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Household Smoking Behavior: Effects on Indoor Air Quality and Health of Urban Children with Asthma

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Abstract

The goal of the study was to examine the association between biomarkers and environmental measures of second hand smoke (SHS) with caregiver, i.e. parent or legal guardian, report of household smoking behavior and morbidity measures among children with asthma. Baseline data were drawn from a longitudinal intervention for 126 inner city children with asthma, residing with a smoker. Most children met criteria for moderate to severe persistent asthma (63%) versus mild intermittent (20%) or mild persistent (17%). Household smoking behavior and asthma morbidity were compared with child urine cotinine and indoor measures of air quality including fine particulate matter ($PM_{2.5}$) and air nicotine (AN). Kruskal–Wallis, Wilcoxon rank-sum and Spearman rho correlation tests were used to determine the level of association between biomarkers of SHS exposure and household smoking behavior and asthma morbidity. Most children had uncontrolled asthma (62%). The primary household smoker was the child's caregiver (86/126,

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68%) of which 66 (77%) were the child's mother. Significantly higher mean $PM_{2.5}$, AN and cotinine concentrations were detected in households where the caregiver was the smoker (caregiver smoker: $PM_{2.5}$ μg/m^3: 44.16, AN: 1.79 μg/m^3, cotinine: 27.39 ng/ml; caregiver non-smoker: $PM_{2.5}$: 28.88 μg/m^3, AN: 0.71 μg/m^3, cotinine:10.78 ng/ml, all $P \leq 0.01$). Urine cotinine concentrations trended higher in children who reported 5 or more symptom days within the past 2 weeks (>5 days/past 2 weeks, cotinine: 28.1 ng/ml vs. <5 days/past 2 weeks, cotinine: 16.2 ng/ml; $P = 0.08$). However, environmental measures of SHS exposures were not associated with asthma symptoms. Urban children with persistent asthma, residing with a smoker are exposed to high levels of SHS predominantly from their primary caregiver. Because cotinine was more strongly associated with asthma symptoms than environmental measures of SHS exposure and is independent of the site of exposure, it remains the gold standard for SHS exposure assessment in children with asthma.

Keywords

Asthma; Children; Cotinine; Particulate matter; Air Nicotine

Introduction

Over 30% of US children are exposed to second hand smoke (SHS) in their homes [1, 2] and 40–46% of children living in poverty are exposed to SHS in their homes [3]. However, impoverished parents are the least likely to quit smoking [4, 5]. Increased child SHS exposure is associated with increased time spent in the home, close proximity to a smoker, and living with a caregiver who smokes [6]. Young children are particularly at risk for high SHS exposure in that they spend up to 90% of their time in the homes [7] and may be more likely exposed to other members of the child's social network who smoke, i.e. extended family, child care workers and neighbors [8].

Exposure to SHS is of particular concern for inner-city African American children with asthma who incur disproportionately higher asthma morbidity [9, 10]. Despite parental awareness that SHS exacerbates asthma, 40–67% of inner-city children with asthma reside in a household with at least one smoker [11–14]. SHS exposure in children with asthma has been associated with increased risk for development and severity of asthma, [15–20] difficulty in managing asthma symptoms, [21, 22] and chronic airway inflammation [23]. Accordingly, avoidance of SHS exposures is a key component of national and international guideline recommendations for management of childhood asthma [24–26].

Assessment of SHS exposure in research studies relies primarily on biomarkers such as cotinine and less often on direct measurement of indoor air quality. The direct measures of indoor air quality that reflect SHS to at least some degree include air nicotine (AN) measurements and measurement of concentration of fine particles suspended in air. Since smoking produces particles with a diameter <2.5 microns, measurement of particulate matter <2.5 microns ($PM_{2.5}$) can be a surrogate measure for SHS exposure. In clinical practice pediatric clinicians often assess SHS exposure sporadically and rely on parent-report without use of biomonitoring [2, 27, 28] or environmental measurement. While it might seem necessary to perform biomonitoring or environmental sampling to gain sufficient evidence for assessing SHS risk, parent response to two simple questions has been proposed to predict cotinine levels in children with asthma [11]. Yet, self-report of SHS exposure often results in underreporting of the actual level of exposure in children.

The first objective of this study was to examine the association between caregiver report of household smoking behavior and both biological and environmental markers of SHS

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exposure. The second objective was to examine the association between measures of SHS and asthma morbidity outcomes among low-income, minority children with asthma residing with a smoker.

Methods

Study Design

The Particulate Reduction Education in City Homes study was a randomized controlled trial designed to test the efficacy of a home-based behavioral SHS reduction training plus home placement of two High Efficiency Particulate Air (HEPA) cleaners in reducing household SHS exposure as compared to either a home-based asthma education plus air cleaners or delayed air cleaner group among inner-city children with asthma. The study protocol was reviewed and received ethical approval by the Johns Hopkins Medical Institutional Review Board. Written informed consent was obtained from the child's primary caregiver. The randomization scheme was created with Stata and embedded in the tracking database to assign children to each group.

Data from the baseline survey, clinical and home environmental evaluations are presented in this manuscript to describe associations between characteristics of household smoking behavior and child urine cotinine concentrations or environmental measures of SHS including $PM_{2.5}$, air nicotine and asthma morbidity. Trained research assistants blinded to study assignment conducted baseline face-to-face surveys ascertaining symptom reports, health care utilization, extensive items regarding household smoking behavior and clinical evaluations for urine cotinine collection. Environmental home monitoring occurred over seven consecutive days within 2 weeks of the clinic evaluation.

Participants

Children with physician diagnosed asthma were recruited during October 2006 through December 2008 from an urban pediatric ED, hospital-based pediatric practices and graduates from two prior pediatric asthma studies. Eligibility criteria were (1) ages 6–12 years, (2) classified with intermittent or persistent asthma severity based on national guidelines using symptom frequency, [24] (3) use of daily controller medication over the past 6 months, (4) sleeping in a home 5 or more days per week and (5) with a smoker in the home. A smoker in the home was defined as someone in the home who smoked more than 5 cigarettes per day and resided in the home at least 4 days per week. Children residing in multiple households with fewer than 5 days per week per household were excluded. For families where the child's primary caregiver was not the mother (19%), we interviewed the child's primary caregiver. Caregiver was defined as the parent or legal guardian who served as the child's primary caregiver on a daily basis and provided data regarding the child's symptom reports, health care utilization, and household smoking behavior.

Measures

Asthma Morbidity and Health Care Utilization—Caregiver reports of asthma symptoms, decreased child activity level, and use of rescue medication (short acting beta agonist) during the past 2 weeks and emergency department (ED) visits or hospitalizations during the past 6 months were collected at baseline. Asthma severity was calculated using day and night symptom frequency and asthma control was calculated using frequency of SABA use within the past 2 weeks [24].

Child SHS Exposure by Caregiver-Report—The child's SHS exposure in the home was based on caregiver report of smoking location, frequency of smoking in the household, estimation of the number of cigarettes smoked in and outside the home and/or car by each

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household member including caregiver. We limited indoor air quality measures of SHS exposure to the child's primary household where the child resided 5 or more days per week. Caregiver report of success in keeping the child away from cigarette smoke was measured using a 4 point Likert scale (not at all successful, unsuccessful, successful and very successful).

Urine Cotinine—Urine cotinine has a half-life from 32 to 38 h in children [29] reflecting SHS exposure up to three to 4 days after exposure [30]. Urine samples (30 cc) were obtained from each child at baseline, labeled and frozen at −60°C and sent to the Center for Disease Control and Prevention (CDC) labs for analysis. Urinary cotinine concentrations were measured using high-performance liquid chromatography and atmospheric pressure chemical ionization tandem mass spectrometry [31, 32]. Briefly, urine aliquots were fortified with trideuterated cotinine and hydrolyzed overnight with *B*-glucuronidase, after which the samples were extracted and total cotinine was measured by liquid chromatographic atmospheric-pressure ionization, tandem mass spectrometry (LC API MS/MS). Each sample run included a set of 14 standards, one or more blanks, and aliquots of quality control materials in addition to the unknowns. All reported data are from runs confirmed as being in statistical control based on standard criteria [33]. Cotinine results are reported as nanograms per milliliter with a limit of detection (LOD) of 0.036 ng/mL.

Environmental Assessment of SHS Exposure consisted of air sampling for fine particulate matter ($PM_{2.5}$) and air nicotine (AN) concentrations. Air sampling was conducted in the child's bedroom and family/TV room because these are major activity rooms representing an indoor environment where the child spends a substantial portion of time. Continuous air sampling was conducted using $PM_{2.5}$ 4 L/min MSP impactors (St. Paul, MN) loaded with 37-mm, 2.0-um pore size, Teflo polytetrafluoroethylene membrane filters with polypropylene support rings (Pall Corporation, Ann Arbor, MI). $PM_{2.5}$ levels reflect particles with an aerodynamic diameter <2.5 um that are deposited in the lower airways and alveoli. The EPA standard ambient $PM_{2.5}$ concentration is 15 $\mu g/m^3$ [34]. Sampling flow rates were calibrated at the beginning and end of each sampling period using primary standards (DryCal; Bios International Corporation, Butler, NJ).

Air Nicotine—Two passive sampling badges were placed in the child's bedroom and the TV/family room at 3–5 feet off the floor. The passive air samplers consist of a sodium-bisulfate-treated filter contained in a 37-mm polystyrene cassette covered with a polycarbonate filter diffusion screen [35, 36]. Nicotine content was analyzed using gas chromatography with a nitrogen-phosphate detector. The limit of detection for the passive air nicotine badges was 0.003 $\mu g/m^3$.

Statistical Analysis

Summary statistics were used to examine the child's asthma morbidity, $PM_{2.5}$ and $PM_{2.5–10}$, air nicotine, urine cotinine levels and characteristics of home SHS exposure. Because of the non-normal distribution of urine cotinine and air sampling data, the Kruskal-Wallis, the Wilcoxon rank-sum test and Cuzick's test for trend across ordered groups were used to test for differences in cotinine, air nicotine and PM concentrations by household smoking and asthma morbidity measures. Spearman correlation coefficients were examined to compare air nicotine and urine cotinine concentrations and air nicotine concentrations by total number of cigarettes smoked by all household members. A 2-sided alpha of <0.05 was considered statistically significant. All analyses were performed using Stata 11.0 (Stata, College Station, TX).

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Results

Baseline Participant Characteristics

One hundred and twenty-six children were enrolled in the randomized controlled trial with baseline data presented in this manuscript. Most children were male (55%), African American (95%) and enrolled in Medicaid (90%). The mean age was 9.1 years. Asthma severity was high with 80% children categorized with persistent asthma (mild persistent, 17%; moderate persistent, 32%; and severe persistent, 31%). (Table 1) Over half of children (62%) met criteria for uncontrolled asthma based on albuterol use more than 4 days/past 2 weeks. Furthermore, three-quarters of children reported albuterol use within the past 2 weeks indicating ongoing symptoms.

Household Smoking Behavior

Almost half (46%) of the households had more than one smoker in the home (Table 2). Most caregivers reported being active smokers (68%), smoking a median of seven cigarettes per day (IQR 5–10 cigarettes) and smoked on a daily basis (45%). Caregivers' primary smoking locations were in their own bedroom (57%), kitchen (30%) or family/TV room (27%). Caregivers reported that children spent the majority of their time indoors (mean: 20.3 h/day). Median number of total cigarettes smoked by all household members was 10 cigarettes per day (IQR 5–20 cigarettes). SHS exposure in a car was moderate (27%). Over one-third (37%) of children were exposed to SHS “almost always” during the past 2 weeks. Caregiver perception of reducing their child's SHS exposure was high; 74% rated themselves as being very successful or successful in keeping their child away from SHS. Most caregivers (88%) reported making changes in household smoking rules due to their child's asthma.

Comparison of Environmental SHS and Urine Cotinine Measures by Household Smoking Behavior

Interpretable air monitoring data was available in most homes (air nicotine: 110/126, 87%; $PM_{2.5}$: 109/126, 87%). Almost all children (123/126, 98%) had urine cotinine samples available for analysis. Both mean air nicotine and urine cotinine concentrations reflected high SHS exposure (air nicotine: 1.43 $\mu g/m^3$ (SD, 2.16); urine cotinine: 22.78 ng/ml (SD 26.40). Three children, aged 6, 9 and 11 years, had urine cotinine concentrations >100 ng/ml however, the children and parents denied they smoked cigarettes. Air nicotine and urine cotinine were highly correlated at $r_s = 0.66$ ($P < 0.001$) (Fig. 1) as were air nicotine and $PM_{2.5}$ ($r_s = 0.76$, $P < 0.0001$). Correlation between caregiver report of SHS exposure, based on number of cigarettes smoked per day by all household smokers, and air nicotine was low ($r_s = 0.28$) as was correlation between caregiver report of SHS exposure and urine cotinine ($r_s = 0.41$) (Figs. 2, 3). A subgroup of children ($N = 21$, 17%) had nicotine concentrations >1 $\mu g/m^3$, yet their caregivers reported less than ½ pack cigarettes/day smoked by all household smokers. Households with two or more smokers and children living with a caregiver who smoked were significantly associated with higher concentrations of air nicotine and urine cotinine (Table 3). Additionally, children residing in homes with a caregiver who smoked had significantly higher concentrations of $PM_{2.5}$. Urine cotinine concentrations did not differ by SHS exposure in a car. Owning a home air conditioner was not associated with differences in environmental SHS measures or urine cotinine concentrations. Median urine cotinine concentrations did not differ significantly across seasons (winter: 24.7 ng/ml, spring: 9.3 ng/ml, summer: 15.6 ng/ml, fall: 12.5 ng/ml; $P = 0.07$ Cuzick's test for trend) yet, winter urine cotinine concentrations were significantly higher as compared to cotinine concentrations for all other seasons combined (winter: 24.7; all other seasons: 12.5; $P \leq 0.01$).

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Comparison of Environmental SHS and Urine Cotinine Measures by Asthma Morbidity

Although urine cotinine concentrations were increased in children who reported 5 or more symptom days or decreased activity days within the past 2 weeks, these findings did not meet criteria for statistical significance (Symptom Days: ≤5 days/past 2 weeks: 16.2 ng/ml, >5 days/past 2 weeks: 28.1 ng/ml; $P = 0.08$; Decreased Activity Days: ≤5 days/past 2 weeks: 16.2 ng/ml, >5 days/past 2 weeks: 18.1 ng/ml; $P = 0.14$). Similarly, environmental measures of SHS exposures were not associated with increased symptom or decreased activity days.

Discussion

In this select population of inner-city children with asthma living with a smoker, fine particulate matter and air nicotine concentrations were highly correlated with urine cotinine concentrations. Additionally, children with a greater number of symptom days had the highest urine cotinine concentrations. Direct environmental measures of SHS, however, had minimal, if any, relationships with asthma symptom outcomes. Taken together, these findings lend support for the continued use of urine cotinine to assess SHS exposure in children.

Although direct environmental measures of SHS are attractive because they are not influenced by individual variability in the metabolism of nicotine as urine cotinine is, these measures of SHS had no correlation with asthma symptoms in our study population. This may be due to the fact that urine cotinine concentrations are explicitly linked to SHS exposure in the child independent of the site of SHS exposure. This is important given that exposure for school age children may occur in a variety of community settings of the child's social network including parents, child care providers, extended family and neighbors.

The predominant household smoker was the child's caregiver in over two-thirds and the mother in over half of the families, comparable to a hospital-based sample of low income children with asthma, [37] yet significantly higher than the 31–39% detected in low-income, predominantly African American families [10, 12, 38]. Our data indicate over a two-fold increase in child urine cotinine concentrations when the caregiver was the household smoker. This confirms that caregiver smoking contributes more to the child's SHS exposure than other household smokers [5, 6, 10, 38]. Caregiver smoking results in close proximity between the smoker and child and prolonged exposure, especially since children spend most time indoors. Moreover, most caregivers reported smoking in their own bedroom, likely in close proximity to the child.

We observed levels of indoor $PM_{2.5}$ that are on average twice as high as the EPA standard for outdoor $PM_{2.5}$ concentrations and are significantly higher than the 13.3 μg/ml $PM_{2.5}$ levels reported in school age children with asthma residing in nonsmoking homes [39]. Although mean air nicotine concentrations of all homes were elevated, air nicotine concentrations were not highly correlated with the number of smokers in the household. Caregiver estimation of child SHS exposure based only on total household number of cigarettes smoked resulted in some misclassification of low SHS exposure in our population. A subgroup of children with high air nicotine concentrations (>1 $\mu g/m^3$) were associated with caregiver reports of low SHS exposure, i.e. less than ½ pack of cigarettes smoked by all household smokers, suggesting the low reliability of caregiver report of SHS exposure and the underestimation of the harm of SHS to their child [40, 41]. Although the use of targeted questions about household smoking to identify significant SHS exposure for children with asthma has been proposed, [10, 35] our data indicate that caregiver report could not be relied upon to accurately distinguish children with high versus low SHS exposure.

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On a local level, pediatric health care providers are in a key position to consistently influence child SHS exposure when parents are willing to disclose their own smoking behavior and quit attempts [28]. Caregivers in our study had a median of greater than two quit attempts over the past year. Nationally 70% of smokers report a desire to quit each year, yet only 34% attempt to quit and only 10% succeed and remain tobacco-free for a year [42]. Moreover, quit rates are lowest in less educated adults [5] represented by over one-third of caregivers in this study population. Higher number of quit attempts are associated with increased likelihood of successful smoking cessation [43, 44]. Therefore, opportunities to address caregiver smoking cessation are numerous with the frequency of well child and asthma care visits that increase the likelihood of a visit coinciding with high caregiver readiness to quit smoking [28, 44].

More importantly, stronger policies are needed for restricting SHS exposure in private spaces for children. Although US tobacco control policies restrict smoking in public spaces including restaurants, workplaces, schools and hospitals, [45] children are not protected from SHS in environments where they spend the majority of their time. A child's social network is often limited to their homes, extended family and neighbor homes and child care settings [8, 46]. Voluntary home smoking bans by parents may not offer complete protection to the child even if neighbors or extended family members smoke outside of the home. Partial home smoking bans have been reported to lower SHS exposure, yet not as significantly as a total home smoking ban [37]. Promoting a larger ecological change in culture that includes community-wide change in restricting smoking in private residences may be more effective in reducing SHS exposure for children than ongoing smoking cessation services for parents [8] as reported by caregivers in this study.

There are potential limitations to this study to consider. Urine cotinine levels reflect SHS exposure at one point in time and may not reflect long term exposure. However, caregiver report of SHS exposure among children with asthma remained relatively stable over time based on cotinine levels up to 9 months later [11]. Factors such as size, layout, air volume and ventilation of the household that influence indoor air nicotine and particulate matter concentrations were not measured in this study and may have influenced our results. Finally, we purposely enrolled high risk children with asthma residing in homes with a smoker to detect a difference in our intervention groups and may have enrolled children with higher SHS exposure levels not applicable to non-urban children who do not live with a smoker.

Conclusion

Urban children with asthma and residing with a smoker were exposed to high levels of SHS that appears to occur primarily by their primary caregiver. Higher levels of SHS exposure were associated with children reporting 5 or more days of asthma symptoms over the past 2 weeks, a definition that is consistent with uncontrolled asthma. Caregiver estimation of child SHS exposure resulted in some misclassification of low SHS exposure in this population. Therefore, cotinine measurement continues to be the gold standard for SHS exposure assessment among children with asthma. Future advocacy needs to address stronger policies to reduce SHS exposure in private spaces for children with a goal of eliminating SHS exposure.

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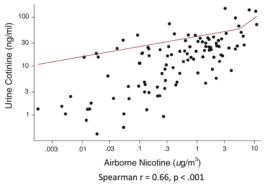

Fig. 1.
Comparison of air nicotine (μg/m^3) by urine cotinine (ng/ml) concentrations

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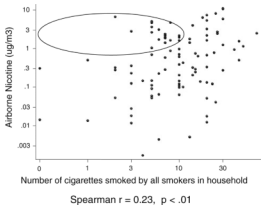

Fig. 2.

Comparison of the number of cigarettes smoked in household/day by all smokers by Air Nicotine (AN) concentration (μg/m^3). Subjects identified in circle (*n* = 21) have AN concentrations >1.0 μg/m^3 with low SHS exposure based on caregiver report of total number of household cigarettes smoked (less than 1/2 pack per day)

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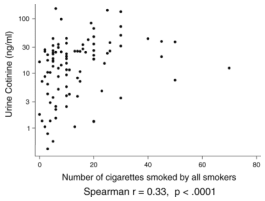

Fig. 3.

Comparison of the number of cigarettes smoked in household/day by all smokers by Urine Cotinine concentrations (ng/ml)

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Table 1

Baseline sociodemographic and health characteristics of participants (n = 126)

Sociodemographic characteristic	*N* (%)
Child's health insurance	
Medicaid (public insurance)	113 (90)
Private	11 (9)
Other	2 (1)
Caregiver	
Birth mother	102 (81)
Birth father	5 (4)
Grandmother/legal guardian	19 (15)
Caregiver marital status	
Single	90 (72)
Married	27 (22)
Separated/divorced/widowed	8 (6)
Caregiver educational status	
<High school degree	44 (35)
High school graduate/GED	64 (51)
Some college/vocational school or college grad	18 (14)
Number household members (Median, IQR)	3 (2–4)
Health characteristics	
Asthma severity	
Intermittent	26 (20)
Mild persistent	21 (17)
Moderate persistent	40 (32)
Severe persistent	39 (31)
Asthma control	
Controlled asthma (albuterol use<4 days in past 2 weeks)	47 (38)
Uncontrolled asthma (albuterol use >4 days in past 2 weeks)	76 (62)
Number of symptom days in past 2 weeks (N = 106)	
0–4 days	86 (81)
5 or more days	20 (19)
Asthma medication use	*N* (%)
Short acting beta agonist use in past 2 weeks	
Yes	96 (76)
Oral corticosteroid medication ever used in past 2 weeks	
Yes	14 (11)
Inhaled corticosteroid medication use in past 2 weeks?	
Yes	65 (52)
Leukotriene modifier use in past 2 weeks	
Yes	43 (34)

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Table 2

Baseline cigarette household smoking characteristics *N* = 126

Characteristic	*N* (%)
Caregiver[a] is smoker in the home	86 (68)
Mother is smoker in the home	66 (52)
Number of Smokers in the home	
1	68 (54)
2	47 (38)
3 or more	10 (8)
Other smokers in the home (YES)	89 (71)
Husband/significant other	40 (45)
Sibling of child	7 (8)
Other relative	46 (52)
Regular visitor or border	15 (13)
Mean hours per day child spent outdoors across all seasons	3.68 (SD 1.8)
Caregiver number cigarettes smoked/day (Median, IQR)	7 (5–10)
Caregiver number of lifetime quit attempts (Median, IQR)	2.5 (2–4)
Total number of cigarettes smoked by all household smokers (Median, IQR)	10 (5–20)
Caregiver frequency of smoking in home *N* = 86	
Always (daily)	39 (45)
Sometimes	29 (34)
Rarely	11 (13)
None	7 (8)
Caregiver smoking location *N* = 86 (Multiple responses reported)	
Caregiver's bedroom	49 (57)
Living room or TV room	23 (27)
Kitchen	26 (30)
Dining room	14 (16)
Child's Bedroom	1 (1)
Other rooms	19 (22)
Child exposed to SHS in car	
Yes	34 (27)
Caregiver make changes in household smoking rules	
Yes	110 (88)
Air conditioner in home	
Yes	72 (57)

[a]Caregiver defined as parent or legal guardian. The mother is the caregiver in most (81%) families and the caregiver is the father, grandmother or other legal guardian in the remaining 19% of families. Most caregivers who smoked were mothers (66/86, 77%)

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Table 3

Baseline mean (SD) $PM_{2.5}$, air nicotine and urine cotinine concentrations by household smoking characteristics

Household smoking characteristic	$PM_{2.5}$ (μg/m^3)	Ari necotinine (μg/ml^3)	Urine cotinine (ng/ml)
Number of household smokers			
1 smoker	33.96 (25.4)[a]	0.99 (1.5)[a]	16.02 (21.6)[b]
2 or more smokers	44.71 (30.5)	1.98 (2.7)	30.10 (29.5)
Caregiver is a smoker			
No	28.88 (22.4)[a]	0.71 (1.5)[a]	12.45 (15.5)[a]
Yes	44.16 (29.8)	1.79 (2.3)	27.39 (28.9)
Child exposed to SHS in car			
No	-	-	22.59 (28.8)
Yes	-	-	23.78 (19.3)
Home air conditioner			
No	38.39 (26.7)	1.20 (1.7)	23.78 (19.3)
Yes	40.10 (29.89)	1.60 (2.4)	23.18 (30.4)

[a] $P \leq 0.05$

[b] $P \leq 0.01$