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Prepregnancy body mass index, smoking during pregnancy, and infant birth weight

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Abstract

Purpose—Smoking during pregnancy is strongly associated with increased risk of small for gestational age (SGA) and low birth weight, while elevated prepregnancy body mass index (BMI) is associated with a decreased risk of SGA and higher birth weight. We investigated the combined effect of prenatal smoking and prepregnancy BMI on risk of SGA and on birth weight.

Methods—A total of 34,928 singleton, term pregnancies in residents of New York City between 1995 and 2003 were evaluated in multivariable regression models of birth weight and risk of SGA.

Results—Increasing prepregnancy BMI reduced the risk of SGA and increased birth weight. The effect of prenatal smoking on birth weight and SGA diminished in women as their prepregnancy BMI increased, such that prenatal smoking did not significantly impact the risk of SGA among women who were overweight or obese prior to pregnancy. Prenatal smoking decreased mean birth weight by 187 grams (95% confidence interval (CI): -337, -37) among underweight women, by 129 grams (95% CI: -170, -87) among normal weight women, by 46 grams (95% CI: -113, +20) among overweight women, and by 75 grams (95% CI: -162, +11) among obese women.

Conclusions—This study suggests that the effect of smoking during pregnancy on SGA and birth weight is present in underweight and normal weight women but markedly reduced among obese and overweight women.

MeSH Headings

birth weight; body mass index; cigarette smoking; fetal growth retardation; infant; small for gestational age

Children who were born with low birth weight or who were small for gestational age (SGA), defined as those with birth weights at or below the tenth percentile for gestational age and gender, are at increased risk of neonatal mortality and morbidities including decreased intelligence, cognition, and obesity (1,2). Mean birth weight has been increasing over the last several decades, which is largely attributed to increasing prepregnancy body mass index

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(BMI) and decreasing prenatal cigarette smoking which act in opposing directions on birth weight (3).

In several studies, prenatal smoking appeared to contribute to the increased risk of SGA and lower birth weight among women who were underweight, normal weight, overweight or obese prior to pregnancy (4,5,6). While these studies report prenatal smoking effects within strata of prepregnancy BMI, there was no explicit test for interaction on either the additive or multiplicative scale in any of these studies (4,5,6). Here we explicitly question whether prepregnancy BMI and prenatal smoking interact on the multiplicative or additive scales to influence risk of SGA in a multiethnic population of New York City residents who delivered at New York State Hospitals (outside of New York City). We also examine the joint effects of prepregnancy BMI and prenatal smoking on mean birth weight.

Materials and Methods

Maternal and neonatal data were obtained through the matching of New York City Department of Health and Mental Hygiene birth certificates database with the hospital discharge database maintained by the Statewide Planning and Research Cooperative System as described elsewhere (7). Only the births of New York City residents who delivered in hospitals outside of New York City during 1995-2003 were included (n = 39,009; (8) because New York City hospitals were not recording maternal height on birth certificates during the study period, precluding calculation of prepregnancy BMI. Women who lived in the boroughs of Bronx and Queens were overrepresented in the analysis due to their proximity to Westchester County- and Long Island- hospitals, respectively, which resulted in less of prenatal smoking (1.0% decrease in prevalence), fewer SGA births (1.6% decrease in prevalence), and higher mean prepregnancy body weight (2.7 lb greater) compared to all births to New York City residents. Prenatal smoking status was available for all births for which BMI was also available. This study was approved by the Institutional Review Board of Mount Sinai School of Medicine.

To examine the association between prepregnancy BMI and prenatal smoking on fetal growth, this population was restricted to term, singleton births (gestational age ≥ 37 weeks; n = 36,118). An additional 8 birth records were removed because of implausible combinations of birth weight and gestation duration: 7 births of 37- 40 gestational weeks with birth weights < 1000 g, and one birth weight of 6,030 g at 43 gestational weeks (n = 36,110; (9). Further, 1,163 birth records did not contain data on maternal race/ethnicity, 18 additional records did not contain maternal birth- place, and 1 birth record did not contain data on infant sex. Therefore, 34,928 term, singleton births were considered in the analysis.

Birth weight and clinical estimates of gestational age were acquired from the birth certificate. From these, SGA was defined as those babies that were below the tenth percentile of body weight for their gestational age and sex, based on the 1999-2000 US Standard (10). Birth certificates were used to identify prenatal smoking, defined by self reported ever smoking during pregnancy, and the quantity of cigarettes smoked per day (none, < ½ pack, ½-1 pack, and > 1 packs). Self reported prepregnancy weight and maternal height were obtained from the birth record to calculate prepregnancy BMI (kg/m^2), where BMI < 18.5 was considered underweight, 18.5 =\≤ BMI < 25 was considered normal, 25 =≤ BMI < 30 was considered overweight, and BMI ≥ 30 was considered obese (11). Maternal demographic characteristics (race/ethnicity, foreign- or United States- born, age, and education), parity, and delivery year, as well as infant sex, were obtained from the birth records.

To characterize the association of prepregnancy BMI and prenatal smoking with SGA, we calculated and presented adjusted risk differences (binomial distribution, link function = identity) as well as risk ratios (binomial distribution, link function = log) with their corresponding 95% confidence intervals (CI) by maximum likelihood estimation using multivariable generalized linear models (PROC GENMOD, SAS version 9.1.3 software, SAS Institute Inc., Cary, North Carolina, USA; (12). The association of prepregnancy BMI and prenatal smoking with birth weight (continuous distribution) was fit by least squares regression using a multivariable general linear model (PROC GLM, SAS). Covariates considered in the multivariable models included maternal race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, Asian and Pacific Islander, and Other; (7), maternal birth place (foreign or US), maternal age (≤ 20, 21-30, 31-40, or >40 years), maternal education (< 12, 12, or > 12 years), parity (0, 1, ≥ 2 viable previous live births), delivery year, and infant sex. The final adjusted multivariable models included covariates that were found to change the parameter estimate of prepregnancy BMI or prenatal smoking by 10% or more or were found to be statistically significant predictors in the saturated model. Goodness of fit of risk models was evaluated by deviance while goodness of fit of the birthweight model was examined by root mean square error.

To assess deviations from multiplicative effects of prepregnancy BMI and prenatal smoking on SGA risk, the interaction of prepregnancy BMI and prenatal smoking was coded as a multiplicative term in the multivariable adjusted models of SGA risk ratios (aRR). To assess any deviations from additivity of prepregnancy BMI and prenatal smoking on SGA risk, the interaction of prepregnancy BMI (underweight, normal weight, overweight, and obese) and prenatal smoking (multi-level and dichotomous) was coded as a multiplicative term in the multivariable adjusted models of SGA risk differences (aRD). Estimate statements were used to calculate the aRD and aRR of SGA between interaction levels, and their CI (PROC GENMOD, SAS). Similarly, to assess deviations from additivity of prepregnancy BMI and prenatal smoking on mean birth weight, the interaction of prepregnancy BMI and prenatal smoking was coded as a multiplicative term in the multivariable adjusted models of birth weights. Estimate statements were used in the analysis of birth weight differences to calculate the adjusted difference in birth weight between interaction levels (PROC GLM, SAS). Interaction contrasts were calculated to predict joint effects of prepregnancy BMI and prenatal smoking in the absence of interaction (13).

Secondary analyses were conducted to calculate the power this study had to detect effect sizes reported in other studies as an aid in data interpretation (PROC POWER, SAS; (4,5). Further, to assess the hypothesis that SGA develops in the absence of metabolic abnormalities (4,14), we excluded women diagnosed with pre-existing chronic hypertension (*International Classification of Diseases*, Ninth Revision (ICD-9) discharge diagnosis codes 401-405, 642.0-642.2, and 642.9, as well as birth records), non-proteinuric gestational hypertension (ICD-9 code 642.3 and birth records), preeclampsia/eclampsia (ICD-9 codes 642.4-642.6), and gestational diabetes (ICD-9 codes 648.81-642.82 and birth records; (7,8). When we examined the subset of births free from these complications (n = 31,615), our results were essentially the same as seen in all births here; therefore these metabolic complications are ignored and cases retained in our multivariable models (n = 34,928).

Results

The majority of women in this population were 21-40 years old (> 90%) and had obtained more than 12 years of education (> 70%, Table 1). Over 40% of the women were born outside the United States. Non-Hispanic whites had the highest prevalence of smoking, the lowest prevalence of SGA, and the highest mean birth weight compared to non-Hispanic blacks, Asian women, and Pacific Islanders. The prevalence of prenatal smoking was 2.3%,

and the prevalence of women who were obese or overweight prior to pregnancy was 36.2%. Women with gestational diabetes, preeclampsia, non-proteinuric gestational hypertension, and pre-existing chronic hypertension had increased prevalence of SGA.

The risk of SGA increased in women who smoked while pregnant in a dose dependent manner (Table 2). Non-smoking mothers had a baseline risk of 6.1 SGA births per 100 births, whereas mothers who smoked over 1 pack per day had 21.5 SGA births per 100 births. Conversely, we observed an inverse dose response relationship between increasing prepregnancy BMI and decreasing SGA. Compared to women who were normal weight prior to pregnancy (6.1 SGA infants per 100 births), women who were overweight or obese prior to pregnancy had decreased risk of SGA, whereas underweight women had 12.2 SGA infants per 100 births.

Despite the clear dose-response relationship between prenatal smoking and risk of SGA (Table 2), there was no significant interaction of prepregnancy BMI and prenatal smoking dose (none, < ½ pack per day, ½ - 1 pack/day, > 1 pack/day) on either the multiplicative or additive scales ($P = 0.14$ and 0.08 respectively, data not shown). Similarly, there was no significant interaction of the effect of prepregnancy BMI and dichotomized prenatal smoking (non-smokers, smokers) on the risk of SGA on the multiplicative scale ($P = 0.10$; Table 3). Reflective of the lack of interaction on the multiplicative scale, there was little difference in the aRR of SGA among obese and overweight women irrespective of their cigarette smoking status (Table 3).

The interaction between the effect of prepregnancy BMI and dichotomized prenatal smoking on the risk of SGA was significant on the additive scale ($P = 0.01$). The risk of SGA was substantially increased among normal weight- and underweight- women who smoked during pregnancy (Table 3). The combined effect of prepregnancy underweight and prenatal smoking on SGA risk (23.3 cases of SGA per 100 births) was greater than the 20.7 SGA births per 100 births predicted by the individual effects of being underweight and smoking during pregnancy in the absence of interaction (Table 3(13). Further, the interaction contrast predicted 12.1 SGA births per 100 births among overweight smokers and 11.7 SGA births per 100 births among obese smokers in the absence of interaction. On the contrary, less than half the expected risk of SGA was observed among overweight and obese smokers; the risk of SGA in overweight smokers was 6.0 SGA births per 100 births (aRD = -0.1, 95% CI: -4.3, +4.0) and was 3.7 SGA births per 100 births in obese smokers (aRD = -2.4, 95% CI: -7.7, +2.8, Table 3). The positive dose response of prepregnancy BMI was more pronounced among smokers, with a wider range of SGA risk associated with prepregnancy BMI among smokers compared to the range of SGA risk associated with prepregnancy BMI among non-smokers (Table 3).

The pattern of the prepregnancy BMI and prenatal smoking interaction on birth weight ($P = 0.04$) was similar to the pattern of their interaction on the risk differences in SGA (Tables 3-4, Figure 1). Birth weight decreased among underweight women, particularly in those who smoked during pregnancy (Table 4, Figure 1). Prenatal smoking decreased mean birth weight in underweight women by 187 grams (95% CI: -337, -37) relative to underweight, non-smoking women (Figure 1). Consistent with the protective effect of high prepregnancy BMI seen on SGA risk, the positive dose-response of prepregnancy BMI and birth weight was attenuated in prenatal smokers compared to non-smokers (Table 4, as indicated by the slopes in Figure 1). As was seen with SGA risk, there was no significant difference in birth weight among women who smoked during pregnancy and had high prepregnancy BMI (overweight and obese) relative to normal weight non-smoking women (Table 4). This is further demonstrated by within-BMI strata estimates of the effect of prenatal smoking on birth weight: prenatal smoking decreased birth weight by 46 grams (95% CI: -113, +20) in

overweight women relative to non-smoking overweight women, and prenatal smoking decreased birth weight by 75 grams (95% CI: -162, +11) in obese women compared to non-smoking obese women (Figure 1).

Discussion

Prepregnancy BMI and prenatal smoking interact on the additive scale to affect SGA risk. The increased risk of SGA and decreased birth weight due to prenatal smoking appeared to be stronger among women who were underweight or normal weight prior to pregnancy and weaker among women who were overweight or obese prior to pregnancy. Similarly, a rodent model of diet induced obesity found that cigarette smoke reduced fat mass only in the leaner, diet control group (15). Furthermore, the influence of BMI on birth weight and SGA was more pronounced in women who smoked compared to non-smoking women, with additive interactions implying that biological interaction may be present (13). This biological interaction may be conceptualized in one of two ways: smoking may attenuate BMI's effects on birth weight, and/or higher BMI may dampen the effect of smoking on birth weight.

It was previously hypothesized that SGA develops in the absence of metabolic abnormalities (4,14). However, we saw an increased prevalence of SGA births among women with gestational diabetes, preeclampsia, and hypertension (Table 1). In our secondary analysis, risk differences of the effects of prenatal smoking on SGA were essentially unchanged by the exclusion of all cases of gestational diabetes, preeclampsia, and pre-existing hypertension. Together these findings suggest that hyperglycemia and hypertension during pregnancy do not drive the results that we found for the pattern of effects for BMI and smoking on SGA occurrence (4,14).

Instead, we hypothesize that hyperlipidemia is responsible for the reduced risk of SGA among women who were overweight or obese prior to pregnancy and smoked prenatally. During the late period of a normal pregnancy, fetal growth peaks and maternal hyperlipidemia (primarily due to hypertriglyceridemia) develops (16,17). Prepregnancy obesity is associated with a further elevation in maternal serum triglycerides, which is likely protective against smoking related toxicity (18,19). For instance, as serum triglyceride levels increase, benzo[a]pyrene metabolism decreases and the quantity of benzo[a]pyrene adducts in aorta endothelial cells decreases (20,21,22). Similarly, obesity dampened the arginine vasopressin response to nicotine, and after substantial weight loss, this response to nicotine returned to normal (23). This is particularly noteworthy given the hypothesized endothelial dysfunction etiology of SGA (4,14).

Relatively few studies have examined the interaction of prepregnancy BMI and prenatal smoking on SGA and birth weight. Similar to our report, the hospital based Collaborative Perinatal Project (1959-1965, n = 44,000) demonstrated that crude birth weights from self-reported obese (>85th percentile for prepregnancy weight) smokers did not differ much from birth weights of all non-smokers (24). Unlike our findings, self-reported prenatal smoking reduced birth weights similarly across clinically measured height-for-weight categories in a small hospital- and clinic- based study (1975-1976, n = 536; (6). Likewise, in a mid-western, urban hospital population (1977-1993, n = 1,343) the odds of SGA due to self-reported prenatal smoking increased similarly among women who were either normal weight or obese prior to pregnancy (1.9-fold (95% CI: 1.0, 3.5) and 1.8-fold (95% CI: 0.9, 3.7) respectively; (5). While we too observed a 1.9-fold increased risk of SGA due to prenatal smoking in normal weight women, the effect of prenatal smoking in obese women reported here was lower than reported in Hellerstedt *et al.* (5). Nevertheless, the prenatal smoking effect was not statistically significant in obese women in both Hellerstedt *et al.* (5) and our

report. In a smaller (*n* = 7,757) Collaborative Perinatal Project based- study of the interaction of prepregnancy BMI and prenatal smoking on SGA occurrence, prenatal smoking increased the odds of SGA in women with prepregnancy BMI ≥ 25, however as was seen in the present study and in Hellerstedt *et al.*, the prenatal smoking effect was weaker in women with prepregnancy BMI ≥ 25 compared to normal weight women (4,5). Ness *et al.* and the present study are reasonably concordant between estimates of effect size of prenatal smoking on SGA in normal weight and underweight women (4). Perhaps Ness *et al.* report stronger prenatal smoking effects in women with prepregnancy BMI ≥ 25 than are reported here because of cigarette formulation changes from 1965 to 1995 in the United States (4).

The primary potential limitation in the present study is exposure misclassification, given that we relied on self-reported prepregnancy weight, height and prenatal smoking, which could plausibly lead to misclassification of prenatal smoking and prepregnancy BMI. As testament to low information bias that likely have resulted from these self-reported exposures, the expected dose response relationship between either prepregnancy BMI or prenatal smoking and SGA was evident (Table 2) and similar to that reported elsewhere (25). Further, the self-report of prenatal smoking appears more common on birth certificates than medical charts (26), and although cotinine levels identified more smokers than self-reported smoking on birth certificates in another study (27), this could reflect active use of smoke-less nicotine products, or environmental tobacco exposure (28). While prepregnancy BMI did not statistically interact with prenatal smoking dose, there was a trend of increased risk of SGA across prenatal smoking levels depicted in Table 2 among underweight women yet there was no trend in risk of SGA across prenatal smoking levels depicted in Table 2 among overweight and obese women (data not shown). BMI self-reports have demonstrated concordance with clinical assessments in several studies (29,30,31,32). Further, the majority of BMI category misclassifications occur within one unit of the BMI category boundary (33). When persons within one unit of the normal weight category boundaries or of the overweight upper boundary were excluded from our analysis, aRD and aRR were nearly identical to estimates presented here (*n* = 28,710). It is also unlikely that the trends reported here result from confounding by differences in prenatal care, which has been associated with increased risk of SGA (34), as late initiation of or absent prenatal care was more common in overweight and obese smokers compared to underweight smokers and thus would predict a greater, not lesser impact of smoking among women with late prenatal care onset. We were also unable to take into account repeat pregnancies to the same mother, and one would predict that evaluating such clustering results in confidence intervals that are somewhat wider than those reported here.

Lastly, because of the limited availability of maternal height data, this study is not population-based, drawing on a peculiar sample of New York City residents who delivered in New York State hospitals. While there is no obvious reason that this sampling mechanism would generate the results reported, the potential for selection bias should be acknowledged. However, All previous studies of the joint effects of prepregnancy adiposity and prenatal smoking were also hospital based (4,5,6,24), and our population was more broadly constituted than most previous studies of the issue. We performed secondary analyses using the prepregnancy BMI and prenatal smoking cut points used in previous estimates of SGA odds to confirm the lack of a prenatal tobacco effect among women who were overweight and obese prior to pregnancy was not an artifact of our unique population structure. We had over 95% power to detect the effects of prenatal cigarette smoking on risk of SGA within BMI strata reported by *Ness et al.* and *Hellerstedt et al.* (4,5).

In conclusion, we examined the effects of prepregnancy BMI and prenatal smoking on SGA risk in over 34,900 singleton term pregnancies in the largest study of this subject to date.

While the study is limited by potential information bias, the present findings support that prenatal smoking increases risk of SGA and decreases birth weight in women who enter pregnancy in the underweight and normal weight BMI categories relative to normal weight non-smoking women in a large, multiethnic cohort of term births. Public health intervention to address prepregnancy underweight or prenatal smoking would be predicted to reduce the burden of SGA more than expected from the singular effects of prepregancy underweight or prenatal smoking on SGA risk. The limited effect on SGA risk and birth weights in term births of women who were overweight or obese prior to pregnancy and smoked prenatally is consistent with the hypothesis that elevated hypertriglyceridemia due to excess adiposity diminishes the toxic effects of prenatal smoking on SGA risk. This does not suggest that women who are overweight or obese prior to pregnancy should smoke or that smokers should gain weight, as overweight and smoking cause numerous other health risks to the mother and her offspring (1,15,35,36,37,38,39,40,41,42).

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Abbreviations

aRD	adjusted risk difference
aRR	adjusted risk ratio
BMI	body mass index
CI	confidence interval
ICD	International Classification of Diseases
SGA	small for gestational age
RD	risk difference
RR	risk ratio

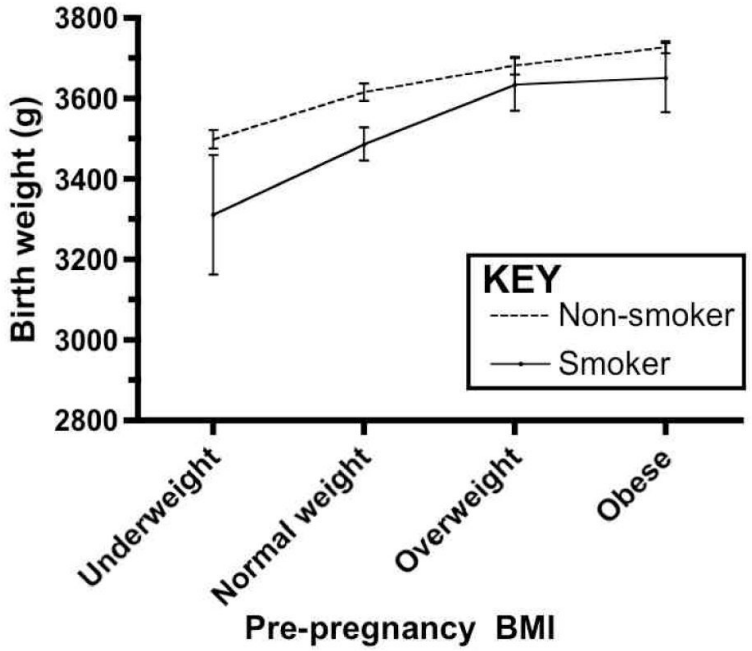

Figure 1.

Interaction between prenatal smoking and prepregnancy BMI in relation to birth weight in 34,928 singleton term births, New York, 1995-2003. The multivariable model was adjusted for race/ethnicity, maternal age (≤ 20, 21-30, 31-40, >40 years), maternal education (< 12, 12 > 12 years), parity (0, 1, ≥ 2), and infant sex. Adjusted birth weights associated with women who did not smoke during pregnancy are depicted by squares/dashed line with 95% confidence intervals. Adjusted birth weights associated with women who smoked during pregnancy are depicted by circles/solid line with 95% confidence intervals.

Table 1

Maternal Characteristics in Relation to Mean Birth Weight and SGA Prevalence in 34,928 Singleton Term Births, New York, 1995-2003.

Characteristic	Full cohort				SGA	
	No.	%	Birth weight (g)		*n* = 3,711	
			Mean	SD	No.	%
Maternal age, years						
≤ 20 years	1,019	2.9	3,287	427	152	14.9
21 – 30 years	15,961	45.7	3,389	458	1,830	11.5
31 – 40 years	16,910	48.4	3,436	474	1,620	9.6
> 40 years	1,038	3.0	3,420	478	109	10.5
Maternal education, years						
< 12	1,849	5.3	3,328	465	273	14.8
12	8,544	24.5	3,404	469	955	11.2
> 12	24,535	70.2	3,418	465	2,483	10.1
Nativity						
US born	20,705	59.3	3,433	469	2,010	9.7
Foreign born	14,223	40.7	3,376	460	1,701	12.0
Ethnic ancestry						
Non-Hispanic white	17,312	49.6	3,472	464	1,408	8.1
Non-Hispanic black	8,345	23.9	3,350	472	1,140	13.7
Hispanic	5,417	15.5	3,378	458	634	11.7
Asia and Pacific Islands	3,555	10.2	3,309	430	478	13.4
Other	299	0.9	3,277	491	51	17.1
Parity						
0	16,228	46.5	3,374	470	2,121	13.7
1	11,741	33.6	3,435	457	1,019	8.7
≥ 2	6,959	19.9	3,450	467	571	8.2
Infant sex						
Male	17,878	51.2	3,469	473	1,948	10.9
Female	17,050	48.8	3,348	452	1,763	10.3
Gestational age, weeks						

Characteristic	Full cohort				SGA	
	No.	%	Birth weight (g)		*n* = 3,711	
			Mean	SD	No.	%
37	2,395	6.9	3,041	451	318	13.3
38	6,027	17.3	3,245	443	730	12.1
39	10,269	29.4	3,391	439	1,234	12.0
40	12,566	36.0	3,504	438	1,218	9.7
≥ 41	3,671	10.5	3,653	442	211	5.8
Prenatal cigarette use						
None	34,121	97.7	3,412	466	3,580	10.5
< ½ pack per day	462	1.3	3,365	465	59	12.8
½ - 1 pack per day	301	0.9	3,252	517	61	20.3
> 1 pack per day	44	0.1	3,252	475	11	25.0
Prepregnancy BMI						
Underweight	1,695	4.8	3,239	427	306	18.0
Normal weight	20,589	59.0	3,382	450	2,327	11.3
Overweight	7,906	22.6	3,459	481	702	8.9
Obese	4,738	13.6	3,509	496	376	7.9
Gestational diabetes						
Absent	34,403	94.8	3,410	463	3,507	10.6
Present	1,821	5.2	3,411	519	204	11.2
Preeclampsia						
Absent	34,403	98.5	3,412	464	3,593	10.4
Present	525	1.5	3,240	564	118	22.5
Non-proteinuric gestational hypertension						
Absent	34,312	98.2	3,411	464	3,612	10.5
Present	616	1.8	3,365	570	99	16.1
Pre-existing chronic hypertension						
Absent	34,364	98.4	3,411	465	3,626	10.6
Present	564	1.6	3,326	539	85	15.1

BMI = body mass index; CI = confidence interval; SGA= small for gestational age birth; SD = standard deviation.

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Table 2

Effect of Prenatal Smoking and Prepregnancy BMI on SGA Risk in 34,928 Singleton Term Births, New York, 1995-2003.

Characteristic	RD, per 100 births			RR		
	Crude	Adjusted[a]	95% CI[a]	Crude	Adjusted[a]	95% CI[a]
Prenatal cigarette use						
None, reference	0[b]	0[c]		1.0	1.0	
< ½ pack per day	+2.3	+2.3	-0.6, +5.3	1.2	1.3	1.0, 1.6
½ - 1 pack per day	+9.8	+9.8	+5.3, +14.4	1.9	2.1	1.6, 2.8
> 1 pack per day	+14.5	+15.4	+2.5, +28.3	2.4	2.8	1.6, 5.2
Prepregnancy BMI						
Underweight	+6.8	+6.1	+4.3, +8.0	1.6	1.5	1.3, 1.7
Normal weight, reference	0[d]	0[b]		1.0	1.0	
Overweight	-2.4	-2.6	-3.3, -1.9	0.8	0.8	0.7, 0.8
Obese	-3.4	-3.0	-3.8, -2.2	0.7	0.7	0.6, 0.8

BMI = body mass index; CI = confidence interval; RD = risk difference; RR = risk ratio.

[a] Adjusted for prepregnancy BMI, prenatal cigarette use, race/ethnicity, maternal age, maternal education, and parity.

[b] The reference crude risk = 10.5.

[c] The reference adjusted risk = 6.1 (95% CI: 5.4, 6.8).

[d] The reference crude risk = 11.3.

Table 3

Interaction Between Prenatal Smoking and Prepregnancy BMI in Relation to SGA Risk in 34,928 Singleton Term Births, New York, 1995-2003.

Prepregnancy BMI	No.		Additive model				Multiplicative Model			
	Non-smoker	Smoker	Non-smoker		Smoker		Non-smoker		Smoker	
			aRD[a]	95% CI[a]	aRD[a]	95% CI[a]	aRR[a]	95% CI[a]	aRR[a]	95% CI[a]
Underweight	1,659	36	+6.1	+4.2, +8.0	+17.2	+2.7, +31.6	1.5	1.3, 1.6	2.5	1.5, 4.1
Normal weight	20,112	477	0[b]		+8.5	+4.9, +12.0	1.0		1.9	1.5, 2.2
Overweight	7,720	186	-2.5	-3.2, -1.8	-0.1	-4.3, +4.1	0.8	0.7, 0.9	1.0	0.6, 1.5
Obese	4,630	108	-2.9	-3.7, -2.1	-2.4	-7.7, +2.8	0.7	0.6, 0.8	0.9	0.5, 1.6

aRD = adjusted risk difference per 100 births; aRR = adjusted risk ratio; BMI = body mass index; CI = confidence interval.

[a]Adjusted for race/ethnicity, maternal age, maternal education, and parity.

[b]The reference adjusted risk = 6.1 (95% CI: 5.4, 6.8).

Table 4

Interaction Between Prenatal Smoking and Prepregnancy BMI in Relation to 34,928 Term, Singleton Birth Weights (g), New York, 1995-2003.

Prepregnancy BMI	Non-smoker		Smoker	
	Difference, g[a]	95% CI[a]	Difference, g[a]	95% CI[a]
Underweight	-117	-140, -94	-304	-453, -156
Normal weight	0[b]		-129	-170, -87
Overweight	+66	+54, +78	+19	-46, +85
Obese	+111	+97, +126	+36	-50, +122

BMI = body mass index; CI = confidence interval.

[a] Adjusted for race/ethnicity, maternal age, maternal education, parity, and infant sex.

[b] The reference birth weight = 3,615 (95% CI: 3,594, 3,637).