Published in final edited form as:

Am J Perinatol. 2019 August ; 36(10): 1039–1044. doi:10.1055/s-0038-1676114.

Interdelivery Interval and Diabetes Mellitus in a Subsequent Pregnancy

Annie M. Dude, MD, PhD[1], **Ashley Battarbee, MD**[2], **Lynn M. Yee, MD, MPH**[1]

[1]Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, Feinberg School of Medicine, Chicago, Illinois

[2]Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina

Abstract

Objective—We determined whether time between deliveries is associated with developing diabetes at the time of a subsequent delivery.

Study Design—This is a case–control study of women who had two consecutive singleton births at the same institution with no pregestational diabetes in the baseline pregnancy. Cases were defined as women who were diagnosed with any type of diabetes at the time of the subsequent delivery. Controls were defined as women who had no diagnosis of diabetes at the time of the subsequent delivery. Interdelivery interval (IDI) was categorized as < 18, 18 to 60, or > 60 months.

Results—Of 12,263 women, 4.1% (N = 501) were diagnosed with diabetes at the subsequent delivery. Women with diabetes were more likely to have an IDI of >60 months than women without diabetes (9.0 vs. 4.2%, $p < 0.001$). After controlling for confounding factors, an IDI > 60 months remained associated with development of pregestational or gestational diabetes by the conclusion of the subsequent pregnancy (adjusted odds ratio = 2.13 compared with an IDI of 18–60 months, 95% confidence interval 1.44–3.15).

Conclusion—A longer IDI is an independent risk factor for the development of diabetes at the time of a subsequent delivery.

Keywords

gestational diabetes; interdelivery interval; pregestational diabetes

Gestational diabetes, defined as carbohydrate intolerance that begins or is first recognized during pregnancy, complicates approximately 6 to 8% of pregnancies in the United States,[1] and pregestational diabetes affects a further 4% of pregnancies.[2] The prevalence of diabetes

Address for correspondence Annie M. Dude, MD, PhD, Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, Feinberg School of Medicine, 250 East Superior Street, Suite 5-2185, Chicago, IL 60611 (annie.dude@gmail.com).

Portions of this article were presented at the 64th Annual Scientific Meeting of the Society for Reproductive Investigation, Orlando, FL, March 15–18, 2017, and the 15th Biannual Meeting of the Diabetes in Pregnancy Study Group of North America, Washington, DC, October 27–29, 2017.

Conflict of Interest
None declared.

in pregnancy has been increasing over time.[3] Diabetes in pregnancy is associated with adverse outcomes for women, including higher order lacerations[4] and cesarean delivery,[5] as well as increased risk of obstetric and neonatal complications including stillbirth[2] and neonatal hypoglycemia.[6] Gestational diabetes is also a risk factor for developing type 2 diabetes mellitus among mothers[7] and obesity in offspring.[8]

A longer interdelivery interval (IDI) has been identified as a risk factor for developing diabetes in a subsequent pregnancy,[9–11] perhaps due to progression of inherent pancreatic β cell dysfunction.[12] However, many existing studies evaluated recurrence of gestational diabetes, rather than de novo onset of gestational diabetes after a previously uncomplicated pregnancy.[9] Others failed to control for important confounders that also vary with time and are associated with the development of diabetes, such as maternal age and weight gain.[10,11] Finally, other data are conflicting, showing that shorter IDIs are also associated with increased risk of gestational diabetes, perhaps due to incomplete weight loss or inadequate time for recovery of baseline endocrine function.[13]

Thus, the limitations of the existing literature leave unresolved the question of the independent relationship of IDI to subsequent gestational or pregestational diabetes. The objective was to evaluate the association between IDI and development of gestational or pregestational diabetes during or before a subsequent delivery, following an index pregnancy without pregestational diabetes, while controlling for other factors such as age and weight change.

Materials and Methods

This is a retrospective case–control study of women aged 18 years or older who delivered two consecutive singleton pregnancies at 24 weeks' gestation or more at Northwestern Memorial Hospital or its affiliated community-based Lake Forest Hospital between January 1, 2005, and December 31, 2015. Women were excluded if they carried a diagnosis of any pregestational type 2 diabetes at the time of the index pregnancy or type 1 diabetes mellitus diagnosed at any time before or during the index or subsequent pregnancy. All data were determined by chart review, with the diagnosis of type 1 versus type 2 diabetes established either through the patient's primary care or endocrine provider, and/or via antibody testing. Additionally, women with missing data on diabetes in either pregnancy were excluded. Women with gestational diabetes mellitus in the index pregnancy were included in the study sample. Cases were defined as women who were diagnosed with either pregestational type 2 diabetes or any type of gestational diabetes at the time of delivery of the subsequent pregnancy. Controls were defined as women who did not carry a diagnosis of pregestational or gestational diabetes at the time of the subsequent delivery. Clinical and demographic data were abstracted from the electronic medical record.

The main exposure of interest was the IDI, measured as a categorical variable coded as < 18 months ("short" IDI), 18 to 60 months, and > 60 months ("long" IDI). We selected this categorization based on previous work related to IDIs and pregnancy outcomes that suggested outcomes in the 18 to 60 months range are similar, whereas shorter and longer intervals are associated with adverse outcomes such as preterm birth, cesarean delivery, low

Apgar scores, and small for gestational age birthweight.[14,15] The primary outcome was pregestational diabetes diagnosed before or during the subsequent pregnancy or gestational diabetes diagnosed at any time during the subsequent pregnancy. Women were diagnosed with gestational diabetes using a two-step process as recommended by the American College of Obstetricians and Gynecologists.[1] Women were determined to have pregestational diabetes if they carried that diagnosis at the beginning of prenatal care for their subsequent pregnancy based on evaluations from a patient's primary care or endocrinology physician, or if she met criteria for pregestational diabetes based on the results of early pregnancy testing.[16]

Other variables considered as clinically important potential confounders include weight change between deliveries, maternal age at the time of index pregnancy (in years), maternal body mass index (BMI) at the time of index pregnancy (kg/m^2), maternal self-reported race/ethnicity (white non-Hispanic, black non-Hispanic, Hispanic, Asian, and other), parity at the time of the index pregnancy (any other live births > 20 weeks' gestation besides the two observed in this study vs. no other previous live births), and whether a woman experienced either diet-controlled or medication-requiring gestational diabetes in the index pregnancy. Weight change was calculated as the difference in BMI based on measured weight on admission for the index delivery compared with weight taken on admission for the subsequent delivery. We chose to use this measure, rather than other measures of weight change such as pre-pregnancy weight or gestational weight gain, for several reasons: patients entered pregnancy at all trimesters, outpatient records were not available for all patients, and pre-pregnancy weight in most cases was self-reported and thus subject to bias. Weight change was further categorized as loss of 2 kg/m^2 or more, loss of 2 kg/m^2 or gain of 2 kg/m^2, gain of 2 to 4 kg/m^2, and gain of 4 kg/m^2 or more. This classification scheme was adapted from Jain et al, who chose a BMI gain or loss of 2 kg/m^2 as a clinically meaningful amount of change (~12 pounds for a 5-foot 4-inch woman).[17]

For bivariable analyses of clinical and demographic variables deemed to be clinically relevant, Wilcoxon's rank-sum and Kruskal–Wallis' tests were used for continuous variables, and chi-square tests were used for categorical variables. Logistic regression was used for multivariable models. Variables were retained in multivariable analyses if they were associated with either the IDI or diabetes at the $p = 0.10$ level or less. All statistical analyses were performed in Stata Release 14.1 (StatCorp, College Station, TX). The Northwestern University Institutional Review Board approved this study with a waiver of informed consent (Protocol No. STU00202774, approved 4/4/16).

Results

Of 13,603 women with data on diabetes in both pregnancies, 0.51% ($N = 69$) had a diagnosis of type 1 diabetes, and 0.28% ($N = 38$) had a diagnosis of type 2 diabetes prior to the index pregnancy; these women were excluded from the analysis sample. Of the remaining 13,496 women, 90.9% ($N = 12,263$) had complete data available and comprised the final sample. The women without complete data ($N = 1,233$) were all missing data on race and ethnicity; these women were excluded from the final sample. Women missing data on race and ethnicity did not differ with regard to likelihood of diabetes in the subsequent

pregnancy ($p = 0.85$) or distribution of IDI ($p = 0.22$). In the final sample, 11.9% ($N =$ 1,457) had an IDI < 18 months, 83.8% ($N = 10,273$) had an IDI of 18 to 60 months, and 4.4% ($N = 533$) had an IDI of 61 months or more. Women with longer IDIs were more likely to have interval BMI increase (►Table 1). Women with longer IDIs were also more likely to have been multiparous at the index pregnancy, younger at the index pregnancy, and were more likely to be Hispanic race.

A total of 500 women (4.1%) developed any type of diabetes at the time of the subsequent delivery. Of these 500 women, 5.4% ($N = 27$) developed pregestational type 2 diabetes, 62.8% ($N = 314$) developed diet-controlled gestational diabetes, and 31.8% ($N = 159$) developed medication-requiring gestational diabetes. Of the 393 women who experienced gestational diabetes in the index pregnancy, 53.4% ($N = 210$) also carried a diagnosis of diabetes at the time of subsequent delivery; of these women with subsequent diabetes after prior gestational diabetes, 8.6% ($N = 18$) developed pregestational diabetes, whereas 47.6% ($N = 100$) developed diet-controlled gestational diabetes and 43.8% ($N = 92$) developed medication-requiring gestational diabetes. A longer IDI was associated with a higher likelihood of developing pregestational or medication-requiring gestational diabetes in both the overall cohort and the subgroup who had gestational diabetes in the index pregnancy, although the frequency of diabetes was substantially higher in the group with prior gestational diabetes (Table 2). Notably, in the subgroup of women with gestational diabetes in the index pregnancy who had an IDI of >60 months, 79% had recurrent diabetes, including 26.3% with pregestational diabetes at the time of the subsequent pregnancy (Table 2).

Women who had diabetes in the subsequent pregnancy were more likely to have a long IDI (9.0 vs. 4.2%, $p < 0.001$) and gestational diabetes in the index pregnancy (Table 3). Additionally, on bivariable analyses, women with diabetes in the subsequent pregnancy were more likely to have gained weight, had higher BMI at the index pregnancy, had greater parity, were older, and less likely to be non-Hispanic white (Table 3). Even after controlling for these potential confounders, a long IDI remained significantly associated with developing diabetes at the time of the subsequent delivery (adjusted odds ratio [aOR] = 2.13, 95% confidence interval [CI] = 1.44–3.15) compared with an 18- to 60-month IDI. Aside from interval BMI change and parity, each of the other factors remained significantly associated with diabetes in the subsequent pregnancy (Table 3). Women with subsequent diabetes were much more likely to have medication-requiring gestational diabetes in the index pregnancy (19.8 vs. 0.2%, $p < 0.001$), which persisted on multivariable analysis (aOR = 113.4, 95% CI = 70.4–182.7). Women who had a short IDI were not different, in terms of their likelihood of diabetes at the time of the subsequent delivery, from women with an intermediate IDI.

To account for the possibility of history of gestational diabetes in a prior (unobserved) pregnancy, which is associated with recurrent gestational diabetes, we conducted a sensitivity analysis restricting the sample to 10,398 women who were nulliparous at the time of the index pregnancy. There was no change in magnitude, direction, or statistical significance between an IDI > 60 months, compared with IDI of 18 to 60 months, and development of diabetes in the subsequent pregnancy (aOR = 2.19, 95% CI = 1.31–3.68).

Comment

Both gestational and pregestational diabetes have substantial implications for maternal and child short- and long-term health, and thus determining potential risk factors for the development of these comorbidities is essential to improving outcomes. In this cohort, although only a minority of women developed pregestational and gestational diabetes during or before delivery of a subsequent pregnancy, we identified time between pregnancies as a potentially modifiable risk factor for the development of diabetes. Additionally, this effect appeared to be most significant among the subgroup of women with gestational diabetes in the index pregnancy, for whom the majority developed pregestational or gestational diabetes with a long IDI. Although previous work has shown an association between a long IDI and development of diabetes in a subsequent pregnancy, they did not adjust for confounding factors such as maternal weight gain over time.[10] In contrast to previous hypotheses and data regarding interpregnancy weight gain as a mechanism increasing the risk of diabetes, our analysis showed weight gain between deliveries was not a statistically significant risk factor for subsequent diabetes diagnosis after accounting for other confounding factors, although BMI at the index pregnancy was a risk factor. In contrast to other studies, our data also did not show a significant difference in the likelihood of developing diabetes between women with a short IDI and women with an IDI of between 18 and 60 months.

The IDI is a risk factor that is potentially modifiable. Previous work has shown that approximately 8 to 27% of women will develop type 2 diabetes within 10 years of a pregnancy complicated by gestational diabetes,[18–20] and thus, shortening the IDI may allow women to complete childbearing in the time period before they develop overt diabetes.[21] To optimize subsequent pregnancy outcomes, contraception and family planning are key; however, previous research indicates women with pregestational and gestational diabetes may be less likely than women without diabetes to use effective contraception.[22,23] One recent national survey indicated that 20 to 40% of pregnancies following the longest interpregnancy intervals were unintended.[24] Unintended pregnancy among women with a history of gestational diabetes may be particularly important. For example, previous work has shown that the risk of developing gestational diabetes is additive across pregnancies, meaning that women who experience gestational diabetes in one pregnancy are at increased risk of gestational diabetes in every subsequent pregnancy.[25] In one previous study, approximately 40 to 60% of women with gestational diabetes developed recurrent gestational diabetes, consistent with our findings here.[26] Every subsequent additional pregnancy affected by gestational diabetes increases the risk of type 2 diabetes development above and beyond the risk associated simply with the passage of time.[27] The obstetric and neonatal risks of gestational diabetes also compound over pregnancies, with women who experience gestational diabetes in two sequential pregnancies exhibiting a higher likelihood of shoulder dystocia and preterm birth than women with gestational diabetes only in the second of two sequential pregnancies.[28] Thus, avoiding unintended pregnancies, especially following long intervals, could reduce risks in future pregnancies as well as the overall risk of progression to diabetes later in life.

This study has many strengths, including a large, ethnically diverse sample of pregnant women. We were able to obtain detailed clinical data regarding diabetes diagnoses

(including in a previous pregnancy), weight change, and clinically important potential confounders, and were able to allow women to serve as their own controls. Additionally, women received care within a stable group of health care providers who diagnose pregestational and gestational diabetes according to standardized protocols. However, there are also several limitations to note. First, we cannot infer causality from this observational study, and as with any large cohort study, there remains the risk of residual confounding. In particular, we cannot control for unobserved pregnancies that may have occurred outside of the two pregnancies studied here, although limiting the sample to women who are nulliparous prior to the index delivery does not change our conclusions. We also cannot control for all confounders of the relationship between delivery interval and diabetes, such as polycystic ovarian syndrome, associated both with infertility (and thus a longer IDI) and diabetes, or genetic predisposition to diabetes. These data come from a single large, tertiary care center, which may have a distinct patient population that limits generalizability to other settings. Indeed, the rate of diabetes in this sample approaches the lower bound of that found in other settings, likely because Northwestern has a high volume of deliveries of relatively low-risk women compared with many other tertiary care centers.

In conclusion, this study affirms that a lengthy IDI is an independent risk factor for development of diabetes in a subsequent pregnancy, even after controlling for history of gestational diabetes as well as maternal age and weight gain. Future directions could explore the use of contraception to modify pregnancy intervals to see if this diminishes the risks of developing de novo or recurrent gestational diabetes. Additionally, although weight gain between pregnancies was not shown to be associated with subsequent diabetes, BMI at the index pregnancy was suggesting that future work on the optimizing maternal health and metabolic status prior to pregnancy, as well as between pregnancies, remains utmost importance for women’s long-term health.

Acknowledgments

Funding

Research reported in this publication was supported, in part, by the National Institutes of Health’s National Center for Advancing Translational Sciences, Grant Number UL1TR001422. L.M.Y. is supported by the NICHD K12 HD050121-11. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

References

1. Committee on Practice Bulletins—Obstetrics. ACOG Practice Bulletin No. 190: gestational diabetes mellitus. Obstet Gynecol 2018; 131(02):e49–e64 [PubMed: 29370047]
2. Starikov R, Dudley D, Reddy UM. Stillbirth in the pregnancy complicated by diabetes. Curr Diab Rep 2015;15(03):11 [PubMed: 25667005]
3. Admon LK, Winkelman TNA, Moniz MH, Davis MM, Heisler M, Dalton VK. Disparities in chronic conditions among women hospitalized for delivery in the United States, 2005-2014. Obstet Gynecol 2017;130(06):1319–1326 [PubMed: 29112666]
4. Hauck YL, Lewis L, Nathan EA, White C, Doherty DA. Risk factors for severe perineal trauma during vaginal childbirth: a Western Australian retrospective cohort study. Women Birth 2015;28(01):16–20 [PubMed: 25476878]

5. Kim SY, Kotelchuck M, Wilson HG, Diop H, Shapiro-Mendoza CK, England LJ. Prevalence of adverse pregnancy outcomes, by maternal diabetes status at first and second deliveries, Massachusetts, 1998-2007. Prev Chronic Dis 2015;12:E218 [PubMed: 26652218]
6. Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS; Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) Trial Group. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. N Engl J Med 2005;352 (24):2477–2486 [PubMed: 15951574]
7. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. Lancet 2009;373(9677):1773–1779 [PubMed: 19465232]
8. Page KA, Romero A, Buchanan TA, Xiang AH. Gestational diabetes mellitus, maternal obesity, and adiposity in offspring. J Pediatr 2014;164(04):807–810 [PubMed: 24388326]
9. Holmes HJ, Lo JY, McIntire DD, Casey BM. Prediction of diabetes recurrence in women with class A1 (diet-treated) gestational diabetes. Am J Perinatol 2010;27(01):47–52 [PubMed: 19806532]
10. Khambalia AZ, Ford JB, Nassar N, Shand AW, McElduff A, Roberts CL. Occurrence and recurrence of diabetes in pregnancy. Diabet Med 2013;30(04):452–456 [PubMed: 23323841]
11. Ehrlich SF, Hedderson MM, Feng J, Davenport ER, Gunderson EP, Ferrara A. Change in body mass index between pregnancies and the risk of gestational diabetes in a second pregnancy. Obstet Gynecol 2011;117(06):1323–1330 [PubMed: 21606742]
12. Xiang AH, Kjos SL, Takayanagi M, Trigo E, Buchanan TA. Detailed physiological characterization of the development of type 2 diabetes in Hispanic women with prior gestational diabetes mellitus. Diabetes 2010;59(10):2625–2630 [PubMed: 20682697]
13. Hanley GE, Hutcheon JA, Kinniburgh BA, Lee L. Interpregnancy interval and adverse pregnancy outcomes: an analysis of successive pregnancies. Obstet Gynecol 2017;129(03):408–415 [PubMed: 28178044]
14. Conde-Agudelo A, Rosas-Bermudez A, Kafury-Goeta AC. Birth spacing and risk of adverse perinatal outcomes: a meta-analysis. JAMA 2006;295(15):1809–1823 [PubMed: 16622143]
15. Yee LM, Truong YN, Caughey AB, Cheng YW. The association between interdelivery interval and adverse perinatal outcomes in a diverse US population. J Perinatol 2016;36(08):593–597 [PubMed: 27031319]
16. ACOG Committee on Practice Bulletins. ACOG Practice Bulletin. Clinical Management Guidelines for Obstetrician-Gynecologists. Number 60, March 2005. Pregestational diabetes mellitus. Obstet Gynecol 2005;105(03):675–685 [PubMed: 15738045]
17. Jain AP, Gavard JA, Rice JJ, Catanzaro RB, Artal R, Hopkins SA. The impact of interpregnancy weight change on birthweight in obese women. Am J Obstet Gynecol 2013;208(03):205.e1–205.e7 [PubMed: 23246318]
18. Kjos SL, Peters RK, Xiang A, Thomas D, Schaefer U, Buchanan TA. Contraception and the risk of type 2 diabetes mellitus in Latina women with prior gestational diabetes mellitus. JAMA 1998;280 (06):533–538 [PubMed: 9707143]
19. Feig DS, Zinman B, Wang X, Hux JE. Risk of development of diabetes mellitus after diagnosis of gestational diabetes. CMAJ 2008;179(03):229–234 [PubMed: 18663202]
20. Kim C, 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]
21. Eades CE, Styles M, Leese GP, Cheyne H, Evans JM. Progression from gestational diabetes to type 2 diabetes in one region of Scotland: an observational follow-up study. BMC Pregnancy Childbirth 2015;15:11 [PubMed: 25643857]
22. Perritt JB, Burke A, Jamshidli R, Wang J, Fox M. Contraception counseling, pregnancy intention and contraception use in women with medical problems: an analysis of data from the Maryland Pregnancy Risk Assessment Monitoring System (PRAMS). Contraception 2013;88(02):263–268 [PubMed: 23245354]
23. Schwarz EB, Braughton MY, Riedel JC, et al. Postpartum care and contraception provided to women with gestational and preconception diabetes in California's Medicaid program. Contraception 2017 ;96(06):432–438 [PubMed: 28844877]

24. Ahrens KA, Thoma M, Copen C, Frederiksen B, Decker E, Moskosky S. Unintended pregnancy and interpregnancy interval by maternal age, National Survey of Family Growth. Contraception 2018; 98(01):52–55 [PubMed: 29501647]

25. Getahun D, Fassett MJ, Jacobsen SJ. Gestational diabetes: risk of recurrence in subsequent pregnancies. Am J Obstet Gynecol 2010;203(05):467.e1–467.e6 [PubMed: 20630491]

26. England L, Kotelchuck M, Wilson HG, et al. Estimating the recurrence rate of gestational diabetes mellitus (GDM) in Massachusetts 1998–2007: methods and findings. Matern Child Health J 2015;19(10):2303–2313 [PubMed: 26045058]

27. Peters RK, Kjos SL, Xiang A, Buchanan TA. Long-term diabetogenic effect of single pregnancy in women with previous gestational diabetes mellitus. Lancet 1996;347(8996):227–230 [PubMed: 8551882]

28. Boghossian NS, Yeung E, Albert PS, et al. Changes in diabetes status between pregnancies and impact on subsequent newborn outcomes. Am J Obstet Gynecol 2014;210(05):431.e1–431.e14 [PubMed: 24361790]

Am J Perinatol. Author manuscript; available in PMC 2020 April 29.

Table 1

Factors associated with differing interdelivery intervals

Variable	Interdelivery interval < 18 months	Interdelivery interval 18–60 months	Interdelivery interval > 60 months	*p*-Value[a]
Diabetes in the index pregnancy				
No diabetes	1,392 (95.5)[b]	9,964 (97.0)	514 (96.4)	0.02
Diet-controlled gestational diabetes	48 (3.3)	211 (2.1)	11 (2.1)	
Medication-requiring gestational diabetes	17 (1.2)	98 (1.0)	8 (1.5)	
BMI change				
Lost > 2 kg/m^2	148 (10.2)	921 (9.0)	33 (6.2)	< 0.001
Lost < 2 to gained < 2 kg/m^2	1,149 (78.9)	8,073 (78.6)	318 (59.7)	
Gained 2–4 kg/m^2 or more	116 (8.0)	884 (8.6)	92 (17.3)	
Gained 4 kg/m^2 or more	44 (3.0)	395 (3.9)	90 (16.9)	
Parity[c]				
One	1,200 (82.4)	8,867 (86.3)	331 (62.1)	< 0.001
More than one	257 (17.6)	1,406 (13.7)	202 (37.9)	
Maternal BMI at index pregnancy (kg/m^2)[d]	28.9 (26.6, 32.8)	28.5 (26.1, 31.7)	29.4 (26.6, 33.1)	< 0.001
Maternal age (years)[d]	31.5 (28.4, 34.3)	31.6 (29.3, 33.9)	28.6 (23.3, 31.9)	< 0.001
Maternal race				
White non-Hispanic	943 (64.7)	7,392 (72.0)	168 (31.5)	< 0.001
Black non-Hispanic	154 (10.6)	544 (5.3)	70 (13.1)	
Hispanic	285 (19.6)	1,663 (16.2)	260 (48.8)	
Asian	75 (5.2)	674 (6.6)	35 (6.6)	
N	1,457	10,273	533	

Abbreviation: BMI, body mass index

[a]The *p*-value for Kruskal–Wallis' tests for continuous variables and chi-square tests for categorical variables.

[b]Data are presented as median ± interquartile range for continuous variable or *N* (%) for categorical variables.

[c]Measured at the time of subsequent delivery.

[d] Measured at the time of index delivery.

Table 2

Prevalence of diabetes in the subsequent pregnancy

Variable	Overall cohort	Interdelivery interval < 18 months	Interdelivery interval 18–60 months	Interdelivery interval > 60 months	*p*-Value[a]
Overall cohort					
No diabetes	11,763 (95.9)[b]	1,397 (95.9)	9,877 (96.2)	489 (91.7)	< 0.001
Diet-controlled gestational diabetes	314 (2.6)	42 (2.9)	253 (2.5)	19 (3.6)	
Medication-requiring gestational diabetes	159 (1.3)	13 (0.9)	128 (1.3)	18 (3.4)	
Pregestational diabetes	27 (0.2)	5 (0.3)	15 (0.2)	7 (1.3)	
N	12,263	1,457	10,273	533	
Women with gestational diabetes in the index pregnancy					
No diabetes	183 (46.6)	34 (52.3)	145 (46.9)	4 (21.1)	< 0.001
Diet-controlled gestational diabetes	100 (25.5)	17 (26.2)	79 (25.6)	4 (21.1)	
Medication-requiring gestational diabetes	92 (23.4)	10 (15.4)	76 (24.6)	6 (31.6)	
Pregestational diabetes	18 (4.6)	4 (6.2)	9 (2.9)	5 (26.3)	
N	393	65	309	19	

[a]The *p*-value for chi-square tests.

[b]Data are presented as *N* (%).

Table 3

Factors associated with interval development of type 2 diabetes or gestational diabetes in a subsequent pregnancy

Variable	Diabetes	No diabetes	*p*-Value[a]	aOR	95% CI
Time between deliveries					
< 18 months	60 (12.0)[b]	1,397 (11.9)	< 0.001	0.76	0.55–1.07
18–60 months	396 (79.0)	9,877 (84.0)		Ref	
> 60 months	45 (9.0)	488 (4.2)		2.13	1.44–3.15
No gestational diabetes in the index pregnancy	290 (58.0)	11,580 (98.4)	< 0.001	Ref	
Diet-controlled gestational diabetes in the index pregnancy	111 (22.2)	159 (1.4)		24.4	18.4–32.4
Medication-requiring gestational diabetes in the index pregnancy	99 (19.8)	24 (0.2)		113.4	70.4–182.7
BMI change					
Lost > 2 kg/m^2	60 (12.0)	1,042 (8.9)	0.003	1.17	0.83–1.63
Lost < 2 to gained < 2 kg/m^2	357 (71.3)	9,183 (78.1)		Ref	
Gained 2–4 kg/m^2 or more	53 (10.6)	1,039 (8.8)		0.85	0.59–1.22
Gained 4 kg/m^2 or more	31 (6.2)	498 (4.2)		1.09	0.68–1.75
Parity[c]					
One	379 (75.7)	10,019 (85.2)	< 0.001	Ref	
More than one	122 (24.4)	1,743 (14.8)		1.19	0.91–1.55
Maternal BMI (kg/m^2)[d]	31.4 (28.0, 36.2)	28.5 (26.1, 31.8)	< 0.001	1.07	1.05–1.09
Maternal age (y)[d]	32.1 (28.7, 34.9)	31.5 (29.1, 33.8)	0.01	1.04	1.01–1.06
Maternal race					
White non-Hispanic	235 (46.9)	8,268 (70.3)	< 0.001	Ref	
Black non-Hispanic	44 (8.8)	724 (6.2)		1.21	0.79–1.85
Hispanic	157 (31.3)	2,051 (17.4)		1.98	1.72–3.41
Asian	65 (13.0)	719 (6.1)		2.42	1.51–2.60
N	500	11,763		12,263	

Abbreviation: BMI, body mass index.

[a]The *p*-value for Wilcoxon's rank-sum tests for continuous variables and chi-square tests for categorical variables.

Am J Perinatol. Author manuscript; available in PMC 2020 April 29.

[b] Data are presented as median (interquartile range) for continuous variable or *N* (%) for categorical variables.

[c] Measured at the time of subsequent delivery.

[d] Measured at the time of index delivery.