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Research Article

PRE-PREGNANCY BODY MASS INDEX AND WEIGHT GAIN DURING PREGNANCY: RELATIONS WITH GESTATIONAL DIABETES AND HYPERTENSION, AND BIRTH OUTCOMES¹Dr.Saira Mahmood, ²Dr.Wardah Rehmat, ³Dr.Mahrukh Saif¹ Sheikh Zayed Medical College, Rahim Yar Khan² Sheikh Zayed Medical College, Rahim Yar Khan³ Sheikh Zayed Medical College, Rahim Yar Khan

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Abstract

To study the relationship between pre-pregnancy body mass index (BMI) and weight gain during pregnancy with pregnancy and birth outcomes, with a focus on gestational diabetes and hypertension and their role in the association with fetal growth. Weight before pregnancy (W1) and weight after delivery (W2) were collected and we calculated BMI and net gestational weight gain, net GWG = (W2 - W1)/(weeks of gestation). Gestational diabetes, hypertension gestational age and birth weight were collected. High BMI was more strongly related to the risk of giving birth to a large-for-gestational-age (LGA) baby than high net GWG (odds ratio OR [95% CI] of 3.23 [1.86–5.60] and 1.61 [0.91–2.85], respectively). Higher net gestational weight gain was significantly associated with an increased risk of LGA only after accounting for blood pressure and glucose disorder. High gestational weight gain should not be neglected in regard to risk of LGA in women without apparent risk factors.

Keywords: Obesity, Pregnancy, Gestational diabetes, Hypertension, Birth weight.**Corresponding author:****Dr. Saira Mahmood,**

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INTRODUCTION:

Overweight and obesity are recognized as significant contributors to ill-health and world-wide burden of disease [1], estimated to affect approximately 1.3 billion adults globally [2]. In the Australian obstetric population 34% of women are overweight or obese [3], although more recent data would suggest that this is approaching 50% [4]. These figures are consistent with international data, which indicate 50–60% of women are overweight or obese on entering pregnancy [5].

Obesity and associated cardio-metabolic complications deserve particular attention in the context of pregnancy. Indeed, it is now well recognized that maternal obesity at conception increases the risk of complications in pregnancy, labor, and birth for both the mother and the neonate [6,7]. In particular, gestational diabetes mellitus and hypertensive disorders are more prevalent in obese pregnant women [8]. These complications themselves confer higher risks of adverse fetal and neonatal outcome, such as large-for-gestational-age (LGA) neonates in mothers with gestational diabetes, or intra-uterine growth restriction in mothers with gestational hypertension [9,10]. Another important factor influencing pregnancy and birth outcomes is weight gain during pregnancy [11]. Most studies show that the higher the pre-pregnancy BMI, the lower the pregnancy weight gain [12]. It has been shown that the risks of gestational diabetes and gestational hypertension could be different according to the amount of weight gain, independent of pre-pregnancy BMI. Increasing maternal body mass index (BMI) is a well recognised risk factor for the development of gestational diabetes [13,14], the two conditions sharing a similar metabolic milieu characterized by insulin resistance, hyperglycaemia, hyperlipidaemia, and a low-grade state of chronic inflammation, which in turn has been documented to influence the availability and transfer of nutrients to the developing foetus [15]. Furthermore, adipose tissue, far from being an inert tissue, has a critical role in innate immune sensing, the production of varying adipocytokines (leptin, TNF- α , IL-6), antagonists to the effect of insulin [16].

2. METHODS:

This prospective cohort study is nested within the LIMIT randomised trial, evaluating the effect of an antenatal dietary and lifestyle intervention for women who are overweight or obese [17]. The methodology of the LIMIT randomised trial has been described in detail previously. The LIMIT trial recruited women with a live singleton pregnancy and a BMI of 25 kg/m² between 10 and 20 weeks' gestation, at the time of their first antenatal appointment. All women provided written

informed consent to participate. Eligible, consenting women were randomized using a central telephone randomization service, and a randomization schedule prepared by non-clinical research staff with balanced variable blocks. Women were randomised to the Dietary and Lifestyle Advice Group or the Standard Care Group. Women in the latter group continued to receive their pregnancy care according to local hospital guidelines and comprise the cohort for this current analysis. Women who are overweight or obese receiving antenatal care according to local hospital guidelines are not routinely provided with lifestyle and behavioral advice. Women were recruited from public maternity hospitals across the South Australian metropolitan area (specifically, Women's and Children's Hospital, Lyell McEwin Hospital, and Flinders Medical Centre). Ethics approval was obtained from all sites.

At the time of trial entry, all women had their height and weight measured, and BMI calculated. Women were then categorised according to their BMI as either overweight (BMI 25.0–29.9 kg/m²) or obese (BMI 30.0 kg/m²), with obesity further classified into subclass 1 (BMI 30.0–34.9 kg/m²), subclass 2 (BMI 35.0–39.9 kg/m²), and subclass 3 (BMI 40.0 kg/m²), utilising World Health Organisation criteria [18]. To coincide with routine antenatal testing, all women were offered a fasting oral glucose tolerance test (OGTT) at 26–28 weeks' gestation. If an OGTT was not undertaken as the initial investigation, an oral glucose challenge test (OGCT) was performed as per routine antenatal care at 26–28 weeks' gestation, with progression to an OGTT if abnormal. A diagnosis of GDM was made for results of fasting blood glucose 5.5 mmol/L or blood glucose 7.8 mmol/L two hours after a loading dose of carbohydrate (75 g of glucose), according to the South Australian state-wide perinatal practice guidelines. These guidelines are based on the previous ADIPS guidelines, which were current at the time of the trial commencement. The cohort assessed in this study is comprised of the women who were randomised to the control group or Standard Care Group of the LIMIT trial. Routine antenatal care of a woman who is diagnosed with GDM includes education by a midwife or diabetic educator regarding diet, home monitoring of blood sugar levels, and referral for treatment as needed. Blood sugar monitoring is initially four times per day aiming for blood glucose between 3.5 and 5.5 mmol/L fasting and 4–7 mmol/L two hours after a meal. If good control is achieved then testing may be reduced. Medical treatment is considered if fasting values are 5.5 mmol/L once or more per week, or if postprandial values are 7.5 mmol/L twice or more per week. It is routine care to plan for delivery at 38 + 0 weeks in women with poor

glycaemic control, polyhydramnios or suspected macrosomia, and at term in women with no spontaneous onset of labour [19].

Clinical outcomes considered included pre-eclampsia (in accordance with recognised Australasian Society for the Study of Hypertension in Pregnancy criteria) [34]; need for induction of labour; caesarean birth; infant macrosomia (defined as birth weight above 4000 g); gestational age at birth; large for gestational age (defined as infant birth weight 90% for gestational age); and admission to the neonatal intensive care unit. Outcomes were abstracted from the woman and infant's case notes after birth by a research assistant.

Statistical analyses were performed with the use of SAS software, version 9.3 (Cary, NC, USA), to evaluate the proportion of women with and without GDM, and the proportion of women experiencing each clinical outcome of interest by both BMI category and presence or absence of GDM. The effect of BMI category (overweight or obese) on GDM, and the effect of both BMI and GDM on each clinical outcome, was assessed using log binomial regression models. Results are presented as relative risks (RR) with 95% confidence intervals. Where there was no significant interaction identified between BMI and GDM on the outcomes considered, the interaction term was removed from the model and the overall effect of BMI and GDM on the risk of each outcome was estimated. A *p* value of less than 0.05 was considered to indicate statistical significance (2-sided)

3. RESULTS:

During the study period, a total of 1030 women were included in the cohort for this analysis, with 445 (43.20%) categorised as overweight, and 585 (56.80%) as obese. Of the women with BMI 30 kg/m², 298 (28.93%) were obese subclass 1, 172 (16.70%) obese subclass 2, and 115 (11.17%) obese subclass 3. A total 709 women underwent an OGTT (68.8%) and 321 women underwent an OGCT (32.2%). Gestational diabetes was diagnosed in 115 women (11.17%), with the baseline characteristics of women at the time of pregnancy booking and clinical outcomes presented by BMI and GDM in Table 1. The incidence of gestational diabetes increased with increasing maternal BMI; 6.74% overweight vs 13.42% obese subclass 1 vs 12.79% obese subclass 2 vs 20.00% obese subclass 3 (Fig 1). Women who were obese were twice as likely to develop GDM compared with women who were overweight (RR 2.16; 95% CI 1.45 to 3.21; *p* = 0.0002).

There were no statistically significant interactions identified between maternal BMI and gestational diabetes for the clinical outcomes of interest, except for gestational age at delivery (GA). Women who were obese were significantly more likely to require caesarean birth (RR 1.27; 95% CI 1.07 to 1.50; *p* = 0.006), and deliver a large for gestational age (LGA) infant (RR 1.38; 95% CI 1.07 to 1.77; *p* = 0.01), independent of GDM. Women who were diagnosed with gestational diabetes were significantly less likely to give birth to an infant with birth weight above 4 kg (RR 0.60; 95% CI 0.36 to 0.1.00; *p* = 0.05), independent of BMI. Both women who were obese and those who had GDM were more likely to deliver at an earlier gestational age, and the interaction between GDM and obesity was significant (0.005). The reduction in gestational age attributable to GDM and obesity was similar. Induction of labor was not significantly increased regardless of BMI or GDM. Infants of women who were diagnosed with GDM were more likely to require admission to the neonatal intensive care unit (NICU) (RR 2.41; 95% CI 1.04, 5.61; *p* = 0.04), independent of maternal BMI. There was a trend towards increased admission to NICU in the infants of women who were obese (RR 2.42; 95% CI 0.98 to 5.99; *p* = 0.06) compared with women who were overweight, independent of GDM.

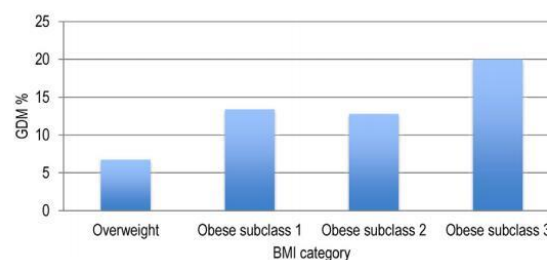


Fig. 1 – Prevalence of GDM by BMI category.

DISCUSSION:

Our results showed that weight gain in pregnancy was lower when pre-pregnancy BMI was higher. In particular, women who were overweight or obese before pregnancy generally gained less weight during pregnancy and very few had an excessive weight gain. Conversely, only 2 of the 161 lean women had a low weight gain. This relationship has been well established [20-22] but the reason for this is not obvious. It can be speculated that medical management and/or personal efforts for not gaining too much weight could be responsible for a part of this association. However, during pregnancy, fat is stored to secure energy supply during fetal growth and lactation. In obese women, no additional storage is necessary, which suggests that pregnancy weight gain could be restricted because of physiological mechanisms.

Our study was based on self-reported measures of weight before pregnancy whereas many studies have found that weight is more likely to be under-reported by women with a high pre-pregnancy BMI [23]. Such a bias would therefore impact on the calculation of gestational weight gain and lead to an over-estimation of weight gain in overweight and obese women. We performed a sensitivity analysis and used weight measured at booking instead of recalled weight for the calculation of gestational weight gain and BMI. Gestational weight gain was divided by the number of remaining weeks of gestation, and then multiplied by 41 to obtain a full-term pregnancy. The results obtained on the 1,351 mothers who booked before 15 weeks were very similar, gestational weight gain was respectively of 10.9, 10.2, 8.4 and 4.3 kg in lean, normal, overweight obese mothers, respectively (results not shown). We also performed all the association analyses presented in the paper with those new two variables and the results were unchanged, even if less significant because of the loss of power (results not shown). As expected, [24], gestational hypertension and gestational diabetes were more frequent in women with higher pre-pregnancy BMI. High weight gain was also associated with an increased risk of gestational hypertension, but in contrast, a tendency for an inverse relation was observed between weight gain and risk of gestational diabetes. This last result was also obtained in previous studies [25], and in particular in a recent Danish study on about 60,000 term pregnancies [26]. This could be attributable to reverse causation, since diagnosis of gestational diabetes may be accompanied with diet recommendations resulting in a decreased weight gain in late pregnancy, as Catalano et al. [27] previously suggested.

On the other hand, it is also possible that common factors such as weak insulin resistance present from the beginning of pregnancy favor both gestational diabetes and low weight gain during pregnancy. Because of different habits in the two centres, the criteria for inviting mothers to a second glucose load test were different. Indeed, the number of women performing this test was higher in Nancy (290) than in Poitiers (179), but the final number of cases was not significantly higher in Nancy (6.6%) than in Poitiers (6%, $P = 0.50$). Therefore, those methodological differences between the centers may not have impacted on our results. Consistent with previous studies, we found that low weight gain was significantly associated with a higher risk of pre-term delivery [28]. We show that this is true even if only maternal weight gain is considered. Moreover, the association was strengthened after excluding women with gestational diabetes and gestational hypertension. The lowest risk of

prematurity was observed in medium–high weight gain (12–16 kg net maternal weight gain for a term pregnancy, 16–20 kg equivalent total weight gain during pregnancy).

Some studies found that the magnitude of the association decreases as pre-pregnancy BMI increases [29], whereas in our study, the interaction with pre-pregnancy BMI was not significant, probably because of the small number of cases in the extreme groups. Recently, Nohr et al. [30] found a statistically significant interaction between the effects of pre-pregnancy BMI group and weight gain on preterm birth: the risk was potentiated at the extremes, namely among underweight women with a low weight gain and obese women with a high weight gain. However, this interaction was observed only for induced preterm deliveries, not for spontaneous preterm births, suggesting the predominant role of obstetric care in obesity related diseases. Low maternal weight gain may indicate deficiencies in nutrients, a lack of expansion of plasma volume, infection, or other unidentified problems. Further understanding of these associations is needed, because it remains unclear whether they are causal and therefore amenable to nutritional interventions.

CONCLUSION:

In conclusion, our analysis using net maternal weight gain and taking into account glucose and blood pressure disorders reinforces the association between low maternal weight gain with premature birth and between high weight gain with LGA compared to that with pre-pregnancy BMI. Regarding the risk of LGA, our results suggest that monitoring weight gain in obese women may reduce but not alleviate the increased risk of LGA amongst these women. Moreover, high weight gain in women without gestational diabetes and/or gestational hypertension increases the risk of LGA, suggesting that more attention should be paid to mothers without apparent risk factors. However, weight gain has to be monitored tightly as low weight gain is also associated with the risk of pre-term delivery.

Conflict of interest

The authors declare that they have no conflict of interest.

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