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Impact of inter-pregnancy BMI change on perinatal outcomes: a retrospective cohort study

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Abstract:

Objective: To examine the patterns and predictors of inter-pregnancy body mass index (BMI) change and its impact on perinatal outcomes in the second pregnancy.

Design: Retrospective cohort study

Setting: Tertiary teaching hospital in Adelaide, Australia

Population: Women with their first and second consecutive, singleton deliveries occurring between 2000 and 2012 (N= 5,371).

Methods: Inter-pregnancy weight change calculated based on difference between BMI at respective antenatal booking visits. Association between inter-pregnancy weight change and perinatal outcomes investigated using multivariate generalised linear models, with stratification according to initial maternal BMI category in first pregnancy.

Main outcome measures: Gestational diabetes (GDM); pregnancy induced hypertensive disorders; small-for-gestational age (SGA); preterm birth; large-for-gestational age (LGA) and macrosomia (>4,500g).

Results: On average, women with a normal BMI gained 1kg/m^2 between first and second pregnancies, while women who were overweight or obese gained 1.37kg/m^2. Among women with a normal BMI in their first pregnancy, a BMI increase of $\geq$4kg/m^2 was associated with increased risk of developing GDM (aRR 1.97; 95% CI 1.22-3.19), a macrosomic (aRR 4.06; 95% CI 2.25-7.34) or LGA infant (aRR 1.31 0.96-1.78) in the second pregnancy, while a reduction in BMI ($\leq$-2kg/m^2) was associated with an increased risk of SGA (aRR 1.94; 1.19-3.16). Among women who were overweight or obese in their first pregnancy, a BMI increase of $\geq$2 to 4 and $\geq$4 kg/m^2 was associated with increased risks of developing GDM in the second pregnancy (aRR 1.39; 95%CI 1.01-1.91 and aRR 1.64 95%CI 1.16-2.31; p_{trend} <0.001), while no associations were observed for a BMI increase and risk of a macrosomic, SGA, or LGA infant. In contrast, reduction in BMI ($\leq$-2kg/m^2) was associated with a reduced risk of GDM (aRR 0.58 95% CI 0.37-0.90) and SGA (aRR 0.47; 95%CI 0.25-0.87).

Conclusion: Increases in BMI between pregnancies is associated with an increased risk for perinatal complications, even in normal-weight women, while a reduction in BMI is associated with improved perinatal outcomes among women who are overweight/obese. Inter-pregnancy weight control is an important target to reduce the risk of an adverse perinatal outcome in a subsequent pregnancy.

Introduction

Obesity is a growing problem worldwide with significant morbidity and public health consequences. In 2008, 56% of Australian women over 20 years old were overweight or obese [1] and 26.2% and 23.8% of pregnant South Australian women were overweight or obese at their 10 week booking visit, respectively [2].

It is well established that entering pregnancy with a high BMI is associated with numerous complications, including hypertensive disorders of pregnancy, thromboembolic disorders, infection, caesarean section and stillbirth [2-10]. Furthermore, infants born to overweight and obese women are more likely to be macrosomic, diagnosed with a congenital anomaly or require neonatal intensive care [2-10].

Villamor and Cnattingius demonstrated in 2006 that increasing weight gain between first and second pregnancies increased the likelihood of developing gestational diabetes (aOR 2.09 (1.68-2.61) per >3 BMI unit increase) and preeclampsia (aOR 1.78 (1.52-2.08) per >3 BMI unit increase), in Swedish women of any initial BMI category. A sub analysis of women with a normal booking BMI <25kg/m^2 demonstrated that the risk of adverse perinatal outcomes increased with significant weight gain, even if the weight gain did not cause the woman to become overweight[11], i.e. women who may have gained more than 3 BMI units but still remained within the normal weight category ($\leq$25kg/m^2).

Subsequently, multiple studies have investigated the effect of inter-pregnancy weight change on the risk of maternal and neonatal complications in Europe and the United States [12-19]. Previous findings have been inconsistent, but generally showed that inter-pregnancy weight gain in women of normal BMI in their first pregnancy was associated with adverse perinatal outcomes in the second pregnancy. To date, there are limited studies specifically examining the impact of BMI loss between pregnancies, which could be expected to have a protective effect on subsequent perinatal outcomes among women who are overweight or obese in their first pregnancy. Children born to women following anti-obesity surgery enjoy a significant reduction in severe obesity, insulin resistance and cardiometabolic markers, when compared to those born prior to maternal surgery, which is sustained into adolescence. This suggests that a benefit is most likely conferred, and suggests that these benefits may extend far beyond the neonatal period [20].

Therefore, the purpose of this study is to investigate patterns and predictors of weight change between pregnancies in a contemporary Australian cohort, and the impact of such changes on subsequent perinatal outcomes in the second pregnancy.

Methods

We conducted a retrospective cohort study utilising routinely collected data on all births occurring in the Women's and Children's Health Network (WCHN) in South Australia, Australia. The project was approved by the Human Research Ethics Committees of the Women's and Children's Health Network and the University of Adelaide in South Australia (ID REC2219/10/14).

All women who had their first two consecutive singleton births between January 2000 and December 2012 at the Women's and Children's Hospital (WCH) in Adelaide, South Australia were included (N=8,368). Women were excluded if their BMI was not recorded during either pregnancy (N=2,596), and if their BMI was < 18.5 kg/m^2 during their first pregnancy (N=218). Outliers were assessed and 183 women were excluded with a height difference of >5cm between pregnancies, leaving a final cohort of 5,371 women. Weight was not routinely recorded at time of delivery, and subsequently, the effect of gestational weight gain was not investigated.

Outcomes were based on data routinely collected and recorded in the WCH Perinatal Statistics Collection. Data is collected on the pregnancy and outcome of every live birth and late fetal death occurring at the WCH. Data are collected according to the guidelines of the Pregnancy Outcome Unit of the South Australian Department of Health for the Supplementary Birth Records (SBRs) and in consultation with the senior clinicians at the hospital. Data collection began in the late 1980s and complete, validated data are available from 1990 onwards. Data are collected by a specially trained research midwife from the women's medical records following delivery through use of a structured coding sheet. It is important to note that included in the medical record is the South Australian Pregnancy Record (SAPR), which is a hand-held antenatal record carried by each woman throughout her pregnancy and contains notes by all health providers consulted during the pregnancy. From the medical records, information is collected on maternal illnesses (e.g. diabetes, gestational diabetes, epilepsy, asthma and psychiatric illness), lifestyle factors (e.g. smoking), obstetric history, course of delivery, pregnancy complications and newborn characteristics (e.g. birth weight). Inter-pregnancy interval was calculated from delivery to conception. All SBRs are checked manually for completeness and data discrepancies and then go through a series of automated validation procedures during data entry. The information in the perinatal statistics collection has been previously validated and has been shown to be very reliable when compared with hospital medical records [21], and has been utilised in previous studies examining perinatal outcomes [22-24]

Weight change was measured in terms of BMI units between the booking visit of the first and second pregnancy, women who had their first antenatal visit after 15 weeks gestation did not have a booking weight recorded.

Baseline characteristics and determinants of inter-pregnancy weight gain were assessed. Outcomes included were gestational diabetes (diagnosis as marked on data collection sheet, OGTTs not); pregnancy induced hypertensive disorders (diagnosis as marked on data collection sheet: preeclampsia, gestational hypertension, chronic hypertension, superimposed preeclampsia) small-for-gestational age (SGA - <10th centile customised for maternal height, weight, fetal sex, gestation and ethnicity); large-for-gestational age (LGA - >10th centile customised for maternal height, weight, gestation and ethnicity), macrosomia >4500g, and preterm birth.

Women were grouped according to the world health organisation (WHO) BMI classifications; normal weight 18.5-25 kg/m^2, overweight 25-30 kg/m^2 obese >30 kg/m^2. We hypothesised that weight change might behave physiologically differently when occurring in a woman who was already overweight, with a possible different magnitude of effect on GDM, hypertensive disorders of pregnancy and birthweight, as she moves either closer to or further away from the healthy weight range. Thus, overweight and obese women were analysed separately to women of normal BMI.

Statistical Analysis

Baseline characteristics and determinants of inter-pregnancy BMI change were assessed using a Kruskal-Wallis test. The impact of BMI change between pregnancies on subsequent perinatal outcomes in the second pregnancy were compared using a generalised linear model (Poisson distribution) with robust variance estimates, with resulting relative risks (RR) and 95% confidence intervals (CIs). Inter-pregnancy BMI change was included as a categorical outcome and defined as ≤-2, >-2 to <2, ≥2 to <4, and ≥4 kg/m^2, with a weight change of >-2 to <2 used as the reference category. Analyses were stratified according to initial maternal BMI category in the first pregnancy (normal weight [BMI<25 kg/m^2] or overweight/obese [BMI≥25 kg/m^2]). Analyses were adjusted for maternal covariates including age, socioeconomic status, maternal BMI in first pregnancy, smoking status, race, pregnancy interval time, and perinatal outcomes in the first pregnancy including gestational diabetes, pregnancy induced hypertensive disorders, birth method, and appropriateness of fetal growth (i.e. LGA or SGA). We also estimated incidence of each outcome during the second pregnancy by categories of change in BMI from the first pregnancy, and tested the linearity of the associations using the Cochran-Armitage test. A linear trend was also investigated in the multivariate generalised linear model using the Wald test for change in BMI when the ordinal variable was introduced into the logistic regression model as a continuous predictor. The potential interaction between baseline BMI category in the first pregnancy and interpregnancy BMI change was investigated by including an interaction term in the multivariate generalised linear model. Statistical analyses were performed using Stata 11.1 (Stata, College Station, TX, USA). Statistical significance was defined as a 2-sided *P* value of < 0.05.

Results

Among the total eligible cohort of 5,371 women, 3247 (61%) were of normal weight, 1302 (24%) were overweight, and 822 (15%) were obese entering their first pregnancy. Maternal characteristics associated with each BMI category are presented in Supplemental Table 1.

Among women who were overweight or obese in their first pregnancy, 94% remained overweight or obese in their second pregnancy (Table 1). Mean gestation at booking visit did not differ between the first and second pregnancy for women included in the study (11.4 vs 11.3 weeks; p=0.62).

Inter-pregnancy BMI change followed an approximate normal distribution among women who were of normal weight, or overweight/obese in their first pregnancy (Figure 2). On average, women with a normal BMI gained 1kg/m^2 between first and second pregnancies, while women who were overweight or obese gained 1.37kg/m^2.

A number of maternal characteristics and perinatal outcomes in the first pregnancy, were associated with changes in inter-pregnancy BMI (Table 2 and 3). Younger women, single women and women who were smokers were more likely to gain weight between pregnancies, while a diagnosis of gestational diabetes in the first pregnancy was protective against inter-pregnancy weight gain (see tables 2 and 3).

Among women with a normal BMI in their first pregnancy, a BMI increase of ≥4kg/m^2 was associated with increased risk of developing GDM (aRR 1.97; 95% CI 1.22-3.19), a macrosomic (aRR 4.06; 95% CI 2.25-7.34) or LGA infant (aRR 1.31 0.96-1.78) in the second pregnancy (Table 4), while a reduction in BMI (≤-2kg/m^2) was associated with an increased risk of SGA (aRR 1.94; 1.19-3.16). Linear trends were observed between increasing BMI change and the prevalence of GDM or macrosomic infant in the second pregnancy.

Among women who were overweight or obese in their first pregnancy, a BMI increase of ≥2 to 4 and ≥4 kg/m^2 was associated with increased risks of developing GDM in the second pregnancy (aRR 1.39; 95%CI 1.01-1.91 and aRR 1.64 95%CI 1.16-2.31; p_{trend} <0.001), while no associations were observed for a BMI increase and risk of a macrosomic, SGA, or LGA infant. In contrast, reduction in BMI (≤-2kg/m^2) was associated with a reduced risk of GDM (aRR 0.58 95% CI 0.37-0.90) and SGA (aRR 0.47; 95%CI 0.25-0.87). Linear trends were observed between increasing BMI change and the prevalence of GDM and SGA in the second pregnancy. Statistically significant interactions between maternal BMI category during the first pregnancy and inter-pregnancy BMI change was observed for the outcomes of SGA (p<0.001) and macrosomic infant (p<0.007).

Discussion

This study demonstrates that inter-pregnancy weight change affects women of a normal weight in their first pregnancy differently to women who are overweight/obese. Importantly, inter-pregnancy weight loss in women who are overweight/obese was associated with substantial reductions in the risk of developing gestational diabetes and small-for-gestational age babies, but not hypertensive disorders of pregnancy, macrosomia or large-for-gestational age babies. In contrast, inter-pregnancy weight gain in women of a normal weight was associated with an increased risk of gestational diabetes, large for gestational age and macrosomia in a subsequent pregnancy. Insulin resistance may be an underlying physiological pathway: as gestational diabetes and increased birthweight are all linked to increasing insulin resistance in pregnancy, independent of BMI, while an increased BMI is widely accepted to predispose to insulin resistance [25]. The only demonstrated effect of inter-pregnancy BMI change on preterm birth was an increased risk in women who were already overweight or obese and who gained between 2 and 4 units of BMI, but not in women who gained more than 4 kg/m^2. No protective effect was seen with weight loss in this group.

Our findings in relation to risk of gestational diabetes (in women of normal and increased BMI) support those of Villamor and Cnattingius, but in contrast to their study, we found no significant result in relation to hypertensive disorders of pregnancy [11].

Another large study, performed by Bogearts et al [13] in Belgium 2009-11 showed an increased risk for macrosomia (aOR 1.42 (1.08-1.87)) with an inter-pregnancy weight gain of >2 kg/m^2 in women who were overweight or obese in their first pregnancy. Our study included a larger proportion of overweight and obese women and our results were not significant for macrosomia, nor did we find a significantly increased risk of LGA in women with a BMI >25 kg/m^2.

Borgearts et al. also showed that weight loss >1 kg/m^2 in women with a BMI <25 increased the odds of low birthweight (aOR 2.22 (1.41-3.51)), while being protective for macrosomia (aOR 0.50 (0.35-0.71)). Our study demonstrated an increased risk of SGA with weight loss in this group, but did not confirm a protective effect for macrosomia.

The current study has a number of strengths compared to previous studies. Both Villamor and Bogearts analysed underweight and normal weight women together and used birthweight measures based on population data, not adjusted for maternal height, weight, and ethnicity.

Until now, significant weight loss among women who are overweight or obese has mostly been studied in relation to risk of developing gestational diabetes and birthweight. Where previous landmark studies have only assessed BMI loss $\geq$ 1 unit [11,13], we were able to assess higher degrees of BMI change. American studies have shown reducing risk of gestational diabetes with increasing degrees of weight loss, and a greater protective effect with a shift from obese to normal weight (aOR 0.21 (0.09-0.50) [14, 15]).

Our findings in relation to birthweight may be compared with Jain et al. [17] and Gethun et al. [18], who studied birthweight in relation to inter-pregnancy BMI change between first and second pregnancies in Missouri, USA. Getahun et al. investigated BMI category shift between 1989-97 and showed that in women who went from obese to normal-weight between pregnancies were less likely to have a large-for-gestational age baby in a subsequent pregnancy (aOR 1.5 (1.1-2.0)) than those who remained obese (aOR 2.3 (2.2-2.4)) when compared to women who maintained a normal BMI. An increased risk was seen in women who shifted from a normal to overweight BMI category (aOR 1.6 (1.5-1.7)), and that risk increased further with a shift to a higher category (aOR 2.0 (1.8-2.3) for women who went from normal weight to obese)[18]. Jain et al. presented BMI unit change in obese women exclusively between 1998 and 2005 and showed that while weight modulation affects the risk of large-for-gestational age, there was no significant effect on the risk of small-for-gestational age [17]. Our study shows a reduction in the incidence of SGA with weight loss in our overweight/obese cohort and showed an increased risk of macrosomia and large-for-gestational age with weight gain in women of normal BMI in their first pregnancy. Like the Villamor and Bogearts studies, Jain et al. and Getahun et al. adjusted for only fetal sex and gestational age when determining whether a baby was appropriate size for dates, whereas we adjusted for maternal height, weight, ethnicity, and fetal sex and gestational age. Adjusting for maternal BMI may have removed some of the effect of inter-pregnancy weight change for birthweight outcomes.

Excessive inter-pregnancy weight gain was demonstrated in young first-time mothers (also shown by Bogearts et al, [13] BMI change 1.10kg/m^2 compared to 0.34-0.81kg/m^2 ($p< 0.001$) in other age groups); smokers (also shown by Villamor and Cnattingius [11] BMI change 0.99kg/m^2 compared to 0.77kg/m^2 in non smokers $p <0.001$) and single women (Table 2). It is also interesting to note that a diagnosis of GDM in a first pregnancy is protective for inter-pregnancy weight gain, and may be at least partially secondary to the acknowledgement of a problem and subsequent healthy life style counseling offered to these women.

Chen et al. [26] and Wallace et al. [27] each investigated the effect of inter-pregnancy weight change on preterm birth. Neither study demonstrated a statistically significant effect of inter-pregnancy weight gain in the overweight/obese women on the incidence of preterm delivery. Wallace et al. investigated women in all BMI categories and found a non-statistically significant increase of 80% in spontaneous pre-term birth with weight loss of $\geq$ 1 kg/m^2 in women with a BMI of <25 kg/m^2 in the index pregnancy, we were not able to confirm this finding.

Ours is a large study in a contemporary cohort studied over a 12-year period, allowing the analysis to measure, examine and adjust for inter-pregnancy interval amongst many other covariates. Given the retrospective nature of our study design, we were limited to utilising data that are routinely collected as part of clinical care and pregnancy outcomes reporting requirements. As we investigated recurrent births occurring at a single tertiary hospital setting, there is a possibility that this group of women represents a higher risk group than that of the general population, which could impact the generalizability of our findings. Excluding women whose first antenatal visit was after 15 weeks gestation may have introduced selection bias. Since we did not have data on paternity or gestational weight gain we were unable to examine these potential effects or adjust for these confounders. As gestational weight gain is strongly associated with inter-pregnancy weight change, being unable to adjust for gestational weight gain makes it impossible to determine the impact of gestational weight gain in the first pregnancy separate to inter-pregnancy weight gain. One should exercise caution in interpreting the findings of our study in relation to preterm birth, as we have not separated iatrogenic preterm birth from spontaneous preterm birth.

The public-health findings of our study are worthy of consideration. For example, if a woman 1.6m tall, weighing 70kg in her first pregnancy (giving her an overweight BMI of 27) lost 6kg (2 BMI units), she would reduce her risk of developing GDM from 8.5% to 4.9%, an absolute reduction of almost 50%. However, if she gained either 7kg (or 3 BMI units) or 12kg (or 5 BMI units) between pregnancies, she would increase her absolute risk to 11.8% or 13.9% respectively.

While weight gain between pregnancies in Australian women is certainly the norm, inter-pregnancy weight control represents an important target in all women to reduce the risk of adverse perinatal outcomes in a subsequent pregnancy.

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Disclosure of Interests
All authors have completed the Unified Competing Interest form at http://www.icmje.org/coi_disclosure.pdf (available on request from the corresponding author) and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous 3 years; no other relationships or activities that could appear to have influenced the submitted work.

Contribution to Authorship
Nil contributors not listed as an author

Details of ethics approval

The project was approved by the Human Research Ethics Committees of the Women's and Children's Health Network and the University of Adelaide in South Australia (ID REC2219/10/14).

Contributors Statement

RDM and LEG conceptualised and designed the study, carried out the initial analyses, and drafted the initial manuscript. GAD, VLC, and BWM helped design the study, assisted in interpretation of results, and reviewed and revised the initial manuscript. All authors approved the final manuscript as submitted.

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Figure 1: Participant Flow Diagram

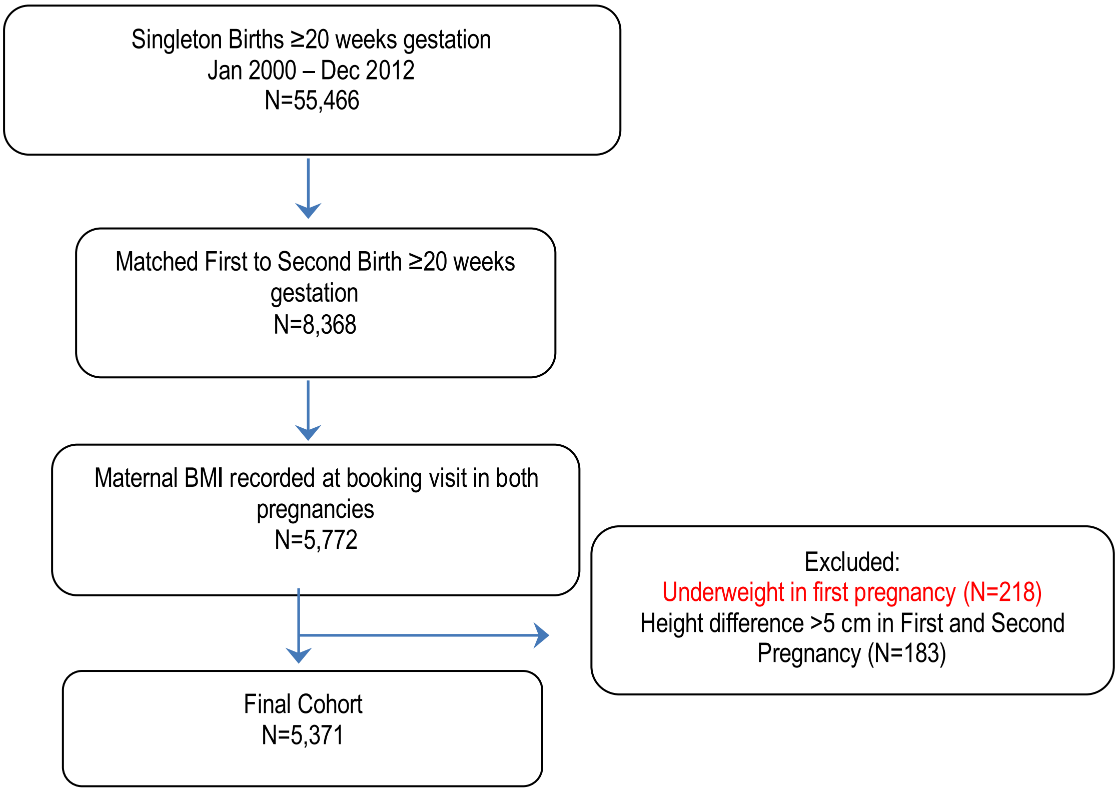

Table 1: Maternal BMI in First and Second Pregnancy

	First Pregnancy		
Second Pregnancy	**Normal Weight BMI≥18.5 & <25**	**Overweight BMI≥25 & <30**	**Obese BMI≥30**
Underweight	67 (2.1)	0	0
Normal Weight	2575 (79.3)	127 (9.8)	3 (0.4)
Overweight	558 (17.2)	807 (62.0)	73 (8.9)
Obese	47 (1.5)	368 (28.3)	746 (90.8)

Figure 2: Inter-pregnancy BMI Change According to BMI in First Pregnancy

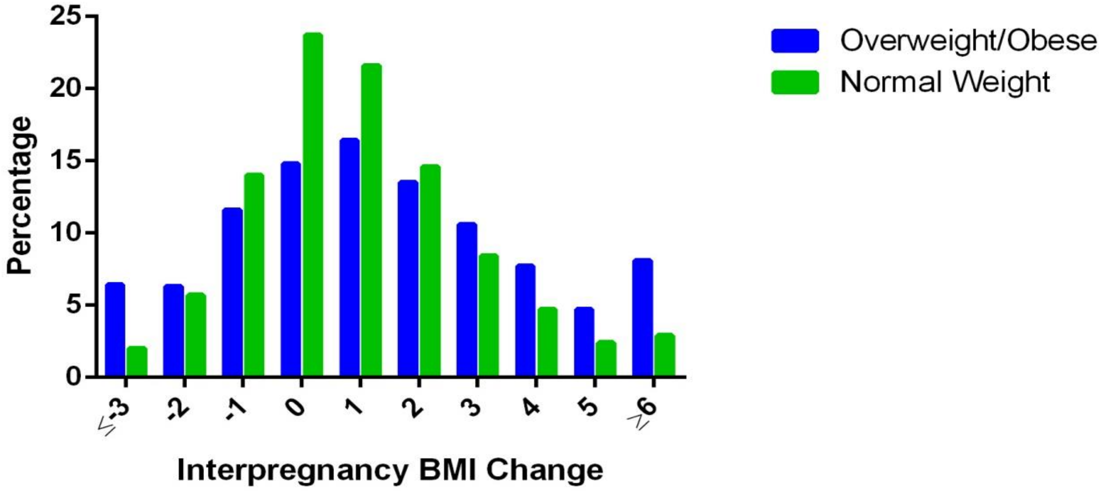

Table 2: Mean Change in Inter-pregnancy Body Mass Index According to Maternal Characteristics of the First Pregnancy

Characteristic	Normal Weight BMI≥18.5 & <25				Overweight/Obese BMI≥25			
	N[#]	Mean BMI Change (kg/m^2)	SD	P[†]	N[#]	Mean BMI Change (kg/m^2)	SD	P[†]
Total	3247	1.00			2124	1.42		
Maternal Age				<0.001				<0.001
≤19	266	1.71	3.05		146	3.19	3.68	
20-24	759	1.38	2.40		499	1.61	3.23	
25-29	1177	0.80	1.78		758	1.26	3.03	
30-34	822	0.74	1.70		546	1.00	2.57	
≥35	223	0.63	1.76		175	0.81	2.88	
Ethnicity				<0.001				0.085
Caucasian	2419	0.91	2.07		1852	1.35	3.09	
Aboriginal/TSI	57	2.04	3.62		46	2.14	3.32	
Asian	583	0.99	1.76		120	0.99	2.47	
Other	188	1.63	2.30		106	1.74	2.82	
Smoking Status				0.005				0.328
Non-Smoker	2689	0.90	1.91		1622	1.39	2.87	
Quit During Pregnancy	177	1.10	2.21		172	1.0	3.09	
Smoker	381	1.51	2.89		330	1.57	3.81	
Socioeconomic Status				0.010				0.081
5 (Highest)	714	0.81	1.91		354	1.07	2.91	
4	733	0.93	1.94		473	1.46	2.64	
3	748	0.92	1.94		523	1.35	2.89	
2	507	0.99	2.24		373	1.19	3.42	
1 (Lowest)	545	1.35	2.42		401	1.72	3.42	
Breastfeeding Infant at Discharge from Hospital				0.014				0.034
No	167	1.38	2.27		167	1.79	3.15	
Yes	3080	0.96	2.06		1957	1.32	3.04	
Married/Defacto				<0.001				<0.001
No	664	1.39	2.67		438	2.46	3.61	
Yes	2576	0.88	1.88		1679	1.09	2.82	

[#] Totals may not sum due to missing data

[†] Kruskal-Wallis tests

Table 3: Mean Change in Inter-pregnancy Body Mass Index According to Perinatal Outcomes of the First Pregnancy

Characteristic	Normal Weight BMI≥18.5 & <25				Overweight/Obese BMI≥25			
	N#	Mean BMI Change (kg/m²)	SD	P†	N#	Mean BMI Change (kg/m²)	SD	P†
Total	3247	1.00			2124	1.42		
Gestational Diabetes				0.476				<0.001
No	3142	0.99	2.09		1988	1.43	3.05	
Yes	105	0.77	1.66		1.36	0.44	2.97	
Hypertensive Disorders of Pregnancy				<0.001				0.166
No	3032	0.95	2.04		1824	1.35	3.04	
Yes	215	1.50	2.49		248	1.24	3.85	
Small-for-Gestational Age				0.980				0.418
No	2976	0.99	2.09		1876	1.39	2.93	
Yes	271	0.93	1.93		248	1.24	3.85	
Large-for-Gestational Age				0.051				0.905
No	2908	0.96	2.07		1875	1.37	3.08	
Yes	339	1.16	2.13		249	1.38	2.82	
Macrosomic Infant >4,500 kg				0.698				0.949
No	3215	0.98	2.07		2087	1.37	3.06	
Yes	32	1.33	3.03		37	1.29	2.61	
Birth Method				0.222				0.620
NVB	1831	0.96	2.05		977	1.44	2.99	
Assisted	736	0.93	2.02		461	1.39	3.01	
Em LSCS	589	1.15	2.26		592	1.35	2.99	
El LSCS	80	0.80	1.73		84	0.44	4.18	
Pregnancy Interval Time				<0.001				<0.001
<1	806	0.86	2.00		548	1.15	2.43	
≥1 to <2	1316	0.70	1.85		770	1.09	2.94	
≥2 to <3	575	1.07	1.97		471	1.53	3.19	
≥3	550	1.73	2.56		335	2.14	3.80	

Totals may not sum due to missing data

† Kruskal-Wallis tests

Table 4: Relative Risks for Adverse Perinatal Outcomes During the Second Pregnancy in Relation to Inter-pregnancy Change in Body Mass Index Between the First and Second Pregnancy Stratified by Pre-pregnancy Body Mass Index at First Pregnancy

Outcome (cases)		Normal Weight (BMI≥18.5 & <25 kg/m²)			Overweight/Obese (BMI≥25 kg/m²)		
	BMI Change (kg/m²)	**n‡**	**%**	**Adjusted RR† (95% CI)**	**n‡**	**%**	**Adjusted RR† (95%CI)**
Gestational Diabetes (n=342)	**≤-2**	5	3.9	1.11 (0.50-2.50)	14	7.1	**0.58 (0.37-0.90)**
	-2 to 2	93	4.1	1.00	98	8.6	1.00
	≥2 to 4	27	4.7	1.36 (0.92-2.00)	45	10.3	**1.39 (1.01-1.91)**
	≥4	17	7.0	**1.97 (1.22-3.19)**	43	12.5	**1.64 (1.16-2.31)**
	p, trend		0.051	**0.008**		**0.011**	**<0.001**
Hypertensive Disorders of Pregnancy (n=222)	**≤-2**	3	2.3	0.77 (0.28-2.14)	8	4.0	0.62 (0.30-1.26)
	-2 to 2	57	2.5	1.00	66	5.8	1.00
	≥2 to 4	15	2.6	0.98 (0.57-1.69)	36	8.2	1.22 (0.79-1.88)
	≥4	11	4.5	1.09 (0.55-2.16)	26	4.6	1.16 (0.70-1.93)
	p, trend		0.234	0.887		0.072	0.372
Macrosomic Infant (>4,500 kg) (n=128)	**≤-2**	2	1.6	0.99 (0.23-4.33)	8	2.5	0.87 (0.34-2.23)
	-2 to 2	35	1.5	1.00	34	3.0	1.00
	≥2 to 4	13	2.2	1.55 (0.83-2.87)	18	4.1	1.59 (0.88-2.88)
	≥4	13	5.4	**4.06 (2.25-7.34)**	8	2.3	0.87 (0.40-1.92)
	p, trend		**<0.001**	**<0.001**		0.931	0.628
Small-for-Gestational Age (n=433)	**≤-2**	16	12.4	**1.94 (1.19-3.16)**	11	5.6	**0.47 (0.25-0.87)**
	-2 to 2	153	6.7	1.00	104	9.1	1.00
	≥2 to 4	36	6.2	0.88 (0.62-1.24)	49	11.2	1.21 (0.87-1.67)
	≥4	24	9.9	1.44 (0.96-2.16)	40	11.6	1.04 (0.73-1.47)
	p, trend		0.848	0.825		**0.015**	**0.023**
Large-for-Gestational Age (n=664)	**≤-2**	16	12.4	1.16 (0.73-1.82)	29	14.7	1.18 (0.80-1.73)
	-2 to 2	263	11.5	1.00	148	12.9	1.00
	≥2 to 4	69	11.9	1.07 (0.84-1.36)	65	14.8	1.20 (0.92-1.57)
	≥4	35	14.4	**1.31 (0.96-1.78)**	39	11.3	0.97 (0.69-1.35)
	p, trend		0.308	0.233		0.518	0.873
Preterm Birth (n=259)	**≤-2**	7	5.4	1.23 (0.60-2.52)	14	7.1	1.40 (0.76-2.57)
	-2 to 2	102	4.5	1.00	54	4.7	1.00
	≥2 to 4	27	4.7	1.00 (0.66-1.52)	34	7.8	**1.59 (1.05-2.41)**
	≥4	7	2.9	0.53 (0.24-1.17)	14	4.1	0.90 (0.53-1.54)
	p, trend		0.343	0.146		0.768	0.982

‡ Number of cases with outcome of interest †Adjusted for maternal age, socioeconomic status, maternal BMI in first pregnancy, smoking status, race, inter-pregnancy interval time, and first pregnancy outcomes (including gestational diabetes, pregnancy induced hypertensive disorders, birth method, LGA/SGA

Supplemental Table 1: Maternal Characteristics According to Body Mass Index Category in First Pregnancy

Characteristic	Normal Weight BMI≥18.5 & <25	Overweight/Obese BMI≥25
Total	N=3,247	N=2,124
Maternal Age, years, N (%)		
≤19	266 (8.2)	146 (6.9)
20-24	759 (23.4)	499 (23.5)
25-29	1177 (36.3)	758 (35.7)
30-34	822 (25.3)	546 (25.7)
≥35	223 (6.9)	175 (8.2)
Ethnicity, N (%)		
Caucasian	2419 (74.5)	1852 (87.2)
Aboriginal/TSI	57 (1.8)	46 (2.2)
Asian	583 (18.0)	120 (5.7)
Other	188 (5.8)	106 (5.0)
Smoking Status, N (%)		
Non-Smoker	2689 (82.8)	1622 (76.4)
Quit During Pregnancy	177 (5.5)	172 (8.1)
Smoker	381 (11.7)	330 (15.5)
Socioeconomic Status, N (%)		
5 (Highest)	714 (22.0)	354 (16.7)
4	733 (22.6)	473 (22.3)
3	748 (23.0)	523 (24.6)
2	507 (15.6)	373 (17.6)
1 (Lowest)	545 (16.8)	401 (18.9)
Pre-Existing Diabetes, N (%)	7 (0.2)	24 (1.1)
Pre-Existing Hypertension, N (%)	16 (0.9)	34 (1.6)
Breastfeeding Infant at Discharge from Hospital, N (%)	3080 (94.9)	1957 (92.1)
Married/Defacto, N (%)	2576 (79.5)	1679 (79.3)
Gestational Diabetes, N (%)	105 (3.2)	136 (6.4)
Hypertensive Disorders of Pregnancy, N (%)	215 (6.6)	300 (14.1)
Small-for-Gestational Age, N (%)	271 (8.4)	248 (11.7)
Large-for-Gestational Age, N (%)	339 (10.4)	249 (11.7)
Macrosomic Infant >4,500 kg, N (%)	32 (1.0)	37 (1.7)
Birth Method, N (%)		
NVB	1831 (56.6)	977 (45.8)
Assisted Vaginal Delivery	736 (22.7)	461 (21.7)
LSCS	669 (20.6)	676 (31.8)
Interpregnancy BMI Change, mean (SD)	0.98 (2.08)	1.37 (3.05)
Pregnancy Interval Time, Years, N (%)		
<1	806 (24.8)	548 (25.8)
≥1 to <2	1316 (40.5)	770 (36.3)
≥2 to <3	575 (17.7)	471 (22.2)
≥3	550 (16.9)	335 (15.8)

Abbreviations: BMI, body mass index; NVB, normal vaginal birth; LSCS, lower segment caesarean section