



POSTPRANDIAL VERSUS PREPRANDIAL BLOOD GLUCOSE MONITORING IN WOMEN WITH GESTATIONAL DIABETES MELLITUS REQUIRING INSULIN THERAPY

Pooja Gahlot*., Alok Gahlot and C.L. Gahlot

Abstract

Background: The fetuses of women with gestational diabetes mellitus are at risk for macrosomia and its attendant complications. The best method of achieving euglycemia in these women and reducing morbidity in their infants is not known. We compared the efficacy of postprandial and preprandial monitoring in achieving glycemic control in women with gestational diabetes.

Methods: We studied 66 women with gestational diabetes mellitus who required insulin therapy at 30 weeks of gestation or earlier. Both groups were also monitored with fasting blood glucose measurements. The goal of insulin therapy was a preprandial value of 60 to 105 mg per deciliter (3.3 to 5.9 mmol per liter) or a postprandial value of less than 140 mg per deciliter (7.8 mmol per liter). Obstetrical data and information on neonatal outcomes were collected.

Results: The mean (\pm SD) change in the glycosylated hemoglobin value was greater in the group in which postprandial measurements were used (-3.0 ± 2.2 percent vs. -0.6 ± 1.6 percent, $P < 0.001$) and the infants' birth weight was lower (3469 ± 668 vs. 3848 ± 434 g, $P = 0.01$). Similarly, the infants born to the women in the postprandial-monitoring group had a lower rate of neonatal hypoglycemia (3 percent vs. 21 percent, $P = 0.05$), were less often large for gestational age (12 percent vs. 42 percent, $P = 0.01$) and were less often delivered by cesarean section because of cephalopelvic disproportion (12 percent vs. 36 percent, $P = 0.04$) than those in the preprandial-monitoring group.

Conclusions: Adjustment of insulin therapy in women with gestational diabetes according to the results of postprandial, rather than preprandial, blood glucose values improves glycemic control and decreases the risk of neonatal hypoglycemia, macrosomia, and cesarean delivery.

Key Words: Gestational diabetes mellitus, Preprandial and post prandial blood glucose, Insulin

INTRODUCTION

Approximately 5 percent of all pregnancies are complicated by gestational diabetes mellitus, which increases both maternal and perinatal morbidity.¹ In treating women with this condition, many have advocated minimizing fluctuations in blood glucose concentrations to avert maternal hyperglycemia and thus decrease the risk of fetal hyperglycemia and its consequences, fetal hyperinsulinemia and excess fetal growth.²⁻⁵ However, despite early diagnosis and aggressive dietary and insulin therapy, perinatal morbidity among the infants born to women with gestational diabetes remains excessive, a fact that may or may not be attributed to suboptimal glycemic control.⁶⁻⁸

In the management of gestational diabetes, various methods of glucose monitoring have been proposed, including the measurement of fasting, preprandial, postprandial, and mean 24-hour blood glucose concentrations.⁹⁻¹¹ In a retrospective pilot study comparing the outcomes of pregnancy among women with gestational diabetes who were followed with preprandial or postprandial glucose measurements, we found that the women's glycosylated hemoglobin values were lower and that there was less macrosomia (defined as a birth weight greater than 4000 g) among their infants when treatment was based on the results of postprandial measurements.¹²

We conducted this prospective, randomized clinical trial to test the hypothesis that blood glucose monitoring at home with use of fasting and postprandial glucose values leads to better glycemic control in women with gestational diabetes who require insulin therapy than the combination of fasting and preprandial monitoring and improves perinatal outcomes by reducing the incidence of neonatal macrosomia and its attendant complications.

METHODS AND MATERIAL

This study was conducted at S.P. Medical College, Bikaner during the period of 1st September 2016 to 30th August 2017.

Study Subjects

At their initial prenatal visits, we screened pregnant women who had risk factors for gestational diabetes including obesity (body weight, >120 percent of ideal value), advanced age (>35 years), glycosuria on dipstick urinalysis ($>2+$), a history of diabetes in first-degree relatives, and a previous unexplained stillbirth or miscarriage. These women were also screened at 24 to 28 weeks of gestation if the results of the initial screening were normal. All women without such risk factors were initially screened at 24 to 28 weeks. The initial screening consisted of the measurement of plasma glucose one hour after the oral administration of 50 g of glucose.

If the plasma glucose value on the initial test was 140 mg per deciliter (7.8 mmol per liter) or higher but below 190 mg per deciliter (10.6 mmol per liter), a three-hour oral glucose-tolerance test (with 100 g of glucose) was performed. Gestational diabetes was diagnosed and dietary therapy was initiated if the women had any two of the following plasma glucose values: fasting, >105 mg per deciliter (5.9 mmol per liter); one hour after the administration of glucose, >190 mg per deciliter (10.6 mmol per liter); two hours, >165 mg per deciliter (9.2 mmol per liter); and three hours, >145 mg per deciliter (8.1 mmol per liter).¹³ Women with elevated fasting values at the time of the three-hour oral glucose-tolerance test were immediately started on insulin therapy. All others in this group were initially treated with diet and monitored with weekly fasting and postprandial (one hour after breakfast) measurements of plasma glucose; insulin therapy was initiated if the values exceeded 105 mg per deciliter or 140 mg per deciliter, respectively.

If the glucose value in the initial screening test was 190 mg per deciliter or higher, a three-hour glucose-tolerance test was not performed. Women with such high concentrations were classified as having gestational diabetes, and fasting and postprandial plasma glucose concentrations were measured in order to determine the need for insulin therapy. Women with fasting values above 105 mg per deciliter or postprandial values above 140 mg per deciliter began to receive insulin therapy.

Women with gestational diabetes were eligible for the study if they required insulin according to the criteria listed above at or before 30 weeks of gestation and were pregnant with a singleton fetus. Women with a history of diabetes before pregnancy or with preexisting hypertension, renal disease, or autoimmune disorders were excluded. The gestational age was estimated from the date of the last menstrual period or early ultrasound dating (at 10 to 20 weeks). Sixty-six women who met these criteria agreed to participate in the study, which was approved by the institutional review boards of the University of California at Irvine and Long Beach Memorial Medical Center.

Study Protocol

The women were assigned to one of two blood-glucose-monitoring protocols for the duration of their pregnancies; permuted-block randomization was used to ensure that equal numbers of women were assigned to each study group throughout its duration. The preprandial-monitoring plan required daily monitoring of fasting, preprandial, and bedtime capillary-blood glucose concentrations. The postprandial-monitoring plan required daily monitoring of blood glucose concentrations before breakfast (fasting) and one hour after each meal. All the women were evaluated weekly by the perinatal-diabetes team (consisting of an obstetrician, a dietitian, a nurse educator, and a counselor) unless complications of pregnancy, including poor glycemic control (usually indicated by hyperglycemia with persistently elevated blood glucose values after two weeks of outpatient therapy), preterm labor, or hypertension made hospitalization necessary. During any hospitalizations, the women were monitored according to their group assignment. A diet was prescribed with a daily allotment of 30 to 35 kcal per kilogram of ideal body weight, divided into three meals and one to three snacks; 40 to 45 percent of the energy was provided by carbohydrate. Calorie intake and food choices

were adjusted at the weekly visits according to weight gain and the blood glucose values measured at home by the women. All the women received split-dose therapy combining short-acting (regular) and intermediate-acting (NPH) human insulin; the doses were adjusted to achieve fasting blood glucose values of 60 to 90 mg per deciliter (3.3 to 5.0 mmol per liter) and preprandial values of 60 to 105 mg per deciliter (3.3 to 5.9 mmol per liter) or postprandial values below 140 mg per deciliter. The initial daily total insulin dose was 0.7 unit per kilogram of body weight for women in the first trimester of pregnancy, 0.8 unit per kilogram for those in the second trimester, and 0.9 unit per kilogram for those in the third trimester. Of the total insulin dose, two thirds was administered in the morning and one third in the evening, with the morning dose (given at about 8 a.m.) split into two thirds intermediate-acting and one third regular insulin and the evening dose given as one half regular insulin at dinner (approximately 6 p.m.) and one half intermediate-acting with dinner or at bedtime (approximately 9 p.m.).

The women measured their blood glucose concentrations using memory-based reflectance glucometers; all the values, as well as insulin doses and dietary intake, were recorded. Adjustments in the insulin doses were made if any of the values were consistently higher than the target blood glucose concentrations; efforts were made to normalize fasting blood glucose first. The insulin doses were usually changed by 2 to 4 units at a time. Total glycosylated hemoglobin was measured at the beginning of the study and in the month before delivery by the method described by Gould *et al.*¹⁴; a value of 8.0 percent or lower was considered normal in pregnancy

RESULTS

The two study groups were similar in age and physical characteristics (Table 1). The results of the one-hour 50-g glucose tests and the fasting plasma glucose values at the time of the three-hour glucose-tolerance tests, the duration of pregnancy at the time of the diagnosis of gestational diabetes requiring insulin treatment, and the week of gestation at the time of the initiation of insulin therapy were also similar (Table 1). Weight gain in both groups of women was similar (Table 2)

Table 1 Characteristics of Pregnant Women with Gestational Diabetes, According to Study Group.

	Preprandial group(N=33)	Postprandial group(N=33)
Age(year)	31+-6	29+-5
gravidity	4.3+-3	3.6+-2.2
Prepregnancy weight (kg)	79+-13	77+-13
Body mass index	29+-3.2	28.4+-3.8
Plasma glucose (mg/dl)At 1 hourFasting	216+-56 137+-38	214+-67 145+-50
Week of gestation at diagnosis	22.9+-7.5	21.8+-6.5
Week of gestation at start of insulin	24.3+-5.2	25.1+-5.1

A review of the patients' records of home blood glucose monitoring during the last four weeks of pregnancy (112 glucose samplings) revealed similar degrees of compliance (>95 percent) and achievement of target blood glucose values in the two groups (Table 2). However, the women in the postprandial-monitoring group received significantly more insulin than those in the preprandial-monitoring group (Table 2). Although the glycosylated hemoglobin values at the time insulin therapy was initiated were similar in the two groups,

Table 2 Obstetrical Data and Outcomes in Women with Gestational Diabetes, According to Study Group.)

	Preprandial	Postprandial	Relative risk(95% CI)	p value
Gestational age at delivery(week)	37.6+3.8	37.9+1.4		0.16
Maternal weight gain (kg)	10.7+5.4	10.5+5.4		0.94
Success in glycemic control(%)	86+4.1	88+5.2		0.62
Compliance with schedule (%)	98+1.9	95+2.2		0.76
Insulin dose				
Units/day	76.8+21.4	100.4+29.5		0.003
Units/kg	0.9+0.1	1.1+0.2		0.001
Glycosylated Hb				
Initial	8.6+2.3	8.9+3.2		0.55
Final	8.1+2.2	6.5+1.4		0.006
Change	-0.6+1.6	-3.0+2.2		<0.001
Cesarean section				
Total	13	8	1.6(0.8-3.4)	0.29
For CPD	12	4	3.0(1.1-8.3)	0.04
Perineal lacerations(3 rd or 4 th degree)	8	3	2.7(0.8-9.4)	0.16
Hospitalization for glycemic control	3	4	0.7(0.2-3.1)	1.00
Preeclampsia	2	2	1.0(0.1-6.7)	1.00

Table 3 Neonatal Outcomes, According to Study Group.

	Preprandial	Postprandial	Relative risk (95%CI)	P value
Birth weight(gm)	3848+434	3469+668		0.01
Large for gestational age	14	4	3.5(1.3-9.5)	0.01
Birth weight>4000gm	12	3	4.1(1.3-13.2)	0.01
Small for gestational age	0	1		1.00
Shoulder dystocia	6	1	6.0(0.8-47.1)	0.10
Neonatal hypoglycemia	7	1	7.0(0.9-53.8)	0.05
Hyperbilirubinemia	4	3	1.3(0.3-5.5)	1.00
Transient tachypnoea	2	2	1.0(0.1-6.7)	1.00
Apgar score at 5 min<8	3	1	3.0(0.3-27.4)	0.61
Still birth	1	0		1.00

the values before delivery were significantly lower in the postprandial-monitoring group; thus, the decrease in glycosylated hemoglobin values during treatment was significantly greater in this group.

The number of women who required hospitalization to optimize glycemic control during pregnancy was similar in the groups (Table 2). Preeclampsia requiring preterm delivery developed in two women in each group. No women were treated with a -adrenergic-agonist drug for preterm labor. There were no other medical complications. There was a trend toward a higher rate of cesarean deliveries in the preprandial-monitoring group, and there was a significant difference between the groups in the frequency of cesarean sections performed for cephalopelvic disproportion during labor or for suspected fetal macrosomia (36 percent in the preprandial-monitoring group vs. 12 percent in the postprandial-monitoring group, P = 0.04) (Table 2). More women in the preprandial-monitoring group were offered an elective cesarean section because the weight of the fetus, estimated by ultrasonography, was more than 4000 g; all these women delivered an infant with a confirmed birth weight greater than 4000 g. There was also a trend toward more third- and fourth-degree perineal lacerations during vaginal deliveries in the preprandial-monitoring group (24 percent, vs. 9 percent in the post-prandial-monitoring group). Despite similar gestational ages at delivery (Table 2), the mean (±SD) birth weight in the preprandial-monitoring group was significantly higher than that in the postprandial-monitoring group (3848±434 vs. 3469±668 g, P = 0.01) (Table 3). The proportion of infants who were large for gestational age (birth weight above the 90th percentile for

gestational age and sex, according to population-specific standards for California) was significantly higher in the preprandial-monitoring group (42 percent, vs. 12 percent in the postprandial-monitoring group; P = 0.01), as was the number of infants weighing more than 4000 g (36 percent vs. 9 percent, P = 0.01). There were more instances of shoulder dystocia during vaginal delivery in the preprandial-monitoring group (18 percent vs. 3 percent, P = 0.10). Although two infants in the preprandial-monitoring group and one in the postprandial-monitoring group were given a diagnosis of Erb's palsy, the palsy resolved before discharge. One infant in each group had a fracture (one of the clavicle and one of the humerus). Only one unexplained stillbirth occurred, and it was in the preprandial-monitoring group (Table 3).

More infants in the preprandial-monitoring group had hypoglycemia (glucose concentration, <30 mg per deciliter) requiring glucagon or dextrose infusion for treatment during the first four days after birth (21 percent vs. 3 percent, P = 0.05) (Table 3). There were no significant differences between the groups in the frequency of other neonatal complications.

DISCUSSION

The results of this study support the hypothesis that postprandial glucose monitoring, in combination with fasting blood glucose measurements, can significantly improve the outcomes of pregnancy in women with gestational diabetes who require insulin therapy. Previous studies of combined preprandial and postprandial glucose monitoring found an

association between fetal macrosomia and suboptimal glycemic control.^{7,16} In one study, blood glucose monitoring before meals in women with insulin-dependent diabetes mellitus did not provide an adequate indication of metabolic control or of the risk of macrosomia; the authors therefore recommended postprandial glucose monitoring in order to optimize glycemic control.¹⁷ In another study, macrosomia was related to postprandial but not to fasting blood glucose values.¹⁸

We found that compliance among patients was similar for both blood-glucose-monitoring plans. Although the adjustment of insulin doses may be simpler when preprandial glucose monitoring is used, we found that more stringent glycemic control could be achieved with postprandial monitoring. The hypoglycemic episodes during gestation that have been described in women who have insulin-dependent diabetes mellitus before pregnancy rarely occur in women with gestational diabetes, because of their hyperinsulinemic, insulin-resistant state after meals. Women in whom preprandial monitoring is used have their blood glucose concentrations measured only at times when they are least likely to be hyperglycemic.

Measurements of glycosylated hemoglobin have proved to be a useful index of long-term (four-to-six-week) glycemic control during pregnancy, and elevated values have been linked to fetal macrosomia.^{19,20} Our results indicate that with tighter glycemic control, a significant decrease in the frequency of neonatal macrosomia can be achieved. Moreover, postprandial glucose values may be a more sensitive indicator of carbohydrate intolerance than fasting or preprandial values, potentially allowing more aggressive insulin treatment.

Large-for-gestational-age infants are delivered in 15 to 45 percent of pregnancies complicated by diabetes.²¹ Gestational diabetes is strongly associated with maternal obesity, and considerable controversy exists as to whether macrosomia is attributable to maternal obesity, poor glycemic control, or both.²²⁻²⁵ Despite the similar body-mass indexes and weight gains during pregnancy in our study groups, significantly fewer infants who were large for gestational age or weighed more than 4000 g were born to the women in the postprandial-monitoring group. Since maternal weight was similar in the two groups, the differences are most readily attributable to differences in the degree of glycemic control. Infants with macrosomia who are born to women with diabetes have a disproportionately increased fetal trunk and shoulder size.²⁶ The decreased incidence of cesarean section for cephalopelvic disproportion, of shoulder dystocia, and of maternal perineal lacerations in the postprandial-monitoring group is thus not surprising.

Neonatal complications, including hypoglycemia, hyperbilirubinemia, and respiratory compromise, have been described in infants born to women with gestational diabetes who require insulin therapy, particularly those in whom glycemic control was poor.^{6,22} The decreased incidence of neonatal hypoglycemia in the infants born to the women in the postprandial-monitoring group is consistent with the better glycemic control documented in this group. There was also a trend toward a lower rate of hyperbilirubinemia in the infants of women in the postprandial-monitoring group. Some limitations of this study must be considered. First, the women were predominantly Hispanic. Race or ethnic group

has been reported to have an independent influence on birth weight and on the prevalence of gestational diabetes, with Hispanics at higher risk for both.²⁷ This factor may limit the extrapolation of our findings to the general population. Second, some of the women probably had previously undiagnosed non-insulin-dependent diabetes mellitus, because their diabetes was identified in early pregnancy. Third, the exclusion of women who started insulin therapy after 30 weeks of gestation increased the likelihood that we would find a difference in perinatal outcome between the groups. Earlier studies may have failed to show benefits of therapeutic intervention in women with gestational diabetes because glucose intolerance is often diagnosed after macrosomia is already apparent, since macrosomia may develop as early as 20 weeks of gestation. Conversely, women in whom gestational diabetes is diagnosed in early pregnancy may have more severe metabolic abnormalities that contribute to accelerated fetal growth. We excluded women with medical complications known to impair fetal growth in order to permit a more accurate assessment of our therapeutic intervention. Fourth, since this was a nonblinded study and some members of the health care team were aware of the hypothesis, bias in the clinical management and the assessment of perinatal outcomes could have been introduced. However, many of the physicians involved believed that preprandial glucose monitoring was as effective as postprandial glucose monitoring.

The fact that the demographic characteristics and details of clinical management in the two groups were similar, as was the degree of compliance with the glucose-monitoring schedules, allowed us to assess the effects of the intervention on perinatal outcomes. Although neonatal macrosomia and other complications are probably multifactorial in origin, in this predominantly Hispanic population of women with gestational diabetes requiring insulin therapy, postprandial glucose monitoring led to better glycemic control than preprandial monitoring. Better control of blood glucose concentrations, in turn, decreased both neonatal risks and perinatal complications.

References

1. Gabbe SG, Mestman JH, Freeman RK, Anderson GV, Lowensohn RI. Management and outcome of class A diabetes mellitus. *Am J Obstet Gynecol* 1977;127:465-469
CrossRef | Web of Science | Medline
2. Goldberg JD, Franklin B, Lasser D, et al. Gestational diabetes: impact of home glucose monitoring on neonatal birth weight. *Am J Obstet Gynecol* 1986;154:546-550
Web of Science | Medline
3. Drexel H, Bichler A, Sailer S, et al. Prevention of perinatal morbidity by tight metabolic control in gestational diabetes mellitus. *Diabetes Care* 1988;11:761-768
CrossRef | Web of Science | Medline
4. Langer O, Mazze R. The relationship between large-for-gestational-age infants and glycemic control in women with gestational diabetes. *Am J Obstet Gynecol* 1988;159:1478-1483
Web of Science | Medline
5. Jovanovic L, Druzin M, Peterson CM. Effect of euglycemia on the outcome of pregnancy in insulin-

- dependent diabetic women as compared with normal control subjects. *Am J Med* 1981;71:921-927
[CrossRef](#) | [Web of Science](#) | [Medline](#)
6. Metzger BE. Summary and recommendations of the Third International Workshop-Conference on Gestational Diabetes Mellitus. *Diabetes* 1991;40:Suppl 2:197-201
[CrossRef](#) | [Web of Science](#) | [Medline](#)
 7. Kitzmiller JL, Cloherty JP, Younger MD, et al. Diabetic pregnancy and perinatal morbidity. *Am J Obstet Gynecol* 1978;131:560-580
[Web of Science](#) | [Medline](#)
 8. Hod M, Merlob P, Friedman S, Schoenfeld A, Ovadia J. Gestational diabetes mellitus: a survey of perinatal complications in the 1980s. *Diabetes* 1991;40:Suppl 2:74-78
[Web of Science](#) | [Medline](#)
 9. Freinkel N. Of pregnancy and progeny. *Diabetes* 1980;29:1023-1035
[CrossRef](#) | [Web of Science](#) | [Medline](#)
 10. Jovanovic L, Peterson CM. Optimal insulin delivery for the pregnant diabetic patient. *Diabetes Care* 1982;5:Suppl 1:24-37
[Web of Science](#) | [Medline](#)
 11. Gabbe SG. Management of diabetes mellitus in pregnancy. *Am J Obstet Gynecol* 1985;153:824-828
[Web of Science](#) | [Medline](#)
 12. Major CA, de Veciana M, Morgan MA, Henry JA. Glucose monitoring in gestational diabetics requiring insulin: preprandial or postprandial? *Am J Obstet Gynecol* 1993;168:406-406 abstract.
 13. O'Sullivan JB, Mahan CM. Criteria for the oral glucose tolerance test in pregnancy. *Diabetes* 1964;13:278-285
[Web of Science](#) | [Medline](#)
 14. Gould BJ, Hall PM, Cook JGH. Measurement of glycosylated hemoglobin using an affinity chromatography method. *Clin Chim Acta* 1982;125:41-48
[CrossRef](#) | [Web of Science](#) | [Medline](#)
 15. Williams RL, Creasy RK, Cunningham GC, Hawes WE, Norris FD, Tashiro M. Fetal growth and perinatal viability in California. *Obstet Gynecol* 1982;59:624-632
[Web of Science](#) | [Medline](#)
 16. Willman SP, Leveno KJ, Guzick DS, Williams ML, Whalley PJ. Glucose threshold for macrosomia in pregnancy complicated by diabetes. *Am J Obstet Gynecol* 1986;154:470-475
[Web of Science](#) | [Medline](#)
 17. Jovanovic-Peterson L, Peterson CM, Reed GF, et al. Maternal postprandial glucose levels and infant birth weight: the Diabetes in Early Pregnancy Study. *Am J Obstet Gynecol* 1991;164:103-111
[Web of Science](#) | [Medline](#)
 18. Combs CA, Gunderson E, Kitzmiller JL, Gavin LA, Main EK. Relationship of fetal macrosomia to maternal postprandial glucose control during pregnancy. *Diabetes Care* 1992;15:1251-1257
[CrossRef](#) | [Web of Science](#) | [Medline](#)
 19. Hahm S, Kaplan S, Nitowsky HM. Hemoglobin A_{1c} levels in mothers of large birth weight infants. *Pediatr Res* 1983;17:315A-315A abstract.
 20. Edidin DV, Mennella J. Increased glycosylated hemoglobin (GHb) in maternal and cord blood of macrosomic infants of diabetic mothers (IDM). *Pediatr Res* 1983;17:288A-288A abstract.
 21. Lavin JP Jr, Lovelace DR, Miodovnik M, Knowles HC, Barden TP. Clinical experience with one hundred seven diabetic pregnancies. *Am J Obstet Gynecol* 1983;147:742-752
[Web of Science](#) | [Medline](#)
 22. Spellacy WN, Miller S, Winegar A, Peterson PQ. Macrosomia -- maternal characteristics and infant complications. *Obstet Gynecol* 1985;66:158-161
[Web of Science](#) | [Medline](#)
 23. Jarrett RJ. Reflections on gestational diabetes mellitus. *Lancet* 1981;2:1220-1221
[CrossRef](#) | [Web of Science](#) | [Medline](#)
 24. Maresh M, Beard RW, Bray CS, Elkeles RS, Wadsworth J. Factors predisposing to and outcome of gestational diabetes. *Obstet Gynecol* 1989;74:342-346
[Web of Science](#) | [Medline](#)
 25. Green JR, Schumacher LB, Pawson IG, Partridge JC, Kretchmer N. Influence of maternal body habitus and glucose tolerance on birth weight. *Obstet Gynecol* 1991;78:235-240
[Web of Science](#) | [Medline](#)
 26. Modanlou HD, Komatsu G, Dorchester W, Freeman RK, Bosu SK. Large-for-gestational-age neonates: anthropometric reasons for shoulder dystocia. *Obstet Gynecol* 1982;60:417-423
[Web of Science](#) | [Medline](#)
 27. Dooley SL, Metzger BE, Cho NH. Gestational diabetes mellitus: influence of race on disease prevalence and perinatal outcome in a U.S. population. *Diabetes* 1991;40:Suppl 2:25-29
