Gestational Weight Gain and Gestational Diabetes Mellitus: Popular Beliefs and Emerging Evidence

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ABSTRACT
The aim of this article is to review the evidence on gestational weight gain (GWG), and its relationship between abnormal foetal growth and the development of gestational diabetes mellitus (GDM). Maternal obesity is a major concern in modern obstetrics and is associated with increased maternal and foetal complications. It is becoming increasingly common and GWG has attracted an upsurge of interest.

In 2009, recommendations from the Institute of Medicine in the United States revised downwards the weight gain recommendations in pregnancy for obese mothers. There is no international consensus or guidelines on GWG. There are concerns with evidence on GWG and many publications have methodological shortcomings.

No causal relationship has been shown between GWG and excessive foetal growth; however, emerging evidence demonstrates a relationship between birth weight and maternal fat-free mass. Interventions in pregnancy aimed at targeting GWG have not shown a decrease in excessive foetal growth and inappropriate intervention may lead to foetal malnutrition with adverse clinical consequences. While an association between pre-pregnancy maternal obesity and GDM has been shown, evidence of the relationship between weight gain during pregnancy and GDM is conflicting.

A recent study has shown that fat mass gain to 28 weeks gestation does not influence the development of GDM. Careful consideration should be paid to the advice that pregnant women are given about GWG and their lifestyle before, during, and after pregnancy and we believe that it may be more appropriate to shift the focus of attention from monitoring maternal weight to increasing physical activity levels and improving nutritional intake.

INTRODUCTION
Maternal obesity is a major concern in modern obstetrics. It is increasingly common, is associated with additional risks for both mother and baby and gives rise to added costs to the health services. The subject of gestational weight gain (GWG) has attracted an upsurge of interest in recent years, particularly as the prevalence of maternal obesity is increasing. It is in this context that this article aims to review the evidence on the relationship between GWG, abnormal foetal growth and the development of gestational diabetes mellitus (GDM).

In 2009, the Institute of Medicine (IOM) in the United States published a review of all existing evidence on GWG. It revised GWG guidelines based on pre-pregnancy body mass index (BMI) independent of age, parity, smoking history, race, and ethnic background. The focus of previous guidelines had been on the attainment of adequate weight gain in pregnancy as to minimise foetal and neonatal complications. The newer publication attempted to provide guidance on the balance between weight gain in pregnancy and outcomes for both mother and baby. This shift of emphasis, taking into account the possible negative outcomes associated with excessive as well as inadequate weight gain, resulted in recommended weight gain for obese women during pregnancy being revised downwards. These guidelines were based on the US population only. There has been no international consensus on recommendations for GWG.

CONCERNS WITH THE CURRENT EVIDENCE
Many publications on GWG have methodological shortcomings. Studies on GWG to date have been fraught with difficulties and high quality scientific evidence is lacking. The IOM report acknowledged that, “Unfortunately, most of the data available to the Committee was not collected with a high level of rigor and most studies relied on recall weight values.” The accuracy of self-reported weight was reviewed in 32 studies involving 57,172 women, and in each study, women underestimated their weight. Self-reported weight leads to faulty categorisation of BMI in 22% of cases and has led to the diagnosis of obesity being missed in 5% of cases. Studies based on self-reporting of maternal weight both under-diagnose obesity and exaggerate the risk of obesity because women with mild obesity report themselves in the overweight category.

There is the additional problem of timing in the calculation of maternal BMI. In the reported studies there is wide variance in the times when BMI is calculated. Some studies utilised the last measured weight before pregnancy while others utilised the first recorded weight in pregnancy. Pre-pregnancy weight has not been defined and in women who are anovulatory a change in weight may stimulate ovulation. For example, ovulation is promoted by weight gain in underweight women and by weight loss in those with polycystic ovarian syndrome.

During pregnancy weight is dependent on gestational age and assessment of weight therefore requires knowledge of gestational age. A study of 1,000 women in early pregnancy showed no change in mean weight or body composition during the first trimester. Measuring weight gain obviously requires the measurement of weight at two different time points. A problem arises with the timing of the repeat measurement in pregnancy. Some studies have used the last measured weight in pregnancy, without account being taken of the importance of gestational age.

In previous studies pregnancies were not dated accurately by ultrasound and so the gestational age at the time of the BMI calculation not known. Some, such as the landmark Hyperglycaemia and Adverse Pregnancy Outcome (HAPO) study, calculated BMI only as late as 24-32 weeks gestation. At this gestation, BMI does not reflect that of early pregnancy and analysis of clinical outcomes cannot distinguish between the influence of BMI and pregnancy weight gain in the second trimester. Gestational age is an important determinant not only of GWG itself but also of the clinical outcomes of GWG. Therefore clinical outcomes can only be compared when there is accurate knowledge of gestational age. This is particularly important when examining outcomes such as birth weight or babies classed as small for gestational age (SGA) or large for gestational age (LGA). Early pregnancy dating by ultrasound should be a prerequisite for any accurate study of GWG.
GWG AND ABERRANT FOETAL GROWTH

Associations between GWG and aberrant foetal growth have been reported, with higher weight gain associated with LGA babies and lower weight gain associated with SGA babies. However, since weight gain in pregnancy includes the weight of the baby, it is hardly surprising that women carrying larger babies have increased their weight more than women carrying smaller babies. Such epidemiological associations do not provide evidence of causation.

Despite a significant rise in the prevalence of maternal obesity no increase in the number of babies born with a birth weight in excess of 4.5 kg has been identified. Studies have found, in spite of an increase in the prevalence of obesity there has been an associated decrease in babies with macrosomia. Therefore, recommendations regarding GWG and excessive foetal growth must take into account the evidence that causal factors for excessive foetal growth have not been well established.

Emerging evidence demonstrates that birth weight is related more closely to maternal fat-free mass than maternal fat mass. A positive correlation was found between birth weight and an increase in maternal fat-free mass but not fat mass, in a study of 63 women. Previous studies showed that maternal water gain is predictive of birth weight. A recent study from Dublin reported a correlation between birth weight and maternal body composition in 2618 women studied prospectively. The mothers’ body composition was measured directly using bioelectrical impedance analysis during the first trimester. Multivariate regression analysis found that birth weight correlated significantly with fat-free mass but not fat mass, suggesting that attempts to reduce maternal fat mass during pregnancy are unlikely to decrease birth weight. Even if evidence for a causal relationship between GWG and excessive foetal growth were established, interventions in pregnancy targeted at limiting GWG have not been shown to prevent excessive foetal growth. Reviews of interventions aimed at optimizing GWG have concluded that the literature is of poor quality and results are inconsistent.

Micronutrient and macronutrient deficiencies in pregnancy are associated with poorer clinical outcomes for both mother and baby. Interventions focused on losing weight during pregnancy should ensure that the growing fetus is not deprived of essential nutrients as a result. The promotion of weight management programmes based on increasing physical activity may be safer than those focusing on dietary restrictions, with an overall emphasis on the promotion of a healthy lifestyle.

Attention has been drawn to an association between obstetric morbidity and high pre-pregnancy BMI. Obesity in pregnancy is associated with an increased risk of a number of serious adverse outcomes, including miscarriage, foetal congenital anomaly, pre-eclampsia, postpartum haemorrhage, stillbirth and neonatal death. There is a higher caesarean section rate and lower breastfeeding rate in this group of women compared to women with a healthy BMI. Recent guidelines recommend that women with diabetes. All of these studies were limited by the methodological problems previously outlined.

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CONCLUSION

GWG is a topic of increased interest however, there are huge gaps in the research and methodological shortcomings of studies and to date little evidence for a causative relationship between GWG and absent fetal growth or the development of GDM has been shown. We recommend that strategies promoting healthy pregnancy should focus on providing resources to optimise pre-pregnancy BMI and to promoting a healthy lifestyle in pregnancy rather than focusing on weight gain during pregnancy.

References