

Comparison of Secondhand Smoke Exposure in Minority and Nonminority Children With Asthma

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Objective: This study determined if secondhand smoke (SHS) exposure is related to asthma-related functional morbidity by examining racial/ethnic differences in non-Latino White (NLW), African American, and Latino families and whether racial/ethnic SHS exposure differences across families persist when accounting for smoking factors. **Methods:** Participants were 305 caregiver smokers of children with asthma. Two passive dosimeters measured secondhand smoke: one in the home and one worn by the child. **Results:** Higher SHS exposure was related to greater asthma-related functional morbidity. African Americans had higher levels of home SHS exposure than did Latinos ($p = .003$) or NLWs ($p = .021$). SHS exposure as assessed by the child-worn dosimeter did not differ across race/ethnicity. African American families were less likely to report a household smoking ban (46.4%) compared to Latinos (79.2%) and NLWs (67.9%; $p < .05$). African Americans were less likely to report having two or more smokers in the home (37.2%) compared to NLWs (53.6%; $p < .05$). NLWs reported the highest number of cigarettes smoked daily ($Mdn = 15.00$) compared to Latinos ($Mdn = 10.00$; $p = .001$) and African Americans ($Mdn = 10.00$; $p < .001$). SHS home exposure levels were regressed on race/ethnicity and relevant covariates. Household smoking ban ($p < .001$) and only one smoker in the home ($p = .005$) were associated with lower levels of SHS in the home; race/ethnicity was not significant. **Conclusions:** Differences in SHS exposure across race/ethnicity exist among children with asthma, possibly due to differential presence of a household smoking ban and number of smokers in the home.

Keywords: asthma, smoking, secondhand smoke, children

Asthma is the most common childhood chronic illness, affecting approximately 9.6% of children and adolescents in the United States (Akinbami, Moorman, & Liu, 2011). Children with asthma

are at increased risk for experiencing significant morbidity due to asthma exacerbations, including missed school days, emergency department visits, and hospitalizations (Akinbami et al., 2011). African American and Latino families are disproportionately affected by asthma and are more likely to have greater asthma morbidity than non-Latino White (NLW) families (Akinbami & Schoendorf, 2002; Canino, McQuaid, & Rand, 2009; Lara, Akinbami, Flores, & Morgenstern, 2006). In particular, African American and Latino children are at increased risk for a greater number of missed school days, more frequent asthma emergency department visits and hospitalizations, and more frequent asthma exacerbations than NLWs (Gupta, Carrión-Carire, & Weiss, 2006; Haselkorn, Lee, Mink, Weiss, & the Tenor Study Group, 2008; Kruse, Deshpande, & Vezina, 2007).

Canino and colleagues (2009) propose a conceptual model for understanding racial/ethnic disparities in asthma. This model posits that health disparities are multicausal and involve several interfacing components between the health care system and individual/community system. The health care system includes health policies (e.g., reimbursement levels), operation of the health system (e.g., cultural sensitivity, use of evidence-based care), and provider/clinician factors (e.g., stereotyping, clinician's training). Alternatively, the individual/community system is posited to in-

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clude the social/environmental context (e.g., indoor/outdoor allergens, poverty) and the individual/family context (e.g., genetics, beliefs, health literacy). According to Canino and colleagues (2009), these two systems interact with each other to affect the process of care (e.g., quality of care) and subsequent asthma treatment outcomes. Secondhand smoke (SHS) exposure is a commonly encountered trigger in a child's environmental context, according to the Canino et al. (2009) model of health disparities in asthma.

Approximately 53.2% of children with asthma between ages 4 and 19 years are exposed to SHS, and 17.6% are exposed in the home (Kit, Simon, Brody, & Akinbami, 2013). Children with asthma who are exposed to SHS show increased respiratory symptoms and are slower to recover after hospitalization for an acute asthma exacerbation (Abulhosn, Morray, Llewellyn, & Redding, 1997; Chilmonczyk et al., 1993). Despite this, caregivers of children with asthma continue to smoke at rates similar to the general population and are not more likely to quit or smoke outside of the home after their child is diagnosed (Liem, Kozyrskyj, Benoit, & Becker, 2007). This does not appear to be related to an information deficit, because many parents report knowing that SHS exposure is related to an asthma diagnosis (Mahabee-Gittens, 2002). These findings have led researchers to target caregivers of children with asthma who smoke to reduce SHS exposure (Borrelli, McQuaid, Novak, Hammond, & Becker, 2010; Hovell et al., 1994; Hovell et al., 2002; Wahlgren, Hovell, Meltzer, Hofstetter, & Zakarian, 1997).

There is evidence for racial/ethnic differences in SHS exposure. In general population studies, compared with children of NLW smokers, children of African American smokers consistently have higher levels of SHS exposure as measured by cotinine levels, a metabolite of nicotine that reflects exposure to nicotine from all sources of tobacco smoke (Knight, Eliopoulos, Klein, Greenwald, & Koren, 1996; Marano, Schober, Brody, & Zhang, 2009). Alternatively, children of Latino smokers may have lower cotinine levels than both NLW and African American children (Marano et al., 2009). Interpretation of these findings, however, is complicated by the fact that racial/ethnic differences in cotinine levels are not consistently related to objective measures of air quality in the home environment (Wilson, Kahn, Khoury, & Lanphear, 2004, 2007). Furthermore, metabolism of nicotine, which may be affected by a range of factors (e.g., age), is metabolized differently by individuals of different race or ethnicity. Despite their caregivers smoking fewer cigarettes, African American children can have higher cotinine levels than their NLW peers (Knight et al., 1996). Additional studies have found that the half-life of cotinine is greater in African Americans compared to NLWs (Pérez-Stable, Herrera, Jacob, & Benowitz, 1998). Because SHS affects asthma symptoms as an inhaled irritant, objective measures of airborne SHS can be useful when investigating the interaction of SHS and race/ethnicity among children with asthma.

Only a small number of studies to date have examined racial/ethnic differences in objectively measured SHS exposure among children with asthma. In two studies, Wilson and colleagues (2004, 2007) found much higher levels of cotinine among African American children with asthma than their NLW counterparts. Racial/ethnic differences in airborne SHS in these studies were less clear. One study reported that homes of NLW families had higher levels of particulate matter < 5 μm in diameter ($\text{PM}_{2.5}$, an indicator of

SHS), whereas the other study indicated that there were no racial/ethnic differences in SHS exposure when assessed by passive dosimeters (Wilson et al., 2007). In addition to these equivocal findings, the studies included households where children were exposed to at least 5 or more cigarettes per day in or around the home, assessed only one context for SHS exposure (i.e., the home), and did not include Latinos, a group known to potentially be at higher risk for asthma morbidity (Canino et al., 2009). Furthermore, given the primary emphasis of these studies on racial/ethnic differences in children's cotinine levels, the previous studies did not examine the how several commonly assessed smoking factors (e.g., household ban status) were related to potential differences in airborne SHS.

Current Study

The current study seeks to determine if SHS exposure is related to asthma-related functional morbidity by examining racial/ethnic differences in a large sample of NLW, African American, and Latino families. Two indicators of objectively measured SHS, as measured by passive dosimeters, were used: one placed in the home (room where the child spends the most time) and one worn by the child. The current study also aimed to examine if potential racial/ethnic differences in SHS exposure across NLW, African American, and Latino families persisted when accounting for several common smoking factors (e.g., household ban status, number of smokers in household). Based on previous literature (Abulhosn et al., 1997; Chilmonczyk et al., 1993; Knight et al., 1996; Marano et al., 2009), it was hypothesized that greater SHS exposure would be related to greater asthma-related functional morbidity and that African American families would have the greatest SHS exposure compared to Latino and NLW families. This study adds to previous work by (a) including a large group of Latino families for comparison and (b) objectively assessing SHS exposure in the home and personal exposure to the child specifically via dosimeters, which is not affected by potential differences in metabolism across racial/ethnic groups.

Method

Participants

Participants included 305 regular smokers (53 Latino, 84 African American, and 168 NLW; Table 1) who were caregivers of children aged 17 years and younger ($M = 5.10$, $SD = 4.51$) with asthma living in Rhode Island and southern Massachusetts who were part of a larger smoking cessation induction study (Borrelli et al., 2010) that included asthma education and motivational interviewing for smoking cessation. Potential participants were told that to be part of the study, they needed to be willing to accept asthma education visits in their home and discuss their smoking. However, they did not have to want to quit smoking to be eligible for inclusion. Once enrolled, those who elected to quit within 30 days received 8 weeks of a transdermal nicotine patch treatment at no cost. This study received approval from our institutional review board.

The present study uses only baseline data from this parent study. The sample includes only those participants whose child had asthma, attended two home visits that occurred prior to random-

Table 1
Asthma, Demographic, and Room Characteristics Across Race/Ethnicity

	Total sample	Latino	African American	Non-Latino White	χ^2	<i>p</i>
Asthma-related functional morbidity score, median (IQR)	1.45 (1.50)	1.50 (1.43)	1.50 (1.55)	1.33 (1.33)	2.80	.247
Caregiver age in years, median (IQR)	32 (13)	30 (11)	36.5 (16)	31 (13)	10.25	.006
Child age in years, median (IQR)	4 (7)	4 (7)	4 (8)	3.5 (7)	2.30	.317
Child gender, <i>n</i> (%)						
Male	181 (59.3)	35 (66.0)	51 (60.7)	95 (56.5)	1.59	.451
Female	124 (40.7)	18 (34.0)	33 (39.3)	73 (43.5)		
Caregiver gender, <i>n</i> (%)						
Male	62 (20.3)	14 (26.4)	17 (20.2)	31 (18.5)	1.58	.454
Female	243 (79.7)	39 (73.6)	67 (79.8)	137 (81.5)		
% on public assistance, <i>n</i> (%)						
No	90 (29.5)	14 (26.9)	23 (27.7)	53 (31.9)	.74	.692
Yes	211 (70.1)	38 (73.1)	60 (72.3)	113 (68.1)		
Adults living in home, <i>n</i> (%)	2 (2)	2 (2)	2 (1)	2 (1)	2.81	.245
Dosimeter room square footage, median (IQR)	197,024 (115, 148)	176,715 (79, 830)	200,200 (107, 447)	203,094 (114, 278)	3.82	.148

Note. Comparisons made across race/ethnicity. Kruskal-Wallis tests were used to compare median values for continuous variables. Chi-square analyses were used to compare categorical variables. There are four missing values for the percentage on public assistance variable. IQR = interquartile range.

ization, and were subsequently randomized to a study treatment arm. In the parent study, of those who were eligible to participate, 13.7% (*n* = 118) refused. Approximately 79% of the remaining caregivers of children with asthma who were enrolled in the parent study completed both study visits and were randomized. Participants whose child had asthma were eligible for the parent study if their child experienced an asthma exacerbation requiring an emergency department or urgent care visit or a hospitalization (within the past 2 months) and were recruited primarily from emergency departments and physician referrals. Participants were eligible if they were (a) a current smoker (smoked ≥ 3 cigarettes per day and more than 100 cigarettes in their lifetime), (b) a primary caregiver of a child with asthma, (c) 18 years of age or older, (d) not currently or planning to become pregnant, (e) fluent in English, (f) reachable by telephone, and (g) not enrolled in another smoking cessation program or using nicotine replacement or medication to help them quit smoking.

Study Design

A research assistant conducted brief phone screenings of potential participants to determine eligibility. Potential participants who were eligible and willing to participate received a home visit from a research assistant to obtain informed consent and place two passive air nicotine monitors (dosimeters): one was placed in the room in which the child spent the most time (home monitor) and a second one was worn by the child (child monitor) or, for children aged 3 years and younger, placed in close proximity to the child (e.g., diaper bag). Participants were told that the dosimeters measured "air quality." Monitors were retrieved from participants' homes 7–10 days after initial placement, and participants then completed a self-report questionnaire, for which they received \$20.00. Participants were given an additional \$5.00 for returning the passive nicotine monitors in good condition.

Measures

Demographics. The age and gender of the caregiver and the child were assessed, along with race/ethnicity and income indicators, including receipt of public assistance.

Asthma functional severity. The Asthma Functional Severity Scale (Rosier et al., 1994), which assessed the degree of functional impairment that asthma imposes on a child's daily life, was completed by caregivers. This scale assesses components of child asthma morbidity, including frequency and intensity of symptoms, interference of asthma with daily activities, and frequency of symptoms between asthma episodes. Total asthma-related functional impairment is calculated by computing a mean score across all completed items, with higher scores reflecting greater impairment.

Smoking-related variables. The number of smokers living in the home (dichotomized as one smoker vs. two or more smokers), number of cigarettes smoked per day by the caregiver during the last 7-day period, and household ban status were assessed via caregiver self-report (Borrelli, Hayes, Gregor, Lee, & McQuaid, 2011; Borrelli, McQuaid, Wagener, & Hammond, 2014). Participants reporting not having a smoking ban in place were coded as "0." Alternatively, participants who reported having a total household smoking ban in place (i.e., a house rule that no one smokes in the home) were coded as "1."

SHS and room characteristics. The average weekly SHS exposure was measured using a Hammond sampler, a passive nicotine monitor (i.e., dosimeter) that samples nicotine from the air as a tracer for SHS (Hammond, Leaderer, Roche, & Schenker, 1987). Nicotine collected in the monitors was analyzed using a previously detailed method (Hammond & Leaderer, 1987) on an HP Agilent 7890A gas chromatograph with a nitrogen-selective detector and a 15-m HP5 0.32-mm, 0.25- μ m column. The laboratory limit of detection is 0.005 μ g per sample, and field samples are corrected for the values found in the field blanks (zero in this study). The effective sampling rate is 24 ml/min, so that the concentration limit of detection for a sample collected for 1 week is 0.02 μ g per cubic meter; longer sampling times have lower limits of detection.

Field blank samples were collected and analyzed throughout the study; the laboratory was blind to the identity of these samples. Laboratory blank filters were analyzed on each analysis day. All blank filters were less than detectable. Nicotine concentration was assessed by two dosimeters. One dosimeter was placed by research

staff in the room where the child spends the majority of his or her time and away from heat sources (e.g., radiators). The other dosimeter was worn by the child or, for children aged 3 years or younger (48.5% of the sample), placed by research staff on an item reported to be in close proximity to the child. Parents were given oral and written instructions on the proper use of child-worn dosimeters. These monitors have been tested in an environmental chamber (Hammond & Leaderer, 1987) and in homes (Leaderer & Hammond, 1991) and have been validated in an intercomparison study demonstrating accurate nicotine detection (Caka et al., 1990); laboratory extraction methods have been previously published (Hammond & Leaderer, 1987). This method was used successfully in a previous study with smoking caregivers of children with asthma (Borrelli et al., 2014). All home monitors (100%) and 98.7% of the child monitors were collected by study personnel and usable for data extraction. Three child monitors were lost and one was damaged. Cubic room volume (in feet) of where the home dosimeter was placed was calculated by multiplying the length, width, and height of the room. Home, $\chi^2(3) = 3.98, p = .264$, and child, $\chi^2(3) = 6.63, p = .085$, nicotine concentrations did not differ across seasons of the year.

Overview of Analyses

Home and child nicotine concentrations were positively skewed (home skewness = 3.98; child skewness = 4.01). Nonparametric tests were used for all primary analyses. First, correlations were conducted to determine if home and child nicotine concentrations were associated with a child's asthma-related functional impairment. Next, the proportion of detectable SHS exposure (airborne nicotine concentrations $> 0.02 \mu\text{g}/\text{m}^3$), as assessed by both home and child monitors, was examined across race/ethnicity. Kruskal-Wallis tests were then conducted to examine whether home and child nicotine concentrations differed across Latino, African American, and NLW groups. Planned pairwise comparisons using Mann-Whitney U tests were conducted to assess significant differences in home or child nicotine concentrations across family ethnicity. Second, smoking variables of interest (i.e., number of cigarettes caregivers smoked, number of smokers in the home, and household smoking ban status) were compared across Latino, African American, and NLW groups through chi-square or analysis of variance tests.

Analyses were performed using SPSS 21 and Stata 12 software. Potential covariates listed in Table 1, such as demographic variables (e.g., caregiver age, number of adults living in the home) and room characteristics (cubic feet of room where the dosimeter was placed), were compared across Latino, African American, and NLW families using Kruskal-Wallis and chi-square tests. Variables that differed significantly between groups were retained as covariates in quantile regression analyses. Multivariate quantile regression analyses that controlled for relevant demographic covariates were conducted to determine if potential group differences in home or child nicotine concentrations were accounted for by differences in smoking behavior (i.e., number of cigarettes caregivers smoked, number of smokers in the home, and household smoking ban status). Quantile regression was used to model the median of the outcome variables because it is a more appropriate measure of central tendency when data are skewed (Koenker & Bassett, 1978). Family ethnicity was dummy coded in the quantile

regression analyses, with Latino families serving as the reference group. Smoking variables were also examined across Latino, African American, and NLW groups through chi-square or Kruskal-Wallis analyses. Planned comparisons were conducted to determine pairwise differences between groups. Due to missing data, sample sizes differed slightly across analyses.

Results

SHS Exposure

Regarding the home monitors, detectable levels of SHS (airborne nicotine concentrations $> 0.02 \mu\text{g}/\text{m}^3$) were found in the homes of 92.3% of NLW families, 95.2% of African American families, and 90.6% of Latino families, with no differences between groups, $\chi^2(2) = 1.21, p = .545$. High levels of detectable SHS were also revealed by analysis of the child monitors (81.9% of NLWs, 85.2% of African Americans, and 75.5% of Latinos), with no differences between groups, $\chi^2(2) = 2.04, p = .361$.

SHS Exposure and Asthma-Related Functional Impairment

Greater reported asthma-related functional morbidity was correlated with increased home nicotine concentrations, $r_s(296) = .19, p = .001$, and child nicotine concentrations, $r_s(296) = .22, p < .001$. Asthma-related functional morbidity did not differ across Latino ($Mdn = 1.50$), African American ($Mdn = 1.50$), and NLW ($Mdn = 1.33$) families, $\chi^2(2) = 2.80, p = .247$.

Differences in SHS Exposure Across Race/Ethnicity

When examining discrete nicotine concentration, Kruskal-Wallis analyses revealed that home nicotine concentrations were different across Latino ($Mdn = .27 \mu\text{g}/\text{m}^3$), African American ($Mdn = .79 \mu\text{g}/\text{m}^3$), and NLW ($Mdn = .35 \mu\text{g}/\text{m}^3$) families, $\chi^2(2) = 9.20, p = .01$. Planned pairwise comparisons revealed that African American families had higher home nicotine concentrations than did either Latino ($p = .003$) or NLW families ($p = .021$; Table 2). Home nicotine concentrations did not differ between Latino and NLW families ($p = .298$). With regard to the samplers worn by the child, planned pairwise comparisons revealed no significant differences across Latino ($Mdn = .15 \mu\text{g}/\text{m}^3$), African American ($Mdn = .35 \mu\text{g}/\text{m}^3$), and NLW ($Mdn = .30 \mu\text{g}/\text{m}^3$) families, $\chi^2(2) = 4.61, p = .10$.

Smoking-Related Variables and SHS Exposure

Household smoking ban status differed among Latino, African American, and NLW groups, $\chi^2(2) = 17.67, p = .008$. Specifically, more Latino (79.2%) and NLW families (67.9%) reported having a smoking ban than did African American families (46.4%; $ps < .05$). The rate of multiple smoker homes was also different across groups, $\chi^2(2) = 6.29, p = .043$. Planned comparisons revealed that African American families were less likely to report having two or more smokers in the home (37.2%) compared to NLW families (53.6%; $p < .05$). There were no statistically significant differences between the proportion of Latino families

Table 2
Median Home and Child Nicotine Concentration Levels by Race/Ethnicity

Nicotine concentration ($\mu\text{g}/\text{m}^3$)	Latino ^a	African American ^b	Non-Latino White ^c	χ^2	<i>p</i>	Planned comparisons
Home	.27	.79	.35	9.20	.010	b > a, c
Child	.15	.35	.30	4.61	.100	a = b = c

Note. Kruskal-Wallis tests were used to compare median values. Mann-Whitney *U* tests were used to test all pairwise planned comparisons across race/ethnicity. Superscripts in planned comparisons refer to race/ethnicity groups.

who reported having two or more smokers in the home (41.3%) compared to African American and NLW families.

Number of cigarettes smoked per day differed across groups, $\chi^2(2) = 25.40$, $p < .001$. NLW caregivers reported the highest number of cigarettes smoked per day ($Mdn = 15.00$) compared to Latino ($Mdn = 10.00$; $p = .001$) and African American ($Mdn = 10.00$; $p < .001$) families. There were no differences in number of cigarettes smoked per day between African American and Latino families (see Table 3). Smoking variables were not found to be highly intercorrelated ($r_s < .18$). Therefore, analyses proceeded with a quantile regression where median home nicotine concentrations were regressed on variables that exhibited differences between groups (see Table 1) and smoking variables of interest. Only household ban status ($b = -.87$, $SE = .19$, $p < .001$) and number of smokers in the home ($b = .50$, $SE = .18$, $p = .005$) were associated with home nicotine concentrations. Specifically, having a household ban and only one smoker in the home (i.e., the caregiver) were independently associated with lower home nicotine concentrations. Race/ethnicity, caregiver age, and number of cigarettes caregivers smoked were unrelated to home nicotine concentrations (see Table 4).

Discussion

Consistent with hypotheses, higher SHS exposure was related to greater asthma-related functional morbidity for children. These results fit well with a large body of previous research indicating the detrimental effects of SHS exposure to child health (U.S. Department of Health & Human Services, 2006), especially among those with asthma or other respiratory illnesses (Abulhosn et al., 1997; Chiltonczyk et al., 1993).

Also, as hypothesized, the results indicated that African American families had higher home nicotine concentrations compared to Latino and NLW families. Home nicotine concentrations did not differ between Latino and NLW families. Differing rates of exposure to home nicotine concentrations among African Americans

and Latinos in our sample, two racial/ethnic groups known to be at risk for poor asthma outcomes, may be linked to institution of a household smoking ban. African American families were less likely to have a household smoking ban in place compared to Latino and NLW families. Household smoking bans have been shown to be related to lower air nicotine and urinary cotinine levels in children with asthma (Berman et al., 2003; Wakefield et al., 2000). Given the well-established link of SHS exposure and asthma morbidity (Chiltonczyk et al., 1993), further examination of reasons for the absence of smoking bans is needed to enable better promotion of household smoking bans and reduction of SHS exposure for children at risk. This may be particularly important for African American caregivers of children with asthma. As related to the Canino et al. (2009) health disparities model, these data may suggest that SHS exposure as an environmental trigger plays a larger role for African Americans than Latinos. Although not assessed in the current study, health disparities experienced by Latino families with asthma may be due to other sources such as difficulties in accessing health care (e.g., insurance difficulties) or lower health literacy, among other factors (Haselkorn et al., 2008) outlined in the Canino et al. (2009) model.

The differing pattern of racial/ethnic differences in SHS exposure found in the current study should be considered in light of the findings reported by Wilson and colleagues (2004, 2007). Counter to Wilson and colleagues (2004), who reported higher levels of $\text{PM}_{2.5}$ in homes of NLW families and no evidence of racial/ethnic differences between African American and NLW families in SHS exposure when assessed by passive dosimeters (Wilson et al., 2007), African American families in the current study had the highest home nicotine concentrations compared to their NLW and Latino counterparts. One explanation for these discrepancies is that an eligibility criterion for participants in Wilson et al. (2004, 2007) was that they smoked five or more cigarettes per day in or around their home and excluded participants with a household smoking ban. The eligibility criterion for the current study was only that

Table 3
Smoking Variables by Race/Ethnicity

	Latino ^a	African American ^b	Non-Latino White ^c	Test statistic	<i>p</i>	Planned comparisons
Household ban status, <i>n</i> (%) yes	42 (79.2)	39 (46.4)	114 (67.9)	17.67	<.001	a, c > b
2+ smokers in home, <i>n</i> (%)	19 (41.3)	29 (37.2)	82 (53.6)	6.29	.043	c > a; a = b; a = c
Number of cigarettes per day, median (IQR; range)	10 (12; 46.50)	10 (8.5; 34.50)	15 (10; 97.00)	25.40	<.001	a, b < c

Note. Chi-square analyses were used to compare categorical variables, and Kruskal-Wallis was used to compare continuous variables. All pairwise planned comparisons were examined across race/ethnicity. Superscripts in planned comparisons refer to race/ethnicity groups. IQR = interquartile range.

Table 4
Quantile Regression Analysis Predicting Home Nicotine Concentration Levels

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>p</i>
Caregiver age in years	.00	.01	0.24	.813
Number of cigarettes per day	.01	.01	0.65	.513
Household ban status	-.87	.19	-4.49	<.001
Number of smokers in home	.50	.18	2.85	.005
Non-Latino White	-.02	.24	-0.07	.946
African American	.19	.28	0.67	.502
Latino	.80	.44	1.83	.068

Note. Household ban status is a dichotomous variable (0 = no ban; 1 = ban in place). Number of smokers in home is a dichotomous variable (0 = 1 smoker in home; 1 = 2 or more smokers in home). Race/ethnicity was dummy coded with Latinos serving as the reference group.

adults smoked three or more cigarettes per day total, regardless of location. Thus, participants in Wilson et al. were likely to be more frequent or heavier in-home smokers. This likely also explains the fact that the SHS exposure reported by Wilson and colleagues (2007) was two to four times higher than in the current study.

There were no statistically significant differences across race/ethnicity in SHS exposure as assessed by child monitors. Our results suggest that a sizable proportion of children is being exposed to detectable levels of SHS. Although there were no differences across race/ethnicity, it is notable that SHS exposure as assessed by child monitors was associated with asthma-related functional morbidity. These data can be interpreted to suggest that reducing child exposure to SHS outside the home should remain a priority (Dove, Dockery, & Connolly, 2011).

The current study had several notable strengths, including objectively assessing SHS exposure via passive dosimeters placed in the home and worn by the child and assessing differences in SHS exposure and smoking-related variables across African American, Latino, and NLW families. Furthermore, very high rates of home (100%) and child monitors (98.7%) were collected by study personnel and usable for data extraction. However, the current study should also be considered in light of several limitations. Data are correlational and cross-sectional in nature, which limits causal inferences. Smoking variables, including smoking ban status, number of cigarettes smoked per day, and number of smokers in the home, are based on self-report data and may reflect inadequate recall in reporting smoking-related data. Although instructions regarding the placement of the child-worn dosimeters were given to families, we do not have objective evidence that child dosimeters were used or worn by children exactly as requested. Furthermore, level of SHS exposure captured by dosimeters may not reflect a child's total exposure to SHS.

Findings from the current study should be considered preliminary and provide a beginning model for future studies that are able to concurrently examine different sources of asthma disparities. Future research with larger sample sizes of minority families that incorporates children's cotinine levels is needed to replicate and expand the current findings. Although there are some questions regarding differences in metabolism and half-life of cotinine levels between African American and NLW youth (Knight et al., 1996; Pérez-Stable et al., 1998), the inclusion of children's cotinine levels would allow examination of potential race/ethnicity differ-

ences in total nicotine exposure and avoid previously noted concerns about child adherence to wearing a passive dosimeter. More detailed assessment of smoking ban status is also needed. Although caregivers reported on the presence of a household smoking ban, smoking outside the home (e.g., porch) and partial household bans were not assessed. Beyond SHS exposure, future studies should incorporate measures of controller medication adherence and a broad range of asthma triggers to determine how these variables contribute to asthma-related functional impairment across NLW, African American, and Latino families.

The current study assessed only some of the numerous known factors that may be related to health disparities in asthma. Given the health disparities present among African American and Latino families with asthma (Akinbami & Schoendorf, 2002; Lara et al., 2006), future studies should aim to disentangle genetic, individual, physical environment, and community-level factors that may lead to differing rates of asthma morbidity and SHS exposure. As indicated in the Canino et al. (2009) model, other variables in the individual/family context known to be related to health disparities in asthma, such as health beliefs, health literacy, and illness management, should be examined as they relate to SHS exposure in minority families. For example, future studies could examine whether there are racial/ethnic differences in the interplay of health beliefs regarding SHS exposure, knowledge of negative effects of SHS exposure, and objectively measured child SHS exposure. Physical environment factors, including exposure to thirdhand smoke and residing in multiunit housing, should be examined as they relate to health disparities in asthma (Matt et al., 2011; Wilson, Klein, Blumkin, Gottlieb, & Winickoff, 2011). Additionally, further research could explore societal and community-level sources of disparity such as health policies (e.g., reimbursement levels), operation of the health system (e.g., cultural sensitivity, use of evidence-based care), and provider/clinician factors (e.g., stereotyping, clinician's training) and smoking behaviors among racial/ethnic minority groups with asthma.

The current study revealed differences across racial/ethnic groups not only in SHS but also in smoking-related variables known to be related to SHS. Latino families had lower home nicotine concentration than did African American families; however, Latino caregivers in our study reported smoking approximately 12 cigarettes per day, and more than 40% reportedly had two or more smokers in the home. Given that previous work has indicated that some Latino subgroups, such as Puerto Ricans, may evidence increased risk (e.g., less social support, higher depressed mood) for smoking treatment failure compared to NLW families (Borrelli et al., 2011), these data may underscore the need for smoking cessation interventions in African American and Latino families (Borrelli, 2010). Previous successful caregiver smoking cessation interventions among Latino and racial/ethnic minority families have included motivational interviewing strategies (Borrelli et al., 2010) and behavioral counseling (Hovell et al., 1994, 2000, 2002) that incorporate feedback regarding children's SHS exposure in the home. It should be noted, however, that because children with asthma can continue to be exposed to SHS in other environments outside the home (Halterman, Fagnano, Conn, & Szilagyi, 2006) and through thirdhand smoke (i.e., residual nicotine and chemicals that remain on surfaces after a cigarette is extinguished) in the home despite the presence of a smoking ban (Matt et al., 2011; Wilson et al., 2011), multiple approaches are

needed to address the impact of smoking: those at the policy level, the community level, and the individual and family health risk levels. Finally, given rates of smoking and SHS exposure levels across racial/ethnic groups, SHS exposure assessment should be conducted as part of routine care when working with families of a child diagnosed with asthma (McQuaid, Walders, & Borrelli, 2003).

References

- Abulhosn, R. S., Morray, B. H., Llewellyn, C. E., & Redding, G. J. (1997). Passive smoke exposure impairs recovery after hospitalization for acute asthma. *Archives of Pediatrics & Adolescent Medicine*, 151, 135–139. <http://dx.doi.org/10.1001/archpedi.1997.02170390025005>
- Akinbami, L. J., Moorman, J. E., & Liu, X. (2011). Asthma prevalence, health care use, and mortality: United States, 2005–2009. *National Health Statistics Reports*, 32, 1–14.
- Akinbami, L. J., & Schoendorf, K. C. (2002). Trends in childhood asthma: Prevalence, health care utilization, and mortality. *Pediatrics*, 110, 315–322. <http://dx.doi.org/10.1542/peds.110.2.315>
- Berman, B. A., Wong, G. C., Bastani, R., Hoang, T., Jones, C., Goldstein, D. R., . . . Lewis, M. A. (2003). Household smoking behavior and ETS exposure among children with asthma in low-income, minority households. *Addictive Behaviors*, 28, 111–128. [http://dx.doi.org/10.1016/S0306-4603\(01\)00221-0](http://dx.doi.org/10.1016/S0306-4603(01)00221-0)
- Borrelli, B. (2010). Smoking cessation: Next steps for special populations research and innovative treatments. *Journal of Consulting and Clinical Psychology*, 78, 1–12. <http://dx.doi.org/10.1037/a0018327>
- Borrelli, B., Hayes, R. B., Gregor, K., Lee, C. S., & McQuaid, E. L. (2011). Differences in smoking behavior and attitudes among Puerto Rican, Dominican, and non-Latino white caregivers of children with asthma. *American Journal of Health Promotion*, 25 (Suppl), S91–S95. <http://dx.doi.org/10.4278/ajhp.100624-ARB-214>
- Borrelli, B., McQuaid, E. L., Novak, S. P., Hammond, S. K., & Becker, B. (2010). Motivating Latino caregivers of children with asthma to quit smoking: A randomized trial. *Journal of Consulting and Clinical Psychology*, 78, 34–43. <http://dx.doi.org/10.1037/a0016932>
- Borrelli, B., McQuaid, E. L., Wagener, T. L., & Hammond, S. K. (2014). Children with asthma versus healthy children: Differences in secondhand smoke exposure and caregiver perceived risk. *Nicotine & Tobacco Research*, 16, 554–561. <http://dx.doi.org/10.1093/ntr/ntt180>
- Caka, F. M., Eatough, D. J., Lewis, E. A., Tang, H., Hammond, S. K., Leaderer, B. P., . . . Fasano, A. (1990). An intercomparison of sampling techniques for nicotine in indoor environments. *Environmental Science & Technology*, 24, 1196–1203. <http://dx.doi.org/10.1021/es00078a007>
- Canino, G., McQuaid, E. L., & Rand, C. S. (2009). Addressing asthma health disparities: A multilevel challenge. *The Journal of Allergy and Clinical Immunology*, 123, 1209–1217. <http://dx.doi.org/10.1016/j.jaci.2009.02.043>
- Chilmonczyk, B. A., Salmun, L. M., Megathlin, K. N., Neveux, L. M., Palomaki, G. E., Knight, G. J., . . . Haddow, J. E. (1993). Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *The New England Journal of Medicine*, 328, 1665–1669. <http://dx.doi.org/10.1056/NEJM199306103282303>
- Dove, M. S., Dockery, D. W., & Connolly, G. N. (2011). Smoke-free air laws and asthma prevalence, symptoms, and severity among nonsmoking youth. *Pediatrics*, 127, 102–109. <http://dx.doi.org/10.1542/peds.2010-1532>
- Gupta, R. S., Carrión-Carire, V., & Weiss, K. B. (2006). The widening black/white gap in asthma hospitalizations and mortality. *The Journal of Allergy and Clinical Immunology*, 117, 351–358. <http://dx.doi.org/10.1016/j.jaci.2005.11.047>
- Halterman, J. S., Fagnano, M., Conn, K. M., & Szilagyi, P. G. (2006). Do parents of urban children with persistent asthma ban smoking in their homes and cars? *Ambulatory Pediatrics*, 6, 115–119. <http://dx.doi.org/10.1016/j.ambp.2005.10.004>
- Hammond, S. K., & Leaderer, B. P. (1987). A diffusion monitor to measure exposure to passive smoking. *Environmental Science & Technology*, 21, 494–497. <http://dx.doi.org/10.1021/es00159a012>
- Hammond, S. K., Leaderer, B. P., Roche, A. C., & Schenker, M. (1987). Collection and analysis of nicotine as a marker for environmental tobacco smoke. *Atmospheric Environment*, 21, 457–462. [http://dx.doi.org/10.1016/0004-6981\(87\)90027-8](http://dx.doi.org/10.1016/0004-6981(87)90027-8)
- Haselkorn, T., Lee, J. H., Mink, D. R., Weiss, S. T., & the TENOR Study Group. (2008). Racial disparities in asthma-related health outcomes in severe or difficult-to-treat asthma. *Annals of Allergy, Asthma & Immunology*, 101, 256–263. [http://dx.doi.org/10.1016/S1081-1206\(10\)60490-5](http://dx.doi.org/10.1016/S1081-1206(10)60490-5)
- Hovell, M. F., Meltzer, S. B., Wahlgren, D. R., Matt, G. E., Hofstetter, C. R., Jones, J. A., . . . Pirkle, J. L. (2002). Asthma management and environmental tobacco smoke exposure reduction in Latino children: A controlled trial. *Pediatrics*, 110, 946–956. <http://dx.doi.org/10.1542/peds.110.5.946>
- Hovell, M. F., Meltzer, S. B., Zakarian, J. M., Wahlgren, D. R., Emerson, J. A., Hofstetter, C. R., . . . Atkins, C. J. (1994). Reduction of environmental tobacco smoke exposure among asthmatic children: A controlled trial. *Chest*, 106, 440–446. <http://dx.doi.org/10.1378/chest.106.2.440>
- Hovell, M. F., Zakarian, J. M., Matt, G. E., Hofstetter, C. R., Bernert, J. T., & Pirkle, J. (2000). Effect of counselling mothers on their children's exposure to environmental tobacco smoke: Randomised controlled trial. *British Medical Journal (Clinical Research Ed.)*, 321, 337–342. <http://dx.doi.org/10.1136/bmj.321.7257.337>
- Kit, B. K., Simon, A. E., Brody, D. J., & Akinbami, L. J. (2013). US prevalence and trends in tobacco smoke exposure among children and adolescents with asthma. *Pediatrics*, 131, 407–414. <http://dx.doi.org/10.1542/peds.2012-2328>
- Knight, J. M., Eliopoulos, C., Klein, J., Greenwald, M., & Koren, G. (1996). Passive smoking in children: Racial differences in systemic exposure to cotinine by hair and urine analysis. *Chest*, 109, 446–450. <http://dx.doi.org/10.1378/chest.109.2.446>
- Koenker, R., & Bassett, G. (1978). Regression quantiles. *Econometrica*, 46, 33–50. <http://dx.doi.org/10.2307/1913643>
- Kruse, L. K., Deshpande, S., & Vezina, M. (2007). Disparities in asthma hospitalizations among children seen in the emergency department. *Journal of Asthma*, 44, 833–837. <http://dx.doi.org/10.1080/02770900701750163>
- Lara, M., Akinbami, L., Flores, G., & Morgenstern, H. (2006). Heterogeneity of childhood asthma among Hispanic children: Puerto Rican children bear a disproportionate burden. *Pediatrics*, 117, 43–53. <http://dx.doi.org/10.1542/peds.2004-1714>
- Leaderer, B. P., & Hammond, S. K. (1991). Evaluation of vapor-phase nicotine and respirable suspended particle mass as markers for environmental tobacco smoke. *Environmental Science & Technology*, 25, 770–777. <http://dx.doi.org/10.1021/es00016a023>
- Liem, J. J., Kozyrskyj, A. L., Benoit, C. M., & Becker, A. B. (2007). Asthma is not enough: Continuation of smoking among parents with an asthmatic child. *Canadian Respiratory Journal*, 14, 349–353.
- Mahabee-Gittens, M. (2002). Smoking in parents of children with asthma and bronchiolitis in a pediatric emergency department. *Pediatric Emergency Care*, 18, 4–7.
- Marano, C., Schober, S. E., Brody, D. J., & Zhang, C. (2009). Secondhand tobacco smoke exposure among children and adolescents: United States, 2003–2006. *Pediatrics*, 124, 1299–1305. <http://dx.doi.org/10.1542/peds.2009-0880>
- Matt, G. E., Quintana, P. J., Destailats, H., Gundel, L. A., Sleiman, M., Singer, B. C., . . . Hovell, M. F. (2011). Thirdhand tobacco smoke: Emerging evidence and arguments for a multidisciplinary research agen-

- da. *Environmental Health Perspectives*, 119, 1218–1226. <http://dx.doi.org/10.1289/ehp.1103500>
- McQuaid, E. L., Walders, N., & Borrelli, B. (2003). Environmental tobacco smoke exposure in pediatric asthma: Overview and recommendations for practice. *Clinical Pediatrics*, 42, 775–787. <http://dx.doi.org/10.1177/000992280304200903>
- Pérez-Stable, E. J., Herrera, B., Jacob, P., III, & Benowitz, N. L. (1998). Nicotine metabolism and intake in Black and White smokers. *Journal of the American Medical Association*, 280, 152–156. <http://dx.doi.org/10.1001/jama.280.2.152>
- Rosier, M. J., Bishop, J., Nolan, T., Robertson, C. F., Carlin, J. B., & Phelan, P. D. (1994). Measurement of functional severity of asthma in children. *American Journal of Respiratory and Critical Care Medicine*, 149, 1434–1441. <http://dx.doi.org/10.1164/ajrccm.149.6.8004295>
- U.S. Department of Health and Human Services. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.
- Wahlgren, D. R., Hovell, M. F., Meltzer, S. B., Hofstetter, C. R., & Zakarian, J. M. (1997). Reduction of environmental tobacco smoke exposure in asthmatic children: A 2-year follow-up. *Chest*, 111, 81–88. <http://dx.doi.org/10.1378/chest.111.1.81>
- Wakefield, M., Banham, D., Martin, J., Ruffin, R., McCaul, K., & Badcock, N. (2000). Restrictions on smoking at home and urinary cotinine levels among children with asthma. *American Journal of Preventive Medicine*, 19, 188–192. [http://dx.doi.org/10.1016/S0749-3797\(00\)00197-5](http://dx.doi.org/10.1016/S0749-3797(00)00197-5)
- Wilson, K. M., Klein, J. D., Blumkin, A. K., Gottlieb, M., & Winickoff, J. P. (2011). Tobacco-smoke exposure in children who live in multiunit housing. *Pediatrics*, 127, 85–92. <http://dx.doi.org/10.1542/peds.2010-2046>
- Wilson, S. E., Kahn, R. S., Khoury, J., & Lanphear, B. P. (2004). Racial differences in exposure to environmental tobacco smoke among children. *Environmental Health Perspectives*, 113, 362–367. <http://dx.doi.org/10.1289/ehp.7379>
- Wilson, S. E., Kahn, R. S., Khoury, J., & Lanphear, B. P. (2007). The role of air nicotine in explaining racial differences in cotinine among tobacco-exposed children. *Chest*, 131, 856–862. <http://dx.doi.org/10.1378/chest.06-2123>

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