Nicotine & Tobacco Research, Volume 13, Number 8 (August 2011) 653–660

Original Investigation

Sources and Frequency of Secondhand Smoke Exposure During Pregnancy

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Received August 30, 2010; accepted March 1, 2011

Abstract

Introduction: This study examined sources of exposure to secondhand smoke (SHS) during pregnancy and misclassification of women as having no SHS exposure if partner smoking was used as the only measure of SHS exposure. We also examined changes in SHS exposure across the three trimesters of pregnancy.

Methods: The sample consisted of 245 pregnant women who were in a serious relationship with a partner and 106 for examination of change over time. Women's smoking status was determined by a combination of self-reports and oral fluid assays. Women's reports of partner smoking, smoking by other social network members, and frequency of exposure to SHS were obtained.

Results: The most common source of SHS exposure during pregnancy was the partner ($n = 245$). However, reliance on the partner smoking measure alone would have misclassified a substantial number of women as having no SHS exposure during pregnancy. The importance of exposure from the general social network was also evident in the finding that among nonsmoking women with nonsmoking partners, 50% reported some level of SHS exposure in the preceding week. Contrary to expectations, there were no changes in SHS exposure across the three trimesters of pregnancy ($n = 106$).

Conclusions: Results highlight the need for treatment plans to target sources of exposure from other members of women's social networks in addition to partners. It may be unrealistic to expect women's cessation efforts to be successful in the face of consistent and continued SHS exposure through pregnancy.

Introduction

Results from studies focusing on maternal exposure to secondhand smoke (SHS) during pregnancy suggest that there may be significant adverse effects on fetal and neonatal health. For instance, in a meta-analysis of studies on SHS exposure during pregnancy, Leonardi-Bee, Smyth, Britton, and Coleman (2008) reported a significant association between SHS exposure and low birth weight at term and small for gestational age status among nonsmoking mothers. Other studies have noted similar results with lower fetal biometry at 20–24 weeks gestation (Hanke, Sobala, and Kalinka, 2004), higher risk for fetal death, preterm delivery, and low birth weight (Gray et al., 2010; Jaddoe et al., 2008). SHS exposure during pregnancy has also been associated with poorer respiratory health among infants over the first 6 months of life (Jedrychowski et al., 2007), and among children with asthma, higher exposure to SHS is associated with higher externalizing behavior problems in the early school years (Yolton et al., 2008). Thus, accurate identification of SHS exposure among pregnant women is an essential first step to preventing negative consequences for maternal and infant health.

The study is guided by social–ecological theory (e.g., Corbett, 2001; Ennett et al., 2010; Green, Richard, amp and Potvin, 1996; Stokols, 1996) suggesting that multiple domains of influence such as household/family, peers, and workplace may provide a context for maintenance of smoking through pregnancy. For example, contexts characterized by the absence of antismoking rules and policies and a high number of individuals who smoke and continue to smoke around the pregnant woman are likely to support the maintenance of smoking during pregnancy. Alternatively, these sources of influence may provide motivation for quitting if social network pressures inhibit access or opportunity and provide social pressures to quit smoking during pregnancy. Finally, this theory would suggest that it is important to consider multiple domains of influence to understand the social context of smoking. Thus, identification of multiple sources of SHS exposure across several contexts is important for understanding the social ecology of pregnancy smoking and of quitting and maintaining quit status.

There is no gold standard for self-report measurement of SHS exposure during pregnancy. Most studies of pregnant women measuring SHS exposure (SHS) use one single item

doi: 10.1093/ntr/ntr053
Advance Access published on April 2, 2011

measure of SHS exposure. The most common methods of measuring SHS exposure among pregnant women have been a single question about partner smoking status or the number of cigarettes smoked by the partner (see Windham, Eaton, and Hopkins, 1999, review). When studies of pregnant smokers have included other questions, they consist of either a question about number of smokers in the household or hours of exposure to SHS (Leonardi-Bee et al., 2008; Newman et al., 2010; Pogodina, Brunner Huber, Racine, and Platonova, 2009; Windham et al.). Few prospective studies of maternal cigarette smoking during pregnancy have examined ongoing SHS exposure using more than single indicator measures of SHS exposure, although studies of postnatal SHS exposure or lifetime SHS exposure have more comprehensive measures (e.g., Edwards, 2009; Rise and Lund, 2005). While it is clear that pregnant smokers are very likely to have partners who also smoke, it is also likely that they will have higher exposure to SHS from other social network sources such as relatives, friends, or coworkers. The first purpose of this study was to provide descriptive data about the sources of SHS exposure during pregnancy.

A second related question is the relationship among the different sources of exposure and the frequency of exposure to SHS. For pregnant women, the most significant predictor of frequency of SHS exposure after accounting for women's own smoking status may be the partner. This relationship is likely to vary according to living status, such that women who live in the same household with their partners will have a higher frequency of SHS exposure than women who live apart from their partners. However, other sources of SHS exposure such as household members, relatives, friends, or coworkers may also account for significant variance in frequency of SHS exposure. In a recent study, Edwards (2009) noted that a large number of women in the general population would be misclassified as having no SHS exposure if only a spousal measure (partner smoking status) of SHS was used. The same could be true of pregnant women as well. Thus, the second goal of this study was to examine partner smoking and other sources of SHS in the social network as predictors of frequency of SHS exposure. In particular, we examine two sides of this issue; (a) the extent to which the other sources of SHS predict SHS exposure after controlling for the women's smoking and their partner's smoking and (b) the extent to which women would be misclassified as having no SHS exposure if only partner smoking status was used as a measure of SHS exposure. If partner smoking was the only predictor of SHS exposure, then perhaps targeting only partner smoking in prevention/intervention studies of SHS exposure would be sufficient to reduce SHS exposure to nonproblematic levels. However, if other sources of exposure (e.g., friends, relatives, etc.) are significant predictors as well, this would be associated with substantial misclassification of SHS exposure and would suggest the need to address the broader social network.

Several studies have examined changes in maternal cigarette smoking throughout pregnancy (Munafò, Heron, and Araya, 2008; Spears, Stein, Koniak-Griffin, 2010). However, little is known about changes in frequency of SHS exposure across the three trimesters of pregnancy. Thus, the final goal of this study was to examine if there were changes in frequency of SHS exposure during pregnancy as a function of women's own smoking status or partner smoking status. If there are no changes in SHS exposure, a one time measurement of SHS exposure during pregnancy would be enough to provide an accurate picture of pregnant women's SHS exposure. From a treatment standpoint, a lack of change in SHS exposure may be a significant barrier to abstinence for pregnant smokers and may need to be addressed in treatment studies. However, if there are significant reductions in SHS exposure across the three trimesters of pregnancy, measurement and treatment implications would be different.

On the basis of this literature, we hypothesized that pregnant smokers and women with smoking partners would be more likely to have household members, relatives, friends, and coworkers who smoked. Moreover, in addition to women's own smoking status, we expected partner smoking to be a significant predictor of frequency of SHS exposure. We expected that reliance on partner smoking alone would misclassify a number of pregnant women who were exposed to SHS as having no SHS exposure. Given the increased media focus on the negative effects of SHS in recent years, we expected general declines in frequency of SHS exposure across pregnancy.

Methods

Sample Selection

Pregnant women who presented for prenatal care at a large city hospital were asked to complete a self-report screening form at their first prenatal appointment. Women who met initial eligibility criteria were invited to participate in an ongoing prospective study of maternal health and child development. Initial eligibility criteria included the following: less than 20 weeks gestation, maternal age of 18 years or older, no illicit drug use (other than cannabis), no heavy alcohol (more than one drink per day or four drinks on one occasion) or cannabis consumption (more than one joint per day or four joints on one occasion) after pregnancy recognition, and no multiple births. Women who agreed to participate were scheduled for a total of four interviews: a prenatal interview at the end of each trimester and a postnatal interview at 2 months of infant age. Once a pregnant smoker was recruited into the study and had completed the first prenatal interview, the most similar nonsmoking woman with regard to age and education was recruited. The study protocol was approved by the appropriate institutional review board. Participants were informed that data confidentiality was protected by a Federal Certificate of Confidentiality issued by the National Institute on Drug Abuse.

Demographic Characteristics

The average age of the women was 24.2 years (SD = 4.9 years) at the time of their first prenatal interview. The sample was 46% African American, 20% Hispanic, 26% Caucasian, and 8% other or mixed race. Fifty-eight percent of the women were married or living with their partner, while the remainder of the sample reported being in a relationship, but not living with their partner. Women who were not living with their partner had been in a relationship with their partner for an average of 5.64 years (SD = 5.10 years) and reported seeing their partner for an average of 5.24 days a week (SD = 2.33 days). Twenty-three percent of the women reported that they were primigravidas, with the remainder of the sample reporting an average gravidity of 3.01 (range 1–12, mode = 1). With regard to education, 28% of the women had less than a high school diploma, 32% had earned their high school diploma, 30% had completed some college courses, 7%

had completed a vocational degree or technical training degree, and 3% had earned a bachelor's degree. Forty-one percent of the women reported working at a paid job for an average of 28.95 hrs a week (SD = 10.81 hrs).

Measures

Participants

The initial sample consisted of 312 pregnant women who were first interviewed at the end of the first trimester between 12 and 20 weeks gestation. However, given that we were interested in partner smoking, we restricted our analyses to include only women who reported that they were in a relationship at the time of the first interview. Thus, our final sample consisted of 245 pregnant women who reported being in a serious relationship. All analyses were conducted with these 245 women with the exception of analyses regarding change in SHS exposure over pregnancy. This is an ongoing longitudinal study, and to date, 106 women have completed all three prenatal interviews and the 2-month postnatal interview (in order to obtain information about smoking between the last prenatal interview and delivery). Accordingly, the sample for the change analysis was restricted to these 106 women. Potential differences between the 245 women with first trimester data and the 106 who have complete data at all three trimesters were examined using independent sample t tests. Results indicated that women with complete data for all three prenatal interviews did not differ from those with first trimester data with regard to gravidity, education, number of smokers living in their home, or any of our SHS measures, with the exception of frequency of smoke exposure in a car ($t_{(242.85)} = 2.09$, $p = .04$, $d = 0.27$). Women with first trimester data reported higher frequency of smoke exposure in a car compared to women with complete data. Chi-square difference tests further revealed that women who had complete data did not differ from those with first trimester data in terms of employment, smoking status, partner smoking status, race, or living status with their partner.

Maternal Substance Use

Participants were interviewed in a private setting by trained interviewers. The timeline followback interview (TLFB; Sobell, Sobell, Klajmer, Pavan, and Basian, 1986) was used to assess maternal substance use at each prenatal and the postnatal interview. Participants were provided a calendar and asked to identify events of personal interest (i.e., holidays, birthdays, vacations, etc.) as anchor points to aid recall. This method has been established as a reliable and valid method of obtaining longitudinal data on substance-use patterns, has good test–retest reliability, and is highly correlated with other intensive self-report measures (Brown et al., 1998). At each prenatal appointment, TLFB was used to gather daily tobacco, alcohol, and cannabis use for the previous 3 months. Women who smoked blunts were asked how many joints they could have rolled from the amount of marijuana in the blunt. Thus, self-reported data spanned 3 months prior to conception through delivery. TLFB yielded a number of different measures of substance use. For the purposes of this study, TLFB data and maternal oral fluids were used to ascertain maternal smoking status during pregnancy.

Maternal oral fluid was collected at each prenatal interview to provide objective evidence of recent exposure. The oral fluid specimens were analyzed by a commercial laboratory for cotinine, the primary nicotine biomarker, with enzyme-linked immunosorbent assay (ELISA) for the first 42 women recruited into the study. Liquid chromatography–tandem mass spectrometry (LC–MSMS) at 5 ng/ml cutoff was used thereafter. In addition to TLFB, maternal oral fluid was used to determine maternal smoking status and was not used for identification of SHS exposure. There were three women who had cotinine below the 5 ng/ml cutoff for active use. All three of these women were in the smoking group due to self-reported cigarette use during pregnancy.

Thus, maternal smoking status was determined by a combination of maternal report and maternal oral fluid results. Mothers were included in the smoking group if self-reports were positive, even if oral fluid results were negative (38% of women in the smoking group). Similarly, mothers who reported that they did not smoke but had positive oral fluid samples (1% of women in the smoking group) were included in the smoking group. Of the women in the smoking group, 76% had a positive oral fluid sample, 99% reported smoking, and 74% had both a positive oral fluid sample and positive self-report.

Social Network Smoking

Potential sources of exposure in the women's social environment consisted of the following: if women had a spouse or partner who lived in the household, if their partner had ever smoked cigarettes, if he was a current smoker, and if the partner smoked inside the home. Women were also asked if their partner smoked other forms of tobacco such as cigars or pipes, with the same follow-up questions. A dummy-coded partner smoking status was determined on the basis of these questions. Other questions regarding sources of SHS exposure consisted of the number of smokers in the household excluding the partner, number of relatives who smoked, and number of friends who smoked.

SHS Exposure

Frequency of SHS exposure in the past 7 days was the primary dependent measure in most analyses. Frequency of exposure is an important indicator of amount of exposure in pregnancy that may be most critical for the fetus. Women were asked about the number of days in the past week that they were in the same room, in the same car, or outside with someone who was smoking. Responses to these three variables were averaged to create a composite measure of average frequency of SHS exposure (range 0–7). This composite measure had high internal consistency, Cronbach's alpha = 0.90.

Data Analyses

First, using SPSS 17 statistical software, descriptive analyses concerning sources of SHS exposure during the women's first trimester of pregnancy were conducted for the entire sample (N = 245) and then conducted separately for smokers (n = 173) and nonsmokers (n = 72). Independent sample t tests and chi-square difference tests were also computed to compare smokers versus nonsmokers on all study variables. The degree of association between partner smoke exposure and other sources of SHS exposure during their first trimester of pregnancy was assessed through a series of bivariate correlations between partner smoke exposure and other exposures in their household, car, outside, and other social settings.

Multiple regression was used to determine the degree of association between partner smoke exposure and frequency of

exposure after accounting for the effects of women's age and smoking status. Age was associated with a couple of SHS exposure measures and was thus included as a covariate. Specifically, a 3-step multiple regression model was tested such that frequency of SHS exposure was regressed on women's age and smoking status on the first step, partner smoking status, partner living status, the number of relatives and friends who smoke, and the number of smokers who live with the women excluding partners on the second step, and the relevant two-way interaction terms (i.e., partner smoke exposure by partner living status, partner smoke exposure by women's smoking status, and partner living status by women's smoking status) on the third step.

We also examined the degree of SHS exposure misclassification through utilizing partner smoking status alone as a measure of SHS exposure by cross-tabulating partner smoking exposure with SHS exposure (dummy coded from SHS frequency as zero vs. nonzero). We were specifically interested in determining the proportion of participants who reported SHS exposure during their first trimester of pregnancy but who reported no partner smoke exposure. Finally, repeated measures analysis of variance (ANOVA) was conducted to evaluate whether frequency of SHS exposure changed throughout pregnancy. It must be noted the repeated measures ANOVA was only conducted for participants who had complete data at each of the three assessment timepoints ($n = 106$).

Results

Sources of SHS Exposure Among Pregnant Women

Overall, 82% ($N = 201$) of the women reported that they were exposed to SHS during their pregnancy. From Table 1, it is clear that more than half of the women reported being exposed to partner smoking, with a substantial proportion revealing that their partner smoked in their home during their pregnancy. Furthermore, 14% of pregnant women reported living with a smoker other than their partner and an additional 5% reported that they lived with two or more smokers in their household. On average, women reported that they were in the same room as someone who was smoking 3–4 days per week, in the same automobile with someone who was smoking 1–2 days per week, and outside with someone who was smoking 3–4 days per week. As expected, there were substantial differences in SHS exposure between pregnant smokers compared to nonsmokers. Pregnant smokers reported that they had more relatives and more friends who smoked compared to nonsmoking women ($t_{(135.46)} = 5.19$, $d = 0.89$, and $t_{(163.45)} = 8.99$, $d = 1.41$, respectively). Furthermore, pregnant smokers reported being in the same room, in the same automobile, or outside with someone who was smoking more frequently than nonsmoking women ($t_{(240.79)} = 13.22$, $d = 1.70$, $t_{(242.48)} = 7.06$, $d = 0.91$, and $t_{(242.99)} = 11.57$, $d = 1.48$, respectively). Finally, results showed that pregnant smokers reported living with more smokers than nonsmoking women ($t_{(228.02)} = 3.82$, $d = 0.51$). Chi-square difference tests further showed that pregnant smokers were more likely to be exposed to partner smoking ($\chi^2_{(1)} = 49.48$, $p < .001$).

Bivariate Association Between Partner Smoking and Other Sources of SHS Exposure

At the level of correlations, results demonstrated that frequency of SHS exposure was positively associated with partner smoking status, such that pregnant women who were with a smoking partner reported higher levels of SHS exposure compared to those with nonsmoking partners (see Table 2). As expected, pregnant women with partners who smoked in their home also reported higher frequency of SHS exposure. Finally, the number of smokers living with the women, along with the number of smoking friends and relatives, were each positively related to frequency of exposure. Partner smoking status was positively associated with the number of smokers living with the women and with the number of the women's smoking friends and relatives. Bivariate correlations were in the small to moderate range.

Contrary to expectations, the correlations between the number of smokers in the home and number of friends and relatives who smoke were near zero, as indicated in Table 2. Our original intent had been to create a composite indicator of social network smoking. However, these measures did not "hang together" well as the Cronbach's alpha for internal consistency was 0.33, which suggested that these measures should not be combined. Thus, the individual measures were used in further analyses.

Table 1. Descriptive Statistics for SHS

	All women ($N = 245$)	Smokers ($n = 173$)	Nonsmokers ($n = 72$)		All women ($N = 245$)	Smokers ($n = 173$)
	M	*SD*	*M*	*SD*	*M*	*SD*
Partner smoking status	57% (yes)	n/a	71% (yes)	n/a	22% (yes)	n/a
Does partner smoke in the woman's home	44% (yes)	n/a	56% (yes)	n/a	14% (yes)	n/a
Number of relatives who smoke	2.47	0.94	2.70	0.90	2.01	0.88
Number of friends who smoke	2.42	1.10	2.75	1.03	1.63	0.83
During past 7 days how often exposed to smoke in a room	2.84	1.66	3.42	1.59	1.46	0.73
During past 7 days how often exposed to smoke in an auto	1.93	1.36	2.21	1.48	1.25	0.65
During past 7 days how often exposed to smoke outside	2.65	1.68	3.17	1.69	1.40	0.71
Average frequency of SHS exposure	2.48	1.26	2.94	1.19	1.37	0.55

Note. *n/a* = not applicable.

Table 2. Bivariate Correlations Among All Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Women's smoking status	—										
2. Partner smoking status	.45*	—									
3. Partner smoke in home	.39*	.77*	—								
4. Partner living status	.02	.10	−.12	—							
5. Number of smokers live in home excluding partner	.19*	.14*	.22*	.02	—						
6. Number of relatives who smoke	.31*	.19*	.15*	.06	.04	—					
7. Number of friends who smoke	.47*	.25*	.23*	.00	.08	.31*	—				
8. SHS exposure room	.54*	.40*	.49*	.05	.25*	.25*	.40*	—			
9. SHS exposure auto	.32*	.31*	.26*	.08	.21*	.25*	.30*	.54*	—		
10. SHS exposure outside	.48*	.30*	.19*	.00	.03	.25*	.28*	.47*	.41*	—	
11. General SHS exposure	.57*	.42*	.39*	.05	.20*	.31*	.41*	.84*	.78*	.80*	—

Note. $N = 245$, $^*p < .05$.

Predicting Frequency of SHS Exposure From Specific Sources of SHS Exposure

A 3-step multiple regression analysis was conducted in which frequency of SHS exposure was regressed on women's age, women's smoking status, partner living status, partner smoking status, number of smokers in the household, excluding partners, number of friends who smoke, number of relatives who smoke, and the proposed two-way interactions. Overall, the model was significant, as this model accounted for 41% of the variability in frequency of SHS exposure among pregnant women ($F_{(11,233)} = 14.21$, $p < .001$). Examination of the residuals indicated that the assumptions of normality and homoscedasticity were tenable for this model. Results of this regression model are presented in Table 3.

From Table 3, it is clear that after accounting for the effects of women's smoking status and age, there was a significant association between partner smoking status and frequency of SHS exposure among pregnant women, such that women who reported being in a relationship with a smoking partner reported higher levels of frequency of SHS exposure compared to those with nonsmoking partners. The number of friends and the number of relatives who smoked were also each positively associated with frequency of SHS exposure. The magnitude of the effects was similar to that of partner smoking, suggesting that sources other than partner smoking may also be important with regard to SHS exposure among pregnant women. None of the interaction terms were significant.

Degree of SHS Exposure Misclassification Utilizing Partner Smoking as the Sole Measure of SHS Exposure

Cross-tabulations were computed to determine the degree of misclassification for SHS exposure when using partner smoking as the sole measure of SHS exposure. From Table 4, it is clear that a substantial proportion (69% or 73/106) of pregnant women whose partners were nonsmokers reported that they were exposed to SHS in the past week. More specifically, 53% of pregnant women whose partners were nonsmokers reported that they were in the same room as someone who was smoking in the past week. Similarly, 26% of pregnant women whose partners were nonsmokers reported that they were in an automobile with someone who was smoking during the past week and 45% reported that they were outside with someone who was smoking. Perhaps, even more striking is the finding that even among the 56 nonsmoking women with nonsmoking partners, 50% reported SHS exposure in the past week.

Table 3. Summary of the Multiple Regression Analyses Predicting Average Frequency of SHS Exposure

Entry	Variable	Beta	ΔR^2	*F* change	*CI*
Step 1	Women's smoking status	0.57**	0.33	117.01	1.29 to 1.86
Step 2	Partner smoking status	0.17**			0.15 to 0.72
	Partner living status	0.03			−0.18 to 0.33
	Number of relatives who smoke	0.12*			0.02 to 0.31
	Number of friends who smoke	0.16*			0.06 to 0.32
	Number of smokers living in the household	0.09	0.08	6.32	−0.03 to 0.61
Step 3	Partner smoke × living status	−0.03			−0.75 to 0.41
	Partner smoke × women smoke	0.17			−0.26 to 1.04
	Women smoke × living status	−0.04	0.01	0.67	−0.67 to 0.58

Note. $N = 245$. *CI*: 95% *CI*s of the unstandardized regression coefficient.
$^*p < .05$, $^{**}p < .01$.

Table 4. General SHS Exposure With Respect to Partner Smoke Exposure Among Pregnant Women

	Entire sample ($N = 245$)		Nonsmokers ($n = 72$)		Smokers ($n = 173$)	
	Nonsmoking partner $N = 106$	Smoking partner $N = 139$	Nonsmoking partner $N = 56$	Smoking partner $N = 16$	Nonsmoking partner $N = 50$	Smoking partner $N = 123$
Exposed to SHS in a room over past week (%)	53	81	30	56	80	84
Exposed to SHS in an auto over past week (%)	**26**	55	**16**	25	**38**	58
Exposed to SHS outside over the past week (%)	45	72	29	31	64	78
Any exposure to SHS in past week (%)	**69**	91	**50**	62	**90**	95

Note. Numbers in bold indicate that a substantial number of women with non-smoking partners were exposed to SHS during pregnancy.

Changes in SHS Exposure During Pregnancy

Results from repeated measures ANOVA indicated that frequency of SHS exposure did not change throughout the women's pregnancy ($F_{(1.96,201.46)} = 0.37$, $p = .69$, $\eta^2 = 0.00$). There was no effect of time on frequency of SHS exposure.

Discussion

Results indicated that the most common source of SHS exposure during pregnancy was the partner. However, an additional 19% of women lived with at least one smoker who was not the partner. Indeed, reliance on the partner smoking measure alone would have misclassified a substantial number of women as having no SHS exposure during pregnancy. The results are similar to those reported by Edwards (2009) using a large cohort of women cancer patients, indicating that 41% of women had been exposed to SHS in their home in their lifetime from sources other than their spouse or parent. Pregnant smokers were more likely to be exposed to SHS compared to nonsmokers, and as would be expected, the different measures of SHS exposure were moderately associated with each other. After accounting for women's own smoking status, partner smoking, relatives, and friends' smoking were all predictive of the frequency of SHS exposure. The magnitude of the associations between relatives/friends smoking and frequency of SHS exposure was similar to that of partner smoking. Thus, all three sources of exposure are important predictors of frequency of SHS exposure, after accounting for women's own smoking status. The importance of other members of the social network is also evident in the finding that among nonsmoking women with nonsmoking partners, 50% reported some level of SHS exposure in the preceding week. Results indicate that more comprehensive measures of SHS exposure that include different potential sources of exposure as well as frequency of exposure in different contexts are important to accurately assess SHS exposure than just the measure of partner smoking or other single indicator measures of SHS during pregnancy.

Contrary to expectations, there were no changes in SHS exposure across the three trimesters. Pregnant smokers and those with smoking partners were exposed to higher levels of SHS throughout pregnancy. Few previous studies have examined potential changes in SHS exposure. Moreover, the sample size for these analyses was restricted to women with complete data through all three trimesters of pregnancy. Thus, the results need to be viewed with caution and replicated before further discussion. However, if these results hold up, they indicate that in spite of general declines in women's own smoking through pregnancy (Bailey, Hill, Hawkins, Catalano, and Abbott, 2008; Carmichael and Ahluwalia, 2000), SHS exposure may remain constant and may have negative health consequences for both maternal and fetal health. Indeed, it may be unrealistic to expect cessation efforts to be successful in the face of continued and consistent SHS exposure during pregnancy.

A number of studies indicate that adult smokers who have lower SHS exposure (e.g., live in smoke-free homes) have more quit attempts and are more likely to remain abstinent than smokers with SHS exposure in the home (see Mills, Messer, Gilpin, and Pierce, 2009, review). Similarly, in a large cohort of pregnant smokers, women's failure to quit or reduce smoking was associated with having a partner who did not quit or reduce smoking and with a higher number of smokers in their social network (Appleton and Pharoah, 1998). In their discussion of the role of smokers in the household in fostering continued smoking during pregnancy, Kahn, Certain, and Whitaker (2002) cited studies in the general adult population indicating that exposure to contextual smoking cues increases the desire to smoke. Continued smoking among friends and other social network members may serve to model undesirable behaviors, especially if these friends and relatives continue to smoke during pregnancy. Thus, continued smoking among relatives and friends and permissive household policies regarding smoking may serve as a barrier to quitting during pregnancy by providing easy access to cigarettes, enhance craving, and provide a context in which smoking during pregnancy is viewed as normative. Continued SHS exposure through pregnancy may also serve to reduce any initial motivation to quit smoking during pregnancy and maintain women's own smoking by lowering motivation to quit. Recent studies have demonstrated that motivation to quit is dynamic, with frequent fluctuations (West and Sohal, 2006), and lack of motivation to quit or declines in motivation predict relapse prospectively (McBride, Pirie, and Curry, 1992; Shiffman, Paty, Gnys, Kassel, and Hickcox, 1996). These results speak to the need to focus on sources of SHS exposure in smoking cessation interventions for pregnant women (Mullen, 2004) and offer cessation interventions to sources of SHS exposure in the women's lives.

This study has several limitations. First is the issue of restricted sample size for examination of changes in SHS exposure, as noted above. Second, the results may only be generalizable to primarily lower socioeconomic status (SES)

smokers with high school or below high school education. It is possible that sources and frequency of SHS exposure through pregnancy differ for higher SES women with different demographic characteristics. However, it should be noted that smoking during pregnancy is more common among younger, lower income women with less education (Gilman, Abrams, and Buka, 2003), suggesting that this may be a particularly important population with respect to pregnancy smoking. Another limitation is that the initial set of 42 oral fluid samples were assayed using ELISA, a less sensitive assay for cotinine. Thus, it is possible that of these 42 women, some were active smokers and were misclassified as nonsmokers in the first trimester. However, oral fluid samples were obtained in each trimester of pregnancy, and ELISA assay was not used beyond the first trimester for these 42 women. On a related note, there may be some concern about potential bias in the cutoffs used for LC–MSMS. The 5 ng/ml cutoff for LC–MSMS was used to discriminate women who were designated as active smokers based on oral fluid samples from nonsmokers. There were only three women who had cotinine levels below this cutoff, and all had been assigned to the smoking group based on self-report. Thus, it is unlikely that the cigarette smoking mothers or those exposed to SHS were assigned to the nonexposed group because of the 5 ng/ml cutoff.

The sample included in the analysis of change in SHS exposure over time was limited to 106 women who had completed all three trimester interviews. Although we examined differences between women with complete data compared to those who had not yet completed all three interviews, there is always the possibility that this group differed from the overall sample on some unmeasured variables. Another limitation was that our measure of SHS exposure was based on number of days of exposure in different contexts, and it is possible that number of hours of exposure is a better indicator of actual exposure. Number of hours of exposure would account for variations from weekdays to weekend days and may provide a more fine-grained measure of SHS exposure than days of exposure. Such a fine-grained approach may yield different conclusions about the sources of SHS exposure, and therefore, this may be a useful direction for future research. Finally, although we examined individual, group, and social network and organization (work)-level influences on SHS exposure, we did not examine other sources of influence suggested by social–ecological theory such as community and population contexts. Community-level influences such as neighborhoods and population-level influences such as taxation and pricing could have influenced exposure to SHS during pregnancy but were unmeasured variables in the study.

Overall findings suggest that reliance on partner smoking alone would underestimate exposure to SHS during pregnancy. Women's reports about the number of relatives and friends who smoke accounted for unique variance in frequency of SHS exposure. It is also important to note that frequencies of SHS exposure in different contexts (room, car, and outside) were only moderately associated with each other. Thus, each context provided important information about the total average frequency of SHS exposure during pregnancy.

Funding

National Institute on Drug Abuse at the National Institutes of Health (R01DA013190).

Declaration of Interests

None declared.

Acknowledgments

We are grateful to the pregnant women who participated in this study and research staff who were responsible for data collection. Special thanks to Dr. Claire Coles and Dr. Nancy Day for consultation on this project. We are extremely grateful to Dr. Amol Lele at Women and Children's Hospital of Buffalo for collaboration on data collection.

References

Appleton, P. L., & Pharoah, P. O. D. (1998). Partner smoking behaviour change is associated with women's smoking reduction and cessation during pregnancy. *British Journal of Health Psychology*, *3*, 361–374.

Bailey, J. A., Hill, K. G., Hawkins, J. D., Catalano, R. F., & Abbott, R. D. (2008). Men's and women's patterns of substance use around pregnancy. *Birth*, *35*, 50–59. doi:10.1111/j.1523-536x.2007.00211.x

Brown, R., Burgess, E. S., Sales, S. D., Whiteley, J. A., Evans, D. M., & Miller, I. (1998). Reliability and validity of a smoking timeline. *Psychology of Addictive Behaviors*, *12*, 101–112. doi:10.1037/0893-164x.12.2.101

Carmichael, S. L., & Ahluwalia, I. B. (2000). Correlates of postpartum smoking relapse results from the pregnancy risk assessment monitoring system (PRAMS). *American Journal of Preventive Medicine*, *19*, 193–196. doi:10.1016/so749-3797(00)00198-7

Corbett, K. K. (2001). Susceptibility of youth to tobacco: A social ecological framework for prevention. *Respiration Physiology*, *128*, 103–118. doi:10.1016/s0034-5687(01)00269-9

Edwards, R. (2009). Spousal smoking as an indicator of total secondhand smoke exposure. *Nicotine and Tobacco Research*, *11*, 606–613. doi:10.1093/ntr/ntp024

Ennett, S. T., Foshee, V. A., Bauman, K. E., Hussong, A., Faris, R., Hipp, J. R., Cai, L. et al. (2010). A social contextual analysis of youth cigarette smoking development. *Nicotine and Tobacco Research*, *12*, 950–962. doi:10.1093/ntr/ntq122

Gilman, S. E., Abrams, D. B., & Buka, S. L. (2003). Socioeconomic status over the life course and stages of cigarette use: initiation, regular use, and cessation. *Journal of Epidemiology and Community Health*, *57*, 802–808. doi:10.1136/jech.57.10.802

Gray, T. R., Eiden, R. D., Leonard, K. E., Connors, G., Shisler, S., & Huestis, M. A. (2010). Nicotine and metabolites in meconium as evidence of maternal cigarette smoking during pregnancy and predictors of neonatal growth deficits. *Nicotine and Tobacco Research*, *12*, 658–664. doi:10.1093/ntr/ntq068

Green, L. W., Richard, L., & Potvin, L. (1996). Ecological foundations of health promotion. *American Journal of Health Promotion*, *10*, 270–281.

Hanke, W., Sobala, W., & Kalinka, J. (2004). Environmental tobacco smoke exposure among pregnant women: Impact on fetal biometry at 20-24 weeks of gestation and newborn child's birth weight. *International Archives of Occupational and Environmental Health*, *77*, 47–52. doi:10.1007/s00420-003-0475-0

Jaddoe, V. W. V., Troe, E-J E W M., Hofman, A., Mackenbach, J. P., Moll, H. A., Steegers, E. A. P., et al. (2008). Active and passive maternal smoking during pregnancy and the risks of low birthweight and preterm birth: The Generation R Study. *Paediatric and Perinatal Epidemiology*, *22*, 162–171. doi:10.1111/j.1365-3016.2007.00916.x

Jedrychowski, W., Galas, A. S., Flak, E., Jacek, R., Penar, A., Spengler, J., et al. (2007). Increased burden of respiratory disease in the first six months of life due to prenatal environmental tobacco smoke: Krakow birth cohort study. *Early Child Development and Care*, *177*, 369–381. doi:10.1080/03004430500507719

Kahn, R. S., Certain, L., & Whitaker, R. C. (2002). A reexamination of smoking before, during, and after pregnancy. *American Journal of Public Health*, *92*, 1801–1808. doi:10.2105/AJPH.92.8.1312

Leonardi-Bee, J., Smyth, A., Britton, J., & Coleman, T. (2008). Environmental tobacco smoke and fetal health: Systematic review and meta-analysis. *Archives of Disease in Childhood. Fetal and Neonatal Edition*, *93*, F351–F361. doi:10.1136/adc.2007.133553

McBride, C. M., Pirie, P. L., & Curry, S. J. (1992). Postpartum relapse to smoking: A prospective study. *Health Education Research*, *7*, 381–390. doi:10.1093/her/7.3.381

Mills, A. L., Messer, K., Gilpin, E. A., & Pierce, J. P. (2009). The effect of smoke free homes on adult smoking behavior: A review. *Nicotine and Tobacco Research*, *11*, 1131–1141. doi:10.1093/ntr/ntp122

Mullen, P. D. (2004). How can more smoking suspension during pregnancy become lifelong abstinence? Lessons learned about predictors, interventions, and gaps in our accumulated knowledge. *Nicotine and Tobacco Research*, *6*(Suppl. 2), S217–S238. doi:10.1080/14622200410001669150

Munafò, M. R., Heron, J., & Araya, R. (2008). Smoking patterns during pregnancy and postnatal period and depressive symptoms. *Nicotine and TobaccoResearch*, *10*, 1609–1620. doi:10.1080/14622200802412895

Newman, R. B., Momirova, V., Dombrowski, M. P., Schatz, M., Wise, R., Landon, M., et al. (2010). The effect of active and passive household cigarette smoke exposure on pregnant women with asthma. *Chest*, *137*, 601–608. doi:10.1378/chest.09-0942

Pogodina, C., Brunner Huber, L. R., Racine, E. F., & Platonova, E. (2009). Smoke-free homes for smoke-free babies: The role of residential environmental tobacco smoke on low birth weight. *Journal of Community Health*, *34*, 376–382. doi:10.1007/s10900-009-9169-1

Rise, J., & Lund, K. E. (2005). Predicting children's level of exposure to environmental tobacco smoke based on two national surveys in Norway in 1995 and 2001. *Addictive Behaviors*, *30*, 1267–1271. doi:10.1016/j.addbeh.2004.12.008

Shiffman, S., Paty, J. A., Gnys, M., Kassel, J. A., & Hickcox, M. (1996). First lapses to smoking: Within-subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology*, *64*, 366–379. doi:10.1037/0022-006x.64.2.366

Sobell, M. B., Sobell, L. C., Klajmer, F., Pavan, D., & Basian, E. (1986). The reliability of a timeline method for assessing normal drinker college students' recent drinking history. *Addictive Behaviors*, *11*, 149–162. doi:10.1016/0306-4603(86)90040-7

Spears, G. V., Stein, J. A., & Koniak-Griffin, D. (2010). Latent growth trajectories of substance use among pregnant and parenting adolescents. *Psychology of Addictive Behaviors*, *24*, 322–332. doi:10.1037/a0018518

Stokols, D. (1996). Translating social ecological theory into guidelines for community health promotion. *American Journal of Health Promotion*, *10*, 282–298.

West, R., & Sohal, T. (2006). "Catastrophic" pathways to smoking cessation: Findings from national survey. *British Medical Journal*, *332*, 458–460. doi:10.1136/bmj.38723.573866.AE

Windham, G. C., Eaton, A., & Hopkins, B. (1999). Evidence for an association between environmental tobacco smoke exposure and birth weight: A meta-analysis and new data. *Paediatric and Perinatal Epidemiology*, *13*, 35–57. doi:10.1046/j.1365-3016.1999.00150x

Yolton, K., Khoury, J., Hornung, R., Dietrich, K., Succop, P., & Lanphear, B. (2008). Environmental tobacco smoke exposure and child behaviors. *Journal of Developmental and Behavioral Pediatrics*, *29*, 450–457. doi:10.1097/DBP.06013e31818d0c21