

Racial Disparities in Lipid Control in Patients With Diabetes

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Patients diagnosed with diabetes mellitus (DM) are at higher risk for cardiovascular disease (CVD) events and mortality than patients with no history of DM.¹⁻⁴ In an effort to reduce this risk, national guidelines recommend strict hypercholesterolemia management, among other measures, in patients with DM. Racial disparities have been observed not only in the prevalence of DM and its complications but also in the management of hypercholesterolemia (lipid testing, treatment, and control/goal attainment).⁵⁻⁷ In 1 published study, investigators found that even among patients treated for hypercholesterolemia, African American patients were less likely to reach their low-density lipoprotein cholesterol (LDL-C) goal compared with white patients.⁸ Several reports have shown that even among patients with coronary heart disease (CHD), DM, or hypertension, African Americans are less likely to receive 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (ie, statin therapy) for dyslipidemia and/or achieve LDL-C control compared with white patients.⁹⁻¹⁴

Disparities in access to healthcare and healthcare-seeking behavior may explain why lipid management impact is better among whites than among African Americans.⁹ These disparities have been attributed to difficulties in accessing healthcare among uninsured minorities, and lower socioeconomic status has been associated with an inferior quality of care received.^{15,16} Even among insured African Americans, quality of care, particularly lipid treatment and control, is inferior to that received by other racial groups.¹⁷⁻²³ However, some findings suggest that patients of differing race and ethnic groups receive equal benefits when treated appropriately.^{9,24} Further complicating matters, previous studies have also shown racial differences in adherence to lipid-lowering medications among patients with diabetes which might contribute to ethnic and racial disparities.²⁵⁻²⁸ This paper builds on previous literature by including information on care processes, clinical outcomes, patient sociodemographic and clinical characteristics, office visit and prescription drug copayments, treatment intensification, and medication adherence in the same study. With its large sample size, high proportion of African Americans, and long observation period, this study strengthens and expands previous findings.

To more fully investigate the question of racial disparities in lipid control, we describe annual rates of testing, treatment, and LDL-C goal

Objectives: To describe lipid management over time in a cohort of insured patients with diabetes and evaluate differences between African American and white patients.

Study Design: Automated claims data were used to identify a cohort of 11,411 patients with diabetes in 1997 to 1998. Patients were followed through 2007.

Methods: Rates of hypercholesterolemia testing, treatment, and goal attainment were measured annually. Treatment was determined by a claim for lipid-lowering agents, and goal attainment was defined as a low-density lipoprotein cholesterol (LDL-C) level <100 mg/dL.

Results: During the study period, LDL-C testing increased from 48% to 70% among African American patients and from 61% to 77% among white patients. Treatment with lipid-lowering drugs increased from 23% to 56% among African American patients and 33% to 61% among white patients. The proportion at goal increased from 35% to 76% and from 24% to 59% among white and African American patients, respectively. African American patients were less likely to be tested for LDL-C (odds ratio [OR] 0.79; 95% confidence interval [CI] 0.73-0.86), treated with lipid-lowering agents (OR 0.72; 95% CI 0.65-0.80), have their medication dosage altered (OR 0.65; 95% CI 0.59-0.73), or attain LDL-C goal (OR 0.59; 95% CI 0.56-0.63) compared with white patients.

Conclusions: Although rates of LDL-C testing, treatment, and goal attainment improved over time, racial disparities in dyslipidemia management continued to exist. Further studies to determine the causes of differences in management by race are warranted.

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Take-Away Points

- Using claims data, we determined that insured African Americans with diabetes were less likely than whites to be treated with lipid-lowering agents, have their medication altered, or reach low-density lipoprotein cholesterol (LDL-C) goal.
- Rates of LDL-C testing, treatment, and goal attainment significantly improved in both races over time.
- Nonadherence to lipid-lowering drugs was higher among African Americans than among whites.

C4N); or (3) a mean glycosylated hemoglobin (A1C) level $\geq 7\%$ or a mean fasting plasma glucose ≥ 126 mg/dL on 2 separate occasions with a mean A1C $\geq 6.5\%$. Patients had to be 18 years or older at baseline and be continuously enrolled in the health plan, with pharmacy benefit coverage, for the 2-year baseline period.

achievement over a 10-year period in a large cohort of insured patients with diabetes receiving care in an integrated health-care delivery system. We also evaluate whether dyslipidemia management differed between African American and white patients after controlling for numerous patient clinical characteristics and sociodemographic factors. This includes controlling for economic barriers beyond the mere presence of health insurance with variables such as prescription drug and physician office visit copayments. Further, we explore whether racial differences in rates of LDL-C goal achievement could be explained by racial differences in treatment intensification of and adherence to lipid-lowering drugs.

METHODS

Study Population and Setting

All study patients received care through a large integrated health system serving southeastern Michigan. This health system includes a 900-member multispecialty salaried medical group that delivers care in Detroit and surrounding communities. We identified a retrospective cohort of patients from multiple sites who were managed by the medical group and had insurance coverage through an affiliated health maintenance organization. All patients had prescription coverage, with tiered copayments based on the covering entity's formulary. We followed cohort members from baseline (January 1, 1997, to December 31, 1998) until the first of either death, health plan disenrollment, or the end of the study period (ie, December 31, 2007). The Institutional Review Board of the Henry Ford Health System approved the study as described.

Inclusion and Exclusion Criteria

We used the National Committee for Quality Assurance's Health Plan Employer Data and Information Set (HEDIS) criteria to identify patients diagnosed with diabetes in the baseline period.²⁹ The patient had to meet at least 1 of the following 3 diagnostic definitions for diabetes: (1) ≥ 1 hospitalization or ≥ 2 outpatient visits with a diagnosis of DM (ICD-9-CM 250.xx); (2) a dispensing for insulin or an oral hypoglycemic medication (therapeutic class codes C4G, C4K, C4L, C4M, and

Data Sources

Information on patient characteristics, including age, sex, marital status, and race, was available from electronic data sources maintained by the health system. At the time of this data collection, race was usually based on self-report, but could have been assigned by administrative staff at the time of the initial clinical encounter. Medical claims and encounter data were used to identify and construct the following: clinical characteristics and diagnosis variables, the Deyo adaptation of the Charlson Comorbidity Index,³⁰ and measures reflective of medical care use (ie, frequency of outpatient visits and cardiology visits). Measures of laboratory test receipt and test results were obtained from an automated clinical laboratory system. Prescription drug claims data were used to compile prescription drug use and adherence measures. Medical group and health plan databases were linked using patients' unique medical record numbers. Use of automated data to identify patients with diabetes has been previously validated.^{31,32}

Analytical Variables

To examine trends in lipid management, we created indicator variables for LDL-C testing, treatment with a lipid-lowering agent (therapeutic class codes D7L, M4E, and M4F), and LDL-C goal attainment during baseline and in each year of follow-up. Patient LDL-C values were based on the average annual value for the 2-year baseline period and for each follow-up year. The clinical laboratory system was also used to derive variables reflective of baseline and annual mean A1C.

Comorbidity scores were calculated using the diagnostic data available during the baseline years to construct the Deyo adaptation of the Charlson Comorbidity Index.³⁰ Diagnostic (≥ 1 inpatient discharge diagnosis or ≥ 2 outpatient diagnoses) and procedural data were used to construct indicators for baseline cardiovascular risk factors and diseases. These included hypertension, left ventricular hypertrophy (LVH), peripheral vascular disease (PVD), cerebrovascular diseases (including stroke or transient ischemic attack), CHD (including unstable angina and myocardial infarction), retinopathy, non-traumatic lower extremity amputation, end-stage renal disease (ESRD), and smoking status. Smoking status was determined by the presence of at least 1 diagnosis of tobacco use

disorder (ICD-9-CM code 305.1) in the 1997-1998 baseline period.

Prescription claims data were used to compute the continuous measure of medication gaps (CMG), a measure of nonadherence in pharmacotherapy.^{33,34} CMG is the sum of treatment gaps in medication refills over the total number of days in the observation period. Since some subjects were taking more than 1 lipid-modifying medication, CMGs for all drugs within the lipid-lowering class were averaged to create a composite CMG. CMG was calculated for 4 classes of hyperlipidemic drugs: statins, fibrates, nicotinic acid, and bile acid sequestrants. Within each drug class, CMG indexes were computed for those patients who filled at least 1 prescription per year (N = 2553 for statins, N = 415 for fibrates, N = 11 for nicotinic acid, and N = 63 for bile acid sequestrants) in the period between the first prescription claim after January 1, 1997, and the last prescription available or December 31, 2007 (ie, if the last prescription extended past the last day of observation).

As we have done previously,^{35,36} medication nonadherence was measured as 1-CMG (ie, reverse-coded) and multiplied by 100 to provide a scale of 0 to 100. Therefore, higher scores reflected better medication adherence. Dichotomous versions of the reverse-coded CMG were created to categorize patients as either “adherent” or “nonadherent.” Patients whose reverse-coded CMG was less than 80% (ie, a gap in therapy greater than 20%) were classified as nonadherent whereas patients with a reverse-coded CMG greater than or equal to 80% were classified as adherent. Patients with no prescribed treatment were considered to have adherence equal to zero in the multivariable models. A cutoff of 80% has been used historically to differentiate adherent from nonadherent behavior³⁷⁻³⁹ and it is associated with the likelihood of achieving LDL-C goal.^{39,40}

Sociodemographic information included age, race, gender, and marital status. Medical claims and encounter data were used to determine clinic, copayment amounts, and number of outpatient and specialty care (cardiology) physician visits during the 2-year baseline period. Race was categorized as “white,” “African American,” or “other.” The “other” population was included in the analyses and is presented in the tables. However, interpretation of these data is difficult due to the heterogeneity of the population and its small sample size (n = 423; 3.7%); therefore, results specific to this subgroup are not described. Residential street address was used to estimate median household income and level of education (as represented by proportion of males and females with high school or lower level of education) using geographical information system technology which assigned values based on the census block-group of residence.⁴¹

Statistical Analyses

Rates of testing, treatment, and LDL-C goal attainment

stratified by race were estimated and compared by χ^2 tests for each follow-up year (1999-2007). Values were declared missing if the patient was not continuously enrolled for the entire year of interest. Multivariable logistic regression models (generalized estimating equation, or GEE)⁴² that account for repeated events (ie, testing, treatment, dose adjustment, and goal achievement during the subsequent follow-up years), while controlling for baseline factors, were used to estimate racial effects on testing, treatment, and goal attainment variables.

We used a stepped approach to modeling the association between race and lipid management among patients with diabetes. Several important covariates could confound and/or mediate an association between race and lipid management. A stepped approach to multivariate modeling allowed us to evaluate the effects of adding important covariates to the model. The first model was adjusted by year of follow-up. In addition to year of follow-up, the second model included baseline sociodemographic characteristics (gender, marital status, estimated household income, level of education, prescription drug and office visit copayments); lipid measures (LDL-C); baseline cardiovascular risk factors, such as hypertension, LVH, retinopathy, smoking status, PVD, CVD, CHD, amputation, ESRD; A1C; Charlson Comorbidity Index; number of outpatient visits; and number of cardiology visits. To determine the role of medication nonadherence on racial differences in LDL-C goal attainment, the third model included a flag for treatment with a lipid-lowering drug along with a dichotomous medication adherence variable. These time-varying variables reflected the annual averages for the previous calendar year (or the last available year if the previous calendar year was not available). Last, to control for African American patients clustering at particular care sites, clinic was added as an additional covariate to the second and third models.

RESULTS

Cohort Characteristics at Baseline

A total of 11,411 patients with diabetes were identified at 28 sites during the baseline period. The mean and median years of observation were 6.2 and 7.0, respectively, for both the overall study population and for each racial category. African American patients comprised 43.0% of the sample. Among African American patients, the mean age at baseline was 56.9 years (SD \pm 12.9 years) and 55.5% were female. Among the white patients, the mean age at baseline was 58.9 years (SD \pm 13.0 years) and 47.3% were female. The majority of cohort members had insurance coverage via an employer and were married; however, a higher proportion of the white patients were married relative to the African American patients. The mean number of outpa-

tient visits during the 2-year baseline period was 12.0 for African Americans and 12.7 for whites; the mean number of cardiology visits during the baseline period was 0.6 for African Americans and 0.8 for whites. Racial distributions across clinics varied considerably. Of the 28 sites, 11 (39%) had >25% African American patients, 6 (21%) had >40% African American patients, and 3 (11%) had >85% African American patients. Hypertension was highly prevalent in the total patient population (77.2%) and African American patients had a prevalence rate at least 10% higher than the other groups. Significant differences in rates were also found among other baseline clinical covariates, including PVD, CVD, CHD, LVH, ESRD, and smoking status. The proportion of African American patients with LDL-C testing in the 2-year baseline period was 56.7%. At baseline, 18.3% of African American patients had an LDL-C level <100 mg/dL and 23% were treated with lipid-lowering therapy. Among white patients, the proportion with LDL-C testing in the 2-year baseline period was 69.9%. At baseline, 25.6% of white patients had an LDL-C level <100 mg/dL and 33% were treated with lipid-lowering therapy. Nonadherence to lipid-lowering agents was higher among African American patients (41% of patients categorized as nonadherent) compared with white patients (26% of patients categorized as nonadherent); these differences were statistically significant ($P < .0001$). These baseline sociodemographic, healthcare access, and clinical characteristics are shown in **Table 1**.

Trends in Lipid Management

Over the follow-up period (1999-2007), 2 trends in lipid management were identified. First, rates of LDL-C testing, treatment, and goal attainment significantly improved in both races over time ($P < 0.001$). Second, the racial disparities in LDL-C testing, treatment, and goal attainment remained highly significant over time ($P < 0.001$).

Among African American patients, LDL-C testing rates increased from 48% in 1999 to 70% in 2007. The overall use of lipid-lowering agents increased from 23% to 39%. Consistent with increased testing and treatment, there was significant improvement in the proportion of patients at LDL-C goal. The proportion of African American patients with average LDL-C levels <100 mg/dL rose by 35% (from 24% to 59%) between 1999 and 2007.

Among white patients, LDL-C testing rates increased from 61% in 1999 to 77% in 2007. The overall use of lipid-lowering agents increased from 32% to 47% while dose adjustment decreased from 9% to 5%. The proportion of white patients with average LDL-C levels <100 mg/dL rose by 41% (from 35% to 76%) between 1999 and 2007. Yearly trends in lipid management are further detailed in **Table 2**.

Testing for Hypercholesterolemia

Without adjusting for any covariates, African American patients were less likely to be tested for LDL-C compared with white patients (OR 0.68; 95% CI 0.65-0.72, **Table 3**). However, after adjusting for sociodemographic and cardiovascular risk variables, year of follow-up, number of visits, clinic, and copayments for visits and prescriptions, the difference was no longer significant ($P = 0.352$). Baseline comorbidities found to be positively associated with testing for LDL-C in the adjusted model were hypertension, CHD, and retinopathy. Evidence of CHD during baseline increased the likelihood of testing by over a third (OR 1.39; 95% CI 1.19-1.63). Significant sociodemographic predictors of LDL-C testing in the adjusted model were increasing age, year of follow-up, increasing median income, and being married. No severity indicators were associated with testing.

Treatment With Lipid-Lowering Agents

After adjusting for sociodemographic and cardiovascular risk variables including year of follow-up, number of visits, clinic, copayments for visits and prescriptions, and LDL-C levels at baseline, African American patients were less likely to be treated with lipid-lowering agents (OR 0.70; 95% CI 0.62-0.79; **Table 3**) compared with white patients. Baseline comorbidities found to be associated with lipid-lowering treatment in the adjusted model were hypertension, CHD, retinopathy, and ESRD. Patients who had evidence of CHD during the baseline period were 1.26 times more likely to receive lipid-lowering treatment than those who showed no evidence of disease (OR 2.26; 95% CI 1.86-2.75). The severity indicator of number of cardiology visits was positively associated with initiation of lipid-lowering treatment; the Charlson Comorbidity Score was not. Significant sociodemographic predictors of treatment in the adjusted model were increasing age, year of follow-up, being male, and being married. Higher prescription copays but lower physician visit copays were positively associated with treatment with lipid-lowering drugs. LDL-C levels were also positive significant predictors of treatment (data not shown).

Treatment Intensification

After adjusting for numerous covariates, African American patients were significantly less likely to have their medication dosage altered or changed within class compared with white patients (OR 0.68; 95% CI 0.60-0.78; **Table 3**). Significant positive predictors of dose adjustment during follow-up in the adjusted model were evidence of baseline hypertension, CHD, ESRD, number of cardiology visits, LDL-C levels, being male, and being married. Visit copayment was inversely associated with dose adjustment.

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■ **Table 1.** Baseline (1997-1998) Characteristics of Patients With Diabetes by Race (N = 11,411)

	African American n = 4912 (43.0%)	White n = 6076 (53.3%)	Other n = 423 (3.7%)
Observation period (mean; median in years)	6.20; 7.0	6.18; 7.0	6.26; 7.0
Demographic variable			
Age at baseline ^a (mean ± SD)	56.9 ± 12.9	58.9 ± 13.0	54.4 ± 12.4
Gender (%)			
Male	44.5	52.7	53.2
Female	55.5	47.3	46.8
Current estimated income ^a \$ (mean ± SD; median)	35,094 ± 13,418; 32,918	52,187 ± 17,486; 49,903	56,803 ± 28,209; 52,088
Insurance type ^a (%)			
Employer sponsored	62.1	54.5	72.6
Medicare risk	13.9	27.3	16.5
Medicare supplemental	19.5	16.8	5.7
Other	4.5	1.4	5.2
Current marital status ^a (%)			
Divorced	9.0	5.9	3.3
Married	57.5	71.1	74.0
Single	19.8	10.5	12.5
Widowed	10.7	11.4	6.1
Legally separated	1.5	0.2	0.5
Unknown/missing	1.5	0.9	3.6
Clinical variable			
Charlson Comorbidity Index (mean ± SD)	2.4 ± 2.2	2.4 ± 2.0	2.0 ± 1.8
Hypertension ^a (%)	83.8	72.9	62.4
Peripheral vascular disease ^a (%)	2.8	4.7	1.7
Cerebrovascular disease ^a (%)	4.6	6.4	3.6
Coronary heart disease ^a (%)	4.2	7.2	4.3
Left ventricular hypertrophy ^a (%)	8.8	3.8	3.3
Tobacco use disorder ^{a,b} (%)	7.2	5.2	4.3
Retinopathy (%)	8.9	9.4	9.7
Non-traumatic lower extremity amputation (%)	0.6	0.4	0.0
End-stage renal disease ^a (%)	5.9	3.5	2.8
Medication adherence			
Adherence to all lipid-lowering drugs			
(1-CMG % mean ± SD; median)	78 ± 23; 86	85 ± 19; 93	82 ± 18; 88
Prevalence of nonadherence (%) ^{a,c}	41.1	26.0	40.4
Physician visits			
Annual outpatient visits (mean ± SD)	12.0 ± 10.0	12.7 ± 9.6	10.6 ± 9.0
Annual cardiology visits (mean ± SD)	0.6 ± 2.1	0.8 ± 2.2	0.5 ± 1.6

CMG indicates continuous measure of medication gaps; SD, standard deviation.

^a*P* ≤ .001. Differences tested only between African American and white patients, due to small "Other" category included for descriptive purposes only.

^bPatients diagnosed at least once in the period 1997-2002 with *ICD-9-CM* code 305.1 (tobacco use disorder).

^cNonadherence defined as 1-CMG × 100 < 80%.

■ **Table 2.** Trends in Lipid Management Within Race During the Follow-up Period (1999-2007)

Year	LDL-C Tested ^a Mean %				LDL-C Treated ^a Mean %				Medication Dose Adjusted ^a Mean %				At LDL-C Goal ^a Mean %			
	N	W	AA	Oth	N	W	AA	Oth	N	W	AA	Oth ^b	N	W	AA	Oth
1999	10,945	61	48	57	10,945	32	23	25	10,945	9	6	7	6031	35	24	35
2000	10,143	65 ^c	51	63 ^c	10,143	37	26	26 ^b	10,143	10	6 ^b	7	5984	38	26	34 ^b
2001	9463	76	68	74	9463	40	32	29 ^c	9463	12	7 ^c	9	6852	40	28	41 ^b
2002	8500	77	71	75	8500	48	40	39	8500	13	8	13	6313	42	31	38 ^b
2003	7645	78	73	76	7645	46	40	41	3838	11 ^b	13	9	5793	60	46	60
2004	6951	78	74	77	6951	50	45	44	3773	14	13	13	5318	64	53	62
2005	6006	78	75	84	6006	57	47	43	3292	11 ^b	10	15	4623	69	57	65
2006	5162	79	74	83	5162	57	50	53	2916	10 ^b	8 ^c	11	3983	74	62	68
2007	3114	77	70	78	3114	47	39	37	1781	5	5 ^b	2	2307	76	59	80

AA indicates African American; LDL-C, low-density lipoprotein cholesterol; Oth, other; W, White.

^aAll $P < .0001$ for comparisons to 1999 unless otherwise specified.

^bNot significant.

^c $P < .05$.

Goal Attainment and the Role of Nonadherence

Measures of medication nonadherence were higher among African American patients than among white patients at baseline (41% vs 26%, $P < .0001$, Table 1). Differences in the apparent treatment effect of lipid-lowering agents by race were partially mitigated after accounting for differences in nonadherence. Three multivariable logistic models were run on the effect of race on LDL-C goal attainment (Table 4). When adjusting for year of follow-up only (Model 1), African American patients were 41% less likely to attain an LDL-C goal of <100 mg/dL compared with white patients (OR 0.59; CI 0.56-0.63). After adjusting for year of follow-up, sociodemographic and cardiovascular risk variables, clinic, number of physician visits, and copayments for visits and prescriptions (Model 2), African American patients were 29% less likely to achieve LDL-C goal compared with white patients (OR 0.71; CI 0.63-0.79). Once the adherence variable and treatment indicator were introduced (Model 3), there was no statistically significant interaction between treatment and race. However, differences in

LDL-C goal attainment between African American and white patients remained statistically significant; African American patients were 22% less likely to achieve LDL-C goal levels compared with white patients (OR 0.78; CI 0.70-0.88).

DISCUSSION

In this study we found that the rates of cholesterol testing, treatment, and goal attainment significantly improved between 1999 and 2007 for patients with diabetes, regardless of race.

While it is apparent that improvements in the diabetes care of this population have occurred with time, improvements have been equal across races. Our data showed that disparities persisted over time even when overall improvements occurred for both races. To reduce the racial disparities, additional efforts and tailored interventions may be required.

In the unadjusted models, all LDL-C management indicators consistently demonstrated health and

■ **Table 3.** Odds Ratio Estimates of the Effect of Race (Relative to White) on Lipid Testing, Treatment, and Dose Adjustment

Variable	Unadjusted OR (95% CI)		Adjusted ^a OR (95% CI)	
	African American	Other	African American	Other
LDL-C testing	0.68 (0.65-0.72)	0.96 (0.84-1.10)	0.96 (0.87-1.05)	1.06 (0.90-1.26)
Lipid treatment	0.75 (0.70-0.79)	0.79 (0.67-0.93)	0.70 (0.62-0.79)	0.83 (0.67-1.02)
Dose adjustment	0.66 (0.61-0.71)	0.72 (0.58-0.90)	0.68 (0.60-0.78)	0.79 (0.61-1.01)

CI indicates confidence interval; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio.

^aAdjusted for clinic, year of follow-up, gender, marital status, level of education, income, age, clinical baseline covariates, glycemic control, baseline LDL-C level, prescription drug and physician visit copay amounts, number of outpatient and cardiology visits, and baseline Charlson Comorbidity Index score.

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■ **Table 4.** Multivariable^a Logistical Models of the Effect of Race (Relative to White) on LDL-C Goal Attainment (OR, CI)

Independent Variable	Model 1 ^a	Model 2 ^b	Model 3 ^c
African American			
OR (CI)	0.59 (0.56-0.63) ^d	0.71 (0.63-0.79) ^d	0.78 (0.70-0.88) ^d
Other race			
OR (CI)	0.89 (0.75-1.05)	1.00 (0.82-1.20)	1.07 (0.88-1.31)

CI indicates confidence interval; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio.
^aModel 1 adjusted for year.
^bModel 2 adjusted for clinic, year of follow-up, gender, marital status, level of education, income, age, clinical baseline covariates, baseline LDL-C level, prescription drug and physician visit copay amounts, number of outpatient and cardiology visits, and baseline Charlson Comorbidity Index Score.
^cModel 3 adjusted for Model 2 variables plus medication adherence.
^d*P* < .001; differences tested between white and African American only.

healthcare disparities between African American and white patients. Relative to white patients, African American patients were less likely to be tested, treated, or to achieve LDL-C goal levels. In the adjusted models, there was no longer any evidence of a racial disparity in LDL-C testing but the disparities in treatment and goal attainment remained. Furthermore, when treated, African American patients were less likely to be adherent to their medications or to have their medication intensified. Both clinical inertia—defined as lack of treatment intensification in a patient not at the recommended goals for care—and patient nonadherence to medication regimens may be important factors in the observed differences in clinical control.^{43,44} Unconscious physician bias may also play a role in the differential rates of treatment.⁴⁵ The demonstrated disparities in medication adjustment implicate clinician behavior; however, a previous study conducted at the same healthcare system clearly demonstrated that delays in treatment intensification are also associated with patient factors.⁴⁶ Previous research has demonstrated that patients who experience adverse side effects from medications or who have difficulty sticking to their dosing regimen were more likely to be nonadherent and less likely to have their treatment intensified.^{26,47} Patients may also be unwilling to consider treatment intensification or a new medication due to cost, lack of trust in their physician, and unresolved concerns about current medications.⁴⁸ Another possible factor for not being at goal is the existence of comorbid chronic conditions, including depression and chronic pain. Comorbid conditions such as depression and pain have been shown to affect patients' adherence to medication and ability to follow a recommended diet.^{49,50} It is apparent that both clinician and patient factors need to be addressed to achieve LDL-C goal levels.

This study should be viewed in light of its limitations. Our reliance on existing automated data precluded measurement of several cardiovascular risk factors including alcohol use, BMI, and family history. Use of ICD-9-CM codes to determine smoking status likely under-ascertained the number of smokers;

however, the magnitude is unknown. Furthermore, “treatment” was a relatively crude measure, as it consisted of only a flag for whether or not a patient had filled any prescription for a lipid-lowering drug in each calendar year. Therefore, we could not further explore whether adherence to a specific treatment, as opposed to any treatment, or treatment duration played a role on observed differential effects of lipid-lowering medication by race. Further, although patient nonadherence was taken into consideration through claims data, primary nonadherence⁵¹ (ie, where prescribed medications are filled but never used or a prescription written but never dispensed) was not measured. There was also loss to follow-up from baseline to the final follow-up year—although there was no evidence of differential attrition by race. Finally, the study sample was all insured and receiving care from 1 integrated delivery system in southeastern Michigan, which limits our ability to generalize findings to populations across the United States and to uninsured individuals.

As has been described by others,^{11,19,52} we found racial disparities in use of cholesterol-lowering medication and recommended goal achievement levels among insured patients with DM. However, this study adds to previous research, as these disparities were found despite controlling for patient nonadherence to medication in addition to numerous access, clinical, and sociodemographic variables. Together, these study findings suggest the importance of physicians and patients becoming more aware of lowering cholesterol among African Americans with DM. Although all patients in the current study had health insurance coverage and hence, a measure of financial access to care, other factors might have contributed to the observed racial disparities. Room for improvement in implementing treatment guidelines in clinical practice is clearly evident. Differences in physician screening,²³ prescribing practices,^{19,53} ability to pay,⁵⁴⁻⁵⁶ or differences in medication adherence by race^{15,47,57} may all play a role in these observed differences and warrant further study. Assessing the relative contribution of these and other potential causes of racial disparities in cholesterol treatment is the next step needed to help mitigate these differences.

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