

Maternal Smoking and Birth Weight

Interaction With Parity and Mother's Own In Utero Exposure to Smoking

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Background: Few studies have reported interactions between maternal smoking and other maternal characteristics and exposures. We examined maternal smoking in a cohort study for which data from 3 generations were available to examine maternal characteristics and exposures from a life-course perspective.

Methods: We had data from 3 generations: women enrolled in the U.S. Collaborative Perinatal Project (CPP) between 1959 and 1965 at the Baltimore site (G1); daughters (G2) of those G1 mothers who were followed to ages 27–33 years in the Pathways to Adulthood study; and children (G3) born to the G2 women who provided pregnancy and birth information during the Pathways study. These data allowed examination of exposures that occurred to the mother during her childhood and in utero.

Results: We found evidence of a 3-way interaction effect on birth weight for maternal smoking in pregnancy, maternal exposure to smoking in utero (grandmaternal smoking), and maternal parity. Maternal smoking reduced birth weight in 3 of the subgroups, with the size of the effect on birth weight moderated by parity and the mother's own in utero exposure to smoking.

Conclusions: A mother's prenatal exposure to smoke may affect the birth weight of her offspring. This effect would be consistent with both the accumulation-of-risk and the fetal-programming hypotheses.

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Maternal smoking is associated with reduced birth weight across a wide range of populations,^{1–10} often with a dose–response trend.^{1,4,7,11–15} A few studies have reported interactions between maternal smoking and other maternal characteristics and exposures. Cnattingius and colleagues have reported a stronger association of smoking with the risks of small-for-gestational age among older mothers^{9,16,17} and low birth weight among multiparous mothers.¹⁸

We examined maternal smoking in a cohort for which we had data from 3 generations: women enrolled in the U.S. Collaborative Perinatal Project study between 1959 and 1965 at the Baltimore site (G1); daughters (G2) born to those G1 mothers and followed to ages 27–33 years in the Pathways to Adulthood study; and children (G3) born to G2 women who provided pregnancy and birth information during the CPP Pathways study. These data allow us to examine exposures that occurred to the mother during her childhood and even in utero. We present here the results of analyses examining the effect of maternal smoking on birth weight, with exploration of interactions with other maternal factors including in utero exposures to the mother.

METHODS

Data from 2 large longitudinal cohort studies were combined to create the intergenerational dataset for the present study. The Johns Hopkins Institutional Review Board approved our use of these data. The Collaborative Perinatal Project was a multicenter prospective study designed to identify risk factors for adverse perinatal and pediatric outcomes. Pregnant women were enrolled from 1959 to 1964, and their offspring were followed to 8 years of age.¹⁹ From 1992 to 1994, investigators at Johns Hopkins Hospital launched a follow-up study of the original Baltimore-based study participants and their children. Johns Hopkins had enrolled 4025 women in the original study from 1959 to 1964. Among those women, 187 had children who died during the follow-up and 404 did not participate in follow-up of the children at age 7 to 8 years. We also excluded 427 who had delivered in 1959 (before the original study protocol had

been finalized), and 66 participated in the pilot. Finally, 247 families were excluded randomly to cut costs. These exclusion criteria left 2694 (67%) of the G1–G2 pairs eligible for follow-up. Among these families, 1482 (55%) had provided complete G1 and G2 interviews.

We then necessarily excluded G2 women with no children ($n = 218$). We also excluded G2 men ($n = 674$). To ensure that the information obtained regarding G2's birth and childhood was as accurate as possible, we excluded those families whose G1 interview was completed by someone other than the biologic mother ($n = 29$). G2s who were twins and G2s of "other" races were also excluded. After our exclusions, 554 G2s remained for study.

A total of 1131 G3 children were reported by these 554 G2 women at their interview at age 27 to 33 years. We excluded 60 children with abnormalities at birth, 34 children with missing birth weights, 1 child whose birth weight was reported to have been less than 500 g, and 10 sets of twins. This left 1016 G3 children for analysis. Some G1 women had more than 1 pregnancy in the original study, and some G2 women had more than 1 child at the time of their interview. This resulted in nonindependent observations on 2 levels: G2s with the same mother and G3s with the same mother or grandmother. Dealing with this multiple clustering is not entirely straightforward using standard software. Therefore, we selected only the eldest female G2 in each sibship and used Generalized Estimating Equations applied to ordinary least squares regression to deal with G3 offspring born to the same G2 mother.^{20,21}

Finally, we excluded 18 infants whose G2 mothers reported having diabetes. Diabetes is associated with higher birth weights but not necessarily healthy perinatal outcomes. The final study sample size was 989 G3 infants born to 500 G2 mothers.

Variable Definition and Construction

Birth weight of the G3 infant was reported in pounds and ounces by the mother (G2) at the time of the interview. Although the length of the recall period varied, studies have shown that mother's recall of infant birth weight is reasonably accurate.^{22,23} Mothers were not asked to provide gestational age for the G3s, precluding the study of intrauterine growth restriction and preterm birth.

G2 variables for the index pregnancy included mother's race (black, white), adult height (inches), age at G3 birth (years), education at G3 birth (some college vs. not), parity before G3 birth (nulliparous, parous), and smoking during G3 pregnancy (yes, no). Age was modeled as a linear and quadratic term. A complete smoking history was obtained, including information on the dates when women began and ended periods of smoking. The unit of time was quarters of a year (eg, January through March). We noted the smoking status of G2 participants during the quarter-years during which the mother was pregnant with G3, and categorized the mothers into those who had never smoked, those who quit before the G3 index pregnancy, those

who smoked during part of the index pregnancy, and those who smoked during the entire index pregnancy. Preliminary analysis indicated that the relevant categories were those who smoked at all during the pregnancy and those who did not. The smokers were therefore combined for final analysis. An advantage of our data was the ability to link data on the childhood and prenatal (in utero) exposures of the mother. We included G2's own birth weight and whether or not the G2 mother had been small for her gestational age (intrauterine growth restriction [IUGR]). These data were available from medical records abstracted for the original study. IUGR was categorized as a dichotomous variable based on a 10th percentile cut-off of birth weight for gestational age using a U.S. standard growth chart.²⁴ Childhood exposures include the receipt of welfare in G2's household at birth or age 7 years and whether G2 was ever hospitalized between birth and age 8 years. Several variables were available for G1 (the mother of G2 and the grandmother of G3): height (inches), prepregnancy body mass index (BMI, kg/m²), education at G2's birth (at least high school diploma versus not), poverty ratio at G2's birth (a ratio of household needs to income, with values below 1 indicating poverty and unmet needs), history of sexually transmitted diseases (yes or no), and smoking during G2 pregnancy (yes or no). The correlation in the original study of self-reported smoking status with biomarkers has been well documented.²⁵

Statistical Methods

We assessed differences in birth weight (grams) by computing the difference in mean birth weight and the accompanying *t* test statistics. To adjust for G3s who were related, we applied GEE to ordinary least squares regression and adjusted the variance estimates for clustering by computing robust standard errors.^{20,21} To determine the significance of the interaction terms, we relied on χ^2 tests of model fit, rather than *t* test statistics for the coefficients, since the standard errors of the latter are subject to substantial collinearity when all possible interactions involving some variables are added to the model.

RESULTS

The mean birth weight for the G3 children in our sample was 3175 gm. Table 1 presents the frequency of the various descriptive characteristics of G1, G2, and G3 individuals for the entire G3 sample and for G3s exposed to maternal smoking compared with those who were not. As expected, the mean birth weight for G3s born to smoking mothers was 132 g lower than for those born to nonsmoking mothers (3251 gm vs. 3119 gm). Adjustment for the other variables included in Table 1 made only a small difference. We were interested a priori in 3 potential effect modifiers: maternal age at G3 birth, parity at G3 birth, and G1 smoking in G2 pregnancy (in utero exposure of the index child's mother). We found no evidence of interaction with maternal

TABLE 1. Descriptive Characteristics for G3 Sample Overall (n = 989) and by Maternal (G2) Smoking

	G3 Sample (n = 989)			Source of Data*
	G3 Total	G2		
		Nonsmoker (n = 417)	Smoker (n = 572)	
Infant (G3) characteristics				
G3 male sex; %	50.4	48.9	51.4	PAS G2 interview
G3 birth weight (grams)				PAS G2 interview
Mean ± SD	3175 ± 637	3251 ± 581	3119 ± 669	
Range	680–5358	992–5358	680–4678	
Maternal (G2) characteristics				
	<i>Adulthood</i>			
G2 white race; %	17	16	17	CPP G1 interview
G2 nulliparous at G3 birth; %	50	52	49	PAS G2 interview
G2 adult height (inches)				PAS G2 interview
Mean ± SD	64.2 ± 2.7	64.0 ± 2.5	64.3 ± 2.8	
Range	57–72	58–70	57–72	
G2 age at G3 birth (years)				PAS G2 interview
Mean ± SD	22.4 ± 4.0	23.1 ± 3.9	21.8 ± 4.0	
Range	14–32	14–32	14–31	
G2 education at G3 birth				PAS G2 interview
At least some college; %	31	33	30	
G2 parity prior to G3 birth				PAS G2 interview
≥ one prior live birth; %	50	48	51	
G2 smoker during G3 pregnancy; %	58	NA	NA	PAS G2 interview
	<i>Birth and Childhood</i>			
G2 birth weight (gm)				CPP medical record
Mean ± SD	2988.6 ± 535.0	2961.8 ± 546.8	3008.2 ± 525.9	
Range	1134–4649	1446–4479	1134–4649	
G2 intrauterine growth restriction; %	17	19	15	CPP medical record
G2 household received welfare at G2 birth or age 7 years; %	42	38	45	CPP G1 interview
G2 ever hospitalized between birth and age 8 years; %	27	28	26	CPP G1 report verified by doctor
Grandmaternal (G1) Characteristics				
G1 adult height (inches)				CPP medical record
Mean ± SD	63.4 ± 2.6	63.5 ± 2.5	63.4 ± 2.6	
Range	51–72	56–72	51–71	
G1 BMI (kg/m ²) at G2 birth				CPP medical record
Mean ± SD	24.0 ± 5.0	23.7 ± 4.7	24.1 ± 5.2	
Range	7.8–45.8	7.8–45.8	8.5–45.8	
G1 education at G2 birth				CPP G1 interview
At least high school diploma; %	26	33	21	
G1 poverty ratio at G2 birth*				CPP G1 interview
Mean ± SD	1.1 ± 0.7	1.2 ± 0.8	1.1 ± 0.6	
Range	0.1–4.7	0.1–4.7	0.1–4.2	
G1 STD history; %	9.0	9.8	8.4	CPP medical history
G1 smoker during pregnancy of G2; %	41	38	44	CPP medical history

*Ratio of household needs to income; values less than 1.0 indicate poverty and unmet needs.

G3, index child; G2, mother of index child; G1, mother of G2.

PAS, Pathways to Adulthood Study; CPP, Collaborative Perinatal Project.

age. We did, however, find a significant interaction of parity (Table 2); among nulliparous women the G3 infants born to smoking mothers were just 38 g lighter than their nonsmoking counterparts while the G3s born to multiparous smoking mothers were 250 gm lighter. We also found interaction of grandmother's (G1's) smoking. Maternal smoking had little effect (21 gm) if the mother had been unexposed in utero (ie, if G1 did not smoke while pregnant with G2), but a large effect (284g) if G1 had smoked during G2's gestation.

We then looked for a possible 3-way interaction among maternal smoking, parity, and grandmaternal smoking. On the basis of χ^2 analyses, including the 3-way interaction improved the fit of the model ($P < 0.05$ for addition of the 3-way interaction to the model with the main effects and both 2-way interactions).

Table 2 presents the crude and adjusted models with the effect of maternal smoking on G3 birth weight stratified into 4 groups: nulliparous G2 mothers where G1 did not smoke; parous G2 mothers where G1 did not smoke; nulliparous G2 mothers where G1 smoked; parous G2 mothers where G1 smoked. As indicated in the table footnotes, results were adjusted for several covariates, including G2's own birth weight. Because there was little difference between adjust-

ment for G2 birth weight and G2 IUGR, we chose to include G2 birth weight (which probably has less measurement error). Maternal smoking had essentially no effect on birth weight for infants born to G2 women who were nulliparous and were unexposed to smoking in utero. However, maternal smoking had a negative effect on the other 3 groups, with the size of the birth weight effect moderated by parity and grandmother's smoking. The strongest effect was in the group of parous women exposed to smoking in utero, for whom the G3 birth weight was reduced by more than 400 gm.

DISCUSSION

Maternal smoking during pregnancy is strongly linked to reduced birth weight and impaired fetal growth.¹⁻¹⁰ However, only a small number of studies have examined the possibility that the effects of maternal smoking depend on the underlying characteristics of the population. The adverse effect of maternal smoking on birth weight and fetal growth appears to be stronger among older mothers.^{9,10,16-18,26} In a follow-up report examining outcomes for nearly half a million births, Cnattingius and colleagues¹⁸ reported that the effect of smoking on the risk of low birth weight increased with both age and parity. Our results are consistent with the parity interaction observed by Cnattin-

TABLE 2. Birth Weight (gm) of Offspring (G3), Stratified by Smoking and Parity of the Mother (G2), and by Smoking of the Grandmother (G1), With Crude and Adjusted Birth Weight Differences

G1 Smoking Status	G2 Parity								
	Nulliparous			Parous			All		
	G2 Smoking Status (n = 204)			G2 Smoking Status (n = 204)			G2 Smoking Status (n = 408)		
	Yes	No	Difference	Yes	No	Difference	Yes	No	Difference
Yes	3167	3330	-163 (-172 to -154) -124* (-289 to 41)	2991	3473	-481 (-497 to -466) -462* (-624 to -300)	3080	3402	-322 (-325 to -320) -284† (-404 to -165)
	G2 Smoking Status (n = 294)			G2 Smoking Status (n = 287)			G2 Smoking Status (n = 581)		
	Yes	No	Difference	Yes	No	Difference	Yes	No	Difference
No	3160	3135	26 (11 to 40) 27* (-108 to 160)	3139	3185	-45 (-66 to -25) -84* (-239 to 72)	3150	3158	-8 (-10 to -7) -21† (-138 to 97)
	G2 Smoking Status (n = 498)			G2 Smoking Status (n = 491)			G2 Smoking Status (n = 989)		
	Yes	No	Difference	Yes	No	Difference	Yes	No	Difference
All	3163	3206	-43 (-46 to -39) -38‡ (-145 to 68)	3077	3300	-224 (-230 to -217) -250‡ (-368 to -132)	3119	3251	-132 (-133 to -131) -129* (-215 to -43)

*Adjusted for G3 sex, G2 race, G2 adult height, G2 age at G3 birth (linear and squared terms), G2 education at G3 birth, G2 birth weight, G2 adult height, G2 household receipt of welfare, G2 childhood hospitalization, G1 adult height, G1 BMI at G2 birth, G1 education at G2 birth, G1 poverty ratio at G2 birth, G1 STD history.

†Adjusted for all factors listed in * as well as G2 parity at G3 birth.

‡Adjusted for all factors included in * as well as G1 smoker during pregnancy of G2.

gius, although we did not find an interaction with maternal age. However, our sample size is much smaller and in particular had few older mothers.

There is a growing body of research on the effect of prenatal environment on adult chronic disease.^{27,28} Much of this research has focused on birth weight and small-for-gestational-age, which are assumed to be proxies for the fetal “programming” that has occurred during critical fetal periods. The exposures that may produce “programming” are unclear. Most studies on outcomes in adulthood have relied on cohorts of men and women with only the most rudimentary information on their gestational history (ie, birth certificates). We had data on the mother’s in utero exposure as well as her birth weight and IUGR status. We included maternal birth weight (Table 2) in our final models and also maternal IUGR (results not shown) but these variables failed to account for the effects of G2’s in utero exposure to smoking. We also adjusted for other variables (mother’s adult height, childhood hospitalizations) that might capture ways in which this very early exposure to smoking might have adversely affected the mother and thus explain the effects on her offspring, but the effects persisted. In utero smoking exposure appears to have an effect on a woman’s offspring that is independent of its effect on her own health in infancy and childhood, thereby supporting the notion of “fetal programming.”

Effects of exposures across the life course have also been proposed to influence later outcomes through an “accumulation of risk.”^{27,29} In utero smoking exposure may add to the cumulative effect of smoking exposure the mother experiences before and during pregnancy. Although these 2 pathways are not mutually exclusive, one pathway may predominate with regard to a particular exposure or outcome. Both the “accumulation of risk” and “fetal programming” models are consistent with our finding of larger effects of maternal smoking on birth weight among women who were themselves exposed in utero to smoking.

The finding of increased birth weight in offspring of nonsmoking mothers who were themselves prenatally exposed to smoke is more puzzling. There may be residual confounding. For example, although smoking in present-day cohorts is associated with decreased socioeconomic status, smoking by women in the 1960s may have been associated with higher socioeconomic status. Therefore, G1s who smoked may have been better off in unmeasured ways than G1s who did not smoke, and thus transferred these advantages to their G2 offspring. Such a difference could potentially explain the apparently beneficial effects of G1 smoking on G3 birth weight among the nonsmoking G2s.

Finally, our findings of no adverse effect of maternal smoking for the subgroup of nulliparas unexposed in utero must be addressed. With the exception of recent work by Cnattingius and a few others discussed above, little has been done to identify subgroups who may experience differential

vulnerability to the effect of smoking. Furthermore, few studies have data on the mother’s in utero exposure to smoking. Our findings must therefore be interpreted cautiously. Selection bias due to attrition and exclusions may contribute to the results, although some of the attrition was random. The exclusions were primarily related to G2 sex and birth order, factors that are unlikely to be associated with effects of smoking and parity on G3 birth weight.

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