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Prenatal Hair Nicotine Analysis in Homes with Multiple Smokers

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Synopsis

Prenatal exposure to secondhand smoke (SHS) is responsible for adverse perinatal outcomes, including preterm birth. Smoking in the home is the primary source of exposure to women during pregnancy. Hair nicotine analysis of mothers and infants was used to describe the relationship between prenatal SHS exposure and number of household smokers. Maternal hair nicotine was strongly correlated with number of household smokers, and a more sensitive measure of household smoking than infant hair. Home smoking bans and focused public media campaigns on the harmful effects of SHS exposure are necessary prevention strategies to avoid adverse perinatal outcomes.

Keywords

Pregnancy; secondhand smoke; biomarker; preterm birth; nicotine

Introduction

Primary smoking during pregnancy places a woman at greater risk for preterm delivery, preterm premature rupture of membranes, and delivering a low birth weight (LBW) or small for gestational age (SGA) infant [1]. Although there is less evidence regarding the effect of secondhand smoke on perinatal morbidity; increase risk for decreased birthweight, smaller head circumference, stillbirth and preterm birth have been consistently reported [2-5].

Secondhand smoke is defined as smoke inhaled by an individual that is not actively engaged in smoking but who is exposed to ambient tobacco smoke [6]. SHS consists of two components: sidestream smoke and mainstream smoke. Sidestream smoke refers to the smoke emitted from tobacco products, while mainstream smoke refers to the smoke exhaled by smokers. SHS contains approximately 4,000 chemicals, is responsible for nearly 3,000 cases of lung cancer deaths among nonsmokers each year, and affects more than 22 million US children annually [7].

Biomarker validation is recommended to confirm smoking and SHS due to a high deception rate for self-report status caused by the social pressures attributed to smoking during pregnancy [8]. Whether a women smokes during pregnancy or is exposed to SHS, biomarkers of exposure can be detected in both the mother and infant [9-11]. Nicotine in

maternal hair is a valid biomarker strongly associated with prenatal reports of SHS exposure [12].

Research examining the association between prenatal smoking, SHS exposure and number of household smokers is limited. The purpose the study is to examine the relationship between maternal and infant hair nicotine levels and number of household smokers in women who smoke and who are exposed to SHS during pregnancy. The study aims propose to: 1) examine the difference in maternal hair nicotine in smoking and SHS-exposed women in homes with one, or two or more additional smoker(s) in the home; 2) to examine the difference in infant hair nicotine in smoking and SHS-exposed women in homes with one, or two or more additional household smokers; and 3) describe the relationship between hair nicotine levels and preterm birth in homes with multiple smokers.

METHODS

Study Design and Population

A correlational, cross-sectional study design was used to determine the association between perinatal exposure to home SHS and hair nicotine levels. Prior to data collection, the study was approved the University of Kentucky Institutional Review Board, and all participants provided informed consent. Potential study participants were identified between December 2006 and April 2007 at the University Hospital. To be included in the study women had to be at least 18 years of age, singleton gestation, and have maternal scalp hair of at least 2 cm in length. Women were excluded if their pregnancy resulted in stillbirth, neonatal death, and/or delivery of an infant with severe congenital anomalies. Women with documented use any drugs of abuse during pregnancy were also excluded. In the four month data collection period, there were 656 births, with nearly 85% meeting eligibility requirements. Quota sampling was used to ensure a representative distribution of mothers who were smokers, nonsmokers/SHS exposed, and nonsmokers/nonexposed during pregnancy.

A total of 210 women were consented to participate within three days of their infant's birth. Mothers were identified via the Labor and Birth daily census report and approached about participating while in the postpartum unit. After obtaining written consent, mothers were asked to complete a questionnaire. After collection of urine and hair samples by trained research assistants, participants were offered a choice of two incentives: payment of $25, or the equivalent of $25 in diapers and wipes.

Classification of smoking and SHS exposure

A woman was classified as a self-reported smoker if she responded "*yes*" to the question, "*Have you smoked a cigarette, even a puff, in the past 7 days*." Mothers who smoked were asked to classify their daily smoking consumption over the last 30 days as: <1 cigarette, 1-5, 6-10, 11-15, 16-20, 21-25, 31-35, 36-40, and > 40. Classification of SHS exposure was based on self-report after confirmation of nonsmoking status. Nonsmokers were defined by urine cotinine ≤ 99 ng/mL and current smokers were defined by urine cotinine ≥ 100 ng/mL. Previous reports on classification of smoking status using urine cotinine have yielded sensitivity and specificity of 88% and 92%, respectively [13]. Number of home SHS exposure sources was defined by number of persons living in participant's home (> one month) that smoked tobacco products. If the participant did not report any prenatal exposures to SHS in home, vehicle or work they were classified as nonsmoking, nonexposed. If a participant answered "*yes*" or quantified exposure (hours or days) to any of the smoking exposure questions, they were classified as nonsmoking/SHS exposed.

Collection of maternal and newborn hair

Maternal hair samples were collected by cutting a proximal segment of hair (approximately 20-25 strands) from the posterior vertex nearest the scalp and placed in a paper envelope. Collection of newborn hair involved cutting a pencil-width segment of hair behind the ear, placed in a paper envelope and stored until being mailed to Wellington Hospital, Wellington, New Zealand for analysis. Additional information regarding maternal and infant hair nicotine collection and analyses were previously reported [14].

Statistical Analysis

With an effective sample size of 210 mothers and an alpha level of .05, the power of Spearman rank correlation to detect a significant association as small as .2 was calculated to be at least 80%. Univariate analyses were used to summarize demographic and socioeconomic characteristics of the participants (see Table 1). Age and number of smokers in the home are presented as mean values. The distribution of hair nicotine levels was positively skewed, thus data were log transformed. Hair nicotine levels are presented as geometric means and 95% CIs. Data were analyzed using SAS version 9.3; an alpha level of .05 was used throughout.

RESULTS

The character demographics of participants are displayed in Table 1. Mean age for the participants who smoked is 24.3 years compared to 24.8 year for those who were SHS exposed. Fifty three of the 210 participants were categorized as smokers and 157 as nonsmokers. Of the nonsmokers, 66 were classified as SHS exposed; all of which reported their primary exposure site as their home. Women who smoked during pregnancy had a mean of 2.4 additional smokers in the home, compared to 1.2 for women who were SHS-exposed.

Maternal and infant hair samples were collected in nearly all of the participants (99% mothers, 98% infants). There was no significant difference in demographics between mothers who smoked and those who were exposed to SHS with the exception of college education. Significant differences in maternal hair nicotine existed between SHS-exposed and women who smoked during pregnancy when comparing hair nicotine with one, or two or more smokers in the home (one smoker in home: 0.0942, 1.9279; and two or more smokers in the home: 0.1094, 1.0501, respectively). Women who smoked during pregnancy had nearly twice the exposure time (hours) compared to women were exposed to secondhand smoke (79.8 and 45, respectively). See Figures 1 and 2 for the associations between household smoking and mean maternal-infant hair nicotine levels.

Maternal hair significantly correlated with all measured smoking behaviors and had stronger association with smoking behavior than infant hair (Table 2). Infant hair demonstrated weaker but significant association in the reported smoking behaviors. Number of cigarettes smoked by other household members in the home ranged between 1- 40 cigarettes per day; with a mean of 12.5 cigarettes per day. Half of the mothers who smoked during pregnancy reported smoking 1 – 10; 28% reported smoking 11 to 20; and 22% reported smoking greater than 20 cigarettes per day. Total number of cigarettes smoked per day strongly correlated with both maternal and infant hair nicotine levels [14].

Over two-thirds of the preterm infants were born from women who smoked (36%) or who were exposed to SHS (33%) during pregnancy. Infants whose mother lived with two or more smokers in the home were nearly twice as likely to be transferred to the neonatal intensive care unit (NICU) compared those with one smoker in the home. There were no significant

differences in demographics in regard to NICU admission when comparing smoking to SHS exposed women.

Discussion

Maternal and infant hair nicotine levels increased as number of smokers in the home increased. Evidence continues to stress the strong association between secondhand smoke exposure and poor birth outcomes [5] [15], however measurement of number of household smokers is seldom reported and/or related with perinatal outcomes. Consistent with a previous report, this study demonstrated a significant association between maternal hair nicotine level and number of smokers in the home [16]. However, the association with infant hair nicotine and number of smokers in the home was weaker than the association with maternal hair.

Other perinatal exposure considerations include dose-response relationships and hair nicotine reference values. Dose response relationships between urine cotinine and home exposure in children and non-pregnant adults have been widely reported [17, 18]. Dose-response relationship between number of smokers in the home and perinatal outcomes is less clear. Fantuzzi et al. (2007) [16] recently noted the significant association between number of home smokers and early preterm birth (< 35 weeks gestation). Previous examination of prenatal segmental hair nicotine analysis reported proximal mean concentrations in three smoking behavior classifications: nonsmoking: 2.8 ng/mL; exposed: 4.3 ng/mL; and 11.08 ng/mL) [19]. Similar trends in the nicotine concentrations (ng/mL) of maternal hair segments were noted in our study (0.33, 1.02, and 9.6, respectively). Hair sampling size may account for lesser nicotine concentrations in the present study. In both studies, maternal hair nicotine (unlike infant hair) was able to significantly differentiate between the three smoking classifications. Also noted are recommended reference values for prenatal hair cotinine levels to distinguish active smokers from passive or unexposed is 0.2 ng/ml and 0.03 ng/ml to distinguish between those exposed and nonexposed ng/mg also exist [20].

During pregnancy and postpartum, a women's primary prenatal SHS exposure locale is in her home, and primary exposure source is her spouse and/or significant other [21]. In this study, her significant other was most often defined as spouse, partner and/or infant's father, followed by mother or mother-in-law. Home smoking restrictions or home smoking bans/ policies can decrease and/or prolong relapse rates in pregnant and/or postpartum women [22] [21][23][24]. Conversely, homes that allow smoking in the home have higher prenatal smoking rates [25]. Having home smoking restrictions has also been reported to impact intention to quit smoking [26]. Although sustained smoking cessation provides the most benefits for decreasing perinatal exposure to SHS, consideration for a "reduction of smoke" could offer health benefits to the family [24]. Few studies evaluate the relationship between a maternal home smoking ban and perinatal outcomes. Yoo et al. (2010) [27]concluded that spouses' who smoked outside the home did not reduce overall exposure to pregnant women compared to nonsmoking spouses.

Limitations in the present study exist and should be addressed. First, self report of smoking and SHS exposure during pregnancy can result in high deception rates; however only 3% of confirmed smokers via urine cotinine reported nonsmoking status. Second, all participants were recruited from a local hospital, which may limit generalizability. Third, sampling error cannot be calculated with quota sampling methods. Fourth, ethnic hair nicotine differences were not accounted for due to sample size and statistical limitations. In the present study, ethnicity would have greatly reduced group sizes when examining SHS and smoking behaviors in homes with one, or two or more smokers in the home. Finally, maternal and infant hair nicotine measurement was performed at one time point. Although, nicotine in

hair is a very stable marker of long-term exposure, collection during each trimester would offer a more comprehensive and valid assessment regarding smoking and exposure status.

Strengths in the study include biochemical validation of smoking status of pregnant women; use of hair nicotine as a valid and long-term biomarker of secondhand smoke exposure; collection of both maternal and infant hair samples; and collection of several self-reported measures of home SHS exposure (hours per day, number of exposure sources, and number of household smokers).

Conclusions

Maternal hair is a more valid and sensitive measure of number of household smokers than infant hair. Pregnant women who smoke or who are exposed to SHS are more likely to experience a multitude of adverse perinatal outcomes than those who do not smoke and/or are not exposed. Furthermore, pregnant women living in households with two or more smokers are more likely to have their infant admitted to the NICU immediately after birth. Health care providers and clinical nurse leaders are strongly encouraged to educate all clients about the adverse perinatal effects of smoking and SHS exposure among pregnant women. Compromised infants face a multitude of acute and lifelong health issues; thus a significant percentage of adverse outcomes could be prevented by avoiding primary and secondary smoking during pregnancy. Prevention strategies including home smoking bans and focused public media campaigns on the harmful effects of SHS exposure are necessary to avoid the adverse perinatal outcomes associated with secondhand smoke.

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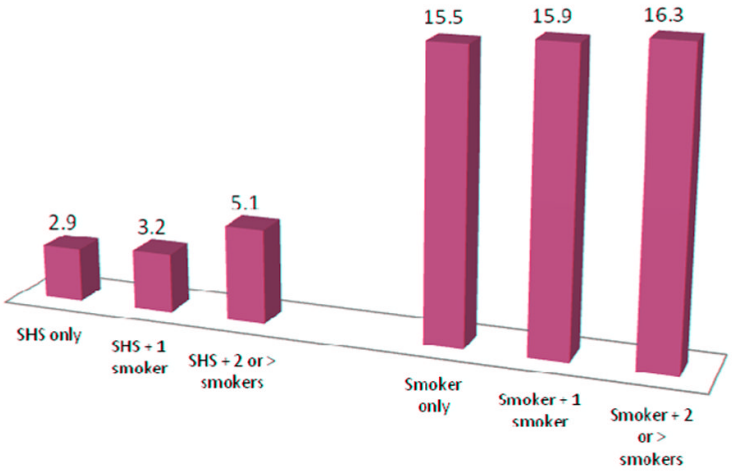

Figure 1.
Mean Nicotine Levels (ng/ml) for Maternal Hair (n=201)

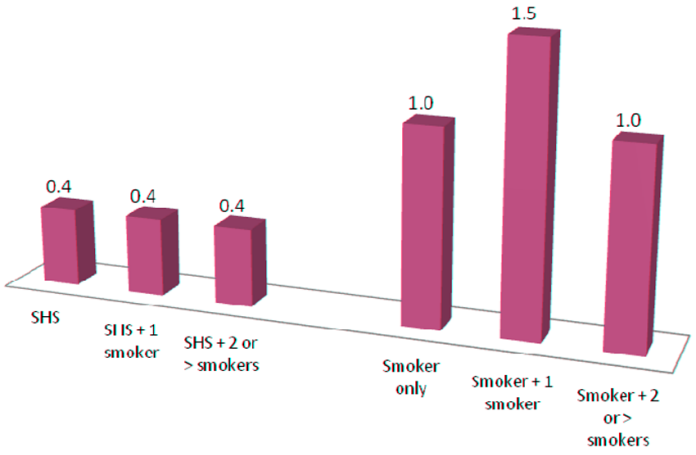

Figure 2.
Mean Nicotine Levels (ng/ml) for Infant Hair (n=178)

Table 1

Demographics and Smoking Classification (n = 210)

	Smoker	NonSmoker/Passive	NonSmoker/NonExposed
Ethnicity/Race	n=53 (%)	n=66 (%)	n=91 (%)
White	41 (19.5)	33 (15.7)	45 (21.4)
African American	10 (4.8)	14 (6.7)	8 (3.8)
Hispanic	0	18 (8.6)	35 (16.7)
Marital Status			
Single	37 (17.7)	40 (18.9)	7 (13)
Married	15 (7.2)	26 (12.4)	64 (30.6)
Highest Grade Completed			
Less than High School	22 (11.1)	13 (6.2)	23 (11.0)
High School/GED	17 (8.1)	25 (12)	20 (9.6)
Some College and above	14 (6.7)	27 (12.9)	48 (23)

Table 2

Spearman rank correlations among hair nicotine and smoking variables ($p < .05$)

Self-reported Smoking Variables	Hair Nicotine	
	Maternal hair	Infant hair
Smoking status	.74	.39
Number of cigarettes per day	.68	.45
Total SHS exposure*	.68	.28
Two or more adults smoking in home	.61	.23

[a]Note: Total SHS exposure accounts for home, work and vehicle