Published in final edited form as:
Am J Perinatol. 2014 March ; 31(3): 249–256. doi:10.1055/s-0033-1347363.

The Effect of Maternal Body Mass Index on Perinatal Outcomes in Women with Diabetes

Nicole E. Marshall, MD, MCR[1], **Camelia Guild, MPH**[2], **Yvonne W. Cheng, MD, MPH**[3], **Aaron B. Caughey, MD, PhD**[1,*], and **Donna R. Halloran, MD, MSPH**[2,*]

[1]Department of Obstetrics and Gynecology, Oregon Health and Science University, Portland, Oregon

[2]Department of Pediatrics, Saint Louis University, St. Louis, Missouri

[3]Department of Obstetrics, Gynecology and Reproductive Sciences, University of California, San Francisco, San Francisco, California

Abstract

Objective—To determine the effect of increasing maternal obesity, including superobesity (body mass index [BMI] ≥ 50 kg/m^2), on perinatal outcomes in women with diabetes.

Study Design—Retrospective cohort study of birth records for all live-born nonanom-alous singleton infants ≥ 37 weeks' gestation born to Missouri residents with diabetes from 2000 to 2006. Women with either pregestational or gestational diabetes were included.

Results—There were 14,595 births to women with diabetes meeting study criteria, including 7,082 women with a BMI > 30 kg/m^2 (48.5%). Compared with normal-weight women with diabetes, increasing BMI category, especially superobesity, was associated with a significantly increased risk for preeclampsia (adjusted relative risk [aRR] 3.6, 95% confidence interval [CI] 2.5, 5.2) and macrosomia (aRR 3.0, 95% CI 1.8, 5.40). The majority of nulliparous obese women with diabetes delivered via cesarean including 50.5% of obese, 61.4% of morbidly obese, and 69.8% of superobese women. The incidence of primary elective cesarean among nulliparous women with diabetes increased significantly with increasing maternal BMI with over 33% of morbidly obese and 39% of superobese women with diabetes delivering electively by cesarean.

Conclusion—Increasing maternal obesity in women with diabetes is significantly associated with higher risks of perinatal complications, especially cesarean delivery.

Keywords

cesarean; diabetes; obesity; superobesity

Obesity is a recognized risk factor for the development of diabetes,[1] even among pregnant women.[2] In 2007 to 2008, 34% of women of childbearing age (20 to 39 years) in the United States met obesity criteria (body mass index [BMI] ≥ 30 kg/m^2).[3] In addition to diabetes, obese women are more likely than normal-weight women to suffer miscarriage,

Address for correspondence Nicole E. Marshall, MD, MCR, 3181 SW Sam Jackson Park Rd., code L458, Portland, OR 97239, (marshani@ohsu.edu).
*Donna R. Halloran and Aaron B. Caughey served as co–senior authors.

This work was presented at the 58th Annual Meeting of the Society for Gynecologic Investigation in Miami Beach, Florida, March 16 to 19, 2011 as an oral presentation—Abstract #O-034.

preeclampsia, cesarean delivery, fetal growth abnormalities, stillbirth, and congenital anomalies.[4]

Pregnant women with diabetes are at risk for similar perinatal complications, including preeclampsia, cesarean delivery, macrosomia, and congenital anomalies,[5,6] and obese women with diabetes are at higher risk for obstetric complications than obese women without diabetes.[7] It has been postulated that the effects of obesity on pregnancy are due, at least in part, to increased insulin resistance,[8] as is also found in diabetes, which may account for some of the common pregnancy complications associated with both obesity and diabetes. However, it is unclear whether increasing obesity among women with diabetes is associated with worse perinatal outcomes compared with normal-weight women with diabetes.

Limited studies have attempted to examine the effect of increasing maternal obesity on perinatal outcomes in women with diabetes.[9–12] Although the evidence is clear that obesity increases the risk of pregnancy complications, study results have varied as to whether obese women with diabetes are at increased risk for complications compared with their normal-weight diabetic counterparts[9,10] or whether increasing BMI class increases the risks.[11,12] These prior studies were hampered by limited sample sizes, restriction to one type of diabetes, and/or comparisons only between obese and normal-weight women.

The objective of this study was to determine the effect of increasing maternal obesity (BMI > 30 kg/m^2), including morbid obesity (BMI 40 to 49.9 kg/m^2) and superobesity (BMI ≥ 50 kg/m^2), on perinatal outcomes in women with diabetes compared with normal-weight women with diabetes (BMI 18.5 to 24.9 kg/m^2). In addition to the risks associated with diabetes in pregnancy, we hypothesized that, compared with normal-weight women with diabetes, increasing maternal prepregnancy BMI is associated with (1) increased risk of maternal complications of pregnancy including preeclampsia and cesarean delivery, (2) greater risk of fetal growth abnormalities including macrosomia, and (3) greater risk of infant complications including neonatal hypoglycemia.

Materials and Methods

This is a population-based retrospective cohort study of all live-born singleton infants ≥ 37 weeks' gestation born to Missouri residents between January 1, 2000, and December 31,2006 (n =502,452). Data was obtained from Missouri Vital Records, which includes birth certificate records linked to hospital discharge information for the available period of 2000 to 2006. As the Missouri birth certificate fails to reliably differentiate between pregestational and gestational diabetes, all women with diabetes were included.[13] Fetuses with major congenital anomalies were excluded (n = 293, 2.0%), and inclusion was limited to term infants to avoid confounding of neonatal outcomes due to complications associated with anomalies and/or prematurity.

The primary predictor of interest was maternal BMI. BMI is based on birth certificate documentation of prepregnancy weight and height, which is typically based on self-report and generally considered to be valid.[14] BMI was calculated by weight in kilograms divided by height in meters squared. The World Health Organization (WHO) BMI categories were followed: normal (18.5 to 24.9 kg/m^2), overweight (25 to 29.9 kg/m^2), and obese (≥ 30 kg/m^2).[15,16] The WHO separates obesity into three classes—class I (30 to 34.9 kg/m^2), class II (35.0 to 39.9 kg/m^2) and class III (≥ 40 kg/m^2). As the primary study objective was to determine the impact of increasing obesity in women with diabetes on perinatal outcomes, we combined class I and II as obese (30 to 39.9 kg/m^2) and separated class III into morbid obesity (40 to 49.9 kg/m^2) and superobesity, defined as BMI ≥ 50 kg/m^2.[17] Normal and overweight categories were unchanged.

The primary outcomes of interest were mode of delivery, preeclampsia, macrosomia (birth weight > 4,500 g), and composite neonatal morbidity, which included low Apgar score (<7 at 5 minutes), birth trauma, infection, neonatal hypoglycemia, respiratory distress syndrome, seizures, neonatal length of stay > 5 days, and/or meconium aspiration syndrome. If a diagnosis such as preeclampsia, birth trauma, or respiratory distress syndrome was documented in the birth certificate, then the condition was considered present. Mode of delivery is categorized on the Missouri birth certificate record as vaginal, operative vaginal, vaginal birth after previous cesarean, repeat cesarean, primary elective cesarean, and primary emergency cesarean. Operative vaginal delivery includes use of outlet/low forceps, mid/high forceps, and vacuum extraction. Although indications for the method of delivery may be poorly identified in birth certificates, studies have shown that the actual method of delivery is accurately reported.[18,19] Hospitals in Missouri are provided with reporting instructions that define a primary elective cesarean as “a first-time cesarean section planned for in the future” and an emergency cesarean as “a first-time cesarean section done out of medical necessity because it is believed that further delay in delivery would seriously compromise the fetus, the mother, or both and/or vaginal delivery is unlikely to be accomplished safely.”[13]

Various maternal sociodemographic characteristics have been shown to be associated with maternal obesity and were evaluated as potential confounders in this study. Maternal education was categorized as high, average, or low based on age and years of education.[20] Greater than 12 years of education was considered high, regardless of maternal age. The R-GINDEX was used to categorize prenatal care based on initiation of care, total number of visits, and gestational age at delivery: no care, inadequate, adequate, intermediate, intensive, or missing.[21] Smoking status was determined by maternal self-report on birth certificate records.

Statistical Analysis

Bivariate analyses were completed using χ^2 tests. Outcomes were assessed using Cochrane Mantel-Haenszel test for linear trend and multivariable logistic regression for adjusted relative risk (aRR) and 95% confidence intervals (CI). Regression models were adjusted for maternal race/ethnicity, age, education, marital status, smoking status, level of prenatal care, insurance status (Medicaid versus private/other), parity (nulliparous versus multiparous), chronic hypertension, repeat cesarean, and primary elective cesarean.

All analyses were completed using SAS version 9.2 (SAS Institute Inc., Cary, North Carolina, United States). Approval for human subject research and a waiver of informed consent were received from the Institutional Review Board at Saint Louis University and the Missouri Department of Health and Senior Services, Section for Epidemiology for Public Health Practice.

Results

There were 14,595 births to women with diabetes meeting study criteria. Of them, 5,020 (34.4%) women were obese (BMI 30 to 39.9 kg/m^2), 1,732 (11.9%) were morbidly obese (BMI 40 to 49.9 kg/m^2), and 330 (2.3%) were superobese (BMI ≥ 50 kg/m^2). Increasing BMI among women with diabetes was associated with African American race, Medicaid insurance, single marital status, low education, increased parity, and chronic hypertension (Table 1).

Preeclampsia increased significantly with increasing maternal BMI (Table 2). Neonatal complications including macrosomia, hypoglycemia, length of stay > 5 days, and composite morbidity increased significantly with increased maternal BMI, but there was no significant

difference in birth trauma. Among superobese women with diabetes, almost 15% developed preeclampsia and 34.2% of infants experienced one or more neonatal morbidities.

Increasing maternal obesity was significantly associated with a decreased risk for vaginal delivery and an increased risk for cesarean delivery, including primary elective cesarean. Overall, only 27.3% of superobese women with diabetes delivered vaginally compared with 60.3% of normal-weight women with diabetes. When limited to nulliparous women with diabetes, only normal-weight and overweight women delivered vaginally (spontaneous or assisted) over half of the time (68% and 56%, respectively) versus 29% of superobese women with diabetes. The incidence of primary elective cesarean increased significantly with increasing maternal BMI with over 33% of morbidly obese and 39% of superobese women with diabetes delivered electively by cesarean. Among multiparous women with diabetes, 10.4% of superobese women with a prior vaginal birth required a primary emergency cesarean during labor versus 3.7% of normal-weight women. Among multiparous superobese women, 49.6% were delivered via repeat cesarean.

After adjusting for confounders including chronic hypertension, increasing BMI category remained significantly associated with increased risk for cesarean delivery, preeclampsia, and macrosomia compared with normal-weight women with diabetes, and the risk for neonatal hypoglycemia and composite neonatal morbidity also increased for each elevated BMI group except for overweight women with diabetes (Table 3). Superobese women with diabetes were at significantly higher risk for preeclampsia (aRR 3.6, 95% CI 2.5, 5.2) compared with normal-weight women with diabetes. Infants of superobese women with diabetes were also at increased risk for macrosomia (aRR 3.0, 95% CI 1.8, 5.4) and composite neonatal morbidity (aRR 2.0, 95% CI 1.6, 2.6) compared with infants of normal-weight women with diabetes. Among women with diabetes, increasing maternal BMI was protective against low birth weight (LBW; < 2500 g) for overweight, obese, and morbidly obese compared with normal-weight women. Among nulliparous women with diabetes, increasing maternal BMI was significantly associated with an increased risk of cesarean delivery compared with normal-weight women, especially for superobese nulliparas (adjusted odds ratio 5.5, 95% CI 3.4, 8.7). Overweight multiparous women were not at increased risk of cesarean delivery, but all classes of obese women with diabetes were at increased risk for cesarean delivery compared with normal-weight women.

A dose–response relationship for risk of cesarean delivery was seen with worsening obesity in women with diabetes, although there was no difference between nulliparous super and morbidly obese women (Table 4). Compared with nulliparous obese women with diabetes, morbidly obese women with diabetes were 1.6 times more likely and superobese women with diabetes were 2.4 times more likely to have a cesarean delivery. Compared with morbidly obese women with diabetes, superobese women with diabetes were 1.5 times more likely to develop preeclampsia and 2.3 times more likely to have a low Apgar score. Although morbidly obese women with diabetes were more likely to have fetal macrosomia than obese women with diabetes (aRR 1.3), there were no other significant differences in risk for macrosomia or low birth weight between obese, morbidly obese, and superobese women with diabetes.

Discussion

Elevated prepregnancy BMI in women with diabetes increases the risk of maternal and infant complications in a dose–response fashion. Among women with diabetes, superobese women experienced the highest rates of perinatal complications including a 67% incidence of cesarean delivery, 34.2% composite neonatal morbidity, 14.6% preeclampsia, and 7.9% macrosomia.

Mode of delivery was significantly impacted by maternal BMI with the incidence of cesarean delivery rising significantly with increasing BMI in women with diabetes and the incidence of operative vaginal delivery decreasing significantly for overweight and obese women with diabetes compared with normal-weight women with diabetes. Perhaps most concerning, less than half of obese nulliparous women with diabetes delivered vaginally and 28 to 40% delivered via elective cesarean. A recent review discussed many of the potential causes for increased cesarean delivery in obese women, including higher induction rates, slower progress during labor, comorbid conditions, and gestational weight gain.[22] As this was a retrospective study, coding limitations prevented the determination of the exact indication for each cesarean delivery. However, indications such as “arrest of dilation/descent” or “failure to progress” may be influenced by caregiver bias if providers, following increased inductions, fail to allow adequate time for labor progression, resulting in earlier interventions in obese women with diabetes. Providers may also be less likely to attempt operative vaginal delivery due to concerns for shoulder dystocia. Previous studies have shown higher rates of cesarean delivery when providers are aware of diabetic status, despite adjustment for macrosomia and other maternal risk factors.[23,24] In our study population, 67% of superobese women with diabetes were delivered via cesarean, including 49.6% of multiparous superobese women with diabetes who underwent repeat cesarean despite the fact that these women face significantly increased risks of anesthetic and operative complications compared with normal-weight women.[25–27] Patients and providers need to be aware of these potential biases and be prepared to discuss and manage labor complications. Future studies need to examine the exact indication for cesarean delivery and investigate the role of provider influence to determine whether mothers and infants are benefiting from these very high cesarean delivery rates.

We determined that the incidence of preeclampsia increased significantly with increasing maternal BMI in women with diabetes: 6% of women with diabetes with a BMI < 30 kg/m^2 developed preeclampsia versus 10.5% of women with diabetes with a BMI ≥ 30 kg/m^2. Obesity has been strongly linked to development of preeclampsia, with the risk doubling for every 5 to 7 kg/m^2 elevation in prepregnancy BMI.[28] The role of abnormal glucose tolerance in preeclampsia has been controversial, with one study finding no increased risk after adjusting for confounders including maternal BMI,[29] and others reporting a significantly increased risk of preeclampsia in women with abnormal glucose tolerance, whether or not they met gestational diabetes criteria.[30–32] Insulin resistance has been strongly implicated in the pathogenesis of preeclampsia.[8,33–36] Although diabetes is the classic example of insulin resistance, obesity is also highly associated with insulin resistance.[35] It is difficult to determine the impact of diabetes plus obesity on the risk of preeclampsia, but our data support an elevated risk for preeclampsia with increasing BMI in women with diabetes.

Diabetes and obesity each have potential long-term health implications for both mothers and their offspring. Women with gestational diabetes are at increased risk to develop type 2 diabetes, many within 5 to 10 years.[37] Birth weight is an important predictor of lifelong health as babies born at the extremes of birth weight, either macrosomic (> 4,500 g) or LBW (< 2,500 g), are at increased risk for chronic diseases later in life. Children born large for gestational age (LGA) to women with either diabetes or obesity are at increased risk of developing metabolic syndrome (obesity, hypertension, dys-lipidemia, and glucose intolerance) by age 11, and half of LGA infants born to women with gestational diabetes developed two or more components of metabolic syndrome by age 11.[38] In our study, superobese women with diabetes had the greatest incidence of fetal macrosomia (7.9%). However, unlike overweight, obese, and morbidly obese women with diabetes with a BMI of 25 to 49.9 who had lower risk of having an LBW infant compared with normal-weight women with diabetes, superobese women with diabetes, despite their significantly increased

weight, had no statistically significant difference in risk for LBW infants compared with normal-weight women. Understanding the link between maternal obesity, glucose status, and birth weight has important implications for decreasing the chronic disease risk for infants born to obese mothers with diabetes.

Limitations of this study include the use of birth certificate and hospital discharge data, which are dependent upon the original quality of the data entered and the opportunity for misclassification. Stillbirths were not assessed as they are not reliably and consistently reported to the state as part of the birth certificate records. Data were limited to 2000 to 2006 as that is the currently available period due to delay in release of data because of extensive verification and may not reflect population changes since that time. As the Missouri birth certificate does not reliably classify the different types of diabetes (type 1, type 2, A1 gestational, and A2 gestational), we chose to combine all women with diabetes. Although this increases the generalizability of the results as women classified as having gestational diabetes may have undiagnosed type 2 diabetes and therefore would have received an incorrect designation,[39] we recognize the limitation of not being able to perform a subanalysis of the different types of diabetes and patient levels of glucose control. However, previous studies have shown similar perinatal outcomes regardless of type of diabetes.[40,41] We are unable to classify outcomes based on diabetic treatment method or compliance as it is also not reliably reported on the birth certificate. Because treatment method has been suggested to play a role in perinatal outcomes,[11] it will be an important factor to include in future studies, which are urgently needed due to the significant increases in obesity and diabetes during pregnancy.

Last, the potential for undercoding a diagnosis such as preeclampsia or neonatal hypoglycemia remains a concern. However, there is no reason to expect that undercoding would be biased by maternal BMI. Patients who were not coded properly would lead to an underrepresentation of the true incidence of these conditions, thus suggesting that the true differences were even larger than stated.

Among women with diabetes, obese women are at significantly increased risk of several pregnancy complications including cesarean delivery, preeclampsia, macrosomia, and composite neonatal morbidity compared with normal-weight women, and the risks increase with increasing maternal obesity. Weight loss prior to pregnancy may be important in preventing the development of gestational diabetes and avoiding the additional burden of diabetes and obesity. By showing an improvement in perinatal outcomes with decreasing obesity class, this study highlights the potential benefit of modest weight loss even in the presence of diabetes and reinforces the importance for providers and communities to encourage healthy weight and preconception weight loss to help decrease maternal and neonatal morbidity. These findings also call attention to the very high cesarean delivery rate, including elective cesarean, in obese women with diabetes, especially women with a BMI > 40 kg/m^2, and emphasize the importance of examining this trend to determine whether the outcomes for these mothers and infants are improved or whether they are experiencing increased morbidity without significant benefit.

Acknowledgments

This work was supported by National Institutes of Health grants KL2RR024141 06 (N.E.M.), KL2RR024994 (D.R.H.), and K08 HD061484-01 (D.R.H.).

References

1. Hossain P, Kawar B, El Nahas M. Obesity and diabetes in the developing world—a growing challenge. N Engl J Med. 2007; 356:213–215. [PubMed: 17229948]

2. Baci Y, Üstüner I, Keskin HL, Ersoy R, Avşar AF. Effect of maternal obesity and weight gain on gestational diabetes mellitus. Gynecol Endocrinol. 2013; 29:133–136. [PubMed: 23110595]
3. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999–2008. JAMA. 2010; 303:235–241. [PubMed: 20071471]
4. American College of Obstetricians and Gynecologists. ACOG practice bulletin no. 105: bariatric surgery and pregnancy. Obstet Gynecol. 2009; 113:1405–1413. [PubMed: 19461456]
5. Yang J, Cummings EA, O'connell C, Jangaard K. Fetal and neonatal outcomes of diabetic pregnancies. Obstet Gynecol. 2006; 108(3 Pt 1):644–650. [PubMed: 16946226]
6. Casey BM, Lucas MJ, Mcintire DD. Pregnancy outcomes in women with gestational diabetes compared with the general obstetric population. Obstet Gynecol. 1997; 90:869–873. [PubMed: 9397092]
7. Knight KM, Pressman EK, Hackney DN, Thornburg LL. Perinatal outcomes in type 2 diabetic patients compared with non-diabetic patients matched by body mass index. J Matern Fetal Neonatal Med. 2012; 25:611–615. [PubMed: 21728737]
8. Roberts JM, Gammill H. Insulin resistance in preeclampsia. Hypertension. 2006; 47:341–342. [PubMed: 16446385]
9. Roman AS, Rebarber A, Fox NS, et al. The effect of maternal obesity on pregnancy outcomes in women with gestational diabetes. J Matern Fetal Neonatal Med. 2011; 24:723–727. [PubMed: 21366395]
10. Ray JG, Vermeulen MJ, Shapiro JL, Kenshole AB. Diabetes Endocrine Pregnancy Outcome Study in Toronto. Maternal and neonatal outcomes in pregestational and gestational diabetes mellitus, and the influence of maternal obesity and weight gain: the DEPOSIT study. QJM. 2001; 94:347–356. [PubMed: 11435630]
11. Langer O, Yogev Y, Xenakis EM, Brustman L. Overweight and obese in gestational diabetes: the impact on pregnancy outcome. Am J Obstet Gynecol. 2005; 192:1768–1776. [PubMed: 15970805]
12. Yogev Y, Langer O. Pregnancy outcome in obese andmorbidly obese gestational diabetic women. Eur J Obstet Gynecol Reprod Biol. 2008; 137:21–26. [PubMed: 17517462]
13. Brockert, J. Recommended standard medical definitions for the U.S. standard certificate of live birth and the U.S. standard report of fetal death; Traverse City, MI. Presented at the 1990 annual meeting of the AVRHS; Jun 25–27. 1989 revision
14. Land G, Vaughan W. Birth certificate completion procedures and the accuracy of Missouri birth certificate data. J Am Med Rec Assoc. 1984; 55:31–34. [PubMed: 10310713]
15. World Health Organization. [Accessed May 14, 2013] Obesity and overweight fact sheet. Available from: http://www.who.int/mediacentre/factsheets/fs311/en
16. Institute of Medicine (US) and National Research Council. (US) Committee to Reexamine IOM Pregnancy Weight Guidelines. Washington DC: National Academics Press; 2009.
17. Mason EE, Doherty C, Maher JW, Scott DH, Rodriguez EM, Blommers TJ. Super obesity and gastric reduction procedures. Gastroenterol Clin North Am. 1987; 16:495–502. [PubMed: 3436656]
18. DiGiuseppe DL, Aron DC, Ranbom L, Harper DL, Rosenthal GE. Reliability of birth certificate data: a multi-hospital comparison to medical records information. Matern Child Health J. 2002; 6:169–179. [PubMed: 12236664]
19. Kahn EB, Berg CJ, Callaghan WM. Cesarean delivery among women with low-risk pregnancies: a comparison of birth certificates and hospital discharge data. Obstet Gynecol. 2009; 113:33–40. [PubMed: 19104357]
20. Petersen DJ, Alexander GR. Seasonal variation in adolescent conceptions, induced abortions, and late initiation of prenatal care. Public Health Rep. 1992; 107:701–706. [PubMed: 1454982]
21. Alexander GR, Cornely DA. Prenatal care utilization: its measurement and relationship to pregnancy outcome. Am J Prev Med. 1987; 3:243–253. [PubMed: 3452362]
22. Wispelwey BP, Sheiner E. Cesarean delivery in obese women: a comprehensive review. J Matern Fetal Neonatal Med. 2013; 26:547–551. [PubMed: 23130683]
23. Naylor CD, Sermer M, Chen E, Sykora K. Cesarean delivery in relation to birth weight and gestational glucose tolerance: patho-physiology or practice style? Toronto Trihospital Gestational Diabetes Investigators. JAMA. 1996; 275:1165–1170. [PubMed: 8609683]

24. Blackwell SC, Hassan SS, Wolfe HW, Michaelson J, Berry SM, Sorokin Y. Why are cesarean delivery rates so high in diabetic pregnancies? J Perinat Med. 2000; 28:316–320. [PubMed: 11031703]
25. Saravanakumar K, Rao SG, Cooper GM. The challenges of obesity and obstetric anaesthesia. Curr Opin Obstet Gynecol. 2006; 18:631–635. [PubMed: 17099334]
26. Gunatilake RP, Perlow JH. Obesity and pregnancy: clinical management of the obese gravida. Am J Obstet Gynecol. 2011; 204:106–119. [PubMed: 21284965]
27. Heit JA, Kobbervig CE, James AH, Petterson TM, Bailey KR, Melton LJ III. Trends in the incidence of venous thromboembolism during pregnancy or postpartum: a 30-year population-based study. Ann Intern Med. 2005; 143:697–706. [PubMed: 16287790]
28. O'Brien TE, Ray JG, Chan WS. Maternal body mass index and the risk of preeclampsia: a systematic overview. Epidemiology. 2003; 14:368–374. [PubMed: 12859040]
29. Joffe GM, Esterlitz JR, Levine RJ, et al. Calcium for Preeclampsia Prevention (CPEP) Study Group. The relationship between abnormal glucose tolerance and hypertensive disorders of pregnancy in healthy nulliparous women. Am J Obstet Gynecol. 1998; 179:1032–1037. [PubMed: 9790393]
30. Schmidt MI, Duncan BB, Reichelt AJ, et al. Brazilian Gestational Diabetes Study Group. Gestational diabetes mellitus diagnosed with a 2-h 75-g oral glucose tolerance test and adverse pregnancy outcomes. Diabetes Care. 2001; 24:1151–1155. [PubMed: 11423494]
31. Carr DB, Newton KM, Utzschneider KM, et al. Gestational diabetes or lesser degrees of glucose intolerance and risk of preeclampsia. Hypertens Pregnancy. 2011; 30:153–163. [PubMed: 21174585]
32. Hod, Yogev C Chen, et al. Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study Cooperative Research Group. Hyperglycemia and adverse pregnancy outcome (HAPO) study: Preeclampsia. Am J Obstet Gynecol. 2010; 202:e1–e7. [PubMed: 20207245]
33. Seely EW, Solomon CG. Insulin resistance and its potential role in pregnancy-induced hypertension. J Clin Endocrinol Metab. 2003; 88:2393–2398. [PubMed: 12788833]
34. Lorentzen B, Birkeland KI, Endresen MJ, Henriksen T. Glucose intolerance in women with preeclampsia. Acta Obstet Gynecol Scand. 1998; 77:22–27. [PubMed: 9492712]
35. Hauth JC, Clifton RG, Roberts JM, et al. Eunice Kennedy Shriver National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal insulin resistance and preeclampsia. Am J Obstet Gynecol. 2011; 204:e1–e6. [PubMed: 21458622]
36. Parretti E, Lapolla A, Dalfrà M, et al. Preeclampsia in lean normo-tensive normotolerant pregnant women can be predicted by simple insulin sensitivity indexes. Hypertension. 2006; 47:449–453. [PubMed: 16446386]
37. Kim C, Newton KM, Knopp RH. Gestational diabetes and the incidence of type 2 diabetes: a systematic review. Diabetes Care. 2002; 25:1862–1868. [PubMed: 12351492]
38. Boney CM, Verma A, Tucker R, Vohr BR. Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus. Pediatrics. 2005; 115:e290–e296. [PubMed: 15741354]
39. American Diabetes Association. Standards of medical care in diabetes—2011. Diabetes Care. 2011; 34(Suppl 1):S11–S61. [PubMed: 21193625]
40. Sacks DA, Chen W, Greenspoon JS, Wolde-Tsadik G. Should the same glucose values be targeted for type 1 as for type 2 diabetics in pregnancy? Am J Obstet Gynecol. 1997; 177:1113–1119. [PubMed: 9396904]
41. Sacks DA, Liu AI, Wolde-Tsadik G, Amini SB, Huston-Presley L, Catalano PM. What proportion of birth weight is attributable to maternal glucose among infants of diabetic women? Am J Obstet Gynecol. 2006; 194:501–507. [PubMed: 16458653]

Table 1

Population Characteristics in Women with Diabetes

	Maternal BMI (kg/m^2)				
	Normal (18.5–24.9), *n* (%)	**Overweight (25–29.9), *n* (%)**	**Obese (30–39.9), *n* (%)**	**Morbidly obese (40–49.9), *n* (%)**	**Superobese (≥ 50), *n* (%)**
Maternal race[a]					
Caucasian	3,091 (78.1)	2,577 (74.0)	3,924 (78.2)	1,361 (78.6)	235 (71.9)
African American	341 (8.6)	502 (14.4)	751 (15.0)	293 (16.9)	84 (25.7)
Hispanic	194 (4.9)	237 (6.8)	252 (5.0)	53 (3.1)	6 (1.8)
Asian/other	333 (8.4)	169 (4.8)	93 (1.9)	25 (1.4)	2 (0.6)
Maternal age (y)[a]					
< 18	48 (1.2)	29 (0.8)	20 (0.4)	0 (0.0)	0 (0.0)
18–34	3,026 (76.2)	2,696 (77.1)	3,830 (76.1)	1,363 (78.5)	266 (80.6)
≥ 35	898 (22.6)	772 (22.1)	1,180 (23.5)	373 (21.5)	64 (19.4)
Education[a]					
High	2,360 (60.0)	1,914 (55.1)	2,697 (54.0)	874 (50.7)	167 (51.2)
Average	1,101 (28.0)	1,054 (30.4)	1,707 (34.2)	638 (37.0)	114 (35.0)
Low	472 (12.0)	503 (14.5)	590 (11.8)	212 912.3)	45 (13.8)
Married[b]	2,968 (74.7)	2,493 (71.3)	3,730 (74.2)	1,253 (72.2)	225 (68.2)
Parity[a]					
0	1,651 941.8)	1,158 (33.3)	1,564 (31.3)	528 (30.6)	96 (29.5)
1	1,170 (29.6)	1,147 (33.0)	1,711 (34.2)	595 (34.5)	104 (31.9)
≥2	1,131 (28.6)	1,168 (33.6)	1,728 (34.5)	603 (34.9)	126 (38.6)
Smoking status					
Yes	584 (14.7)	545 (15.6)	828 (16.5)	276 (15.9)	53 (16.1)
No	3,361 (84.6)	2,938 (84.0)	4,179 (83.1)	1,451 (83.6)	276 (83.6)
Unknown	27 (0.7)	14 (0.4)	23 (0.5)	9 (0.5)	1 (0.3)
Medicaid[a]	1,358 (34.3)	1,355 (38.9)	2,127 (42.4)	820 (47.4)	179 (54.4)
Prenatal care utilization[a]					

	Maternal BMI (kg/m^2)				
	Normal (18.5–24.9), *n* (%)	**Overweight (25–29.9), *n* (%)**	**Obese (30–39.9), *n* (%)**	**Morbidly obese (40–49.9), *n* (%)**	**Superobese (≥ 50), *n* (%)**
Missing	62 (1.6)	48 (1.4)	62 (1.3)	27 (1.6)	7 (2.2)
None	4 (0.1)	7 (0.2)	5 (0.1)	0 (0.0)	0 (0.0)
Inadequate	95 (2.5)	102 (3.0)	108 (2.2)	38 (2.3)	13 (4.2)
Adequate	2,017 (52.1)	1,731 (51.0)	2,387 (48.7)	784 (46.5)	127 (40.6)
Intermediate	1,053 (27.2)	849 (25.0)	1,189 (24.3)	370 (21.9)	71 (22.7)
Intensive	644 (16.6)	658 (19.4)	1,146 (23.4)	469 (27.8)	95 (30.4)
Male infant	2,047 (51.5)	1,834 (52.4)	2,557 (50.8)	878 (50.6)	173 (52.4)
Chronic hypertension[a]	45 (1.1)	100 (2.9)	302 (6.0)	160 (9.2)	47 (14.2)

Abbreviation: BMI, body mass index.

[a] $p < 0.0001$.

[b] $p < 0.001$.

Table 2

Perinatal Outcomes in Women with Diabetes by Parity Across Maternal BMI Category

	Maternal BMI (kg/m^2)				
	Normal (18.5–24.9), *n* (%)	**Overweight (25–29.9), *n* (%)**	**Obese (30–39.9), *n* (%)**	**Morbidly obese (40–49.9), *n* (%)**	**Superobese (≥ 50), *n* (%)**
Maternal					
Nulliparous					
Vaginal delivery[a]	890 (53.9)	531 (45.9)	618 (39.5)	164 (31.1)	19 (19.8)
Operative vaginal delivery[a]	253 (15.3)	130 (11.2)	157 (10.0)	38 (7.2)	9 (9.4)
Primary elective cesarean[a]	256 (15.5)	264 (22.8)	439 (28.1)	179 (33.9)	38 (39.6)
Primary emergency cesarean[a]	252 (15.3)	231 (20.0)	350 (22.4)	145 (27.5)	29 (30.2)
Multiparous					
Vaginal delivery[a]	1,494 (64.9)	1,369 (59.1)	1,732 (50.4)	467 (39.0)	71 (30.9)
Operative vaginal delivery	108 (4.7)	95 (4.1)	147 (4.3)	53 (4.4)	9 (3.9)
Vaginal birth after cesarean[b]	49 (2.1)	69 (3.0)	63 (1.8)	20 (1.7)	1 (0.4)
Primary elective cesarean[a]	112 (4.9)	118 (5.1)	254 (7.4)	122 (10.2)	11 (4.8)
Primary emergency cesarean[a]	85 (3.7)	90 (3.9)	173 (5.0)	88 (7.4)	24 (10.4)
Repeat cesarean[a]	453 (19.7)	574 (24.8)	1,070 (31.1)	448 (37.4)	114 (49.6)
Preeclampsia[a]	182 (4.6)	267 (7.6)	513 (10.2)	183 (10.5)	48 (14.6)
Neonatal					
Macrosomia[a]	65 (1.6)	135 (3.9)	222 (4.4)	111 (6.4)	26 (7.9)
Low birth weight[b]	120 (3.0)	72 (2.1)	105 (2.1)	25 (1.4)	11 (3.3)
Birth trauma[a]	157 (4.0)	144 (4.1)	170 (3.4)	68 (3.9)	14 (4.2)
Neonatal hypoglycemia[a]	406 (10.2)	408 (11.7)	764 (15.2)	299 (17.2)	75 (22.7)
Length of stay > 5 d[a]	156 (3.9)	166 (4.8)	275 (5.5)	141 (8.1)	38 (11.5)
Low Apgar[b]	31 (0.8)	42 (1.2)	41 (0.8)	26 (1.5)	10 (3.0)
Composite morbidity[a,c]	685 (17.3)	675 (19.3)	1,132 (22.5)	457 (26.3)	113 (34.2)

Abbreviation: BMI, body mass index.

[a] $p < 0.0001$.

[b] $p < 0.001$.

[c] Low Apgar score (<7 at 5 min), birth trauma, infection, neonatal hypoglycemia, respiratory distress syndrome, seizures, neonatal length of stay > 5 d, and/or meconium aspiration syndrome.

Table 3

Adjusted Risk of Adverse Perinatal Outcomes by Maternal BMI Category

	Maternal BMI (kg/m^2)			
	Overweight (25–29.9), aRR[a] (95% CI)	**Obese 30.39.9), aRR[a] (95% CI)**	**Morbidly obese (40–49.9), aRR[a] (95% CI)**	**Superobese (≥ 50), aRR[a] (95% CI)**
Maternal				
Nulliparous				
Cesarean	1.7 (1.5, 2.0)[b]	2.3 (2.0, 2.6)[b]	3.6 (3.0, 4.5)[b]	5.5 (3.4, 8.7)[b]
Operative vaginal	0.7 (0.6, 0.9)[c]	0.7 (0.5, 0.8)[b]	0.5 (0.3, 0.6)[b]	0.7 (0.3,1.34)
Multiparous[d]				
Cesarean	1.1 (0.8, 1.4)	1.4 (1.1, 1.8)[c]	2.6 (1.9, 3.5)[b]	4.7 (2.8, 7.7)[b]
Operative vaginal	0.9 (0.7, 1.2)	1.0 (0.7, 1.3)	1.1 (0.8, 1.5)	1.0 (0.5, 2.0)
Preeclampsia	1.8 (1.5, 2.2)[b]	2.5 (2.1, 3.0)[b]	2.5 (2.0, 3.2)[b]	3.6 (2.5, 5.2)[b]
Neonatal				
Macrosomia	2.0 (1.5, 2.7)[b]	2.0 (1.5, 2.7)[b]	2.7 (2.0, 3.7)[b]	3.0 (1.8, 5.0)[b]
Low birth weight	0.7 (0.5, 0.9)[c]	0.7 (0.5, 0.9)[c]	0.4 (0.3, 0.6)[c]	0.7 (0.4, 1.5)
Hypoglycemia	1.1 (0.9, 1.3)	1.4 (1.2, 1.6)[b]	1.5 (1.3, 1.8)[b]	1.9 (1.4, 2.6)[b]
Length of stay > 5 d	1.1 (0.9, 1.4)	1.2 (1.0, 1.5)	1.7 (1.3, 2.2)[b]	2.0 (1.3, 3.0)[c]
Low Apgar	1.6 (1.0, 2.6)	1.1 (0.7, 1.8)	1.9 (1.1, 3.3)[c]	4.1 (1.9, 8.6)[c]
Composite morbidity[e]	1.1 (1.0, 1.3)	1.3 (1.2, 1.4)[b]	1.5 (1.3, 1.7)[b]	2.0 (1.6, 2.6)[b]

Abbreviations: aRR, adjusted relative risk; BMI, body mass index; CI, confidence interval.

[a] Referent normal weight (BMI 18.5–24.9 kg/m^2), adjusted for maternal age, black race, average education, Medicaid, adequate prenatal care, smoking, marital status and chronic hypertension.

[b] $p < 0.0001$.

[c] $p < 0.05$.

[d] Also adjusted for repeat cesarean and primary scheduled cesarean.

[e] Low Apgar score (<7 at 5 min), birth trauma, infection, neonatal hypoglycemia, respiratory distress syndrome, seizures, neonatal length of stay > 5 d, and/or meconium aspiration syndrome.

Table 4

Perinatal Outcome—Comparisons Between Obesity Groups in Women with Diabetes

	Morbid versus obese, aRR (95% CI)	Super versus obese, aRR (95% CI)	Super versus morbid, aRR (95% CI)
Cesarean			
Nulliparous[a]	1.6 (1.3, 2.0)[e]	2.4 (1.5, 3.8)	1.5 (0.9, 2.5)
Multiparous[b]	1.8 (1.4, 2.3)[e]	3.2 (2.0, 5.2)[e]	1.7 (1.1, 2.9)[c]
Preeclampsia[b]	1.0 (0.9, 1.2)	1.5 (1.0, 2.0)[c]	1.5 (1.1, 2.2)[c]
Macrosomia[b]	1.3 (1.1, 1.7)[c]	1.5 (1.0, 2.4)	1.1 (0.7, 1.7)
Low birth weight[b]	0.6 (0.4, 1.0)	1.2 (0.6, 2.4)	2.0 (0.9, 4.3)
Length of stay > 5 d[b]	1.4 (1.1, 1.7)[d]	1.7 (1.1, 2.5)[d]	1.3 (0.8, 1.9)
Low Apgar[b]	1.7 (1.0, 2.8)	3.3 (1.6, 6.9)[d]	2.3 (1.1, 4.9)[c]
Neonatal hypoglycemia[b]	1.1 (0.9, 1.3)	1.4 (1.0, 1.8)[c]	1.2 (0.9, 1.7)
Composite morbidity[b]	1.2 (1.0, 1.3)[c]	1.6 (1.2, 2.1)[e]	1.3 (1.0, 1.7)

Abbreviations: aRR, adjusted relative risk; BMI, body mass index; CI, confidence interval.

[a] Adjusted for maternal age, black race, average education, Medicaid, adequate prenatal care, smoking, marital status and chronic hypertension.

[b] Also adjusted for repeat cesarean, primary scheduled cesarean.

[c] $p < 0.05$.

[d] $p < 0.01$.

[e] $p < 0.001$.