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Estimating the Recurrence Rate of Gestational Diabetes Mellitus (GDM) in Massachusetts 1998–2007: Methods and Findings

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Abstract

Objectives—Women with gestational diabetes mellitus (GDM) may be able to reduce their risk of recurrent GDM and progression to type 2 diabetes mellitus through lifestyle change; however, there is limited population-based information on GDM recurrence rates.

Methods—We used data from a population of women delivering two sequential live singleton infants in Massachusetts (1998–2007) to estimate the prevalence of chronic diabetes mellitus (CDM) and GDM in parity one pregnancies and recurrence of GDM and progression from GDM to CDM in parity two pregnancies. We examined four diabetes classification approaches; birth certificate (BC) data alone, hospital discharge (HD) data alone, both sources hierarchically combined with a diagnosis of CDM from either source taking priority over a diagnosis of GDM, and both sources combined including only pregnancies with full agreement in diagnosis. Descriptive statistics were used to describe population characteristics, prevalence of CDM and GDM, and recurrence of diabetes in successive pregnancies. Diabetes classification agreement

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was assessed using the Kappa statistic. Associated maternal characteristics were examined through adjusted model-based *t* tests and Chi square tests.

Results—A total of 134,670 women with two sequential deliveries of parities one and two were identified. While there was only slight agreement on GDM classification across HD and BC records, estimates of GDM recurrence were fairly consistent; nearly half of women with GDM in their parity one pregnancy developed GDM in their subsequent pregnancy. While estimates of progression from GDM to CDM across sequential pregnancies were more variable, all approaches yielded estimates of ≤5 %. The development of either GDM or CDM following a parity one pregnancy with no diagnosis of diabetes was <3 % across approaches. Women with recurrent GDM were disproportionately older and foreign born.

Conclusion—Recurrent GDM is a serious life course public health issue; the inter-pregnancy interval provides an important window for diabetes prevention.

Keywords

Diabetes; Gestational diabetes mellitus; Diabetes recurrence; Sequential pregnancies; Pregnancy

Introduction

Gestational diabetes mellitus (GDM), defined as carbohydrate intolerance leading to hyperglycemia with onset or first recognition during pregnancy, is a common complication of pregnancy [1]. It is associated with increased maternal and infant complications, including increased cesarean delivery [2–5], infant macrosomia [3–5], respiratory distress [6], neonatal hypoglycemia [4], and perinatal mortality [4]. While some 5–10 % of women have abnormal hyperglycemia that persists after delivery (reflecting preexisting diabetes or impaired glucose tolerance and/or impaired fasting glucose), most women revert to normal carbohydrate metabolism [1, 7]. However, women with a history of GDM have a 30–84 % increased risk of developing GDM in subsequent pregnancies [8] and a 35–60 % chance of developing diabetes in the next 10–20 years, usually type 2 diabetes [9, 10].

Women with GDM may be able to reduce their risk of recurrent GDM and progression to type 2 diabetes mellitus through lifestyle change. A number of randomized trials have demonstrated that medication [11–13] and/or weight loss [12, 13] and increased physical activity [13] reduce the risk of type 2 diabetes in high-risk individuals, including women with previous GDM [14]. However, very limited epidemiological information exists on the recurrence rates for GDM; the few extant studies use very different types of samples, methodologies and GDM criteria and then report greatly varying rates of GDM recurrence [5, 8, 15–19]. In a recent study from 20 Utah hospitals from 2002 to 2010, Boghossian et al. [5] used medical records to identify cases of GDM. Their results indicated that the risk of developing recurrent GDM was 65 % overall and 62 % among women with sets of parity 1 and 2 births. In contrast, Getahun et al. [17], using the Southern California Kaiser Permanente data base from 1991 to 2008, reported a 41 % GDM recurrence rate and an increased GDM prevalence following a prior GDM pregnancy compared to a prior non-affected pregnancy (OR = 13.2, 95 % CI 12.0–14.6). To our knowledge, the only U.S. population-based study [15] was conducted more than 20 years ago, using data from

Washington state birth certificate (BC) data from 1984 to 1991. McGuire et al. [15] found that the risk of developing GDM in the pregnancy following two GDM-affected pregnancies was greatly increased compared with women without GDM in the previous two pregnancies (OR = 23.2, 95 % CI 17.2–31.2). A more recent population-based study from New South Wales Australia from 2001 to 2008 reported a GDM recurrence rate of 41 % [19]. Progression of GDM to chronic diabetes mellitus (CDM) across sequential pregnancies is even less well studied, especially in population-based data [10]. More current and larger, U.S. population-based estimates of GDM prevalence and the incidence of recurrence and progression to type 2 diabetes are needed to estimate the potential public health impact of diabetes prevention interventions, especially in light of increasing obesity, type 2 diabetes, and GDM [20–25].

The primary sources for surveillance of diabetes in pregnancy in the U.S. are BC and hospital discharge (HD) records; these sources have sometimes been combined to increase sensitivity [26, 27]. Detailed comparisons of the agreement of chronic diabetes diagnoses (CDM), which includes type 1 and type 2 diabetes, and GDM between these two sources or combinations of the two sources have not been performed.

In this study, we used a population of women delivering two sequential live singleton infants in Massachusetts (1998–2007) to examine the prevalence of CDM and GDM in parity one pregnancies, the recurrence rate of GDM, and the progression from GDM to CDM between parity one and parity two pregnancies. We examined and contrasted four different approaches to classifying women's diabetes status: BC data alone, HD data alone, both sources hierarchically combined in which a diagnosis of CDM from either source took priority over a diagnosis of GDM, and both sources combined [18] in which only pregnancies with complete agreement in diagnosis between sources were included. We then examined selected characteristics of women with different patterns of GDM/CDM recurrence and non-recurrence.

Methods

Data Source

We used a population-based cohort of pregnant women who delivered a live birth from 1998 through 2007 in Massachusetts derived from the Pregnancy to Early Life Longitudinal (PELL) data system, a longitudinally linked data system of mothers and their children from birth to early childhood [28]. Deterministic and probabilistic methodologies were used to link records from the data sets. Consecutive birth files were linked to identify sequential pregnancies in individual women. A more detailed description of the PELL data system is described elsewhere [28].

For this analysis, we first selected sets of two sequential pregnancies in our dataset to individual women which met the following criteria: both pregnancies resulted in a live singleton delivery in a Massachusetts hospital to a Massachusetts resident and BC and HD records could be linked for both deliveries (n = 181,030 sets of births). We then restricted the sample to only first and second parity births (n = 141,233 sets of births). We then subjected the two birth sets to closer scrutiny to assure that all deliveries were truly

sequential using the following criteria: (1) absence of a date of last live birth in the first parity pregnancy; (2) exact or close agreement between the date of birth of the first parity pregnancy and the date of last live birth for the second parity pregnancy; (3) the parity numbers from the BC for the two pregnancies were one and two or parity numbers could be inferred if they didn't match one and two (for example, if parity numbers were one and something other than two, but the date of last live birth for the second parity pregnancy matched the date of delivery for the first parity pregnancy); and (4) the time between the two deliveries was plausible. Questionable sets were deleted (n = 6563). Based on these criteria, we identified 134,670 record sets that we judged to be first and second pregnancies of parity one and two.

Diabetes Definition and Classification

Data from PELL HD records and BC records were used to identify potential cases of GDM and CDM during pregnancy. Up to nine ICD 9-CM discharge codes could be listed for a delivery hospitalization. Hospital discharge ICD 9-CM codes for CDM were 648.0 (CDM excluding GDM), and 250.0–250.9 [CDM without complications (250.0) or with complications (250.1–.9)]. The ICD 9-CM code for GDM was 648.8. On the Massachusetts 1996 standard certificate of live birth either or both of the CDM or GDM boxes could be checked. A separate check box for "no diabetes" is not available. For both BC and HD records, in the absence of an indication of diabetes (no diabetes box checked or no ICD-9CM code for diabetes) it was assumed that the pregnancy was not complicated by diabetes.

GDM and CDM diagnoses were classified based on data source: BC alone, HD alone, and BC and HD combined. For BC alone and for HD alone, if an individual pregnancy had diagnoses for both CDM and GDM, CDM was the default diagnosis. When BC and HD were combined, two different approaches were taken. In the first, referred to as the "combined hierarchical" approach, the presence of a CDM diagnosis from either source (HD or BC) resulted in the pregnancy diagnosis categorized as CDM; among the remaining pregnancies, a GDM diagnosis from either source resulted in a pregnancy diagnosis of GDM; and those pregnancies without a diagnosis of diabetes from either source were categorized as "no diabetes." In the second, referred to as the "combined full agreement" approach, only records with complete agreement between HD and BC diagnoses were included; all other records (deliveries with diabetes status 'both' in either the BC or HD record of either delivery, and deliveries for which the BC and HD diabetes status do not agree for both deliveries) were excluded from analysis (N = 4982 records (3.6 %)).

Analysis

Descriptive statistics were used to describe population characteristics, prevalence of CDM and GDM, and recurrence of diabetes in pregnancy. Results from each of the four diabetes classification methods were compared. The extent of diabetes classification agreement was assessed using the Kappa statistic and plausibility of sequential diagnoses. (Kappa statistics from 0.00–0.20, 0.21–0.40, 0.41–0.60; 0.61–0.80; to 0.81–1.00 are considered slight, fair, moderate, substantial, and almost perfect comparability respectively [29]). Finally, selected maternal characteristics were compared across eight categories of diabetes status that

grouped cases with and without GDM and CDM in parity one and two births (No DM/No DM, No DM/GDM, No DM/CDM, GDM/No DM, GDM/GDM, GDM/CDM, CDM/No DM or GDM, CDM/CDM), with a focus on women with recurrent GDM and those without, using Chi square tests and model-based *t* tests, adjusted for multiple comparisons. The adjustments were made by a method described by Jin and Wang [30] for categorical variables and by the Tukey–Kramer method [31] for numeric variables; an adjusted *p* value of <0.05 was considered statistically significant. Statistical analyses were performed using SAS software V.9 (SAS Institute Inc., Cary, NC) for Windows.

The study protocol was reviewed and approved or exempted by the institutional review boards (IRBs) of the Centers for Disease Control and Prevention and the Massachusetts Department of Public Health.

Results

There were 134,670 Massachusetts resident women with two sequential deliveries in Massachusetts hospitals with parity one and two births in 1998–2007. Characteristics of these women are described in Table 1. Based on information from parity one births, the median maternal age was 29 years, and nearly 10 % of women were age 35 or older. Over two-thirds had over 12 years of education (68.6 %), almost three-fourths had private insurance (73.5 %), and nearly 20 % were foreign born.

Diabetes Classification

There was substantial agreement on diabetes classification between HD records and BC, primarily due to the high number of women with no DM in either pregnancy in both data sets. The Kappa statistic for diabetes diagnostic agreement was 0.69 for parity one and 0.69 for parity two pregnancies, with 98.2 and 97.6 % concordance respectively. If women with no DM for both BC and HD are excluded, there is only slight agreement on GDM classification, the Kappa statistic decreases to 0.09 in parity one and to 0.05 in parity two pregnancies and the concordance level drops to 52.2 and 70.6 %. Among parity one pregnancies with a diagnosis of GDM by HD alone, only 60.8 % also had a diagnosis of GDM by BC; among those with a diagnosis of CDM by HD alone, 63.3 % also had a diagnosis CDM by BC; and of those with no diagnosis of DM by HD alone, 99.4 % had no diagnosis of CDM by BC (Table 2). The percentage of pregnancies with a diabetes diagnosis determined by BC that also had the same diagnosis by HD were 79.4, 63.2, and 99.0 % for GDM, CDM, and no diabetes, respectively. Findings were similar for parity two pregnancies.

Using the combined hierarchical approach, 4102 women were classified as having GDM in the parity one pregnancy and the source of GDM diagnosis was as follows: 31 % had a diagnosis of GDM from the HD and not the BC, 13 % from the BC and not the HD, and 57 % had a diagnosis from both sources (Table 3). Of the 1030 women classified as having CDM in the parity one pregnancy, 18 % had a diagnosis of CDM from the HD and not the BC, 43 % had a diagnosis from the BC and not the HD, and 39 % had a diagnosis from both sources. A similar pattern was seen for the parity two pregnancies.

Diabetes Prevalence

The four diabetes classification approaches provide varying estimates of GDM and CDM prevalence in parity one and two pregnancies. According to HD alone, 2.8 % of parity one pregnancies were complicated by GDM and 0.4 % were complicated by CDM (Table 4). When diabetes diagnosis in the first pregnancy was examined using BC alone, the estimate was slightly lower for GDM (2.2 %) but similar for CDM (0.4 %). Diabetes prevalence estimates based on the combined hierarchical approach were higher (3.0 % GDM; 0.8 % CDM) than those using either the BC or HD source alone. Diabetes prevalence estimates based on the combined full agreement approach were lower than in each of the three other approaches (1.8 % GDM; 0.3 % CDM). Up to 1.7-fold differences in initial GDM rates and 2.3 fold differences in initial CDM rates were noted across the different classification approaches.

Comparisons across the four classification approaches for parity two pregnancies were similar to comparisons for parity one pregnancies. All diabetes prevalence estimates for parity two were slightly higher than for parity one, regardless of the categorization approach (Table 4).

Diabetes in Sequential Pregnancies

Despite variations in diabetes estimates across classification methods, estimates of GDM recurrence across sequential pregnancies among women with GDM in the parity one pregnancy were fairly consistent across approaches, and ranged from 38.4 (BC alone) to 47.7 % (combined hierarchical) (Table 5). Estimates of progression from GDM in the parity one pregnancy to CDM in the parity two pregnancy ranged from 2.4 (combined full agreement) to 5.1 % (combined hierarchical). Among women with no diabetes in the parity one pregnancy, estimates of GDM in the parity two pregnancy ranged from 1.5 (combined full agreement) to 2.6 % (combined hierarchical). Very few women with no diabetes in the parity one pregnancy had a diagnosis of CDM in the parity two pregnancy (0.0 % using combined full agreement and 0.4 % using combined hierarchical). Because diabetes prevalence increased over the study period, we repeated our analysis using data from 2003 to 2007 only; the resultant patterns of diabetes diagnosis across pregnancies were similar to those using the full multi-year data set.

Examination of diabetes diagnosis in the parity two pregnancy among women with a diagnosis of CDM in the parity one pregnancy revealed evidence of significant misclassification in three of the classification approaches; using HD alone, 24.7 % had no diagnosis of diabetes or had GDM in the parity two pregnancy, using BC alone, 53.5 % had no diagnosis of diabetes or had GDM in the parity two pregnancy, and using combined hierarchical approach, 42.3 % had no diagnosis of diabetes or had GDM in the parity two pregnancy. In contrast, when using the combined full agreement approach, only 9.8 % had no diagnosis of diabetes or GDM in the parity two pregnancies (Table 5). Further examination of data using the combined hierarchical approach revealed that for women with CDM in the parity one pregnancy and no diabetes in the parity two pregnancy, nearly half (42 %) had had the diagnosis of CDM from the BC and no diagnosis of diabetes from the HD. For women with CDM in the parity one pregnancy and GDM in the parity two

pregnancy, there was no single particular combination that accounted more than 14 % of women in this category (data not shown).

Maternal Characteristics

Patterns of diabetes diagnosis across pregnancies varied by selected maternal characteristics (Table 6). Women with No GDM or CDM in either pregnancy (group 1) were younger, less educated and had a different racial distribution than women in most other GDM/CDM diagnosis categories. They were more often White and less often Asian. Among women with GDM in the parity one pregnancy, those with recurrent GDM (group 5) were older at parity one than those who did not have GDM (group 4) in the parity two pregnancy ($p < 0.001$) and were more likely to be foreign born ($p < 0.05$); and the same pattern was seen for those women with either recurrent GDM or CDM together (groups 5 and 6) (data not shown).

Discussion

In this population-based study, we examined patterns in diabetes across consecutive pregnancies using different approaches to disease categorization. We found that estimates of GDM recurrence were fairly consistent, regardless of the categorization approach; nearly half of women with GDM in their parity one pregnancy developed GDM in their subsequent pregnancy. While estimates of progression from GDM to CDM across sequential pregnancies were somewhat variable, all four approaches yielded estimates of ≤5 %. Similarly, the development of either type of diabetes (GDM or CDM) following a parity one pregnancy with no diagnosis of diabetes was rare (<3 %) in all approaches.

There are few published studies of GDM recurrence rates. Our estimated recurrence rates (34–48 %) are similar to the ~40 % recurrence rates noted in the study of Kaiser Permanente enrollees [17] and the large population-based study in New South Australia [19], as well as in White non-Hispanic mothers from several non-population-based studies conducted within and outside of the U.S [8]. In contrast, the current study finding is much lower than the 62 % recurrence rate reported in the recent Utah hospitals' case series study [5].

Because GDM is believed to be a manifestation of increased insulin resistance and decreased beta cell compensation for insulin resistance [32–34], one might expect the recurrence rate to be very high. Several factors could have contributed to the finding that over half of women with GDM in the first pregnancy did not have GDM in the second pregnancy. First, women with a history of GDM may have modified their lifestyle between pregnancies or during their second pregnancy, resulting in weight loss, less weight gain during pregnancy, improved nutrition and/or increased physical activity. Second, the finding could reflect errors in the initial GDM diagnosis on the BC or HD records (e.g. an over-estimation in initial GDM diagnosis). For example, providers may record a diagnosis of GDM or possible GDM in the medical records of women with elevated blood glucose levels even if a glucose tolerance test did not meet criteria for GDM; and clinicians may use different standards for GDM diagnoses. Alternatively, a diagnosis of GDM in one pregnancy may not be a perfect predictor of progression toward type 2 diabetes; progression toward type 2 diabetes might not be a linear process, especially when observed over short periods of time. The initial GDM diagnosis may still reflect some elevated future risk,

though not enough to merit a GDM diagnosis in the subsequent pregnancy. Finally, the pattern we observed could reflect selection bias if women at highest risk for recurrence were less likely than lower risk women to have a second pregnancy during our study interval.

Our lower estimates of progression from GDM to CDM using combined full agreement (2.4 %) and HD alone (3.1 %) are similar to the estimate of Khambalia et al. [19]. The higher estimates of CDM progression based on BC data may be less reliable and prone to over-estimation. The combined full agreement approach likely has lower sensitivity because of the high percentage of records excluded, but its specificity is superior to the other approaches. New CDM diagnoses in parity two pregnancies without prior GDM indications were rare.

As evidence has shown that weight loss after pregnancy has the potential to reduce a woman's future risk for type 2 diabetes [35], women with a GDM-affected pregnancy should be encouraged to continue their lifestyle modification after pregnancy to return to their healthy pre-pregnancy BMI, and if they were overweight or obese when entering pregnancy to continue to lose weight to prevent postpartum weight retention or entering in the next pregnancy obese. Future studies to better understand the etiology of those who do and do not develop GDM in a subsequent pregnancy or progress to type 2 diabetes would be useful for public health efforts in diabetes prevention.

Examination of methodological variations in GDM classification was a planned, critical part of this study, though we found more substantial variations than we had initially anticipated– which in part led us to utilize alternative combinational approaches, including the combined full agreement approach. Two major methodological findings are of particular note; we found large variations in initial classification of diabetes status between BC and HD records and evidence of substantial misclassification in CDM diagnosis. First, variation up to 166 % in GDM classification by source is problematic for surveillance. Lydon-Rochelle et al. [27] and others have noted that clinical risk factors for poor pregnancy outcomes are generally more often documented in the HD records than in BC records. Here we document that 31 % of GDM cases identified in the HD records were not noted in the BC data. Since use of BC data for state (and national) estimates of GDM is wide-spread, our findings suggests caution in its usage for that purpose as it likely substantially under-estimates GDM prevalence.

Second, this study detected serious CDM measurement issues among pregnant women, using information either from the BC or HD or their combinations. Potential CDM over-identification and implausible sequential birth diagnoses were both widely noted (e.g., CDM in the parity one pregnancy and no CDM or GDM in the parity two pregnancy). In particular, the current study found that the BC alone or in combined hierarchical approach had the largest number of implausible diagnosis combinations. This finding is also consistent with an earlier validation study conducted by Lydon-Rochelle and colleagues in which medical records in Washington State were compared with data from BC, HD, and a combination of these sources and showed a substantial over estimation of CDM [27]. Their medical record-based estimate of CDM was 1.1 % while that based on combined data from BC and HD was 1.7 %, an overestimation of more than 50 %. When combined with the findings of the current study, the Lydon-Rochelle et al. data suggest that combining data

from BC and HD to determine CDM diagnosis may be especially problematic and could result in an increase in false positive diabetes diagnoses and an overestimation of CDM prevalence during pregnancy.

More generally, BC and HD data may not be suitable sources of data for identifying or monitoring CDM among population-based cohorts of women giving birth. The inconsistencies across data sources noted here may simply reflect the widespread uncertainty with initial recording of CDM/GDM diagnoses in parity one births. Some of the GDM associated births may reflect undiagnosed CDM cases among women who had never have been preconceptionally monitored for diabetes prior to entry into prenatal care. Interestingly, most of the other sequential GDM researchers chose restrictive methodologic solutions to avoid dealing directly with any potential CDM misclassification problems; either by excluding or omitting all CDM cases from their analyses [17] or by assuming CDM stability/chronicity and ignoring any implausible changes in subsequent diagnoses [5]. Known CDM/GDM diagnostic confusion during pregnancy is manifest in our public surveillance data systems.

The characteristics of women with sequential GDM, older maternal age and higher foreign-born prevalence, is consistent with the findings of Khambalia and colleagues, who identified maternal age >35 and maternal country of birth Middle East/North Africa and Asia as risk factors for recurrent GDM, as well as for pregnancy associated hypertension, large for gestational age/preterm birth in the first pregnancy, and longer birth interval [19]. The maternal characteristics for GDM recurrence seem consistent with the major risk factors for GDM itself.

Our study has several limitations. First, the older age, better educated demographics of Massachusetts births may restrict the generalizability of the study findings. Second, identifying the first and second sequential delivery combinations may be subject to error; some of our birth chains may not actually reflect parity one and parity two. Third, there is no “no diabetes” diagnostic category on either the BC or the HD record, it is a residual classification and may be subject to error. Fourth, we were not able to validate the study’s administrative record GDM/CDM diagnoses with medical record reviews—and so could not calculate the sensitivity, specificity, positive predictive values, and negative predictive values of the four categorization approaches. Further, we cannot determine if there are any differentially misclassification biases across parity that might further alter our sequential GDM estimates. Moreover, the right censoring of the study period may have limited the current analysis to women with shorter inter-pregnancy intervals, excluding the full fertility experiences of the women who have not finished their child-bearing efforts, and hence potentially under-estimating the sequential GDM/CDM prevalence. In addition, we did not have data on maternal body mass index, which is an important predictor of both GDM and CDM. Strengths of this study include that it is population based and that the sample is large enough to examine combinations of diabetes diagnoses across pregnancies.

Conclusions

In a MA population-based cohort, using a variety of data sources and GDM/CDM classification approaches, we consistently found that women with GDM in their parity one pregnancy have a risk of recurrence approaching 50 % and 2–5 % risk of progression to CDM in their parity two pregnancy. The risk of GDM or CDM in the parity two pregnancy in the absence of a diagnosis of diabetes in the initial pregnancy is very low. Substantial variations in diabetes classification surveillance measures exist. There was only slight agreement on GDM classification between HD and BC records, suggesting the potential for under-reporting of GDM from either data base alone. Further, substantial levels of CDM diagnostic implausibility and misclassification from BC and HD data, alone or in hierarchical combinations, suggest that these data sources may not be suitable for studying chronic diabetes in pregnancy. Women with sequential GDM pregnancies are older and more likely foreign born, similar to risk factors for GDM itself. GDM recurrence remains a serious life course public health issue in this era of obesity and diabetes epidemics. Given that over half of women with GDM diagnosis in their first pregnancy have no DM diagnosis in the subsequent pregnancy suggests that the inter-conception period may therefore provide an important window for diabetes prevention. Clinical and lifestyle interventions may be efficacious for some women. Future studies from this series will examine the impact of GDM recurrence on maternal and birth outcomes.

References

1. Centers for Disease Control and Prevention. National diabetes statistics report: Estimates of diabetes and its burden in the United States, 2014. U.S. Department of Health and Human Services; Atlanta, GA: 2014.
2. Casey BM, Lucas MJ, Mcintire DD, Leveno KJ. Pregnancy outcomes in women with gestational diabetes compared with the general obstetric population. Obstetrics and Gynecology. 1997; 90(6): 869–873. [PubMed: 9397092]
3. Xiong X, Saunders LD, Wang FL, Demianczuk NN. Gestational diabetes mellitus: Prevalence, risk factors, maternal and infant outcomes. International Journal of Gynaecology and Obstetrics. 2001; 75(3):221–228. [PubMed: 11728481]
4. Jensen DM, Sorensen B, Feilberg-Jorgensen N, Westergaard JG, Beck-Nielsen H. Maternal and perinatal outcomes in 143 Danish women with gestational diabetes mellitus and 143 controls with a similar risk profile. Diabetic Medicine. 2000; 17(4):281–286. [PubMed: 10821294]
5. Boghossian NS, Yeung E, Albert PS, Mendola P, Laughon SK, Hinkle SN, Zhang C. Changes in diabetes status between pregnancies and impact on subsequent newborn outcomes. American Journal of Obstetrics and Gynecology. 2014; 210(5):431.e1–14. [PubMed: 24361790]
6. Barahona MJ, Sucunza N, Garcia-Patterson A, et al. Period of gestational diabetes mellitus diagnosis and maternal and fetal morbidity. Acta Obstetricia et Gynecologica Scandinavica. 2005; 84(7):622–627. [PubMed: 15954869]
7. Kim SC, Newton KM, Knopp RH. Gestational diabetes and the incidence of type 2 diabetes: A systematic review. Diabetes Care. 2002; 25(10):1862–1868. [PubMed: 12351492]
8. Kim C, Berger DK, Chamany S. Recurrence of gestational diabetes mellitus: A systematic review. Diabetes Care. 2007; 30(5):1314–1319. [PubMed: 17290037]
9. Kim SY, Dietz PM, England L, Morrow B, Callaghan WM. Trends in pre-pregnancy obesity in nine states, 1993–2003. Obesity Research. 2007; 15(4):986–993.
10. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: A systematic review and meta-analysis. Lancet. 2009; 373:1773–1779. [PubMed: 19465232]

11. Buchanan TA, Xiang AH, Peters RK, et al. Preservation of pancreatic beta-cell function and prevention of type 2 diabetes by pharmacological treatment of insulin resistance in high risk Hispanic women. Diabetes. 2002; 51:2796–2803. [PubMed: 12196473]
12. Buchanan TA. Pancreatic beta-cell loss and preservation in type 2 diabetes. Clinical Therapeutics. 2003; 25(Suppl 2):B32–B46. [PubMed: 14553865]
13. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. New England Journal of Medicine. 2002; 346:393–403. [PubMed: 11832527]
14. Ratner RE. Prevention of type 2 diabetes in women with previous gestational diabetes. Diabetes Care. 2007; 30(12):3154.
15. McGuire V, Rauh MJ, Mueller BA, Hickock D. The risk of diabetes in a subsequent pregnancy associated with prior history of gestational diabetes or macrosomic infant. Paediatric and Perinatal Epidemiology. 1996; 10(1):64–72. [PubMed: 8746432]
16. Major CA, deVeciana M, Weeks J, Morgan MA. Recurrence of gestational diabetes: Who is at risk? American Journal of Obstetrics and Gynecology. 1998; 179(4):1038–1042. [PubMed: 9790394]
17. Getahun D, Fassett MJ, Jacobsen SJ. Gestational diabetes: Risk of recurrence in subsequent pregnancies. American Journal of Obstetrics and Gynecology. 2010; 203:467.e1–6. [PubMed: 20630491]
18. Holmes HJ, Lo JY, McIntire DD, Casey BM. Prediction of diabetes recurrence in women with class A1 (diet-treated) gestational diabetes. American Journal of Perinatology. 2010; 27(1):47–52. [PubMed: 19806532]
19. Khambalia AZ, Ford JB, Nassar N, Shand AW, McElduff A, Roberts CL. Short report: Epidemiology occurrence and recurrence of diabetes in pregnancy. Diabetic Medicine. 2013; 30:452–456. [PubMed: 23323841]
20. Fisher SC, Kim SY, Sharma AJ, Rochat R, Morrow B. Is obesity still increasing among pregnant women? Prepregnancy obesity trends in 20 states, 2003–2009. Preventive Medicine. 2013; 56(6): 372–378. [PubMed: 23454595]
21. Albrecht SS, Kuklina EV, Bansil P, Jamieson DJ, Whiteman MK, Kourtis AP, et al. Diabetes trends among delivery hospitalizations in the U.S., 1994–2004. Diabetes Care. 2010; 33(4):768–773. [PubMed: 20067968]
22. Ferrara A, Kahn HS, Quesenberry CP, Riley C, Hedderson MM. An increase in the incidence of gestational diabetes mellitus: Northern California, 1991–2000. Obstetrics and Gynecology. 2004; 103:526–533. [PubMed: 14990417]
23. Dabelea D, Snell-Bergeon JK, Hartsfield CL, Bischoff KJ, Hamman RF, McDuffie RS. Increasing prevalence of gestational diabetes mellitus (GDM) over time and by birth cohort: Kaiser Permanente of Colorado GDM Screening Program. Diabetes Care. 2005; 28(3):579–584. [PubMed: 15735191]
24. Lawrence JM, Contreras R, Chen W, Sacks DA. Trends in the prevalence of preexisting diabetes and gestational diabetes mellitus among a racially/ethnically diverse population of pregnant women, 1999–2005. Diabetes Care. 2008; 31:899–904. [PubMed: 18223030]
25. Getahun D, Nath C, Ananth CV, Chavez MR, Smulian JC. Gestational diabetes in the United States: Temporal trends 1989 through 2004. American Journal of Obstetrics and Gynecology. 2008; 195:525e1–6. [PubMed: 18279822]
26. Savitz DA, Janevic TM, Engel SM, Kaufman JS, Herring AH. Ethnicity and gestational diabetes in New York City, 1995–2003. BJOG. 2008; 115(8):969–978. [PubMed: 18651880]
27. Lydon-Rochelle MT, Holt VL, Cárdenas V, Nelson JC, Easterling TR, Gardella C, Callaghan WM. The reporting of pre-existing maternal medical conditions and complications of pregnancy on birth certificates and in hospital discharge data. American Journal of Obstetrics and Gynecology. 2005; 193(1):125–134. [PubMed: 16021070]
28. Clements KM, Barfield WD, Kotelchuck M, Lee KG, Wilber N. Birth characteristics associated with early intervention referral, evaluation for eligibility, and program eligibility in the first year of life. Maternal and Child Health Journal. 2006; 10(5):433–441. [PubMed: 16710765]

29. Landis JR, Koch GG. The measurement of observer agreement for categorical data. Biometrics. 1977; 33(1):159–174. [PubMed: 843571]

30. Jin, M.; Wang, B. Implementing multiple comparisons on Pearson Chi square for an RxC contingency table in SAS. The SAS Institute; Cary, NC: 2014. SAS Technical Paper 1544-2014, SAS Global Forum, March 2014

31. Kramer CY. Extension of multiple range tests to group means with unequal numbers of replications. Biometica. 1956; 12:307–310.

32. Catalano PM, Tyzbir ED, Wolfe RR, et al. Carbohydrate metabolism during pregnancy in control subjects and women with gestational diabetes. American Journal of Physiology. 1993; 264:E60–E67. [PubMed: 8430789]

33. Xiang AH, Peters RK, Trigo E, Kjos SL, Lee WP, Buchanan TA. Multiple metabolic defects during late pregnancy in women at high risk for type 2 diabetes. Diabetes. 1999; 48:848–854. [PubMed: 10102703]

34. Homko C, Sivan E, Chen X, Reece EA, Boden G. Insulin secretion during and after pregnancy in patients with gestational diabetes mellitus. Journal of Clinical Endocrinology and Metabolism. 2001; 86:568–573. [PubMed: 11158010]

35. Catanzaro RB, Gavard JA, Mostello DJ, Friganza JC. A lifestyle intervention of weight-gain restriction: Diet and exercise in obese women with gestational diabetes mellitus. Applied Physiology, Nutrition and Metabolism. 2007; 32(3):596–601.

Significance

What is already known on this subject?

Gestational diabetes mellitus (GDM) is increasing in the US. Lifestyle changes can reduce risks of recurrent GDM and progression to type 2 chronic diabetes mellitus (CDM). However, few published studies describe recurrence rates across pregnancies.

What this study adds?

This study provides population-based estimates of GDM recurrence in Massachusetts. Nearly 50 % of women with GDM in their parity one pregnancy developed GDM in their next pregnancy, and under 5 % progressed to CDM. However, this study found limited agreement in GDM and CDM status across different diabetes classification approaches using birth certificate and hospital discharge records.

Table 1

Maternal characteristics of study population at parity one delivery among 134,670 sets of sequential first and second deliveries, Massachusetts 1998–2007

Maternal characteristics	Number 134,670	Percent
Age in years		
<20	16,273	12.08
20–24	22,057	16.38
25–29	39,266	29.16
30–34	43,841	32.55
35–39	12,334	9.16
40+	899	0.67
Median		29.00
Education <High School		
High school (12 years)	15,915	11.83
Some college (<4 years)	26,321	19.57
≥4 years of college	26,511	19.71
	65,742	48.88
Interval between parity 1 and parity 2 pregnancies (*in years*)		
Median		2.42
Race/ethnicity		
White	103,223	76.73
Black	7343	5.46
Hispanic	13,121	9.75
Asian	8346	6.20
Native American/other	2493	1.85
Foreign born		
No	108,112	80.29
Yes	26,545	19.71
Payment source		
Private	98,940	73.47
Public	34,694	25.76
Self-pay	574	0.43
Free care	462	0.34
Year of first delivery		
1998–1999	40,447	30.03
2000–2001	37,794	28.06
2002–2003	33,138	24.61
2004–2005	21,099	15.67
2006–2007	2192	1.63
Year of second delivery		
1998–1999	2234	1.66
2000–2001	22,627	16.80

Maternal characteristics	Number 134,670	Percent
2002–2003	34,381	25.53
2004–2005	36,876	27.38
2006–2007	38,552	28.63

Table 2

Diabetes diagnosis from birth certificate records compared to diabetes diagnosis from hospital discharge records for sets of sequential parity one pregnancy (N = 134,670) and parity two pregnancy (N = 134,670), Massachusetts 1998–2007

Hospital discharge diagnosis (N, column %)					
Birth certificate diagnosis	**No DM**	**GDM**	**CDM**	**Both**	**Total**
Parity one pregnancy					
No DM	129,538 (99.4)	1266 (33.2)	90 (16.3)	11 (28.9)	130,905 (97.2)
GDM	515 (0.4)	2321 (60.8)	72 (13.1)	14 (36.8)	2922 (2.2)
CDM	142 (0.1)	55 (1.4)	349 (63.3)	6 (15.8)	552 (0.4)
Both GDM/CDM	68 (0.1)	176 (4.6)	40 (7.3)	7 (18.4)	291 (0.2)
Total	130,263 (100.0)	3818 (100.0)	551 (100.0)	38 (100.0)	134,670 (100.0)
Parity two pregnancy					
No DM	127,955 (99.2)	1524 (30.5)	127 (19.6)	8 (33.3)	129,614 (96.2)
GDM	807 (0.6)	3122 (62.6)	106 (16.3)	10 (41.7)	4045 (3.0)
CDM	174 (0.1)	104 (2.1)	382 (58.9)	2 (8.3)	662 (0.5)
Both GDM/CDM	70 (0.1)	241 (4.8)	34 (5.2)	4 (16.7)	349 (0.3)
Total	129,006 (100.0)	4991 (100.0)	649 (100.0)	24 (100.0)	134,670 (100.0)

Kappa = 0.69, 95 % CI = 0.68–0.70

Table 3

Diabetes category based on combined hierarchical approach, by source of diagnosis, for parity one pregnancy and parity two pregnancy, Massachusetts 1998–2007

	Diabetes status based on combined hierarchical categorization (N, column %)	
Parity 1 pregnancy		
Source of diabetes diagnosis	GDM (N = 4102)	CDM (N = 1030)
HD	1266 (31)	187 (18)
BC	515 (13)	441 (43)
Both HD and BC	2321 (57)	402 (39)
Parity 2 pregnancy		
Source of diabetes diagnosis	GDM (N = 5453)	CDM (N = 1262)
HD	1524 (28)	251 (20)
BC	807 (15)	589 (47)
Both HD and BC	3122 (57)	422 (33)

Table 4

Prevalence of diabetes diagnoses for parity one and parity two pregnancy by four approaches using hospital discharge and birth certificate records, Massachusetts 1998–2007

Diabetes status	HD alone		BC alone		Combined BC-HD hierarchical		Combined BC-HD full agreement	
	N	%	N	%	N	%	N	%
Parity one pregnancy								
No DM	130,263	96.7	130,905	97.2	129,538	96.2	129,538	98.0
GDM	3818	2.8	2922	2.2	4102	3.0	2321	1.8
CDM	551	0.4	552	0.4	1030	0.8	349	0.3
Both GDM/CDM	38	0.0	291	0.2			7	0.0
Total	134,670	100.0	134,670	100.0	134,670	100.0	132,215	100.0
Parity two pregnancy								
No DM	129,006	95.8	129,614	96.2	127,955	95.0	127,955	97.3
GDM	4991	3.7	4045	3.0	5453	4.0	3122	2.4
CDM	649	0.5	662	0.5	1262	0.9	382	0.3
Both GDM/CDM	24	0.0	349	0.3			4	0.0
Total	134,670	100.0	134,670	100.0	134,670	100.0	131,463	100.0

Table 5

Prevalence of diabetes status from parity one to parity two pregnancy by four approaches to assign diabetes status, Massachusetts 1998–2007

Parity one birth	Parity two birth	Approach for assigning DM status (N, column %)			
		HD alone[a]	BC alone[b]	BC-HD combined hierarchical	BC-HD combined full agreement[c]
N		134,608	134,155	134,670	129,688
GDM	No DM	1922 (50.4)	1673 (57.9)	1938 (47.2)	964 (53.4)
	GDM	1769 (46.4)	1108 (38.4)	1955 (47.7)	798 (44.2)
	CDM	120 (3.1)	107 (3.7)	209 (5.1)	44 (2.4)
No DM	No DM	127,009 (97.5)	127,636 (97.6)	125,737 (97.1)	125,737 (98.5)
	GDM	3134 (2.4)	2816 (2.2)	3342 (2.6)	1866 (1.5)
	CDM	107 (0.1)	280 (0.2)	459 (0.4)	0 (0.0)
CDM	No DM	63 (11.5)	210 (39.3)	280 (27.2)	13 (5.3)
	GDM	72 (13.2)	76 (14.2)	156 (15.1)	11 (4.5)
	CDM	412 (75.3)	249 (46.5)	594 (57.7)	220 (90.2)

[a] Excludes 62 deliveries with diabetes status ‘both’ in the hospital discharge record of either delivery

[b] Excludes 515 deliveries with diabetes status ‘both’ in the birth certificate record of either delivery

[c] Excludes 9892 deliveries with either (1) diabetes status ‘both’ in either the BC or HD record of either delivery, or (2) deliveries for which the BC and HD diabetes status do not agree for both deliveries

Table 6

Selected characteristics of women by various sequential GDM/CDM combinations, using combined hierarchical approach for diabetes classification, Massachusetts 1998–2007 (N = 134,670)

	Diabetes status across parity one and parity two births							
Group Maternal trait	**1 No DM No DM**	**2 No DM GDM**	**3 No DM CDM**	**4 GDM No DM**	**5 GDM GDM**	**6 GDM CDM**	**7 CDM No DM/ GDM**	**8 CDM CDM**
N	125,737	3342	459	1938	1955	209	436	594
Age at parity one birth (%)								
<20	12.5	8.3	9.6	6.1	3.2	4.8	7.6	5.7
20–24	16.5	16.1	17.2	15.2	11.8	11.5	13.1	14.1
25–29	29.1	30.4	26.1	32.0	30.7	29.7	28.7	27.1
30–34	32.4	34.1	32.9	33.8	38.7	36.4	36.2	38.4
35–39	9.0	10.0	13.3	12.0	13.9	16.3	14.0	13.3
40+	0.6	1.1	0.9	1.0	1.6	1.4	0.5	1.3
Mean age[1]	27.5	28.3[a]	28.2[a]	28.7[a,b]	29.7[a,b,c,d]	29.6[a,b,c,d]	28.9[a]	29.1[a,b]
Birth interval (years)	2.8	3.2[a]	3.2[a]	2.7[a,b,c]	2.7[a,b,c]	3.0[a]	2.8[b,c]	2.7[b,c]
Education at parity one birth (%)								
<HS (<12 years)	12.5	8.3	9.6	6.1	3.2	4.8	7.6	5.7
HS (12 years)	16.5	16.1	17.2	15.2	11.8	11.5	13.1	14.1
Some college (13–15 years)[2]	29.1	30.4	26.1	32.0	30.7	29.7	28.7	27.1
4 + years of college (16 + years)	32.4	34.1[a]	32.9	33.8[a]	38.7[a,b,c]	36.4[a]	36.2[b,e,f]	38.4[a,b]
Race/ethnicity at parity one birth (%)								
Hispanic	9.8	9.5	17.0	7.3	8.1	13.9	14.3	10.4
White	77.2	70.8	59.6	72.9	72.0	63.5	63.4	73.9
Black	5.4	6.1	11.6	6.4	5.8	11.1	9.2	8.9
Asian	5.8	11.6	8.7	11.7	12.6	10.6	11.3	5.9
Native American	1.9	2.0[a]	3.1[a,b]	1.7[a,c]	1.5[a,c]	1.0[a,d,e]	1.8[a,d,e]	0.8[a,b,c,d,e],
Foreign born (%)	19.3	26.4[a]	27.5[a]	25.3[a]	28.2[a,d]	30.6[a]	28.4[a]	17.2
Payment source at parity one birth (%)								
Private[2]	73.4	73.0	67.5[a]	77.0[a,b,c]	78.6[a,b]	76.6[c]	73.4	71.5
Public	25.9	26.1	31.4	22.1	20.5	23.0	25.5	26.9
Self-pay	0.4	0.5	0.4	0.6	0.6	0.5	0.7	0.7
Free care	0.3	0.4	0.7	0.3	0.4	0.0	0.5	0.8

[a] Different from group 1, $p<.05$

[b] Different from group 2, $p<.05$

[c] Different from group 3, $p<.05$

[d] Different from group 4, $p<.05$

[e] Different from group 5, $p<.05$

[f] Different from group 6, $p<.05$

[1] Comparison of Mean Age

[2] Comparison of Public versus Private payment source only