Impact of Restricted Maternal Weight Gain on Fetal Growth and Perinatal Morbidity in Obese Women With Type 2 Diabetes

Björg Ásbjörnsdóttir[1,2]
Signe S. Rasmussen, phd[1,2]
Louise Kelstrup, md[1,3]
Peter Damm, dmsc[1,3,4]
Elisabeth R. Mathiesen, dmsc[1,2,4]

OBJECTIVE—Since January 2008, obese women with type 2 diabetes were advised to gain 0–5 kg during pregnancy. The aim with this study was to evaluate fetal growth and perinatal morbidity in relation to gestational weight gain in these women.

RESEARCH DESIGN AND METHODS—A retrospective cohort comprised the records of 58 singleton pregnancies in obese women (BMI ≥30 kg/m^2) with type 2 diabetes giving birth between 2008 and 2011. Birth weight was evaluated by SD *z* score to adjust for gestational age and sex.

RESULTS—Seventeen women (29%) gained ≤5 kg, and the remaining 41 gained >5 kg. The median (range) gestational weight gains were 3.7 kg (−4.7 to 5 kg) and 12.1 kg (5.5–25.5 kg), respectively. Prepregnancy BMI was 33.5 kg/m^2 (30–53 kg/m^2) vs. 36.8 kg/m^2 (30–48 kg/m^2), $P = 0.037$, and median HbA_{1c} was 6.7% at first visit in both groups and decreased to 5.7 and 6.0%, $P = 0.620$, in late pregnancy, respectively. Gestational weight gain ≤5 kg was associated with lower birth weight *z* score ($P = 0.008$), lower rates of large-for-gestational-age (LGA) infants (12 vs. 39%, $P = 0.041$), delivery closer to term (268 vs. 262 days, $P = 0.039$), and less perinatal morbidity (35 vs. 71%, $P = 0.024$) compared with pregnancies with maternal weight gain >5 kg.

CONCLUSIONS—In this pilot study in obese women with type 2 diabetes, maternal gestational weight gain ≤5 kg was associated with a more proportionate birth weight and less perinatal morbidity.

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Pregestational diabetes is associated with various pregnancy complications. The prevalence of large-for-gestational-age (LGA) infants born to women with type 2 diabetes has been reported as ~50% (1,2). Elevated maternal glucose crosses the placental barrier, leading to fetal hyperinsulinemia, which stimulates growth both directly and indirectly (3,4). Maternal hyperglycemia and hypertriglyceridemia are characteristic features of pregnancies complicated by diabetes and are established risk factors for fetal LGA or macrosomia (4,5). Moreover, women with pregestational diabetes are at increased risk of pregnancy-induced hypertension, pre-eclampsia, preterm delivery, and caesarean section, and pregestational diabetes can also lead to neonatal morbidity (6,7). The treatment of pregnant women with type 2 diabetes tends to focus on achieving euglycemia, before, during, and after pregnancy (8).

In pregnant women without diabetes, obesity is associated with complications such as perinatal mortality and LGA/macrosomia, the risk rising with rising prepregnancy BMI (9,10). High maternal weight gain during pregnancy in women without diabetes increases the risk of excessive fetal growth (11), whereas low maternal gestational weight gain is associated with birth of small-for-gestational-age (SGA) infants and preterm birth (9). Obesity management during pregnancy includes recommendations of appropriate weight gain. The U.S. Institute of Medicine (IOM) recommends a total weight gain of 5–9 kg for healthy obese women (12), and Cedergren (13) suggests that obese women should gain <6 kg to reduce the risk of adverse pregnancy outcomes.

Obesity is a common characteristic of pregnant women with type 2 diabetes. To our knowledge, there are no specific recommendations regarding weight gain during pregnancy for this group, and there is little literature on the subject. One study concluded that gestational weight gain in women with type 2 diabetes greater than that recommended by IOM guidelines was associated with higher odds both of infants being LGA and macrosomic and caesarean delivery (14). Based on results from a study in obese women without diabetes (15), since 2008, the procedure in our clinic for pregnant women with diabetes has been to advise obese (BMI ≥30 kg/m^2) women with type 2 diabetes to gain 0–5 kg in total during pregnancy in an attempt to minimize the frequency of LGA infants.

Here, we aimed to evaluate fetal growth and perinatal morbidity in relation to gestational weight gain in obese women with type 2 diabetes. Additionally, we investigated whether it seems safe to advise this group of women to gain ≤5 kg regarding maternal health, including diabetes management, and pregnancy outcomes.

RESEARCH DESIGN AND METHODS

Study design and population

Since January 2008, all obese pregnant women with type 2 diabetes attending our

From the [1]Center for Pregnant Women with Diabetes, Rigshospitalet, Copenhagen, Denmark; the [2]Department of Endocrinology, Rigshospitalet, Copenhagen, Denmark; the [3]Department of Obstetrics, Rigshospitalet, Copenhagen, Denmark; and the [4]Faculty of Health Sciences, University of Copenhagen, Copenhagen, Denmark.
Corresponding author: Björg Ásbjörnsdóttir, bjorga@gmail.com.
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clinic have been advised to gain 0–5 kg body weight during pregnancy. All 117 pregnant women with type 2 diabetes giving birth at our center during January 2008 to October 2011 were included in this retrospective cohort study. The inclusion criteria were as follows: type 2 diabetes, defined as diabetes diagnosed before pregnancy where the initial management was diet or oral antidiabetic drugs; referral before 22 gestational weeks with a singleton pregnancy; prepregnancy BMI ≥30 kg/m^2; and no concurrent diseases that could affect weight gain. Women of all ethnicities were included.

We excluded 59 women due to BMI <30 kg/m^2 (n = 47), former bariatric surgery (n = 2), incomplete data in the medical records on weight gain during pregnancy (n = 8), and other prepregnancy medical problems (n = 2), leaving 58 participants. If a woman had had more than one pregnancy during the study period, only the most recent pregnancy was included.

Demographic and clinical data on the mother and newborn were obtained from the original medical records.

The main outcome variable was birth weight z score. Secondary outcomes were LGA infants and perinatal morbidity.

Treatments during pregnancy

At the woman's first pregnancy visit to dedicated diabetes nurses and dietitians, she received guidance and motivation to follow a healthy lifestyle. All women had a 1-h tailored dietary consultation concerning the diabetes diet, which was supplemented with educational handouts to aid learning. Further, all women received both oral and written information advising them to gain between 0 and 5 kg body weight overall during pregnancy, with the main weight gain in the second half of pregnancy. Moderate physical activity for about 30 min/day was advised. The women were weighed at each clinical visit, and their weight was used to reinforce the aim of restricted weight gain. Self-monitoring of plasma glucose was recommended six to seven times daily. The targets for plasma glucose were as follows: fasting, 4–6 mmol/L, and 90-min postprandial, 4–8 mmol/L.

The pregnant women consulted the clinic at least six times during pregnancy, usually at 10, 14, 22, 27, 33, and 37 gestational weeks. At each visit, blood pressure and HbA_{1c} were measured, and a dipstick of sterile urine was screened for ketone bodies and protein. If a woman presented with ketonuria, her plasma glucose and carbohydrate intake were evaluated. If necessary, women were advised to have an additional carbohydrate intake during the evening. In general, the women were seen by a diabetes caregiver every 2 weeks, either at our clinic or in cooperation with their local diabetes center. In those treated with oral antidiabetic drugs (usually metformin) at first visit, this treatment was discontinued. Insulin treatment was commenced if the women did not obtain adequate glycemic control from diet alone. A basal-bolus regimen or two daily injections of insulin NovoMix 30 were used.

Definitions and measurements

Gestational weight gain was calculated as the difference between self-reported weight before pregnancy and the last weight measured before delivery. Proteinuria was defined as occurrence of one of the following: proteinuria ≥300 mg/24 h, urine albumin excretion ≥190 mg/24 h, proteinuria greater than or equal to +1 on a dipstick of sterile urine, or urine albumin-creatinine mass ratio ≥190 mg/g. Pre-eclampsia was defined as blood pressure ≥140/90 mmHg accompanied by proteinuria after 20 gestational weeks. If a woman had hypertension in early pregnancy, a sudden increase of ≥15% in systolic or diastolic blood pressure and urine albumin excretion ≥190 mg/24 h were required. Diabetic nephropathy was noted if urine albumin-creatinine mass ratio was ≥300 mg/g. An ophthalmologist evaluated the presence or absence of diabetic retinopathy at first visit and around 27 weeks of gestation. Ethnic origin was defined as Nordic Caucasian when the pregnant woman originated from Northern Europe.

Preterm delivery was defined as birth before 37 completed gestational weeks. Caesarean section was classified as emergency when carried out within 8 h of clinical decision.

Birth weight z score is a measure of how far in SDs the birth weight deviates from the mean of a standard Nordic population, adjusted for gestational age and sex (16). LGA and SGA were defined as a birth weight >90th or <10th percentile, respectively, adjusted for gestational age and sex (16). Ponderal index was calculated as weight divided by the third power of body length. Macrosomia was defined as birth weight ≥4,000 g.

Perinatal mortality was fetal death later than 22 weeks of gestation or death within 1 week of delivery. Major congenital malformations were those leading to death, causing a substantial future handicap or requiring surgery. Jaundice was registered when phototherapy was required, and transient tachypnea of the newborn (TTN) was defined as a need for continuous positive airway pressure for >60 min. Neonatal hypoglycemia was defined as plasma glucose <2.5 mmol/L, measured 2 h after birth. We designed a combined end point, perinatal morbidity, defined as the occurrence of at least one of the following complications: perinatal mortality, major congenital malformation, jaundice, TTN, neonatal hypoglycemia, or admission to neonatal special care unit (NSCU).

Statistical analysis

Continuous variables were reported as median (range) and categorical data with number (%). Differences between the two exposure groups were analyzed with χ^2 test, Fischer exact test, or Mann-Whitney U test when appropriate.

To control for effects of confounding, multiple linear regression analysis was applied using maternal weight gain in pregnancy ≤5 or >5 kg as exposure variable and birth weight z score as outcome variable. Based on theoretical considerations, we included six possible confounders: prepregnancy BMI (kg/m^2), smoking (yes vs. no), HbA_{1c} (%) at last visit, Nordic Caucasian (yes vs. no), preeclampsia (yes vs. no), and nulliparity (yes. vs. no). All tests were two tailed, and a P value of <0.05 was considered statistically significant. Data were analyzed in SPSS Statistics version 19 (SPSS Inc., Chicago, IL).

Ethics

The Danish Data Protection Agency approved the protocol. According to Danish law, the protocol did not need approval from the regional ethics committees as the study was a register study without biological material and medical intervention.

RESULTS—Table 1 shows the basic characteristics of the study population. The median weight gain in the total cohort was 9.2 kg (range −4.7 to 25.5 kg); 17 women (29%) gained ≤5 kg with a median weight gain of 3.7 kg, and 41 (71%) gained >5 kg with a median weight gain of 12.1 kg (Table 2). Women gaining ≤5 kg had a lower median prepregnancy BMI and higher median diastolic blood pressure at first visit compared with

Table 1—*Maternal characteristics at first visit*

	Weight gain ≤5 kg	Weight gain >5 kg	*P* value[a]
n (%)	17 (29)	41 (71)	
Maternal age (years)	35 (23–44)	34 (20–45)	0.199
Nordic Caucasians	11 (65)	30 (75)	0.433
Nullipara	6 (35)	14 (34)	0.934
Duration of type 2 diabetes (years)	3 (0.25–10)	3 (0.5–10)	0.648
Smokers	3 (18)	5 (12)	0.587
Prepregnancy weight (kg)	95 (72–156)	106 (76–140)	0.055
Prepregnancy BMI (kg/m^2)	33.5 (30–52.7)	36.8 (30–48.2)	0.037
Diabetic retinopathy	4 (24)	8 (20)	0.733
Diabetic nephropathy	0	1 (2)	0.527
Gestational age at booking (days)	86 (53–144)	69 (36–144)	0.074
Systolic blood pressure (mmHg)	129 (112–165)	124 (88–178)	0.235
Diastolic blood pressure (mmHg)	83 (70–95)	75 (55–98)	0.036

Data shown as *n* (%) or median (range). [a]Mann-Whitney *U* test, χ^2, or Fisher exact test as appropriate.

the remaining women (Table 1). Median HbA_{1c} was 6.7% at first visit in both groups but declined to 5.7 and 6.0%, respectively, and the prevalence of ketonuria was low in both groups (Table 2). In both groups, the main weight gain occurred in the second half of pregnancy (Table 2), but five women lost weight during pregnancy, with a median weight loss of 1.9 kg (−4.7 to −0.8 kg). Nearly all women were insulin treated from early pregnancy, but significantly less insulin was needed in the group of women gaining ≤5 kg (Table 2).

A gestational weight gain of ≤5 kg was significantly associated with a more proportionate birth weight with lower median birth weight *z* score (−0.44 [range −3.31 to 1.98] vs. 0.84 [−2.32 to 4.02], $P = 0.008$) as well as a lower rate of LGA infants ($P = 0.041$) compared with those gaining >5 kg during pregnancy (Table 3). The prevalence of SGA infants was comparable. Women gaining ≤5 kg delivered closer to term ($P = 0.039$) and had infants with less perinatal morbidity compared with the remaining women ($P = 0.024$) (Table 3).

In the multiple linear regression analysis, a gestational weight gain of ≤5 kg was significantly associated with a lower birth weight *z* score when adjusted for prepregnancy BMI, smoking, HbA_{1c} at last visit, ethnicity, preeclampsia, and nulliparity, ($\beta = -0.978$ [95% CI −1.831 to −0.126], $P = 0.025$), corresponding to a one SD lower birth weight.

Two infants had a major congenital malformation: one with a curved femur, requiring surgery (>5-kg group), and one with cardiac and renal malformations leading to stillbirth (≤5-kg group). In addition, one LGA infant (>5-kg group) died shortly after birth due to severe shoulder dystocia (Table 3).

Table 2—*Maternal HbA_{1c}, urine ketone bodies, weight gain, and insulin treatment during pregnancy*

	Weight gain ≤5 kg	Weight gain >5 kg	*P* value[a]
n (%)	17 (29)	41 (71)	
HbA_{1c}			
At first visit (%)	6.7 (5.1–8.1)	6.7 (5.3–13.2)	0.365
At 22 weeks (%)	5.7 (5.1–6.6)	5.6 (4.6–7.7)	0.537
At last visit (%)	5.7 (5.4–6.6)	6 (4.8–8.2)	0.620
Ketone bodies detected			
At first visit	0	2 (5)	1.000
At 22 weeks	0	0	
At last visit	0	0	
Weight changes			
Total weight gain (kg)[b]	3.7 (−4.7 to 5)	12.1 (5.5–25.5)	<0.001
Total weight gain/week (g)[b]	97 (−123 to 132)	320 (150–687)	<0.001
Weight gain/week in first half of pregnancy (g)	20 (−317 to 210)	175 (−147 to 605)	0.001
Weight gain/week in second half of pregnancy (g)	147 (−57 to 587)	506 (96–1,316)	<0.001
Insulin			
Treatment before first visit	5 (29)	11 (27)	0.841
Treatment after first visit	12 (71)	35 (85)	0.191
Treatment at last visit	17 (100)	38 (93)	0.256
Dose at last visit (IU/kg)	0.72 (0.12–1.80)	1.29 (0.50–2.75)	0.003

Data shown as *n* (%) or median (range). [a]Mann-Whitney *U* test, χ^2, or Fisher exact test as appropriate. [b]From self-reported prepregnancy weight to last weight measured.

CONCLUSIONS—In this retrospective cohort study evaluating how the medical advice regarding restricting maternal weight gain to 0–5 kg during pregnancy in obese women with type 2 diabetes affects fetal growth and perinatal morbidity in an unselected cohort of patients, we found that those who gained 5 kg or less had infants with a more proportionate birth weight, represented by a smaller *z* score, a lower proportion of LGA infants, and a marginally lower ponderal index. Further, we found that perinatal morbidity was lower and the prevalence of SGA unchanged in offspring of women who gained 5 kg or less during pregnancy.

We believe this is a novel study. Its strengths stem from the exclusion of women with concurrent diseases and all relevant pregnancy data having been recorded and therefore being easy to collect retrospectively. The cases of malformation and perinatal mortality were reviewed. Potentially positive effects of weight restriction as well as potentially negative effects were considered. At their first pregnancy visit, all women received education about the increased risk of complications in pregnancies with diabetes and were motivated toward a healthy lifestyle. At their subsequent routine visits, they received tailored advice on diet adjustments. This approach was pragmatic and feasible for an everyday clinical setting. We are not aware of publications on more intensive intervention strategies in obese pregnant women with type 2 diabetes or of randomized controlled studies.

Table 3—*Pregnancy-related outcomes*

	Weight gain ≤5 kg	Weight gain >5 kg	*P* value[a]
n (%)	17 (29)	41 (71)	
Pre-eclampsia	0	4 (10)	0.310
Gestational age at delivery (days)	268 (221–284)	262 (206–280)	0.039
Preterm delivery	2 (12)	11 (27)	0.307
Emergency caesarean section	1 (6)	11 (27)	0.088
Elective caesarean section	6 (35)	14 (34)	1.000
Female offspring	9 (53)	17 (41)	0.424
Birth weight (g)	3,134 (1,278–3,870)	3,364 (1,070–4,432)	0.120
Birth weight *z* score (SD)	−0.44 (−3.31 to 1.98)	0.84 (−2.32 to 4.02)	0.008
LGA (>90th percentile)	2 (12)	16 (39)	0.041
SGA (<10th percentile)	3 (18)	4 (10)	0.407
Ponderal index (kg/m^3)	23.9 (20.4–29.2)	25.8 (20.1–30.1)	0.099
Macrosomia (birth weight >4,000 g)	0	8 (20)	0.090
Perinatal morbidity[b]	6 (35)	29 (71)	0.024
Perinatal mortality	1 (6)	1 (2)	0.504
Major congenital malformations	1 (6)	1 (2)	0.504
Jaundice[c]	0	8 (20)	0.089
TTN[c]	2 (13)	6 (15)	1.000
Neonatal hypoglycemia[c]	3 (19)	20 (51)	0.036
Admission to NSCU[c]	3 (19)	14 (34)	0.339

Data shown as *n* (%) or median (range). [a]Mann-Whitney *U* test, χ^2, or Fisher exact test as appropriate. [b]Morbidity was defined as the occurrence of at least one of the following complications: perinatal mortality, malformation, jaundice, TTN, neonatal hypoglycemia, or admission to NSCU. [c]Only living infants included (*n* = 16 and *n* = 40).

Weight loss, fasting, or poorly controlled diabetes can cause ketonemia and/or ketonuria (12,17). The presence of ketonemia in pregnant women has been associated with lower cerebral function in the offspring (12). Therefore, it is encouraging that the prevalence of women with morning ketonuria was very low and comparable in the two groups. This is in accordance with the finding that a 33% reduction in caloric intake was not associated with an increase in ketonuria in women with gestational diabetes mellitus (18). It would have added to our study if the women had tested for ketonuria at home or if we had checked for ketonemia at clinical visits; nevertheless, few of the routine clinical urine tests were positive.

Our findings are in accordance with a large register study (14) including 2,310 overweight and obese women with type 2 diabetes, which found that overweight and obese women with type 2 diabetes who gained >9 kg in total during pregnancy were at increased risk of having macrosomic or LGA infants. In the same study (14), 4.2% of the women who gained <5 kg during pregnancy had intrauterine fetal demises, but the specific causes were not given, and women with concurrent diseases were not excluded. In our study, only one stillbirth occurred, arising from multiple fetal anomalies. Although this is reassuring, due to the relatively low number of women included, our findings on rare outcomes have limited statistical power.

Since weight gain in early pregnancy is related to fat deposition (19), it is relevant to note that the weight gain in our patients was seen mainly in the second half of pregnancy. From our study, we cannot conclude which level of weight gain should be recommended for obese women with type 2 diabetes, but our data point toward a negligible gain in the first half of pregnancy and a gain of about 150 g per week in the second.

The prevalence of LGA infants is comparable to that in other studies (20). Lower gestational weight gain in women with the highest pregestational BMI, as previously found in obese women without diabetes (15), was not present in our material, where women who gained ≤5 kg were less obese before their current pregnancy than those who gained more. However, it is important to note that the significant association between maternal weight gain and fetal *z* score was independent of prepregnancy BMI in our study.

At our clinic, we discontinue all oral antidiabetic drugs at first visit because of their potentially harmful fetal effects. Women who gained 5 kg or less obtained excellent glycemic control by receiving almost half the insulin doses per kilogram needed in the remaining women. It is well known that insulin sensitivity increases during weight loss and that additional weight gain of adipose tissue often induces more severe insulin resistance. However, the women in our study who gained >5 kg during pregnancy were already more obese at pregnancy onset than the group gaining ≤5 kg, which might explain part of the difference in exogenous insulin requirement. Moreover, the group gaining ≤5 kg might have been more compliant, with a higher level of exercise, a more restricted diet, and more frequent self-monitoring of blood glucose, thereby better adapting their insulin doses. One study showed that women with gestational diabetes mellitus and low gestational weight gain were more likely to remain on diet control; however, they were also more likely to have a higher prevalence of SGA neonates (21). We did not find an increased rate of SGA infants among women gaining ≤5 kg, and all these women received insulin treatment.

We used self-reported weight before pregnancy to calculate the total gestational weight gain, and therefore there is a risk of recall bias. We did not use body weight measured at the first clinic visit because the women attended the clinic at different times in their pregnancy, i.e., between 6 and 22 gestational weeks. Another possible bias arises from the women who delivered preterm being included in the study (*n* = 13); obviously, these women had a shorter pregnancy in which to gain weight than those who delivered at term. However, because there were only two women who delivered preterm in the group who gained ≤5 kg, we do not consider the bias crucial for the outcome of this study.

This study is limited by its retrospective nature and small sample size, giving it a character of a pilot study. We included all eligible patients in our clinic and did not perform sample size calculations before commencing the study. Moreover, the descriptive nature of our study cannot give firm conclusions, but it can provide a basis for hypotheses and new studies. As far as we are aware, no dietary intervention studies targeting weight-gain restriction have been performed in obese pregnant women with type 2 diabetes; accordingly, randomized clinical trials are needed to evaluate the effect of weight-gain restriction in this population.

In conclusion, we found that in obese women with type 2 diabetes, maternal gestational weight gain ≤5 kg was associated with a more proportionate birth weight and less perinatal morbidity without increased risk of SGA infants. Our data suggest that it is seemingly safe to advise obese women with type 2 diabetes to gain ≤5 kg during pregnancy.

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B.Á. researched data, wrote the manuscript, and contributed to discussions. S.S.R. and P.D. contributed to discussions and reviewed and edited the manuscript. L.K. researched data, contributed to discussions, and reviewed and edited the manuscript. E.R.M. contributed to the idea, researched data, contributed to discussions, and reviewed and edited the manuscript. E.R.M. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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