ORIGINAL INVESTIGATION

Secondhand Smoke Exposure Among Nonsmoking Pregnant Women in New York City

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Received July 24, 2013; accepted February 11, 2014

ABSTRACT

Introduction: Although secondhand smoke (SHS) exposure during pregnancy has detrimental effects on fetal health, little is known about levels of SHS in nonsmoking pregnant women. We examined disparities in SHS exposure among nonsmoking, ethnic minority pregnant women in New York City.

Methods: We used self-reported smoking and serum cotinine collected from 244 pregnant women from the Bronx who self-identified as African American, Caribbean American, or Black Hispanic to examine smoking prevalence (>3 ng/ml) and, in an adjusted logistic regression model, risk factors for SHS (≥ 0.05 ng/ml and ≤ 3 ng/ml).

Results: Although only 4.1% of women self-reported they were smokers, 10.7% had serum cotinine levels indicating they were smokers. Among the 218 nonsmokers, 46.8% had serum cotinine levels indicating SHS exposure. Women at highest risk included those with less than a high school degree (66.7%) and those who were U.S.-born Black Hispanic (63.2%) or African American (63.0%). Women with more than 12 years of education were less likely to have detectable SHS exposure than women with fewer than 12 years (adjusted odds ratio 0.39, 95% CI = 0.17, 0.91). Compared with African American U.S.-born women, those who were African American foreign-born or Caribbean American and either U.S.-born or foreign-born were less likely to have detectable SHS exposure (all $p \leq .05$).

Conclusions: Nearly half of nonsmoking pregnant women in New York City had elevated cotinine levels despite living in a city with comprehensive tobacco control policies. Health professionals need to assess sources of SHS exposure during pregnancy and promote smoke-free environments to improve maternal and fetal health.

INTRODUCTION

Despite an increasing proportion of the United States protected by smoke-free legislation (American Nonsmokers Rights Foundation, 2013) and national targets to reduce nonsmokers' exposure to secondhand smoke (SHS) (U.S. Department of Health and Human Services [USDHHS], 2011), in 2007–2008, 40% of the nonsmoking population (≥ 3 years old) had serum cotinine levels indicating SHS exposure (Centers for Disease Control and Prevention [CDC], 2010). The dramatic decline in SHS seen in past decades (Pirkle, Bernert, Caudill, Sosnoff, & Pechacek, 2006) may have stalled as studies have shown a plateau in levels over recent years (CDC, 2010; Chen, Burton, Baker, Mastey, & Mannino, 2010). Children, non-Hispanic Blacks, and more disadvantaged groups continue to have higher levels of exposure than other populations (CDC, 2010; Chen

et al., 2010; Ellis et al., 2009; USDHHS, 2006). As there are no known safe levels of SHS (USDHHS, 2006), prevention is critical.

Public health campaigns (CDC, 2013; USDHHS, 2013) and clinical guidelines (American College of Obstetricians and Gynecologists, 2011; Best, Committee on Environmental Health, Committee on Native American Child Health, & Committee on Adolescence, 2009; Fiore et al., 2008) have focused on encouraging women to quit smoking during pregnancy and reduce children's exposure to SHS because of the known harmful effects on infants' and children's health (USDHHS, 2004, 2006). An increasing body of evidence suggests that SHS during pregnancy has similar, albeit less pronounced, effects on fetal health as smoking directly. The 2006 Surgeon General report concluded that SHS exposure during pregnancy was associated with a small reduction in birth

doi:10.1093/ntr/ntu034

Advance Access publication March 18, 2014

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weight and suggestive of an increased risk for preterm delivery (USDHHS, 2006). More recent systematic reviews and meta-analyses (Nieuwenhuijsen, Dadvand, Grellier, Martinez, & Vrijheid, 2013) have demonstrated that SHS exposure during pregnancy decreases birth weight by at least 33 g (Leonardi-Bee, Smyth, Britton, & Coleman, 2008), increases the risk of low birth weight (Leonardi-Bee et al., 2008; Salmasi, Grady, Jones, & McDonald, 2010), stillbirth (Leonardi-Bee, Britton, & Venn, 2011), and congenital anomalies (Leonardi-Bee et al., 2011; Salmasi et al., 2010).

In 2003, New York City (NYC) implemented a comprehensive tobacco control program including smoke-free workplaces, restaurants, and bars; an increase in the price of cigarettes; free nicotine replacement therapy for smokers; and local antismoking advertising campaigns (Ellis et al., 2009). An evaluation one and a half years later demonstrated that while smoking rates in NYC were lower than rates nationally, 51% of nonsmoking women in NYC had elevated cotinine levels compared with 40% of nonsmoking women nationally (Ellis et al., 2009). Ellis and colleagues (2009) found groups at risk for SHS were New Yorkers who were younger, male, Asian, and had lower educational attainment and household income. However, whether similar differences in SHS exposure may be apparent among pregnant women is unknown.

Our overall aim was to examine disparities in SHS exposure measured by serum cotinine among a sample of nonsmoking low-income, ethnic minority pregnant women in NYC nearly 10 years after the implementation of comprehensive tobacco control policies.

MATERIALS AND METHODS

Sample

The sample was derived from a broader study examining the mechanisms that underlie preterm labor and preterm birth in minority women. Women were recruited from three hospitals located in the Bronx. Women who participated in the study were 18 years of age or older, between 22 and 34 weeks pregnant with a singleton pregnancy, free from chronic illness and free from uterine, cervical, or fetal anomalies, and self-identified as Black. Women were excluded if they could not read or speak English or Spanish. Although demographic information was only collected at the first study visit, serum cotinine was collected from patients at each of three possible timepoints depending on the gestational age of the patient at the time of recruitment. For our purposes, only the serum cotinine at Time 1, between 22 and 24 weeks of gestational age, was analyzed to minimize missing data.

From the 269 women enrolled in the study from August 2009 through May 2012, 244 were included in data analyses. Women were excluded if they had missing cotinine values at Time 1 (21) or self-reported being a smoker but had cotinine levels ≤ 3 ng/ml (1). Women were also excluded if they identified as mixed race (5) or were Black Hispanic foreign born (2) due to small sample sizes. For four women with missing cotinine at Time 1, we used their cotinine sample at Time 2 (between 26 and 29 weeks of gestational age).

Institutional review board approval for this study was obtained through Boston College, Albert Einstein College of Medicine, Lincoln Medical and Mental Health Center, and the New York State Department of Health.

Sociodemographic Characteristics and Self-Report of Smoking

At the first study visit, women were interviewed to determine study eligibility and collect sociodemographic characteristics. Women reported their race/ethnicity, age, last grade of school attended, annual household income, marital status, hours worked per week (being employed was defined as working 1+ hr/week), number of previous live births, and the number of adults they lived with. Women also reported whether they currently smoked.

Women self-identified themselves as either Black, African American, Caribbean American, Black Hispanic, or mixed race. As only five women reported their race/ethnicity as Black, we included them with African American women. Information on place of birth (U.S. born vs. foreign born) was extracted from the medical record. We constructed a variable combining women's race/ethnicity and nativity based on previous studies demonstrating differences in smoking during pregnancy by country of birth (Elo & Culhane, 2010; Pereira & Cortes, 2006): African American U.S. born, African American foreign born, Caribbean American U.S. born, Caribbean American foreign born, and Black Hispanic U.S. born.

Laboratory Methods

Serum samples collected from patients at each timepoint were shipped frozen to New York State Wadsworth Laboratory identified only by study numbers and were accessioned and reported using this identifier. Serum cotinine levels were determined using a high-throughput 96-well plate format sample preparation and then analyzed using an isotope dilution, liquid chromatography/tandem mass spectrometry (LC/MS/MS) method. The analysis was a modification of techniques used by the Centers for Disease Control and Prevention for the national Health and Nutrition Examination Survey (HANES) and New York State Wadsworth Laboratories for the NYC HANES studies (Ellis et al., 2009).

In summary, 250 μ l of each serum sample was equilibrated with a trideuterated cotinine internal standard solution and extracted using a 96-well Bond Elut Plexa Solid Phase Extraction plate (Varian). The acetonitrile sample extract was taken to dryness and reconstituted in 50 μ l of 96%/4% acetonitrile/water solution, and a 5- μ l injection volume was used for LC/MS/MS analysis using electrospray ionization (ESI). The instrumental systems comprised a Shimadzu Prominence LC with a Phenomenex Luna Hilic (100 $\times$ 2.00 mm) column and a AB Sciex API 4000 triple quadrupole mass spectrometer operated in ESI-positive-ion mode using multiple reaction monitoring detection. The ratio of the signal for the native cotinine (m/z 177 $\rightarrow$ 80) to the internal standard (m/z 180 $\rightarrow$ 80) was used for quantitation. Calibration standards were prepared from authentic standards of cotinine and d3-cotinine obtained from Sigma-Aldrich Chemical Co.

Typical batches included 60 serum samples, at least 9 blanks and 12 quality control samples. Three quality control (QC) pools were used at low-, medium-, and high-target cotinine concentrations of 0.173, 1.61, and 15.7 ng/ml. During specimen analysis, the instrument was calibrated using a multipoint calibration curve, and also, each Day 3 blanks, low, medium, and high QC were analyzed for quality control purposes. Final results were blank corrected using the mean batch blank value. The average blank for this study was 0.025 ng/ml ($n = 69$).

Quality control charts for the three QC levels were evaluated to ensure that data were reported only when the analysis was within control limits and that signals did not exceed the calibration range, otherwise, the analysis was repeated. The limit of detection for this method was 0.05 ng/ml cotinine in serum.

Data Analysis

We defined smokers as those women with a serum cotinine level higher than 3 ng/ml and nonsmokers as those with a serum cotinine level 3 ng/ml or lower (Benowitz, Bernert, Caraballo, Holiday, & Wang, 2009). Women with SHS exposure were defined as nonsmokers with serum cotinine levels ≥ 0.05 and ≤ 3 ng/ml.

We first compared women's self-report of smoking status with serum cotinine levels to examine the congruency between measures. Women who were smokers, as defined by serum cotinine levels, were excluded from further analyses. We then examined sociodemographic characteristics associated with SHS exposure using Pearson's chi-squared tests; subsequently, those factors $p \leq .05$ were included in an adjusted logistic regression model. Missing values for education and household income were coded to be included in the analyses. We conducted analyses using Stata Statistical Software, version 12.1 SE.

RESULTS

In this sample of pregnant women in NYC, 57% were African American and a further 35% were Caribbean American, 34% of women were foreign born, approximately half were 18–24 years old or had 12 years of education or less, 59% reported an annual household income of less than \$5K, and 79% were single (Table 1).

Although 4.1% of women self-reported they were smokers, 10.7% had serum cotinine levels indicating they were current smokers (Table 2). The overlap suggests that among smokers, as determined by serum cotinine levels, 61.5% (16/26) did not disclose they were smokers.

Among women who were nonsmokers based on serum cotinine levels, 46.8% (102/218) had levels indicating SHS exposure (Table 2). However, more than 60% of women from some subgroups had detectable serum cotinine levels (Table 3). Women at the highest risk included those who were U.S. born and either Black Hispanic or African American, had less than 12 years of education, did not report an annual household income, or did not have any other adults in their household. Gradients were evident across most factors, such that a lower proportion of women who were older, had at least 12 years of education, a household income of at least \$5K, were married, and employed had detectable SHS. There were no differences in SHS exposure by parity or the number of adults in the household.

In a mutually adjusted model, the strongest predictors of SHS were race/ethnicity and nativity and education (Table 3). Compared with African American U.S.-born women, those who were African American foreign born or Caribbean American and either U.S. or foreign born were less likely to have detectable SHS exposure (all $p \leq .05$). There were no differences between U.S.-born women who were Black Hispanic and African American. Women with more than 12 years of

education were also less likely to have detectable SHS exposure than women with less than 12 years ($p \leq .05$). There were no significant differences in SHS exposure by age, household income, marital status, or employment status.

DISCUSSION

Despite living in a city with comprehensive tobacco control policies, we found that nearly half of nonsmoking, low-income, ethnic minority pregnant women in NYC had detectable levels of SHS as measured by serum cotinine. Disparities were evident even within this group, such that approximately 65% of U.S.-born Black Hispanic and African American women and those with less than a high school degree had SHS exposure. These differences by race/ethnicity, nativity, and education remained after adjustment for a range of potential confounding factors.

We showed that 62% of pregnant women whose serum cotinine indicated they were current smokers denied they were smokers during our study interview. This is potentially an even higher estimate than Dietz and colleagues (2011) who reported that approximately one in four pregnant smokers did

Table 1. Sociodemographic Characteristics of Pregnant Women in New York City ($N = 244$)

	No.	%
Race and nativity		
African American U.S.-born	101	42.3
African American foreign-born	34	14.2
Caribbean American U.S.-born	36	15.1
Caribbean American foreign-born	48	20.1
Black Hispanic U.S.-born	20	8.4
Age (years)		
18–24	116	47.5
25–29	63	25.8
30+	65	26.6
Education (years)		
<12	52	21.3
12	81	33.2
>12	103	42.2
Missing coded	8	3.3
Annual income		
<\$5K	144	59.0
$\geq$ \$5K	88	36.1
Missing coded	12	4.9
Marital status		
Single	192	78.7
Married	52	21.3
Employment status		
Not employed	125	52.1
Employed	115	47.9
Parity		
Nulliparous	112	45.9
Multiparous	132	54.1
Number of adults in household		
0	32	13.3
1	147	61.3
2+	61	25.4

Note. Missing values: race and nativity (5), employment status (4), and number of adults in household (4).

Secondhand smoke exposure

not disclose they were active smokers by comparing serum cotinine levels to self-report. Although they found that younger pregnant women were more likely to not disclose their smoking status than older women (Dietz et al., 2011), we were unable to examine characteristics due to our small sample size of smokers. Serum cotinine has a half-life of 16 hr (Benowitz, Pérez-Stable, Gong, Modin, Herrera, & Jacob, 1999), and there

is the potential for misclassification using a single measure. In our data, we compared measures at Time 1 and Time 2 during pregnancy among a subset of mothers ($N = 178$), and the estimates of smokers and SHS exposure were consistent (data not shown).

In our study of African American, Caribbean American, and Black Hispanic pregnant women from the Bronx, nearly half had elevated serum cotinine levels indicating SHS; however, we did not have a comparable self-report measure of exposure nor information on sources of exposure. A small-scale study of African American and Dominican pregnant women from Washington Heights, Harlem, and the Bronx a few years prior to the implementation of smoke-free policies in NYC found that almost half of African American women reported the presence of a smoker in the home compared with one-third of Dominican women; the percent of detectable plasma cotinine in cord blood differed by 78% and 32%, respectively (Perera et al., 2004). A study using data on an ethnically diverse sample of pregnant nonsmokers from California in 1992 found that 72% of women reported no SHS exposure, but nearly all

Table 2. Women's Self-Report of Smoking Status by Serum Cotinine Level Categories ($N = 244$)

Serum cotinine level categories	Self-report of smoking (no.)		Total
	No	Yes	
Below detection limit	116	0	116
Secondhand smoke exposure	102	0	102
Smoker	16	10	26
Total	234	10	244

Table 3. Sociodemographic Characteristics and Adjusted Odds of Secondhand Smoke Exposure Evaluated Through Serum Cotinine Levels

	% Secondhand smoke exposure ($n = 218$)	p value	Adjusted OR ^a (95% CI) ($n = 214$)
Race and nativity		<.001	
African American U.S.-born	63.0		1
African American foreign-born	20.6		0.19 (0.07, 0.54)**
Caribbean American U.S.-born	40.0		0.41 (0.18, 0.97)*
Caribbean American foreign-born	36.2		0.31 (0.14, 0.72)**
Black Hispanic U.S.-born	63.2		0.97 (0.32, 2.91)
Age (years)		.005	
18–24	57.7		1
25–29	42.1		0.89 (0.41, 1.90)
30+	31.6		0.52 (0.23, 1.21)
Education (years)		.02	
<12	66.7		1
12	48.6		0.51 (0.22, 1.23)
>12	37.8		0.39 (0.17, 0.91)*
Missing coded	33.3		0.36 (0.05, 2.89)
Annual income		.03	
<\$5K	52.0		1
≥\$5K	35.8		0.72 (0.31, 1.67)
Missing coded	66.7		3.47 (0.85, 14.12)
Marital status		.02	
Single	50.9		1
Married	32.7		0.87 (0.39, 1.95)
Employment status		.02	
Not employed	54.1		1
Employed	38.3		0.67 (0.31, 1.46)
Parity		.08	
Nulliparous	52.8		
Multiparous	41.1		
Number of adults in household		.5	
0	57.7		
1	44.7		
2+	46.4		

Note. OR = odds ratio; CI = confidence interval.

Missing values: race and nativity (2), employment status (3), and number of adults in household (5).

^aModel only includes variables with $p \leq .05$.

* $p \leq .05$; ** $p \leq .01$.

women had some detectable serum cotinine levels (DeLorenze, Kharrazi, Kaufman, Eskenazi, & Bernert, 2002). Despite the growing literature on SHS exposure and fetal health (Leonardi-Bee et al., 2008, 2011; Nieuwenhuijsen et al., 2013; Salmasi et al., 2010; USDHHS, 2006), estimates of SHS vary by reporting method (biochemical measure vs. self-report) and often appear to be context specific—country, urban versus rural, and the sociodemographic composition of the sample. SHS exposure has been shown to be high in outdoor settings (Sureda, Fernandez, Lopez, & Nebot, 2013), public housing (Winickoff, Gottlieb, & Mello, 2010), and cultural differences in smoking patterns impact SHS exposure from partners and the greater community (CDC, 2011; Karlamangla, Merkin, Crimmins, & Seeman, 2010).

The implications of nondisclosure of smoking status or under-reporting of SHS exposure for clinical purposes are similar to those for public health surveillance. Serum cotinine is expensive to analyze and often not available for health professionals to assess smoking status and SHS exposure in the clinic or through large-scale data collection in population-level surveys. Despite serum cotinine being a more accurate measure, self-report is sometimes the only option. As the meta-analyses of SHS on fetal health used both self-report and biochemical measures of SHS and no differences in outcomes were noted, self-report of SHS exposure is likely to be a valid option (Leonardi-Bee et al., 2008, 2011; Nieuwenhuijsen et al., 2013; Salmasi et al., 2010). Further research is needed to help identify pregnant women who are at higher risk of both smoking during pregnancy and SHS exposure in order to focus prevention efforts. This will likely include a combination of population-based analyses to identify risk factors for smoking during pregnancy and SHS exposure as well as speaking with pregnant women directly to identify reasons for nondisclosure and sources of SHS.

National HANES data from 2007–2008 showed that 37% of females in the United States had detectable levels of serum cotinine (CDC, 2010) in contrast to 47% in our sample of non-smoking pregnant women in NYC. Non-Hispanic Black non-smokers have higher cotinine levels than nonsmokers of other racial/ethnic groups at the same level of SHS exposure (Chen et al., 2010; USDHHS, 2006), which may partially explain this discrepancy. Despite the implementation of a comprehensive tobacco control program in NYC in 2003, the proportion of pregnant women from the Bronx with SHS exposure in our sample was only slightly lower than that found among females in the NYC HANES in 2004 (47% vs. 51%) (Ellis et al., 2009). A follow up of the NYC HANES is planned for 2013 (New York City Department of Health and Mental Hygiene, 2013), which will help evaluate change in SHS exposure and disparities in serum cotinine levels.

Consistent with other studies, we found socio-economic and racial/ethnic gradients in SHS. Lower educational attainment was associated with higher risk of exposure (CDC, 2010; Ellis et al., 2009). We also showed that a higher proportion of U.S.-born women who were African American or Caribbean American had detectable serum cotinine than their foreign-born counterparts. Although U.S.-born women are more likely to smoke during pregnancy than foreign-born women (Elo & Culhane, 2010; Pereira & Cortes, 2006), we are not aware of other studies that have examined SHS exposure by nativity among pregnant women. Interestingly, Ellis and colleagues (2009) did not find a difference in SHS exposure by nativity

in their study of adults in NYC; however, the prevalence of smoking was lower among adults who were foreign born than those who were U.S. born, which could influence SHS exposure among partners, family members, and their community.

The high proportion of SHS exposure among our sample of nonsmoking, low-income ethnic minority pregnant women has direct implications for fetal health (Leonardi-Bee et al., 2008, 2011; USDHHS, 2006) and potentially for their infants postpartum as well (USDHHS, 2006). Our results highlight that health professionals need to actively assess risk of SHS exposure among pregnant women and counsel about strategies to limit SHS exposure during the perinatal period. These discussions can take place in the context of on-going prenatal care, particularly exposure in their home and work environments. Health care providers caring for women postnatally also need to counsel about strategies to reduce the risk of SHS exposure for the new baby and all members of the family. The promotion of strategies that stress smoke-free environments in the home, work, and car should occur even in environments with comprehensive policies (Best et al., 2009). Despite the success of public health policies (USDHHS, 2011), programs (CDC, 2013; USDHHS, 2013), and clinical guidelines (American College of Obstetricians and Gynecologists, 2011; Best et al., 2009; Fiore et al., 2008) to target smoking during pregnancy and SHS exposure in children, our findings suggest that more is needed to raise awareness about SHS during pregnancy to improve maternal and fetal health.

FUNDING

This work was supported by grants from the National Institutes of Health—Eunice Kennedy Shriver National Institute of Child Health & Human Development (R00HD068506 to SSH) and National Institute of Nursing Research (R01NR010552 to SG).

DECLARATION OF INTERESTS

None declared.

ACKNOWLEDGMENTS

We would like to thank Dr. T. Welsh, MD, FACOG, Co-Director of Family Planning in the Department of Obstetrics/Gynecology at Lincoln Medical & Mental Health Center, and J. Daly, NY State Department of Health, Wadsworth Center. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

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