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Intimate Partner Violence, Small for Gestational Age Birth and Cigarette Smoking in the Pregnancy Risk Assessment Monitoring System

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Abstract

Background: Exposure to intimate partner violence (IPV) in the perinatal period is associated with obstetric complications, poor maternal mental health, neonatal complications, and increased risk of infant mortality and morbidity. Less is known about how IPV may influence small for gestational age (SGA) birth.
Materials and Methods: Data were obtained for 231,081 United States mothers who delivered neonates from 2004 to 2011 and completed the Pregnancy Risk Assessment Monitoring System survey 2–9 months after delivery. Weighted descriptive statistics and multivariate logistic regression models were used.
Results: IPV in the year before or during pregnancy was related to SGA bivariately (odds ratio 1.39, 95% confidence interval [CI] 1.28, 1.51), and after adjustment for demographic and obstetric factors, this association attenuated after further adjustment for perinatal smoking patterns, (adjusted odds ratio [aOR] 1.06, 95% CI 0.97, 1.15). Compared with nonabused women, women experiencing perinatal IPV were more than twice as likely to smoke before pregnancy (aOR 2.34, 95% CI 2.19, 2.49), and nearly 1.5 times as likely to report sustained smoking into the last 3 months of pregnancy (aOR 1.45, 95% CI 1.32, 1.59). In turn, among prepregnancy smokers, sustained smoking was associated with delivery of a SGA neonate (aOR 1.87, 95% CI 1.72, 2.03), fully attenuating the association of perinatal IPV with SGA.
Conclusion: Women who experienced perinatal IPV were significantly more likely to smoke prepregnancy and sustain smoking into the last 3 months of pregnancy. Through behavioral and physiological pathways, smoking cessation may be uniquely challenging for women experiencing IPV, yet critical to address clinically to mitigate risk for SGA.

Keywords: intimate partner violence, smoking, reproductive health

Introduction

Intimate partner violence (IPV) is a significant public health issue, affecting an estimated 1.5 million women in the United States each year.[1,2] Women of childbearing age in particular are at highest risk of IPV with nearly 15% of women aged 18–24 experiencing rape, physical violence, or stalking by an intimate partner within the past year. In the United States, ~4%–8% of women experience IPV during pregnancy, with well-established factors (*e.g.*, young age, low education, race/ethnicity, poverty) placing some women at significantly increased risk.[2–4]

Exposure to IPV in the perinatal period, defined here as 12 months before and during pregnancy, is associated with obstetric complications,[4,5] poor mental health,[4,6,7] and increased risk of infant mortality and morbidity.[4,8] Less is known about how IPV may influence small for gestational age (SGA) birth. Infants born SGA have increased morbidity and mortality,[9] and suffer increased risk of cognitive delays and socioemotional problems in childhood.[10] Additionally, the consequences of SGA birth may extend into adulthood with increased risk of coronary heart disease, obesity, and diabetes.[11,12]

Limited research demonstrates that perinatal IPV may be a risk factor for SGA, although results are mixed, in part,

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reflecting a complex relationship with substance use. Where IPV is associated with SGA, causal pathways appear to be confounded or potentially mediated by substance use. In a Canadian sample ($n = 4{,}750$), IPV during pregnancy was significantly associated with SGA; this association attenuated after controlling for substance use.[13] By contrast, IPV was significantly associated with SGA even after controlling for self-reported substance use in an urban United States sample of predominantly low-income women.[14] In a community sample of low-income perinatal women living in rural and urban areas in the United States, SGA risk increased with increases in IPV severity.[8]

Smoking may serve to mediate or explain, rather than confound, the relationship between IPV and SGA. In a recent population-based analysis of pregnant women, experiencing IPV was associated with significantly higher rates of prepregnancy smoking, and sustained smoking.[15] The strength of the relationship between IPV victimization and cigarette smoking has found a stronger association in pregnant women as compared with nonpregnant women.[16] In animal models, cotinine, the main metabolite of nicotine, is associated with decreased anxiety and depressive-like behavior in mouse models of post-traumatic stress disorder (PTSD).[17] Smokers with trauma-related symptoms have more withdrawal symptoms and are more likely to relapse compared with smokers without a trauma history.[18] Furthermore, several studies suggest a graded relationship between trauma severity, current smoking, heavy smoking, and initiating smoking at a young age, all suggesting a mediating role of smoking.[19–23]

In turn, maternal smoking during pregnancy is one of the most common preventable causes of infant morbidity and mortality.[24,25] Maternal smoking increases risk for pregnancy complications and adverse neonatal outcomes (*e.g.*, preterm delivery, low birthweight, SGA, congenital malformations, and sudden infant death syndrome).[24,25] Indeed, delivery of a SGA neonate is one of the most well-documented adverse outcomes associated with smoking during pregnancy.[26]

Taken together, our understanding of the association between IPV and SGA is limited, and research in the United States is largely drawn from community samples of perinatal women. Thus, the population-level patterns of IPV and SGA birth remain unclear. We advance this knowledge base through analysis of United States population-based data to examine the association between perinatal IPV and SGA birth while exploring the mediating role of perinatal smoking patterns.

Materials and Methods

Data were obtained from the 2004 to 2011 Pregnancy Risk Assessment Monitoring System (PRAMS), an ongoing multistate surveillance project conducted by the United States Centers for Disease Control and Prevention (CDC) in collaboration with participating state health departments. As per CDC guidelines, data are released for states meeting a minimum response rate, specifically ≥70% for 2004–2006, and ≥65% for 2007–2011. Our analysis includes data from New York City and 35 states.

PRAMS utilizes stratified systematic random sampling of 100–250 mothers who delivered a live birth from participating states each month, employing birth certificates as the sampling frame, with oversampling of high-risk populations, including women who delivered low birthweight neonates. Questionnaires are mailed to participants 2–4 months postpartum, with three additional attempts through mail before telephone contact is made. Self-reported survey responses are linked to birth certificate data. Weighting accounts for survey design, nonresponse, and noncoverage. Further information is available on PRAMS methodology.[27]

The exposure variable, perinatal IPV, was self-reported. Participants were asked if they were ''pushed, hit, slapped, kicked, choked, or physically hurt'' by a current or ex-husband or partner. To ensure that perinatal IPV exposure was temporally before delivery, the referent period for perinatal was limited to the 12 months before pregnancy or during pregnancy. SGA was derived from birth certificate data, and defined as birthweight <10th percentile adjusted for gestational age, sex, and maternal race/ethnicity. Confounders drawn from birth certificate data included maternal age, race/ethnicity, educational level, adequacy of prenatal care as per the Kotelchuck Index,[28] and gestational weight gain, categorized as inadequate, normal, or excessive, taking into account prepregnancy body mass index, as per 2009 Institute of Medicine guidelines.[29] Self-reported PRAMS data provided additional variables, including income, categorized as <100%, 101%–200%, or >200% of the federal poverty guideline by year and family size, and perinatal smoking.

Perinatal cigarette smoking, and its timing before and during the pregnancy, was assessed as follows: A PRAMS survey item asked participants if they smoked at least 100 cigarettes in the last 2 years. If not, they skipped the remaining smoking-related questions. Participants who smoked at least 100 cigarettes in the last 2 years were asked how many cigarettes they smoked on an average day during the 3 months before they were pregnant and during the last 3 months of pregnancy. For both questions, response options were categorical and included ''Less than one cigarette'' and ''I didn't smoke then.'' In the present analysis, respondents who did not smoke at least 100 cigarettes in the last 2 years, or those who reported not smoking during the 3 months before pregnancy and the last 3 months of pregnancy, were classified as nonsmokers. Respondents who smoked any number of cigarettes (including <1 per day) during the 3 months before pregnancy but did not smoke during the last 3 months of pregnancy were classified as having quit smoking during pregnancy. Respondents who reported smoking during the last 3 months of pregnancy (including <1 cigarette per day) were classified as experiencing sustained smoking into late pregnancy.

Prevalence and 95% confidence intervals (CIs) for perinatal IPV were calculated for the total population and by demographics and pregnancy-related behavioral risks assessed. Significance of bivariate associations with perinatal IPV was assessed based on the p value of the Pearson Chi-squared statistic with Rao and Scott's correction for survey design.[30] Significance for all analyses was set at $p < 0.05$. Bivariate associations with SGA birth were assessed using weighted crude odds ratios (ORs) and corresponding 95% CIs.

To assess potential mediation of the IPV–SGA association by perinatal smoking pattern in the full population, the effect estimate and associated 95% CI were compared in two logistic regression models: (i) adjusted for demographic and obstetric variables (age, educational level, income, adequacy of prenatal care, and gestational weight gain); and (ii) adjusted for perinatal smoking pattern in addition to

demographic and obstetric variables. Similarly, to assess the role of perinatal smoking pattern in the IPV–SGA association specifically among prepregnancy smokers, the previously described models were fitted in the analytic subpopulation limited to prepregnancy smokers. The effect estimate and associated 95% CI of the IPV–SGA association were compared before and after adjustment for sustained smoking. Maternal race/ethnicity was not included in multivariate models because SGA as operationalized by PRAMS is already adjusted for race. Mediation analyses utilized women's reports of timing of exposures within the cross-sectional data to approximate temporal data. Sensitivity analyses examined whether results differed by PRAMS phase (2004–2008 or 2009–2011). Sensitivity analyses also examined the effect of adjusting for alcohol consumption during late pregnancy (categorized as none, <1 drink per week, 1–6 drinks per week, or 7 or more drinks per week; the PRAMS survey does not inquire about alcohol consumption during the first 6 months of pregnancy.)

Of a total 323,926 participants, 2.3% ($n=7,336$) were excluded for lack of data on IPV, an additional 7.7% ($n=24,408$) lacked SGA data, and of those remaining, 20.9% ($n=61,101$) were excluded for missing data on one or more variables of interest for a final sample size of 231,081. As per the complex sample survey design of the PRAMS, all analyses were weighted and conducted using STATA 13.0. The Johns Hopkins Medical Institutional Review Board reviewed the study protocol, and qualified the study as exempt research given its utilization of publicly available, deidentified surveillance data.

Results

Prevalence of IPV and relations to demographics, behavioral risk factors, and SGA

Demographic and obstetric sample characteristics, overall and by IPV exposure status, are shown in Table 1.

Table 1. Maternal Characteristics and Small for Gestational Age Birth by Perinatal Intimate Partner Violence Exposure

	No perinatal IPV	*Perinatal IPV*	*Total*
Observations	216,066	15,015	231,081
Weighted counts	10,421,207	624,377	11,045,584
% of population	94.3 (94.2, 94.5)	5.7 (5.5, 5.8)	100.0
Column percent (95% CI)[a]			
SGA birth[b]			
No	90.9 (90.7, 91.0)	87.7 (86.9, 88.6)	90.7 (90.5, 90.9)
Yes	9.1 (9.0, 9.3)	12.3 (11.4, 13.1)	9.3 (9.1, 9.5)
Age[b]			
<20	7.0 (6.8, 7.1)	16.4 (15.4, 17.5)	7.5 (7.3, 7.7)
20–24	22.2 (22.0, 22.5)	38.2 (36.9, 39.6)	23.1 (22.9, 23.4)
25–29	29.9 (29.6, 30.2)	25.7 (24.5, 26.9)	29.7 (29.4, 30.0)
≥30	40.9 (40.6, 41.2)	19.7 (18.6, 20.8)	39.7 (39.4, 40.0)
Race/ethnicity[b]			
White or Other NH	67.7 (67.4, 68.0)	54.4 (53.0, 55.7)	66.9 (66.7, 67.2)
Black NH	12.8 (12.6, 13.0)	23.5 (22.4, 24.6)	13.4 (13.2, 13.6)
Hispanic	13.8 (13.6, 14.0)	18.0 (16.8, 19.2)	14.0 (13.8, 14.2)
AI/AN NH	0.9 (0.9, 0.9)	1.7 (1.5, 1.9)	0.9 (0.9, 1.0)
Asian NH	4.8 (4.7, 4.9)	2.5 (2.2, 2.8)	4.7 (4.6, 4.8)
Education			
<12 years	13.0 (12.8, 13.2)	25.0 (23.8, 26.2)	13.7 (13.4, 13.9)
≥12 years	87.0 (86.8, 87.2)	75.0 (73.8, 76.2)	86.3 (86.1, 86.6)
Income—% federal poverty guidelines[b]			
<100	31.0 (30.7, 31.4)	65.4 (64.1, 66.8)	33.0 (32.7, 33.3)
101–200	21.1 (20.9, 21.4)	19.8 (18.7, 20.9)	21.1 (20.8, 21.3)
≥200	47.8 (47.5, 48.1)	14.8 (13.9, 15.8)	45.9 (45.6, 46.3)
Perinatal smoking pattern[b]			
Nonsmoker	77.3 (77.0, 77.6)	52.2 (50.8, 53.6)	75.9 (75.6, 76.1)
Quit during pregnancy	11.5 (11.2, 11.7)	17.6 (16.6, 18.7)	11.8 (11.6, 12.0)
Sustained smoking	11.3 (11.1, 11.5)	30.1 (28.9, 31.4)	12.3 (12.1, 12.6)
Adequacy of prenatal care[b]			
Inadequate	10.4 (10.1, 10.6)	18.7 (17.7, 19.8)	10.8 (10.6, 11.1)
Intermediate	13.5 (13.2, 13.7)	14.1 (13.1, 15.1)	13.5 (13.3, 13.7)
Adequate	47.1 (46.7, 47.4)	39.4 (38.1, 40.8)	46.6 (46.3, 47.0)
Adequate plus	29.1 (28.8, 29.4)	27.8 (26.6, 29.0)	29.0 (28.7, 29.3)
Gestational weight gain[b]			
Inadequate	20.3 (20.0, 20.5)	22.4 (21.3, 23.5)	20.4 (20.1, 20.7)
Normal	33.7 (33.4, 34.0)	30.5 (29.3, 31.8)	33.5 (33.2, 33.8)
Excessive	46.1 (45.7, 46.4)	47.1 (45.7, 48.5)	46.1 (45.8, 46.4)

[a]All percentages and test statistics reported are from survey-weighted procedures.
[b]Design-based F statistic $p<0.0001$.
SGA, small for gestational age; IPV, intimate partner violence; CI, confidence interval; AN, Alaska Native; AI, American Indian; NH, Non-Hispanic.

Approximately 1 in 17 (5.7%) mothers delivering live neonates reported experiencing perinatal IPV. Younger women were at greatest risk for perinatal IPV with 54.6% of women experiencing perinatal IPV aged 24 and younger. Women who experienced perinatal IPV were more likely to be non-Hispanic Black, Hispanic, or American Indian/Alaska Native compared with those who did not experience perinatal IPV. Women who experienced perinatal IPV were also more likely to have received less than a high school education, and report an income <100% of the federal poverty guidelines. Nearly 12.3% of women who experienced perinatal IPV delivered a neonate classified as SGA, compared with 9.1% of women who did not experience perinatal IPV ($p < 0.0001$). Women experiencing perinatal IPV were more likely to report inadequate prenatal care and inadequate gestational weight gain. All demographic and obstetric variables were significantly associated with perinatal IPV ($p < 0.0001$).

Perinatal smoking patterns differed by experiences of perinatal IPV ($p < 0.0001$). Nearly 48% of women who experienced perinatal IPV, compared with 22.7% of nonabused women, reported smoking during the perinatal period (3 months before becoming pregnant and/or the last 3 months of pregnancy). Among women who reported sustained smoking into the last 3 months of pregnancy, 30.1% experienced perinatal IPV, compared with 11.3% of nonabused women. After adjustment for demographic and obstetric variables, women who experienced perinatal IPV had 2.27 (95% CI 2.12, 2.42) times the odds of smoking before pregnancy compared with women who did not experience perinatal IPV. Among prepregnancy smokers, women who experienced perinatal IPV had 1.52 (95% CI 1.38, 1.66) times the adjusted odds of sustained smoking into late pregnancy, rather than quitting smoking during pregnancy, compared with women who did not experience perinatal IPV (data not shown).

Predictors of SGA birth

In bivariate logistic regression analyses, perinatal IPV was associated with SGA (OR 1.39, 95% CI 1.28, 1.51; Table 2). Perinatal smoking pattern had a significant bivariate association with SGA birth, driven by sustained smoking: compared with 8.2% of nonsmokers and 8.7% of those who quit smoking during pregnancy, nearly 17% of women who reported sustained smoking into late pregnancy delivered a neonate classified as SGA. In the logistic model adjusted for maternal age, education, income, adequacy of prenatal care, and gestational weight gain, the association between IPV and SGA remained significant with an adjusted odds ratio (aOR) 1.18 (95% CI 1.08, 1.28). After additionally adjusting for perinatal smoking pattern, the association of IPV with SGA attenuated to nonsignificance (aOR 1.05, 95% CI 0.97, 1.15), whereas sustained smoking was associated with 2.06 (95% CI 1.95, 2.18) times the adjusted odds of SGA birth as compared with nonsmoking.

Illustrated solely among prepregnancy smokers, women experiencing perinatal IPV were 1.15 times more likely to deliver a neonate classified as SGA as compared with their nonabused counterparts after adjusting for maternal age, education, income, adequacy of prenatal care, and gestational weight gain (aOR 1.15, 95% CI 1.03, 1.29). After further adjusting for sustained smoking in the last 3 months of pregnancy, the relationship between IPV and SGA among prepregnancy smokers was fully attenuated (aOR 1.10, 95% CI 0.98, 1.23), whereas sustained smoking was associated with 1.87 (95% CI 1.73, 2.03) times the adjusted odds of SGA birth (compared with quitting smoking during pregnancy). Results were not sensitive to PRAMS phase or adjustment for alcohol consumption during late pregnancy (data not shown).

Discussion

Using women's reports of exposures within the perinatal period, results demonstrate a significant association between perinatal IPV and SGA birth. A central finding of our study is the significant association between perinatal IPV and sustained smoking into late pregnancy and the resultant effect on delivery of an SGA neonate. Compared with nonabused women, women experiencing perinatal IPV were nearly 1.5 times more likely to smoke during the last 3 months of pregnancy. Smoking during late pregnancy, in turn, more than doubled a woman's odds of delivering a neonate classified as SGA. These findings are not explained by the higher rates of prepregnancy smoking among women who experience perinatal IPV.

In analyses limited to prepregnancy smokers, women who experienced perinatal IPV were significantly more likely to continue smoking until late pregnancy which in turn was associated with delivery of an SGA neonate. In both the full population and the subpopulation of prepregnancy smokers, the significant adjusted association of IPV and delivery of an SGA neonate was attenuated after addition of perinatal smoking pattern to the model. Taken together, our results identify a mediating role of maternal smoking in the IPV–SGA association, and illustrate that smoking cessation support for pregnant women is a critical component of mitigating the harm of IPV to women and their children.

Importantly, our results suggest that women experiencing perinatal IPV are significantly less likely to quit smoking early in pregnancy as compared with their nonabused counterparts. Women who have experienced IPV utilize various coping strategies in an attempt to mitigate the impact of abuse in their lives. Smoking is one such maladaptive coping strategy used to reduce symptoms of anxiety and depression. Qualitative research demonstrates that women describe the ''constant stress'' of violence as a significant impediment to smoking cessation.[31] Extant research supports stress response pathways as a plausible mechanism linking trauma symptoms and tobacco use.[18]

Research with animal models demonstrates that induced stress increases nicotine-seeking behavior.[32] Similarly, in nicotine-deprived smokers, exposure to a stress condition was associated with significantly higher tobacco cravings and relapse as compared with exposure to neutral condition.[33] Thus, women experiencing trauma-related symptoms represent a particularly vulnerable group for tobacco use disorder.

Limited research has demonstrated a significant association between IPV and SGA birth after controlling for maternal smoking, although findings were limited by small sample sizes, and high-risk samples.[8,14] Results extend past research demonstrating a link between maternal smoking and SGA birth,[34] by clarifying the role of maternal smoking pattern during the prenatal period. Our findings demonstrated that the prevalence of SGA was more than twice as high among women who sustained smoking into the last 3 months

Table 2. Predictors of Small for Gestational Age Birth in Bivariate and Multivariate Models

	Not SGA	*SGA*[a]	*OR (95% CI)*	*Full population; model 1*	*Full population; model 2*	*Prepregnancy smokers only; model 1*	*Prepregnancy smokers only; model 2*
Row percent (95% CI)[b]							
Perinatal IPV							
No	90.9 (90.7, 91.0)	9.1 (9.0, 9.3)	REF	REF	REF	REF	REF
Yes	87.7 (86.9, 88.6)	12.3 (11.4, 13.1)	1.39 (1.28, 1.51)	1.18 (1.08, 1.28)	1.05 (0.97, 1.15)	1.15 (1.03, 1.29)	1.10 (0.98, 1.23)
Age							
<20	87.0 (86.3, 87.8)	13.0 (12.2, 13.7)	1.70 (1.58, 1.83)	1.40 (1.28, 1.52)	1.40 (1.29, 1.53)	1.01 (0.88, 1.16)	1.11 (0.97, 1.28)
20–24	88.9 (88.4, 89.3)	11.1 (10.7, 11.6)	1.43 (1.36, 1.51)	1.27 (1.19, 1.35)	1.21 (1.14, 1.29)	1.02 (0.91, 1.13)	1.05 (0.95, 1.17)
25–29	91.3 (91.0, 91.6)	8.7 (8.4, 9.0)	1.09 (1.03, 1.15)	1.06 (1.01, 1.12)	1.03 (0.98, 1.09)	0.92 (0.83, 1.02)	0.94 (0.85, 1.04)
≥30	92.0 (91.7, 92.2)	8.0 (7.8, 8.3)	REF	REF	REF	REF	REF
Education							
<12 years	88.1 (87.6, 88.7)	11.9 (11.3, 12.4)	1.37 (1.29, 1.46)	1.05 (0.98, 1.12)	0.99 (0.93, 1.06)	1.15 (1.04, 1.27)	1.05 (0.95, 1.16)
≥12 years	91.1 (90.9, 91.3)	8.9 (8.7, 9.1)	REF	REF	REF	REF	REF
Income—% federal poverty guidelines							
<100	88.2 (87.9, 88.6)	11.8 (11.4, 12.1)	1.59 (1.52, 1.67)	1.33 (1.25, 1.41)	1.19 (1.12, 1.26)	1.40 (1.26, 1.55)	1.18 (1.06, 1.31)
101–200	91.0 (90.6, 91.4)	9.0 (8.6, 9.4)	1.18 (1.12, 1.25)	1.07 (1.01, 1.14)	1.01 (0.95, 1.07)	1.13 (1.01, 1.26)	1.01 (0.90, 1.13)
≥200	92.3 (92.0, 92.5)	7.7 (7.5, 8.0)	REF	REF	REF	REF	REF
Perinatal smoking pattern							
Nonsmoker	91.8 (91.6, 92.0)	8.2 (8.0, 8.4)	REF	—	REF	—	—
Quit during pregnancy	91.3 (90.7, 91.8)	8.7 (8.2, 9.3)	1.07 (1.00, 1.15)	—	1.09 (1.02, 1.18)	—	REF
Sustained smoking	83.1 (82.5, 83.8)	16.9 (16.2, 17.5)	2.28 (2.16, 2.40)	—	2.06 (1.95, 2.18)	—	1.87 (1.73, 2.03)
Adequacy of prenatal care							
Inadequate	89.4 (88.8, 90.0)	10.6 (10.0, 11.2)	1.24 (1.15, 1.33)	0.99 (0.92, 1.06)	0.98 (0.91, 1.05)	1.00 (0.89, 1.12)	0.98 (0.87, 1.10)
Intermediate	90.0 (89.4, 90.5)	10.0 (9.5, 10.6)	1.16 (1.09, 1.24)	1.10 (1.03, 1.18)	1.10 (1.03, 1.18)	1.14 (1.01, 1.29)	1.14 (1.01, 1.29)
Adequate	91.3 (91.0, 91.5)	8.7 (8.5, 9.0)	REF	REF	REF	REF	REF
Adequate plus	90.6 (90.3, 90.9)	9.4 (9.1, 9.7)	1.09 (1.03, 1.14)	1.06 (1.01, 1.11)	1.04 (0.99, 1.09)	1.03 (0.94, 1.12)	1.01 (0.93, 1.10)
Gestational weight gain							
Inadequate	85.8 (85.4, 86.3)	14.2 (13.7, 14.6)	1.49 (1.41, 1.57)	1.42 (1.35, 1.50)	1.41 (1.34, 1.49)	1.47 (1.34, 1.62)	1.43 (1.30, 1.57)
Normal	90.0 (89.7, 90.3)	10.0 (9.7, 10.3)	REF	REF	REF	REF	REF
Excessive	93.3 (93.1, 93.5)	6.7 (6.5, 6.9)	0.65 (0.61, 0.68)	0.63 (0.60, 0.66)	0.62 (0.59, 0.66)	0.58 (0.53, 0.64)	0.61 (0.56, 0.66)

[a]SGA as operationalized in PRAMS is adjusted for maternal race/ethnicity.
[b]All percentages, odds ratios, and confidence intervals reported are from survey-weighted procedures.
PRAMS, Pregnancy Risk Assessment Monitoring System.

of pregnancy as compared with nonsmokers in a population-based study. These findings are consistent with other research demonstrating that women who smoke during pregnancy have double the risk of fetal growth restriction as compared with nonsmokers.[34,35]

Taken together, our results suggest that public health initiatives and interventions focused on improving pregnancy outcomes must address both smoking cessation and perinatal IPV. Women who experience perinatal IPV are more likely to continue to smoke throughout pregnancy, and may have a more difficult time quitting smoking at any point during pregnancy.[15] Women experiencing perinatal IPV represent a high-priority population for smoking cessation, and treatment approaches should acknowledge the additional challenges in cessation for women experiencing trauma-related symptoms.

Importantly, many interventions targeting maternal smoking during pregnancy fail to address the complex reasons for women's smoking patterns. There are multiple barriers to smoking cessation, including psychosocial, cultural, socioeconomic, and biological factors that may be particularly salient in the perinatal period.[36] Thus, interventions must focus on psychosocial barriers to cessation, and our results demonstrate that perinatal IPV is a significant factor associated with both prepregnancy smoking, and sustained smoking into late pregnancy. Research demonstrates that women with posttraumatic stress disorder have higher rates of cigarette use than their nontraumatized counterparts,[20,37] suggesting an important role for implementing trauma-informed care.[38] Limited research has provided support for mindfulness and acceptance-based interventions for individuals experiencing posttraumatic stress disorder and tobacco use disorder.[39] This may be a promising treatment for women experiencing IPV and future research should evaluate the effectiveness of these treatments.

Universal screening for perinatal IPV is recommended by multiple organizations, including the United States Preventive Services Taskforce, the American College of Obstetricians and Gynecologists (the College), and the Association of Women's Health, Obstetric, and Neonatal Nurses (AWHONN).[40–42] Screening and counseling for IPV is effective in identifying women experiencing abuse, and may increase safety behaviors, decrease abuse, and improve maternal outcomes.[43,44] Women who screen positive for perinatal IPV should receive brief counseling during the visit as well as appropriate referrals to local organizations and support services with a detailed follow-up plan. Healthcare providers report shared barriers to screening and counseling for both smoking and IPV, including time constraints, and inadequate knowledge.[45,46] Thus, there is a significant need for healthcare provider training around best-practice guidelines, decision-support tools, and education on area-specific resources.

There were several limitations in the current study. First, the PRAMS IPV assessment is limited to physical abuse, although evidence links psychological and sexual abuse with adverse health behaviors and outcomes.[47] Additionally, information on IPV and smoking was limited to 1 year before pregnancy. The chronicity of IPV may be an important consideration in smoking initiation as well as preconception health status. As with all self-reported data, reporting and recall biases may generate misclassification. SGA data may suffer inaccuracies related to maternal recall or misidentification of last menstrual period for establishing gestational age. Furthermore, we were unable to assess whether the association between perinatal IPV and SGA contributes to well-documented disparities in birth outcomes because the SGA variable, as operationalized by PRAMS, is adjusted for race/ethnicity. Finally, data on substance use and mental health disorders during pregnancy, both important predictors of adverse neonatal outcomes, are limited in PRAMS.

Conclusion

Our study provides important insights into the possible mechanisms by which IPV may influence neonatal outcomes and in turn provide clinical direction to mitigate the harm of IPV and smoking. Women who experienced perinatal IPV were more than twice as likely to report prepregnancy smoking and nearly 1.5 times more likely to continue to smoke into the last 3 months of pregnancy as compared with their nonabused counterparts. Importantly, women who reported sustained smoking into the last 3 months of pregnancy had more than twice the risk of an SGA birth than those women who did not smoke before or during pregnancy. Our findings highlight the critical role that IPV plays in both maternal smoking during pregnancy and SGA birth.

Screening for violence is an important first step in improving maternal and child outcomes. Healthcare providers caring for women during the perinatal period are in the unique position to assist women experiencing perinatal IPV given the nature of the patient–provider relationship as well as the multiple contacts during this period. An enhanced understanding of the psychosocial barriers by which women are confronted may assist in addressing other health risks such as smoking which jeopardize maternal and infant outcomes.

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Author Disclosure Statement

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