REVIEW ARTICLE

Weight gain in pregnancy: is less truly more for mother and infant?

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Summary: Although more than 50% of women gain weight above the Institute of Medicine (IOM) guidelines for weight gain in pregnancy and excessive weight gain is an independent risk factor for significant maternal and neonatal morbidity and offspring obesity, there is little consensus over the ideal weight gain during pregnancy. Surprisingly, the 2009 IOM guidelines varied minimally from the 1990 IOM guidelines, and many critics advocate lower weight gain recommendations. This review explores the energy costs of pregnancy, the relationship between gestational weight gain and birth weight, and considers what gestational weight gain minimizes both large-for-gestational age as well as small-for-gestational age infants. An extensive examination of the current data leads this author to question whether the current weight gain recommendations are too liberal, especially for obese pregnant women.

Keywords: obesity, high-risk pregnancy, maternal–fetal medicine, metabolism, nutrition

INTRODUCTION

Although obesity in pregnancy is the leading health risk responsible for the greatest maternal and neonatal morbidity[1,2] and excessive gestational weight gain (GWG) is an independent risk factor for childhood obesity, unabated controversy persists over the ideal weight gain during pregnancy. The strongest predictor of postpartum weight retention is GWG and >60% of previous normal weight gravidas become overweight with their subsequent pregnancy.[3,4] Pregnant women often receive contradictory advice regarding weight gain recommendations in pregnancy and it is not surprising that interventions to minimize excessive GWG have been largely disappointing. In the face of the growing obesity epidemic, it was perplexing for many clinicians that the 2009 Institute of Medicine (IOM) recommendations for weight gain in pregnancy[4,5] were not very different from the recommendations in 1990 (Tables 1 and 2). In fact, the only significant modifications of the guidelines were to use the World Health Organization (WHO)/National Heart Lung Blood Institute (NHLBI) criteria for body mass index (BMI) cut-offs and to add an upper limit of weight gain for obese women (5–9 kg instead of at least 6.8 kg), but there was still no distinction between weight gain recommendations with regard to severity of obesity (Class I: BMI 30–34.9; Class II: BMI 35–39.9; and Class III: BMI of ≥40). What, in fact, are the energy costs of pregnancy? What weight gain is considered obligate for a pregnant woman? What is the relationship between GWG and birth weight? At what GWG can both small-for-gestational age (SGA) infants and large-for-gestational age infants (LGA) be minimized? An examination of the current data may suggest that the most recent weight gain guidelines are subject to further modification and may be too generous for obese women.

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RATIONALE SUPPORTING THE 1990 IOM GUIDELINES

Weight gain recommendations for pregnant women have been subject to changing concerns and demographics since the first recommendations were made in the 1930s that all pregnant women should gain approximately 6.8 kg (15 lbs), irrespective of weight status.[6] This sentiment changed in 1970 with the realization that overly restricting weight gain could be harmful and result in low birth weights and a new recommendation of 9–12.3 kg (20–27 lbs) was proposed. During the years between 1970 and 1990, it was increasingly recognized that one size weight gain might not fit all BMIs. In 1990, the IOM published recommendations based on maternal BMI category and a major priority at that time was preventing SGA given the exceedingly high costs related to the care of SGA infants and their long-term health consequences. The data from the 1980s that served as the primary basis of the 1990 guidelines demonstrated that nearly as many women were underweight as overweight (20–30% in each group) and that the prevalence of obesity in women 20–39 years was only 12.3%.[7] The 1990 guidelines primarily considered the data to minimize low birth weight or preterm birth.[8]

OBSERVATIONS NECESSITATING AN UPDATE OF THE 1990 IOM GUIDELINES

In 1994, the prevalence of obesity in women of child-bearing age in the USA jumped to nearly 21% and by the year 2006, 33% of

Table 1 **1990 Institute of Medicine Guidelines**

Prepregnancy BMI	Total weight gain (kg)	Rate weight gain 2nd/3rd trimester
Low BMI (<19.8)	12.5–18 (28–40 lbs)	0.5 (~1 lb/week)
Normal BMI (19.8–26)	11.5–16 (25–35 lbs)	0.4 (1 lb/week)
Overweight BMI (>26–29)	7–11.5 (15–25 lbs)	0.3 (0.66 lb/week)
Obese BMI (≥29)	≥6.8 (≥15 lbs)	Not specified

BMI, body mass index

women in the USA were considered obese, with nearly two-thirds of USA women either overweight or obese.[7,9] In fact, 8% of women made criteria for severe obesity with a BMI ≥ 40.[7] The obesity rate in the UK was reported at ~23%. At the same time, the rapidly increasing prevalence of childhood obesity was being recognized and the National Health and Nutrition Examination Surveys (NHANES 2007–2008) estimated that nearly 17% of children and adolescents were obese in 2008 compared with only ~5% in 1980. Globally, an increasing trend of LGA infants was being recognized in Denmark, Sweden, Canada and the USA, which constituted a 20% rise from 1990 to 2000. In the USA, by 2006, nearly 20% of all infants were considered LGA compared with only 7.6% SGA. Soon thereafter, it was appreciated that the majority of LGA infants were born not to women with pre-existing diabetes (DM) or gestational diabetes (GDM), but to obese women. Importantly, the increase in birth weights appeared directly related to the increase in maternal weights during the same time period.[10] Further, infants born to obese women were not simply larger in respect to both lean and fat mass, but primarily due to an increase in fat mass which accounted for nearly 50% of the variance in birth weight.[10] As the prevalence of maternal obesity and LGA infants continued to overwhelm the prevalence of underweight mothers and SGA infants, the IOM reconvened to consider maternal and infant outcome data according to GWG in the face of the adult and childhood obesity epidemic.[5,11]

'EATING FOR TWO' MAY NOT BE SUCH A GOOD IDEA

Data from 2007 demonstrated that in the USA, the obesity rate was >25% in 30 states.[12] Possibly the most concerning statistics that mobilized the IOM to re-examine the guidelines cited that ~38% of normal weight women, ~63% of overweight women and ~46% of obese women gained more than the 1990 IOM recommendations.[13] Additionally, gestational diabetes, pre-eclampsia and caesarean delivery rates were rising and clearly related to maternal BMI and excessive weight gain[1,12]

Table 2 **2009 Institute of Medicine Guidelines**

Prepregnancy BMI	Total weight gain (kg)	Rate weight gain 2nd/3rd trimester
Low BMI (<18.5)	12.5–18 (28–40 lbs)	0.5 (~1 lb/week)
Normal BMI (18.5–24.9)	11.5–16 kg (25–35 lbs)	0.4 (1 lb/week)
Overweight BMI (25–29.9)	7–11.5 kg (15–25 lbs)	0.3 (0.66 lb/week)
Obese BMI (≥30)	5–9 kg (11–20 lbs)	0.2 (0.5 lb/week)

BMI, body mass index

as was the risk of increasing BMI categories between pregnancies due to postpartum weight retention.

EFFECT OF MATERNAL WEIGHT GAIN ON INFANT ADIPOSITY AND CHILDHOOD METABOLIC SYNDROME

Soon after the recognition of the Barker hypothesis that an intrauterine environment characterized by nutritional deficiency not only resulted in growth restriction at birth but also carried an increased risk of later diabetes and cardiovascular disease, the Developmental Origins of Disease hypothesis[14–18] was expanded to include intrauterine nutrient excess as a risk factor for later obesity and metabolic disease. The discovery of epigenetics provided a conceptual framework that explained how metabolic factors (glucose, lipids, amino acids, growth factors, cytokines, hormones) in the intrauterine environment could alter DNA methylation and histone modification to change gene expression modifying number, growth and function of many cells, promote adipogenesis and later impact appetite regulation via the hypothalamus and alter mitochondrial function of the offspring.[19–24] Although pre-gravid BMI was being increasingly recognized as the strongest independent risk factor for LGA, infant adiposity, and later childhood obesity,[8,10,25] increasing data emerged that GWG also independently contributed to infant adiposity and the risk of metabolic syndrome in childhood. A number of mother–infant cohorts demonstrated an effect of excessive GWG and offspring adiposity at 5–7 years of age[26] and some studies showed more of an effect of GWG in the first and second trimester than the third on childhood BMI.[27]

In a large study ($n = 175$) which examined the correlates of metabolic syndrome in children aged 6–11, the risks of maternal obesity or being born LGA posed a higher risk than being born from a mother with GDM.[28] An analysis of 1044 mother–child pairs from the Harvard Project Viva which aimed to ascertain the specific effect of GWG on adiposity in the offspring at three years of age[29] was published shortly thereafter. Fifty-four percent of the mothers gained excessive weight according to the 1990 IOM guidelines and one-third of the women had a BMI > 26. However, the most provocative finding was that women who gained according to the guidelines still had a 3.8-fold increased risk of having a three-year-old at or above the 95th percentile for weight compared with those who gained less than the guidelines recommended. This risk increased to 4.3-fold in those who gained an excessive amount of weight. Further, although women who gained weight according to the IOM guidelines had an increased risk of LGA infants (as did women who gained more than the guidelines), women who gained less than the recommended guidelines did not increase their risk of having an SGA infant. Such unfavourable offspring data further brought into question the appropriateness of the 1990 weight gain guidelines.

POSTPARTUM WEIGHT RETENTION AND SUBSEQUENT MATERNAL BMI

Although the majority of studies demonstrate that maternal BMI is the strongest risk factor for childhood obesity,[25,30] a strong driver of BMI in the next pregnancy is postpartum weight retention. The strongest predictor of one year postpartum weight retention is GWG[11,31] and weight retention at one

year predicts weight at 15 years. In fact, excessive GWG increases the risk of being overweight by an odds ratio of 2.2 and obesity by 4.5 at 21 years after pregnancy.[32] The majority of women never lose their GWG and go into each subsequent pregnancy at a higher BMI. The greater the weight gain, the greater the weight retention at one year. The ethnic group that is at the most extreme risk for weight retention is black women who gain more than 6.8 kg (15 lbs) during their pregnancy.[33] In the Avon Longitudinal Study of Parents and Children (ALSPAC) of 2356 mothers in the UK, women with a high GWG had a three-fold increased odds of overweight and central adiposity 16 years after pregnancy.[34]

ENERGY COSTS OF PREGNANCY AND OBLIGATE WEIGHT GAIN REQUIREMENTS

If one were to better understand the energy costs of pregnancy, perhaps a more logical basis for weight gain recommendations could be made. The calories required for synthesizing the actual products of conception contribute the least to the energy costs of pregnancy because fetal growth is slow and is extended over nine months. Hence, the greatest energy cost is actually maintaining the pregnancy. The additional energy deposited as maternal fat is what accounts for the tremendous variation in the energy costs of pregnancy between lean women who store little fat compared with women who store up to 50% of their GWG as fat. As a direct consequence of larger fat stores, the basal metabolic rate (BMR) is driven upward, further increasing the energy cost of pregnancy. Many clinicians are taught that pregnant women require an additional ~300 kcal per day to maintain the pregnancy (~77,000 kcal), which was derived from the estimated energy costs of a well-nourished woman in UK[35] who gained ~10 kg (~22 lbs). However, when one examines where those estimated energy costs come from, only ~5000 kcal accounts for the energy deposited in the conceptus as new tissue compared with ~36,000 kcal deposited as fat which requires another ~36,000 kcal to maintain the new tissue and extra maternal weight, estimated as the increase in BMR over the pregnancy. In comparison with an ~19 kg (~41 lbs) weight gain which is not uncommon in Swedish pregnant women, the estimated energy costs would be ~5000 kcal for the conceptus, ~75,000 kcal as fat and another ~64,000 kcal for the increase in BMR over pregnancy or an extra 540 kcal per day (144,000 kcal total). This is in contrast to the typical 5.5 kg (12 lbs) weight gain for a less affluent mother in Thailand for which only 12,000 kcal is directed to maternal fat deposits, 25,000 kcal is required by the increase in BMR and 5000 kcal for the conceptus (160 extra kcal per day or 42,000 kcal total). Thus, the quantity of maternal fat deposition and the increase in BMR required to maintain it is the driving factor for the energy cost of pregnancy.[35] Women in even less affluent countries who gain almost no weight are in a negative energy balance in pregnancy and burn their own fat but are at risk for severe ketonaemia.

A slightly different perspective to consider is the maternal compartmental location of weight gain in pregnancy, i.e. 'where weight gain goes.' The mother who delivers a healthy term infant would typically gain 3400 g (7.5 lbs) for the fetus, 650 g for the placenta (1.4 lbs), 970 g for the increase in uterus size (2.1 lbs), 405 g for the increase in breast development (0.9 lbs), 800 g for amniotic fluid (1.8 lbs) and 1450 g for the increase in blood volume (3.2 lbs), which are all considered obligate weight gain requirements. In addition, most women increase their extravascular water by 1480 g (3.3 lbs) and fat mass by 3345 g (7.4 lbs). Thus, the obligate weight gain is ~7800 g (17 lbs) which would require ~60,000 or 225 kcal extra per day. The obligate weight gain plus the increase in extravascular water and fat drives the caloric requirement to 100,000 kcal, the total weight gain to ~12.5 kg (28 lbs) or an extra 375 kg/day.

RELATIONSHIP BETWEEN GWG AND BIRTH WEIGHT

As early as 1986, it was reported that although there was a clear relationship between maternal weight gain and birth weight in underweight and normal weight women, this relationship was not the case for obese women.[36] Obese women did not appear to need to gain any significant weight in order to have a normally grown infant (~3500 g) and even overweight women who did not gain any weight had, on average, an infant weighing at least 3200 g (~7 lbs). Therefore, although underweight women and normal weight women were at risk of having an SGA infant without adequate weight gain, this was not the case with overweight and especially obese women. Many recent subsequent studies have demonstrated that the relationship between SGA and inadequate weight gain only holds for normal weight and underweight women.[18,37–42] In a systematic review of outcomes of the 35 highest quality studies drawn from the report conducted for the Agency for Healthcare Research and Quality (AHRQ), the authors concluded that there was strong support between excessive weight gain and LGA but only strong support between inadequate weight gain and SGA in normal and underweight women.[6] SGA infants born to overweight or obese women appear to be related to maternal morbidities resulting in placental insufficiency rather than inadequate weight gain.

NEWER STUDIES SUPPORTING LESS WEIGHT GAIN THAN 2009 IOM GUIDELINES

A number of studies, both immediately before[29,37,38,43] and soon after[6,18,39–41,44–47] the IOM announced their 2009 recommendations, were published supporting less weight gain, especially for overweight and obese women. Critics of the guidelines[4] began to publish their opposition to the guidelines. The IOM committee did not include studies that included pre-eclampsia and GDM as adverse outcomes in their analysis because they stated that such outcomes are confounded by other factors that are not related to weight gain. Yet, the committee included caesarean delivery as an outcome in their analysis, which is confounded by even more factors than simply maternal weight gain. Concerns that the committee did not give enough credence to the role of GWG as a driving factor for postpartum weight retention and subsequent BMI in the next pregnancy were also voiced. Not adequately adjusting for smoking and low socioeconomic status, and especially for not stratifying the weight gain recommendations for more severe levels of obesity when ~8% of US women have a BMI $\geq$ 40, received sharp criticism.

Many of the major studies that supported less weight gain than the 2009 IOM recommendations examined adverse outcomes other than only SGA or LGA. DeVader *et al.*[43] examined Missouri birth certificate data in ~95,000 newborns in normal

weight women (BMI 19.8–26) and noted that women who gained $<\sim 11.4$ kg (25 lbs) had lower odds of pre-eclampsia, cephalic-pelvic disproportion, failed induction, caesarean delivery and LGA but had a 5% increased risk in SGA (9.3% to 14.3%). Women who gained ~16 kg (~35 lbs) had higher odds of pre-eclampsia, fetal distress, failed induction, caesarean delivery and LGA but a lower risk of SGA. Cedergren[37] evaluated nearly 300,000 singletons from the Swedish Birth Registry and examined all BMI groups using a composite of many adverse maternal and fetal/neonatal outcomes to make weight gain recommendations. To minimize the composite adverse maternal and offspring outcomes, much lower weight gain recommendations were made in normal weight women of 2–10 kg (5–22 lbs), overweight women of 0–9 kg (<20 lbs) and obese women of 0–6 kg (<13 lbs). Potti *et al.*[44] applied the Cedergren and IOM recommendations to the New Jersey PRAMS database of 9125 subjects and concluded that while less macrosomia and caesarean deliveries would be expected applying the Cedergren recommendations, a slightly higher rate of preterm delivery, SGA and NICU admissions would result and that the recommended weight gain should be somewhere between the Cedergren and IOM recommendations.

Kiel *et al.*[38] evaluated the Missouri birth certificate data of ~120,000 offspring of obese mothers and determined where both LGA and SGA would be minimized according to the severity of obesity and also included pre-eclampsia and caesarean delivery as outcomes. Minimizing these risks led the authors to conclude that although for Class I obesity, ~6.8 kg (~15 lbs) was appropriate, the ideal weight gain for women with Class II obesity (35–39.9 kg) was only 0–4 kg (<9 lbs) and for women with a BMI $\geq$ 40 (Class III obesity), a weight *loss* of 0–4 kg (up to 9 lbs) was ideal. As noted previously, after the IOM guidelines were reported, the AHRQ published their systematic review in 2009 of the 35 highest quality studies and underscored that the data suggest that overweight and obese women who gained below the IOM recommendations do not have a higher risk of SGA and that the guidelines for these groups of women should be re-examined. Subsequently, Beyerlein *et al.*[39] examined 177,000 deliveries in Bavaria and sought to determine what GWG resulted in a joint predicted risk for both SGA and LGA of $\leq$20% and would also ensure that the SGA rate was $\leq$10%. The investigators determined that a wider range of weight gain would ensure this risk for underweight and normal weight women than what was recommended by the IOM. However, lower weight gain targets could be applied for overweight women of 0–12 kg (~0–26 lbs) and especially for obese women of −7 to 2 kg (−15 to 4 lbs), suggesting that a modest weight loss does not increase the risk of SGA. Oken[18] examined the relationship between GWG and five adverse outcomes including LGA, SGA, preterm delivery, postpartum weight retention and childhood obesity in ~2000 mother–child pairs in Project Viva and determined that the lowest predicted prevalence of the five adverse outcomes was associated with a weight gain of ~11.2 kg (~25 lbs) in normal weight women, a weight loss of 1.2 kg (~3 lbs) in overweight women and a weight loss of 7.6 kg (~17 lbs) in obese women. Hinkle *et al.*[41] similarly concluded from examining ~122,000 birth records from obese women reported in the Pregnancy Nutrition Surveillance System that Class I obese women should optimally gain 0.1–9 kg (0.2–20 lbs) but that optimal range for Classes II and III obese women was −4.9 to 4.9 kg (−11 to 11 lbs) to avoid both SGA and LGA.

Bodnar *et al.*[46] reviewed the available records from the Magee Obstetrical and Infant database born to women with Classes I, II and III obesity. Outcomes included LGA, SGA, spontaneous and indicated preterm births. Caucasian obese women who gained only ~50% of the IOM recommendations had a slightly higher adjusted odds ratio of SGA (1.1, 1.2 and 1.2 for Class I, II and III obesity, respectively) but a lower risk of LGA (0.9, 0.9 and 0.8, respectively). However, black women with obesity Class III who gained only ~50% of the IOM recommendations had a slightly *lower* risk of SGA and women who gained 300% of the IOM recommendations actually had a *higher* risk of SGA (OR = 1.4). Spontaneous preterm births were minimally affected by weight gain or weight loss in any of the categories although induced preterm births were significantly increased in women who gained 200–300% of the IOM recommendations. Limitations in the study included missing data which differed in some important characteristics among groups of women and no reporting of rates of pre-eclampsia, gestational diabetes, caesarean delivery or postpartum weight retention.

A recent population cohort study in Sweden[47] examined weight loss in women with Classes I, II or III obesity and found that women with Class II or III obesity who lost weight had a decreased or unaffected risk for caesarean delivery, pre-eclampsia, LGA, excessive postpartum bleeding, instrumental delivery, low Apgar scores and fetal distress. Although there was a small increase of SGA in those who lost weight (3.7%), it was only slightly higher than the overall prevalence of SGA births in Sweden (3.6%). The authors concluded that it may be reasonably safe for classes II and III women to lose a modest amount of weight due to the potential benefits at reducing caesarean delivery, pre-eclampsia and LGA at only a minimal expense of increasing SGA and with no evidence of other unfavourable fetal outcomes.

In an attempt to also examine longer term childhood outcomes with GWG, Margerison *et al.*[40] analysed the data in nearly 5000 children aged 14–22 from the 1979 National Longitudinal Survey of Youth. In order to minimize SGA, LGA, caesarean delivery, postpartum weight retention as well as childhood obesity, the investigators determined that GWG clearly increased LGA, postpartum weight retention and child overweight but that SGA only decreased with GWG in underweight and normal weight mothers. The investigators recommended an optimal GWG of ~5 kg for overweight mothers (11 lbs) but an optimal GWG of 0–5 kg in obese mothers (0–11 lbs). Lastly, in the nine-year follow-up of the body composition and biomarkers of offspring from ~3500 mothers in the Avon Longitudinal Study Parents/Children in the UK,[34,45] the investigators concluded that GWG above the 2009 IOM recommendations increased offspring BMI, fat mass, leptin, systolic blood pressure, CRP, Il-6 and decreased HDL. However, GWG less than the recommendations reduced offspring adiposity without the unfavourable biomarkers for metabolic syndrome.

WHY SO LITTLE CHANGE IN THE 2009 GUIDELINES?

Some of the data cited above which support less weight gain during pregnancy, especially in the overweight and obese groups, were published after the 2009 IOM guidelines. However, there were a number of studies supporting less weight gain, especially in the overweight and obese groups prior to the publication of the guidelines.[29,37,38,43] The

committee members published a commentary justifying the weight gain recommendations for the guidelines[5] citing that they felt strongly about trying to balance the risk of low versus high GWG and that SGA may be more important than LGA. They based their recommendations primarily on the basis of a primigravida, age 25–29, non-smoker, high social status and no exercise. They acknowledged that they did not consider the studies that used pre-eclampsia and GDM as adverse outcomes because they believed that the data to support these outcomes were weaker[8] and cited that most investigators did not measure weight gain before the diagnosis of GDM. There was not enough new evidence to change previous weight gain recommendations for adolescents, twins or women carrying higher order multiple fetuses.

The committee primarily considered the outcomes of SGA, LGA, unplanned caesarean delivery and excessive ($\geq$5 kg) postpartum weight retention. They also stated that since maternal BMI appeared to be the the most important risk factor for infant adiposity, that pre-pregnancy BMI and weight loss before pregnancy should be focused on the most. They also cited that the majority of women do not gain according to the guidelines, and that attempts to get women to gain according to the guidelines would be a major step in the right direction and represent a radical change. Lastly, they acknowledged that the committee evaluated data that demonstrated good outcomes among obese women, especially those with Class II or Class III obesity, who gained below the obligatory maternal tissue accretion and products of conception requirements (~7.5 kg). However, they cautioned that the data were limited, insufficient to support specific weight gain recommendations for women with a BMI $\geq$ 35 and that they had reservations about any weight loss recommendations due to concerns about possible ketonaemia, especially in women with glucose intolerance. The weight gain per week recommendations were simply constructed as linear interpolations between the approximately 1 and 2 kg weight gain that most women gain in the first trimester and the target total GWG for each BMI category divided by the number of weeks in the remaining two trimesters of pregnancy.

WHAT CAN BE DONE TO MINIMIZE EXCESSIVE WEIGHT GAIN AND DO INTERVENTIONS WORK?

There are clearly groups of women at highest risk of gaining excess weight in pregnancy who might be targeted. Although overweight and obese women do not tend to gain more weight than normal weight women, they often gain more than the guidelines recommended for their BMI class. Approximately 40% normal weight, 60% overweight and 25% of obese women gain more than the IOM recommendations.[13] Younger women, including adolescents, and those with lower education also tend to gain more weight. Although women who smoke or with short inter-pregnancy intervals gain less weight and have a higher risk of SGA, their infants have a higher percent fat mass compared with fat-free mass. There have been a number of randomized controlled trials[5,48–51] using diet and physical activity to minimize excess GWG. However, the results have been modestly effective and more likely to be positive in the studies which utilized the most intensive interventions, started earliest in pregnancy and targeted overweight women.[51]

Attempts to facilitate weight loss in the postpartum period may be slightly more effective and certainly breastfeeding should be encouraged and is associated with less postpartum weight retention and less childhood obesity.[52] However, barriers that should be considered during this period include sleep deprivation which increases ghrelin and decreases leptin,[53] time constraints, and postpartum depression.

CONCLUSIONS

Clearly stressing the benefits of weight reduction before pregnancy should be paramount in overweight and obese women who seek preconception counselling, given the increased maternal and neonatal morbidity and mortality associated with obesity in pregnancy, and the strength of pre-gravid BMI in predicting infant adiposity. Stressing the importance of increased physical activity and a healthy diet that is both low in simple carbohydrates and saturated fats is also critical, especially given the increasing data that maternal triglycerides are correlated with excess fetal fat accretion in humans.[54–56] Further, a high fat maternal diet in non-human primates results in lipid deposition in the liver, changes in appetite regulation, behaviour and mitochondrial oxidation in the offspring, predisposing them to obesity and insulin resistance.[57,58]

Most women (>50%) do not gain within the IOM guidelines, thus emphasizing the importance of avoiding excessive weight gain and its adverse effects on LGA, pre-eclampsia, GDM, caesarean delivery, postpartum weight retention and childhood obesity is an enormously important first step. However, this author concurs that the 2009 IOM weight gain recommendations for overweight and obese women may be too generous given consistent data that although GWG is related to birth weight and SGA in underweight and normal weight women, this same relationship does not hold for overweight and obese women. Given the recently cited data, the significant contribution of weight gain to postpartum weight retention, and the rising maternal and paediatric obesity epidemic, one could at least argue that normal weight and overweight women should strive to gain weight at the lower end of the IOM recommendations (~11 kgs or 25 lbs for normal weight women and 6.8 kg or 15 lbs for overweight women). Further, obese women with a BMI of 30–34.9 do not appear to be at risk of SGA without any significant weight gain and weight gain recommendations of no more than 5 kg (10–12 lbs) should be strongly considered. For women with a BMI of $\geq$35, there are increasing data that no weight gain is safe and it is possible that modest weight loss might be of benefit, although caution must be exerted in advocating this during the time of conception and pregnancy until carefully controlled studies are completed. In addition, most of the literature suffers from a lack of long-term metabolic outcomes in the offspring. It is hoped that the IOM will re-evaluate their weight gain recommendations, especially for overweight and obese women, and urgently advocate for the careful execution of prospective studies which investigate both the short- and long-term maternal and child outcomes associated with no weight gain, or even modest weight loss.

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