



Obesity in pregnancy: Complications and maternal management

Authors: [Patrick S Ramsey, MD, MSPH](#), [Robert S Schenken, MD](#)

Section Editors: [Charles J Lockwood, MD, MHCM](#), [F Xavier Pi-Sunyer, MD, MPH](#)

Deputy Editor: [Vanessa A Barss, MD, FACOG](#)

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INTRODUCTION — Obese pregnant women are at increased risk for an array of maternal and perinatal complications, and the risks are amplified with increasing degrees of maternal obesity [1-4]. Among the adverse effects on the fetus are an increased risk of childhood and adult obesity [5,6]. Obstetric providers should be aware of these risks and modify patient care before pregnancy, during pregnancy, and postpartum to optimize maternal and offspring outcomes [7,8].

This topic will discuss issues related to obesity and pregnancy. Obesity in the nonpregnant population is reviewed separately. (See "[Obesity in adults: Prevalence, screening, and evaluation](#)" and "[Overweight and obesity in adults: Health consequences](#)" and "[Obesity in adults: Overview of management](#)".)

PATHOBIOLOGY — Adipose tissue is an active endocrine organ; when present in excess, it can have dysregulatory effects on metabolic, vascular, and particularly inflammatory pathways in many organ systems, and thereby affect obstetric outcomes [9]. For example, obesity-related insulin resistance and abnormalities in inflammatory pathways can affect placental growth and function [10], and have been linked to development of preeclampsia [11,12]. The observation that some obesity-related pregnancy complications increase with greater degrees of obesity support this hypothesis [13]. Maternal genotype may also play a role [14].

Epigenetic changes induced by fetal exposure to increased levels of glucose, insulin, lipids, and inflammatory cytokines may play a role in the long-term outcome of offspring. These in utero effects may result in permanent or transient changes in metabolic programming, leading to adverse health outcomes in adult life (fetal origins of adult disease theory [Barker hypothesis]) [15]. The potential programming effects of maternal overnutrition are difficult to study, however, because of the complex relationships between the maternal metabolic milieu and the developing fetus and the influence of postnatal factors, including lifestyle and environment [16].

DEFINITION OF OBESITY — Obesity is defined as body mass index (BMI) ≥ 30 kg/m² ([table 1](#)) [8]. It is further stratified by class: class I (BMI 30.0 to 34.9 kg/m²), class II (BMI 35.0 to 39.9 kg/m²), and class III (BMI ≥ 40 kg/m²).

This standard definition for the nonpregnant population does not adapt well to the pregnant population since a pregnant woman's weight increases over a relatively short interval of time and much of the weight gain is related to accretion of mass that will be lost at delivery: the fetus, amniotic fluid, and blood. Since a pregnancy-specific definition of obesity has not been standardized, pregnant women are often considered obese or nonobese based on their prepregnancy BMI. (See "[Weight gain and loss in pregnancy](#)", [section on 'Change in weight related to pregnancy'](#).)

PREVALENCE OF OBESITY IN REPRODUCTIVE AGED WOMEN — The prevalence of obesity in reproductive-aged and pregnant women varies widely depending upon the definition used, year, and

characteristics of the study population, but has increased in concordance with the increased prevalence of obesity in the general population [17,18]. United States National Center for Health Statistics data for 2011 to 2014 showed 34.4 percent of women aged 20 to 39 years were obese (body mass index [BMI] ≥ 30 kg/m²); the prevalence was highest in non-Hispanic black women (56.9 percent) [19]. Among US women with live births in 2015, 25.8 percent were overweight and 25.6 percent were obese prepregnancy, according to National Vital Statistics System (NVSS) natality data [20]. By comparison, in 1980 (before routine calculation of BMI), only 7 percent of women weighed over 200 pounds at their first prenatal visit [21].

POTENTIAL ISSUES IN PREGNANCY

Overall risk of severe morbidity or mortality — In a population-based study including over 740,000 pregnant women, the composite "severe maternal morbidity or mortality" rates per 10,000 women by body mass index (BMI) were: normal BMI (143), overweight (160), class 1 obesity (168), class 2 obesity (178), and class 3 obesity (203) [4]. Compared with women with BMIs in the normal range, a small but statistically significant increase in the composite outcome was observed in overweight women and women with class 1, 2, and 3 obesity: adjusted odds ratio 1.1, 1.1, 1.2, and 1.4, respectively. Severe morbidity included hemorrhage requiring transfusion; serious cardiac, respiratory, cerebrovascular, or hematologic complications; venous thrombosis/embolism; sepsis; shock; hepatic or renal failure; anesthesia-related complications; and uterine rupture.

Antepartum

Early pregnancy loss — In a 2011 systematic review including six retrospective studies and a total of 28,538 women (3800 obese [BMI ≥ 28 or 30 kg/m²], 3792 overweight [BMI 25 to 29 kg/m²], and 17,146 normal weight [BMI <25 kg/m²]), the percentages of spontaneously conceiving women with ≥ 1 miscarriage were 16.6 percent for obese women, 11.8 for overweight women, and 10.7 percent for normal-weight women [22]. The odds of having ≥ 1 miscarriage were increased for obese women (odds ratio [OR] 1.31, 95% CI 1.18-1.46) and overweight women (OR 1.11, 95% CI 1.00-1.24) when compared with women with normal BMI. The cohort of women with recurrent miscarriage was small but showed a higher risk for recurrent early miscarriage in obese versus normal-BMI women (0.4 versus 0.1 percent, OR 3.51, 95% CI 1.03-12.01). Differences in patient characteristics and study designs, however, limit the validity of these findings.

Euploid embryos accounted for the observed increased risk of miscarriage among overweight and obese women in one study [23] but not in another [24]. If the early excess loss of euploid embryos is confirmed, one mechanism may be an unfavorable hormonal environment related to obesity and resulting in poorer endometrial receptivity [25]. Another mechanism may involve inflammatory changes related to polycystic ovary syndrome (PCOS). PCOS has been associated with a miscarriage rate 20 to 40 percent higher than the baseline in the general obstetric population [26]. A prospective study suggested that the low-grade chronic inflammation commonly seen in PCOS patients worsens during pregnancy and may account, at least in part, for the excess risk of adverse pregnancy outcomes such as miscarriage [27].

Occult type 2 diabetes — Although obesity is a risk factor for development of type 2 diabetes and screening is recommended in this population, screening may not have been performed or not performed recently. Screening in early pregnancy will detect these women. (See '[First trimester](#)' below.)

Gestational diabetes — The prevalence of gestational diabetes mellitus (GDM) is significantly higher in obese women than in the general obstetrical population ([table 2](#) and [table 3](#)) [28,29], and the risk increases with increasing maternal weight and BMI [1,30,31]. The increased risk of GDM is related to an exaggerated increase in insulin resistance in the obese state [32]. In a systematic review of studies on prepregnancy BMI and risk of GDM, the prevalence of GDM increased by 0.92 percent for every 1 kg/m² increase in BMI [1].

Pregnancy associated hypertension — An association between obesity and hypertensive disorders during pregnancy has been consistently reported ([table 2](#) and [table 3](#)). In particular, maternal weight and BMI are independent risk factors for preeclampsia, as well as other hypertensive disorders [33-40]. In a systematic review of 13 cohort studies comprising nearly 1.4 million women, the risk of preeclampsia doubled

with each 5 to 7 kg/m² increase in prepregnancy BMI [36]. This relationship persisted in studies that excluded women with chronic hypertension, diabetes mellitus, or multiple gestations, or after adjustment for other confounders. Cohort studies of women who underwent bariatric surgery suggest that weight loss significantly reduces the occurrence of preeclampsia [41]. (See "[Preeclampsia: Clinical features and diagnosis](#)".)

The mechanism whereby obesity imparts an increased risk for preeclampsia is not well-defined. The pathophysiologic changes associated with obesity-related cardiovascular risk, such as insulin resistance, hyperlipidemia, and subclinical inflammation, may also be responsible for the increased incidence of preeclampsia in obese gravidas [42-44]. (See "[Overweight and obesity in adults: Health consequences](#)", [section on 'Hypertension'](#).)

Indicated and spontaneous preterm birth — Obesity increases the risk of medically indicated preterm delivery, primarily due to obesity-related maternal disorders, such as hypertension, preeclampsia, and diabetes. In a 2010 systematic review of maternal overweight and obesity and risk of preterm birth, overweight and obese women were at increased risk of induced preterm birth compared with women of normal BMI (relative risk [RR] 1.30, 95% CI 1.23-1.37, five studies) and the risk increased with increasing weight [45].

Whether obesity increases the risk for spontaneous preterm birth is less clear. The 2010 systematic review found no difference in the rate of spontaneous preterm birth between groups; however, heterogeneity was high. A subsequent population-based cohort study from Sweden including over 1.5 million singleton deliveries confirmed that overweight and obese women were at increased risk of medically indicated preterm deliveries at all gestational ages, but also observed a significant dose-response relationship between severity of obesity and risk of spontaneous extremely preterm delivery (22 to 27 weeks), but not for very preterm (28 to 31 weeks) or moderately preterm (32 to 36 weeks) delivery after adjustment of confounders [46]. The authors hypothesized that the obesity-related inflammatory up-regulation, as well as other factors (eg, subclinical genital tract infection), increased the risk of spontaneous extremely preterm delivery.

Several studies have also demonstrated an independent association between PCOS and spontaneous preterm birth, including cervical insufficiency [47,48]. The precise mechanism by which PCOS modulates the risk for spontaneous preterm birth or cervical insufficiency, independent from or as part of the obesity effects, has not been well defined but may be related to changes in the hormonal milieu (eg, increased relaxin levels) that weaken the cervical collagen matrix.

Post-term pregnancy — The body of evidence supports an association between obesity and post-term pregnancy (OR 1.2 to 1.7 in four population-based studies [28,30,49,50]) [28,30,49-53].

The mechanism by which obesity prolongs pregnancy has not been determined. One hypothesis is that gestational age calculated from last menstrual period overestimates true fetal age because obese women tend to be oligo-ovulatory; this hypothesis is supported by studies of early ultrasound assessment of gestational age in this population that found the EDD by LMP was earlier than the EDD by ultrasound [54-56]. Others have hypothesized that hormonal changes associated with obesity may interfere with the hormonal changes that initiate the onset of parturition.

Multifetal pregnancy — An increased incidence of dizygotic but not monozygotic twin gestation has been observed among obese gravidas [29,57,58]. In an analysis of 51,783 pregnancies (561 twin) in the Collaborative Perinatal Project, the incidence of dizygotic twins in women with BMI ≥ 30 kg/m² and < 25 kg/m² was 1.1 and 0.5 percent, respectively [58]. These data were derived from patients in 12 hospitals in the United States from 1959 to 1966, before widespread use of agents for ovulation induction.

The association of maternal weight with dizygotic twinning has been attributed to elevated follicle-stimulating hormone (FSH) levels in obese women. Although a direct relationship between obesity and elevated FSH has not been demonstrated, mean FSH levels are highest in women who have had two sets of twins previously, intermediate in women who have had one set of twins, and lowest in women who have only delivered singletons [59].

Obstructive sleep apnea — Obstructive sleep apnea (OSA) may be precipitated or exacerbated during pregnancy and may increase the risk for preeclampsia and GDM. Postoperative respiratory depression can be particularly problematic in patients with OSA receiving opiates after cesarean delivery. (See "[Obstructive sleep apnea in pregnancy](#)".)

Carpal tunnel syndrome — Both obesity and pregnancy are associated with an increased risk of carpal tunnel syndrome. (See "[Neurologic disorders complicating pregnancy](#)", [section on 'Carpal tunnel syndrome'](#).)

Intrapartum

Induction — Obese women are at increased risk for labor induction due to their increased risk for pregnancy complications [49,53,60]. They are also at increased risk for induction failure. In one study, for example, obese women overall were twice as likely to experience a failed induction as normal-weight women and the risk increased with increasing class of obesity (ORs for class I, II, and III: 1.85, 2.30, and 2.89, respectively) [60]. In another study of induction in nulliparas, the rate of cervical dilation and labor duration were inversely associated with maternal weight (eg, a 0.3 hour increase in the oxytocin to delivery interval was observed for each 10-kg increment in weight) [61].

Progress of labor — Maternal obesity appears to have a modest impact on labor progression that is independent of fetal size [61-70] but related to maternal size. In a cohort study of 612 nulliparas who delivered following spontaneous labor, the median duration of labor from 4 to 10 cm was significantly longer for both overweight and obese women compared with normal-weight women (7.5, 7.9, and 6.2 hours, respectively) [62].

The duration of the second stage of labor does not appear to be affected by increasing BMI [66].

Cesarean delivery — Obesity is a risk factor for both elective and emergency cesarean delivery, and the risk increases with increasing maternal weight [71-73]. In one study, each unit increase in prepregnancy BMI resulted in a 7 percent increase in risk of cesarean delivery [73]. Obesity-related pregnancy complications, higher infant birth weight, and increased frequency of preterm and post-term delivery account for some of the excess risk of cesarean delivery [17,74-78]. However, obesity also appears to be an independent risk factor, possibly because of the adverse effects on labor progress described above.

Cesarean delivery of the obese gravida is associated with numerous perioperative concerns, which are reviewed separately. (See "[Cesarean delivery of the obese woman](#)".)

Trial of labor after cesarean delivery — Observational studies have consistently reported that a trial of labor after a cesarean delivery is less likely to result in vaginal birth in obese gravidas. (See "[Choosing the route of delivery after cesarean birth](#)", [section on 'Obese women'](#).)

Difficulties with anesthesia — Obese women undergoing neuraxial obstetric anesthesia have been reported to have higher rates of multiple attempts at placement [79], inadvertent dural puncture [79], failed analgesia requiring a repeat procedure [80], and hypotension [81]. If general anesthesia is required, obesity is one of the factors predictive of a difficult airway [82,83]. (See "[Anesthesia for the obese patient](#)" and "[Cesarean delivery of the obese woman](#)", [section on 'Anesthesia'](#).)

Complications related to macrosomia — Macrosomia (birth weight >4000 g) is more common in offspring of obese women (table 3) and an important risk factor for shoulder dystocia. (See "[Shoulder dystocia: Risk factors and planning delivery of high-risk pregnancies](#)", [section on 'High birth weight'](#) and "[Shoulder dystocia: Risk factors and planning delivery of high-risk pregnancies](#)", [section on 'Maternal obesity and high gestational weight gain'](#).)

Other potential intrapartum complications of macrosomia include dysfunctional labor, operative intervention (forceps or vacuum vaginal delivery, cesarean delivery), maternal genital tract laceration, and postpartum hemorrhage. (See "[Fetal macrosomia](#)", [section on 'Risk factors'](#).)

Postpartum

Venous thromboembolism — Obesity, the pregnant/postpartum state, and cesarean delivery are independent risk factors for venous thromboembolism (VTE), which is a major cause of maternal morbidity and mortality [84]. In one review, the risk for postpartum VTE in women with class I, II, and III obesity was OR 2.5, 2.9, and 4.6, respectively, compared with women whose BMI was normal [85]. The absolute risk of VTE in women with a combination of risk factors depends on the combination and is unclear. (See "[Deep vein thrombosis in pregnancy: Epidemiology, pathogenesis, and diagnosis](#)", section on 'Risk factors'.)

Infection — The obese gravida is at higher risk for postpartum infection (wound, episiotomy, endometritis), regardless of mode of delivery and despite use of common prophylactic antibiotic regimens [30,33,82,86-88]. Poor vascularity of subcutaneous adipose tissue and formation of seromas and hematomas account, at least in part, for the increased risk of wound infection.

Postpartum depression — A 2014 meta-analysis of 62 studies of obesity and mental disorders during pregnancy and postpartum noted an increased incidence of postpartum depression in obese women (OR 1.30, 95% CI 1.20-1.42) [89].

Offspring

Congenital anomalies — Obese women are at increased risk for having a fetus with congenital anomalies, including neural tube defects, cardiac malformations, orofacial defects, and limb reduction abnormalities [90]. The risk appears to increase with an increasing degree of maternal obesity. The mechanism for these associations is not well defined but is likely related to an altered nutritional milieu during fetal development.

The following systematic reviews/meta-analyses illustrate the magnitude of risk:

- A 2009 systematic review (39 studies) and meta-analysis (18 studies) evaluated observational studies that estimated prepregnancy or early pregnancy weight or BMI and provided data on congenital anomalies [90]. Compared with pregnancies in women with a normal BMI, pregnancies in obese mothers were at increased risk of:
 - Neural tube defects (NTDs) (OR 1.87, 95% CI 1.62-2.15), spina bifida (OR 2.24, 95% CI 1.86-2.69), hydrocephaly (OR 1.68, 95% CI 1.19-2.36)
 - Cardiovascular anomalies (OR 1.30, 95% CI 1.12-1.51), septal anomalies (OR 1.20, 95% CI 1.09-1.31)
 - Cleft palate (OR 1.23, 95% CI 1.03-1.47), cleft lip and palate (OR 1.20, 95% CI 1.03-1.40)
 - Anorectal atresia (OR 1.48, 95% CI 1.12-1.97)
 - Limb reduction anomalies (OR 1.34; 95% CI 1.03-1.73)

In contrast, the risk of gastroschisis was significantly reduced (OR 0.17; 95% CI 0.10-0.30).

Other systematic reviews have demonstrated that as the severity of maternal obesity increased, the risk for NTDs [91] and congenital heart defects [92] also increased. While these observations are consistent among studies, authors have not consistently adjusted for diabetes status in their analyses or excluded women with diabetes. As diabetes is an established risk factor for fetal anomalies, incompletely accounting for diabetes or first trimester glycemic control may explain some or all of the increased risks reported in these studies.

Of note, one study noted that obese women did not experience the typical reduction in NTD risk associated with standard doses of [folic acid](#) supplementation, suggesting that folate deficiency may not be the underlying etiology of NTDs in obese women [93] (see "[Folic acid supplementation in pregnancy](#)", section on 'Treatment failures'). Others have not demonstrated a decrease in congenital anomalies after bariatric surgery, although

this may be related to the relatively small numbers of patients and low absolute risk of congenital abnormality. (See "[Fertility and pregnancy after bariatric surgery](#)", section on 'Pregnancy outcomes'.)

There are several other limitations to these data. For example, congenital anomalies are more difficult to detect with prenatal ultrasonography in obese women, resulting in fewer optimal examinations and antepartum diagnoses and, in turn, fewer pregnancy terminations [94-102]. Maternal obesity reduces the detection of fetal anomalies by at least 20 percent compared with women with a normal BMI [98,101,103]. Although most studies attempted to adjust for confounders, adjustment factors varied among the studies. Lastly, criteria for obesity and ascertainment of obesity were not consistent across all the studies.

Asphyxia and death — Risks for birth asphyxia, fetal death, stillbirth, perinatal death, neonatal death, and infant death are all increased in the setting of maternal obesity. A population-based cohort study from Sweden reported increased risks of perinatal asphyxia-related complications (Apgar 0 to 3 at five minutes, meconium aspiration, neonatal seizures) with increasing maternal BMI in singleton term infants [104]. A 2014 systematic review and meta-analysis of cohort studies found that even modest increases in maternal BMI increased the risk of fetal death, stillbirth, and infant death [105]. The 44 publications included >10,000 fetal deaths, >16,000 stillbirths, >4000 perinatal deaths, >11,000 neonatal deaths, and almost 5000 infant deaths. The major findings were:

- Relative risk per 5 unit increase in maternal BMI: fetal death 1.21 (95% CI 1.09-1.35, n = 7 studies), stillbirth 1.24 (95% CI 1.18-1.30, n = 18 studies), perinatal death 1.16 (95% CI 1.00-1.35, n = 11 studies), neonatal death 1.15 (95% CI 1.07-1.23, n = 12 studies), and infant death 1.18 (95% CI 1.09-1.28, n = 4 studies).
- Absolute risk per 10,000 pregnancies for women with BMI of 20, 25, and 30 were: fetal death 76, 82, and 102, respectively; stillbirth 40, 48, and 59, respectively; perinatal death 66, 73, and 86, respectively; neonatal death 20, 21, and 24, respectively; and infant death 33, 37, 43, respectively.

Several hypotheses have been proposed to explain the above observations. Obese women have higher rates of diabetes and hypertension than nonobese women; however, a proportion of the excess risk of death remains after adjustment for these factors. Other potential etiologies include metabolic changes associated with obesity (hyperlipidemia with reduced prostacyclin production) and nocturnal apnea with transient oxygen desaturation. It is also possible that confounding factors, such as maternal age, smoking, and infants with congenital anomalies, account for the disparity. Antepartum, external fetal heart rate monitoring may not provide a clear tracing in severely obese women; intrapartum, clinicians may delay performance of emergency cesarean delivery in such women. Although most studies attempted to adjust for confounders, adjustment factors varied among the studies.

Another concerning observation is the increased risk for stillbirth with increasing degrees of maternal obesity. Previous work has demonstrated that the risk for stillbirth increases with increasing obesity (stillbirth adjusted hazard ratio [95% CI]: class I obesity 1.3 [1.2-1.4], class II obesity 1.4 [1.3-1.6], class III obesity 1.9 [1.3-1.6]) and higher stillbirth risk in African American obese women (1.9 [1.7-2.1]) than in Caucasian obese women (1.4 [1.3-1.5]) [8,106]. Among women with class III obesity and women with a BMI ≥ 50 kg/m², the risk for stillbirth increased with advancing gestational age: 30 to 33 weeks, hazard and risk ratios 1.40 and 1.69, respectively; 37 to 39 weeks, hazard and risk ratios 3.20 and 2.95, respectively; and 40 to 42 weeks, hazard and risk ratios 3.30 and 8.95, respectively [8,107].

A decrease in perinatal mortality has not been reported after bariatric surgery, although this may be related to the relatively small numbers of patients and very low absolute risk of perinatal death. (See "[Fertility and pregnancy after bariatric surgery](#)", section on 'Pregnancy outcomes'.)

Prematurity — (See '[Indicated and spontaneous preterm birth](#)' above.)

Large for gestational age — Prepregnancy obesity and maternal weight gain both play an important role in determining infant birth weight. Several studies have reported that increasing prepregnancy weight has a

linear relationship with birth weight [51,108,109]; as a result, the obese gravida is at increased risk of delivering a large for gestational age (LGA) infant [21,28,29,37,49,51,76,82,86,87,110]. This relationship is independent of the increased prevalence of GDM in obese women, but may relate to maternal and fetal hyperinsulinemia [38,74,111]. In a prospective longitudinal study, fetuses of obese women without major chronic diseases had longer femur and humerus lengths and larger head circumferences than those of nonobese women, beginning in the second half of pregnancy [112]. Beginning at 32 weeks of gestation, they also had greater abdominal circumferences than those of normal-weight women. These relationships persisted after exclusion of women with pregnancy complications that can impact fetal growth (eg, gestational diabetes, preeclampsia).

Two potential sequelae of being LGA are:

- Shoulder dystocia (see "[Shoulder dystocia: Risk factors and planning delivery of high-risk pregnancies](#)")
- Predisposition to obesity later in life (see "[Large for gestational age newborn](#)", section on 'Potential long-term effects')

Asthma — A 2014 meta-analysis of observational studies noted an association between maternal obesity in pregnancy and risk of current asthma/wheeze in offspring (OR 1.35, 95% CI 1.08-1.68, five studies); maternal asthma history did not account for the findings [113]. The biologic mechanisms for this association have not been elucidated but may involve obesity-related changes in inflammatory pathways and dietary exposures.

Childhood obesity — Having one obese parent increases the risk of obesity by two- to threefold, and up to 15-fold if both parents are obese. Maternal obesity is a particular concern because in-utero nutritional excess and development in an obesogenic environment may lead to permanent changes of fetal metabolic pathways and thereby increase the risk of childhood and adult diseases related to these pathways, such as obesity, diabetes, hypertension, and cardiovascular disease. However, shared genetic or familial lifestyle also plays a role. (See "[Definition; epidemiology; and etiology of obesity in children and adolescents](#)".)

Neurodevelopment — There is a growing body of evidence that prenatal and lactational exposure to maternal obesity and high-fat diet are associated with neurodevelopmental and psychiatric disorders in offspring, such as cognitive impairment, autism spectrum disorders, attention deficit hyperactivity disorder, anxiety and depression, schizophrenia, and eating disorders [114-116]. Most studies have not controlled for antenatal maternal anxiety and depression, which can impact the frequency of psychopathologic disorders in offspring. (See "[Antenatal depression: Risks of cognitive impairment and psychopathology in the offspring](#)".) Genetic, social, and environmental risk factors may also contribute to development of behavioral problems in offspring [117]. Adjustment for these factors attenuates reported associations between maternal obesity and neuropsychological development in offspring.

An increased risk of cerebral palsy (CP) has been observed in term singletons born to overweight and obese women in Sweden [118]. In this population-based cohort study, adjusted hazard ratios for CP increased progressively from 1.26 at BMI 25 to 29.9 kg/m² to 1.86 at BMI ≥40 kg/m² compared with women of normal BMI, but the absolute incidence of CP in offspring of these women was low: 2.22 to 3.41 cases/10,000 child-years. The association was not observed in preterm births. Part of the increased risk appeared to be mediated by asphyxia-related neonatal complications, which have been reported to be more prevalent in overweight and obese women [104]. The same group reported a predisposition to childhood epilepsy that was attenuated but persisted after adjusting for multiple maternal and neonatal confounders [119].

Possible mechanisms for adverse effects in offspring include neuroinflammation; increased oxidative stress; and dysregulated insulin, glucose, leptin, serotonergic, and dopaminergic signaling.

PREPREGNANCY MANAGEMENT

Preconception counseling, evaluation, and care — Ideally health care providers and obese women should discuss reproductive planning well before conception. This discussion should include:

- Information about the adverse effects of obesity on fertility. (See "[Optimizing natural fertility in couples planning pregnancy](#)", [section on 'Overweight and obesity'](#).)
- Information about the potential pregnancy complications associated with obesity, as described above.
- Evaluation for obesity-associated medical comorbidities (eg, diabetes, hypertension) and appropriate intervention to optimize maternal health status. (See "[Overweight and obesity in adults: Health consequences](#)" and "[Obesity in adults: Prevalence, screening, and evaluation](#)", [section on 'Assessing obesity-related health risk'](#).)
- Counseling about the benefits of weight loss before attempting to conceive. (See "[Prepregnancy weight loss](#)" below.)

Prepregnancy weight loss — Obese women are encouraged to undertake a weight reduction program (diet, exercise, behavior modification), and possibly adjunctive medical therapy or bariatric surgery, if indicated, before attempting to conceive because weight loss appears to have beneficial effects on reproductive function, pregnancy outcome, and overall health [[8,120,121](#)]. Importantly, almost all drugs prescribed for weight reduction have adverse fetal effects and should **not** be used during pregnancy. (See "[Obesity in adults: Overview of management](#)" and "[Obesity in adults: Drug therapy](#)".)

Although randomized trials have not demonstrated that dietary and lifestyle interventions during pregnancy improve maternal and infant outcomes, the trials have generally been of poor to fair methodological quality [[122,123](#)]. Data from observational studies support the benefits of prepregnancy weight loss [[124-129](#)]. For example:

- Data from prospective cohort studies suggest that a decrease in pregravid weight from obese to normal between first and second pregnancies reduced the risk of cesarean delivery and delivery of a large for gestational age (LGA) infant [[124,125](#)].
- Population-based retrospective cohort studies have observed that obese women who reduced their body mass index (BMI) between pregnancies (eg, by at least one or two units) substantially reduced their risk of having a LGA infant, improved the likelihood of vaginal birth after cesarean delivery (VBAC), and reduced risks of gestational diabetes mellitus (GDM) and stillbirth compared with obese women who maintained their weight [[126-129](#)]. As an example, interpregnancy weight loss ≥ 2 BMI units was associated with 40 percent reduction in risk of an LGA infant in one study [[128](#)].
- Among women who undergo bariatric surgery, the risks for preeclampsia, GDM, and LGA/macrosomia are reduced compared with obese women who have not undergone a bariatric procedure. The risks for spontaneous preterm delivery, cesarean delivery, and congenital anomalies appear to be the same or lower than in obese women who have not undergone a bariatric procedure, but data are less consistent than for other outcomes. These data are reviewed separately. (See "[Fertility and pregnancy after bariatric surgery](#)", [section on 'Pregnancy outcomes'](#).)

PREGNANCY MANAGEMENT — Given the associations between obesity and adverse pregnancy outcome described above, modifications to routine prenatal care (see "[Prenatal care: Initial assessment](#)") have been suggested for this population, as described below. An appropriate multidisciplinary team includes the obstetric provider, high-risk pregnancy specialist, nutritionist, and, during labor and delivery, the anesthesiologist.

First trimester

Baseline assessments

- Maternal weight and body mass index (BMI).
- Blood pressure using an appropriately sized cuff.
- Early ultrasound to establish gestational age and determine whether there is a multifetal gestation.
- Medication review, particularly exposure to weight loss medications, which should be discontinued, and use of oral anti-hyperglycemic drugs, which are often discontinued in favor of insulin therapy. (See ["Pregestational diabetes mellitus: Glycemic control during pregnancy", section on 'Women on oral anti-hyperglycemic agents prior to pregnancy'.](#))
- Diabetes screening (see ["Diabetes mellitus in pregnancy: Screening and diagnosis", section on 'Identification of overt diabetes in early pregnancy'.](#)). Screening procedures may need to be modified in patients who have undergone bariatric surgery. (See ["Fertility and pregnancy after bariatric surgery", section on 'Gestational diabetes'.](#))
- Consider quantitative urine protein, creatinine concentration, platelet count, and liver function tests, which can be useful for comparison with laboratory values later in pregnancy if the patient undergoes evaluation for preeclampsia. Obesity is a known risk factor for nonalcoholic fatty liver disease (NASH). (See ["Epidemiology, clinical features, and diagnosis of nonalcoholic fatty liver disease in adults".](#))
- In women who have undergone bariatric surgery, evaluate for and treat nutritional deficiencies. (See ["Fertility and pregnancy after bariatric surgery", section on 'Micronutrient supplementation'.](#))

Counseling — The patient should receive information regarding potential pregnancy risks associated with obesity (as described above), diet and gestational weight gain, and exercise. Her diet, weight gain/loss, and physical activity should be reviewed frequently throughout pregnancy and the postpartum period.

Gestational weight gain — The Institute of Medicine (IOM) provides guidance for recommended total and rate of weight gain in pregnancy ([table 4](#)) [[130](#)]. For the obese gravida, limiting gestational weight gain may reduce the risk of some adverse pregnancy outcomes, such as macrosomia. However, weight loss has been associated with an increased risk of small for gestational age newborns [[131](#)].

Working with a nutritionist throughout pregnancy and the puerperium can help patients plan meals for optimum gestational weight gain and avoidance of postpartum weight retention [[132](#)]. (See ["Nutrition in pregnancy"](#) and ["Weight gain and loss in pregnancy"](#).)

Exercise — Exercise has multiple health benefits. Pregnant women can initiate an exercise program or continue most prepregnancy exercise programs, which can help control gestational weight gain [[133,134](#)] and may improve some pregnancy outcomes (eg, reduced risk of gestational diabetes) [[135](#)]. (See ["Exercise during pregnancy and the postpartum period"](#) and ["Diabetes mellitus in pregnancy: Screening and diagnosis", section on 'Preventive approaches for risk reduction'.](#))

Fetal aneuploidy screening — Our approach to fetal aneuploidy screening in the obese gravida is the same as that for the general population and irrespective of BMI. Limitations and challenges of each testing modality should be recognized and patients counseled appropriately.

Obese women are not at increased risk for fetal aneuploidy; however, obesity can affect screening test performance. Cell-free fetal DNA screening is more likely to result in test failure or an inaccurate result in obese women because they may have a lower fetal fraction of the cell-free DNA. (See ["Prenatal screening for common aneuploidies using cell-free DNA", section on 'Obese women'.](#))

First and second trimester serum-based screening tests are adjusted for maternal weight; thus, obesity does not affect test performance. However, accurate nuchal translucency measurement may be more difficult to obtain and, in one study, the failure rate increased with increasing maternal BMI category [[136](#)]. While the authors did not attempt to delineate the potential benefits of transvaginal over transabdominal approach,

theoretically, use of transvaginal sonography may offer improved ascertainment of nuchal translucency measurements irrespective of degree of obesity.

Diagnostic procedures (amniocentesis, chorionic villus sampling) are more challenging technically. Avoiding imaging through the pannus, repositioning the transducer to improve the scanning angle, or even using a vaginal probe in the umbilicus may be helpful to optimize visualization. Use of a low-frequency transducer and a narrow sector width may also help to increase tissue penetration and improve imaging success during diagnostic procedures [137].

Referrals — If underlying cardiopulmonary disease is suspected, cardiology or pulmonology referral should be considered for additional testing and diagnosis.

Referral to a sleep specialist should be individualized based on BMI, comorbidities, and symptomatology, such as snoring, witnessed apneas, and/or falling asleep while driving. While there are no universal guidelines for screening for obstructive sleep apnea solely based on BMI, the risk in women with BMI >30 kg/m² was 15.4 percent in one study [138]. (See "[Obstructive sleep apnea in pregnancy](#)".)

Referral to a registered dietician for ongoing consultation can provide patients with dietary plans and goals as well as guidance about healthy lifestyle changes.

Second trimester

Low-dose aspirin — Obese women with additional risk factors for development of preeclampsia may benefit from treatment with low dose [aspirin](#) (81 mg). We follow the approach suggested by the United States Preventative Services Task Force (USPSTF) [139] and endorsed by the American College of Obstetricians and Gynecologists [140]. In 2014, the USPSTF reviewed the available literature and concluded that obesity, defined as BMI \geq 30 kg/m², was a moderate risk factor for preeclampsia and recommended consideration of low-dose aspirin if the patient has several moderate risk factors. Other moderate risk factors included nulliparity, family history of preeclampsia (mother or sister), sociodemographic characteristics (African American race, low socioeconomic status), maternal age \geq 35, and personal factors (eg, low birth weight or small for gestational age, previous adverse pregnancy outcome, >10-year pregnancy interval) [139]. Identification of obese women with an appropriate combination of moderate risk factors to be considered high risk is subjective and determined case by case, as the data describing the magnitude of the association between each of these risk factors and development of preeclampsia vary widely and lack consistency.

These data and use of low dose [aspirin](#) for prevention of preeclampsia are discussed in more detail separately. (See "[Preeclampsia: Prevention](#)".)

Fetal ultrasound survey — A detailed (not basic) fetal anatomic survey is performed at 18 to 24 weeks of gestation to screen for fetal anomalies [141]. Because abdominal adiposity may make interpretation difficult, follow-up ultrasound assessment(s) may be needed. (See "[Ultrasound examination in obstetrics and gynecology](#)", [section on 'Detailed examination'](#) and "[Ultrasound examination in obstetrics and gynecology](#)", [section on 'Obesity'](#).)

Due to the limitations of ultrasound with increasing degrees of obesity, we suggest concomitant use of maternal serum alpha fetoprotein to screen for neural tube and other relevant congenital defects. (See "[Open neural tube defects: Risk factors, prenatal screening and diagnosis, and pregnancy management](#)", [section on 'Screening tests'](#).)

In our opinion, the increased risk for fetal cardiac malformations and lower detection rates for congenital cardiac malformations with maternal obesity are sufficient to support the use of fetal echocardiography in these pregnancies, unless the detailed obstetric ultrasound assessment of the heart (four chambers, outflow tracts, three-vessel and tracheal view [141]) is optimal and normal. The American Heart Association Scientific Statement on Diagnosis and Management of Fetal Cardiac Disease does not specifically cite maternal obesity as an indication for fetal echocardiography [142]. (See "[Fetal cardiac abnormalities: Screening](#),"

[evaluation, and pregnancy management](#)", [section on 'Basic fetal cardiac evaluation'](#) and ["Fetal cardiac abnormalities: Screening, evaluation, and pregnancy management"](#), [section on 'Advanced fetal cardiac evaluation'](#).)

Screening for gestational diabetes — If diabetes has not been previously identified, screening for gestational diabetes is recommended at 24 to 28 weeks of gestation. (See ["Diabetes mellitus in pregnancy: Screening and diagnosis"](#).)

For women with Roux-en-Y gastrojejunostomy or other malabsorptive bypass forms of bariatric surgery, a glucose load may incite maternal dumping syndrome so a different approach is used for diabetes screening. (See ["Fertility and pregnancy after bariatric surgery"](#), [section on 'Gestational diabetes'](#).)

Third trimester

Assessment of fetal growth — Clinical assessment of fetal size by abdominal palpation and measurement of fundal height is more challenging in the obese gravida. Ultrasound assessment of fetal growth every four to six weeks is reasonable.

Assessment of fetal well-being — Although the frequency of fetal demise appears to be increased in pregnancies of obese women, the mechanism is unclear and the value of antenatal fetal surveillance with nonstress tests or biophysical profile scoring in this setting has not been studied. We initiate antepartum fetal assessment for standard indications. (See ["Overview of antepartum fetal surveillance"](#), [section on 'Indications for fetal surveillance'](#) and ["Cesarean delivery: Preoperative planning and patient preparation"](#), [section on 'Indications'](#).)

External cephalic version — While study results have been mixed regarding the effect of obesity on the success of external cephalic version (ECV), obesity is not a contraindication to ECV [143]. A successful ECV is particularly beneficial in obese women, given the significant surgical risks of cesarean delivery in these patients. (See ["External cephalic version"](#).)

Labor and delivery

Equipment and instruments — Ensure that the labor and delivery unit has appropriate physical resources (eg, gowns, beds, stirrups, operating room tables, scales, transport vehicles and equipment, lifting equipment, floor-anchored toilet, surgical instruments) for caring for severely obese women. As an example, disposable retractors that anchor the pannus cephalad increase exposure during cesarean and free the surgeons' hands. (See ["Hospital accreditation, accommodations, and staffing for care of the bariatric surgical patient"](#).)

Fetal monitoring — Obtaining a continuous fetal heart rate tracing with an external Doppler ultrasound transducer can be difficult in obese women. Placement of an internal fetal scalp electrode avoids this problem. Use of maternal abdominal electrodes to detect the fetal electrocardiogram is an uncommon and novel approach but also appears to be reliable in this population [144].

Anesthesia consultation — Evaluation by an anesthesiologist prior to labor or in early labor is recommended for all obese parturients because of their higher risk of anesthetic challenges and complications. (See ["Preanesthesia medical evaluation of the obese patient"](#) and ["Anesthesia for the obese patient"](#).)

For patients planning a vaginal birth, early placement of an epidural or intrathecal catheter may obviate the need for general anesthesia if emergency cesarean is needed [83].

Timing and route of delivery — Delivery by the estimated due date has been recommended to reduce the risk of stillbirth and complications from continued fetal growth. In one protocol, women meeting any of the following criteria were delivered by their estimated due date: (1) prepregnancy BMI ≥ 40 kg/m², (2) prepregnancy BMI 35 to 39.9 kg/m² plus gestational diabetes mellitus or large for gestational age fetus, or (3)

prepregnancy BMI 30 to 34.9 kg/m² plus gestational diabetes mellitus and large for gestational age fetus [145]. Adoption of this protocol did not increase the cesarean delivery rate (it went down) and reduced the incidence of macrosomia. Fetal death rates did not change, but the study had insufficient power to detect a clinically significant reduction. This remains a controversial area. Induction by the estimated date of delivery is reasonable and does not appear to increase cesarean delivery rate; however, each institution should evaluate its approach and develop guidelines to help guide timing of delivery in obese women at or approaching term. (See "[Induction of labor with oxytocin](#)", [section on 'Obstetrical and medical'](#)".)

The route of delivery should be based on standard obstetric indications. Planned cesarean delivery is not associated with less morbidity than planned vaginal delivery, including among the superobese [146,147].

Cesarean delivery

Thromboprophylaxis — Obesity, the postpartum state, and cesarean delivery are independent risk factors for venous thromboembolism (VTE). The American College of Obstetricians and Gynecologists endorses universal use of pneumatic compression devices at the time of cesarean delivery and both mechanical and pharmacologic thromboprophylaxis in women at high risk of VTE undergoing cesarean delivery [148]. Evidence is lacking as to whether obese women undergoing cesarean delivery benefit from the addition of pharmacologic prophylaxis. Use of pharmacologic thromboprophylaxis and duration of postpartum prophylaxis depend on several patient-specific factors. Specific recommendations for thromboprophylaxis are reviewed separately. (See "[Cesarean delivery: Preoperative planning and patient preparation](#)", [section on 'Thromboembolism prophylaxis'](#)".)

Antibiotic prophylaxis — An appropriate dose of prophylactic antibiotics should be administered based on maternal weight. (See "[Cesarean delivery of the obese woman](#)", [section on 'Surgical-site infection prevention'](#)".)

Technical issues — Surgical technique needs to be modified to obtain adequate exposure and reduce the risk of postoperative infection and abdominal wall dehiscence. Increasing BMI is associated with increasing incision-to-delivery time, which may increase neonatal morbidity, particularly in emergency situations [149-151]. (See "[Cesarean delivery of the obese woman](#)", [section on 'Operative procedure'](#)".)

Postpartum

- If cesarean was performed, postcesarean care should be modified to reduce the risk of obesity-associated postsurgical complications. (See "[Cesarean delivery of the obese woman](#)", [section on 'Postoperative care'](#)".)
- Encourage breastfeeding and provide additional support. Contact with a lactation consultant before discharge from the hospital and soon after discharge can be helpful since obese women are prone to difficulty with lactation [152]. (See "[Initiation of breastfeeding](#)".)
- Intrauterine contraception is safe and effective, and may be safer and more effective in this population than estrogen-progestin contraceptives, although the latter are also an acceptable choice [153]. (See "[Contraception counseling for obese women](#)".)
- Women with a gestational diabetes should be screened for glucose intolerance 6 to 12 weeks after delivery. (See "[Gestational diabetes mellitus: Glycemic control and maternal prognosis](#)", [section on 'Follow-up and prevention of type 2 diabetes'](#)".)
- Support women their efforts to lose gestational weight gain, avoid postpartum weight gain, and achieve a healthy body mass index. Provide counseling and referrals, as appropriate. (See "[Obesity in adults: Overview of management](#)".)

GUIDELINES FROM NATIONAL ORGANIZATIONS

- [Centre for Maternal and Child Enquiries/Royal College of Obstetricians and Gynecologists](#)
- [American College of Obstetricians and Gynecologists](#)
- Society of Obstetricians and Gynaecologists of Canada [154]

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Beyond the Basics topics (see ["Patient education: Weight loss treatments \(Beyond the Basics\)"](#) and ["Patient education: Evaluation of the infertile couple \(Beyond the Basics\)"](#) and ["Patient education: Ovulation induction with clomiphene \(Beyond the Basics\)"](#))

SUMMARY AND RECOMMENDATIONS

- Obesity in pregnancy is best defined as prepregnancy body mass index (BMI) ≥ 30 kg/m², since the pregnant woman's weight increases over a relatively short interval of time and much of this weight gain is related to accretion of matter that will be lost at delivery. (See ["Definition of obesity"](#) above.)
- Adipose tissue is an active endocrine organ; when present in excess, it can have dysregulatory effects on metabolic, vascular, and inflammatory pathways in many organ systems, and thereby lead to a variety of reproductive and medical problems. (See ["Pathobiology"](#) above.)
- Compared with pregnant women with BMI < 25 kg/m², pregnancies among obese women are at increased risk of several adverse outcomes, including increased rates of early pregnancy loss, congenital anomalies, stillbirth, pregnancy-associated hypertension, preterm and post-term birth, gestational diabetes mellitus (GDM), multifetal gestation, and birth of a large for gestational age infant. Macrosomia may result in shoulder dystocia and its sequelae (brachial plexus injury) or cesarean delivery. Cesarean delivery in obese women is associated with increased rates of wound infection and thromboembolism.

Obese pregnant women are also at increased risk for maternal disorders, such as sleep-related breathing disorders, carpal tunnel syndrome, postpartum depression, and venous thromboembolism (VTE). (See ["Potential issues in pregnancy"](#) above.)

- Ideally, health care providers and obese women should discuss reproductive planning well before conception, including (see ["Pregpregnancy management"](#) above):
 - Information about the adverse effects of obesity on fertility.
 - Information about the potential pregnancy complications associated with obesity, as described above.
 - Evaluation for obesity-associated medical comorbidities (eg, diabetes, hypertension) and appropriate intervention to optimize maternal health status.
 - Counseling about the benefits of weight loss before attempting to conceive. Significant preconception weight loss likely reduces the risks of pregnancy complications. Some obese women

could benefit from referral for bariatric surgery. (See ['Prepregnancy weight loss'](#) above.)

- Given the associations between obesity and adverse pregnancy outcome, modifications to routine prenatal care have been suggested for this population. (See ['Pregnancy management'](#) above.)

Some of these modifications include:

- Screening for diabetes in early pregnancy. (See ['Baseline assessments'](#) above.)
 - Limiting gestational weight gain ([table 4](#)) (see ['Gestational weight gain'](#) above). Encouraging regular exercise can help control gestational weight gain. (See ["Exercise during pregnancy and the postpartum period"](#).)
 - Routine ultrasound examination to establish an accurate gestational age and for fetal anatomic survey. (See ['Fetal ultrasound survey'](#) above.)
 - Fetal echocardiography unless the standard basic obstetric ultrasound assessment of the four chambers and outflow tract is optimal and normal. (See ['Fetal ultrasound survey'](#) above.)
 - Discussing the additional challenges and limitations of fetal aneuploidy screening. Obese women are not at increased risk for fetal aneuploidy; however, obesity can affect screening test performance. Cell-free fetal DNA screening is more likely to result in test failure or an inaccurate result in obese women because they have a lower fetal fraction of the cell-free DNA. (See ['Fetal aneuploidy screening'](#) above.)
 - Referral to a sleep medicine specialist in selected women based on BMI and other comorbidities and/or symptomatology (eg, snoring, witnessed apneas, and/or falling asleep while driving). (See ['Obstructive sleep apnea'](#) above and ['Referrals'](#) above.)
 - Low-dose [aspirin](#) to reduce the risk of preeclampsia in women with multiple risk factors. (See ['Low-dose aspirin'](#) above.)
 - Ensuring that appropriate equipment is available. (See ['Equipment and instruments'](#) above and ['Technical issues'](#) above.)
 - Early evaluation by an anesthesiologist. (See ['Anesthesia consultation'](#) above.)
- Although stillbirth risks are increased in the setting of maternal obesity, there is no evidence demonstrating improved pregnancy outcome with routine use of antenatal fetal surveillance with nonstress tests or biophysical profile scoring; thus, a recommendation for or against use of antenatal testing cannot be made at this time. Testing should be initiated for standard indications. (See ['Assessment of fetal well-being'](#) above.)
 - Delivery timing and indications for labor induction should not be altered solely based on maternal obesity; however, induction by the estimated date of delivery is reasonable and does not appear to increase cesarean delivery rates. (See ['Timing and route of delivery'](#) above.)
 - For women undergoing cesarean delivery, an appropriate dose of prophylactic antibiotics should be administered based on maternal weight. (See ['Antibiotic prophylaxis'](#) above.)
 - Pneumatic compression devices should be used to prevent postpartum VTE in all women undergoing cesarean delivery, and both mechanical and pharmacologic thromboprophylaxis should be administered to women at high risk of VTE. (See ["Cesarean delivery: Preoperative planning and patient preparation", section on 'Thromboembolism prophylaxis'.](#))

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Topic 433 Version 102.0

GRAPHICS

Classification of body mass index

Underweight – BMI <18.5 kg/m ²
Normal weight – BMI ≥18.5 to 24.9 kg/m ²
Overweight – BMI ≥25 to 29.9 kg/m ²
Obesity – BMI ≥30 kg/m ²
Obesity class I – BMI 30 to 34.9 kg/m ²
Obesity class II – BMI 35 to 39.9 kg/m ²
Obesity class III – BMI ≥40 kg/m ² (also referred to as severe, extreme, or massive obesity)

Body mass index (BMI) classifications are based upon risk of cardiovascular disease. These classifications for BMI have been adopted by the National Institutes of Health (NIH) and World Health Organization (WHO) for Caucasian, Hispanic, and black individuals. Because these cut-offs underestimate risk in the Asian and South Asian population, WHO and NIH guidelines for Asians define overweight as a BMI between 23 and 24.9 kg/m² and obesity as a BMI >25 kg/m².

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Graphic 97661 Version 2.0

Results of multivariable logistic regression analysis showing the relation between obesity and selected maternal outcomes, singleton deliveries, Nova Scotia, 1988 to 2002

Maternal morbidity	Moderate obesity (n = 9355)	
	Adjusted OR*	95% CI
Antepartum		
PIH	2.38	2.24-2.52
Severe PIH (including HELLP)	1.56	1.35-1.8
Gestational diabetes	2.8	2.54-3.08
Intrapartum		
Very PTB (<32 weeks)	0.89	0.7-1.12
PTB (<37 weeks)	0.96	0.87-1.05
Post-term (>41 weeks)	1.18	1.08-1.28
Labor induction	1.94	1.86-2.04
Cesarean delivery	1.6	1.53-1.67
Anesthesia complications	1.18	1-1.39
Augmentation	1.25	1.18-1.33
Prolonged second stage (>3 hours)	0.96	0.88-1.05
Shoulder dystocia	1.51	1.32-1.74
Third- or fourth-degree laceration ¶	0.9	0.72-1.11
Postpartum		
Postpartum hemorrhage	1.12	1.02-1.22
Pyrexia	1.07	0.95-1.19
Puerperal morbidity	1.12	0.9-1.4
Endometritis	1.04	0.77-1.42
Urinary tract infection	1.15	0.95-1.39
Wound infection	1.67	1.38-2
Wound dehiscence	1.85	1.1-3.11
Prolonged hospital stay (>3 days)	0.89	0.85-0.93

Comparison group = 79,005 non-obese women.

OR: odds ratio; PIH: pregnancy-induced hypertension; HELLP: syndrome of hemolysis, elevated liver enzymes, and low platelets; PTB: preterm birth.

* Adjusted for maternal age, marital status, smoking, parity, and socioeconomic status.

¶ Third- or fourth-degree laceration limited to those delivered vaginally.

Data from: Robinson HE, O'Connell CM, Joseph KS, McLeod NL. Maternal outcomes in pregnancies complicated by obesity. *Obstet Gynecol* 2005; 106:1357.

Graphic 80972 Version 6.0

Obstetric complications by maternal body mass index

Outcome	Control (n = 13,752), percent	Obese (n = 1473), percent	Morbidly obese (n = 877), percent
Gestational hypertension	4.8	10.2	12.3
Preeclampsia	2.1	3.0	6.3
Gestational diabetes	2.3	6.3	9.5
Preterm premature rupture of membranes	1.7	2.1	2.2
Preterm delivery	3.3	4.0	5.5
Fetal growth restriction	1.1	1.0	0.8
Birth weight >4000 g	8.3	13.3	14.6
Birth weight >4500 g	1.0	2.1	2.6
Placenta previa	0.6	0.8	0.5
Placental abruption	0.8	0.8	0.8
Operative vaginal delivery	10.5	8.5	11.1

Modified from: Weiss JL, Malone FD, Emig D, et al. Obesity, obstetric complications, and cesarean delivery rate: A population-based screening study. Am J Obstet Gynecol 2004; 190:1091.

Graphic 59438 Version 6.0

Recommendations for total and rate of weight gain during pregnancy by prepregnancy BMI

Pregpregnancy BMI	Total weight gain		Rates of weight gain* second and third trimester	
	Range in kg	Range in lb	Mean (range) in kg/week	Mean (range) in lb/week
Underweight (<18.5 kg/m ²)	12.5 to 18	28 to 40	0.51 (0.44 to 0.58)	1 (1 to 1.3)
Normal weight (18.5 to 24.9 kg/m ²)	11.5 to 16	25 to 35	0.42 (0.35 to 0.50)	1 (0.8 to 1)
Overweight (25.0 to 29.9 kg/m ²)	7 to 11.5	15 to 25	0.28 (0.23 to 0.33)	0.6 (0.5 to 0.7)
Obese (≥30.0 kg/m ²)	5 to 9	11 to 20	0.22 (0.17 to 0.27)	0.5 (0.4 to 0.6)

BMI: body mass index.

* Calculations assume a 0.5 to 2 kg (1.1 to 4.4 lb) weight gain in the first trimester.

Weight Gain During Pregnancy: Reexamining the Guidelines. Institute of Medicine (US) and National Research Council (US) Committee to Reexamine IOM Pregnancy Weight Guidelines, Rasmussen KM, Yaktine AL (Eds), National Academies Press (US), The National Academies Collection: Reports funded by National Institutes of Health, Washington (DC) 2009. Reprinted with permission from the National Academies Press, Copyright © 2009 National Academy of Sciences.

Graphic 75820 Version 17.0

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