratio, 0.73; 95% CI, 0.56-0.95). Independent predictors of caregiver smoking included a protective effect of higher education (odds ratio, 0.35; 95% CI, 0.15-0.83) and a positive association with potential problematic drug/alcohol use (odds ratio, 2.30; 95% CI, 1.39-3.83).

Conclusions: Tobacco smoke exposure was high in this urban sample of asthmatic children. Caregiver smoking was strongly associated with child exposure and also was associated with lower socioeconomic status, non-Hispanic ethnicity, and depression symptoms. (J Allergy Clin Immunol 2008;122:754-9.)

Key words: Inner-city asthma, child, tobacco smoke exposure, cotinine

Exposure to second-hand cigarette smoke is associated with increased asthma incidence,¹⁻⁴ increased rates of health care use, and respiratory morbidity.^{5,6} Despite the strength of the associations between tobacco smoke exposure and child health, little is known about the level of tobacco smoke exposure among urban children with persistent asthma, a population at high risk for tobacco-related asthma morbidity and at high risk to become smokers.⁷ Rates of smoking in the inner-city and the inner-city minority populations have been reported to be around 40%, depending on sex and ethnicity.⁸⁻¹⁰ In what appears to be the largest study of this topic, for children in 6 US cities, the presence of a child with asthma living in the home was not associated with lower rates of smoking.¹¹ However, that study did not measure biomarkers from children to ascertain actual smoking exposure.

The purposes of this study were to objectively determine the prevalence and degree of tobacco smoke exposure (as measured by means of child salivary cotinine levels) among children with persistent asthma living in a major US city. Secondarily, we also characterize how sociodemographic, economic, and psychologic factors might influence caregiver smoking.

METHODS

Sample

This analysis is based on the cohort established by the Chicago Initiative to Raise Asthma Health Equity (CHIRAH) study. The CHIRAH cohort is a community-based longitudinal cohort study of urban children and adults with persistent asthma. CHIRAH was designed to examine the effect of socioeconomic and psychosocial stressors on asthma health disparities. Subjects were enrolled from February 2004 to July 2005. The cohort was established by a broad community-based screening for households with persons with asthma by using a school-based sampling technique.¹² (Further details of sampling are available in the Methods section in this article's Online Repository at www.jacionline.org.)

Children were eligible if they were age 8 to 14 years and had persistent symptomatic asthma, which is defined as requiring at least 8 weeks of asthma medication over the previous 12 months. This analysis examined 482 child/caregiver dyads (85.9% of the cohort). Only 1 dyad was recruited per household. Pairs were excluded from this analysis if there were incomplete caregiver-reported smoking data, a salivary cotinine value was not obtained, or salivary cotinine levels were higher than the designated cutoff point for active smoking on the part of the child (>8 ng/mL). Subjects who were not included

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Abbreviations used

CES-D: Clinical Epidemiological Survey of Depression
CHIRAH: Chicago Initiative to Raise Asthma Health Equity
SES: Socioeconomic status

in the analyses because of refusal or inability to obtain a sample ($n = 57$) did not differ from those included in this analysis ($n = 482$) in any of the variables described below.

Data presented herein come from the initial face-to-face interviews with participants, which were carried out in a community setting. At that visit, trained research assistants administered a standardized questionnaire, which collected data on the demographic, economic, and psychosocial characteristics of the individual and household. It also collected data on asthma-related symptoms, knowledge, behaviors, use of health services, and health literacy in adults.

Study subjects also had blood drawn for Immunocap testing (Pharmacia, Uppsala, Sweden) for dust mite and cockroach allergens, saliva collected for assessment of cotinine levels, spirometric analysis performed, and height and weight documented.

Salivary cotinine measurements

Saliva was collected with the Quantisal (Immunoassay, Pomona, Calif) collection pad, and the extracted cotinine concentration was determined by using a commercially available ELISA kit (Salimetrics, State College, Pa). This assay has a lower limit of detection of 0.05 ng/mL. Children with cotinine levels of 8 ng/mL or greater were excluded from the analyses ($n = 22$, or 4.4% of the 504 children with cotinine levels measured) because reports suggest that a level of 8 to 15 ng/mL is associated with active smoking on the part of the child.^{13,14} A recent report showed that a level of 8 ng/mL or greater afforded a sensitivity of 0.975 and a specificity of 0.968.¹⁴ Reports have suggested that levels ranging from 1 to 2 ng/mL might be the result of passive smoking.¹⁵⁻¹⁷ Levels in the range of 2 to 8 ng/mL were still considered passive smoking, given the reference levels in the literature.

Self-reported smoking variables

Smoking by the caregiver was determined by self-report as "not at all," "some days of the week," or "every day of the week." For the purposes of this analysis, caregivers were classified as smoking if they responded either "some days of the week" or "every day of the week." We also determined the number of smokers in the house aside from the caregiver and the number of rooms in the house, providing us with the number of smokers per room. The children were not asked whether they actively smoked because the questionnaire was administered with the parent present, and there were no questions about exposure outside of the home.

Demographic variables

Ethnicity was based on self-report as per National Center for Health Statistics categories. Subjects could choose more than 1 race/ethnicity. For the purposes of this analysis, we classified individuals into 3 race/ethnicity categories based on their responses: Hispanic; African American, non-Hispanic; and white/other, non-Hispanic, and non-African American. This final group was 91% white. Rather than excluding the data from child/caregiver dyads classified as "other" racial groups, we combined them with the white group on the basis of sensitivity analyses with and without these subjects. There were no changes in magnitude or significance of the associations to warrant their exclusion. Individuals who recorded both Hispanic and African American ancestry were coded as Hispanic because this might have bearing on patterns of smoking. The demographic variables of caregiver age and sex were also considered in the models.

Socioeconomic status variables

The socioeconomic variables available included income, education, work status, and home ownership, as well as the insurance status of the child. Of these, we used self-reported income based on level of significance in the model. Self-reported income was assessed as annual household income in one of 4 categories: less than \$15,000, \$15,000 to \$30,000, \$30,000 to \$50,000, and greater than \$50,000 per year.

Psychosocial variables

Other variables included a validated score of depressive symptoms: the Clinical Epidemiological Survey of Depression (CES-D). The CES-D is a 20-item depression-screening tool designed for use in the general population.¹⁸ The total number of negative stressors from the Crisis in Family Systems instrument, a validated measure of life stress,¹⁹ was also considered. A positive screen result for the use of alcohol and other drugs was determined by using a previously published standardized questionnaire that was adapted from the CAGE-Adapted to Include Drugs (CAGE-AID) questionnaire.²⁰

Statistical analysis

Our primary analysis sought to determine what factors were associated with higher cotinine levels in children. Because cotinine levels were not normally distributed, the outcome variable was log-transformed to satisfy assumptions for linear regression. Consequently, arithmetic means do not estimate the central tendency of these data, and geometric means (computed as the anti-log of the mean of the log-transformed variable) and 95% CIs were reported. The anti-log of the coefficients derived from the regression models are presented (β^*) because this results in a value that is expressed in the original units of nanograms per milliliter. A β -coefficient that was additive on a log scale serves a multiplicative function on a non-log scale when it is converted to its anti-log. As such, all β^* values represent the multiplicative factor by which average levels of cotinine change between groups when this level is expressed in nanograms per milliliter.

Univariate regression analyses were used to assess the relationship between the transformed cotinine levels and all variables of interest, including household smoking, demographics, socioeconomic status (SES), and psychosocial characteristics. We first looked at which of the household smoking variables had the greatest effect on cotinine levels. Once this was determined, demographic, SES, and psychosocial variables were added to the model. Manual backwards stepwise techniques were used to detect collinearity and determine the best variables to remain in the final multivariate model.

A secondary analysis was carried out to evaluate the psychosocial and demographic factors associated with caregiver smoking. Univariate logistic regression analyses were used to assess which of the abovementioned variables had the greatest association with caregiver smoking. The same manual backward stepwise techniques used in the primary analysis were used to determine the final model.

In all models robust variance estimators were computed to account for potential within-school clustering of study subjects. All analyses were done with Stata version 9.2 (StataCorp, College Station, Tex).

The protocol was approved by the institutional review boards of Northwestern University, Children's Memorial Hospital and the Cook County Bureau of Health Services.

RESULTS

Baseline characteristics

Table I displays the child and caregiver characteristics of the 482 child/caregiver dyads. The mean age of the children was 10.5 ± 1.8 years (49.4% ≥ 11 years), and 58.5% were boys. More than 90% of caregivers were female, with an average age of 38.3 ± 8.0 years. The caregivers were predominantly mothers (87%). Only 6.4% of primary caregivers were fathers. The remainder of the caregivers were other relatives, the largest fraction of which (2.5% of the total) were grandparents. Interestingly, 36%

TABLE I. Child and caregiver/household characteristics in an inner-city sample of families with asthmatic children (n = 482)

	Children (n = 482)	Caregiver/ household
Demographics		
Age (y), mean (SD)	10.5 (1.8)	38.3 (8.0)
Sex (% female)	41.5	93.2
Race/ethnicity (%)		
HA	26.4	23.6
AA	55.8	54.8
W/O	17.8	21.6
SES		
Household income (%)		
<\$15,000		19.1
\$15,000–\$30,000		28.0
\$30,000–\$50,000		18.9
>\$50,000		34.0
Education (%)		
<High school		11.8
High school		26.8
Some college		42.5
BA/BSc or higher		18.9
Private insurance (%)		50.2
Home ownership (%)		36.7
Caregiver employed (%)		65.6
Asthma control and severity		
Duration of asthma (y), mean (SD)	7.1 (3.4)	
Exacerbations over last 12 mo, median (IQR)	2 (0–4)	
Subjects with exacerbations over last 12 mo (%)	64	
Percent predicted FEV ₁ , median (IQR)	96.9 (87.6–107.1)	
No. of days with β -agonist use in the last 2 wk, median (IQR)	2 (0–6)	
Percentage of children on an inhaled steroid	40	
Smoking		
Child exposed to tobacco smoke (≥ 1 ng/mL), no. (%)	330 (68.5)	
Log cotinine level, geometric mean (95% CI)	1.4 (1.3–1.5)	
Any smokers in household (%)		49.6
Caregiver smoking (%)		31.3
Presence of smokers other than caregiver (%)		29.7
No. of smokers in home with a smoker, mean (SD)		1.4 (0.66)
Percentage of caregivers with asthma		36
Psychosocial		
Caregiver drug or alcohol use (%)		14.3
CES-D, mean (SD)		13.8 (11.2)
CRISYS, mean (SD)		8.5 (5.9)

HA, Hispanic American; AA, African American, non-Hispanic; W/O, white/other, non-Hispanic, non-African American; CRISYS, Crisis in Family Systems.

*Exacerbations represent the total number of asthma exacerbations over the last 12 months requiring any of the following: hospitalization, emergency department care, or same-day medical care.

of the caregivers themselves had asthma, and of these, 34% reported smoking. The racial distribution of the children and caregivers was similar, with more than half being of self-described African American ethnicity. By design, this sample disproportionately enrolled lower-income participants and African American participants. The children included in this sample had asthma for a mean duration of 7.1 years and a median of 2 exacerbations (hospitalizations, emergency department visits, or same-day urgent care visits) over the last year, despite relatively normal pulmonary function in a large proportion of the children. Only 40% of these children were taking an inhaled steroid.

Tobacco smoke exposure

Overall, 68.5% children demonstrated evidence of tobacco smoke exposure (with a cotinine level >1 ng/mL), with a geometric mean cotinine level of 1.14 ng/mL (95% CI, 1.32–1.52). Levels of salivary cotinine did not significantly differ by age group, with 8- to 10-year-olds having levels of 1.40 ng/mL (95% CI, 1.27–1.55) and 11- to 15-year-olds having levels of 1.43 ng/mL (95% CI, 1.29–1.58; $P = .78$). However, levels were slightly higher for girls (1.54 ng/mL; 95% CI, 1.39–1.71) than boys (1.33 ng/mL; 95% CI, 1.21–1.47), although these differences were of borderline significance ($P = .05$).

More than 31.3% of caregivers reported being active smokers: this was similar among female/male caregivers (27.3% vs 31.6%, respectively) and younger (<40 years of age) versus older caregivers (31.1% vs 31.6%, respectively). Household smoking was not infrequent, with 49.6% reporting the presence of a smoker in the home, despite the higher percentage of children who have objective evidence of smoke exposure (68.5%).

Relationship between child smoking exposure and caregiver and household factors

Table II characterizes the univariate and multivariate regression results between childhood exposure and a number of possible risk factors. The results in Table II are presented as the anti-log (β^*). This value can be thought of as a multiplicative factor by which average levels of cotinine change (in nanograms per milliliter) compared with the reference group. For example, children whose caregivers smoked had average cotinine levels that were 1.87 times greater than those whose caregivers did not smoke in the home.

In univariate analyses the presence of any smokers in the household predicted cotinine levels as well as or better than knowing the number of smokers in the household. Caregiver smoking ($\beta^* = 1.87$; 95% CI, 1.59–2.20; $P < .001$) affected childhood exposure more than the presence of other smokers in the household ($\beta^* = 1.52$; 95% CI, 1.29–1.79; $P < .001$). Therefore caregiver smoking and other household members' smoking were both included in the model. Hispanic ethnicity and higher SES (education, income, insurance status, home ownership, and employment) were also associated with lower levels of exposure. Because of strong relationships between home ownership, employment, and household income causing collinearity, the less traditional measures of SES were not included in the model in favor of annual household income. The same was seen in the relationship between caregiver depression (CES-D) and negative life stressors (Crisis in Family Systems). As such, the latter measure was not included in analyses.

TABLE II. Unadjusted and multivariate comparisons of children's log cotinine levels to baseline demographic and psychosocial characteristics

Baseline characteristic	Unadjusted β^* value (anti-log of coefficient) for Salivary cotinine [95% CI]	Adjusted β^* value (anti-log of coefficient) for salivary cotinine [95% CI]
No. of smokers	1.41 (1.26-1.57)§	
Smokers per room	6.55 (3.64-11.8)§	
Any smokers in household	1.81 (1.55-2.14)§	
Caregiver smoking	1.87 (1.59-2.20)§	1.68 (1.45-1.95)§
Presence of smokers other than caregiver	1.52 (1.29-1.79)§	1.40 (1.22-1.62)§
Increasing age of child	1.01 (0.98-1.04)	1.00 (0.97-1.04)
Increasing age of caregiver	1.00 (0.99-1.01)	
Child sex (female)	1.16 (1.02-1.31)†	1.13 (1.01-1.27)†
Child ethnicity		
W/O vs HA	1.27 (0.99-1.62)	
W/O vs AA	0.85 (0.67-1.07)	
AA vs HA	1.49 (1.17-1.91)‡	
Caregiver ethnicity		
W/O vs HA	1.42 (1.08-1.88)‡	1.59 (1.18-2.14)‡
W/O vs AA	0.87 (0.69-1.10)	
AA vs HA	1.63 (1.25-2.13)§	1.55 (1.16-2.06)‡
Caregiver education		
<High school	Reference	—
High school	0.98 (0.79-1.22)	1.03 (0.86-1.22)
Some college	0.78 (0.63-0.97)†	0.94 (0.77-1.16)
BA/BSc or higher	0.71 (0.57-0.89)‡	0.96 (0.76-1.22)
Income		
<\$15,000	Reference	—
\$15,000–\$30,000	0.86 (0.71-1.04)	0.87 (0.72-1.05)
\$30,000–\$50,000	0.84 (0.65-1.10)	0.88 (0.68-1.14)
>\$50,000	0.61 (0.49-0.77)§	0.73 (0.56-0.95)
Private insurance	0.74 (0.63-0.87)§	0.99 (0.85-1.15)†
Home ownership	0.69 (0.58-0.83)§	
Employment	0.81 (0.67-0.97)†	
CES-D	1.01 (1.01-1.02)§	1.01 (1.00-1.01)†
CRISYS	1.02 (1.00-1.03)†	

HA, Hispanic American; AA, African American, non-Hispanic; W/O, white/other, non-Hispanic, non-African American; CRISYS, Crisis in Family Systems.

*This value represents the anti-log of the β value for the log-transformed cotinine variable used in analysis. The anti-log of β has a multiplicative effect relating to the average non-log-transformed cotinine levels between the groups compared.

Significance: † $P < .05$, ‡ $P \leq .01$, § $P \leq .001$.

The final multivariate model examining factors associated with exposure is presented in the adjusted column in Table II. Caregiver smoking was the strongest determinant ($\beta^* = 1.68$; 95% CI, 1.45-1.95; $P < .001$) of increased child cotinine levels, and the presence of other smokers in the home also remained strongly associated with increased cotinine levels ($\beta^* = 1.40$; 95% CI, 1.22-1.62; $P < .001$). Higher SES measured based on an annual household income of greater than \$50,000 was protective for cotinine exposure ($\beta^* = 0.73$; 95% CI, 0.56-0.95; $P = .02$). Both African American and white/other subjects had higher cotinine levels than those with Hispanic ethnicity ($\beta^* = 1.55$; 95% CI, 1.16-2.06; $P = .003$ and $\beta^* = 1.59$; 95% CI, 1.18-2.14; $P = .003$, respectively). A sensitivity analysis was performed, excluding the individuals who were designated as "other" in the white/other group. Exclusion of these individuals did not alter the

associations of caregiver smoking with ethnicity. Caregiver depressive symptoms were also significantly related to increased child cotinine levels ($\beta^* = 1.01$; 95% CI, 1.00-1.01; $P < .04$).

Relationship between caregiver smoking and other caregiver and household factors

Given that caregiver smoking was the major correlate for higher cotinine levels in children, a secondary multivariate model explored possible caregiver and household factors that could be related to caregiver smoking (Table III). Controlling for other factors, only lower levels of caregiver education and a positive screen result for problematic drug or alcohol use were significantly associated with caregiver smoking, with caregiver age being borderline significant. Having other smokers in the household did not modify the effects of drug/alcohol use or increase/modify the effects of depressive symptoms on caregiver smoking. Given that asthma, diagnosis of depression, and smoking can have a complex relationship, we also carried out a sensitivity analysis whereby we included caregiver asthma in the models. Caregiver asthma was not significant and did not alter any associations.

DISCUSSION

In our community-based, low-income, urban sample of asthmatic children and their caregivers, we found a distressingly high prevalence of clinically important tobacco smoke exposure in school-age children. Caregiver smoking and the presence of other smokers in the home were major correlates of the degree of child exposure, as measured by means of salivary cotinine level. To a lesser degree, exposure was also associated with socioeconomic, ethnic/cultural, and caregiver depression symptoms.

Our findings are in keeping with prior literature, which also suggested that there are higher degrees of exposure in socioeconomically disadvantaged populations. Specifically, in low-income women aged 18 to 44 years of age, the rate of smoking is as high as 27%.⁸ This is similar to the rates of self-reported smoking in our study, in which the rate of household smoking was 49.5%, with 31.3% of caregivers admitting to smoking. Overall, in low-income African American populations, rates of smoking have been reported to be up to 40%.^{9,10} However, our study found a much greater level of exposure (68.5% with a salivary cotinine level of >1 ng/mL) than was previously documented in the aforementioned studies, which relied on self-report. Our findings reveal a self-reported prevalence of exposure similar to that seen in the National Cooperative Inner-City Asthma Study, in which 59% of inner-city children were living with a smoker and 39% had a caregiver who smoked.¹¹ However, in contrast to the National Cooperative Inner-City Asthma Study study, our study objectively quantified exposure by salivary cotinine level, with a much higher level of exposure (68.5%) when objectively measured. This is important because it establishes that exposure levels in inner-city asthmatic children are higher than previously suspected, regardless of reporting bias or whether the source of exposure was inside or outside of the home. Our study represents a community-based multiracial cohort. This type of cohort is more likely to represent a range of low- to medium-income families in urban areas.

The nature of our sample allows us to better evaluate the correlates of exposure in the community as opposed to hospital-based samples, which might not be as representative of

TABLE III. Multivariate comparisons of caregiver smoking status with demographic, socioeconomic, stress, and depression variables

Characteristic	OR of caregiver smoking	95% CI	P value
Caregiver age	1.02	1.00-1.05	.05
Caregiver ethnicity			
W/O vs HA	1.60	0.88-2.93	.13
AA vs HA	1.04	0.61-1.77	.89
Caregiver education			
<High school	Reference	—	—
High school	0.89	0.49-1.62	.70
Some college	0.68	0.38-1.21	.19
BA/BSc or higher	0.35	0.15-0.83	.02
Income			
<\$15,000	Reference	—	—
\$15,000–\$30,000	0.89	0.51-1.57	.69
\$30,000–\$50,000	1.20	0.60-2.41	.60
>\$50,000	0.65	0.30-1.41	.28
Private insurance	0.73	0.43-1.24	.24
Drug or alcohol abuse	2.30	1.39-3.83	.001
Other smokers in household	1.46	0.91-2.34	.11
CES-D	1.01	0.99-1.03	.28

OR, Odds ratio; HA, Hispanic American; AA, African American, non-Hispanic; W/O, white/other, non-Hispanic, non-African American.

community levels of smoking. In this regard, although household smoking was a major correlate of exposure, Hispanic ethnicity and higher SES were associated with less smoke exposure in children, which is consistent with a prior study²¹ and with the fact that rates of tobacco use are lower in this population.²² Also, depressive symptoms, as represented by the CES-D, were only modestly associated with increased child cotinine levels.

Prior literature suggests that individuals with higher depressive symptoms²³ or diagnoses of depression²⁴⁻²⁶ are more likely to use tobacco products and that poorer social support might be associated with smoking in chronically ill populations.²⁷ Also, depression is common in caregivers of children with asthma and might result in increased pediatric health care use, including hospitalizations for asthma.^{28,29} Given the high number of depressive symptoms claimed by our adult caregivers, we expected more intense smoking behavior, with subsequent higher cotinine levels in the children. The effect on childhood cotinine levels was small per unit change in CES-D score. One proviso of the use of these scales is that there is no literature establishing a clinically significant difference on these instruments. This was not a problem for our analysis because these measures were used as continuous variables to evaluate association and not as end points. Also, only caregiver depressive symptoms were assessed in this study and not the symptoms of any other smokers in the home. It is possible that measurement of depressive symptoms of other household members who smoke would have been also associated with child cotinine levels.

Given that caregiver smoking showed a stronger association with childhood exposure than the presence of other household smokers, our secondary analysis evaluated the determinants of caregiver smoking. This analysis suggested that lower SES and concomitant use of other substances, including alcohol and illicit drugs, are associated with caregiver smoking. The finding that lower SES is a risk factor for smoking is consistent with the literature, as reviewed above. The use of other illicit drugs and

alcohol has also been shown to be associated with smoking³⁰⁻³² and decreased ability to quit.³² It is therefore not unexpected that these are markers of caregiver smoking behavior.

Our study findings must be interpreted in the light of a number of considerations. First, the cotinine level reflects exposure at a point in time. In this our study is like most other studies in the area,¹³⁻¹⁷ which also use a single-point measurement of salivary cotinine. This method will still identify smokers with more than minimal ongoing exposure and be preferable to self-report given that subjects might misclassify their smoking status in studies.³³⁻³⁵ Furthermore, objective biomarkers of smoking might be more predictive of health outcomes than self-reported smoking status.³⁶

A third limitation to our study is that there were no measures of secondhand smoke outside of the home. Although household smoking was a common source of exposure, there was a discrepancy between reported smoking and salivary cotinine levels that indicate exposure. This raises the question of whether this discrepancy is due to exposures outside of the home in these young children and early teens or underreporting of in-home exposures. Adolescent boys with levels of less than the 8 ng/mL cutoff point for active smoking had lower salivary cotinine levels than girls. In inner-city teens estimates of active smoking have ranged from 7% to 28%,^{7,37-39} and adolescent girls might smoke more frequently.³⁹ Given the pattern of cotinine levels, we suspect that some of the exposure in the older children might be out-of-home exposure, but we cannot confirm this. Regardless of the source, exposure in these children is much more prevalent than previously appreciated.

The results of our study suggest that in urban low-income families, tobacco smoke exposure is more prevalent than was previously recognized in these school-age asthmatic children and that most tobacco smoke exposure in the home is from the caregiver. Finally, we have identified some characteristics of caregivers in this population who smoke. This is important because screening caregivers for smoking and intense intervention might be an important public health venue for decreasing the morbidity associated with asthma in low-income urban populations. Further research is also needed to determine the role of exposures outside of the home as opposed to underreported exposures in the home for these young, asthmatic, inner-city children.

Clinical implications: Interventions targeting caregiver smoking might reduce childhood asthma morbidity in urban environments.

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METHODS

Details of sampling procedures

A systematic population-proportionate sampling method was used to establish a cohort of 50% African American and two-thirds low-income families. Schools were first stratified by African American race (>50% African American) and then by income (low income as classified by >70% of the students receiving subsidized school lunch). This resulted in 92 schools identified. Also, 5 randomly selected schools in each race-income sampling group were selected as community anchors. Then the 2 geographically closest schools to each anchor school were selected to form a geographic cluster, adding a total of 40 additional schools. Of the selected 132 schools, 27 refused to participate, and 1 of the selected cluster schools had been previously selected, yielding our final sample of 105 schools.

School screening of 62,005 elementary school-aged children led to 48,917 surveys returned, with 10,143 persons with possible asthma (either children or adults in the household) on the initial screen. Of these surveys, 4900 individuals with asthma representing 3676 households agreed to be contacted for research. After an initial telephone interview, 839 children and 519 adults with asthma were considered eligible and agreed to a detailed face-to-face interview and testing. This report includes only child/caregiver dyads of this cohort. Only 1 child/caregiver dyad was recruited per household.

Children were eligible if they were age 8 to 14 years and had persistent symptomatic asthma, which was defined as requiring at least 8 weeks of asthma medication over the previous 12 months. Asthma medication requirement was chosen as a criterion (as opposed to a 2-week recall of symptoms) because ongoing medication use implies a physician's diagnosis and reflects severity beyond mild intermittent disease over a longer period of time. Child/caregiver dyads were excluded if the caregiver was not fluent in spoken English (many of the measurement instruments were only validated in English), had no telephone (needed for follow-up), or was not planning on living in Chicago for the duration of the study follow-up (18 months). Ultimately, of the 839 children with asthma and their primary caregivers screened eligible, 561 (67%) completed written informed consent and were enrolled in the study.

Further description of psychosocial variables

For use of the CES-D, participants rated each question on a 4-point scale (0 = "rarely/none" to 3 = "most of the time") in terms of the frequency with which the symptom addressed by each question occurred in the last 7 days. For this analysis, the participant's score on the CES-D was used as a continuous variable, with possible values ranging from 0 to 60.