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Change in Body Mass Index Between Pregnancies and the Risk of Gestational Diabetes in a Second Pregnancy

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Abstract

Objective—To estimate the association between inter-pregnancy change in body mass index (BMI) and the risk of gestational diabetes (GDM) in a second pregnancy.

Methods—In a retrospective cohort analysis of 22,351 women, logistic regression models provided adjusted estimates of the risk of GDM in women gaining 3.0 or more, 2.0-2.9, and 1.0-1.9 BMI units or losing 1.0-2.0 and more than 2.0 units between pregnancies [one BMI unit corresponds to 5.9 pounds for the average height (5 feet 4 inches) of the study population]. Women with stable BMI ($\pm$ 1.0 BMI unit) comprised the reference.

Results—Compared to women who remained stable, inter-pregnancy BMI gains were associated with an increased risk of GDM in the second pregnancy [OR= 1.71 (95% confidence interval 1.42-2.07) for gaining 1.0-1.9 BMI units, OR= 2.46 (95% confidence interval 2.00-3.02) for 2.0-2.9 BMI units, and OR= 3.40 (95% confidence interval 2.81-4.12) for 3.0 or more BMI units]. The loss of BMI units was associated with a lower risk of GDM only among women who were overweight/obese in the first pregnancy [OR= 0.26 (95% confidence interval 0.14-0.47) for the loss of more than 2.0 BMI units]. In overweight/obese women, those with GDM in the first pregnancy that did not develop the condition again gained fewer BMI units than those experiencing recurrent GDM [mean change 0.66 (95% confidence interval 0.25-1.07) vs. 2.00 (95% confidence interval 1.56-2.43) BMI units, respectively].

Conclusion—Inter-pregnancy increases in BMI may increase a woman's risk of GDM pregnancy; reductions in BMI may be protective, particularly in overweight/obese women.

Introduction

Gestational diabetes mellitus (GDM), defined as carbohydrate intolerance with first onset or recognition in pregnancy,(1) is associated with an increased risk of adverse perinatal outcomes (2;3) as well as subsequent diabetes in women and their offspring.(4;5) The prevalence of GDM in the U.S. is 4-7%,(6;7) with ~10-95% increases in prevalence reported over the last two decades.(8-12) Emulating established risk factors for type 2 diabetes,(13) risk factors for GDM include advanced age, race-ethnicity, a family history of diabetes and obesity.(3;14)

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Excessive postpartum weight retention and lifestyle changes have been associated with a woman being overweight years after pregnancy,(15) thereby increasing her risk of developing type 2 diabetes. Pre-gravid weight gain (16) and gestational weight gain (17) have similarly been shown to increase the risk of GDM. Yet whether pregravid weight loss reduces the risk of GDM remains unknown. A pregnancy complicated by GDM is associated with a high risk of recurrent GDM in a subsequent pregnancy,(18) but potential modification of this risk by inter-pregnancy weight gain and loss has also not been explored.

The present study examines a diverse cohort of women from Kaiser Permanente Northern California (KPNC), with and without GDM in their first pregnancy, to estimate the association between inter-pregnancy change in BMI and the risk of GDM in a second pregnancy. It was hypothesized that the loss of BMI units between pregnancies would reduce the risk of complication recurrence, while gaining BMI units would increase the risk of GDM in a second pregnancy.

Materials and Methods

The study setting is KPNC, a large group-practice prepaid health plan that provides comprehensive medical services to members residing in a 14 county region. Approximately 30% of the population residing in the area served by KPNC is enrolled in the health plan. In this setting, 94% of women delivering live-born singletons undergo the recommended 50-g, 1-hr GCT screening for GDM (19) (hereafter referred to as the screening test). Women with plasma glucose values ≥ 7.8 mmol/L (140 mg/dl) on the screening test go on to receive a diagnostic 100-g, 3-hr OGTT (hereafter referred to as the diagnostic test). GDM was defined according to the American Diabetes Association (ADA) plasma glucose thresholds (20) for the diagnostic test, two or more values meeting or exceeding the following cut points: fasting 5.3 mmol/L (95 mg/dl); 1-hour 10.0 mmol/L (180 mg/dl); 2-hour 8.6 mmol/L (155 mg/dl); 3-hour 7.8 mmol/L (140 mg/dl). Plasma glucose values were obtained from the KPNC clinical laboratory database, which captures the results of all laboratory tests performed at any KPNC facility. A previous study during the study period in this setting (19) conducted a medical chart review on over 2,500 pregnancies and reported glucose values in the database to be 99.4% accurate. All plasma glucose measurements were performed using the hexokinase method at the KPNC regional laboratory, which participates in the College of American Pathologists' accreditation and monitoring program.

Using the KPNC Pregnancy Glucose Tolerance Registry (12), 48,534 women without recognized diabetes prior to pregnancy who were 15 to 45 years of age at first pregnancy and delivered their first and second live-born singletons at KPNC between 1996 and 2006 were identified. Women missing data on weight at either pregnancy were excluded [n= 5,409 missing weight (11.1%) in the first pregnancy, n= 7,266 (15.0%) missing weight in the second pregnancy, and n= 6,002 (12.4%) missing weight in both pregnancies]. An additional 6,131 women (12.6%) missing data on height were also excluded. Women missing plasma glucose values for the screening test or the diagnostic test and those with abnormal screening results but no diagnostic test on record were also excluded (n= 1,325; 2.7%), as were those missing data on gestational age at the weight measurements and mother's place of birth (n= 50; 0.1%), leaving a final analytic cohort of 22,351 women (Figure 1).

The distribution of potential outcomes (GDM in the first pregnancy/second pregnancy: yes/no, no/no, yes/yes and no/yes) did not differ significantly between women with and those without data on weight and height in the electronic medical records (data not shown). African American and Hispanic women were slightly more likely to be excluded from the

analytic cohort because they were missing these data (43.7% and 45.9% with weight and height data vs. 56.3% and 54.1% without these data, respectively).

Pregnancy body weight and maternal height were obtained from the KPNC electronic medical records. Pregnancy weight was measured by a KPNC clinician in conjunction with the Alpha-fetoprotein (AFP) test that occurs in the second trimester [mean gestational age at the AFP test in the first pregnancy was 16.7 weeks (SD 1.3); in the second pregnancy, it was 16.9 weeks (SD 1.3)]. Body mass index (BMI) at each pregnancy was calculated as the maternal weight (kilograms) in that pregnancy divided by the height (meters) squared. Body mass index in the first pregnancy was subtracted from that in the second pregnancy to estimate the inter-pregnancy change in BMI units (kg/m^2). Gestational age at the weight measurements for both pregnancies were also taken from the KPNC electronic medical records. A previous study in this setting during the same period (21) reported 98.5% agreement between gestational age in the electronic medical records and estimates from ultrasonographic data collected prior to 24 weeks. The interval between pregnancies was calculated as the years between delivery dates. Self reported California birth certificate. Data on smoking were only available in the electronic medical records starting in 2001, thus sensitivity analyses were conducted among those whose first pregnancies occurred in 2001 or beyond ($n = 17,768$ or 79.5%).

The direct method of adjustment, with the study cohort as the standard, was used to calculate the age-adjusted risk of GDM in a second pregnancy. To estimate the risk of GDM in a second pregnancy associated with inter-pregnancy changes in maternal BMI, we constructed a logistic regression model that adjusted for maternal age at the first delivery (continuous), race-ethnicity (Caucasian as the reference, African American, Asian, Hispanic, Other and Unknown), and place of birth (outside the U.S. vs. the U.S.), GDM (yes/no) and BMI (continuous) in the first pregnancy, gestational age (continuous) at the weight ascertainment of both pregnancies and the time interval between pregnancies. Women were divided into the following exposure 13 categories: those who gained 3.0 or more, 2.0-2.9, or 1.0-1.9 BMI units, and those who lost 1.0-2.0 or more than 2.0 BMI units, with women who remained stable between pregnancies (± 1.0 unit) serving as the reference. For the average height of the study population, 5 feet 4 inches, one BMI unit corresponded to 5.9 pounds. Sensitivity analyses with further adjustment for smoking between pregnancies were conducted within the subset with smoking data available.

We explored variation in the association between BMI change and the risk of GDM in a second pregnancy by maternal age at first delivery (≤ 30 or > 30 years of age), race-ethnicity (Caucasian, Hispanic, African American, and Asian), and overweight/obese status in the first pregnancy ($\text{BMI} < 25.0 \text{ kg/m}^2$ vs. $\geq 25 \text{ kg/m}^2$) by separately adding interaction terms to fully-adjusted models. The interaction terms for age and race-ethnicity were not significant (p values 0.53 and 0.32, respectively), but the interaction term for overweight/obese status in the first pregnancy did attain statistical significance (P value = 0.04); thus we present results both pooled and stratified by overweight/obese status in the first pregnancy.

We also compared the mean change in BMI units between pregnancies for the following categories of women: women with GDM in the first pregnancy but not the second, those free of GDM in both pregnancies, women with GDM in both pregnancies, and women without GDM in their first pregnancy that developed the condition in their second, with additional stratification by overweight/obese status in the first pregnancy ($\text{BMI} < 25.0 \text{ kg/m}^2$ vs. $\geq 25 \text{ kg/m}^2$). Mean BMI change was estimated for each group after adjustment for age, race-ethnicity, place of birth, gestational age at the weight measurements, and time interval between pregnancies.

SAS 9.1 (SAS Institute Inc., Cary, NC) was used for all analyses. This study was approved by the human subjects committees of KPNC and the State of California.

Results

GDM occurred in 4.6% of the cohort in the first pregnancy, 5.2% in the second pregnancy and 1.8% in both pregnancies. Table 1 displays cohort characteristics assessed at the first pregnancy by GDM status in the second pregnancy. Overall, women who had GDM in the second pregnancy were older, less likely to be Caucasian, and more likely to be born outside of the U.S., to be overweight or obese at the first pregnancy and to gain BMI units between pregnancies compared to women who did not have GDM in the second pregnancy.

Less than 10% of the cohort lost BMI units between pregnancies and 53% gained BMI units. Caucasian women and those who had attained higher levels of education were more likely to lose BMI units between pregnancies. African American and Hispanic women, as well as those who had attained lower levels of education, were more likely to have gained BMI units between pregnancies (data not shown).

For those with GDM in their first pregnancy, the age-adjusted risk of GDM in the second pregnancy was 38.19% (95% CI 34.96-41.42) and for those whose first pregnancy was not complicated by GDM, the risk was 3.52% (95% CI 3.27-3.76). In an analysis adjusted for age, race-ethnicity, place of birth, GDM and BMI in the first pregnancy, gestational age at both pregnancy weight measurements and the interval between pregnancies, women with GDM in the first pregnancy had 17 times the risk of developing GDM again in the second pregnancy [OR= 16.55 (95% CI 14.08-19.45)] as compared to those without GDM in their first pregnancy. The risk of GDM recurrence did not differ between normal weight (BMI < 25.0 kg/m²) and overweight/obese (BMI ≥ 25.0 kg/m²) women (data not shown).

Adjusted risk estimates for GDM in the second pregnancy by change in BMI units between pregnancies are presented in Table 2. In the adjusted analysis that included all women, those who gained BMI units between pregnancies had an increased risk of GDM in the second pregnancy relative to those whose BMI remained stable, and the risk estimates increased with increasing gains in BMI units; women who lost BMI units between pregnancies significantly decreased their risk of GDM in the second pregnancy (*P* value for linear trend across groups <0.001). However, maternal overweight/obesity status (BMI ≥ 25.0 kg/m² vs. < 25.0 kg/m²) in the first pregnancy significantly modified the association (*P* value = 0.04). Stratification by overweight/obesity status in the first pregnancy revealed that the loss of BMI units was only significantly associated with a reduced risk of GDM in the second pregnancy among women who were overweight or obese in their first pregnancy. Among women with BMI ≥ 25.0 kg/m² in their first pregnancy, the loss of > 2.0 BMI units between pregnancies decreased the risk of GDM in the second pregnancy by 74% [OR = 0.26 (95% CI 0.14-0.47)]. Increases in the risk of GDM in the second pregnancy were observed for increasing gains in BMI between pregnancies, regardless of overweight/obese status in the first pregnancy. Risk estimates were slightly higher among women who were normal weight (BMI < 25.0 kg/m²) in their first pregnancy and gained BMI units between pregnancies as compared to overweight/obese women (BMI ≥ 25.0 kg/m²), but not significantly so; normal weight women almost doubled their risk of GDM in the second pregnancy with gains of only 1.0-1.9 BMI units [OR = 1.90 (95% CI 1.44-2.49)].

Sensitivity analyses were conducted amongst a subset of women for whom smoking data were available (*n*= 17,768, 79.5%). The results of fully adjusted models for these subsets did not differ substantially from those presented for the full analytic cohorts (data not shown).

Figure 2 presents the adjusted mean change in BMI units between pregnancies for all combinations of GDM in the first and the second pregnancies, by maternal overweight/obesity status ($\text{BMI} \geq 25.0 \text{ kg/m}^2$ vs. $< 25.0 \text{ kg/m}^2$) in the first pregnancy. Overall, women who had GDM in the first pregnancy but not the second pregnancy gained the fewest BMI units and those who did not have GDM in the first pregnancy that went on to develop GDM in the second pregnancy gained the most BMI units. However, among women who were overweight or obese, those with GDM in their first pregnancy that did not develop the condition again in their second pregnancy gained the fewest BMI units between pregnancies of any group [mean change= 0.66 units (95% CI 0.25-1.07)]; these women also gained significantly fewer BMI units than the overweight or obese women experiencing recurrent GDM [mean change= 2.00 BMI units (95% CI 1.56-2.43)]. In normal weight women, those with GDM in their first pregnancy that did not develop the condition again in their second pregnancy gained a mean 1.46 BMI units (95% CI 1.17-1.76); this was similar to the mean gain in BMI units observed among women with GDM in both pregnancies. Women free of GDM in the first pregnancy that went on to develop the condition in their second pregnancy gained the most BMI units in both women who were normal weight and those who were overweight or obese in the first pregnancy [pooled mean change = 2.84 units (95% CI 2.61-3.08)].

Discussion

Our findings suggest that women who gain BMI units between their first and second pregnancies are at an increased risk for developing GDM in the second pregnancy. Women who lose BMI units between pregnancies appear to have a decreased risk of GDM in their second pregnancy, but there was significant variation by maternal overweight/obese status in the first pregnancy: the loss of BMI units was only significantly protective of GDM in a second pregnancy among women who were overweight or obese in their first. Although most women gained BMI units between pregnancies, the group with the lowest average gain was women who were overweight or obese and had GDM in their first pregnancy but did not develop GDM again in their second. Our results also suggest that the effects of body mass gains may be greater among women of normal weight in their first pregnancy, while the effects of losses in body mass appear greater among overweight/obese women. Taken together, these results support the avoidance of gestational weight retention and postpartum weight gain to decrease the risk of GDM in a second pregnancy as well as the promotion of postpartum weight loss in overweight/obese women, particularly those with a history of GDM.

In this study, GDM in the first pregnancy was the strongest predictor of recurrent GDM in the second pregnancy. Getahun et al. (18) reported that compared to women who were free of GDM in their first pregnancy, those experiencing the condition in their first pregnancy had a 13 fold increased risk of GDM in a second pregnancy. They also found that compared to women free of GDM in their first and their second pregnancies, those with GDM in their first but not their second had a 6 fold increased risk of GDM in the third pregnancy, those without GDM in the first pregnancy who experienced GDM in their second had a 15 fold increased risk of GDM in the third pregnancy, and those with GDM in their first two pregnancies had a 26 fold increased risk of GDM in the third pregnancy. The association between inter-pregnancy changes in body mass and GDM risk in third and higher order pregnancies remains to be investigated.

Our results are also in accordance with the only other study to examine inter-pregnancy changes in BMI and the risk of GDM. Villamor et al. (22) reported that among women who were free of GDM in their first pregnancy, the risk of GDM in a second pregnancy began to rise with inter-pregnancy gains of 1.0-2.0 BMI units and the risk continued to increase with

additional gains. Although increasing gains in BMI elevated the risk of GDM for all women, significantly stronger associations were observed for those with a normal BMI in their first pregnancy as compared to overweight/obese women. Villamor et al. did not report an association between inter-pregnancy decreases in BMI and the reduction of GDM risk and were unable to assess inter-pregnancy BMI change in relation to recurrent GDM.

The second half of pregnancy is characterized by progressive insulin resistance, hyperinsulinemia, and mild postprandial hyperglycemia. Most women are able to increase their insulin secretion to compensate for this insulin resistant state and maintain normal glucose tolerance.(13) However, those women requiring the hypersecretion of insulin to compensate for pregnancy induced insulin resistance may experience β -cell exhaustion and GDM.(13) Yet there is some evidence that weight gain may also result in insulin resistance in the non-pregnant state.(23) Therefore, gains of 2 or more BMI units (which in this cohort, corresponds to 11.8 pounds) between pregnancies may contribute to added β -cell exhaustion and result in an inadequate secretion of insulin for the level of insulin resistance induced by a second pregnancy. On the contrary, inter-pregnancy reductions in body mass may improve insulin sensitivity, leaving women with improved β -cell function and thus better able to compensate for the insulin resistance brought on by their second pregnancy. In regards to the modification of effect by maternal overweight/obesity status, it should be noted that pre-pregnancy overweight or obesity are strong, well-established risk factors for GDM,(14) thus normal weight women experiencing the condition are likely to be more genetically susceptible. The finding that decreases in body mass did not significantly reduce the risk of GDM in a second pregnancy among normal weight women may be due to their increased genetic risk.

There are several limitations to the current study. First, body weight measured during pregnancy was used to calculate BMI and thus, estimate the change in BMI units between pregnancies. This strategy obscures the effect of gestational weight gain, which may affect the risk of GDM.(24) Height and weight data were not universally available, due to the usual confines of retrospective studies and because not all women had the AFP test. Also characteristic of retrospective studies, data on several potential confounding factors were unavailable, including physical activity, diet, and breastfeeding. Yet there are several strengths to be noted. The availability of plasma glucose values from a single regional laboratory suggests that the misclassification of GDM is unlikely. Previous work (22) has relied on International Classification of Diseases (ICD) codes to ascertain GDM status, a source that is more prone to misclassification. Lastly, the racial and ethnic diversity of this cohort make our results highly generalizable.

Our findings suggest that gains in body mass prior to pregnancy could increase a woman's risk of GDM while reductions in body mass, particularly in overweight/obese women, could protect against the complication. These results are particularly relevant to women with a history of GDM, who are at increased risk of developing GDM again in a subsequent pregnancy.(25) Randomized trials investigating the efficacy of weight loss/weight maintenance interventions in preventing subsequent GDM remain to be conducted.

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Figure 1.
Assembly of the analytic cohort, Kaiser Permanente Northern California Pregnancy Glucose Tolerance Registry.

Figure 2.

Mean interpregnancy change in body mass index (BMI) units, by BMI in the first pregnancy and gestational diabetes status in the first and second pregnancies. Adjusted for age, race-ethnicity, place of birth, gestational age at the weight measurements, and interval between pregnancies. Kaiser Permanente Northern California Pregnancy Glucose Tolerance Registry, 1996 to 2006. * $P < .001$ for difference in the mean change in BMI units between women who were overweight or obese in the first pregnancy and women who were of normal weight in the first pregnancy among women who had gestational diabetes in the first pregnancy, but not the second pregnancy.

Table 1

Women's characteristics at first pregnancy by gestational diabetes status in the second pregnancy; Kaiser Permanente Northern California Pregnancy Glucose Tolerance Registry, 1996-2006.

	GDM in the Second Pregnancy		
	No n = 21,201	Yes n = 1,150	p-value *
Characteristic	n (%)	n (%)	
Age at first delivery (years)			<0.01
< 20	1,924 (9.1%)	44 (3.8%)	
20-24	4,978 (23.5%)	163 (14.2%)	
25-29	8,144 (38.4%)	468 (40.7%)	
30-34	5,533 (26.1%)	400 (34.8%)	
35-39	592 (2.8%)	70 (6.1%)	
40+	30 (0.1%)	5 (0.4%)	
Race-ethnicity			<0.01
Caucasian	9,836 (46.4%)	328 (28.5%)	
Hispanic	4,926 (23.2%)	301 (26.2%)	
African American	1,393 (6.6%)	55 (4.8%)	
Asian	4,961 (23.4%)	462 (40.2%)	
Other	59 (0.3%)	3 (0.3%)	
Unknown	26 (0.1%)	1 (0.1%)	
Education			0.10
Less than high school	1,545 (7.3%)	91 (7.9%)	
High school graduate	4,948 (23.3%)	248 (21.6%)	
College	10,185 (48.0%)	585 (50.9%)	
Graduate studies	4,135 (19.5%)	201 (17.5%)	
Missing	388 (1.8%)	25 (2.2%)	
Mother's place of birth			<0.01
Outside the United States	7,156 (33.8%)	606 (52.7%)	
United States	14,045 (66.2%)	544 (47.3%)	
Smoking between pregnancies			0.89
No	14,985 (70.7%)	829 (72.1%)	
Yes	1,853 (8.7%)	101 (8.8%)	
Missing	4,363 (20.6%)	220 (19.1%)	
Time between Pregnancies (years)			<0.01
< 2	6,144 (29.0%)	278 (24.2%)	
2 to 4	11,065 (52.2%)	568 (49.4%)	
4 to 6	2,908 (13.7%)	216 (18.8%)	
≥ 6	1,084 (5.1%)	88 (7.7%)	
BMI (kg/m²) in the first			<0.01

	GDM in the Second Pregnancy		
	No n = 21,201	Yes n = 1,150	p-value*
pregnancy			
≤ 18.4	521 (2.5%)	13 (1.1%)	
18.5-24.9	11,776 (55.5%)	463 (40.3%)	
25.0-29.9	5,837 (27.5%)	365 (31.7%)	
≥ 30.0	3,067 (14.5%)	309 (26.9%)	
Change in BMI units between first and second pregnancies			<0.01
Lost ≥ 2.0	774 (3.7%)	20 (1.7%)	
Lost 1.0-1.9	1,228 (5.8%)	37 (3.2%)	
Remained stable (± 1.0)	8,236 (38.8%)	295 (25.7%)	
Gained 1.0-1.9	4,317 (20.4%)	264 (23.0%)	
Gained 2.0-2.9	2,785 (13.1%)	204 (17.7%)	
Gained ≥ 3.0	3,861 (18.2%)	330 (28.7%)	

*
p-value does not include missing category

GDM = gestational diabetes mellitus

BMI = body mass index

Table 2

Odds ratios for gestational diabetes mellitus in a second pregnancy associated with change in BMI units between the first and second pregnancies, for all women and stratified by body mass index in the first pregnancy, Kaiser Permanente Northern California Pregnancy Glucose Tolerance Registry, 1996-2006.

	N	Unadjusted OR OR (95% CI)	Adjusted ^{a,b} (95% CI)
All women ^a	22,351		
Change in BMI units			
Lost > 2.0	794	0.72 (0.46-1.14)	0.32 (0.20-0.54)**
Lost 1.0-2.0	1,265	0.84 (0.59-1.19)	0.61 (0.42-0.90)*
Remained stable ($\pm$ 1.0)	8,531	1.00	1.00
Gained 1.0-1.9	4,581	1.71 (1.44-2.02)**	1.71 (1.42-2.07)**
Gained 2.0-2.9	2,989	2.05 (1.70-2.46)**	2.46 (2.00-3.02)**
Gained $\geq$ 3.0	4,191	2.39 (2.03-2.80)**	3.40 (2.81-4.12)**
<i>P</i> for trend		<0.001	<0.001
BMI < 25 kg/m² at first pregnancy ^b	12,773		
Change in BMI units			
Lost > 2.0	162	2.00 (0.92-4.36)	1.18 (0.48-2.91)
Lost 1.0-2.0	651	0.62 (0.31-1.23)	0.53 (0.26-1.10)
Remained stable ($\pm$ 1.0)	5,534	1.00	1.00
Gained 1.0-1.9	2,838	2.04 (1.59-2.63)**	1.90 (1.44-2.49)**
Gained 2.0-2.9	1,644	2.66 (2.02-3.50)**	2.91 (2.16-3.93)**
Gained $\geq$ 3.0	1,944	2.92 (2.26-3.78)**	4.47 (3.34-6.00)**
BMI $\geq$ 25 kg/m² at first pregnancy ^b	9,578		
Change in BMI units			
Lost > 2.0	632	0.34 (0.19-0.61)**	0.26 (0.14-0.47)**
Lost 1.0-2.0	614	0.78 (0.52-1.17)	0.62 (0.39-0.98)*
Remained stable ($\pm$ 1.0)	2,997	1.00	1.00
Gained 1.0-1.9	1,743	1.41 (1.12-1.78)**	1.50 (1.16-1.96)**
Gained 2.0-2.9	1,345	1.47 (1.15-1.88)**	2.11 (1.59-2.78)**
Gained $\geq$ 3.0	2,247	1.68 (1.37-2.07)**	2.84 (2.21-3.64)**

OR, odds ratio; CI, confidence interval.

^aModel adjusted for age, race-ethnicity, place of birth, GDM and BMI in the first pregnancy, gestational age at the weight measurements, and time interval between pregnancies.

^bModels adjusted for age, race-ethnicity, place of birth, GDM in the first pregnancy, gestational age at the weight measurements, and time interval between pregnancies.

* Significant at $p < 0.05$

** Significant at $p < 0.01$