Atlantic DIP: Is weight gain less than that recommended by IOM safe in obese women with Gestational Diabetes Mellitus? – a retrospective study

Delia Bogdanet[1], Mohamad Mustafa[2], Aftab Khattak[2], Paula O'Shea[3], and Fidelma Dunne[1]

[1]National University of Ireland Galway College of Medicine Nursing and Health Sciences
[2]University Hospital Galway
[3]Galway University Hospitals

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Abstract

Objective The study objective was to examine maternal and infant outcomes for obese GDM women who lost weight or gained 0-5 kg during pregnancy. Design A 7-year retrospective study of pregnancy outcomes for obese GDM women. Setting The ATLANTIC DIP consists of 5 antenatal centres along the Irish Atlantic seaboard. Population A total of 754 women met the inclusion criteria. Methods Women were stratified into 3 distinct groups according to their weight gain status: lost weight or gained less than 5 kg (Group 1, n=237 (31.4%)), gained 5-9kg (Group 2, n=77 (10.2%)) or gained >9kg (Group 3 n=440 (58.4%)). The groups were further subdivided according to treatment modality: diet alone (GDM-D) or diet and insulin (GDM-I). Main outcome measures Maternal (eg.preeclampsia, pregnancy induced hypertension (PIH)) and infant outcomes (eg.mortality, prematurity, macrosomia, large for gestational age (LGA)) were assessed. Results Women in Group 1 were older with a higher booking BMI compared to Groups 2 and 3. Logistic regression analysis adjusted for baseline BMI, insulin use, smoking status, parity, family history, ethnicity and age determined no significant difference in maternal or infant outcomes for women in Group1 compared to those in Group 2. Women with excessive weight gain had higher rates of PIH, macrosomia and LGA. Conclusion In our population, weight gain less than IOM guideline does not appear to be associated with adverse outcomes. However, further validation through a prospective study with a larger obese GDM cohort is required before these findings could be recommended for routine clinical use.

Design

A 7-year retrospective study of pregnancy outcomes for obese GDM women.

Setting

The ATLANTIC DIP consists of 5 antenatal centres along the Irish Atlantic seaboard.

Population

A total of 754 women met the inclusion criteria.

Methods

Women were stratified into 3 distinct groups according to their weight gain status: lost weight or gained less than 5 kg (Group 1, n=237 (31.4%)), gained 5-9kg (Group 2, n=77 (10.2%)) or gained >9kg (Group 3 n=440 (58.4%)). The groups were further subdivided according to treatment modality: diet alone (GDM-D) or diet and insulin (GDM-I).

Main outcome measures

Maternal (eg.preeclampsia, pregnancy induced hypertension (PIH)) and infant outcomes (eg.mortality, prematurity, macrosomia, large for gestational age (LGA)) were assessed.

Results

Women in Group 1 were older with a higher booking BMI compared to Groups 2 and 3. Logistic regression analysis adjusted for baseline BMI, insulin use, smoking status, parity, family history, ethnicity and age determined no significant difference in maternal or infant outcomes for women in Group1 compared to those in Group 2. Women with excessive weight gain had higher rates of PIH, macrosomia and LGA.

Conclusion

In our population, weight gain less than IOM guideline does not appear to be associated with adverse outcomes. However, further validation through a prospective study with a larger obese GDM cohort is required before these findings could be recommended for routine clinical use.

Tweetable abstract

Weight gain less than the IOM guidelines in obese pregnant women with gestational diabetes does not appear to be associated with adverse outcomes but further validation is required.

Keywords: gestational diabetes, weight gain, weight loss, Institute of Medicine, pregnancy outcomes

Abbreviations

Ante-partum haemorrhage APH

ATLANTIC Diabetes in Pregnancy Group ATLANTIC DIP

Caesarean Delivery CS

Diastolic blood pressure DBP

Elective caesarean delivery ELCS

Emergency caesarean section EMCS

Gestational diabetes mellitus GDM

Gestational weight gain GWG

Institute of Medicine IOM

International Association of the Diabetes in Pregnancy Study Group IADPSG

Large for gestational age LGA

Neonatal intensive care unit NICU

Oral glucose tolerance test OGTT

Post-partum haemorrhage PPH

Pre-eclampsia PET

Pregnancy induced hypertension PIH

Small for gestational age SGA

Systolic blood pressure SBP

World Health Organisation WHO

Introduction

The World Health Organization defines obesity as a body mass index (BMI) of 30 kg/m^2 or more (1). Worldwide, over one third of women of reproductive age are now obese (2). In the United States, the reported prevalence of obesity in women of reproductive age between 1999-2002 was 29% (3). More than a decade later this prevalence had risen to 38% (4). United Kingdom, has a reported prevalence of obesity in in women of reproductive age of 10.9% (5), Australia of 28% (6) and Asia of 22% (7).

At the same time, the reported prevalence of gestational diabetes mellitus (GDM) in Europe varies considerably, and in certain populations is reported to occur in more than 20% of pregnancies (8-10) raising to as high as 52% in women with a BMI [?] 29kg/m^2 (11).

With such high and rising prevalence, obesity and GDM have become the most common clinical risks in obstetric practice increasing the probability of a variety of pregnancy-related complications compared to women with a normal BMI and normal glucose tolerance(5, 12-16).

Beyond the impact of a high baseline BMI on pregnancy outcomes, the amount of weight gained during pregnancy can affect the immediate and future health of a woman and her infant. The Institute of Medicine (IOM) guidelines for weight gain in pregnancy recommend weight gain of 5-9kg for all obese women (17). Suboptimal gestational weight gain (GWG), either excessive or inadequate, is also associated with reported maternal and neonatal complications (18-20). Current research indicates that excessive GWG and high pre-pregnancy BMI are associated with increased risks for adverse pregnancy outcomes (19, 21).

Thus, the question remains whether more stringent recommendations for weight gain may improve GDM related outcomes, by reducing the additive effect of diabetes, obesity and excessive weight gain.

Recent studies have shown that in GDM women, minimal GWG led to higher rates of small for gestational age infants (SGA)(18). However, a study of overweight and obese GDM Asian women reported that minimal GWG and tight blood glucose control during pregnancy may eliminate most of the adverse pregnancy outcomes experienced (22).

The primary aim of this study was to investigate the effects of GWG below the IOM recommendation on pregnancy outcomes in women with GDM and a BMI [?]30 kg/m^2.

As a secondary aim, we compared pregnancy outcomes in obese GDM women with insufficient GWG, the IOM recommended GWG and excessive GWG.

Methods

The ATLANTIC Diabetes in Pregnancy Group (ATLANTIC DIP) consists of a number of antenatal centres along the Irish Atlantic seaboard and offers pre-pregnancy, antenatal and postnatal care to women with pre-gestational diabetes and GDM. Patient information is recorded in real time on the diabetes information system (DIAMOND, Hicom Woking, UK).

This current study is a retrospective cohort study of pregnancy outcomes for obese GDM women recorded in the Atlantic DIP database over a 7-year period, 2010 to 2016. Out of a total of 1319 women with GDM diagnosed according to International Association of the Diabetes and Pregnancy Study Groups (IADPSG) criteria and treated either with medical nutritional therapy (GDM-D) only or diet and insulin (GDM-I), we identified 754 women with a BMI [?]30 kg/m^2. Women were stratified according to their GWG status into three distinct groups; Group 1: women with weight loss and/or weight gain of <5kgs (n= 237); GDM- D (n=91); GDM-I (n= 146); Group 2: women with weight gain of 5-9kgs (n= 77); GDM-D (n=29); GDM-I (n=48); and group 3: women with weight gain >9kgs (n= 440); GDM-D (n=159), GDM-I (n=281), (Figure 1 and Table 1).

The IADPSG criteria confirm a diagnosis of GDM when fasting glucose is [?]5.1 mmol/L (92 mg/dL), 1-hour glucose is [?]10.0 mmol/L (180mg/dL) or 2-hour glucose is [?]8.5 mmol/L (153mg/dL) following a standard 2 hours 75-g OGTT. Women diagnosed with GDM are managed in a combined diabetes antenatal clinic and reviewed every 2-4 weeks by a multidisciplinary team including an obstetrician, diabetologist, and midwife/diabetes nurse specialist. Each patient receives a consultation on diet changes at GDM diagnosis and

additional consultations as required. During this consultation, the patient receives advice about carbohydrate intake and distribution. This is supplemented by written material and online access to other materials for consolidation of dietary advice. In addition, women have access by phone to a midwife/diabetes nurse specialist for advice during the standard working week.

BMI was calculated at first antenatal visit (weeks 11-14 of pregnancy) and stratified according to WHO guidelines as underweight (<18.5 kg/m^2), normal weight (18.5-24.9 kg/m^2), overweight (25-29.9 kg/m^2), and obese ([?]30 kg/m^2). Weight was measured at each clinic visit by the attending physician as per the local best practice weight measurement guidelines.

Consistent with local evidence-based guidelines, women are advised to monitor their blood glucose levels 7 times per day (fasting, pre-meals, 1-hour post meals, and at bedtime). Blood glucose targets are set at [?]5.3 mmol/L (95 mg/dL) for fasting / pre-meal, and [?]7.8 mmol/L (140 mg/dL) 1-hour post meals. Insulin is commenced when blood glucose readings are outside these ranges on more than 3 successive days. Women are commenced on a long acting analogue insulin (insulin detemir) titrating the dose every 3 days to achieve a fasting blood glucose level of [?]5.3 mmol/L (95mg/dL) and a short acting analogue insulin (insulin aspart) to achieve 1h post prandial blood glucose level [?]7.8 mmol/L (140 mg/dL).

The following maternal outcomes: caesarean section (CS), preeclampsia (PET),pregnancy induced hypertension (PIH), polyhydramnios, ante partum haemorrhage (APH) and post-partum haemorrhage (PPH) and infant outcomes: congenital malformations, neonatal mortality, admission to the Neonatal Intensive Care Unit (NICU), prematurity, large for gestational age (LGA), macrosomia, SGA, neonatal hypoglycaemia, respiratory distress and shoulder dystocia are recorded. PET is defined as new onset systolic blood pressure (SBP) of at least 140 mmHg and/or diastolic blood pressure (DBP) of at least 90 mmHg at more than 20 weeks gestation with proteinuria of greater than 300 mg/day. PIH is defined as new-onset BP at least 140/90mmHg after 20 weeks gestation with no proteinuria. Prematurity is defined as a baby born alive before 37 completed weeks of pregnancy. Mortality includes stillbirth and neonatal death. LGA is defined as an infant birth weight greater than the 90th percentile for sex and gestational age plotted on the WHO growth chart and macrosomia as an infant birth weight greater than 4000g. SGA is defined as an infant birth weight less than the 10th percentile for sex and gestational age plotted on the WHO growth chart. Neonatal hypoglycaemia is defined as a plasma glucose level of less than 1.65 mmol/L (30 mg/dL) in the first 24 hours of life and less than 2.5 mmol/L (45 mg/dL) thereafter. The decision to proceed with a caesarean delivery is made by the woman's obstetrician. Polyhydramnios is diagnosed when the amniotic fluid index measured is greater than 24cm on foetal ultrasound. Shoulder dystocia is defined as a vaginal cephalic delivery that requires additional obstetric manoeuvres to deliver the foetus after the head has delivered and gentle traction has failed.

Statistical analysis

Data were analysed using SPSS version 20 (Armonk, NY, IBM Corp). No imputations were carried out for missing data. The Kolomogorov-Smirnov test was used to evaluate data distribution. Differences in normally distributed data between the two groups were assessed by the independent t-test, with the Mann-Whitney U test used for non-normally distributed data. Chi-square was used for qualitative data to compare the two groups. Multivariate analysis was performed using multiple logistic regression to model relationships between less than recommended GWG (reference group: women with the IOM recommended GWG (Group 2)) and maternal and infant outcomes, correcting for age, smoking status, ethnicity, and family history of diabetes (first-degree relatives). Differences between the two groups were reported in adjusted odds ratio (aOR) and 95% confidence interval (CI). A $p<0.05$ was deemed statistically significant. Three-way ANOVA/ Kruskall Wallis were used to assess the differences in baseline characteristics and pregnancy outcomes between women with insufficient GWG, the IOM recommended GWG and excessive GWG.

Results

Maternal characteristics of women with insufficient GWG and women with the IOM recommended GWG are detailed in Table 1. In the total group (n = 314), age, ethnicity, family history of diabetes, smoking

status, starting BMI, booking SBP and DBP week of GDM diagnosis, parity and first recorded HbA_{1C} were not statistically significant between groups. On the 75g OGTT the 1-hour glucose value was higher (189mg/dL(10.5mmol/L) vs 181.8mg/dL (10.1 mmol/L) in women with GWG <5kg vs those with ideal GWG trending towards but not reaching statistical significance (p=0.05).

The groups were divided for further analysis into those treated with lifestyle intervention only (GDM-D) compared to those receiving insulin (GDM –I). In the GDM-I subgroup there were no differences in any of the baseline characteristics. In the GDM-D subgroup booking SBP (123.6mmHg vs 117.3mmHg; P<0.03) and DBP (73mmHg vs 68 mmHg; p<0.01) were significantly higher in women with GWG of <5kg compared to those with the recommended GWG. Both the 1-hour (189mg/dL(10.5mmol/L) vs 167.4 mg/dL(9.3mmol/L); p<0.01) and 2-hour (133.2mg/dL(7.4mmol/L) vs 118.8mg/dL(6.6 mmol/L); p=0.02) glucose values on the OGTT were significantly higher in those with GWG <5kg compared to those with the recommended GWG. Gestational week of delivery (38.9 vs 39.8, p=0.01) was lower in those with GWG <5kg compared to those with the recommended GWG. All other characteristics assessed were similar. On the 3-way analysis between women with insufficient GWG, the recommended amount of GWG and excessive amount of GWG, women with GWG <5kg were more likely to be older (34.4 years ±5.2 vs 33.8 years±4.5 vs 33.1 years ±5.4, p=0.01), to have a higher baseline BMI (37.1 kg/m^2±5.4 vs 36.4 kg/m^2±5.1 vs 35.5 kg/m^2±4.5, p<0.01) and deliver earlier (38.8 weeks ± 2.1 vs 39.2weeks ±1.4 vs 39.2 weeks ±2.3, p<0.01).Women with GWG above the IOM recommendations were more likely to have a higher DBP at booking (73.4mmHg±9.1 vs 72.7mmHg±9.7 vs 70.3mmHg±9.4, p=0.01) and higher HbA_{1c} levels (37.3mmol/mol ± 16.6 vs 35.3mmol/mol ± 8.8 vs 35.5mmol/mol ± 16.7) compared to women with GWG <5kg and women with GWG 5-9 kg.

Maternal outcomes are reported in Table 2. In the total group, women with GWG <5kg were more likely to have PPH (10% vs. 5.2%, p<0.01) or polyhydramnios (14.8% vs 5.3, p=0.03) compared to women with ideal GWG. On subgroup analysis, in the GDM-I group, women with GWG <5kg had higher rates of PPH (7.9% Vs 0%, p=0.05). In the GDM-D group, women with GWG <5kg were more likely to have PPH (13.2% Vs. 0%, p=0.03) and PIH (15.4% Vs 0%, p=0.02) compared to the ideal weight gain group. On the 3-way analysis between women with insufficient GWG, the recommended amount of GWG and excessive GWG, women with GWG <5kg were more likely to have higher rates of PPH (10%vs 5.2% vs 8.2%, p=0.02) and higher rates of polyhydramnios (14.8% vs 5.3% vs 5.2%, p<0.01). The higher rates of polyhydramnios were also found in the subgroup analysis in the GDM-I subgroup (20.1% vs 8.7% vs 5.2%, p< 0.01). Women with GWG above the IOM recommendations, were also more likely to have higher rates of PIH in the total group (18.5% vs 13.9% vs 6.6%, p=0.02) and in the GDM-D subgroup (20.1% vs 15.4% vs 0%, p=0.02).

Infant outcomes are shown in Table 3. The rates of LGA were higher in infants of GDM women with a GWG of <5kg compared to those who gained an ideal weight(17.3% vs 7.9%, p=0.04). On subgroup analysis, in the GDM-D group, the rate of prematurity was greater in infants of women with GWG <5kg (14.3% vs 0%, p=0.03). On the 3-way analysis between women with insufficient GWG, the recommended amount of GWG and excessive amount of GWG, infants of women with GWG <5kg were more likely to have a lower birth weight (3517.9g ± 566.6 vs 3563.2g ± 388.8 vs 3662.21± 604, p<0.01). However, there was no difference between groups in SGA rates (3.5% vs 1.3% vs 2.6%, p=0.50). Infants of women with GWG >9kg were more likely to have higher rates of macrosomia (26.6% vs 18.5% vs 15.6%, p=0.01) and LGA (23.5% vs 17.3% vs 7.9%, p<0.01) compared to the other 2 groups).

Logistic regression analysis (Table 4) determined that women with weight loss or GWG <5kg had higher odds ratios to develop PIH and polyhydramnios (OR 3, 95%CI 1.1-8.9, p=0.04; OR 3, 95%CI 1.1-8.1, p=0.04 respectively). However, when adjusted for baseline BMI, insulin use, smoking status, parity, family history, ethnicity and age, GWG <IOM recommendation in obese women with GDM was not associated with adverse outcomes.

Discussion

Main Findings and Interpretation

In our study we focused on the difference in baseline characteristics and pregnancy outcomes between obese

pregnant women diagnosed with GDM who lost weight or gained up to 5 kg compared to those who gained the IOM recommended weight of 5-9kg. A secondary analysis evaluated the differences between all 3 groups in our cohort: women with GWG <5kg, those with GWG of 5-9 kg and women with GWG>9 kg. We did not further subdivide the groups according to obesity category because that would have generated a very small number of study participants in each subcategory. To our knowledge, this study is one of a few aiming to investigate the relationship between high maternal pre-pregnancy BMI and GWG outwith the IOM recommendations.

Few studies have specifically addressed weight loss or insufficient GWG in pregnancy, as this is generally not promoted in pregnancy (23-28).

Our study did not find higher rates of SGA or early prematurity in women with weight loss or insufficient GWG. A retrospective study (23) found that weight loss in obese pregnant women diagnosed with GDM is associated with higher odds for SGA (aOR 1.69, 95% CI 1.32–2.17) and preterm delivery <34 weeks (aOR 1.71, 95% CI 1.23–2.37). This study, despite having a very large cohort, used different GDM diagnostic criteria (Carpenter and Coustan) to our study (IADPSG) and had a different population profile in terms of baseline characteristics; their study included overweight and obese women while our study focused only on obese only women. Similar to the findings of these authors, we found higher rates of prematurity in women with weight loss/insufficient GWG treated with diet alone, but this was not statistically significant on adjusted logistic regression. Our finding that weight loss after a GDM diagnosis in obese women is not associated with a lower birth weight is supported in a recent study by Katon *et al* (25). However, the latter study had a relatively small sample size and did not analyse markers of foetal growth such as SGA and LGA. Bauer *et al* (26) also found no increased odds for SGA or prematurity in obese GDM women who lost weight or maintained their weight during pregnancy. Recently, Kurtzhals *et al* (29) found improved foetal growth in women with restricted GWG with no increased rates of SGA or LGA. A direct comparison to our study findings however is difficult as women were diagnosed by the Danish Criteria which are higher compared to the IADPSG criteria, the baseline BMI was self-reported with an inevitable risk of recall bias and women were not stratified according to their BMI.

Conversely, in our population of obese women with GDM, the rates of LGA babies were higher in those who lost weight or gained <5kg as compared to women who gained 5-9kg although this difference was eliminated on logistic regression analysis. This may be due to the higher baseline BMI in the GWG <5kg group suggesting that in our population pre-pregnancy BMI has a greater impact on foetal growth not compensated by weight loss or minimal GWG during pregnancy. This finding albeit controversial, is supported by other studies (30-32).

Another interesting finding was the higher rates of PPH and polyhydramnios in GDM women with low GWG although again the significance is lost on adjusted logistic regression. It is known the polyhydramnios is associated with higher rates of PPH due to uterine stretching. The current literature examining the link between obesity and PPH is contradictory (33, 34). Studies that have assessed potential links between weight loss in obese women (without GDM) and PPH (27, 35) found no association. A recent study (23) found improved rates of polyhydramnios in obese GDM women with weight loss but this study concentrated on gestational weight change related outcomes in a BMI heterogenous population and there was no sub analysis on obese only study participants.

In evaluating adverse outcomes between women who gained insufficient weight, women who gained 5-9 kg and women who gained >9kgs, we found that women in the first group were older and had a higher pre-pregnancy BMI compared to the other 2 groups, this finding is supported by others (23, 36). Moreover, and supporting the results of our primary analysis, the insufficient GWG group had higher rates of polyhydramnios and PPH, even compared to the excessive GWG group but lost on adjusted logistic regression analysis. A recent study (37) found higher rates of polyhydramnios in euglycemic women that were older and had higher pre-pregnancy weight gain. Another study (38) also found higher rates of polyhydramnios in women with a higher pre-pregnancy BMI. These studies were not restricted to GDM patients but complement the results of our study and suggest that baseline BMI may plays a greater role in the physiopathology of polyhydramnios

beyond dysglycemia and GWG.

Women with excessive GWG had higher rates of PIH, macrosomia and LGA and this is supported by a large body of current literature ((23, 27, 39, 40)

Strengths and limitations

Our study is not without limitations. This is a retrospective convenience sample and prone to confounding bias, while every effort was made to control for potential confounding factors by implementing multivariable logistic regression analyses to estimate the association between gestational weight loss and pregnancy outcomes, there may be residual variables for which we did not have information. Furthermore, given the study design, we acknowledge that some outcomes are underpowered and that a higher number of study participants might reveal stronger associations.

Another study limitation is the procedure for assessment of patient's weight. At our centres, equipment is calibrated on a regular basis. Due to the retrospective nature of the study it is difficult to guarantee the standardisation of the weighing process.

Moreover, we do not know the reasons for weight loss in this population. It is possible that some women lost weight because of factors that placed them at risk of adverse outcomes, but we are unable to fully explore this. We assumed that women who lost/gained insufficient weight likely made lifestyle changes subsequent to their GDM diagnosis. We acknowledge that further investigation is required to clarify the reasons for weight loss in this population. In addition, due to the observational nature of this data, we cannot state a causal relationship between gestational weight loss and adverse outcomes. Since it is not possible to perform a randomized controlled study of weight loss/insufficient GWG versus adequate GWG (as recommended by the IOM), we believe that the current analysis provides valuable information that may be useful in caring for this high-risk population.

Lastly, we used HbA1c as a measure of glycemic control as individual readings were recorded in a paper-based diary retained by the study participant and thus not available to the research team.

Conclusion

This study found that GWG of <5kg is not associated with any substantial neonatal risk. On adjusted logistic regression analysis maternal outcomes also were not different. Notwithstanding, validation through a prospective study with a larger obese GDM cohort is required before the findings presented here could be recommended for routine clinical use and further research is required to better understand how to perform successful interventions to optimize pregnancy weight change.

Data availability

Data is available on request

Conflict of Interest

The authors have no conflict of interest

Author Contributions

DB, POS and FPD designed the study. DB analysed the data and drafted the initial manuscript. All authors (DB, MM, AK, POS and FPD) made substantial contributions to the acquisition and interpretation of data, revised the manuscript critically for important intellectual content and approved the final version to be published. DB is responsible for the integrity of the work as a whole.

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Figure 1. Schematic detailing of study participants, numbers and sub-categories

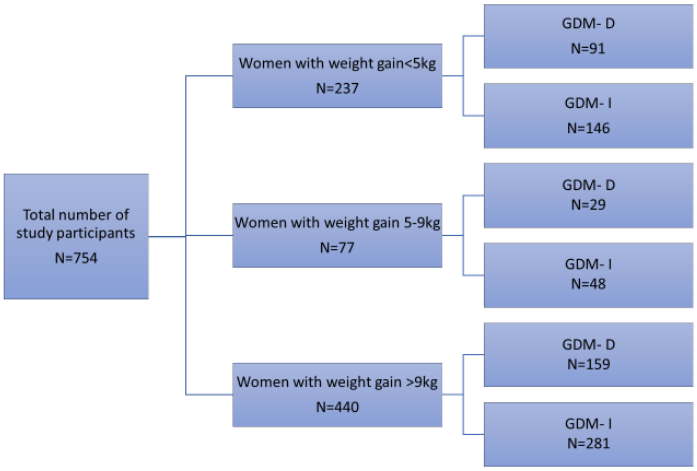

Table 1. Baseline characteristics of women with GDM and BMI [?] 30 kg/m^2

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
Agpar 1 min ^	8.7±0.9	8.8±0.6	0.90	8.7±0.7	0.9
Agpar 5 min ^	9.4±1.1	9.3±0.7	0.20	9.3±0.5	0.1
Birthweight (grams)^	3517.9±566.6	3563.2 ± 388.8	0.70	3662.21± 604	**<0.01**
Mortality	1/235 (0.4%)	0/77	0.50	2/440 (0.5%)	0.8
NICU	88/234 (37.6%)	24/77 (31.2%)	0.30	140/435 (32.2%)	0.3
Shoulder dystocia	1/232 (0.4%)	0/77	0.50	6/435 (1.4%)	0.3
Malformations	5/233 (2.1%)	2/76 (2.6%)	0.80	14/436 (3.2%)	0.7
Hypoglycaemia	12/235 (5.1%)	1/77 (1.3%)	0.10	21/440 (4.8%)	0.3

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
Respiratory distress	16/235 (6.8%)	4/77 (5.2%)	0.60	22/440 (5%)	0.6
Macrosomia	43/233 (18.5%)	12/77 (15.6%)	0.50	116/436 (26.6%)	**0.01**
LGA	40/231 (17.3%)	6/76 (7.9%)	**0.04**	101/430 (23.5%)	**<0.01**
SGA	8/231 (3.5%)	1/76 (1.3%)	0.30	11/430 (2.6%)	0.5
Prematurity	29/235 (12.3%)	6/77 (7.8%)	0.20	44/440 (10%)	0.4
Insulin treated	**n=146**	**n=48**		**n=281**	
Agpar 1 min ^	8.7±0.9	8.7 ±0.7	0.90	8.7±0.7	0.9
Agpar 5 min ^	9.4±1.3	9.3 ± 0.8	0.50	9.4±0.5	0.1
Birth weight (grams) ^	3580.5±531.5	3586± 443	0.90	3722.3±593.3	0.03
NICU	72/143 (50.3%)	19/48 (39.6%)	0.10	2/281 (0.7%)	0.8
Mortality	1/144 (0.7%)	0/48	0.50	106/276 (38.4%)	0.05
Shoulder dystocia	1/141 (0.7%)	0/48	0.50	3/276 (1.1%)	0.7
Malformations	4/142 (2.8%)	2/47 (4.3%)	0.60	10/277 (3.6%)	0.8
Hypoglycaemia	9/144 (6.3%)	1/48 (2.1%)	0.20	12/281 (4.3%)	0.4
Respiratory distress	10/144 (6.9%)	3/48 (6.3%)	0.80	14/281 (5%)	0.7
Macrosomia	31/142 (21.8%)	9/48 (18.8%)	0.60	86/278 (30.9%)	0.05
LGA	28/140 (20%)	5/47 (10.6%)	0.10	72/272 (26.5%)	**0.03**
SGA	5/140 (3.6%)	1/47 (2.1%)	0.60	5/272 (1.8%)	0.5
Prematurity	16/144 (11.1%)	6/48 (12.5%)	0.70	27/281 (9.6%)	0.7
Diet treated	**n=91**	**n=29**		**n=159**	
Agpar 1 min ^	8.7±1	8.9±0.4	0.80	8.7±0.8	0.9
Agpar 5 min ^	9.4±0.7	9.3±0.5	0.10	9.3±0.6	0.2
Birth weight (grams) ^	3420.2±607.8	3525.7±280.6	0.50	3556.3± 610	0.1
NICU	16/91 (17.6%)	5/29 (17.2%)	0.90	0/159	N/a
Mortality	0/91	0/29	N/a	34/159 (21.4%)	0.7
Shoulder dystocia	0/91	0/29	N/a	3/159 (1.9%)	0.3
Malformations	1/91 (1.1%)	0/29	0.50	4/159 (2.5%)	0.5
Hypoglycaemia	3/91 (3.3%)	0/29	0.30	9/159 (5.7%)	0.3
Respiratory distress	6/91 (6.6)	1/29 (3.4%)	0.50	8/159 (5%)	0.7
Macrosomia	12/91 (13.2%)	3/29 (10.3%)	0.60	30/158 (19%)	0.3
LGA	12/91 (13.2%)	1/29 (3.4%)	0.10	29/158 (18.4%)	0.09

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
SGA	3/91 (3.3%)	0/29	0.30	6/158 (3.8%)	0.5
Prematurity	13/91 (14.3%)	0/29	**0.03**	17/159 (10.7%)	0.09

Group 1 = lost weight or gained 0-5 kg; Group 2 = gained 5-9kg; Group 3 = gained >9kg. ˆ = *mean±SD;*# p-values represent the significance levels for comparison between Group 1 and Group 2. *p-values represent the significance levels for comparison between Groups 1,2 and 3. There are missing cases in each category

Table 2. Maternal Outcomes

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
Agpar 1 min ˆ	8.7±0.9	8.8±0.6	0.90	8.7±0.7	0.9
Agpar 5 min ˆ	9.4±1.1	9.3±0.7	0.20	9.3±0.5	0.1
Birthweight (grams)ˆ	3517.9±566.6	3563.2 ± 388.8	0.70	3662.21± 604	**<0.01**
Mortality	1/235 (0.4%)	0/77	0.50	2/440 (0.5%)	0.8
NICU	88/234 (37.6%)	24/77 (31.2%)	0.30	140/435 (32.2%)	0.3
Shoulder dystocia	1/232 (0.4%)	0/77	0.50	6/435 (1.4%)	0.3
Malformations	5/233 (2.1%)	2/76 (2.6%)	0.80	14/436 (3.2%)	0.7
Hypoglycaemia	12/235 (5.1%)	1/77 (1.3%)	0.10	21/440 (4.8%)	0.3
Respiratory distress	16/235 (6.8%)	4/77 (5.2%)	0.60	22/440 (5%)	0.6
Macrosomia	43/233 (18.5%)	12/77 (15.6%)	0.50	116/436 (26.6%)	**0.01**
LGA	40/231 (17.3%)	6/76 (7.9%)	**0.04**	101/430 (23.5%)	**<0.01**
SGA	8/231 (3.5%)	1/76 (1.3%)	0.30	11/430 (2.6%)	0.5
Prematurity	29/235 (12.3%)	6/77 (7.8%)	0.20	44/440 (10%)	0.4
Insulin treated	**n=146**	**n=48**		**n=281**	
Agpar 1 min ˆ	8.7±0.9	8.7 ±0.7	0.90	8.7±0.7	0.9
Agpar 5 min ˆ	9.4±1.3	9.3 ± 0.8	0.50	9.4±0.5	0.1
Birth weight (grams) ˆ	3580.5±531.5	3586± 443	0.90	3722.3±593.3	0.03
NICU	72/143 (50.3%)	19/48 (39.6%)	0.10	2/281 (0.7%)	0.8
Mortality	1/144 (0.7%)	0/48	0.50	106/276 (38.4%)	0.05
Shoulder dystocia	1/141 (0.7%)	0/48	0.50	3/276 (1.1%)	0.7
Malformations	4/142 (2.8%)	2/47 (4.3%)	0.60	10/277 (3.6%)	0.8
Hypoglycaemia	9/144 (6.3%)	1/48 (2.1%)	0.20	12/281 (4.3%)	0.4
Respiratory distress	10/144 (6.9%)	3/48 (6.3%)	0.80	14/281 (5%)	0.7

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
Macrosomia	31/142 (21.8%)	9/48 (18.8%)	0.60	86/278 (30.9%)	0.05
LGA	28/140 (20%)	5/47 (10.6%)	0.10	72/272 (26.5%)	**0.03**
SGA	5/140 (3.6%)	1/47 (2.1%)	0.60	5/272 (1.8%)	0.5
Prematurity	16/144 (11.1%)	6/48 (12.5%)	0.70	27/281 (9.6%)	0.7
Diet treated	**n=91**	**n=29**		**n=159**	
Agpar 1 min ˆ	8.7±1	8.9±0.4	0.80	8.7±0.8	0.9
Agpar 5 min ˆ	9.4±0.7	9.3±0.5	0.10	9.3±0.6	0.2
Birth weight (grams) ˆ	3420.2±607.8	3525.7±280.6	0.50	3556.3± 610	0.1
NICU	16/91 (17.6%)	5/29 (17.2%)	0.90	0/159	N/a
Mortality	0/91	0/29	N/a	34/159 (21.4%)	0.7
Shoulder dystocia	0/91	0/29	N/a	3/159 (1.9%)	0.3
Malformations	1/91 (1.1%)	0/29	0.50	4/159 (2.5%)	0.5
Hypoglycaemia	3/91 (3.3%)	0/29	0.30	9/159 (5.7%)	0.3
Respiratory distress	6/91 (6.6)	1/29 (3.4%)	0.50	8/159 (5%)	0.7
Macrosomia	12/91 (13.2%)	3/29 (10.3%)	0.60	30/158 (19%)	0.3
LGA	12/91 (13.2%)	1/29 (3.4%)	0.10	29/158 (18.4%)	0.09
SGA	3/91 (3.3%)	0/29	0.30	6/158 (3.8%)	0.5
Prematurity	13/91 (14.3%)	0/29	**0.03**	17/159 (10.7%)	0.09

Group 1 = lost weight or gained 0-5 kg; Group 2 = gained 5-9kg; Group 3 = gained >9kg. APH = Antepartum haemorrhage; PPH= Post-partum haemorrhage PET=Pre-eclampsia; PIH= pregnancy induced hypertension; C section = caesarean section; ˆ = mean±SD;# p-values represent the significance levels for comparison between Group 1 and Group 2. *p-values represent the significance levels for comparison between Groups 1,2 and 3. There are missing cases in each category.

Table 3. Infant outcomes

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
Agpar 1 min ˆ	8.7±0.9	8.8±0.6	0.90	8.7±0.7	0.9
Agpar 5 min ˆ	9.4±1.1	9.3±0.7	0.20	9.3±0.5	0.1
Birthweight (grams)ˆ	3517.9±566.6	3563.2 ± 388.8	0.70	3662.21± 604	**<0.01**
Mortality	1/235 (0.4%)	0/77	0.50	2/440 (0.5%)	0.8
NICU	88/234 (37.6%)	24/77 (31.2%)	0.30	140/435 (32.2%)	0.3
Shoulder dystocia	1/232 (0.4%)	0/77	0.50	6/435 (1.4%)	0.3
Malformations	5/233 (2.1%)	2/76 (2.6%)	0.80	14/436 (3.2%)	0.7

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
Hypoglycaemia	12/235 (5.1%)	1/77 (1.3%)	0.10	21/440 (4.8%)	0.3
Respiratory distress	16/235 (6.8%)	4/77 (5.2%)	0.60	22/440 (5%)	0.6
Macrosomia	43/233 (18.5%)	12/77 (15.6%)	0.50	116/436 (26.6%)	**0.01**
LGA	40/231 (17.3%)	6/76 (7.9%)	**0.04**	101/430 (23.5%)	**<0.01**
SGA	8/231 (3.5%)	1/76 (1.3%)	0.30	11/430 (2.6%)	0.5
Prematurity	29/235 (12.3%)	6/77 (7.8%)	0.20	44/440 (10%)	0.4
Insulin treated	**n=146**	**n=48**		**n=281**	
Agpar 1 min ^	8.7±0.9	8.7 ±0.7	0.90	8.7±0.7	0.9
Agpar 5 min ^	9.4±1.3	9.3 ± 0.8	0.50	9.4±0.5	0.1
Birth weight (grams) ^	3580.5±531.5	3586± 443	0.90	3722.3±593.3	0.03
NICU	72/143 (50.3%)	19/48 (39.6%)	0.10	2/281 (0.7%)	0.8
Mortality	1/144 (0.7%)	0/48	0.50	106/276 (38.4%)	0.05
Shoulder dystocia	1/141 (0.7%)	0/48	0.50	3/276 (1.1%)	0.7
Malformations	4/142 (2.8%)	2/47 (4.3%)	0.60	10/277 (3.6%)	0.8
Hypoglycaemia	9/144 (6.3%)	1/48 (2.1%)	0.20	12/281 (4.3%)	0.4
Respiratory distress	10/144 (6.9%)	3/48 (6.3%)	0.80	14/281 (5%)	0.7
Macrosomia	31/142 (21.8%)	9/48 (18.8%)	0.60	86/278 (30.9%)	0.05
LGA	28/140 (20%)	5/47 (10.6%)	0.10	72/272 (26.5%)	**0.03**
SGA	5/140 (3.6%)	1/47 (2.1%)	0.60	5/272 (1.8%)	0.5
Prematurity	16/144 (11.1%)	6/48 (12.5%)	0.70	27/281 (9.6%)	0.7
Diet treated	**n=91**	**n=29**		**n=159**	
Agpar 1 min ^	8.7±1	8.9±0.4	0.80	8.7±0.8	0.9
Agpar 5 min ^	9.4±0.7	9.3±0.5	0.10	9.3±0.6	0.2
Birth weight (grams) ^	3420.2±607.8	3525.7±280.6	0.50	3556.3± 610	0.1
NICU	16/91 (17.6%)	5/29 (17.2%)	0.90	0/159	N/a
Mortality	0/91	0/29	N/a	34/159 (21.4%)	0.7
Shoulder dystocia	0/91	0/29	N/a	3/159 (1.9%)	0.3
Malformations	1/91 (1.1%)	0/29	0.50	4/159 (2.5%)	0.5
Hypoglycaemia	3/91 (3.3%)	0/29	0.30	9/159 (5.7%)	0.3
Respiratory distress	6/91 (6.6)	1/29 (3.4%)	0.50	8/159 (5%)	0.7
Macrosomia	12/91 (13.2%)	3/29 (10.3%)	0.60	30/158 (19%)	0.3

Variable	Group 1 n=237	Group 2 n=77	#p value	Group 3 n=440	*p value
LGA	12/91 (13.2%)	1/29 (3.4%)	0.10	29/158 (18.4%)	0.09
SGA	3/91 (3.3%)	0/29	0.30	6/158 (3.8%)	0.5
Prematurity	13/91 (14.3%)	0/29	**0.03**	17/159 (10.7%)	0.09

Group 1 = lost weight or gained 0-5 kg; Group 2 = gained 5-9kg; Group 3 = gained >9kg. LGA = Large for gestational age; SGA = Small for gestational age; NICU= Neonatal intensive care unit; ˆ = mean±SD;

N/a = Not applicable; #p-values represent the significance levels for comparison between Group 1 and Group 2. *p-values represent the significance levels for comparison between Groups 1, 2 and 3. There are missing cases in each category.

Table 4 . Logistic regression unadjusted and adjusted for baseline BMI, insulin use, smoking status, parity, family history, ethnicity and age for the women who lost weight or gained 0-5 kg (Group 1).

Variable	OR	95% CI	#P value	aOR
Maternal Outicomes	**Maternal Outicomes**	**Maternal Outicomes**	**Maternal Outicomes**	**Maternal Outico**
PET	1.7	0.5-6.3	0.3	2.4
PIH	3.0	1.1-8.9	**0.04**	7.4
APH	0.7	0.5-7.1	0.3	0.3
PPH	0.9	0.1-1.1	0.9	1.1
C section total	1.2	0.7-2	0.4	1.3
C section elective	1.1	0.6-1.8	0.8	1.3
C section emergency	1.3	0.6-2.6	0.4	1.2
Polyhydramnios	3.0	1.1-8.1	**0.04**	1.7
Infant Outcomes	**Infant Outcomes**	**Infant Outcomes**	**Infant Outcomes**	**Infant Outcomes**
Macrosomia	1.2	0.5-2.4	0.5	0.8
LGA	2.4	1.1-6.1	0.05	1.2
SGA	2.7	0.3-21	0.3	0.2
Prematurity	1.6	0.6-4.1	0.2	2.4
Mortality	0.2	0.2-0.9	0.9	0.2
Respiratory distress	0.6	0.2-2.3	0.7	1.1
NICU	0.7	0.4-1.3	0.3	0.8
Shoulder dystocia	0.1	0.1-0.7	0.5	N/a
Malformations	1.2	0.2-6.4	0.8	1.6
Hypoglycaemia	0.1	0.03-1.9	0.1	0.2

OR = Odds Ratio; aOR= Adjusted Odds Ratio;# p-values represent the significance levels for OR;

*p-values represent the significance levels for aOR; N/a = Not applicable; PET=Pre-eclampsia.

PIH= pregnancy induced hypertension; APH = Ante-partum haemorrhage; PPH= Post-partum haemorrhage; LGA = Large for gestational age; SGA = Small for gestational age; NICU= Neonatal intensive care unit.