Consequences of Smoking during Pregnancy on Maternal Health

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Abstract

Objective: To estimate the incidence of maternal cardiovascular and pulmonary events and the prevalence of other comorbid conditions among pregnant smokers.

Methods: We queried the Nationwide Inpatient Sample (NIS) from the Healthcare Cost and Utilization Project (HCUP) of the Agency for Healthcare Research and Quality (AHRQ) for pregnancy-related discharge codes for the years 2000–2004. The prevalence of various conditions and the incidence of various complications were compared between smokers and nonsmokers.

Results: The majority of smokers were young and white and had public insurance. Smokers were more likely to have experienced deep vein thrombosis (odds ratio [OR] 1.3, 95% confidence interval [CI] 1.1, 1.6), stroke (OR 1.7, 95% CI 1.2, 2.5), pulmonary embolus (OR 2.5, 95% CI 2.1, 3.0), and myocardial infarction (OR 4.6, 95% CI 3.3, 6.4). They were 3 times more likely to have experienced influenza or pneumonia (OR 2.9, 95% CI 2.7, 3.2) and 15 times more likely to have bronchitis (OR 15.2, 95% CI 12.8, 18.2). They were more likely to suffer from a number of comorbidities, including asthma (OR 4.0, 95% CI 3.7, 4.2) and gastrointestinal ulcers (OR 3.7, 95% CI 2.6, 5.5). Although they were less likely to have experienced gestational diabetes (OR 0.9, 95% CI 0.9, 1.0), preeclampsia (OR 0.8, 95% CI 0.8, 0.9), or eclampsia (OR 0.7, 95% CI 0.6, 0.9), they were more than 5 times as likely to have experienced an ectopic pregnancy (OR 5.4, 95% CI 4.6, 6.3).

Conclusions: Smoking has a negative impact on maternal health. Counseling about the risks of smoking in pregnancy should include not only fetal risks but maternal risks as well.

Introduction

ACCORDING TO ESTIMATES from the United States Department of Health and Human Services (DHHS), >20% of U.S. adults (45.1 million people) smoke cigarettes.¹ Although the rate of smoking decreases among women when they become pregnant, it is estimated that about 11% continue.² Smoking in a nonpregnant woman increases her risk for cardiovascular morbidity, such as acute myocardial infarction (MI),³ stroke,^{4–6} and pulmonary embolism (PE).⁷ Besides increasing the risk of cardiovascular morbidity,⁸ smoking in a nonpregnant woman also increases her risk of pulmonary morbidity.^{8–11} The effects of smoking on the outcomes of pregnancy are well documented and include an increased risk of preterm premature rupture of the membranes (PPROM), preterm birth, low birth weight, placenta previa, and placental abruption⁸; however, there are fewer data on the effects of smoking on the health of the women themselves

during pregnancy. The purpose of this study was primarily to estimate the extent of maternal cardiovascular and pulmonary morbidity among pregnant smokers and secondarily to estimate adverse pregnancy outcomes.

Materials and Methods

The research protocol used in this study was reviewed and approved by the Duke University Medical Center Institutional Review Board. We queried the Nationwide Inpatient Sample (NIS) from the Healthcare Cost and Utilization Project (HCUP) of the Agency for Healthcare Research and Quality (AHRQ) for all pregnancy-related discharge codes for the years 2000–2004 (the latest data available at the inception of the study). The NIS contains data from approximately 1000 hospitals and is the largest all-payer inpatient care database in the United States. It is a 20% stratified sample from a sampling frame that comprises 90% of all U.S. hospital discharges.^{12,13}

Information included in the NIS is what can be derived from a typical discharge abstract, with safeguards to protect the privacy of individual patients, physicians, and hospitals. Although the data are limited, the NIS is the most reliable source of U.S. data on hospital admissions and discharges.^{12,13} Reliability is supported by agreement among the NIS, a telephone survey, and the National Health Interview Survey (NHIS) (a national, door-to-door survey). Invalid or inconsistent diagnostic codes are flagged and dealt with by the HCUP.^{12,13} Missing data were excluded from this analysis. For instance, if a record was missing data on race, that record was excluded from analyses of race.

All pregnancy-related discharge records in the NIS from 2000 through 2004 were identified using the *International Classification of Diseases Ninth Revision* (ICD-9) codes 630–648 for admissions during pregnancy; codes 72–75, v27, and 650–659 for admissions for delivery; and codes 670–677 for readmissions postpartum. The presence of a delivery code (72–75, v27, or 650–659) was used to distinguish the records of hospitalizations for childbirth. The ICD-9 codes 305.1 and V15.82 were used to identify smokers. The prevalence of various conditions and the incidence of various complications were compared between smokers and nonsmokers. For various conditions and complications, both the ICD-9 code for a particular condition or complication in pregnancy and the general ICD-9 code for that condition or complication were used.

Hospital discharge data for the years 2000–2004 released from the U.S. AHRQ were analyzed based on the NIS sampling design, a multistaged sampling frame consisting of three stages. Because of the possibility of repeated admissions, only pregnancy discharges with a delivery code were used in the analysis. The weighted total number of pregnancy-related discharges was 21,207,981, and the unweighted count was 4,387,959. The three stages are strata (geographic region, urban vs. rural location, teaching status, type of ownership, and bed size), hospitals within the strata, and individual discharges weighted by population counts and controlled for missing data. STATA 9.0 (Stata Corp LP, College Station, TX) with its SVY (survey data) commands using these three stages were used for all analyses, both descriptive and inferential. Computational estimates used by STATA allowed the sampled data to estimate population counts for all hospital discharge records for the 5 years analyzed.

Two-way chi-square analyses were performed, accounting for the complex survey design of the NIS. The two-way chi-square analyses weighted yielded cell frequencies for smokers and nonsmokers for discharges with or without the condition under study. Cardiovascular and pulmonary events were tallied over all admissions. Repeat admissions could occur during pregnancy and postpartum, where as delivery admissions likely involve unique individuals. To avoid overcounting the prevalence of comorbid conditions, comorbid conditions were tallied from delivery admissions only. Cell frequencies were divided by the number of deliveries to compute rates (ratios). Because this was an exploratory investigation, logistic regression analyses were used to compute odds ratios (ORs) with 95% confidence intervals (95% CI) for each of the age and racial groups, medical conditions, and obstetric complications. *p* values for each variable under study were included. Because of the large sample size, *p* < 0.01 (type I error) should be considered significant. An OR

TABLE 1. RACIAL DISTRIBUTION OF PREGNANT SMOKERS WHO DELIVERED, BY YEAR^a

Year	Weighted counts smokers	Weighted counts nonsmokers	% White	% Black	% Hispanic	% Other or unknown
2000	95,496	4,128,033	57	8	3	32
2001	111,978	4,010,139	42	7	3	48
2002	126,162	4,149,294	38	7	3	52
2003	143,474	4,060,320	45	7	4	44
2004	163,702	4,219,382	46	7	4	43
Total	640,813	20,567,169				

^aRace code missing for 20,281, 45,150, 55,252, 50,602, and 53,705 smoking discharges for years 2000, 2001, 2002, 2003, and 2004, respectively.

can be very close to 1 yet have significant digits beyond 1 and a *p* value of 0.01.

Results

From 2000 to 2004, there were 21,207,981 weighted pregnancy-related discharges with delivery codes included in this database. Of these, 640,813 (4%) included the ICD-9 code for smoking, 305.1 or V15.82. Table 1 illustrates the racial distribution of the women who delivered coded as smokers. The data from AHQR allow each state to determine if it wishes to include racial codes in its records. Because some states do not include racial codes, there are approximately 50,000 discharges with this information missing. As shown in Table 2, most of the women were white, <24 years of age, and had public insurance.

Smokers were more likely than nonsmokers to experience cardiovascular events (Table 3). Compared with nonsmokers, the odds of a deep vein thrombosis (DVT) were 1.3 (95% CI 1.1, 1.6), the odds of a stroke were 1.7 (95% CI 1.2, 2.5), the odds of a PE were 2.5 (95% CI 2.1, 3.0), and the odds of an MI were 4.6 (95% CI 3.3, 6.4). Although smokers were no more likely to develop sepsis or require mechanical ventilation,

TABLE 2. SMOKING AMONG WOMEN AT TIME OF CHILDBIRTH, BY AGE AND RACE

Characteristic	Weighted counts smokers	Weighted counts nonsmokers	Odds ratio	95% confidence interval	p value
Age					
<20	95,150	2,168,512	1.0	—	—
20–24	226,053	4,974,166	1.0	(1.0, 1.1)	0.01
25–29	148,325	5,442,213	0.6	(0.6, 0.6)	<0.01
30–34	101,774	4,980,966	0.5	(0.4, 0.5)	<0.01
35–39	54,836	2,450,962	0.5	(0.5, 0.5)	<0.01
≥40	14,673	550,324	0.6	(0.5, 0.6)	<0.01
Race ^a					
White	325,829	7,931,703	1.0	—	—
Black	50,713	2,061,822	0.6	(0.5, 0.7)	<0.01
Hispanic	24,155	3,620,285	0.2	(0.1, 0.2)	<0.01
Insurance					
Private	222,990	11,742,196	1.0	—	—
Public	398,933	8,247,165	2.5	(2.3, 2.8)	<0.01

^aCalculated only for discharges where race was known.

TABLE 3. MATERNAL CARDIOVASCULAR EVENTS, PULMONARY EVENTS, AND INFECTIOUS MORBIDITY AMONG SMOKERS COMPARED TO NONSMOKERS

Event	ICD-9 codes	Weighted counts smokers	Weighted counts nonsmokers	Odds ratio	95% confidence interval	p value
Thrombotic events						
Stroke	431, 434, 436, 325, 674	184	2,936	1.7	(1.2, 2.5)	<0.01
Myocardial infarction	410	207	1,216	4.6	(3.3, 6.4)	<0.01
Pulmonary embolus	673.2, 415.1	849	9,102	2.5	(2.1, 3.0)	<0.01
Deep vein thrombosis	671.3, 451.1, 451.2	623	13,778	1.3	(1.1, 1.6)	<0.01
Pulmonary complications						
Pneumonia/influenza	480-83, 486-87	4,108	41,755	2.9	(2.7, 3.2)	<0.01
Bronchitis	491, 492	1,252	2,489	15.2	(12.8, 18.2)	<0.01
Infectious						
Bacterial infection	646.5-6, 590.1	28,475	692,786	1.2	(1.1, 1.3)	<0.01
Pyelonephritis	590.1	6,123	15,0378	1.2	(1.2, 1.3)	<0.01

they were 3 times more likely to experience influenza or pneumonia (OR 2.9, 95% CI 2.7, 3.2) and 15 times more likely to develop bronchitis (OR 15.2, 95% CI 12.8, 18.2).

Besides experiencing an increased number of cardiovascular and pulmonary events, smokers were more likely to suffer from a number of comorbidities, particularly pulmonary and cardiovascular comorbidities (Table 4). Compared with nonsmokers, smokers were slightly more likely to have hypertension, cardiomyopathy, and pulmonary hypertension. They were more than 4 times more likely to have asthma (OR 4.0, 95% CI 3.7, 4.2), 3 times more likely to be obese (OR

3.2, 95% CI 2.8, 3.5), and 14 times more likely to abuse other substances (OR 14.1, 95% CI 13.1, 15.2). Smokers were more likely to experience gastrointestinal ulcers, with an OR of 3.7 (95% CI 2.6, 5.5). Smokers were slightly more likely to have anemia and HIV infection. Smokers were less likely to experience gestational diabetes (OR 0.9, 95% CI 0.9, 1.0), gestational hypertension (OR 0.8, 95% CI 0.8, 0.9), and eclampsia (OR 0.7, 95% CI 0.6, 0.9).

There was a higher risk of placental abruption, intrauterine fetal demise, intrauterine (fetal) growth restriction (IUGR), preterm labor, placenta previa, and PPROM (Table 5).

TABLE 4. VARIOUS COMORBID CONDITIONS AMONG SMOKERS COMPARED TO NONSMOKERS

Condition	ICD-9 codes	Weighted counts smokers	Weighted counts nonsmokers	Odds ratio	95% confidence interval	p value
Cardiovascular						
Cardiomyopathy	425	339	5,596	1.4	(1.1, 1.9)	0.01
Heart disease	648.5	7,195	20,0373	1.1	(1.0, 1.2)	0.17
Hypertension	401-405	48,826	12,0431	1.2	(1.1, 1.3)	<0.01
Pulmonary hypertension	416.8	527	10,716	1.3	(0.8, 2.0)	0.24
Other						
Asthma	493	6,000	322,132	4.0	(3.7, 4.2)	<0.01
Diabetes	648.0, 250	27,398	689,241	1.2	(1.1, 1.3)	0.01
Gastrointestinal ulcers	531, 532, 533	148	1,231	3.7	(2.6, 5.5)	<0.01
Anemia	648.2, 280, 281	65,510	1,416,044	1.4	(1.4, 1.5)	<0.01
Thrombocytopenia	287.3-4, 287.5	2,706	93,000	0.9	(0.8, 1.0)	0.01
Social stressors						
Obesity	278.0	14,733	156,201	3.2	(2.8, 3.5)	<0.01
HIV	04.2, V08	1,182	23,807	1.5	(1.3, 1.8)	<0.01
Alcohol and substance abuse	648.3, 305.0, 305.2-3, 305.5-7	56,872	131,135	14.1	(13.1, 15.2)	<0.01
Malnutrition	648.9	195,170	3,184,733	2.4	(2.2, 2.7)	<0.01
Pregnancy specific						
Ectopic pregnancy	633	2,122	11,566	5.4	(4.6, 6.3)	<0.01
Antepartum bleeding	640.0, 640.8-9, 641.1, 641.3, 641.8-9	6,809	160,136	1.3	(1.2, 1.3)	<0.01
Hyperemesis	643	1,607	40,322	1.2	(1.1, 1.4)	<0.01
Gestational diabetes	648.8	27,398	881,819	0.9	(0.9, 1.0)	0.02
Gestational hypertension/preeclampsia	642.0, 642.2-642.7	65,510	2,482,829	0.8	(0.8, 0.9)	<0.01
Eclampsia	642.6	527	18,319	0.7	(0.6, 0.9)	0.01
Postpartum hemorrhage	666, 669.1	23,537	704,830	1.1	(1.0, 1.1)	<0.01
Transfusions	99.02, 99.04, 99.00, 99.07	2,818	106,711	1.3	(0.9, 1.8)	0.15

TABLE 5. ADVERSE FETAL OUTCOMES AT TIME OF DELIVERY

<i>Outcome</i>	<i>ICD-9 codes</i>	<i>Weighted counts smokers</i>	<i>Weighted counts nonsmokers</i>	<i>Odds ratio</i>	<i>95% confidence interval</i>	<i>p value</i>
Placental abruption	641.2	13,102	222,153	1.4	(1.3, 1.5)	<0.01
Preterm labor	644	74,961	168,3009	1.3	(1.2, 1.5)	<0.01
Intrauterine (fetal) growth restriction	656.5	26,498	310,099	2.6	(2.5, 2.8)	<0.01
Intrauterine fetal death	656.4	3771	88,011	1.1	(1.0, 1.2)	0.01
Placenta previa	641.0, 641.1	452	11,837	1.2	(1.1, 1.3)	<0.01
PPROM	658.1, 658.23	2,619	41,790	1.7	(1.5, 1.9)	<0.01

Pregnant smokers were also more than 5 times more likely to experience an ectopic pregnancy (OR 5.4, 95% CI 4.6, 6.3).

Discussion

Pregnant women are already at an increased risk of cardiovascular events, including acute MI,¹⁴ stroke,¹⁵ and venous thromboembolism,¹⁶ compared with women who are not pregnant. In previous case-control studies we performed using the same database and comparing women who experienced cardiovascular events during pregnancy with those who did not, we identified smoking as a risk factor.^{14,15,17} In this study we found that pregnant smokers had a >4-fold increased risk for acute MI, a 2-fold increased risk for PE, and a 1.3-fold increased risk for DVT compared with pregnant nonsmokers. The additional risk of cardiovascular events conferred by smoking is over and above the risk conferred by pregnancy.

We found multiple other effects on maternal health. We found a higher incidence of pulmonary complications, such as asthma, bronchitis, and pneumonia. Nonpregnant smokers are more likely to experience gastrointestinal ulcers,¹⁸ and this was true of the pregnant smokers in this study, with an OR of 3.7 (95% CI 2.6, 5.5). Smokers were slightly more likely to have anemia and HIV infection, possibly reflecting the lower socioeconomic status of the pregnant smokers. There was a higher incidence of ectopic pregnancies among smokers. Other studies have also found a 1.4–4-fold increased incidence of ectopic pregnancies among smokers,^{19,20} with heavier smokers having the highest risk.¹⁹ We found a 5-fold increased risk, perhaps because the smokers who were identified were heavier smokers. Theories on how smoking causes ectopic pregnancy include fallopian tube ciliary damage causing decreased ovum transit times and inflammation interfering with fertilization.²¹

Like other studies in the literature, this study found that smokers have a higher incidence of poor pregnancy outcomes, including PPRM,^{20,22} preterm labor,^{22,23} placental abruption,^{20,24} IUGR,^{25,26} and intrauterine fetal demise.^{27,28}

There are several limitations to this study. First, the data were collected from a database of all hospital discharges. Therefore, there is no verification of actual smoking status by biochemical markers and no supplemental data to confirm pulmonary or cardiovascular complications. Data are limited to events and comorbidities identified during hospital admissions in pregnancy or at the time of childbirth. Comorbidities may have been underestimated. Studies that have

used linked birth and death certificates have found that a more complete assessment of maternal mortality includes maternal deaths up to 1 year postdelivery. Obtaining data on these late maternal deaths was not possible using the deidentified data in the NIS database.

Second, there is a possibility that bias could be introduced if smoking is a known risk factor for certain conditions. Smoking could be coded more often, exaggerating the effects. For example, more physicians might label a patient a smoker if the patient were diagnosed with a PE. However, we did find a negative association with preeclampsia and a positive association with ectopic pregnancy, conditions for which a bias in coding for smoking would not be anticipated.

Lastly, the smoking rate in our dataset is around 4%, and we know that the smoking rate in pregnant women nationwide is around 11%.² The rate of smoking appears to be underreported in this database. Pregnant women do underreport smoking, perhaps because of the social undesirability of smoking in pregnancy.²⁹ As most of the hospital admissions were at the time of childbirth, it is possible that women quit smoking during the pregnancy. It is also possible that physicians are not identifying behavioral modifiers, such as smoking, when admitting patients or patients are not identifying themselves as smokers when they are admitted.

The discrepancy between smoking rates in this sample and in national samples is curious. The national samples used birth certificate records to ascertain smoking status and included women who were giving birth at hospitals. Linking of birth certificate data with NIS data might have provided a more complete enumeration of women who reported smoking during pregnancy and, therefore, provided a more complete picture of the effects of smoking on maternal health. Again, however, the data in the NIS are deidentified, which eliminates the possibility of linking the data to records from other sources. Because the smoking rate in this database is much lower than anticipated, there are likely to be smokers in the control group, and the effects are likely diluted. Therefore, the association between smoking and poor maternal outcomes could be higher than we are reporting. On the other hand, because cardiovascular events are rare in pregnant women, no one medical center would have enough numbers to establish an incidence and identify risk factors, which is possible with a large database, such as the NIS.

There are several implications from the findings from this study. Our study stands in contrast to a tendency to focus primarily on how maternal disease affects neonatal outcome while failing to notice maternal health as an end in itself. Al-

though the American College of Obstetricians and Gynecologists (ACOG) Committee Opinion on smoking in pregnancy reviews the detrimental consequences of maternal smoking on pregnancy outcomes (ectopic pregnancy, IUGR, placenta previa, placental abruption, PPRM, low birth weight, perinatal mortality, sudden infant death syndrome [SIDS], and increased risks to children's health), there is no mention of the increased risk of cardiovascular or pulmonary complications.³⁰ The oversight may be attributed to a paucity of data regarding maternal outcomes associated with smoking, but the lack of data itself highlights the need for data on maternal as well as fetal outcomes of pregnancy in the conduct of future clinical and epidemiological research during pregnancy.

Our findings also have several implications for clinical care. First, practice guidelines should be revised so that clinicians will recognize that smokers are at risk for cardiovascular and pulmonary complications during pregnancy. Second, our data suggest that counseling about smoking cessation should be a priority both preconceptionally and during pregnancy. As physicians, this should include educating women about the increased risks associated with smoking while pregnant—not only the fetal and neonatal complications but also the short-term and long-term consequences to maternal health. Because smokers are more likely to have significant comorbidities, adequate information about maternal and fetal risks of smoking may be critical not only to decisions about smoking cessation but also to decisions about when and whether to pursue pregnancy. Data from smoking cessation studies have shown that patient education and empowerment have a higher success rate than drug therapy alone.

In the end, maternal health cannot be separated from fetal health. A stroke or an MI during pregnancy will have immediate consequences for maternal and fetal health, and it will most certainly also have a long-term impact on the health of the woman, her children, and her family. Often, the best way to ensure the health of the fetus is to ensure the health of the pregnant woman. Identifying and preventing maternal complications of smoking during pregnancy are important components of improving the health of both women and the fetuses they carry.

Conclusions

Smoking has a negative impact on maternal health. Counseling about the risks of smoking in pregnancy should include not only the fetal risks but the maternal risks as well.

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Disclosure Statement

The authors have no conflicts of interest to report.

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