

The short- and long-term implications of maternal obesity on the mother and her offspring

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Obesity's increasing prevalence has reached epidemic proportions in the USA, with close to one-third of the adult population affected in 2000. Additionally, there is increasing prevalence of obesity in other industrialised areas of the world such as Europe. Of potentially more concern is the potential risks associated with obesity and related metabolic complications in the developing world. The maternal, fetal, peripartum and neonatal complications of obesity in pregnancy have far-reaching implications for both mother and offspring. Of alarming interest is the increasing rate of

obesity among adolescents and the cycle of obesity in future generations it portends. The purpose in this review is to briefly review the maternal perinatal morbidities associated with maternal pregravid obesity. Additionally, we will review evidence of both short- and long-term effect of maternal obesity on the *in utero* environment as it relates to fetal growth, neonatal body composition and adolescent obesity.

Keywords Body composition, maternal, neonatal, obesity, pregnancy.

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Introduction

Obesity is an epidemic not only in developed countries but also in the developing world. 'Indeed, it is now so common that it (obesity) is replacing the more traditional public healthcare concerns including under nutrition and infectious disease as one of the most significant contributors to ill health'.¹ In the USA, the prevalence of adult obesity, defined as a body mass index (BMI; wt/ht²) > 30, rose to an alarming 30.5% in 2000, compared with 22.9% from 1994 to 1998. The proportion of the population meeting the definition of overweight (BMI > 25) increased from 55.9 to 64.5% during the same period.² Potentially more important, the prevalence of obesity in adolescents in the USA has increased by 11.3% between 1994 and 2000.³ This increased prevalence has been especially notable among Mexican-American and non-Hispanic black adolescents.³

In our own inner city urban population, we have seen a significant increase in obesity at delivery from 1986–1996 through 1997–2001.⁴ The percent of women who weighed more than 200 lb increased from 21 to 28%. When analysed by delivery weights stratified by 50-lb increments, there were significant increases in each group (201–250 lb: 16–20%; 251–300 lb: 3.7–5.5% and >300 lb: 1.2–1.6%). The risk of

increasing obesity is disproportionate among the races, with the prevalence of obesity increasing most among African American women. The epidemic of obesity is not unique to the USA, as a significant increase in the incidence of obesity in women of reproductive age has been noted over the past decade in Europe, approaching that reported in the USA in some countries (Figure 1).⁵ In this review, unless otherwise noted, we will define obesity based on the World Health Organization¹ and National Institutes of Health criteria.⁶ Normal weight is defined as a BMI of 18.5–24.9, overweight as a BMI 25–29.9 and obesity as a BMI ≥ 30. Obesity can be further characterised by BMI as class I (30–34.9), class II (35–39.9) and class III (≥ 40).

Obstetric problems associated with maternal obesity

The complications of maternal obesity in pregnancy generally relate to pregravid weight rather than gestational weight gain or weight at delivery. Based on the Institute of Medicine guidelines, recommended weight gain in pregnancy for normal weight women is 25–35 lb, 15–25 lb for overweight women and 15 lb for obese women;⁷ however, these guidelines are currently under review. In our experience, average

weight women gain more weight during pregnancy as compared with women who are obese. In a small prospective longitudinal study, Ehrenberg *et al.* reported that lean subjects gained a greater percent fat as compared with obese subjects (3.3 versus 0.1%, $P = 0.004$) but gained similar amounts of fat mass (4.7 versus 4.2 kg, $P = 0.58$) and lean body mass (7.6 versus 8.8 kg, $P = 0.18$). Although during pregnancy there is generally an increased accretion of subcutaneous adipose in a central distribution (between the midthorax through the suprailiac and upper thigh), the accrual of fat mass in pregnancy in lean women is more peripheral (biceps and triceps area) as compared with obese women.⁸

The obese woman is at risk for a multitude of potential medical and obstetric problems during gestation, which may have adverse short-term effects on her fetus. There is an increased risk of miscarriage in obese women, whether they conceived after natural conception⁹ or assisted reproductive measures.¹⁰ Later in gestation, the obese woman is at significant risk for gestational hypertension, pre-eclampsia and gestational diabetes (GDM).¹¹ Based on a prospective multicentre study of 16 102 women, all initially evaluated at 10–14 weeks, 85% were controls (BMI < 30), 9% were obese (BMI 30–34.9) and 6% were morbidly obese (BMI ≥ 35). Obese women and morbidly obese women were 2.5 and 3.2 times (respectively) more likely to develop gestational hypertension than the control group. Similarly, pre-eclampsia was 1.6 and 3.3 times more likely to develop in obese and morbidly obese women, respectively. After adjusting for potential covariables using multivariable analysis, the adjusted odds ratio (OR) for the risk of GDM was 2.6 for obese and 4.0 for morbidly obese women, respectively. Because of these maternal obstetric problems, the morbidly obese women had a significant increased risk (OR 1.5, 95% CI 1.1–2.1) of preterm delivery, less than 37 weeks.

In addition to the increased risk of antenatal obstetric problems in obese women, there is an increased risk of caesarean delivery and associated morbidities.¹² Regional and general anaesthesia are both concerns in this population. There can be difficulty with placement of epidural or spinal anaesthesia in obese women, requiring multiple attempts. Additionally, general anaesthesia carries the risk of difficult intubation and the increased incidence of sleep apnoea postpartum. In the multicentre study of Weiss *et al.*,¹¹ the caesarean delivery rate for nulliparous women was 20.7% for women with a BMI of 29.9 or less, 33.8% for women with a BMI of 30–34.9 and 47.7% for women with a BMI of 35–39.9. Similar data were reported by Durnwald *et al.*¹³ in women attempting vaginal birth after caesarean delivery (VBAC). Of 510 women attempting a trial of labour, 337 (66%) were successful and 173 (34%) required repeat caesarean delivery. The greatest success rate for VBAC was in underweight (BMI < 19.8) women (84.7%) as compared with those in normal weight women ($P = 0.04$). Decreased VBAC success

was observed in obese women (54.6%) but not in overweight women (65.5%) compared with normal weight women (70.5%), $P = 0.003$ and $P = 0.36$, respectively. Additionally, normal weight women who gained weight between pregnancies to become overweight during their attempted VBAC had decreased success rates compared with those women whose BMI remained average (56.6 versus 74.2%, $P = 0.006$). Unfortunately, the converse was not true, in that weight loss resulting in a status change from overweight to average did not significantly improve their VBAC success (64.0 versus 58.4%, $P = 0.67$). The increased caesarean rate in overweight and obese women is also associated with an increase in post-operative complications such as wound infection/breakdown, excessive blood loss and postpartum endometritis. In summary, overweight and obese women are at risk of increased medical and obstetric problems in pregnancy, which then in turn increase her risk of preterm delivery, caesarean delivery and attendant operative morbidities.

Fetal risks associated with maternal obesity

Maternal obesity is a significant risk factor for neural tube defects in neonates of obese women.¹⁴ Although the risk of other congenital anomalies are less clear, the increased risk of neural tube defects in obese women appears to be independent of the risk of maternal diabetes and despite folic acid fortification in the flour in some countries.¹⁴ It is well-known among practitioners that ultrasound visualisation of fetal anatomy is more difficult in women carrying a predominance of centralised adipose tissue, and decreased sensitivity of ultrasound for cardiac anatomy has been previously documented.¹⁵ It is possible that this decreased sensitivity for evaluation of anatomy has contributed to a higher proportion of liveborn infants affected by malformations in this population.

In addition to the increased fetal mortality and morbidity associated with obstetric complications in the obese woman, fetal overgrowth is also a major concern, albeit much more subtle in the short term. Recent studies from both North America and Europe have reported an increase in mean birthweights, particularly those infants either greater than the 90th centile for gestational age (large for gestational age [LGA]) or macrosomic (birthweight > 4 kg).^{16,17} In Denmark, the percentage of macrosomic newborns increased from 16.7% in 1990 to 20.0% in 1999.¹⁸ Factors such as decreased maternal smoking, an increased incidence of diabetes and increasing maternal BMI have all been implicated. In our own population, we have observed a mean increase of 116 g in term singleton birthweight over the past 30 years. The increase in maternal weight at delivery was the factor most strongly correlated with the increase in birthweight.¹⁹ However, in order to better understand the relationship between maternal

obesity and fetal overgrowth, a review of factors relating to fetal growth are in order.

There are multiple factors associated with fetal growth. Maternal nutrition is of course an important factor. Based on experiences in pregnancies during the Dutch famine of 1944–1945, the period of gestation at which nutritional deprivation occurs is important.^{20,21} Nutritional deprivation in early pregnancy, followed by increased access to food in later pregnancy actually resulted in babies being heavier at birth as compared with babies born either before or after the famine. In contrast, if the famine occurred during late gestation, the babies weighed less and thinner at birth, with no change in length. Nutritional supplementation can improve birthweight. Based on the Guatemalan studies, the type of supplementation, i.e. protein or carbohydrate, may not make a difference in the increase in birthweight, assuming minimal protein requirements are achieved.²²

Maternal anthropometric variables are important factors relating to fetal growth. Maternal pregravid weight has a very strong correlation with birthweight.²³ Although maternal height is also associated with an increase in birthweight, when adjusted for weight, there was no longer a significant correlation between maternal height and birthweight.²⁴ Maternal weight gain during gestation is positively correlated with birthweight.²⁵ The correlation is stronger in nulliparous women ($r = 0.26$) compared with parous women ($r = 0.16$). The interaction of maternal pregravid weight and weight gain was examined by Abrams and Laros.²⁶ There was a progressively stronger correlation between maternal weight gain and birthweight in moderately overweight, ideal body weight and underweight women. In women with >135% of ideal weight for height before conception, there was no correlation between weight gain during pregnancy and birthweight. Last, maternal age and parity have independently been reported to have a positive correlation with birthweight. However, McKeown and Gibson²⁷ reported that when maternal age was adjusted for parity, there was no longer a significant correlation between maternal age and birthweight. Parity has been shown by Thompson *et al.*²⁸ to be associated with a mean 100- to 150-g increase in birthweight in subsequent pregnancies. However, the additional effect of parity on birthweight is diminished with increasing parity.

Relative to maternal factors, paternal anthropometric factors have limited impact on fetal growth. Morton reported that in half-siblings with the mother as the common parent, the correlation between birthweight and the half-siblings was $r = 0.58$. In contrast, the correlation of birthweight in half-siblings with the father as the common parent was only $r = 0.10$.²⁹ Animal cross-breeding studies support these findings. Walton and Hammond cross-bred Shetland ponies with Shire horses. The size of the foals was roughly the same as the foals of the maternal pure breed. Thus, maternal regulation was more important in determining intrauterine growth than

were paternal factors.³⁰ Klebanoff *et al.*,³¹ using a Danish population registry, reported that paternal birthweight, adult height and adult weight together explained approximately 3% of the variance in birthweight, compared with 9% for the corresponding maternal factors. In summary, maternal factors, most importantly maternal pregravid weight, has the strongest correlations with birthweight.

In our studies of fetal overgrowth and macrosomia, we have elected to concentrate on measures of body composition, i.e. fat and fat-free or lean body mass. The rationale for this approach stems from work performed in the previous century. As early as 1923, research by Moulton described that the variability in weight within mammalian species was explained by the amount of adipose tissue whereas the amount of lean body mass was relatively constant and changed in a consistent manner over time.³² In the human fetus, Sparks used autopsy data and chemical analysis in 169 stillborns and described a relatively comparable rate of accretion of lean body mass in small-for-gestational-age (SGA), average-for-gestational-age (AGA) and LGA fetuses but considerable variation in the accretion of fetal fat. Fat accretion in the SGA fetus was considerably less than in the AGA fetus, which in turn was less than that of the LGA fetus.³³ Last, the term human fetus at birth has the greatest percent body (approximately 12%) fat as compared with other mammals.³⁴ For these reasons, we have elected to assess fetal growth in our studies using estimates of body composition. The methodologies we have employed include anthropometric, stable isotope and total body electrical conductivity (TOBEC). These methods have been previously described.^{35–37}

The utility of using body composition in understanding fetal growth is exemplified by a previous study by our group evaluating the proportion of the variance in birthweight explained by body composition analysis of the fetus, and particularly fat and fat-free mass. The mean birthweight of the population was 3553 ± 462 g and the mean percent body fat was $13.7 \pm 4.2\%$. Fat-free mass, which accounted for ~86% of mean birthweight accounted for 83% of the variance in birthweight. In contrast, body fat which accounted for only ~14% of birthweight explained 46% of the variance in birthweight.³⁸

We have published a series of studies comparing the body composition analysis of infants of women with normal glucose tolerance (NGT) and GDM within 48 hours of birth (Table 1).³⁹ These studies used both TOBEC and anthropometric methodologies. Although there was no significant difference in birthweight or fat-free mass between the groups, there was a significant increase in fat mass and percent body fat in the infants of the GDM mothers. The body composition analyses were confirmed by the anthropometric/skinfold measures. These data were adjusted for potential confounding variables such as parity and gestational age without any significant change in results.

Table 1. Neonatal body composition (TOBEC) and anthropometrics in infants of women with GDM and NGT

	GDM (n = 195)	NGT (n = 220)	P value
Weight (g)	3398 ± 550	3337 ± 549	0.26
Fat-free mass (g)	2962 ± 405	2975 ± 408	0.74
Fat mass (g)	436 ± 206	362 ± 198	0.0002
Body fat (%)	12.4 ± 4.6	10.4 ± 4.6	0.0001
Tricep (mm)	4.7 ± 1.1	4.2 ± 1.0	0.0001
Subscapular (mm)	5.4 ± 1.4	4.6 ± 1.2	0.0001
Flank (mm)	4.2 ± 1.2	3.8 ± 1.0	0.0001
Thigh (mm)	6.0 ± 1.4	5.4 ± 1.5	0.0001
Abdomen (mm)	3.5 ± 0.9	3.0 ± 0.8	0.0001

We further analysed the data after stratification of the group into birthweight subsets, AGA³⁹ and LGA.⁴⁰ In Table 2, there are no significant differences in birthweights between the AGA infants of the GDM and NGT groups. However, there was again a significant increase in fat mass, percent body fat and skinfold measures in the infants of the mothers with GDM as compared with infants of women with NGT. Interestingly, the fat-free mass in the infants of the mothers with GDM was significantly less compared with the infants in the NGT group. The similar results were obtained when we limited the analysis to only LGA neonates⁴⁰ (Table 3). This relative increase in fat mass but not body weight may have obstetric implications such as the increased incidence of shoulder dystocia in GDM as compared with NGT neonates in similar birthweight categories. Based on these results, we conclude that birthweight alone may not be a sensitive enough measure to recognise subtle difference in fetal growth in certain populations such as infants of the mother with GDM.

In order to assess the relative strength of maternal metabolic factors both pregravid and in early and late gestation associated with body composition at birth, we evaluated 16

Table 2. Neonatal body composition (TOBEC) and anthropometrics in AGA infants of women with GDM and NGT

	GDM (n = 132)	NGT (n = 175)	P value
Weight (g)	3202 ± 357	3249 ± 372	0.27
Fat-free mass (g)	2832 ± 286	2919 ± 287	0.008
Fat mass (g)	371 ± 163	329 ± 150	0.02
Body fat (%)	11.4 ± 4.6	9.9 ± 4.0	0.002
Tricep (mm)	4.5 ± 0.9	4.1 ± 0.8	0.0002
Subscapular (mm)	5.1 ± 1.1	4.5 ± 1.0	0.0001
Flank (mm)	4.0 ± 1.2	3.7 ± 0.8	0.007
Thigh (mm)	5.7 ± 1.2	5.2 ± 1.3	0.002
Abdomen (mm)	3.3 ± 0.9	3.0 ± 0.8	0.002

Table 3. Neonatal body composition (TOBEC) in LGA infants of women with GDM and NGT

	GDM (n = 50)	NGT (n = 52)	P value
Weight (g)	4060 ± 380	4120 ± 351	0.13
Fat-free mass (g)	3400 ± 312	3564 ± 310	0.0009
Fat mass (g)	662 ± 163	563 ± 206	0.02
Body fat (%)	16.2 ± 3.3	13.5 ± 4.5	0.002

infants of lean/average weight woman with NGT ($n = 6$) and GDM ($n = 10$) in a prospective longitudinal fashion.⁴¹ We incorporated multiple measures of maternal metabolism, such as the euglycaemic clamp estimates of insulin sensitivity, as well as maternal and paternal anthropometric factors. The results of the stepwise multiple logistic regression analyses are shown in Table 4. There was a strong correlation between insulin sensitivity in late gestation and both birthweight and fat-free mass. This may reflect the significant decrease in maternal insulin sensitivity required in order to provide nutrients for fetal fat-free mass (amino acids) and energy needs (carbohydrates). Surprisingly, maternal pregravid insulin sensitivity had the strongest correlation with fat mass at birth. We speculate that the maternal pregravid metabolic milieu, characterised by decreased insulin sensitivity and increased beta cell response, may affect early placental functional development as it relates to lipid and cytokine gene expression, which in later pregnancy affect both maternal lipid metabolism and placental transport of nutrients. Evidence for both increased placental cytokine expression and lipid metabolism in gene array studies of placenta of macrosomic (obese) newborns of women with GDM have been published by our group.⁴²

In an effort to better understand the potential independent effect of maternal obesity on growth of infants of NGT and

Table 4. Stepwise regression analysis of factors correlated with neonatal body composition³⁹

	r²	Δr²
Birthweight		
Insulin sensitivity (late pregnancy)	0.28	—
Maternal weight gain	0.48	0.20
Fat-free mass		
Insulin sensitivity (late pregnancy)	0.33	—
Maternal weight gain	0.53	0.20
Fat mass		
Insulin sensitivity (pregravid)	0.15	—
Parity	0.29	0.14
Neonatal sex	0.39	0.10
Insulin sensitivity (late pregnancy)	0.46	0.07

GDM mothers, we performed a stepwise logistic regression analysis on the 220 infants of mothers with NGT and 195 term infants of mothers with GDM previously described.³⁹ The results are given in Table 5. Not surprisingly, gestational age at term was the independent variable with the strongest correlation with both birthweight and fat-free mass. Maternal smoking had a negative correlation with both birthweight and fat-free mass and paternal weight had a weak correlation with only fat-free mass. In contrast, maternal pregravid BMI had the strongest correlation with fat mass ($r^2 = 0.066$) and percent body fat ($r^2 = 0.072$), therefore explaining approximately 7% of the variance in both fat mass and percent body fat. Although approximately 50% of the subjects had GDM, only 2% of the variance ($r^2 = 0.016$) in fat mass in this population was explained by a mother having GDM. Furthermore, Ehrenberg *et al.*,⁴³ from our institution, reported that the risk of having a LGA neonate was greatest for women with a history of diabetes (OR 4.4) when compared with maternal obesity (OR 1.6). However, there was four-fold greater number of LGA babies born of obese women than women with diabetes because the relative prevalence of overweight/obesity

Table 5. Stepwise regression analysis of factors relating to fetal growth and body composition in infants of women with GDM ($n = 195$) and NGT ($n = 220$)

	r^2	Δr^2	P value
Birthweight			
EGA	0.114	—	
Pregravid weight	0.162	0.048	
Weight gain	0.210	0.048	
Smoking (—)	0.227	0.017	
Parity	0.239	0.012	0.0001
Lean body mass			
EGA	0.122	—	
Smoking (—)	0.153	0.031	
Pregravid weight	0.179	0.026	
Weight gain	0.212	0.033	
Parity	0.225	0.013	
Maternal height	0.241	0.016	
Paternal weight	0.250	0.009	0.0001
Fat mass			
Pregravid BMI	0.066	—	
EGA	0.136	0.070	
Weight gain	0.171	0.035	
Group (GDM)	0.187	0.016	0.0001
%Body fat			
Pregravid BMI	0.072	—	
EGA	0.116	0.044	
Weight gain	0.147	0.031	
Group (GDM)	0.166	0.019	0.0001

EGA, estimated gestational age.

Pregravid maternal obesity has the strongest correlation with neonatal measures of fat mass/%body fat in contrast to lean body mass.³⁹

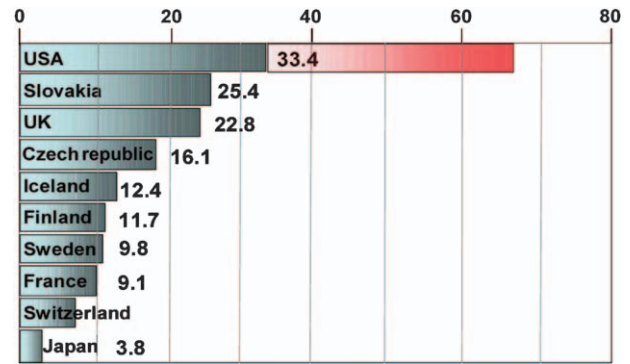


Figure 1. The percentage of women with a BMI > 30 in the USA and other countries. The data from the USA includes %obese (solid) and %overweight (shaded).

to diabetes was 47 and 5%, respectively. Therefore, at least in our population, maternal obesity and not diabetes appears to be the more important factor contributing to the population's increase in mean birthweight.

In a continuing effort to understand the relationship between maternal pregravid factors and fetal adiposity, we compared the longitudinal changes in maternal insulin sensitivity using the hyperinsulinemic–euglycaemic clamp among women with a pregravid BMI < 25, 25–30 and > 30 (Figure 2). Although there was a 50–60% decrease in insulin sensitivity in both groups from before conception through late pregnancy ($P = 0.0001$), the obese subjects were significantly less insulin sensitive, or more insulin resistant, than the lean women ($P = 0.0001$) and overweight women ($P = 0.004$), particularly pregravid and in early gestation. We propose that the increase in birthweight and fat mass in infants of obese women when compared with average weight women is related to maternal metabolic alterations in early pregnancy affecting fetoplacental growth and metabolism in the *in utero* environment.

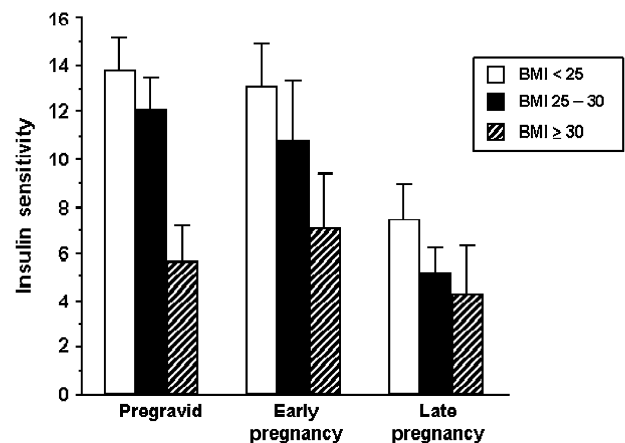


Figure 2. The longitudinal changes in insulin sensitivity in lean overweight and obese women, before conception (pregravid) and in early (12–14 weeks) and late (34–36 weeks) gestation.

Long-term risks for the fetus of the obese mother

Although much has been written about the increased risk of the metabolic syndrome (obesity, hypertension, insulin resistance and dyslipidaemia) in infants born as SGA, recent evidence points towards an increase in adolescent and adult obesity in infants born either as LGA or macrosomic.⁴⁴ There is abundant evidence linking higher birthweights to increased obesity in adolescents as well as adults for at least 25 years.^{45,46} Large cohort studies such as the Nurses Health Study⁴⁷ and the Health Professional Follow-up Study⁴⁸ report a J-shaped curve, i.e. a slightly greater BMI among subjects born small but a much greater prevalence of overweight and obesity in those born large.⁴⁹ The increased prevalence of adolescent obesity is related to an increased risk of the metabolic syndrome. The increased incidence of obesity accounts for much of the 33% increase in type II diabetes particularly among the young. Fifty to 90% of adolescents with type II diabetes have a BMI > 27⁵⁰ and 25% of obese children of 4–10 years of age have impaired glucose tolerance.⁵¹ Hence, the epidemic of obesity and subsequent risk of diabetes and components of the metabolic syndrome may begin *in utero* with fetal overgrowth/adiposity rather than undergrowth.

A recent retrospective cohort study by Whitaker⁵² in over 8400 children in the USA in the early 1990s reported that children who were born to obese mothers (based on BMI in the first trimester) were twice as likely to be obese by 2 years of age. If a woman had a BMI ≥ 30 in the first trimester, the prevalence of childhood obesity (BMI > 95th percentile based on Center for Disease Control criteria) at ages 2, 3 and 4 years was 15.1, 20.6 and 24.1%, respectively. This was between 2.4 and 2.7 times the prevalence of obesity observed in children of mothers whose BMI was in the normal range (18.5–24.9). This effect was only slightly modified by birthweight.

There is an independent effect of maternal pregravid weight and diabetes not only on birthweight but also on the adolescent risk of obesity. Langer *et al.*⁵³ reported that in obese women with GDM, whose glucose was well controlled on diet alone, the odds of fetal macrosomia (birthweight > 4000 g) was significantly increased (OR 2.12) compared with those in women having a well-controlled (diet only) GDM with normal BMI. Similar results were reported in women with GDM, which was poorly controlled on diet or insulin. In well-controlled, insulin-requiring GDM, there was no significantly increased risk of macrosomia with increasing pregravid BMI. Additionally, Dabelea *et al.*⁵⁴ also reported that the mean adolescent BMI was 2.6 kg/m² greater in sibling offspring of diabetic pregnancies compared with the index siblings born when the mother had previously had NGT. Hence, both maternal pregravid weight and the presence of maternal

diabetes may independently affect the risk of adolescent obesity in the offspring.

This risk of developing the metabolic syndrome in adolescents was addressed by Boney *et al.*⁵⁵ in a longitudinal cohort study of AGA and LGA infants of women with NGT and GDM. The metabolic syndrome was defined as the presence of two or more of the following components: obesity, hypertension, glucose intolerance and dyslipidaemia. Maternal obesity was defined as a pregravid BMI > 27.3. Children who were LGA at birth had an increased hazard ratio for metabolic syndrome (2.19 [95% CI: 1.25–3.82; *P* = 0.01]) by 11 years of age, as did children of obese women (1.81 [95% CI 1.03–3.19, *P* = 0.04]). The presence of maternal GDM was not independently significant, but the risk of the development of metabolic syndrome was significantly different between LGA and AGA offspring of mothers with GDM by age 11 (relative risk = 3.6).

Summary and opinion

There has been a significant increase in the prevalence of obesity in the past two decades in developed countries. This increased prevalence of obesity is not confined to adults and is beginning to affect our youth, having been reported with increasing frequency in adolescents and children as young as 2 years of age. Although there is significant morbidity and mortality associated with obesity in and of itself, there are additional risks associated with the metabolic syndrome, which is a component of and may in itself be a consequence of obesity.

Although lifestyle measures of diet and exercise have been useful in the treatment of obesity, they often fail when subjects revert with increased weight once intensive management concludes. Bariatric surgery has been employed with increased frequency in developed countries but is expensive, associated with potential surgical complications, and lacking in significant long-term outcome data.

Therefore, prevention rather than treatment has gained newfound interest, particularly with the increasing incidence of obesity and risk of attendant complications in developing countries. Based on this review, there appears to be an opportunity to potentially break the cycle of obesity during pregnancy. Having obese women lose weight and achieve a normal BMI prior to conception would be the ideal goal, but realistically quite difficult to achieve, given the known problems of achieving optimal glucose control in women planning to become pregnant. What other options exist? Given the expense and potential long-term risks of pharmacological therapy, more than likely lifestyle interventions offer the only viable alternative. Treatment modalities such as minimisation of weight gained during pregnancy, combined with the institution of healthy eating and exercise regimens, may represent an ideal option for these interventions. Until we

attain a better understanding of the underlying genetic predispositions, physiology and mechanisms relating to maternal and fetoplacental interactions relating to fetal growth and development, all treatments must by necessity be empiric. However, we need to build upon the information we currently possess because waiting may not be an option for much longer.

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