Gestational weight gain: an ounce of prevention is still worth a pound of cure

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Abbreviations

CDC	Centers for Disease Control and Prevention
GDM	Gestational diabetes mellitus
GWG	Gestational weight gain
HAPO	Hyperglycemia and Adverse Pregnancy Outcome
IOM	Institute of Medicine
ISI	Insulin sensitivity index
LGA	Large for gestational age
SGA	Small for gestational age

Introduction

Gestational weight gain (GWG) is an important factor in the antenatal management of pregnancy. Most obstetrics professional societies [1–5] address the issue of appropriate weight gain during pregnancy. Recommendations for GWG, however, have varied over the years, often without the benefit of sound scientific evidence, ranging from encouraging pregnant women to ‘eat for two’, to recommending minimal weight gain to prevent complications of pregnancy. In 1990 the Institute of Medicine (IOM) of the National Academy of Science in the United States convened a committee to recommend dietary intake and specific GWG recommendations with the goal of delivery of a healthy full-term infant of appropriate size [6]. The 1990 IOM committee noted that there was a positive relationship of both pre-pregnancy maternal weight and GWG with birthweight. Their recommendations were in part a response to the increased risk of the perinatal morbidity and mortality of

infants with low birthweight related to poor GWG. The committee report advised an average GWG of 9.1–11.4 kg and advised against the then-current practice of limiting GWG to 4.5–6.4 kg. In 2009, in part because of the increase in numbers of overweight and/or obese people in the population and the lack of specific GWG guidelines for obese women (the 1990 committee recommended target for women with a BMI >29.0 was at least 6.8 kg), the IOM convened a committee to re-examine the GWG guidelines [7]. The results of the report were published in 2009 based on the evidence available at that time (see Table 1). A further goal was to recommend support for researchers to conduct studies on the determinants and impact of GWG, and pattern of weight gain on maternal and child outcomes. This, in the context of the Developmental Origins of Health and Disease (DOHaD) hypothesis, has led to a substantial increase in the number of publications on GWG and impact on pregnancy outcomes over the last decade.

Relevance

Excessive GWG has implications for mothers: for example, excessive GWG increases the risk of maternal gestational diabetes mellitus (GDM) [8] and postpartum weight retention [9]. Excessive GWG represents accrual of adipose tissue in overweight and obese women, which increases the long-term risk of being overweight or obese [10]. This commentary, however, will focus on the short- and long-term effects of inadequate and excessive maternal GWG on the offspring. The positive relationship between GWG and birthweight is much stronger in women who are lean compared with obese pre-pregnancy, i.e. the higher the BMI before pregnancy, the less GWG is a determinant of birthweight. By contrast, the lower the pre-pregnancy BMI the greater the GWG required to achieve an adequate birthweight. This relationship was described by Abrams and Laros more than 30 years ago [11]. Similarly, our group reported that increasing GWG above 2009 IOM

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Table 1

IOM 2009 gestational weight gain recommendations		
Pre-pregnancy BMI category	Total weight gain (kg)	Rate of weight gain in 2nd and 3rd trimesters (kg/week) ^a
Underweight (<18.5 kg/m ²)	12.5–18	0.51 (0.44–0.58)
Normal weight (18.5–24.9 kg/m ²)	11.5–16	0.42 (0.35–0.50)
Overweight (25–29.9 kg/m ²)	7–11.5	0.28 (0.23–0.33)
Obese ^b (≥30 kg/m ²)	5–9	0.22 (0.17–0.27)

^a Calculations assume a first trimester weight gain of 0.5–2.0 kg. Data are presented as mean (range)

^b 1990 IOM recommendation: for obese women (BMI >29), weight gain at least 6.8 kg

Adapted from Committee Opinion Number 548: Weight gain during pregnancy, The American College of Obstetricians and Gynecologists (2013) [1]

recommendations resulted in higher adiposity in newborn offspring of women with normal weight, but the degree of GWG in obese women was not related to neonatal adiposity [12]. The relationship between GWG lower than the 2009 IOM recommendations and fetal health is generally well accepted for underweight and normal weight women because of the increased risk of a small for gestational age (SGA) newborn with poor GWG. There is more controversy regarding the suggestion that GWG in overweight and obese women should be lower than the IOM guidelines. Some investigators report that, for overweight and obese women, GWG lower than the IOM guidelines, or even weight loss in pregnancy, should be considered, in order to minimise adverse health outcomes for mothers and their offspring [13]. However, a recent meta-analysis of over 1,000,000 women reports that, while obese women who gain less than the IOM GWG guidelines decrease the risk of a large for gestational age (LGA) or macrosomic baby, there is an increased risk of having an SGA baby or of preterm birth [14]. The impact of excessive GWG on the offspring of those with GDM is associated with an increased risk of LGA, macrosomia and gestational hypertension [15]. However, the effect of GWG lower than the IOM guidelines in women with GDM is less well characterised, but was addressed in two publications in this issue of *Diabetologia* [16, 17].

Knowledge gained from the two papers

In the report by Kurtzhals et al, women with less than 34 weeks' gestation were diagnosed with GDM on the basis of local Danish criteria (and HbA_{1c} ≤6.5% [48 mmol/mol]) if diagnosed before 20 weeks, to avoid including women with pre-existing diabetes) [16]. All women were encouraged to follow an energy-restricted diet of approximately 6000 kJ/

day and an exercise programme of 30 min of physical activity/day. After the diagnosis of GDM, all women were asked to gain weight within guidelines similar to those of the IOM. In this retrospective study, women were stratified into three groups based on GWG over approximately 11 weeks of treatment: restricted GWG (53%), appropriate GWG (16%), and excessive GWG (31%) based on second and third trimester IOM GWG/week guidelines for pre-pregnancy BMI. Measures of glucose tolerance, HbA_{1c}, GWG and fetal abdominal circumference standard deviation (AC-SD; as a measure of in utero growth) were comparable between the three groups before treatment at 27[±]5¹ weeks. The average weight gain was 0 kg, 3 kg and 5 kg, respectively, in the three groups. In women in the restricted GWG group, in addition to a decrease in GWG during treatment, there was a significant decrease in total GWG, HbA_{1c}, need for insulin treatment, and birthweight SD score compared with the excessive GWG group. In the multivariate linear regression analysis, excessive GWG during dietary treatment and HbA_{1c} in late pregnancy were identified as potentially modifiable clinical risk factors for infant birthweight SD score. Of note there was no significant difference between the restricted GWG and appropriate GWG groups in relation to preterm delivery, Caesarean section, AC-SD score, birthweight, birthweight SD score, SGA or LGA. One take-home message from this study is that it is possible to institute lifestyle interventions in women with GDM resulting in only 31% of women gaining excessive weight over the latter stages of pregnancy.

The study by Tam et al followed 905 mother–child pairs who participated in the original Hong Kong cohort of the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study [17]. The aim of the study was to evaluate the relationship between GWG and cardiometabolic risk factors in offspring at age 7. The analyses were adjusted for multiple

covariates: model 1 (the child's sex, age and/or height); model 2 (model 1 + maternal pre-pregnancy BMI and/or family history of diabetes and hypertension); model 3 (model 2 + environmental factors during pregnancy [including maternal age, glucose area under the curve (AUC) during the OGTT, mode of delivery and gestational age at delivery], breastfeeding and child exercise level); model 4 (model 3 + birthweight); and model 5 (model 4 + childhood BMI). Mean pre-pregnancy BMI was 20.9 ± 2.9 kg/m² and 17.2% gained less than the IOM GWG guidelines, 41.8% within the guidelines, and 41.0% gained more than the guidelines.

After model 1 adjustment, offspring of mothers who gained more weight than the IOM GWG guidelines had a higher BMI, waist circumference, blood pressure and insulin AUC, and were more insulin resistant (higher HOMA-IR and lower Matsuda insulin sensitivity index [ISI]) with a greater insulin response than offspring of women who gained weight within the IOM guidelines. When adjusted for models 2–4, the associations remained significant except for HOMA-IR and systolic blood pressure.

Among women gaining less weight than the IOM guidelines, the offspring had higher diastolic blood pressure, insulin AUC and pancreatic beta cell function, and a lower Matsuda ISI than women who gained weight within the guidelines. When adjusted for models 2–4, higher diastolic blood pressure and pancreatic beta cell function and lower Matsuda ISI were mitigated but remained significant.

The authors then performed a quadratic analysis between maternal GWG and childhood metabolic traits. In model 1 there was a U-shaped relationship between standardised GWG and pancreatic beta cell function, diastolic blood pressure, higher weight and hip circumference, insulin AUC and Matsuda ISI (inverted U). Adjustments for Models 2–4 attenuated but did not eliminate the quadratic associations. However, all the associations for both inadequate and excessive GWG were notably attenuated (with the exception of diastolic blood pressure) when adjusted for model 5 (including childhood BMI). Although GDM per se was not identified in the Tam et al study, in the original HAPO study there was a linear relationship between maternal levels of glucose and adverse pregnancy outcomes [18]. Because the investigators assessed the AUC for glucose and insulin relative to GWG, one can infer that the relationship follows what would be expected in women with untreated GDM, which was the case in the HAPO study. The significance of this study is in the U-shaped relationship between inadequate and excessive GWG and long-term cardiometabolic function in the offspring at age 7.

Limitations

Although Kurtzhals et al conclude that restricted GWG was associated with more appropriate fetal growth in women with

GDM, there was a significant difference only in comparison with the women with GDM who had excessive GWG [16]; there were no significant differences in neonatal outcomes between the restricted GWG and the appropriate GWG groups. Furthermore, birthweight or birthweight SD score may not detect subtle differences in growth among groups, such as differences in lean or fat mass. Although there were no differences in head circumference scores based on ultrasound measures before and after treatment in the Kurtzhals et al study, decreased birthweight, lean mass (including head circumference and length) and fat mass have been reported in neonates of overweight and obese women, including women with GDM, who had inadequate GWG, defined as less than 5 kg [19]. On the basis of the data from the Tam et al study, there may be increased cardiometabolic risks in children of those women who had restricted GWG [17]. The population in the Tam et al study was exclusively Chinese and, in line with norms for this population, the mean pre-pregnancy BMI was 20.9 ± 2.9 and the prevalence of overweight and obesity was only 8.3%, which is lower than most Western populations. Whether these results would apply to a more overweight/obese population is unknown. Finally, in model 5, adjusting for childhood BMI, the relationships among GWG and cardiometabolic outcomes at age 7, other than for diastolic blood pressure percentile, were no longer significant. The data from both these studies emphasise the importance of factors beyond GWG relating to long-term childhood metabolic outcomes, including but not limited to environmental, genetic, epigenetic, paternal, nutrition, and lifestyle factors before, during, and after pregnancy.

Where to go from here?

On the basis of the available evidence, what should the recommendations be for those women who are already pregnant and have GDM or risk factors for GDM, such as a positive family history of diabetes and/or are overweight or obese? Healthy eating and increased physical activity, as demonstrated by Kurtzhals et al, can help avoid excessive GWG regardless of maternal pre-pregnancy BMI. Although encouraging such behaviour early in pregnancy, even before a diagnosis of GDM, may not mitigate perinatal morbidities, lifestyle intervention to avoid excessive GWG will make returning to pre-pregnancy weight less onerous for women. Based on data from the Centers for Disease Control and Prevention (CDC) in the United States, only 26.3% of women reported receiving GWG advice based on IOM guidelines from their healthcare provider [20]. Hence, there are multiple opportunities for advances in achieving appropriate GWG and avoiding inadequate or excessive GWG, based on IOM guidelines, including use of technologies such as antenatal electronic health records and multiple apps for tracking GWG for use at home.

What about prevention? We know, based on CDC data, that 38% of women with normal weight fall within the appropriate GWG category, compared with only 24% of overweight/obese women. Further, only 37% of normal weight women have excessive GWG, in contrast to 50–60% of overweight/obese women [21]. Hence, every effort should be made to improve metabolic function by optimising BMI prior to a planned pregnancy, as maternal obesity prior to pregnancy is a very important risk factor for fetal overgrowth/adiposity [22]. It is not necessary to achieve a normal BMI in women who are obese, in order to improve metabolic health: there is evidence that a 5–7% reduction in weight can improve metabolic function in overweight and obese individuals [23]. There have been concerted efforts to convey this message to both healthcare providers and patients, with multiple research projects now in progress to assess the utility of this approach. However, these efforts must not be limited to the periconception period but should follow a life-course approach involving not just healthcare providers but also the larger community where our patients live and work [24].

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