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Original Research

Racial and Ethnic Inequities in Development of Type 2 Diabetes After Gestational Diabetes Mellitus

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OBJECTIVE: To estimate racial and ethnic disparities in type 2 diabetes mellitus after gestational diabetes mellitus (GDM) and to investigate baseline pregnancy clinical and social or structural characteristics as mediators.

METHODS: We conducted a retrospective cohort of individuals with GDM using linked 2009–2011 New York City birth and hospital data and 2009–2017 New York City A1c Registry data. We ascertained GDM and pregnancy characteristics from birth and hospital records. We classified type 2 diabetes as two hemoglobin A_{1c} test results of 6.5% or higher. We grouped pregnancy characteristics into clinical (body mass index [BMI], chronic hypertension, gestational hypertension, preeclampsia, preterm delivery, caesarean, breastfeeding, macrosomia, shoulder dystocia) and social or structural (education, Medicaid insurance, prenatal care, and WIC [Special Supplemental Nutrition Program for Women, Infants, and Children] participation). We used Cox proportional hazards models to estimate associations between race and ethnicity and 8-year type 2 diabetes incidence, and we tested mediation of pregnancy characteristics, additionally adjusting for age and nativity (U.S.-born vs foreign-born).

RESULTS: The analytic data set included 22,338 patients with GDM. The 8-year type 2 diabetes incidence was 11.7% overall and 18.5% in Black, 16.8% in South and Southeast Asian, 14.6% in Hispanic, 5.5% in East and Central Asian, and 5.4% in White individuals with adjusted hazard ratios of 4.0 (95% CI 2.4–3.9), 2.9 (95% CI 2.4–3.3), 3.3 (95% CI 2.7–4.2), and 1.0 (95% CI 0.9–1.4) for each group compared with White individuals. Clinical and social or structural pregnancy characteristics explained 9.3% and 23.8% of Black, 31.2% and 24.7% of Hispanic, and 7.6% and 16.3% of South and Southeast Asian compared with White disparities. Associations between education, Medicaid insurance, WIC participation, and BMI and type 2 diabetes incidence were more pronounced among White than Black, Hispanic, and South and Southeast Asian individuals.

CONCLUSION: Population-based racial and ethnic inequities are substantial in type 2 diabetes after GDM. Characteristics at the time of delivery partially explain disparities, creating an opportunity to intervene on life-course cardiometabolic inequities, whereas weak associations of common social or structural measures and BMI in Black, Hispanic and South and Southeast Asian individuals demonstrate the need for greater understanding of how structural racism influences postpartum cardiometabolic risk in these groups.

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Gestational diabetes mellitus (GDM) is diagnosed in about 8% of pregnant people in the United States, and the incidence has increased in all racial and ethnic subgroups in the past decade.[1] Racial and ethnic disparities in GDM exist, with South Asian and Hispanic individuals at the greatest risk.[2,3] These GDM disparities have profound implications for life-course disparities in type 2 diabetes mellitus and cardiovascular disease. Several recent meta-analyses have highlighted the increased risk of type 2 diabetes after GDM, estimating a type 2 diabetes risk of 10% at 5 years postpartum,[4] 20% at 10 years, and 50% at 40 years.[5] However, despite substantial evidence of racial and ethnic disparities in both GDM and type 2 diabetes, data are scarce on racial and ethnic differences in progression to type 2 diabetes after GDM.

Current mechanisms explaining racial and ethnic disparities in transition to type 2 diabetes after GDM are unknown. Structural racism, or interlocking systems of disadvantage attributable to historical and current racial oppression,[6] is likely a root cause of racial and ethnic disparities. Structural disadvantage intertwines with cultural differences between groups, such as diet, to shape diabetes risk.[7] From the vantage point of the clinician at the time of delivery, characteristics of pregnancy that are associated with differences in progression to type 2 diabetes may elucidate targets for intervention. Health care during pregnancy is a rare point of care with the health system for many people and therefore serves as an opportunity to intervene early to reduce life-course differences in type 2 diabetes.[8] However, research going beyond documenting differences to explaining them is scarce.

To fill this gap, we created the APPLE (A1c in Pregnancy and Postpartum Linkage for Equity) study, a novel 8-year retrospective cohort of 22,853 pregnancies in New York City. The primary objective of this analysis was to investigate population-level racial and ethnic disparities in time to type 2 diabetes after GDM. Our secondary objective was to estimate the contribution of pregnancy characteristics of the baseline pregnancy with GDM to incident type 2 diabetes in 8 years of follow-up. Finally, we examined whether differences exist in the relative importance of each pregnancy characteristic between racial and ethnic groups.

METHODS

We constructed a retrospective population-based cohort, the APPLE study, by linking vital registry records for all 2009–2011 New York City births with 2009–2017 A1c Registry data. Laboratory reporting of hemoglobin A_{1c} (Hb A_{1c}) results for New York City residents to the New York City Department of Health and Mental Hygiene has been mandated since 2005.[9] Hospital discharge data from the Statewide Planning and Research Cooperative System were linked with birth record data for the same period to strengthen measures for GDM and other pregnancy-related comorbidities. The Department of Health and Mental Hygiene performed data linkages using a systematic matching algorithm incorporating maternal identifiers and birth date. IRB approval was obtained by the Department of Health and Mental Hygiene, the New York State Department of Health, and the Icahn School of Medicine at Mount Sinai.

We identified individuals with GDM at their index pregnancy by whether GDM was indicated on neonatal birth records (n=16,089) or on the delivery hospital record (International Classification of Diseases, Ninth Revision [ICD-9] codes 648.01–648.04 for the same birth [n=19,814], Appendix 1, available online at available online at http://links.lww.com/AOG/D335). The two sources showed high agreement (κ=0.70), and we classified individuals as cases if GDM was indicated in either source[10] (6.7% of all births, n=23,027). We excluded individuals with Hb A_{1c} test results with values of 6.5% or higher in the first trimester and individuals with known diabetes before the start of the second trimester (pregestational diabetes indicated on the hospital record [ICD-9 codes 250.x, 362.01, 362.02, 363.04–363.07, 366.41] or birth record).

Following the American Diabetes Association recommendations for using Hb A_{1c} to screen for type 2 diabetes,[11] we identified the date of type 2 diabetes onset as the second date of an Hb A_{1c} test with a value of 6.5% or higher. (We also conducted a sensitivity analysis using the date of the first of the two elevated Hb A_{1c} test results to determine onset.) To allow for the distinction in the transition from GDM to type 2 diabetes, we began observation at 12 weeks postpartum. We calculated survival time in years by subtracting the date at 12 weeks postpartum from the date of type 2 diabetes onset.

We defined race and ethnicity as a social construct, representing intergenerational experiences of racism.[12] We ascertained self-reported race and Hispanic ethnicity from the birth record. We categorized individuals identifying as Hispanic of any race as Hispanic. We categorized individuals not of Hispanic origin as Black, East and Central Asian, South and Southeast Asian, and White. We created the two groups of Asian individuals because of previous research demonstrating large differences in GDM risk between South and East Asian groups, whereas groups of differing geographic

origin of Black and Hispanic individuals had more similar risk.[3] We excluded individuals in categories different from these to avoid combining individuals with no theoretical basis.[13] We chose clinical and social or structural characteristics as potential mediators on the basis of previous literature on predictors of type 2 diabetes after GDM and social or structural determinants of diabetes.[14] Clinical characteristics, including previous live births (0, 1, 2, or 3), multiple gestation, prepregnancy body mass index (BMI, calculated as weight in kilograms divided by height in meters squared), macrosomia (birth weight more than 4,500 g), cesarean delivery, and exclusive breastfeeding at discharge, were obtained from the birth record of the index pregnancy. Other clinical characteristics were ascertained from a combination of the hospital and birth records.[15] We ascertained prepregnancy hypertension using ICD-9 codes 401.x–405.x, 642.0x–642.2x, or chronic hypertension indicated on the birth record. Preeclampsia was identified as preeclampsia with or without severe features (42.4x–642.6x), preexisting hypertension with superimposed preeclampsia (642.7x), or eclampsia indicated on the birth record. Gestational hypertension was identified by codes 642.3x or indication on the birth record. Neonatal shoulder dystocia was indicated by codes 660.4, 600.41, and 600.43. Social or structural characteristics included education (less than college degree vs college graduate or higher), insurance status (Medicaid insurance vs private insurance), early prenatal care (care initiated in the first trimester), and enrollment in the WIC (Special Supplemental Nutrition Program for Women, Infants, and Children) program as a proxy for income level. We adjusted all models for maternal age and nativity (U.S.-born vs foreign-born). We obtained alcohol, tobacco, or drug use from the birth record; however, we omitted them from multivariable models because of low prevalence.

Cumulative incidence rates of type 2 diabetes among those with no prior diagnosed diabetes over a 6- to 8-year period by race and ethnicity were estimated by the Kaplan–Meier method.[16] Stratified Cox proportional hazards models were used to estimate the hazard of type 2 diabetes diagnosis for each racial and ethnic group relative to White respondents, with adjustment for age and nativity. For all models, we examined the proportional hazard assumption using log(–log) plots and by examining Schoenfeld residuals. Missing values were less than 1% for variables except BMI (3.4%) and early prenatal care (1.7%) and were excluded from multivariable models.

Next, we examined causal mediation between race and ethnicity and time to type 2 diabetes diagnosis by clinical and social or structural variable sets using inverse odds ratio weights.[17] The inverse odds ratio weight is a semiparametric weight-based approach to causal mediation analysis that can be accommodated within survival models. We applied the inverse odds ratio weight for each racial and ethnic group relative to White individuals. The approach applies the inverse odds ratio estimate of the association between the mediator(s) on the exposure as a weight to decompose the total effect in a weighted Cox regression. The difference between the total effect (ie, the standard Cox regression model of exposure on outcome omitting the inverse odds ratio weight) and direct effect (the same model Cox model but applying the inverse odds ratio weight) is the indirect effect (ie, the effect estimate attributed to the mediators).[18] We calculated the percentage of the total effect explained by mediators by dividing the indirect effect by the total effect and used bootstrapping to calculate 95% CIs (5,000 iterations).

Finally, we added exposure–mediator interaction terms to the overall Cox model. Although the inverse odds ratio weight is robust to exposure–mediator interaction, we were interested in testing whether the strength of associations between pregnancy characteristics and type 2 diabetes varied by race and ethnicity. To account for multiple comparisons, we calculated the Holm–Bonferroni P value. For interaction terms with $P<.05$, we present adjusted hazard ratios (HRs) stratified by race and ethnicity.

Because our cohort was created by linking administrative data sets, we used probabilistic bias analysis to assess the potential effect of type 2 diabetes misclassification or loss to follow-up on our estimated measures of association.[19,20] Details of the probabilistic bias analysis are given in Appendix 2, available online at http://links.lww.com/AOG/D335. Finally, we conducted a sensitivity analysis by estimating the Cox proportional hazard model using only people with Hb A_{1c} measures.

RESULTS

The analytic data set included 22,338 patients with GDM: 4,687 Black (20.5%), 2,450 East and Central Asian (11.0%), 7,062 Hispanic (30.9%), 2,429 South and Southeast Asian (10.9%), and 4,225 White (18.5%) individuals (Table 1). Of individuals identified with GDM, 78.3% had an Hb A_{1c} test during the follow-up period. Among Black individuals, the percent was 82.3%; White individuals, 67.9%; Hispanic individuals, 81.9%; and Asian individuals, 77.8%. Those with and without Hb A_{1c} values had similar characteristics (Appendix 3, available online

Table 1. Baseline Characteristics of Individuals With Gestational Diabetes Who Gave Birth Between 2009 and 2011, by Race and Ethnicity (N=22,338)

Characteristic	Total	Race and Ethnicity				
		Black [4,687 (20.5)]	East and Central Asian [2,450 (11.0)]	Hispanic [7,062 (30.9)]	South and Southeast Asian [2,429 (10.9)]	White [4,225 (18.5)]
Age (y)						
10–19	316 (1.4)	105 (2.2)	2 (0.1.)	174 (2.5)	6 (0.3)	20 (0.5)
20–29	7,129 (31.9)	1,582 (33.8)	814 (31.4)	2,604 (36.9)	914 (37.6)	862 (20.4)
30–39	12,705 (56.9)	2,500 (53.3)	1,544 (59.6)	3,681 (52.1)	1,369 (56.4)	2,726 (64.5)
40 and older	2,188 (9.8)	500 (10.7)	232 (9.0)	603 (8.5)	140 (5.8)	617 (14.6)
Nativity						
U.S.-born	7,204 (32.2)	1,990 (42.5)	7 (0.3)	2,288 (32.4)	15 (0.6)	2,557 (60.5)
Foreign-born	15,134 (67.8)	2,697 (57.5)	2,585 (99.7)	4,774 (67.6)	2,414 (99.4)	1,668 (39.5)
Education level						
Less than high school	5,925 (26.5)	900 (19.4)	893 (34.5)	2,947 (41.8)	561 (23.1)	338 (8.0)
High school	5,121 (22.9)	1,324 (28.2)	496 (19.1)	1,692 (23.9)	518 (21.3)	680 (16.1)
Some college	3,433 (15.4)	1,073 (23.9)	233 (9.0)	1,124 (15.9)	275 (11.2)	525 (12.4)
College or higher	8,282 (37.1)	1,349 (28.8)	970 (37.4)	1,281 (18.1)	1,070 (44.1)	2,671 (63.2)
Missing	77 (0.8)	41 (0.9)	1 (0.01)	18 (0.3)	5 (0.2)	12 (0.3)
Insurance						
Private	7,846 (35.4)	3,279 (70.0)	837 (32.4)	5,516 (78.1)	1,862 (76.7)	2,836 (67.1)
Medicaid or none	14,294 (64.6)	1,329 (28.3)	1,747 (67.6)	1,496 (21.2)	540 (22.2)	1,368 (32.4)
Missing	198 (0.8)	79 (1.7)	0 (0.0)	50 (0.7)	27 (1.1)	21 (0.5)
Prenatal care in 1st trimester						
No	6,387 (28.6)	1,629 (34.7)	639 (25.4)	2,083 (29.5)	828 (34.4)	813 (19.2)
Yes	15,187 (68.0)	2,840 (60.6)	1,878 (74.6)	4,723 (66.9)	1,554 (64.0)	3,281 (77.7)
Missing	764 (3.4)	220 (4.7)	0 (0.0)	257 (3.6)	47 (1.9)	131 (3.1)
WIC participation						
No	13,045 (58.4)	3,372 (71.9)	969 (37.5)	4,952 (70.1)	1,515 (62.4)	3,127 (75.1)
Yes	9,166 (41.0)	1,278 (27.3)	1,613 (62.5)	2,081 (29.5)	894 (36.8)	1,070 (25.3)
Missing	127 (0.6)	37 (0.8)	0 (0.0)	29 (0.4)	20 (0.8)	31 (0.7)
BMI category						
Underweight	690 (3.1)	54 (1.2)	310 (12.0)	88 (1.2)	67 (2.8)	122 (2.9)
Normal	8,634 (38.7)	1,017 (21.7)	1,854 (71.5)	1,964 (27.8)	1,159 (47.7)	1,959 (46.4)
Obesity	6,341 (28.4)	1,400 (29.9)	321 (12.4)	2,331 (33.0)	826 (34.0)	1,075 (25.4)
Morbid obesity	6,301 (29.1)	2,103 (44.9)	90 (3.5)	2,538 (35.9)	350 (14.4)	1,009 (23.9)
Missing	372 (1.7)	113 (2.4)	17 (0.7)	141 (2.0)	27 (1.1)	3 (0.1)
Multiple gestation pregnancy						
No	21,859 (97.9)	76 (1.6)	45 (1.7)	105 (1.5)	36 (1.5)	182 (4.3)
Yes	479 (2.1)	4,611 (98.4)	2,547 (98.3)	6,957 (98.5)	2,393 (98.5)	4,043 (95.7)
Previous live births						
0	9,237 (41.4)	1,773 (37.8)	1,277 (49.3)	2,378 (33.7)	921 (38.0)	2,164 (51.3)
1	6,767 (30.3)	1,355 (28.9)	987 (38.1)	2,113 (29.9)	866 (35.6)	1,034 (24.5)
2	3,626 (16.3)	822 (17.5)	274 (10.6)	1,531 (21.7)	412 (17.0)	456 (10.8)
3 or more	2,689 (12.1)	728 (15.5)	53 (2.1)	1,035 (14.7)	228 (9.4)	568 (13.5)
Missing	20 (0.1)	9 (0.2)	1 (0.04)	5 (0.1)	2 (0.1)	3 (0.1)
Prepregnancy hypertension*						
No	20,983 (93.9)	4,135 (88.2)	2,553 (98.5)	6,640 (94)	2,308 (95)	4,063 (96.2)
Yes	1,355 (6.1)	552 (11.8)	39 (1.5)	422 (6)	121 (5)	162 (3.8)
Gestational hypertension*						
No	20,456 (91.6)	4,217 (90)	2,495 (96.3)	6,331 (89.7)	2,288 (94.2)	3,885 (92)
Yes	1,882 (8.4)	470 (10)	97 (3.7)	731 (10.4)	141 (5.8)	340 (8.1)

(*continued*)

Table 1. Baseline Characteristics of Individuals With Gestational Diabetes Who Gave Birth Between 2009 and 2011, by Race and Ethnicity (N=22,338) (*continued*)

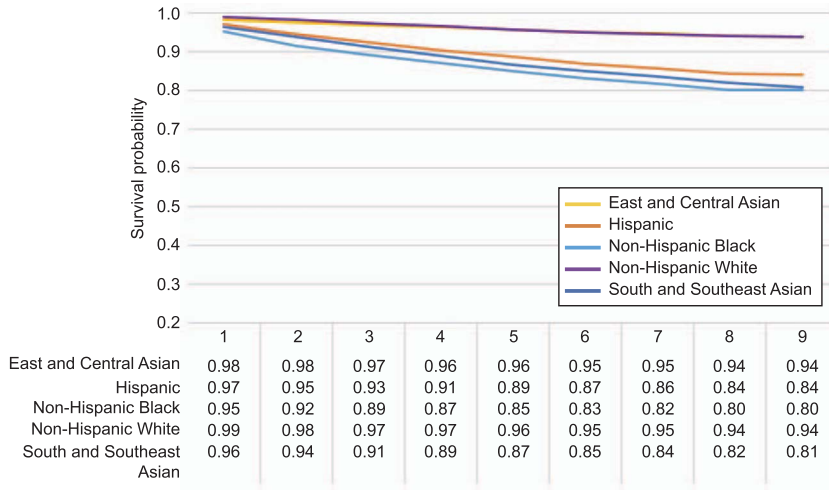
Characteristic	Total	Race and Ethnicity				
		Black [4,687 (20.5)]	East and Central Asian [2,450 (11.0)]	Hispanic [7,062 (30.9)]	South and Southeast Asian [2,429 (10.9)]	White [4,225 (18.5)]
Preeclampsia*,†						
No	20,321 (90.8)	4,021 (85.8)	2,507 (96.7)	6,306 (89.3)	2,288 (94.2)	3,940 (93.3)
Yes	2,017 (9.0)	666 (14.2)	85 (3.3)	756 (10.7)	141 (5.8)	285 (6.8)
Smoked in 3 mo before or during this pregnancy						
No	21,807 (97.6)	4,526 (96.6)	2,563 (98.9)	6,885 (97.5)	2,422 (99.7)	4,082 (96.6)
Yes	506 (2.3)	148 (3.2)	28 (1.1)	168 (2.4)	6 (0.3)	141 (3.3)
Missing	26 (0.1)	13 (0.3)	1 (0.04)	9 (0.1)	1 (0.04)	2 (0.05)
Alcohol use during this pregnancy						
No	22,151 (99.2)	4,622 (99.2)	2,573 (99.4)	7,006 (99.3)	2,409 (99.2)	4,191 (99.2)
Yes	143 (0.6)	35 (0.8)	13 (0.50)	43 (0.6)	16 (0.7)	27 (0.67
Missing	26 (0.2)	2 (0.04)	2 (0.08)	13 (0.2)	3 (0.12)	6 (0.1)
Cesarean birth						
No	12,549 (56.2)	2,389 (51)	1,724 (66.5)	3,913 (55.4)	1,322 (54.4)	2,412 (57.1)
Yes	9,789 (43.8)	2,298 (49)	868 (33.5)	3,149 (44.6)	1,107 (45.6)	1,813 (42.9)
Preterm delivery						
No	19,538 (87.5)	3,887 (82.9)	2,357 (90.9)	6,180 (87.5)	2,152 (88.6)	3,787 (89.6)
Yes	2,797 (12.5)	800 (17.1)	235 (9.1)	882 (12.5)	277 (11.4)	438 (10.4)
Macrosomia						
No	20,283 (90.8)	4,158 (88.9)	2,451 (94.6)	6,272 (88.8)	2,320 (95.5)	3,805 (90.1)
Yes	2,041 (9.1)	519 (11.1)	141 (5.4)	789 (11.2)	109 (4.5)	418 (9.9)
Shoulder dystocia*						
No	22,330 (99.2)	4,650 (99.2)	2,582 (99.6)	6,987 (98.9)	2,413 (99.3)	4,201 (99.4)
Yes	179 (0.8)	37 (0.8)	10 (0.4)	75 (1.1)	16 (0.7)	24 (0.6)
Exclusive breastfeeding						
No	16,331 (73.1)	3,666 (78.2)	2,156 (83.2)	5,411 (76.6)	1,811 (74.6)	2,403 (56.9)
Yes	6,007 (26.9)	1,021 (21.8)	436 (16.8)	1,651 (23.4)	618 (25.4)	1,822 (43.1)

WIC, Special Supplemental Nutrition Program for Women, Infants, and Children; BMI, body mass index.
Data are n (%).
* Indicated by hospital discharge codes or on birth certificate.
† Superimposed hypertension, mild, moderate, or severe.

at http://links.lww.com/AOG/D335). We conducted sensitivity analyses in this Hb A_{1c}–screened subsample (n=14,392).

Characteristics of study participants are given in Table 1. The cumulative incidence of type 2 diabetes was 11.7% (n=2,675) by the end of the 8-year follow-up (Fig. 1). The cumulative incidence of type 2 diabetes among individuals by race and ethnicity was 18.5% of Black, 16.8% of South and Southeast Asian, 14.6% of Hispanic, 5.5% of East and Central Asian, and 5.4% of White individuals. After adjustment for age and nativity, the following groups had elevated type 2 diabetes incidence relative to White individuals: Black (adjusted HR 4.0, 95% CI 2.4–3.9), South and Southeast Asian (adjusted HR 3.3, 95% CI 2.7–4.2), and Hispanic (adjusted HR 2.9, 95% CI 2.4–3.3) individuals (Table 2).

Combined clinical and social or structural factors explained the largest percentage of the association between race and ethnicity on type 2 diabetes among Hispanic (45.8%, 95% CI 28.6–58.0%) followed by Black (26.7%, 95% CI 11.5–32.5%) individuals (Table 2). In contrast, combined mediators explained the least percentage among South and Southeast Asian individuals (14.1%, 95% CI 3.9–24.8%). Among both Hispanic and Black individuals, clinical mediators related to pregnancy mediated a larger percentage of association than social or structural factors. For example, among Black individuals, clinical mediators explained 23.8% (95% CI 12.9–29.9%) of association, and social or structural mediators accounted for only 9.3% of the mediated effect (95% CI 4.0–16.5%). In contrast, among South and Southeast Asian individuals, social or structural mediators accounted for a larger percentage of association in diabetes risk (16.3%, 95% CI 9.6–23.8%) than clinical mediators (7.6%, 95% CI 1.8–18.8%). We did not conduct mediation analysis on East and Central Asian compared



	1	2	3	4	5	6	7	8	9
East and Central Asian	0.98	0.98	0.97	0.96	0.96	0.95	0.95	0.94	0.94
Hispanic	0.97	0.95	0.93	0.91	0.89	0.87	0.86	0.84	0.84
Non-Hispanic Black	0.95	0.92	0.89	0.87	0.85	0.83	0.82	0.80	0.80
Non-Hispanic White	0.99	0.98	0.97	0.97	0.96	0.95	0.95	0.94	0.94
South and Southeast Asian	0.96	0.94	0.91	0.89	0.87	0.85	0.84	0.82	0.81

Fig. 1. Kaplan–Meier product limit estimates for transition from gestational diabetes mellitus to type 2 diabetes mellitus, stratified by race and ethnicity, New York City, 2009–2017.

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with White individuals estimates because no disparity was found.

Interaction terms were significant for coefficients for maternal education, Medicaid insurance, WIC participation, and BMI. These characteristics were associated with a higher relative hazard of type 2 diabetes after GDM among White individuals than in Black, Hispanic, and South and Southeast Asian individuals (Table 3).

Adjusting for hypothetical screening bias with 15% underdiagnosis of type 2 diabetes among Black, Hispanic, and South and Southeast Asian groups

Table 2. Cumulative Incidence of Type 2 Diabetes Stratified by Race and Ethnicity and Percentage of Effect Explained by Pregnancy Characteristics

	Type 2 Diabetes		Adjusted HR (95% CI) Effect of Race and Ethnicity*		Percentage of Disparity (95% CI) Explained by†		
Race and Ethnicity	Total	Cumulative Incidence	Total Effect	Direct Effect Not Through Social or Structural and Clinical Mediators	Clinical Mediators‡	Social or Structural Mediators§	Social or Structural and Clinical Mediators
Black	4,687	866 (18.8)	4.0 (2.4–3.9)	2.8 (2.0–3.3)	23.8 (12.9–29.9)	9.3 (4.0–16.5)	26.7 (11.5–32.5)
East and Central Asian‖	2,450	143 (5.5)	1.0 (0.9–1.4)		NA	NA	NA
Hispanic	7,062	1,029 (14.6)	2.9 (2.4–3.3)	1.8 (1.4–2.2)	31.2 (15.4–39.4)	24.7 (13.9–36.7)	45.8 (28.6–58.0)
South or Southeast Asian	2,429	408 (16.8)	3.3 (2.7–4.2)	2.8 (2.1–3.8)	7.6 (1.8–18.8)	16.3 (9.6–23.8)	14.1 (3.9–24.8)
White	4,225	230 (5.4)	1.00	1.00	1.00	1.00	1.00

HR, hazard ratio; NA, not applicable.
Data are n or n (%) unless otherwise specified.
* All models are adjusted for age and nativity.
† Percentage of total effect explained by mediators was calculated using the following equation: [total effect−direct effect (weighted by inverse odds ratio weight)]/total effect, where the total effect is the effect estimate unadjusted by mediators, direct effect is the proportion of effect not explained through mediators (as applied in inverse odds ratio weight), and the difference between total and direct effect is the indirect effect (the decomposed effect operating through mediators). The percent effect mediated was measured on the log scale; bootstrapping was used to obtain SEs for 95% CI calculation.
‡ Preeclampsia, prepregnancy body mass index, prehypertension, gestational hypertension, preterm, caesarean vs vaginal delivery, exclusive breastfeeding at hospital discharge (yes or no), macrosomia, and neonatal shoulder dystocia during delivery.
§ Maternal education (less than college degree vs college or higher), Medicaid insurance or none vs private insurance, timely antenatal care (within first trimester), and enrollment in WIC (Special Supplemental Nutrition Program for Women, Infants, and Children).
‖ Mediation analysis not conducted on East and Central Asian vs White HR because of a lack of disparity.

Table 3. Associations Between Pregnancy Characteristics and Type 2 Diabetes After Gestational Diabetes for Characteristics for Which Effect Modification Is Present by Race and Ethnicity

	Adjusted HR (95% CI)*			
Characteristic	Black	Hispanic	South or Southeast Asian	White
Education level				
Less than college degree	1.3 (1.1–1.5)	1.4 (1.2, 1.6)	1.7 (1.4–2.1)	2.7 (2.2–3.6)
College degree or higher	1	1	1	1
Insurance				
Medicaid or none	1.1 (0.9–1.3)	1.6 (1.3–1.9)	1.5 (1.1–1.9)	3.6 (2.6–4.9)
Private insurance	1	1	1	1
WIC participation				
Yes	1.3 (1.1–1.5)	1.5 (1.3–1.8)	1.5 (1.2–1.9)	2.8 (2.1–3.7)
No	1	1	1	1
BMI	1.05 (1.04–1.06)	1.07 (1.06–1.08)	1.10 (1.08–1.12)	1.13 (1.11–1.15)

HR, hazard ratio; WIC, Special Supplemental Nutrition Program for Women, Infants, and Children; BMI, body mass index.
* All models adjusted for 5-year age group and nativity.

relative to 10% underdiagnosis of type 2 diabetes among White individuals would moderately bias HRs toward the null, although a strong magnitude of effect persisted (Appendix 4, available online at http://links.lww.com/AOG/D335). Results were very similar for HRs in the sensitivity analysis when the cohort was restricted to only those with Hb A_{1c} measures (Appendix 5, available online at http://links.lww.com/AOG/D335). In the sensitivity analysis, using the first of two elevated Hb A_{1c} test dates as disease onset results in racial and ethnic disparities of slightly greater magnitude (Appendix 6, available online at http://links.lww.com/AOG/D335).

DISCUSSION

We identified substantial racial disparities in the emergence of type 2 diabetes after GDM, with fourfold increased risk among Black individuals and threefold increased risk among Hispanic and South and Southeast Asian individuals relative to White individuals. Clinical and social or structural characteristics of pregnancy combined explained only 14% of disparities among South and Southeast Asian people compared to White people, 27% of disparities among Black people compared to White people, and 46% of disparities among Hispanic people compared to White people. No disparities were identified between East and Central Asian and White individuals. Measured social and social structural factors and BMI had less pronounced associations with type 2 diabetes for Black, Hispanic, and South and Southeast Asian individuals than for White individuals.

Our work builds on limited research on racial and ethnic disparities in type 2 diabetes after GDM. Similar to a study of a health insurance claims cohort in California, we found that the risk of type 2 diabetes after GDM was highest in Black individuals, second highest in Hispanic individuals, and lowest in a broadly defined category of Asian individuals.[21] In addition, as in our study, an administrative cohort in Canada found a higher risk of type 2 diabetes among South Asian women relative to White women.[22] The fact that Black postpartum individuals have been identified in multiple cohorts as being at high risk of type 2 diabetes after GDM, despite little to no heightened risk of GDM, is noteworthy. It is unclear whether this pattern is attributable to different phenotype of GDM or type 2 diabetes or whether the postpartum period after GDM fuels emergent disparities. For example, in our study, hypertension accompanying GDM was more prevalent among Black individuals. These findings highlight the key role that large population-based cohorts such as the APPLE cohort play in bringing to light populations at high risk.

Our results can be considered in the context of previous literature on predictors of type 2 diabetes after GDM. Recent meta-analyses of clinical predictors of type 2 diabetes among individuals with GDM found BMI, advanced maternal age, multiparity, hypertensive disorders, and preterm delivery to be important predictors of type 2 diabetes after GDM.[23] All of these factors besides advanced maternal age were more prevalent in Black and Hispanic individuals than in White individuals in our cohort. Our finding that clinical characteristics were not as important for South Asian individuals follows previous work showing that the population attributable risk of obesity for GDM was lower in Asian groups.[24] Very few studies have examined prenatal social or structural determinants of type 2 diabetes after GDM,

but one U.S. study found an association between lower maternal education during a pregnancy with GDM and higher risk of later type 2 diabetes.[25] We found that associations between education, Medicaid insurance, and WIC participation and later type 2 diabetes were less pronounced in Black, Hispanic, and South and Southeast Asian individuals than in White individuals. This finding is in line with previous literature suggesting that these common proxies for social or structural determinants of health may not capture comparable experiences across different racial and ethnic groups; eg, loan debt or assets may better explain socioeconomic experience across groups.[26, 27] Another potential explanation is that, regardless of socioeconomic status, exposure to racism is deleterious to maternal outcomes even among educated, privately insured individuals. Our prospective cohort contains large numbers of individuals from diverse racial and ethnic groups, enabling us to identify for the first time the gap in understanding racial and ethnic differences in the progression to type 2 diabetes.

Overall, our findings underscore the opportunity for GDM as an intervention point for life-course type 2 diabetes inequities and stress the importance of racial and ethnic disparities in GDM outcomes beyond the current pregnancy. For example, previous research on clinical outcomes of individuals with GDM has found an increased risk of adverse pregnancy outcomes among Black but not Asian or Hispanic individuals,[28–31] citing the "healthy immigrant" paradox. In contrast, our research highlights the heightened postpartum risk of type 2 diabetes after GDM among South and Southeast Asian and Hispanic individuals, the majority of whom are immigrants in our study population. Our findings regarding clinical and social or structural mediators suggest that targeting these risk factors might reduce but not eliminate life-course inequities in type 2 diabetes. Furthermore, our inability to explain disparities using a robust group of mediators suggests that further work is needed to define multilevel strategies (eg, clinical interventions that target individuals, along with policy-level changes that target structural disparities) to counter structural racism and to eliminate disparities. An example is current state policies expanding Medicaid until 12 months postpartum, which has the promise of increasing access to follow-up primary care among postpartum people with GDM.[32] Such policies could be paired with implementation strategies to ensure that evidence-based postpartum type 2 diabetes prevention programs[33–35] are equitably available.

Other potential mechanisms rooted in structural racism that may drive disparities in progression to type 2 diabetes after GDM among Black, Hispanic, and South and Southeast Asian communities include the social and built environment of one's neighborhood and chronic stress. Some environmental features previously associated with type 2 diabetes include neighborhood deprivation, access to care, availability of green spaces, the food environment, and social cohesion.[28–30,36] Chronic high levels of stress attributable to structural barriers, interpersonal discrimination, and psychological distress are associated with cardiometabolic and glucose dysregulation.[37] A pregnancy complicated by GDM already constitutes a "stress test" of cardiometabolic regulation. Added stress from social and economic challenges could exacerbate cardiometabolic and glycemic dysregulation or impede postpartum recovery, facilitating type 2 diabetes pathogenesis.[38] The neighborhood environment or psychosocial stress can also lead to negative health behaviors, such as unhealthy eating and physical activity patterns, or restrict health care utilization. The social construct of race and ethnicity reflects the totality of these mechanisms over the life course.

Our study has several limitations. We do not have information on individual risk factors such as physical activity and diet; these factors might have additionally mediated disparities but are not needed for the validity of our study findings. Because some of the social or structural mediators we tested may be antecedent causes of the clinical mediators, our mediation results most likely underestimate the total effect of social or structural factors. In addition, our big data cohort did not contain instruments to assess interpersonal or structural racism. We also did not have information on gender identity, so it is unknown whether our findings generalize to all pregnancy-capable genders. Despite these limitations, our study boasts several strengths. It is the largest cohort study to date on racial and ethnic disparities after GDM, providing the power necessary for rigorous mediation analyses. We used both the birth and hospital records to define our GDM cohort and to ascertain covariates, an approach with high validity. We leveraged a unique surveillance database, the New York City A1c Registry, to create the APPLE cohort. Finally, we applied probabilistic bias analysis, an underused but rigorous approach, which allowed us to estimate a range of plausible effect estimates given varying degrees of screening and loss-to-follow-up bias.

We found substantial population-level racial and ethnic disparities in the progression to type 2 diabetes. Pregnancy characteristics explained only a modest proportion of disparities, suggesting that further

investigation of levers to intervene and reduce disparities is warranted. Nonetheless, these findings can be used by clinicians to target GDM as an opportunity to intervene to advance life-course health equity.

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