Maternal tobacco use: A third-trimester risk factor for small-for-gestational-age pregnancy outcome

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ABSTRACT

Background: Small for gestational age (SGA) is a well-known consequence of maternal smoking. Here, we newly examine the magnitude of SGA risk by week of gestational age.
Methods: Singleton live births (N = 3,032,928) with recorded birth weight, gestational age (22–44 weeks), and maternal tobacco use (Y/N) were categorized as to SGA (Y/N), based on 10th percentile gender-specific weights-for-age.
Results: SGA prevalence among tobacco users (19.5%) and non-users (9.1%) yielded a significant SGA prevalence rate ratio of 2.15 (2.13–2.16) and a significant adjusted odds ratio of 2.36 (2.34–2.38). The tobacco non-users' rate was steadily near 9% across the week 22–44 gestational age range. The tobacco users' rate was steady until week 33 when it rose monotonically through week 37 to about 20% at week 38 and remained high. This pattern for SGA by gestational week was similar for prevalence rates and adjusted ORs. Tobacco use only through week 33 was not seen to be an SGA risk factor. The magnitude of tobacco use as an SGA risk factor for late third trimester births increased during the period of preterm birth and became fully evident with a two-fold risk for full term infants.
Conclusion: We newly report the temporal pattern of tobacco-related SGA by week of gestational age. Tobacco-related SGA was only seen for late third trimester births – increasing during weeks 33–37 with a doubling during weeks 38–44. This pattern, informative for issues of mechanism, highlights the potential benefit of extending tobacco cessation programs through the third trimester of pregnancy.

1. Introduction

Epidemiological and clinical studies of pregnancy have well established that tobacco smoking increases the risk of poor neonatal health outcomes. Deleterious health outcomes observed at birth and linked to maternal tobacco smoking include, but are not limited to, an elevated risk of Small for Gestational Age (SGA) or Intrauterine Growth Restriction (IUGR), Low Birthweight (LBW), and Preterm Birth (PTB) (Jaddoe et al., 2007). Small for gestational age (SGA) as a measure accounts for both low birthweight and preterm birth and provides for a continuous measure across both birthweight and gestational age

(Association of Maternal and Child Health Program, 2014). Previous studies have demonstrated a 2–3 fold SGA risk with maternal smoking (Mitchell et al., 2002; Chiolero et al., 2005; Tong et al., 2017).

Small for gestational age (SGA) infants are at an increased risk of perinatal deaths and SIDS and later deficits in development including the social and cognitive domains (U.S. Department of Health and Human Services (US DHHS), 2004). Furthermore, SGA children are at higher risk for long term consequences involving cardiovascular disease, chronic lung disease, and neurological and metabolic conditions in adult life (Hales et al., 1991; Barker et al., 1993; Noakes et al., 2007).

Despite the known risks, women continue to smoke during

pregnancy. In 2002, an estimated 11.5% of US infants were exposed to maternal smoking during pregnancy (Dietz et al., 2010). In 2010, the Pregnancy Risk Assessment Monitoring System (PRAMS) reported that 23% of women reported smoking in the 3 months before pregnancy; 11% in the last 3 months of pregnancy; and 16% 2–6 months after delivery (Tong et al., 2013). Cigarette smoking remains one of the most preventable causes of infant morbidity and mortality in the United States. Reducing maternal smoking during pregnancy will remain a critical component of public health in improving maternal and infant health.

The purpose of this study is to examine the temporal pattern of tobacco-related SGA with respect to gestational age by week using live birth certificate information for singleton infants born (1990–2002) to mothers who reside in the four central Appalachian states.

2. Method

The study analyzes the SGA data in the 1990–2002 US live birth certificate dataset for the four states (Kentucky, Tennessee, Virginia, and West Virginia) in our neonatal health study of central Appalachian States. The 1989 standard live birth certificate was used by all four states during this period. The subsequent revision was released in 2003. These files, containing 3,206,343 live birth records, were obtained from the National Association for Public Health Statistics and Information Services (NAPHSIS) with the approval from the individual state departments of health and included both county of birth occurrence and county of maternal residence in addition to the information available on the public use data files. The analyses here are based on the state and county of maternal residence rather than on the state and county of birth. Thus, out-of-state births to mothers from these four states are included, while in-state births to mothers whose residency was outside of these four states are not included. The leading authors (SHL, HF) had unrestricted access to the data and vouch for accuracy and completeness of the data and all analyses.

2.1. Outcome measure

The outcome, small-for-gestational age (SGA), was derived from recorded birthweight and gestational ages for each gestational age in weeks using the gender-specific 10%tile levels in Oken et al. (2003). The birth certificate specifically asks for the "Clinical Assessment of Gestation (Weeks)". Birthweights were recorded by medical professionals at the time of birth. Oken et al. (2003) is a refinement of the analyses of Alexander et al. (1996) that were based on the US national birth certificates for 1991 (Oken et al., 2003). Oken et al. (2003) had developed gestational age-specific, gender-specific birthweight distribution norms from U.S. live birth certificates (1999–2000) for male and female singleton live births with gestational ages 22 to 44 weeks (Oken et al., 2003). We used the cutoff values for male and for female live births with birthweights less than the 10%-ile birthweight level at each gestational age for determining SGA and SGA prevalence. Both birthweight, and gestational age in weeks (22–44 weeks) entered as discrete variables which allowed for an examination of SGA rate by gestational week.

2.2. Study population

Our analytic data set contained 3,067,914 records that met the Oken criteria, of which 3,032,098 (99%) also had recorded maternal tobacco use history. These 3,032,928 singleton live births comprised our study population.

2.3. Exposure

Information on maternal tobacco use (yes or no) was obtained at the time of delivery and was the primary exposure of interest.

2.4. Co-variables

Analytic co-variables, based on the published SGA literature, included maternal factors (marital status, age, race, ethnicity, and education), location (state of maternal residence and place of maternal birth), and prenatal care adequacy. Gestational age by week, birth year, and child's gender were additional co-variables. The analysis was conducted using all the co-variables that had been recorded for at least 98% of the live births.

Paternal characteristics were not included in the analysis as the documentation was incomplete (20–70% missing data). Nor were the average number of cigarettes smoked (17% missing) available. Similarly, alcohol use was not included as it was markedly under reported – 1.5% prevalence compared to national rates of 16.3% (Centers for Disease Control and Prevention (CDC), 2002). Data on medical risk factors, e.g., diabetes, hypertension, and renal disease, were not extracted as they, unlike tobacco use, have not been validated from the birth certificate (Nielsen et al., 2014).

Place of maternal birth entered the analytic dataset as a dichotomous variable (foreign or domestic), as did marital status (unmarried or married), maternal age (< 18 yr or 18+ yr), tobacco use (yes or no), child sex (female or male), ethnicity of mother (Hispanic or Non-Hispanic), maternal education (> 12 years of schooling or not), and pre-natal care adequacy (PCA) (adequate or not). Maternal race entered as white, black, or other. Prenatal care adequacy is a constructed co-variable that includes both the trimester when prenatal visits were initiated and the number of prenatal visits using a nomogram (modified Kessner criterion) developed by IOM (1973) and was predetermined in the birth certificate data set (Kessner et al., 1973). State of maternal residence was limited to the four states in the study [KY, TN, VA, and WV]. Birth year (1990–2002) entered as a discrete variable.

2.5. Data analyses

SGA prevalence and relative risks were calculated for tobacco users and tobacco non-users. Both unadjusted and adjusted odds ratios were calculated with adjustments for the co-variables above.

Multivariable logistic models were developed using Stata SE-13 with SGA as the dependent variable, maternal tobacco use as the independent variable of interest, and the above co-variables of maternal and infant risk factors as additional independent variables of interest (Stata Statistical Software). Results for all the analyses were considered significant when the p value was < 0.05.

SGA prevalence stratified by gestational week (weeks 22–44) and overall were graphically presented, as were gestational week-specific rate differences between tobacco users and tobacco non-users. Gestational week-specific adjusted odds ratios were graphed with 95% confidence intervals. State-specific gestational week-specific odds ratios were simultaneously graphed using 3-week rolling averages.

3. Results

3.1. Cohort development

The four state (KY, TN, WV, VA) live birth certificate (1990–2002) data file contained 3,206,343 live births, of which 3,067,914 met the reference populations criteria (Oken et al., 2003). Tobacco use histories were known for 3,028,928 98.9% of the eligible live births. The final analytic cohort of 3,032,928 was comprised of 1,555,669 male newborns and 1,477,259 female newborns.

3.2. SGA risks and ratios by tobacco use history

SGA infants are those with birthweight below the 10th percentile for their gender-specific gestational age. Study newborns were classified as being SGA based on the male and female 10th percentile gestational

Table 1

Small-for-gestational age (SGA) by maternal tobacco use – numbers, prevalences, and ratios.

Population	SGA	Non-SGA	Total	SGA Prevalence (%)	95% CI
Total	332,542	2,700,386	3,032,928	11.0%	(10.9%-11.0%)
Tobacco Use					
Yes	106,391	438,617	545,008	19.5%	(19.4%-19.6%)
No	226,151	2,261,769	2,487,920	9.1%	(9.05%-9.13%)
% Tobacco Use	32.0%	16.2%	18.0%		
	Prevalence Rate Ratio			2.15	(2.13–2.16)
	Crude Odds Ratio			2.43	(2.41–2.45)
	Adjusted* Odds Ratio			2.36	(2.34–2.38)

*Adjusted for child sex, prenatal care adequacy, maternal characteristics (married, race, Hispanic, age, education, foreign-born), state of residence, year of birth, and gestational age (weeks).

week-specific birthweight cutoff-values developed from the 1999–2000 US birth certificates for singleton births (Oken et al., 2003).

Overall, the SGA prevalence for the four-state dataset was 11.0%, significantly higher than the expected prevalence of less than 10% (Table 1). The SGA prevalence for tobacco users was 19.5% and for tobacco non-users was 9.1%, yielding a prevalence rate ratio (PRR) of 2.15 (95% CI, 2.13–2.16; $p < 0.0001$) and a crude odds ratio (cOR) of 2.43 (95% CI, 2.41–2.45; $p < 0.0001$). The adjusted odds ratio (aOR) for tobacco use was 2.36 (95% CI, 2.34–2.38; $p < 0.0005$). Alternative analyses might have presented adjusted prevalence rate ratios or risk ratios.

The tobacco use prevalence for the four-state area was 18.0% (545,008/3,032,928 = 18.0%). Tobacco use prevalence for SGA births (32.0%) was twice that for non-SGA births (16.2%).

3.3. Co-variables

The prevalence of co-variables for tobacco users and non-users are shown in Table 2. Tobacco use had the greatest odds ratio among the strongly significant ($p < 0.005$) independent variables in the multi-variable logistic regression with the greatest odds ratio. The other strongly significant ($p < 0.005$) independent variables were marital status, maternal race, maternal age, maternal education, maternal for-eign-born, prenatal care adequacy, and state of residence (KY, TN, VA, WV). The odds ratios were statistically significant for each gestational age from week 34 through week 44.

3.4. Gestational age stratification and SGA prevalence for tobacco users and non-users

With over 300,000 SGA infants in our study population, it was feasible to examine the SGA prevalence for tobacco users and non-users by week of gestational age at birth (Fig. 1). The overall SGA prevalence was 19.5% (0.195) for tobacco users and was 9.1% (0.091) for tobacco non-users, but these varied by gestational age.

The SGA prevalence for maternal tobacco non-users stayed between 8% (0.08) and 10% (0.10) independent of gestational age.

In contrast, SGA prevalence for tobacco users, which was similar to that of non-users through 33 weeks of gestational age, rose thereafter. The SGA prevalence for tobacco users showed a monotonically in-creasing prevalence from gestational week 33 to week 37, i.e., the period of pre-maturity, and then reached a plateau of 20% (0.20) that extended throughout the full-term period. The multivariate logistic regression revealed the same patterns as seen in the unadjusted pre-valence analyses.

3.5. SGA prevalence by gestational age, tobacco use, and state

To explore further, SGA prevalence was analyzed by gestational age and tobacco use for each of the 4-states (Kentucky, Tennessee, Virginia, and West Virginia) (Fig. 2). Our findings were consistent for all 4-states

Table 2

Maternal characteristics by tobacco use – number, proportion, and prevalence.

Co-Variable	Number*		Proportion		Prevalence	
	User	Non-User	User	Non-User	% of Users	% of Non-Users
N	545,008	2,487,920	18%	82%		
Maternal Birth*						
Domestic	535,336	2,259,754	19%	81%	98%	91%
Foreign	9,145	226,247	4%	96%	2%	9%
Maternal Residence						
Kentucky	163,414	480,300	25%	75%	30%	19%
Tennessee	172,808	765,860	18%	82%	32%	31%
Virginia	140,491	1,048,377	12%	88%	26%	42%
West Virginia	68,295	193,383	26%	74%	13%	8%
Marital Status						
Married	295,463	1,810,962	14%	86%	54%	73%
Unmarried	249,545	676,958	27%	73%	46%	27%
Maternal Age						
< 18 y/o	31,050	121,355	20%	80%	6%	5%
18–35 y/o	486,006	2,171,687	18%	82%	89%	87%
> 35 y/o	27,952	194,878	13%	87%	5%	8%
Maternal Race						
White	481,598	1,924,893	20%	80%	88%	77%
Black	60,608	490,692	11%	89%	11%	20%
Other	2,802	72,335	4%	96%	1%	3%
Maternal Hispanic*						
Hisp	2,905	100,218	3%	97%	1%	4%
Non-Hisp	541,696	2,385,449	19%	81%	99%	96%
Maternal Education*						
> 12	89,453	1,187,784	7%	93%	16%	48%
< = 12	453,544	1,290,011	26%	74%	84%	52%
PCAdequacy*						
Adequate	363,416	1,933,122	16%	84%	68%	79%
< Adequate	169,159	509,851	25%	75%	32%	21%
Child Gender						
Female	279,738	1,275,931	18%	82%	51%	51%
Male	265,270	1,211,989	18%	82%	49%	49%

*Where the sum is less than the total, the difference is due to missing values.

combined and for each state independently. The temporal pattern ob-served for the 4-states combined was thus independently replicated in each of the four states (3-week rolling averages). This indicated a generic pattern of association with tobacco use rather than a pattern unique to any one state.

4. Discussion

While maternal cigarette smoking is well-known to be a major risk factor for small-for-gestational-age (SGA) infants, the age at which this occurs is not clear. This study, based on more than 3 million singleton live birth certificates from four US states, has found it to be demon-strably seen only in late third trimester. For those born prior to 33 weeks of age, the prevalence of SGA among tobacco users and non-tobacco users was similar. The prevalence showed a monotonic increase until 37 weeks of age and then plateaued through 44 weeks of age.
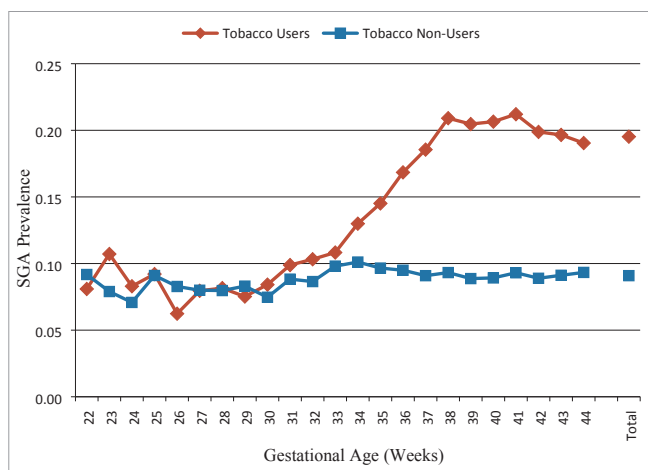
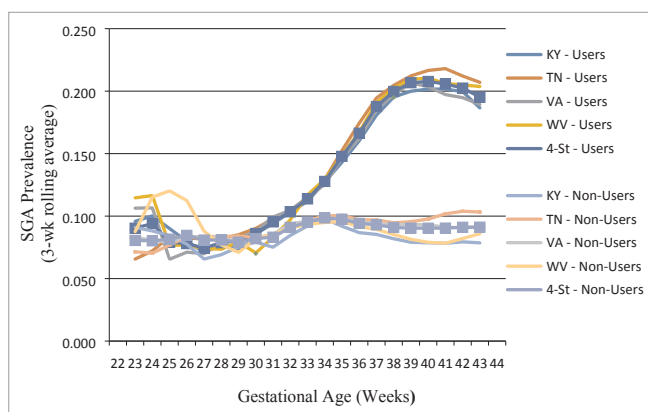**Fig. 1.** SGA prevalence by gestational age for tobacco users and non-users.

Fig. 2. SGA prevalence by gestational age, tobacco use, and state (3-wk rolling average).

These findings may suggest that the SGA effect of cigarette smoking is an acute effect of smoking during the period of rapid growth rather than a long-term effect of early exposure. If this were so, it would further suggest that programs that stress smoking cessation in the third trimester may be influential in reducing the prevalence of SGA.

This is the first study to examine SGA prevalence on a gestational week basis. Previous studies have examined SGA risk by trimester of smoking cessation. Some have found that the smoking-associated SGA risk is eliminated with smoking cessation in the first trimester (Yan and Groothuis, 2015; Polakowski et al., 2009; Blatt et al., 2015; Lieberman et al., 1994; Abraham et al., 2017; Bernstein et al., 2005), and others that the risk is eliminated or reduced with cessation during the second trimester (Kovo et al., 2013; Yan and Groothuis, 2015; Polakowski et al., 2009; Blatt et al., 2015; Lieberman et al., 1994; Abraham et al., 2017). These studies have generally analyzed by trimester. Many studies that have specifically looked at the third trimester have found a marked reduction or elimination of smoking-associated SGA where cessation has occurred before the third trimester. Both Baba et al. (2013) and Yan and Groothuis (2015) reported no increased risk of SGA for women who smoked during the 1st-trimester but had quit by the 3rd-trimester (Baba et al., 2013; Yan and Groothuis, 2015). Polakowski et al. (2009) showed a significant (41%) reduction in SGA risk with quitting in the second trimester for full-term births but less so for preterm (week 28–36) births (Polakowski et al., 2009). Blatt et al. (2017) in an analysis of 7 years (2006–2012) of Ohio birth certificates found that ceasing cigarette smoking in the third trimester reduced the SGA risk from 2.26 to 1.67 (Blatt et al., 2015). Mitchell et al. (Mitchell et al., 2002) was one of the few studies to report risk by month of

cessation and reported an increased risk of SGA only for those women who continued smoking into their last month of pregnancy (Mitchell et al., 2002). The data from Lieberman et al. (1994), a study from Boston Hospital for Women that obtained trimester-specific smoking histories, had shown that the increased LBW at term came entirely from the mothers who smoked during the 3rd trimester (Lieberman et al., 1994). A meta-analysis of prenatal ultrasound studies found that significant differences in intrauterine SGA prevalences were found only in the third trimester (Abraham et al., 2017).

Bernstein et al. (2005) conducted a prospective study on smoking cessation during well-dated pregnancies at the University of Vermont. They found among the smoking parameters examined that the strongest predictor of birth weight percentile was the maternal third-trimester cigarette consumption. They found a linear correlation between newborn birth weight and the number of cigarettes smoked in the third-trimester (Bernstein et al., 2005).

The third trimester is the period of maximum intrauterine growth and the period of maximum stress on the placenta. During this period, the fetal weight increases on average from about 1000 g to more than 3000 g. Thus, it is the time period in which a reduction in birth weight is most likely to be expected to occur (Oken et al., 2003). Naeye (1981) examined 8000 placentas in the Collaborative Perinatal Project (CPP) seeking the etiology of the fetal growth retardation associated with cigarette smoking (Naeye, 1981). He concluded that it could not be explained by chronic undernutrition, genetic factors, or under-perfusion. While pathological effects have been observed from smoking both in the placenta and in the uterine arteries, these effects did not seem to explain the increased SGA prevalence (Naeye, 1981). More recently, Kovo et al. (2013) has examined and compared the pathology in early-onset fetal growth retardation (≤ 34 weeks gestation) with that in late-onset fetal growth retardation (> 34 weeks gestation) (Kovo et al., 2013). They reported that the pathological difference for the late-onset fetal growth retardation was found in the fetal vasculature rather than in the placenta. Our suggestion here might be that smoking-related SGA may manifest as late onset fetal growth retardation and, thus, that its underlying pathology may be more related to fetal effects at that time period than to placental effects.

Alternative hypotheses relate to reduced tissue oxygenation or specific toxicities. Fetal tissue oxygenation is critical for normal fetal growth with higher levels required in the later trimesters (Naeye, 1981). It may be that the acute hypoxemic effects of carbon monoxide are relevant. Evidence does show that carbon monoxide levels in the serum of pregnant women who smoke are three times higher than those in pregnant women who don't smoke, and fetal concentrations of carbon monoxide have been found to be twice those of their mothers (Rogers, 2009). Additional acute exposures include nicotine and the myriad of other toxins in cigarette smoke (Rogers, 2009). The mechanism is not known and may be multifactorial.

The strength of this study includes that all data on more than 3 million live births and that those data have been collected on the same standardized form. The critical variables of birth weight and of gestational age were obtained in the same manner throughout and were susceptible to little observer variation. Tobacco use histories were obtained on nearly 99% of the newborns, and all co-variables were obtained on more than 98%. The same pattern was observed in each of four separate states. An additional strength of this study is that it has been conducted in four central Appalachian states that includes three of the four states in the USA with in 2003 the highest prevalence ($> 24\%$) of cigarette smoking among women (Ferdosi et al., 2018; Morbidity Mortality, 2003).

Limitations of this study included that smoking history was self-reported and not specified by trimester. The analysis did not account for non-validated or non-recorded co-variables (e.g., paternal characteristics, medical risk factors, maternal height and BMI, and social drugs [e.g., alcohol, snuff, and narcotics]). Further, this analysis applies only to SGA as a consequence of maternal smoking and not specifically to the

other adverse effects of maternal smoking. These data were obtained using the standard birth certificate of 1989. It would be appropriate to replicate these analyses using the standard birth certificate of 2003 with the specificity of smoking history and the variety of co-variables that it contains. Such replication elsewhere and with broader covariate data is urged.

In summary, we newly report the temporal pattern of tobacco-related SGA by week of gestational age in the third trimester. As a result, we are able to observe a narrow exposure window in which tobacco-related SGA of newborns seems to occur primarily in the late 3rd trimester (i.e., after the 33 week of gestation). This includes the time period for most pre-term births and all full-term births and the period of greatest medical attention in pregnancy.

This pattern is consistent with the observations that smoking cessation prior to the third trimester eliminates or greatly reduces the tobacco-related increased risk of SGA and leads to the speculation that the mechanisms of action are acute from third-trimester exposure rather than a later manifestation of earlier exposure. Further, it suggests that smoking cessation begun by the beginning of the late third trimester (i.e., week 33) should be considered in the design of public health education programs focused on maternal smoking cessation to eliminate at least one of the consequences of maternal smoking.

CRediT authorship contribution statement

Steven H. Lamm: Conceptualization, Methodology, Writing - original draft, Writing - review & editing, Supervision. **Hamid Ferdosi:** Methodology, Data curation, Validation, Formal analysis, Resources, Investigation. **Isabella J. Boroje:** Methodology, Writing - review & editing. **Nana Ama Afari-Dwamena:** Resources, Validation. **Lu Qian:** Resources, Validation. **Elisabeth Dissen Dash:** Resources, Investigation, Validation. **Ji Li:** Methodology, Formal analysis. **Rusan Chen:** Methodology, Formal analysis. **Manning Feinleib:** Conceptualization.

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