Increasing Prevalence of Gestational Diabetes Mellitus (GDM) Over Time and by Birth Cohort

Kaiser Permanente of Colorado GDM Screening Program

DANA DABELEA, MD, PHD[1]
JANET K. SNELL-BERGEON, MPH[1]
CYNTHIA L. HARTSFIELD, PHD[2]

KIMBERLY J. BISCHOFF, MSPH[2]
RICHARD F. HAMMAN, MD, PHD[1]
ROBERT S. MCDUFFIE, MD[3]

OBJECTIVE — The prevalence of gestational diabetes mellitus (GDM) varies in direct proportion with the prevalence of type 2 diabetes in a given population or ethnic group. Given that the number of people with diabetes worldwide is expected to increase at record levels through 2030, we examined temporal trends in GDM among diverse ethnic groups.

RESEARCH DESIGN AND METHODS — Kaiser Permanente of Colorado (KPCO) has used a standard protocol to universally screen for GDM since 1994. This report is based on 36,403 KPCO singleton pregnancies occurring between 1994 and 2002 and examines trends in GDM prevalence among women with diverse ethnic backgrounds.

RESULTS — The prevalence of GDM among KPCO members doubled from 1994 to 2002 (2.1–4.1%, $P < 0.001$), with significant increases in all racial/ethnic groups. In logistic regression, year of diagnosis (odds ratio [OR] and 95% CI per 1 year = 1.12 [1.09–1.14]), mother's age (OR per 5 years = 1.7 [1.6–1.8]) and ethnicity other than non-Hispanic white (OR = 2.1 [1.9–2.4]) were all significantly associated with GDM. Birth year remained significant (OR = 1.06, $P = 0.006$), even after adjusting for prior GDM history.

CONCLUSIONS — This study shows that the prevalence of GDM is increasing in a universally screened multiethnic population. The increasing GDM prevalence suggests that the vicious cycle of diabetes in pregnancy initially described among Pima Indians may also be occurring among other U.S. ethnic groups.

***Diabetes Care* 28:579–584, 2005**

G estational diabetes mellitus (GDM) is defined as glucose intolerance with onset or first recognition during pregnancy (1,2). Approximately 135,000 cases of GDM, representing on average 3–8% of all pregnancies (1), are diagnosed annually in the U.S (3). Marked variation in GDM prevalence among different racial/ethnic groups has been documented, with higher prevalence among Native-American, Asian, African-American, and Hispanic populations than among non-Hispanic whites (4–8,9).

Exposure to maternal hyperglycemia during pregnancy is associated with birth defects (10) and effects on childhood growth and glucose regulation (11–13). Among the Pima Indians, the population with the world's highest incidence and prevalence of type 2 diabetes in children (14) and adults (15), most of the increased prevalence of childhood type 2 diabetes in the last 30 years is attributable to increasing exposure to maternal diabetes during pregnancy (14).

There is a general impression that the prevalence of GDM has increased over time (1), along with the increase in the prevalence of obesity (16). To date, however, few studies have examined trends in GDM in populations other than Native Americans (5,14,17). Characterizing trends in GDM is critical for understanding some of the mechanisms responsible for the increasing rates of obesity and type 2 diabetes in youth.

We hypothesized that an increasing prevalence of GDM would exist in populations other than the Pima Indians. We analyzed GDM screening results among women of several ethnic groups who delivered singleton infants between 1994 and 2002 and were members of the Kaiser Permanente of Colorado (KPCO) health plan.

RESEARCH DESIGN AND
METHODS — The study was conducted at KPCO, a nonprofit staff-model health maintenance organization that serves ~380,000 members in the Denver metropolitan area. The study was approved by the KPCO Research Review Committee and Institutional Review Board. The cohort of women described below was identified through the KPCO perinatal database. The database was developed in 1992 and contains data that define delivery events for each woman represented, including perinatal, demographic, and behavioral information (18,19). GDM is coded as present if diag-

Table 1—*Characteristics of pregnancies screened between 1994 and 2002*

	1994–2002	1994	2002	P*
Pregnancies (*n*)	36,403	3,644	4,079	
Maternal age	27.9 ± 6.0	28.1 ± 6.0	28.3 ± 6.0	0.4
Race/ethnicity (%)				
Non-Hispanic white	21,444 (60.6)	2,516 (68.8)	2,373 (58.2)	<0.0001
Hispanic	5,920 (16.3)	396 (10.8)	831 (20.4)	<0.0001
African American	2,293 (6.3)	158 (4.3)	251 (6.2)	<0.0001
Asian	1,465 (4.0)	89 (2.4)	280 (6.6)	<0.0001
Other	498 (1.4)	59 (1.6)	4 (0.1)	<0.0001
Missing	4,178 (11.5)	441 (12.1)	340 (8.3)	<0.0001

Data are means ±SD or *n* (%). *2002 versus 1994.

nosed through the standard KPCO screening protocol (described below) and absent if screening was negative or not performed.

GDM screening
Since the late 1980s, KPCO has routinely screened for GDM in all nondiabetic pregnancies using a two-step standard protocol. At 24–28 weeks, all pregnant women without previously diagnosed diabetes are offered screening for GDM with a 1-h 50-g oral glucose tolerance test (OGTT). A value ≥140 mg/dl identifies patients who undergo a 3-h 100-g diagnostic OGTT. GDM is diagnosed when two or more glucose values during the diagnostic OGTT meet or exceed the criteria for a positive test, as recommended by the National Diabetes Data Group (NDDG) (20). Virtually all patients with a positive screening 1-h value undergo a diagnostic 3-h test, and no GDM diagnosis is made solely on the basis of a single positive test. Since 1994, the KPCO screening and diagnostic protocols have remained constant. The proportion of women undergoing this screening protocol was estimated on a random sample of 2,328 pregnant women who were KPCO members in 1996 and was found to be 98% (21). Between 2001 and 2002, 96% of all KPCO eligible pregnant women were screened for GDM.

Statistical analysis
Included in this analysis were all pregnant women who delivered singleton infants between 1994 and 2002 (*N* = 36,403) and who were screened for GDM as described above. Pregnancies ending before 20 weeks of gestation were not included. Births from women diagnosed with nongestational diabetes before the index

pregnancy were not included in the analysis (*n* = 140 over the study period). The annual prevalence of GDM was computed in four race/ethnic groups (non-Hispanic whites, Hispanics, African Americans, and Asians) and was age adjusted to the age distribution of KPCO pregnant women in 1994 using the direct method (22). Data are also presented as age-adjusted 3-year moving averages (seven time periods: 1994–1996, 1995–1997, etc.) for each race/ethnic group. This averaging method is used to smooth data and is particularly useful for examining time trends where fluctuations during shorter time periods make it difficult to determine whether trends exist (23,24). A χ^2 Cochran-Armitage linear trend test, adjusted for age, was used to test for time trends in GDM prevalence in each race/ethnic group (25). Pregnancies occurring among women whose race/ethnic backgrounds were "other" (*n* = 498 over the study period) and "missing" (*n* = 4,178 over the study period) were excluded from the race/ethnic-specific analyses but were included in the logistic regression models. Multiple logistic regression analysis was used to examine the independent effect of calendar time (year of GDM diagnosis) on GDM prevalence, controlling for maternal age, race/ethnicity, prior GDM diagnosis, parity, and gravidity. The effects of race/ethnicity on the prevalence of GDM were explored using three categories, with non-Hispanic whites as the reference group: *1*) non-Hispanic whites, *2*) other than non-Hispanic whites (including Hispanics, African Americans, Asians, and others), and *3*) missing. A potential interaction between time (year of GDM diagnosis) and race/ethnicity on the prevalence of GDM was tested by adding an interaction term in the regression

model. Finally, to estimate a potential cohort effect, age-specific prevalence of GDM in 2-year age-intervals was determined among four maternal birth cohorts (1946–1955, 1956–1965, 1966–1975, and 1976–1990) and multiple logistic regression analysis was used to examine the independent effect of birth cohort (per 10 years) on the prevalence of GDM, adjusted for confounders.

RESULTS — A total of 36,403 eligible pregnancies occurred among 30,216 women who were screened for GDM between 1 January 1994 and 31 December 2002. The mean maternal age of screened pregnancies (28 years) did not change over time; however, the proportion of pregnancies from minority women increased from 1994 to 2002 (Table 1). A total of 1,183 pregnancies were complicated by GDM between 1994 and 2002. Overall, the prevalence of GDM doubled from 2.1% in 1994 to 4.1% in 2002 (*P* < 0.001).

The age-adjusted prevalence of GDM is shown both as annual estimates and standard deviations (Table 2) and as 3-year moving averages (Fig. 1) for the four race/ethnic groups included in this analysis. The prevalence of GDM (3-year moving averages, Fig. 1) increased significantly from 1994–1996 to 2000–2002 among non-Hispanic whites (1.9 to 3.4%), Hispanics (2.8 to 5.1%), African Americans (2.5 to 4.6%), and Asians (6.3 to 8.6%). Every year from 1994 to 2002, the prevalence of GDM was significantly higher among minority women than among non-Hispanic white women (Table 2). Over the study period, pregnant women of racial/ethnic backgrounds other than non-Hispanic white had twofold higher prevalence of GDM than non-Hispanic whites (Table 3, models 2 and 3), independent of differences in maternal age, year of GDM diagnosis, and prior history of GDM. Pregnant women whose racial/ethnic background was unknown (missing) had GDM prevalence similar to that of non-Hispanic white women.

Table 3 shows the effect of calendar time on the prevalence of GDM. There was a 12% increase per year (95% CI = 9–14%; *P* < 0.0001) over the period from 1994 to 2002 (Table 3, model 1). The increase was independent and virtually unchanged (10% per year) after controlling for changes in maternal age and in the race/ethnicity mix of the study popu-

Table 2—*Annual race/ethnicity-specific age-adjusted prevalence (per 100) of gestational diabetes: 1994–2002*

Year pregnant	Non-Hispanic white		Hispanic		African American		Asian		P
	n†	Prevalence	n†	Prevalence	n†	Prevalence	n†	Prevalence	
1994	43	1.7 ± 0.26	11	2.8 ± 0.83	6	3.8 ± 1.52	7	7.9 ± 2.85	<0.001
1995	35	1.9 ± 0.32	5	1.6 ± 0.71	2	1.3 ± 0.98	6	5.8 ± 2.39	<0.001
1996	54	2.0 ± 0.27	20	3.4 ± 0.75	5	2.1 ± 0.94	8	5.4 ± 1.81	<0.001
1997	66	2.4 ± 0.29	22	3.2 ± 0.66	9	3.1 ± 1.01	7	4.4 ± 1.65	<0.001
1998	79	2.9 ± 0.32	27	3.4 ± 0.63	14	4.1 ± 1.08	9	5.6 ± 1.78	<0.001
1999	85	3.5 ± 0.37	34	4.9 ± 0.82	8	2.6 ± 0.94	2	2.2 ± 1.56	<0.001
2000	101	4.2 ± 0.41	39	4.8 ± 0.75	18	5.9 ± 1.34	23	11.5 ± 2.26	<0.001
2001	74	3.2 ± 0.37	41	5.1 ± 0.78	8	2.9 ± 0.99	21	8.8 ± 1.84	<0.001
2002	73	3.1 ± 0.35	45	5.4 ± 0.79	14	5.5 ± 1.44	19	6.8 ± 1.50	<0.001
P value (trend)		0.002		<0.001		<0.001		<0.001	

Data are given as percentage ± SD unless noted. *Other than non-Hispanic white versus non-Hispanic white; †number of pregnancies with GDM.

lation (Table 3, model 2). Moreover, this increase was still significant (6% per year) after additionally controlling for a previous history of GDM (the strongest risk factor for subsequent GDM in these models) in the subgroup of women with multiple pregnancies (Table 3, model 3). Parity and gravidity were not significantly associated with GDM and had no effect on the GDM increase over time (data not shown). No significant time-by-race interaction on the prevalence of GDM was noted, indicating that the increase in GDM over time was similar among minority and non-Hispanic white women.

Figure 2 shows age-specific (women's ages at diagnosis) prevalence estimates of GDM for four cohorts of pregnant women grouped according to their own birth period (birth cohorts): 1946–1955, 1956–1965, 1966–1975, and 1976–1990. For a given age at delivery, the prevalence was higher in younger generation women than in older cohorts, although the prevalence of GDM was similar in the most recent birth cohorts (1976–1990 and 1966–1975). In multiple logistic regression, the prevalence of GDM was significantly higher (OR per 10 years [95% CI] = 1.4 [1.2–1.7]), $P < 0.0001$) for women born later compared with those born 10 years earlier, regardless of their age at GDM diagnosis, race/ethnic background, and gravidity.

CONCLUSIONS — This study shows that the prevalence of GDM doubled between 1994 and 2002 among women of varied ethnic/racial backgrounds living in Colorado. Very little of this increase was due to changes in the age and ethnic distribution of screened pregnancies or a previous history of diagnosed GDM. Importantly, the data show an increasing prevalence of GDM among four race/ethnic groups: non-Hispanic whites, Hispanics, African Americans, and Asians, thus adding to previous reports among American Indians (14,26).

There are several strengths to this study: *1*) the availability of a computerized database that provided consistency in data collection, tracking, and management over time (18); and *2*) an objective, standardized screening and diagnostic procedure with very high effectiveness that has remained constant throughout the study period (21,27). The observed increase in GDM prevalence in this population was not due to changes in ascertainment because screening has been routinely offered to all KPCO women without known diabetes, and the same
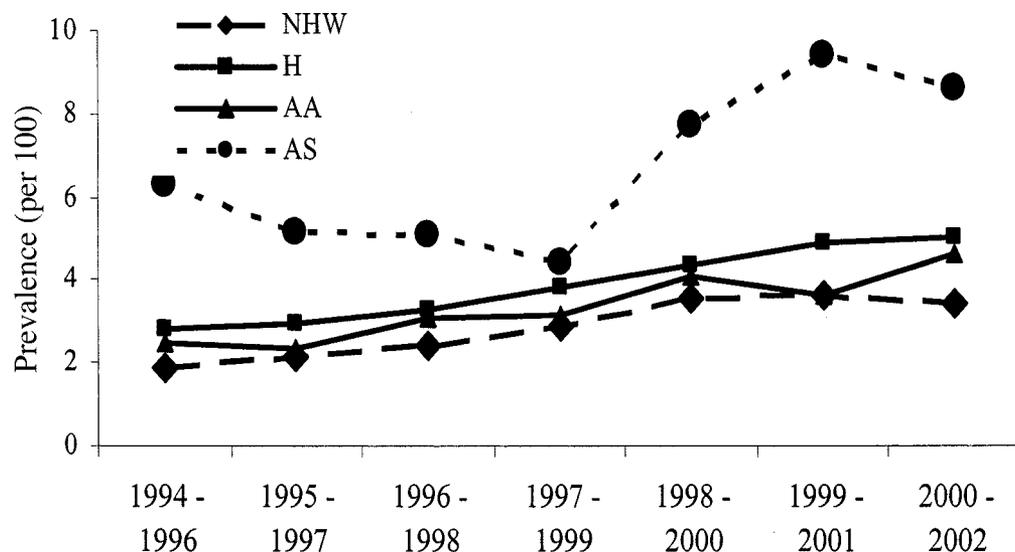

Figure 1—*Age-adjusted prevalence of GDM by race/ethnicity as 3-year moving averages. NHW, non-Hispanic whites; H, Hispanics; AA, African Americans; AS, Asians.*

Table 3—*The effect of time (year of diagnosis) on the prevalence of gestational diabetes using multiple logistic regression*

	Model 1	Model 2	Model 3
Year of diagnosis (per 1 year)	1.12 (1.09–1.14)	1.10 (1.07–1.12)	1.06 (1.01–1.10)
Race/ethnicity			
Other than NHW vs. NHW		2.1 (1.9–2.4)	2.0 (1.7–2.3)
Missing vs. NHW		1.0 (0.8–1.2)	0.9 (0.7–1.3)
Maternal age (per 5 years)		1.7 (1.6–1.8)	1.6 (1.5–1.7)
Prior GDM (yes/no)			20.7 (16.1–26.5)

Model 1 is unadjusted; model 2 is adjusted for race/ethnicity and maternal age (36,388 pregnancies with available data); model 3 is adjusted for race/ethnicity, maternal age, prior diagnosis of gestational diabetes (24,870 multiple pregnancies; information on previous GDM missing in 71 multiple pregnancies). NHW, non-Hispanic white.

standard criteria (20) were used to diagnose GDM since 1994. The observed trends were not due to increased access to the screening protocol over time, given that the acquisition of screening by KPCO women has not substantially changed over time. Although the geographical area served by KPCO has not changed over time, there was a substantial increase in the proportion of screened pregnancies among minority women (Table 1). However, the overall increased GDM prevalence was not caused by a higher proportion of screened high-risk pregnancies in later versus earlier time periods, as the trends were independent of changes in the ethnic distribution of the population (Table 3). Moreover, the increasing trends were significant in populations with both high risk (Hispanic, African American, and Asian) and low risk (non-Hispanic white) for GDM and type 2 diabetes. We were not able to assess the contribution to the observed trends of potential changes in the socioeconomic status of the KPCO population. It is, however, unlikely that the observed trends were due to decreasing socioeconomic status, given that the trends were not explained by changes in ethnicity, and previous data do not suggest an effect of socioeconomic status on GDM risk, independent of race/ethnicity. It is also very unlikely that socioeconomic status changes were present and important enough to determine an increase in GDM prevalence in all racial/ethnic groups.

The present study describes a strong cohort effect on the prevalence of GDM. Regardless of age and ethnicity, women born more recently were at increased risk for GDM diagnosis compared with women born earlier. This finding probably reflects an increased exposure to risk

factor(s) operating before childbearing age. One of the strongest risk factors for GDM (28) is obesity, the prevalence of which has been dramatically increasing over the last several decades (16,29). Although Colorado has the lowest estimated prevalence of obesity in the nation, obesity among Colorado women more than doubled between 1990 and 2001 (29). Coincidently, the prevalence of self-reported (nongestational) diabetes increased markedly among Colorado adults, from 3.4% in 1994 to 5.1% in 2000, an increase observed in both sexes and all age and racial/ethnic groups (30). Our results on increasing GDM prevalence over the same time period are consistent with the reported trends in obesity and type 2 diabetes in Colorado. The fact that GDM prevalence was similar for the two most recently born cohorts in our study has several possible explanations: *1*) not all the women belonging to the

1976–1990 cohort reached the childbearing age as of the date of this analysis, so the prevalence of GDM in the young age-groups with data available for comparison may be artificially low; *2*) the effect of increasing obesity on GDM prevalence is not apparent at very young ages; and *3*) the increase in GDM in the younger population might have reached a plateau.

Early data from a maternity hospital in Australia demonstrated a significant increase in GDM in all racial groups, from 3.3% during 1979–1983 to 7.5% during 1984–1988 (5). However, the authors failed to control for the changing age distribution among pregnant women. A recent report from the Kaiser Permanente Health Care Plan in Northern California found a 35% overall age-ethnicity–adjusted increase in GDM cumulative incidence between 1991 and 2000, an ~4% increase per year (17). Although the proportion of screened pregnancies also increased over time and there was no formal testing for significant effects within each race/ethnic group, this study provided evidence that GDM is increasing in Northern California among women of diverse ethnic/racial backgrounds. Similar trends over the same time period were observed among pregnant women who were members of the Kaiser Permanente Health Care Plans in Northern California and Colorado. The age-adjusted annual prevalence of GDM, however, was lower in Colorado than in Northern California in all race/ethnic groups considered. Part of the difference is due to different diagnostic
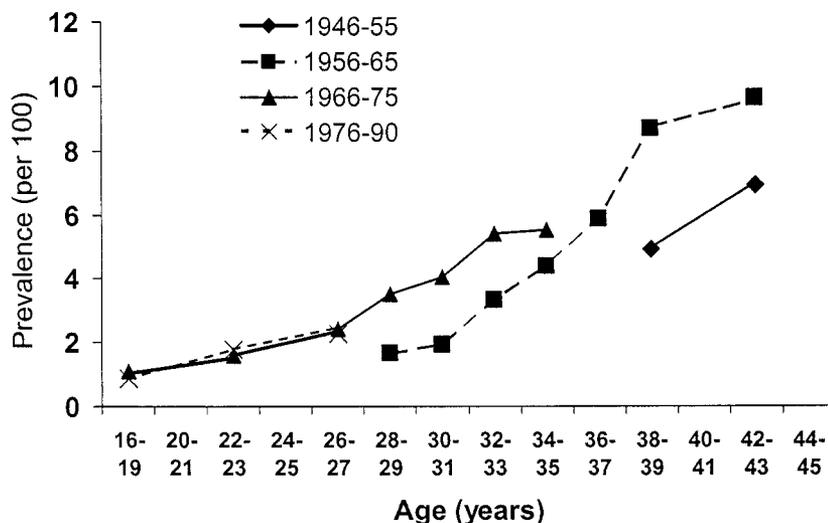
Figure 2—*Age-specific prevalence of GDM by birth cohort.*

criteria. Ferrara et al. (17) used lower plasma glucose thresholds for GDM diagnosis, as recommended by the American Diabetes Association (31), the American College of Obstetricians and Gynecologists (32), and the World Health Organization (33), whereas our protocol was based on higher plasma glucose thresholds recommended by the NDDG (20). When Ferrara and associates used the higher NDDG thresholds in an alternate analysis, the prevalence of GDM in the Northern California study (3.5% in 1990 and 5.1% in 2000) was more similar to that in Colorado (2.1% in 1994 and 4.1% in 2002). The remaining differences may, in part, reflect the substantially lower prevalence of obesity in Colorado compared with that in Northern California (34).

Our analysis was not performed on a geographically based sample of Colorado women; however, the KPCO membership is representative of the population living in the Denver metropolitan area with regard to demographics and ethnicity, and this has not changed over the period of the study (35,36). Another disadvantage is the small number of pregnancies among minority women (especially Asian) available for the race/ethnic-specific analysis. Nevertheless, the increase in GDM prevalence was significant for all racial/ethnic groups (Table 2), and the increase was similar among minority and non-Hispanic white women.

An important limitation of this study is the lack of information about maternal obesity. Data on maternal BMI is missing at the present time from the KPCO perinatal database, and it was also missing in the Kaiser Permanente Study in Northern California (17). This information may be essential for explaining the observed GDM trends. It is also possible that part of the increase in GDM prevalence is due to an obesity-associated increase in type 2 diabetes, undiagnosed before pregnancy. If this is correct, then the proportion of GDM women with normal glucose tolerance after the index pregnancy should decrease over time; unfortunately no data are available at the present time to test this hypothesis. There were no trends in the prevalence of diagnosed preexistent diabetes in the KPCO database (data not shown). However, the number of cases was small and all patients (except one) were insulin treated.

As many as 50% of women with GDM may develop type 2 diabetes within 5 years of the index pregnancy (37). Further, increasing exposure to diabetes during pregnancy may be an important determinant of the increasing prevalence of obesity and type 2 diabetes in youth, as demonstrated among Pima Indian children (14,38). This closes the postulated cross-generational vicious cycle of diabetes in pregnancy, wherein maternal diabetes begets more diabetes in offspring (12,39). Unfortunately, with a few significant exceptions (12,13), no large, ethnically diverse study has followed a group of children whose mothers had GDM and a comparison group whose mothers did not have GDM long enough to demonstrate whether this vicious cycle also operates among other U.S. ethnic groups.

Our data provide evidence that GDM may be increasing among U.S. women with diverse racial/ethnic backgrounds (17). Given the etiology of type 2 diabetes (40,41), the observed increase probably reflects the well-documented obesity epidemic (42). The increase in GDM prevalence may represent a major determinant of the recent increase in obesity and type 2 diabetes among U.S. children, which may lead to further increases in GDM as these youths mature.

References

1. Metzger BE, Coustan DR: Summary and recommendations of the Fourth International Workshop-Conference on Gestational Diabetes Mellitus: the Organizing Committee. *Diabetes Care* 21:B161–B167, 1998
2. World Health Organization Study Group: *Prevention of diabetes mellitus*. Geneva, World Health Org., 1980 (Tech. Rep. Ser., no. 844)
3. American College of Obstetricians and Gynecologists: ACOG Practice Bulletin: Assessment of risk factors for preterm birth: clinical management guidelines for obstetrician-gynecologists: number 31, October 2001. *Obstet Gynecol* 98:709–716, 2001
4. Green JR, Pawson IG, Schumacher LB, Perry J, Kretchmer N: Glucose tolerance in pregnancy: ethnic variation and influence of body habitus. *Am J Obstet Gynecol* 163:86–92, 1990
5. Beischer NA, Oats JN, Henry OA, Sheedy MT, Walstab JE: Incidence and severity of gestational diabetes mellitus according to country of birth in women living in Australia. *Diabetes* 40 (Suppl. 2):S35–S38, 1991
6. Dooley SL, Metzger BE, Cho NH: Gestational diabetes mellitus: influence of race on disease prevalence and perinatal outcome in a U.S. population. *Diabetes* 40 (Suppl. 2):S25–S29, 1991
7. Berkowitz GS, Lapinski RH, Wein R, Lee D: Race/ethnicity and other risk factors for gestational diabetes. *Am J Epidemiol* 135:965–973, 1992
8. Murphy NJ, Bulkow LR, Schraer CD, Lanier AP: Prevalence of diabetes mellitus in pregnancy among Yup'ik Eskimos, 1987–1988. *Diabetes Care* 16:315–317, 1993
9. King H: Epidemiology of glucose intolerance and gestational diabetes in women of childbearing age. *Diabetes Care* 21 (Suppl. 2):B9–B13, 1998
10. Kjos SL, Buchanan TA: Gestational diabetes mellitus. *N Engl J Med* 341:1749–1756, 1999
11. Dabelea D, Knowler WC, Pettitt DJ: Effect of diabetes in pregnancy on offspring: follow-up research in the Pima Indians. *J Matern Fetal Med* 9:83–88, 2000
12. Pettitt DJ, Knowler WC: Diabetes and obesity in the Pima Indians: a crossgenerational vicious cycle. *J Obesity Weight Regul* 7:61–65, 1988
13. Silverman BL, Rizzo T, Green OC, Cho NH, Winter RJ, Ogata ES, Richards GE, Metzger BE: Long-term prospective evaluation of offspring of diabetic mothers. *Diabetes* 40:S121–S125, 1991
14. Dabelea D, Hanson RL, Bennett PH, Roumain J, Knowler WC, Pettitt DJ: Increasing prevalence of type II diabetes in American Indian children. *Diabetologia* 41:904–910, 1998
15. Knowler WC, Bennett PH, Hamman RF, Miller M: Diabetes incidence and prevalence in Pima Indians: a 19-fold greater incidence than in Rochester, Minnesota. *Am J Epidemiol* 108:497–505, 1978
16. Flegal KM: The obesity epidemic in children and adults: current evidence and research issues. *Med Sci Sports Exerc* 31 (Suppl. 11):S509–S514, 1999
17. Ferrara A, Kahn HS, Quesenberry CP, Riley C, Hedderson MM: An increase in the incidence of gestational diabetes mellitus: Northern California, 1991–2000. *Obstet Gynecol* 103:526–533, 2004
18. Meikle SF, Lyons E, Hulac P, Orleans M: Rehospitalizations and outpatient contacts of mothers and neonates after hospital discharge after vaginal delivery. *Am J Obstet Gynecol* 179:166–171, 1998
19. Lynch A, McDuffie R, Murphy J, Faber K, Leff M, Orleans M: Assisted reproductive interventions and multiple birth. *Obstet Gynecol* 97:195–200, 2001
20. National Diabetes Data Group: Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 28:1039–1057, 1979
21. McDuffie RS Jr, Bischoff KJ, Beck A, Orleans M: Does reducing the number of

prenatal office visits for low-risk women result in increased use of other medical services? *Obstet Gynecol* 90:68–70, 1997

22. Breslow NE, Day NE: *Statistical Methods in Cancer Research, Vol. II—The Design and Analysis of Cohort Studies*. Lyon, France, International Agency for Research on Cancer, 1987 (IARC Sci. Publ. No. 82)
23. Ding K, Torabi MR, Bailey WJ: Trend analysis of cigarette smoking by Indiana adolescents, 1991–2000. *Am J Health Behav* 27:35–42, 2003
24. Neter J, Kutner MH, Nachtseim CJ, Wasserman W: *Applied Linear Statistical Models*. New York, McGraw-Hill, 1996
25. Agresti A: *Categorical Data Analysis*. New York, John Wiley and Sons, 1990
26. Moum K, Holzman G, Parsons S, Harwell TS, MT-MD Working Group on Diabetes in Pregnancy: Increasing rate of diabetes in pregnancy among American Indian and White mothers in Montana and North Dakota (Abstract). *Diabetes* 52 (Suppl.1):1773P, 2003
27. Innes KE, Wimsatt JH, McDuffie R: Relative glucose tolerance and subsequent development of hypertension in pregnancy. *Obstet Gynecol* 97:905–910, 2001
28. Jovanovic L, Pettitt DJ: Gestational diabetes mellitus. *JAMA* 286:2516–2518, 2004
29. Rosenblatt B: Adult obesity in Colorado: results from the behavioral risk factor surveillance system [article online], 2002. *Health Watch* 48. Available from www.cdphe.state.co.us/hs/Briefs/obesity2002
30. Rosenblatt B: *Prevalence, Health Behaviors, and Preventive Health Practices among Adult Coloradans with Diagnosed diabetes: Results from the Behavioral Risk Factor Surveillance System, 1997–2000*. Denver, Colorado Department of Public Health. 2002
31. American Diabetes Association: Gestational diabetes mellitus. *Diabetes Care* 23 (Suppl. 1):S77–S79, 2000
32. American College of Obstetricians and Gynecologists: Gestational diabetes. *Obstet Gynecol* 98:525–538, 2001
33. World Health Organization: *Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complications. Report of a WHO Consultation. Part 1: Diagnosis and Classification of Diabetes Mellitus*. Geneva, World Health Organization 1999 (WHO/NCD/NCS/99.2)
34. Ahluwalia IB, Mack KA, Murphy W, Mokdad AH, Bales VS: State-specific prevalence of selected chronic disease-related characteristics: Behavioral Risk Factor Surveillance System, 2001. *MMWR Surveill Summ* 52:1–80, 2003
35. Amthauer H, Gaglio B, Glasgow RE, Dortch W, King DK: Lessons learned: patient recruitment strategies for a type 2 diabetes intervention in a primary care setting. *Diabetes Ed* 29:673–681, 2003
36. Caplan LS, McQueen DV, Qualters JR, Leff M, Garrett C, Calonge N: Validity of women's self-reports of cancer screening test utilization in a managed care population. *Cancer Epidemiol Biomarkers Prev* 12:1182–1187, 2003
37. Kim C, Newton KM, Knopp RH: Gestational diabetes and the incidence of type 2 diabetes: a systematic review. *Diabetes Care* 25:1862–1868, 2002
38. Dabelea D, Hanson RL, Lindsay RS, Pettitt DJ, Imperatore G, Gabir MM, Roumain J, Bennett PH, Knowler WC: Intrauterine exposure to diabetes conveys risks for type 2 diabetes and obesity: a study of discordant sibships. *Diabetes* 49:2208–2211, 2000
39. Freinkel N: Banting Lecture: Of pregnancy and progeny (Review). *Diabetes* 29:1023–1035, 1980
40. Colditz GA, Willett WC, Stampfer MJ, Manson JE, Hennekens CH, Arky RA, Speizer FE: Weight as a risk factor for clinical diabetes in women. *Am J Epidemiol* 132:501–513, 1990
41. Haffner SM, Stern MP, Mitchell BD, Hazuda HP, Patterson JK: Incidence of type II diabetes in Mexican Americans predicted by fasting insulin and glucose levels, obesity, and body-fat distribution. *Diabetes* 39:283–288, 1990
42. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL: Overweight and obesity in the United States: prevalence and trends, 1960–1994. *Int J Obes Relat Metab Disord* 22:39–47, 1998