The Impact of Maternal Obesity on Maternal and Fetal Health

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The increasing rate of maternal obesity provides a major challenge to obstetric practice. Maternal obesity can result in negative outcomes for both women and fetuses. The maternal risks during pregnancy include gestational diabetes and preeclampsia. The fetus is at risk for stillbirth and congenital anomalies. Obesity in pregnancy can also affect health later in life for both mother and child. For women, these risks include heart disease and hypertension. Children have a risk of future obesity and heart disease. Women and their offspring are at increased risk for diabetes. Obstetrician-gynecologists are well positioned to prevent and treat this epidemic.
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The worldwide prevalence of obesity has increased substantially over the past few decades. Economic, technologic, and lifestyle changes have created an abundance of cheap, high-calorie food coupled with decreased required physical activity. We are eating more and moving less. There is evidence for metabolic dysregulation among obese individuals that has been linked with a number of possible environmental factors, including contaminants from modern industry. Obesity is a significant public health concern and is likely to remain so

for the foreseeable future. Maternal obesity increases the risk of a number of pregnancy complications, including preeclampsia, gestational diabetes mellitus (GDM), and cesarean delivery (Table 1).[1] Excessive weight gain during pregnancy and postpartum retention of pregnancy weight gain are significant risk factors for later obesity in women.[2] Additionally, maternal health can have a significant impact on the in utero environment and, thus, on fetal development and the health of the child later in life (Table 1).[3]

According to the in utero fetal programming hypothesis (Barker hypothesis), size at birth is related to the risk of developing disease later in life.[4] Although the Barker hypothesis originally focused on low birth weight, there is evidence that high birth weight may have its own set of complications later in life. A link between maternal obesity in the first trimester and obesity in children has been demonstrated. Whitaker[5] found that the relative risk of childhood obesity associated with maternal obesity in the first trimester of pregnancy was 2.0 (95% confidence interval [CI], 1.7-2.3) at 2 years of age, 2.3 (95% CI, 2.0-2.6) at 3 years of age, and 2.3 (95% CI, 2.0-2.6) at 4 years of age. Birth weight has also been shown to be directly correlated with body mass index (BMI) later in life.[6]

One mechanism thought to underlie these relationships is in utero fetal programming by nutritional stimuli. Fetuses have to adapt to the supply of nutrients crossing the placenta whether a deficit or an overabundance, and these adaptations may permanently change their physiology and metabolism.[3] These programmed changes may serve as the origins of a diverse array of diseases that arise later in life, including heart disease, hypertension, and non–insulin-dependent diabetes (Figure 1). Moreover, because of fetal programming, obesity may become a self-perpetuating problem. Daughters of obese women may themselves be vulnerable to becoming obese and more likely to have offspring who share this vulnerability.

Table 1
Obstetric Complications in Obese Pregnant Women

Complication	OR (95% CI) or % vs Normal Weight	*P*
Early pregnancy		
Spontaneous abortion (miscarriage)		
After spontaneous conception	1.2 (1.1-1.5)	.04
After IVF conception	1.8 (1.1-3.0)	< .05
Recurrent miscarriage	3.5 (1.1-21.0)	.04
Congenital anomalies		
Neural tube defects	1.8 (1.1-3.0)	< .05
Spina bifida	2.6 (1.5-4.5)	< .05
Congenital heart disease	1.2 (1.1-1.3)	< .05
Omphalocele	3.3 (1.0-10.3)	< .05
Late pregnancy		
Hypertensive disorder of pregnancy		
Gestational nonproteinuric hypertension	2.5 (2.1-3.0)	< .0001
Preeclampsia	3.2 (1.8-5.8)	.007
Gestational diabetes mellitus	2.6 (2.1-3.4)	< .001
Preterm birth	1.5 (1.1-2.1)	< .05
Intrauterine fetal demise (stillbirth)	2.8 (1.9-4.7)	< .001
Peripartum		
Cesarean delivery	47.7% vs 20.7%	< .01
Decreased VBAC success	84.7% vs 66%	.04
Operative morbidity	33.8% vs 20.7%	< .05
Anesthesia complications		
Excessive blood loss		
Postpartum endometritis		
Wound infection/breakdown		
Postpartum thrombophlebitis		
Fetal/neonatal complications		
Fetal macrosomia (EFW ≥ 4500 g)	2.2 (1.6-3.1)	< .001
Shoulder dystocia	3.6 (2.1-6.3)	< .001
Birth weight > 4000 g	1.7 (1.4-2.0)	.0006
Birth weight > 4500 g	2.0 (1.4-3.0)	< .0001
Childhood obesity	2.3 (2.0-2.6)	< .05

95% CI, 95% confidence interval; EFW, estimated fetal weight; IVF, in vitro fertilization; OR, odds ratio; VBAC, vaginal birth after cesarean.

Definitions of Obesity

The most commonly used measurement for defining obesity is BMI, which refers to an individual's weight in kilograms divided by the square of his or her height in meters. Individuals are deemed *overweight* when they have a BMI between 25 and 30 kg/m^2; *obesity* is defined as a BMI greater than or equal to 30 kg/m^2, and *extreme obesity* is defined as a BMI greater or equal to 40 kg/m^2. It is important to note, however, that BMI can be misleading. For example,

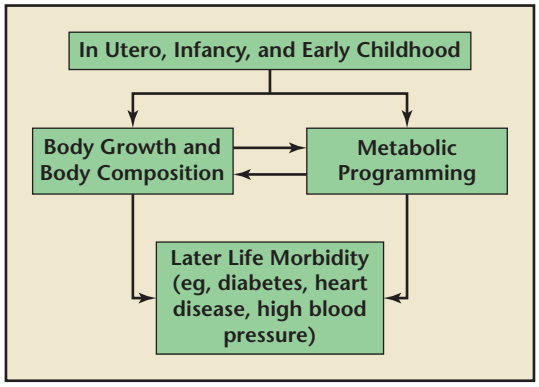

Figure 1. *The impact of malnutrition during early development.*

weight lifters and professional athletes tend to have high BMI because they have a high muscle mass, not excess fat. These individuals are not at risk for metabolic health problems because the health consequences of obesity come from excess adipose tissue, not the size of one's body. Despite this limitation, BMI continues to be used today because it is easily calculated and is the best tool available from a broad-based health policy perspective.[7]

Biology of Adipose Tissue

Fat (lipid) is an essential tissue and performs multiple and diverse functions, including providing nutritional, hormonal, and even structural support. The main fat depots in the body are in adipose tissue. Adipocytes are cells specifically adapted for fat storage, serve as a future energy source, and help to avoid the negative metabolic consequences of excess cellular lipid deposits in organs such as muscle, liver, and heart. However, adipose tissue is not a passive organ. It actively regulates metabolism through multiple distinct but overlapping pathways. Adipose tissue also contains a large number of nonfat cells, including fibroblasts and immune cells such as mast cells, macrophages, and leukocytes.[8] Both adipocytes and nonfat cells synthesize and secrete numerous peptide and steroid hormones as well as cytokines and chemokines, and such factors are known to influence local and systemic physiology (Table 2).[8,9] In this way, adipose tissue functions as an endocrine organ,[9] and it is the metabolic function of adipose tissue that causes much of the pathology associated with obesity.

Adipose tissue functions as an endocrine organ in a number of ways. It stores and releases preformed steroid hormones, converts precursors to biologically active hormones, and converts active hormones to inactive metabolites. To this end, adipocytes express a number of enzymes critical

Table 2
Enzymes and Hormones Produced by Adipose Tissue

Enzyme/Hormone	Function	Changes Associated With Obesity
Aromatase	Converts androgens to estrogens	No change with obesity, but increased fat mass results in greater total conversion
17β-hydroxysteroid hydrogenase	Converts estrone to estradiol and androstendione to testosterone	No change
5α-reductase	Inactivates cortisol	No change
11β-hydroxysteroid dehydrogenase type 1	Converts cortisone to cortisol	Activity is increased in obese women
Leptin	Affects food intake, timing of puberty, bone development, and immune function	Circulating leptin levels are increased in obese women
Tumor necrosis factor α (TNFα)	Represses genes involved in the uptake and storage of nonesterfied fatty acids and glucose	Expression of TNFα is increased in the adipose tissue of obese women
Adiponectin	Enhances insulin action	Circulating levels of adiponectin are decreased in obese women

to steroid hormone biosynthesis and metabolism (Table 2). For example, estrone is converted to estradiol in peripheral adipose tissue. Indeed, most if not all circulating estradiol in postmenopausal women comes directly from adipose tissue.[9] Adipose tissue expresses 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1), which converts cortisone to cortisol, as well as 5α-reductase, which converts cortisol to 5α-tetrahydrocortisol. Thus, adipose tissue regulates the local concentration of glucocorticoids and contributes to their metabolic clearance. Finally, adipose tissue secretes a large number of bioactive peptides and cytokines, collectively known as adipokines (Table 2).

Fat in our diet and on our bodies is beneficial as long as it exists in moderation. Too much fat becomes maladaptive, and normal physiology pushed beyond adaptive function becomes pathology, a concept referred to as *allostatic overload*. In the setting of obesity, pathology develops because of an increase in adipose tissue beyond the tolerable functional range. In this way, the metabolic consequences of obesity are analogous to the endocrine dysfunction seen in hyperplasia of any endocrine organ. Consider for a moment the metabolic and health consequences if a person's liver, thyroid, or adrenal gland doubled in size.

Source of Data on Obesity

The primary source of national data on obesity and overweight in the United States is the National Health and Nutrition Examination Survey (NHANES), which includes both an extensive take-home questionnaire and a physical examination in a mobile examination center (http://www.cdc.gov/nchs/about/major/nhanes/hlthprofess.htm). A key feature of NHANES is that it allows for standardized measurements of height and weight, and, thus, an accurate calculation of BMI. Another source of obesity data is the Pregnancy Risk Assessment Monitoring System (PRAMS), an ongoing population-based surveillance system that examines trends in prepregnancy obesity by maternal demographic and behavioral characteristics. PRAMS collects self-reported data from maternal questionnaires on behaviors associated with pregnancy (http://www.cdc.gov/prams). The National Vital Statistics System (NVSS) (http://www.cdc.gov/nchs/nvss.htm) contains data on all births in the United States as reported on state birth certificates and is an easy way to collect an abundance of data. Unfortunately, all of these data sources have their limitations. For example, PRAMS only includes 9 states (which represent 18.5%, or 1 in 5, of all live births in the United States[10]) and tracks trends in obesity only over a 10-year period. Similarly, national birth certificate data collected by the NVSS includes maternal weight but not height, and so BMI cannot be calculated.

Patterns of Maternal Obesity

Data from PRAMS has shown that the prevalence of prepregnancy obesity increased by 69% over a 10-year period, from 13% in 1993-1994 to 22% in 2002-2003.[10] In this report, maternal obesity increased across all categories of age; race; education; smoking status; Special Supplemental Nutrition Program for Women, Infants, and Children enrollment; and parity.[10]

The Institute of Medicine (IOM) and the National Heart, Lung, and Blood Institute of the National Institutes of Health established guidelines for healthy ranges of weight gain in pregnancy (Table 3). PRAMS data showed that only 1 out of 3 women had weight gain consistent with the recommendations of the IOM.[11] Racial and ethnic factors clearly affect weight gain during pregnancy. According to Brawarsky and colleagues,[12] African American women are more likely to be overweight prior to pregnancy and were most likely to gain weight in excess of the IOM guidelines, white females were most likely to report target weight gain, Hispanic women were least likely to report target gains, and Asian women were more likely to gain less than the recommend weight.

The postpartum period may be a critical time for long-term weight gain and the development of maternal obesity. Excess weight gain during pregnancy and persistent weight retention 1 year postpartum are strong predictors of overweight a decade or more later.[2] According to the National Maternal and Infant Health Survey, more than 30% of women retained 14 lb or more when compared with their recall of their prepregnancy weight, with African American women

Table 3
Recommendations for Weight Gain in Pregnancy

Body Mass Index	Recommended Weight Gain
18.5-24.9 kg/m^2 (normal weight)	25-35 lb (11.2-15.9 kg)
25-29.9 kg/m^2 (overweight)	15-25 lb (6.8-11.2 kg)
$>$ 30 kg/m^2 (obese)	15 lb (6.8 kg)

reporting a larger weight gain during pregnancy and less weight loss postpartum.[13] A more recent study showed that 12% of women retained at least 11 lb 1 year postpartum.[14] These women were more likely to have gained excessive weight during pregnancy and to be younger, heavier prior to pregnancy, nonwhite, unmarried, primiparous, and of lower socioeconomic status. For multiparous women, weight retention from previous pregnancies and the quality of health care received between pregnancies appear to be important determinants of subsequent prepregnancy weight.[14] Some authorities have suggested that more intensive postpartum care in women who are overweight or obese (such as graded exercise and weight loss programs) may be able to significantly impact subsequent pregnancy outcome, but this remains to be definitively demonstrated. Importantly, in a large epidemiologic study in Sweden, an increase in interpregnancy BMI (by at least 3 kg/m^2) was associated with a higher risk of adverse pregnancy outcomes.[15]

Effect of Obesity on Maternal Complications in Pregnancy

Maternal obesity increases the risk of a number of pregnancy complications (Table 1) and, as such, requires adjustment to routine prenatal care (summarized in Table 4). Maternal obesity is a risk factor for spontaneous abortion (for both spontaneous conceptions and conceptions achieved through assisted reproductive technology), as well as for unexplained stillbirth (intrauterine fetal demise). A recent meta-analysis of 9 studies revealed that obese pregnant women

Table 4
Adjustments to Routine Prenatal Care in Obese Pregnant Women

Risk Factor	Recommended Care
Increased risk of neural tube defect	• Preconception folic acid supplementation (4 mg daily ideally 3 months prior to pregnancy through the first trimester) • Maternal serum AFP (15-20 weeks) • Detailed fetal anatomy survey (18-20 weeks)
Increased risk of hypertensive disorders of pregnancy, including preeclampsia	• Baseline 24-hour urinalysis in second trimester • Baseline liver and renal function tests in second trimester • Blood pressure and urine dip for protein at each prenatal visit • There is no effective way to prevent preeclampsia
Increased risk of gestational diabetes (GDM)	• Consider early screening with 1-hour nonfasting 50-g glucose load test (GLT) at 16-20 weeks. If positive, check a definitive 3-hour 100-g glucose tolerance test (GTT) to confirm the diagnosis of GDM. If negative, repeated GLT at the usual gestational age of 24-28 weeks
Increased risk of unexplained stillbirth	• Consider weekly antepartum fetal testing with NST and/or BPP beginning at 36 weeks, especially in women with a BMI $\geq$ 40 kg/m^2 (although this has not been shown to definitively improve perinatal outcome)
Increased risk of anesthesia complications	• ACOG recommends a prelabor or early intrapartum anesthesia consultation for all women with a BMI $\geq$ 40 kg/m^2 • Consider early epidural placement in labor • Recheck epidural placement if the patient is transferred to the operative room for cesarean delivery because of increased risk of migration of the epidural catheter
Failure to lose weight after delivery is associated with subsequent adverse maternal health problems, including complications of future pregnancies	• Continue nutrition counseling and exercise program after delivery • Consider consulting a weight loss specialist to optimize postpartum weight loss before attempting another pregnancy • If complicated by GDM, check 2-hour 75-g GTT at or after 6-week postpartum visit

ACOG, The American College of Obstetricians and Gynecologists; AFP, α-fetoprotein; BMI, body mass index; BPP, biophysical profile; NST, non-stress test.

have an estimated risk of stillbirth that is twice that of normal weight pregnant women.[16] Several mechanisms have been proposed for this relationship, including the increased risks of hypertensive disorders and gestational diabetes that are associated with maternal obesity during pregnancy.

Maternal obesity is associated with an increased risk of hypertensive disorders of pregnancy, including preeclampsia (gestational proteinuric hypertension), with an odds ratio (OR) of between 2 and 3.[17] The risk increases linearly as BMI increases. For each increase in BMI of 5 to 7 kg/m^2, there is a corresponding 2-fold increase in the risk of developing preeclampsia.[18]

Obese women are at increased risk of complications at the time of labor and delivery. The rate of successful vaginal delivery decreases progressively as maternal BMI increases. A meta-analysis of 33 studies showed that the ORs of cesarean delivery were 1.46 (95% CI, 1.34-1.60), 2.05 (95% CI, 1.86-2.27), and 2.89 (95% CI, 2.28-3.79) among overweight, obese, and severely obese women, respectively, compared with normal weight pregnant women.[19] According to Ehrenberg and coworkers, the cesarean delivery rate for women weighing less than 200 lb was 18%, versus 39.6% in women who were classified as extremely obese.[20] This 2- to 3-fold increase in cesarean delivery rate is true for both primigravid and multigravid women.[1] Whether this is secondary to increased fetal size or another maternal characteristic is not known.

Maternal obesity also influences the success rate of attempted vaginal birth after cesarean (VBAC). Carroll and colleagues[21] found that women weighing less than 200 lb had a VBAC success rate of 81.8% compared with 57.1% for women weighing 200 to 300 lb and 13.3% for women heavier than 300 lb. A similar relationship was observed in a subsequent study using BMI rather than absolute maternal weight, with VBAC success rates ranging from 84.7% in women with a BMI lower than 19.8 kg/m^2 to 54.6% in those with a BMI higher than 30 kg/m^2.[22]

In addition to an increased rate of operative delivery, obese women are also at increased risk of intraoperative complications, including increased infectious morbidity and thromboembolic events (Table 1). There is also an increased risk of anesthetic complications, such as failed intubation at the time of general endotracheal anesthesia.[23] A number of specific recommendations have been proposed to minimize intraoperative complications in obese pregnant women (summarized in Table 5).

The reason obese pregnant women are more likely to end up with a cesarean delivery is not known, but a theory is that obese women are more likely to experience dysfunctional

Table 5
Recommendations Before, During, and After Surgery in Obese Pregnant Women

- Consider preoperative **cardiac evaluation,** especially if the patient has diabetes or chronic hypertension. This should include a baseline electrocardiogram and, if abnormal, an echocardiogram and cardiology consultation.
- Give preoperative **broad-spectrum antibiotics** 20-30 minutes before the skin incision to reduce the risk of postpartum endometritis and wound infection.
- Consider using a **large operating table** (especially if the patient is > 300 lb) and having additional personnel in the delivery room.
- Because of the increased risk of intrapartum blood loss, consider having **additional blood products** available in the operating room.
- If indicated, **tape the pannus** out of the surgical field to facilitate visualization and avoid a through-and-through skin incision.
- **Close the subcutaneous layer**. There is extensive evidence that seroma formation and postoperative wound disruption can be decreased in obese women (defined as adipose layer > 2 cm) if the subcutaneous tissues are closed using layers of running sutures.
- **Avoid subcuticular skin closure** to allow serous fluids from the subcutaneous fat to drain out of the incision rather than accumulate in the subcutaneous layer.
- Place **pneumatic compression stockings** on the lower extremities of all obese parturients prior to and during surgery as prophylaxis against deep vein thrombosis (DVT).
- The compression stockings should remain in place until the patient is fully ambulatory. Additional prophylaxis against DVT with prophylactic low-molecular-weight heparin should be considered in women with a body mass index ≥ 40 kg/m^2.
- Begin **early ambulation** to prevent DVT formation.
- Consider **delaying removal of staples or sutures** for a full week to allow the skin to heal completely.

labor. For example, Vahratian and colleagues[24] found that the rate of cervical dilation in nulliparous women in spontaneous labor decreased as maternal BMI increased. In this study, normal weight women (BMI 19.8-26.0 kg/m^2) took a median duration of 5.43 hours to dilate from 4 to 10 cm, whereas obese women (BMI > 29.0 kg/m^2) took 6.98 hours. This appears to be true also in women undergoing induction of labor at term. Nuthalapaty and colleagues[25] demonstrated that, although multiparous women progressed faster during induced labor than nulliparous women, in both groups an increase in maternal weight quartile was associated with a decreased rate of cervical dilation and an increase in the duration of labor. Denison and colleagues[26] showed that a higher maternal BMI in the first trimester and a greater increase in BMI throughout pregnancy were associated with a reduced likelihood of spontaneous labor at term, an increased risk of post-term pregnancy, and an increased rate of intrapartum complications.

Effect of Maternal Obesity on Perinatal Outcome

Maternal obesity is associated with abnormal fetal growth. Women who are heavier are less likely to have a pregnancy complicated by a small-for-gestational age infant or intrauterine growth restriction, but this protective effect appears to dissipate once the maternal BMI reaches the level of obesity (> 30 kg/m^2). The major concern in obese pregnant women is fetal macrosomia (defined as an estimated fetal weight of greater than or equal to 4500 g), which appears to be increased 2- to 3-fold in obese parturients.[27] Moreover, there appears to be a dose-dependent relationship between maternal obesity and fetal macrosomia. In a recent meta-analysis, the prevalence rates of fetal macrosomia were 13.3% and 14.6% for obese and morbidly obese women, respectively, compared with 8.3% for the normal weight control group.[16] In the United States, the mean birth weight between 1985 and 1998 increased from 3423 to 3431 g among whites and from 3217 to 3244 g among blacks.[28] In Canada during the same time period, the mean birth weight increased from 3391 to 3427.[28] In Denmark, the mean birth weight between 1990 and 1999 increased from 3474 g to 3519 g (an increase of 45 g) and macrosomia rates increased from 16.7% to 20%.[29] During a similar time period (1992-2001) in Sweden, there was a 3% increase in the incidence of large-for-gestational-age newborns (defined as birth weight > 2 standard deviations from the mean for a given gestational age).[30] Although a number of factors may explain this global increase in the prevalence of fetal macrosomia, the prevailing data suggest that maternal obesity is the main factor, followed by maternal diabetes status.[27]

Fetal macrosomia in obese women is associated not only with an increase in the absolute size of the fetus, but also in a change in body composition.[31,32] Sewell and coworkers[31] found that the average fat mass of infants born to mothers with a normal BMI (< 25 kg/m^2) was 334 g, giving a body fat composition of 9.7%. The offspring of women with a BMI > 25 kg/m^2, on the other hand, had a mean fat mass of 416 g, or a body fat composition of 11.6%. Of note, the majority of this effect appears to be a result of weight gain during pregnancy. Indeed, prepregnancy BMI appears to account for only 6.6% of the observed variation in infantile fat mass and only 7.2% of body fat composition.[33]

Maternal obesity is associated also with an increased risk of neural tube defect (NTD) in the offspring, even after controlling for ethnicity, maternal age, education, and socioeconomic status.[34-36] Watkins and coworkers[35] concluded that a 1 kg/m^2 increase in BMI is associated with a 7% increased risk of having an infant with NTD. A recent meta-analysis by Rasmussen and colleagues[36] reported that the OR for delivering an infant with NTD was 1.22 (95% CI, 0.99-1.49), 1.70 (95% CI, 1.34-2.15), and 3.11 (95% CI, 1.75-5.46) among overweight, obese, and morbidly obese women, respectively, compared with normal weight women. The mechanism underlying the increased risk of NTD in pregnancies complicated by maternal obesity is unknown. However, a number of theories have been proposed, including a reduction in the amount of folic acid reaching the developing embryo due to insufficient absorption and greater maternal metabolic demands, chronic hypoxia, and increased circulating levels of triglycerides, uric acid, estrogen, and insulin (due, in part, to increased insulin resistance).[34,35]

Maternal Obesity and Diabetes

Maternal obesity is associated with an increased risk of diabetes, both pregestational diabetes and GDM.[37,38] Compared with normal weight women (BMI < 25 kg/m^2), a recent meta-analysis of 20 studies demonstrated that the OR of developing GDM was 2.14 (95% CI, 1.82-2.53), 3.56 (95% CI, 3.05-4.21), and 8.56 (95% CI, 5.07-16.04) among overweight (BMI 25-30 kg/m^2), obese (BMI > 30 kg/m^2), and severely obese women (BMI > 40 kg/m^2), respectively.[38] A recent study found that weight gain in the 5 years prior to becoming pregnant, even at a rate of 1.1 to 2.2 kg per year, increases the risk of developing GDM, and that this was especially true for women who were not initially overweight.[37] In addition to prepregnancy BMI, a number of

other demographic factors affect the incidence of GDM. Hedderson and colleagues[37] found that GDM was more likely in women who were older than 35 years of age and who were of Hispanic or Asian ethnicity. In this cohort, GDM was also more common in women with 12 years or less of schooling and with 2 or more previous live births.

The reason obese women are at higher risk of developing GDM has yet to be fully delineated, but is likely related to an increase in insulin resistance. As a result of the continued production of counterregulatory (anti-insulin) hormones by the growing placenta, insulin resistance increases progressively throughout pregnancy. At any single point in pregnancy, however, obese women have higher insulin resistance (lower insulin sensitivity) than women of normal weight, which results in increased availability of lipids for fetal growth and development.[33] Gene microarray profiling of the placentae of obese women with GDM demonstrates increased expression of genes related to lipid metabolism and transport,[39] which likely accounts for the increase in birth weight and fat mass observed in the offspring of such women.

The development of GDM has a number of adverse maternal and fetal implications. For women, these include an increased risk of hyperglycemia, cesarean delivery, and diabetes in later life, with more than 50% of women with GDM acquiring diabetes within 20 years of delivery.[40] The implications for the offspring may be even more severe. Pregnancies complicated by GDM have a 4-fold increased risk of perinatal mortality and a 3-fold increased risk of macrosomia. In addition to being larger, infants born of pregnancies complicated by GDM also have significantly larger skin folds at all areas of measurement (triceps, subscapular, flank, thigh, abdomen) and, as such, are at increased risk of shoulder dystocia and resultant birth injury.[41] Moreover, offspring born of GDM pregnancies are more likely to develop childhood and adult obesity (OR 1.4 [95% CI, 1.2-1.6] for every 1-kg increment in birth weight) as well as type 2 diabetes mellitus.[42]

Physician Responsibility

With the known adverse consequences of maternal obesity, it is important that physicians address this issue with their patients. Disconcertingly, Honda[43] found that, over a period of 1 year, only 21.3% and 24.5% of adults who visited their physician received advice about diet and exercise, respectively. On a positive note, a recent survey of 900 obstetrician-gynecologists by The American College of Obstetricians and Gynecologists showed that 80% routinely counsel their pregnant patients about weight control, although only 35% believe that such prenatal counseling will significantly affect the incidence of obesity.[44]

Conclusions

The incidence of maternal obesity and its attendant comorbid conditions (diabetes, cardiovascular disease) continues to increase at an alarming rate, with major public health implications. Not only does maternal obesity affect the woman, but it also impacts the health of the child, leading to increased childhood obesity and diabetes. Despite improvements in our understanding of this endocrinopathy, there are still many barriers to the clinical care for such women. Obstetrician-gynecologists are in a key position to prevent and treat this epidemic. ■

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