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Racial and Ethnic Differences in Long-Term Ocular Outcomes Among Individuals With Gestational Diabetes

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Abstract

Introduction: Gestational diabetes mellitus (GDM) disproportionately affects Black and Hispanic or Latino birthing populations, but racial and ethnic differences in long-term ocular complications after GDM are not well characterized. We compared the risk of diabetic retinopathy (DR) and diabetic cataracts (DC) among individuals with GDM across racial and ethnic groups over 10 years.

Methods: We conducted a retrospective cohort study using de-identified electronic health record data from the TriNetX US Collaborative Network (N = 182,086 with GDM across 67 healthcare organizations). Individuals with pre-existing type 1 or type 2 diabetes or prior GDM were excluded. We calculated cumulative incidence and incidence rates of DR and DC by race and ethnicity between January 2000 and July 2026. We then performed 1:1 propensity score matching for Black or African American vs. White patients and for Hispanic or Latino vs. White patients, balancing demographics, body mass index (BMI), blood pressure, laboratory values, and medications (standardized mean difference < 0.10). Outcomes were compared using risk ratios (RR), Kaplan-Meier analysis, and hazard ratios (HR).

Results: Overall, 1,078 individuals developed DR (0.593%), and 505 developed DC (0.278%). Crude cumulative incidence was highest among Black patients for DC (0.70%) and among Native Hawaiian or Other Pacific Islander (1.32%), Black (1.23%), and American Indian or Alaska Native (1.18%) patients for DR. In matched cohorts of 24,121 patients per group, Black patients had a higher risk than White patients of both DR (RR, 1.62; 95% confidence interval (CI), 1.21-2.18) and DC (RR, 2.26; 95% CI, 1.44-3.56). Time-to-event analyses were consistent (DR HR, 0.68 for White vs. Black patients; 95% CI, 0.50-0.91; DC HR, 0.47; 95% CI, 0.30-0.75). In matched cohorts of 17,600 patients per group, Hispanic or Latino and White patients did not differ significantly in DR (RR, 1.21; 95% CI, 0.77-1.90) or DC (RR, 0.94; 95% CI, 0.46-1.90).

Conclusions: Among individuals with GDM, Black patients had a higher risk of DR and DC than matched White patients, although absolute risks were low. We found no significant difference between Hispanic or Latino and White patients, but power was limited by rare outcomes and shorter follow-up. These findings are hypothesis-generating and support further study of ocular outcomes after GDM, including studies with a non-GDM comparison group and longer follow-up.

Categories: Obstetrics/Gynecology, Ophthalmology
Keywords: diabetic cataract, diabetic retinopathy, gestational diabetes mellitus, racial and ethnic disparities, trinetx

Introduction

Gestational diabetes mellitus (GDM) is hyperglycemia first recognized during pregnancy. Its incidence has risen in the United States and varies by race and ethnicity [1]. Among Hispanic or Latino individuals, age-standardized rates rose from 50.0 per 1,000 live births in 2011 to 66.6 per 1,000 live births in 2019 [2]. Black or African American birthing populations also carry a disproportionate burden of GDM, linked to long-standing inequities in healthcare access, socioeconomic conditions, and metabolic risk [3].

Although GDM usually resolves after delivery, it marks lasting metabolic risk. Individuals with GDM are at increased risk of later type 2 diabetes, and this risk differs by race and ethnicity. After adjustment for age and nativity, Black individuals had a four times higher risk of developing type 2 diabetes within eight years of GDM than White individuals [3].

Chronic hyperglycemia damages both the retina and the lens. In the retina, it causes endothelial dysfunction, pericyte loss, and breakdown of the blood-retina barrier, leading to diabetic retinopathy (DR) [4]. In the lens, glucose metabolism through the polyol pathway and the formation of advanced glycation end-products cause osmotic and oxidative damage that can lead to diabetic cataract (DC) [5,6]. These pathways are well described in pre-existing diabetes. It is less clear how often DR and DC are recorded in the

years after a pregnancy complicated by GDM, when some individuals progress to type 2 diabetes.

Most evidence on diabetic eye disease around pregnancy comes from individuals with pre-existing diabetes, in whom DR can progress during pregnancy and the first year postpartum [7]. Guidelines therefore recommend retinal examination for pregnant individuals with pre-existing diabetes. Routine eye examination is not recommended for GDM, which has traditionally been considered transient and at lower risk of microvascular complications [7]. Racial disparities in DR screening and treatment are documented in general diabetes populations [8]. However, few studies have examined ocular outcomes after GDM, and whether these outcomes differ by race and ethnicity is not well characterized.

We therefore compared the risk of DR and DC across racial and ethnic groups within a large cohort of individuals with GDM, using electronic health record data from the TriNetX US Collaborative Network. The primary analyses were propensity score-matched (PSM) comparisons of Black or African American vs. White patients and of Hispanic or Latino vs. White patients, with outcomes counted from the index GDM diagnosis for up to 10 years. Crude cumulative incidence across all recorded racial and ethnic groups was a secondary, descriptive analysis. Because all patients had GDM, this design compares groups within a GDM population; it does not estimate the effect of GDM itself on ocular outcomes.

Materials And Methods

Cohort identification and baseline characteristics

Pregnant individuals were identified in the TriNetX US Collaborative Network using a two-step algorithm requiring evidence of pregnancy followed by evidence of GDM. Patients first had to meet at least one "pregnancy" criterion (Table *1*) using ICD-10-PCS and ICD-10-CM codes [9,10]. Patients with evidence of pre-existing (pre-gestational) type 1 or type 2 diabetes mellitus, or a personal history of prior gestational diabetes, were excluded from the pregnancy criterion (Table *1*), so that only incident GDM in the index pregnancy would be captured.

Must have (any of)			
Code system	Code	Description	Patients
ICD-10-PCS	10E0	Pregnancy/delivery/products of conception	1,409,999
ICD-10-PCS	10E	Delivery	1,409,999
ICD-10-PCS	10	Pregnancy	2,358,895
ICD-10-CM	Z33.1	Pregnant state, incidental	939,960
Cannot have (any of)			
Code system	Code	Description	Patients
ICD-10-CM	O24.1	Pre-existing type 2 diabetes mellitus, in pregnancy, childbirth, and the puerperium	84,487
ICD-10-CM	O24.0	Pre-existing type 1 diabetes mellitus, in pregnancy, childbirth, and the puerperium	29,569
ICD-10-CM	Z86.32	Personal history of gestational diabetes	174,874

TABLE 1: Pregnancy inclusion and exclusion criteria [9,10]

Among patients meeting the pregnancy criterion, GDM was defined using a broad set of ICD-10-CM codes covering GDM diagnosed in pregnancy, childbirth, or the puerperium, across all documented control/management subtypes (diet-controlled, insulin-controlled, and oral hypoglycemic agent-controlled), as well as related codes for abnormal glucose complicating pregnancy and unspecified diabetes mellitus in pregnancy (Table *2*). The first instance of GDM was required to occur within a window from three months before to one year after the first instance of pregnancy, in order to temporally anchor the GDM diagnosis to the index pregnancy.

Must have (any of)			
Code system	Code	Description	Patients
ICD-10-CM	O24.4	Gestational diabetes mellitus	471,117
ICD-10-CM	O99.810	Abnormal glucose complicating pregnancy	373,948
ICD-10-CM	O24.911	Unspecified diabetes mellitus in pregnancy, first trimester	56,286
ICD-10-CM	O24.919	Unspecified diabetes mellitus in pregnancy, unspecified trimester	48,940
ICD-10-CM	O24.439	Gestational diabetes mellitus in the puerperium, unspecified control	15,221
ICD-10-CM	O24.41	Gestational diabetes mellitus in pregnancy	443,696
ICD-10-CM	O24.419	Gestational diabetes mellitus in pregnancy, unspecified control	351,878
ICD-10-CM	O24.42	Gestational diabetes mellitus in childbirth	222,442
ICD-10-CM	O24.429	Gestational diabetes mellitus in childbirth, unspecified control	92,060
ICD-10-CM	O24.43	Gestational diabetes mellitus in the puerperium	27,700
ICD-10-CM	O24.410	Gestational diabetes mellitus in pregnancy, diet controlled	205,117
ICD-10-CM	O24.414	Gestational diabetes mellitus in pregnancy, insulin controlled	93,895
ICD-10-CM	O24.420	Gestational diabetes mellitus in childbirth, diet controlled	86,179
ICD-10-CM	O24.424	Gestational diabetes mellitus in childbirth, insulin controlled	49,017
ICD-10-CM	O24.430	Gestational diabetes mellitus in the puerperium, diet controlled	7,288
ICD-10-CM	O24.434	Gestational diabetes mellitus in the puerperium, insulin controlled	4,991
ICD-10-CM	O24.415	Gestational diabetes mellitus in pregnancy, controlled by oral hypoglycemic drugs	45,890
ICD-10-CM	O24.425	Gestational diabetes mellitus in childbirth, controlled by oral hypoglycemic drugs	24,813
ICD-10-CM	O24.435	Gestational diabetes mellitus in puerperium, controlled by oral hypoglycemic drugs	2,114

TABLE 2: Gestational diabetes inclusion criteria [10]

No exclusion ("cannot have") criteria were applied to the gestational diabetes definition.

Racial and ethnic stratification

The overall GDM cohort (N = 182,086 across 67 healthcare organizations (HCOs)) was stratified by documented race and ethnicity to construct three analytic groups: White, Black or African American, and Hispanic or Latino patients with GDM. Two pairwise cohorts were then built for comparison - Black or African American vs. White (18,111-24,900 patients per group; 56 HCOs) and Hispanic or Latino vs. White (17,600 patients per group) - reflecting the two racial/ethnic contrasts of interest for this analysis.

Baseline characteristics and PSM

For each pairwise comparison, baseline characteristics were summarized across four domains: demographics (age at index, mean ± SD), diagnoses (ICD-10 code E66, overweight and obesity), laboratory results (Logical Observation Identifiers Names and Codes (LOINC)-coded categorical distributions of body mass index (BMI), triglycerides, systolic blood pressure, serum/plasma glucose, hemoglobin A1c, total cholesterol, and serum iron), and medications (antihyperglycemic agents, insulin, and oral hypoglycemic agents). To account for baseline imbalances between racial/ethnic groups, 1:1 PSM was performed for each comparison. Covariate balance before and after matching was assessed using the standardized mean difference (SMD), with SMD < 0.10 considered indicative of adequate balance, consistent with conventional thresholds for propensity-matched cohort studies.

Black or African American vs. White patients

Before matching, the White GDM cohort comprised approximately 75,300 patients, and the Black or African American GDM cohort comprised approximately 24,900 patients. Black patients had a higher prevalence of overweight/obesity (31.4% vs. 22.7%; SMD = 0.20) and were more likely to have BMI ⩾ 35 kg/m² (42.9% vs.

32.2%; SMD = 0.22). Glycemic markers were also less favorable among Black patients before matching, including a higher proportion with hemoglobin A1c 5.7%-6.4% (11.5% vs. 3.5%; SMD = 0.31) and glucose 126-200 mg/dL (27.2% vs. 17.6%; SMD = 0.23). After 1:1 PSM, matched cohorts of approximately 24,100-24,300 patients per group were retained, and SMDs for nearly all characteristics fell below 0.10 (e.g., overweight/obesity SMD = 0.011; BMI ⩾ 35 kg/m² SMD = 0.023; hemoglobin A1c 5.7%-6.4% SMD = 0.015), indicating adequate covariate balance (Table 3).

Baseline characteristics						
Characteristic	White (before)	Comparator (before)	SMD (before)	White (after)	Comparator (after)	SMD (after)
Age at index, mean (SD)	30.74 (±5.72)	30.06 (±6.28)	0.11	29.84 (±5.84)	30.05 (±6.28)	0.034
Overweight and obesity (E66)	17,067 (22.66%)	7,814 (31.39%)	0.2	7,442 (30.85%)	7,321 (30.35%)	0.011
BMI 18.5-24.9 kg/m²	22,683 (30.12%)	4,954 (19.90%)	0.24	4,722 (19.58%)	4,909 (20.35%)	0.019
BMI 25-29.9 kg/m²	31,491 (41.81%)	8,461 (33.99%)	0.16	8,150 (33.79%)	8,309 (34.45%)	0.014
BMI 30-34.9 kg/m²	27,399 (36.38%)	9,578 (38.48%)	0.043	9,317 (38.63%)	9,258 (38.38%)	0.005
BMI ≥ 35 kg/m²	24,256 (32.20%)	10,681 (42.91%)	0.22	10,386 (43.06%)	10,111 (41.92%)	0.023
Triglycerides ≤ 160 mg/dL	10,409 (13.82%)	3,168 (12.73%)	0.032	2,741 (11.36%)	2,951 (12.23%)	0.027
Triglycerides ≥ 160 mg/dL	2,890 (3.84%)	421 (1.69%)	0.13	432 (1.79%)	413 (1.71%)	0.006
Systolic BP ≤ 120 mmHg	58,666 (77.89%)	18,717 (75.20%)	0.064	18,149 (75.24%)	18,138 (75.20%)	0.001
Systolic BP 121-130 mmHg	49,972 (66.35%)	16,543 (66.46%)	0.002	16,153 (66.97%)	15,948 (66.12%)	0.018
Systolic BP 131-140 mmHg	35,746 (47.46%)	12,676 (50.93%)	0.069	12,362 (51.25%)	12,152 (50.38%)	0.017
Systolic BP 141-180 mmHg	21,683 (28.79%)	8,315 (33.41%)	0.1	8,061 (33.42%)	7,879 (32.66%)	0.016
Systolic BP ≥ 181 mmHg	747 (0.99%)	567 (2.28%)	0.1	447 (1.85%)	483 (2.00%)	0.011
Glucose 60-100 mg/dL	39,343 (52.23%)	14,453 (58.06%)	0.12	13,812 (57.26%)	13,796 (57.20%)	0.001
Glucose 101-125 mg/dL	21,453 (28.48%)	8,644 (34.73%)	0.13	8,165 (33.85%)	8,128 (33.70%)	0.003
Glucose 126-200 mg/dL	13,224 (17.56%)	6,758 (27.15%)	0.23	6,152 (25.50%)	6,274 (26.01%)	0.012
Glucose ≥ 201 mg/dL	1,712 (2.27%)	1,287 (5.17%)	0.15	1,059 (4.39%)	1,119 (4.64%)	0.012
HbA1c 0%-5.7%	16,587 (22.02%)	5,152 (20.70%)	0.032	4,949 (20.52%)	4,983 (20.66%)	0.003
HbA1c 5.7%-6.4%	2,622 (3.48%)	2,849 (11.45%)	0.31	2,102 (8.71%)	2,207 (9.15%)	0.015
HbA1c 6.5%-7.9%	477 (0.63%)	607 (2.44%)	0.15	412 (1.71%)	465 (1.93%)	0.016
HbA1c ≥ 8%	330 (0.44%)	315 (1.27%)	0.09	235 (0.97%)	266 (1.10%)	0.013
Cholesterol ≤ 155 mg/dL	3,415 (4.53%)	1,350 (5.42%)	0.041	1,153 (4.78%)	1,240 (5.14%)	0.017
Cholesterol 155-250 mg/dL	9,579 (12.72%)	2,430 (9.76%)	0.094	2,174 (9.01%)	2,301 (9.54%)	0.018
Cholesterol ≥ 250 mg/dL	838 (1.11%)	131 (0.53%)	0.065	129 (0.54%)	124 (0.51%)	0.003

Iron ≤ 11 µg/dL	30 (0.04%)	25 (0.10%)	0.023	21 (0.09%)	19 (0.08%)	0.003
Iron ≥ 11 µg/dL	5,554 (7.37%)	2,535 (10.18%)	0.099	2,198 (9.11%)	2,309 (9.57%)	0.016
Antihyperglycemics	11,223 (14.90%)	4,620 (18.56%)	0.098	4,285 (17.77%)	4,365 (18.10%)	0.009
Insulin	7,372 (9.79%)	2,950 (11.85%)	0.066	2,605 (10.80%)	2,748 (11.39%)	0.019
Oral hypoglycemic agents	4,550 (6.04%)	1,816 (7.30%)	0.05	1,656 (6.87%)	1,679 (6.96%)	0.004

TABLE 3: Baseline characteristics - Black or African American vs. White patients, before and after propensity score matching

"Comparator" = Black or African American + GDM. SMD < 0.10 indicates adequate covariate balance.

SMD: standardized mean difference; BMI: body mass index; BP: blood pressure; GDM: gestational diabetes mellitus

Hispanic or Latino vs. White patients

Before matching, the Hispanic/Latino GDM cohort comprised approximately 17,700 patients, compared with the same White reference cohort of approximately 75,300 patients. Hispanic/Latino patients showed a notably lower prevalence of elevated systolic blood pressure across multiple ranges (e.g., 131-140 mmHg: 33.0% vs. 47.5%; SMD = 0.30) and a lower prevalence of BMI in the normal range (18.5-24.9 kg/m²: 16.6% vs. 30.1%; SMD = 0.32), reflecting meaningful baseline differences in cardiometabolic risk-factor distribution between groups. Following 1:1 PSM, matched cohorts of approximately 17,600 patients per group were retained, and SMDs for essentially all characteristics were reduced below 0.10 (e.g., systolic blood pressure 131-140 mmHg SMD = 0.002; BMI 18.5-24.9 kg/m² SMD = 0.007), confirming successful balance of baseline covariates (Table *4*). Across both comparisons, PSM substantially reduced covariate imbalance, with post-matching SMDs falling below the conventional 0.10 threshold for the large majority of demographic, diagnostic, laboratory, and medication-related characteristics, supporting the validity of subsequent between-group outcome comparisons.

Characteristic	White (before)	Comparator (before)	SMD (before)	White (after)	Comparator (after)	SMD (after)
Age at index, mean (SD)	30.74 (±5.72)	30.68 (±6.32)	0.009	30.73 (±5.86)	30.68 (±6.32)	0.009
Overweight and obesity (E66)	17,067 (22.66%)	3,553 (20.08%)	0.063	3,458 (19.65%)	3,541 (20.12%)	0.012
BMI 18.5-24.9 kg/m²	22,683 (30.12%)	2,937 (16.60%)	0.32	2,893 (16.44%)	2,937 (16.69%)	0.007
BMI 25-29.9 kg/m²	31,491 (41.81%)	6,182 (34.94%)	0.14	5,918 (33.62%)	6,160 (35.00%)	0.029
BMI 30-34.9 kg/m²	27,399 (36.38%)	6,498 (36.73%)	0.007	6,375 (36.22%)	6,445 (36.62%)	0.008
BMI ≥ 35 kg/m²	24,256 (32.20%)	5,055 (28.57%)	0.079	5,006 (28.44%)	5,029 (28.57%)	0.003
Triglycerides ≤ 160 mg/dL	10,409 (13.82%)	1,142 (6.46%)	0.25	1,099 (6.24%)	1,136 (6.46%)	0.009
Triglycerides ≥ 160 mg/dL	2,890 (3.84%)	399 (2.25%)	0.092	393 (2.23%)	399 (2.27%)	0.002
Systolic BP ≤ 120 mmHg	58,666 (77.89%)	13,076 (73.91%)	0.093	12,832 (72.91%)	12,996 (73.84%)	0.021
Systolic BP 121-130 mmHg	49,972 (66.35%)	9,366 (52.94%)	0.28	9,154 (52.01%)	9,341 (53.07%)	0.021
Systolic BP 131-140 mmHg	35,746 (47.46%)	5,840 (33.01%)	0.3	5,812 (33.02%)	5,832 (33.14%)	0.002
Systolic BP 141-180 mmHg	21,683	3,025 (17.10%)	0.28	3,017 (17.14%)	3,021 (17.16%)	0.001

	(28.79%)					
Systolic BP ≥ 181 mmHg	747 (0.99%)	81 (0.46%)	0.063	76 (0.43%)	81 (0.46%)	0.004
Glucose 60-100 mg/dL	39,343 (52.23%)	8,071 (45.62%)	0.13	7,952 (45.18%)	8,017 (45.55%)	0.007
Glucose 101-125 mg/dL	21,453 (28.48%)	4,609 (26.05%)	0.055	4,511 (25.63%)	4,566 (25.94%)	0.007
Glucose 126-200 mg/dL	13,224 (17.56%)	2,914 (16.47%)	0.029	2,830 (16.08%)	2,883 (16.38%)	0.008
Glucose ≥ 201 mg/dL	1,712 (2.27%)	464 (2.62%)	0.023	452 (2.57%)	452 (2.57%)	0
HbA1c 0%-5.7%	16,587 (22.02%)	3,875 (21.90%)	0.003	3,764 (21.39%)	3,847 (21.86%)	0.011
HbA1c 5.7%-6.4%	2,622 (3.48%)	1,245 (7.04%)	0.16	1,206 (6.85%)	1,164 (6.61%)	0.01
HbA1c 6.5%-7.9%	477 (0.63%)	196 (1.11%)	0.051	188 (1.07%)	191 (1.08%)	0.002
HbA1c ≥ 8%	330 (0.44%)	87 (0.49%)	0.008	95 (0.54%)	86 (0.49%)	0.007
Cholesterol ≤ 155 mg/dL	3,415 (4.53%)	507 (2.87%)	0.088	475 (2.70%)	501 (2.85%)	0.009
Cholesterol 155-250 mg/dL	9,579 (12.72%)	1,015 (5.74%)	0.24	1,023 (5.81%)	1,014 (5.76%)	0.002
Cholesterol ≥ 250 mg/dL	838 (1.11%)	69 (0.39%)	0.084	69 (0.39%)	69 (0.39%)	0
Iron ≤ 11 µg/dL	30 (0.04%)	20 (0.11%)	0.026	16 (0.09%)	15 (0.09%)	0.002
Iron ≥ 11 µg/dL	5,554 (7.37%)	983 (5.56%)	0.074	889 (5.05%)	976 (5.54%)	0.022
Antihyperglycemics	11,223 (14.90%)	2,635 (14.89%)	0	2,455 (13.95%)	2,615 (14.86%)	0.026
Insulin	7,372 (9.79%)	1,778 (10.05%)	0.009	1,700 (9.66%)	1,758 (9.99%)	0.011
Oral hypoglycemic agents	4,550 (6.04%)	1,653 (9.34%)	0.12	1,469 (8.35%)	1,603 (9.11%)	0.027

TABLE 4: Baseline characteristics - Hispanic or Latino vs. White patients, before and after propensity score matching

"Comparator" = Hispanic or Latino + GDM. SMD < 0.10 indicates adequate covariate balance.

SMD: standardized mean difference; BMI: body mass index; BP: blood pressure

Outcome definitions

The two outcomes were DR and DC, identified by ICD-10-CM diagnosis codes in the TriNetX network (Table 5). DR was defined as any of the codes E11.31 to E11.35, covering type 2 diabetes mellitus with unspecified, mild, moderate, or severe nonproliferative, or proliferative DR, including subcodes. DC was defined as code E11.36, type 2 diabetes mellitus with DC. Each outcome was counted from the first recorded instance of pregnancy and GDM through the end of available follow-up. Patients with the outcome recorded before this window were excluded from the time-to-event analyses for that outcome.

Outcome	Code system	Code	Description	Patients
Diabetic retinopathy	ICD-10-CM	E11.31	Type 2 diabetes mellitus with unspecified diabetic retinopathy	484,949
Diabetic retinopathy	ICD-10-CM	E11.32	Type 2 diabetes mellitus with mild nonproliferative diabetic retinopathy	337,643
Diabetic retinopathy	ICD-10-CM	E11.33	Type 2 diabetes mellitus with moderate nonproliferative diabetic retinopathy	116,339
Diabetic retinopathy	ICD-10-CM	E11.34	Type 2 diabetes mellitus with severe nonproliferative diabetic retinopathy	52,364
Diabetic retinopathy	ICD-10-CM	E11.35	Type 2 diabetes mellitus with proliferative diabetic retinopathy	205,997
Diabetic cataract	ICD-10-CM	E11.36	Type 2 diabetes mellitus with diabetic cataract	475,670

TABLE 5: Outcome definitions [10]

Patient counts are totals across the TriNetX network at the time of the query.

All outcome codes fall within the E11 category, so each outcome requires a recorded diagnosis of type 2 diabetes. Retinopathy or cataract codes without a diabetes code were not included, such as H35.0x (background retinopathy) and H25-H26 (other cataract). Codes for other types of diabetes (E08, E09, E10, E13) were not included either. The outcomes therefore capture ocular complications recorded in patients who received a type 2 diabetes diagnosis after GDM. They depend on progression to recognized type 2 diabetes as well as on the eye condition itself.

In addition, E11.36 is often assigned to any cataract in a patient with a diabetes diagnosis. DC therefore represents coded DC rather than confirmed diabetes-related lens disease. Outcome codes were not validated against chart review or ophthalmic examination, and outcome severity was not assessed.

Results

Incidence of DR and DC

Between January 2000 and July 2026, a total of 1,078 individuals in the GDM cohort developed DR, corresponding to a cumulative incidence of 0.59%. The overall incidence rate was 2×10^{-6} cases per person-day. Stratified by race, the cumulative incidence was highest among Native Hawaiian or Other Pacific Islander patients (1.32%, n = 29), followed by Black or African American (1.23%, n = 325) and American Indian or Alaska Native (1.18%, n = 24) patients. Intermediate cumulative incidence was observed among individuals with unknown race (0.69%, n = 143), whereas lower cumulative incidence was observed among patients of Other Race (0.48%, n = 57), White patients (0.45%, n = 453), and Asian patients (0.26%, n = 47). Consistent with these findings, incidence rates were highest among Native Hawaiian or Other Pacific Islander, Black or African American, and American Indian or Alaska Native patients (3×10^{-6} cases per person-day each), followed by individuals of Other Race and those with unknown race (2×10^{-6} cases per person-day), White patients (1×10^{-6} cases per person-day), and Asian patients ($<1 \times 10^{-6}$ cases per person-day). When stratified by ethnicity, cumulative incidence was highest among patients with unknown ethnicity (0.79%, n = 250), followed by non-Hispanic or Latino patients (0.57%, n = 652), while Hispanic or Latino patients had the lowest cumulative incidence (0.48%, n = 176). Similarly, incidence rates were highest among patients with unknown ethnicity (3×10^{-6} cases per person-day), followed by Hispanic or Latino patients (2×10^{-6} cases per person-day) and non-Hispanic or Latino patients (1×10^{-6} cases per person-day).

During the same study period, 505 individuals developed DC, corresponding to a cumulative incidence of 0.28%. The overall incidence rate was below the reporting threshold of TriNetX ($<1 \times 10^{-6}$ cases per person-day). Stratified by race, cumulative incidence was highest among Black or African American patients (0.69%, n = 183), followed by American Indian or Alaska Native patients (0.49%, n = 10), Native Hawaiian or Other Pacific Islander patients (0.46%, n = 10), individuals with unknown race (0.30%, n = 63), patients of Other Race (0.24%, n = 29), White patients (0.20%, n = 197), and Asian patients (0.10%, n = 19). Correspondingly, incidence rates were highest among Black or African American patients (2×10^{-6} cases per person-day), followed by American Indian or Alaska Native patients and individuals with unknown race (1×10^{-6} cases per person-day each). Incidence rates among Asian, Native Hawaiian or Other Pacific Islander, White, and Other Race patients remained below the TriNetX reporting threshold ($<1 \times 10^{-6}$ cases per person-day).

Overall, these descriptive analyses demonstrate variation in the cumulative incidence of DR and DC across racial and ethnic groups within the GDM cohort. Black or African American patients exhibited the highest observed cumulative incidence of DC and among the highest cumulative incidence of DR, while White and Asian patients generally demonstrated lower cumulative incidence for both outcomes. These descriptive findings provide context for the subsequent comparative Kaplan-Meier and risk ratio (RR) analyses, which evaluated racial and ethnic differences in ocular outcomes after adjustment for potential confounding

factors.

Follow-up duration

Outcomes were assessed over 10 years after the index date, but observed follow-up was considerably shorter. The PSM White and Black or African American GDM cohorts each included 24,121 individuals. The White cohort had a mean follow-up of 3.90 ± 3.43 years (median, 3.04 years; IQR, 5.97 years), and the Black or African American cohort had a slightly longer mean follow-up of 4.29 ± 3.59 years (median, 3.55 years; IQR, 6.65 years).

The PSM White and Hispanic or Latino GDM cohorts each included 17,600 individuals. The White cohort had a mean follow-up of 3.96 ± 3.50 years (median, 3.08 years; IQR, 6.20 years), whereas the Hispanic or Latino cohort had a shorter mean follow-up of 3.31 ± 3.47 years (median, 2.00 years; IQR, 5.61 years).

Mean follow-up across all matched cohorts was therefore 3-4 years, short relative to the time over which DR and DC typically develop. Follow-up also differed between groups, being longer in Black patients and shorter in Hispanic or Latino patients than in their White comparators. Because cumulative RR do not account for these differences, the time-to-event analyses were treated as primary.

PSM comparison of White and Black or African American individuals with GDM

Following PSM, 24,121 Black or African American individuals with GDM were matched to 24,121 White individuals with GDM. Compared with the White cohort, Black or African American individuals demonstrated significantly greater risks of developing both DR and DC during follow-up.

For DR, 71 of 24,091 White patients (0.30%) and 115 of 24,087 Black or African American patients (0.48%) developed DR during follow-up. Black or African American individuals had a significantly higher cumulative risk of DR than White individuals (RR, 1.62; 95% confidence interval (CI), 1.21-2.18; p = 0.001), corresponding to an absolute risk difference of 0.18% (95% CI, 0.07%-0.29%). Kaplan-Meier analysis likewise demonstrated significantly lower DR-free survival among Black or African American individuals, p = 0.010 (Figure *1*). The estimated hazard ratio for White versus Black or African American individuals was 0.68 (95% CI, 0.50-0.91), corresponding to an approximately 47% higher hazard of DR among Black or African American individuals. Thirty White patients and 34 Black or African American patients were excluded from the survival analysis because the outcome occurred before the start of the observation period.

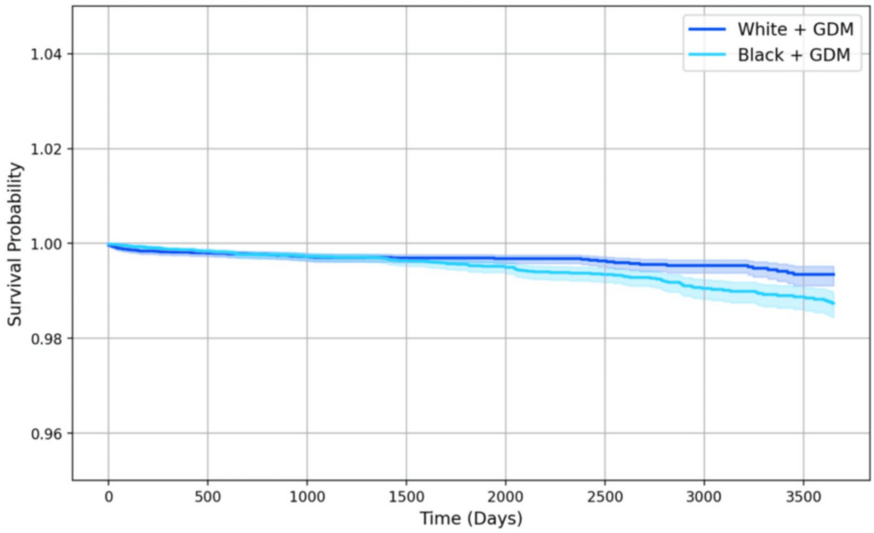


FIGURE 1: Kaplan-Meier curves comparing time to diabetic retinopathy (DR) among White and Black pregnant individuals with gestational diabetes mellitus (GDM)

For DC, 27 of 24,108 White patients (0.11%) and 61 of 24,098 Black or African American patients (0.25%) developed DC during follow-up. Black or African American individuals had more than twice the cumulative risk of developing DC compared with White individuals (RR, 2.26; 95% CI, 1.44-3.56; p < 0.001), corresponding to an absolute risk difference of 0.14% (95% CI, 0.07%-0.22%). Kaplan-Meier analysis similarly demonstrated significantly lower DC-free survival among Black or African American individuals, p

= 0.001 (Figure 2). The estimated hazard ratio for White versus Black or African American individuals was 0.47 (95% CI, 0.30-0.75), corresponding to an approximately 2.11-fold higher hazard of DC among Black or African American individuals. Thirteen White patients and 23 Black or African American patients were excluded from the survival analysis because the outcome occurred before the start of the observation period. The proportional hazards assumption was satisfied (proportionality test, p = 0.62).

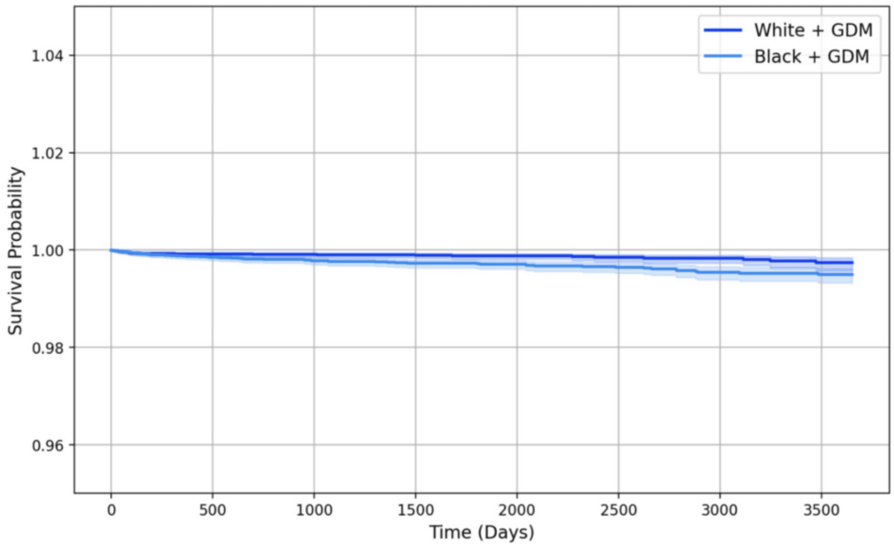


FIGURE 2: Kaplan-Meier curves comparing time to diabetic cataracts (DC) among White and Black pregnant individuals with gestational diabetes mellitus (GDM)

Overall, both cumulative risk and time-to-event analyses consistently demonstrated significantly greater long-term risks of DR and DC among Black or African American individuals with GDM compared with PSM White individuals.

PSM comparison of White and Hispanic or Latino individuals with GDM

Following PSM, 17,600 White individuals with GDM were matched to 17,600 Hispanic or Latino individuals with GDM. No statistically significant differences in long-term ocular outcomes were observed between the matched cohorts.

For DR, 41 of 17,586 White patients (0.23%) and 34 of 17,591 Hispanic or Latino patients (0.19%) developed DR during follow-up. The cumulative risk of DR did not differ significantly between the cohorts (RR, 1.21; 95% CI, 0.77-1.90; risk difference, 0.04%; 95% CI, −0.06% to 0.14%; p = 0.418). Kaplan-Meier analysis likewise demonstrated no significant difference in DR-free survival between the groups (p = 0.907). The estimated hazard ratio was 1.03 (95% CI, 0.65-1.62). However, the proportional hazards assumption was not satisfied (proportionality test, p = 0.0008), indicating that the hazard ratio should be interpreted with caution.

Similarly, DC developed in 15 of 17,597 White patients (0.09%) and 16 of 17,594 Hispanic or Latino patients (0.09%). There was no significant difference in cumulative risk between the cohorts (RR, 0.94; 95% CI, 0.46-1.90; risk difference, −0.006%; 95% CI, −0.068% to 0.056%; p = 0.857). Kaplan-Meier analysis also demonstrated no significant difference in DC-free survival (p = 0.51). The estimated hazard ratio was 0.79 (95% CI, 0.39-1.60), and the proportional hazards assumption was satisfied (proportionality test, p = 0.52). Overall, these findings indicate no statistically significant differences in the cumulative risk or time to event for DR or DC between PSM White and Hispanic or Latino individuals with GDM.

Discussion

In this retrospective cohort of individuals with GDM, Black or African American patients had a higher recorded risk of DR and coded DC than PSM White patients. In the primary time-to-event analyses, the hazard of DR was 1.47 (95% CI, 1.10-2.00) times higher and the hazard of DC 2.13 (95% CI, 1.33-3.33) times higher in Black patients. Cumulative RR were consistent (DR: RR, 1.62; 95% CI, 1.21-2.18; DC: RR, 2.26; 95% CI, 1.44-3.56).

Absolute risks were low in both groups (0.1%-0.5%), and the absolute risk differences were small: 0.18%

(95% CI, 0.07%-0.29%) for DR and 0.14% (95% CI, 0.07%-0.22%) for DC. These are relative differences in rare outcomes over 3-4 years of follow-up, not evidence of a high individual burden. Matched Hispanic or Latino and White patients did not differ significantly in either outcome. In unmatched descriptive analyses, crude DR incidence was also higher among Native Hawaiian or Other Pacific Islander and American Indian or Alaska Native patients, but these groups had few events (29 and 24 DR cases) and were not formally compared.

These findings describe an association between race or ethnicity and recorded ocular outcomes within a GDM population. Because the study had no comparison group without GDM, they do not show that GDM causes DR or DC.

Interpreting the difference between Black and White patients

Our design cannot identify why the difference exists, but several explanations are consistent with the data. The first is undiagnosed pre-existing diabetes. After matching, about 3% of patients in each group had an HbA1c of 6.5% or higher before the index date, and 30 White and 34 Black patients had DR recorded before follow-up began. We excluded these patients because these conditions developed before our study period following the index event: pregnancy and GDM diagnosis.

The second is progression to type 2 diabetes. Type 2 diabetes after GDM is more common in Black individuals [3] and is a plausible pathway to later eye disease. Additionally, differences in healthcare use could change how often eye conditions are detected and coded, in either direction. Finally, follow-up was longer in Black patients (median, 3.55 vs. 3.04 years), which may inflate their cumulative risk. The time-to-event analyses account for this difference and gave consistent results.

Beyond these data, prior studies have documented racial disparities in the screening and treatment of DR in general diabetes populations [8]. Proposed contributors include limited access to ophthalmic care, delayed intervention, barriers to glycemic management, chronic stress, perceived discrimination in medical care, and environmental exposures [8,11-14]. Our data contained no measures of healthcare access, socioeconomic status, or psychosocial exposures. These remain hypotheses, and our results should not be read as evidence for any one of them.

Interpreting the outcomes between Latino or Hispanic and White patients

The absence of a significant difference should not be read as equivalence. Point estimates were close to the null for DR (RR, 0.83; 95% CI, 0.53-1.30) and DC (RR, 1.07; 95% CI, 0.53-2.17), but CI were wide because events were few (75 DR and 31 DC events across both matched cohorts). We therefore cannot exclude a modest difference in either direction. The proportional hazards assumption was violated for DR in this comparison, so the hazard ratio (0.97) should be interpreted with caution.

Two features of the data also bias this comparison toward the null. Median follow-up was shorter in Hispanic or Latino patients (2.00 vs. 3.08 years), which reduces the ability to detect outcomes that develop over many years. These findings do not support extending the Black-White difference to Hispanic or Latino patients, and larger studies with longer follow-up are needed.

Eye screening after GDM

Current guidelines recommend retinal examination for pregnant individuals with pre-existing diabetes, with follow-up intervals based on retinopathy severity [12,15,16]. Routine eye examination is not recommended for GDM, which has traditionally been considered transient and at lower risk of microvascular complications [12,16]. Our study did not evaluate screening, so it cannot show whether screening after GDM would change outcomes. Programs using nonmydriatic retinal photography have improved screening rates among pregnant patients with pre-existing diabetes in safety-net settings [17]. Such approaches could be studied after GDM if a need is established.

Strengths and limitations

This study drew on a large multicenter cohort of 182,086 individuals with GDM from 67 HCOs. PSM achieved good balance on measured covariates, and both cumulative risk and time-to-event analyses gave consistent results. The study also has several limitations.

First, it is a retrospective analysis of electronic health record data. Exposures and outcomes depend on diagnostic coding and on patients' contact with the healthcare system, both of which may differ across racial and ethnic groups. GDM was defined with a broad set of codes, including O99.810 and unspecified diabetes in pregnancy, which may misclassify some patients. Exclusion of pre-existing diabetes was also incomplete, as elevated baseline HbA1c in some patients shows.

Second, both outcomes depend on diagnosis codes and on a recorded diabetes diagnosis. Coded DC in

particular may not reflect diabetes-related lens disease. We did not assess outcome severity, so we cannot draw conclusions about vision-threatening disease.

Third, PSM balanced only measured covariates. Socioeconomic status, insurance, and access to care were not available, and parity, hypertensive disorders of pregnancy, and calendar year and site were not included in matching. Additionally, race and ethnicity are social constructs, and in this study, they serve as proxies for exposures we did not measure.

Fourth, follow-up was short (mean, 3-4 years) for conditions that develop over many years, and it differed across groups. Outcomes were rare, which limited precision, particularly in the Hispanic or Latino comparison. We focused on the short term to limit the influence of later progression to type 2 diabetes, giving a clearer picture of racial and ethnic differences in the development of DR and DC among individuals with GDM.

Finally, the study had no comparison group without GDM, so it cannot determine whether GDM itself increases ocular risk. We also did not perform matched comparisons for Asian, Native Hawaiian or Other Pacific Islander, or American Indian or Alaska Native patients, so findings for these groups are descriptive only.

Future directions

Our findings are hypothesis-generating and do not by themselves support changes to screening guidelines. Future studies should include a non-GDM comparison group, longer follow-up, measured social determinants of health, data on progression to type 2 diabetes, and outcome severity. Such studies could test whether risk-stratified eye evaluation after GDM improves outcomes, particularly among Black individuals. Because GDM is usually diagnosed in the late second trimester, any such evaluation would more plausibly occur after diagnosis or postpartum, for example, at postpartum glucose testing [12,17].

Conclusions

In this retrospective cohort of individuals with GDM from the TriNetX US Collaborative Network, Black or African American patients had a higher recorded risk of DR and coded DC than PSM White patients over 3-4 years of follow-up. The hazard was 1.47 times higher for DR and 2.13 times higher for DC, and cumulative RR were consistent. Absolute risks were low (0.1%-0.5%), and absolute differences were small (0.18% for DR and 0.14% for DC). These results reflect relative differences in rare outcomes, not a high individual burden. Matched Hispanic or Latino and White patients did not differ significantly in either outcome. This should not be read as equivalence: events were few, CI were wide, and follow-up was shorter in the Hispanic or Latino cohort.

All patients in this study had GDM, so these findings describe differences between racial and ethnic groups within a GDM population. They do not show that GDM causes DR or DC, and they cannot identify why the Black-White difference exists. Undiagnosed pre-existing diabetes, later progression to type 2 diabetes, and differences in healthcare use or coding could each contribute, and the DC outcome in particular may reflect coding practice rather than diabetes-related lens disease. These findings are hypothesis-generating. Future studies with a non-GDM comparison group, longer follow-up, data on type 2 diabetes progression, and measured social determinants of health are needed to explain these differences. Such studies could also test whether eye evaluation after GDM, particularly postpartum, improves outcomes for Black patients.

Additional Information

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All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Eva M. Nelson, Shloka Homa, Emily Ho, Marina Gad El Sayed, Madeline Chung, Daniel Novak, Jennifer Zamora, Eugene Albright

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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