**ORIGINAL REPORTS: DIABETES**

**DEMOGRAPHIC DIFFERENCES IN THE TREATMENT AND CONTROL OF GLUCOSE IN TYPE 2 DIABETIC PATIENTS: IMPLICATIONS FOR HEALTH CARE PRACTICE**

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**INTRODUCTION**

The US population is becoming older and more ethnically diverse with a high prevalence of overweight and obesity. Diabetes is more prevalent with increasing age and in non-White ethnic minorities and is often associated with obesity. Thus, the prevalence of type 2 diabetes in the United States continues to rise along with costs, estimated at $174 billion US in 2007.

Diabetes is the single largest contributor to end-stage renal disease. Better diabetes control, assessed by glycosylated hemoglobin (HbA1c), improves microvascular outcomes including nephropathy. Type 2 diabetes is strongly linked with macrovascular disease, which ultimately impacts most diabetic patients. Yet, evidence that tight diabetes control reduces macrovascular complications is inconsistent although macrovascular benefits may be evident after longer follow-up periods than most clinical trials.

Disparities between outcomes of Black and White patients with type 2 diabetes are well documented and especially prevalent in the Southeast United States. Moreover, while control of cardiovascular risk factors including diabetes improved across race/ethnicity groups, disparities persist. Thus, insight on modifiable clinical factors that contribute to racial disparities in diabetes control could inform strategies for improving health equity.

Some evidence suggested that tight diabetes control does not improve or might worsen outcomes, which led to an HbA1c goal of <8% for diabetic patients ≥65 years and younger patients with advanced clinical diseases. After considering the evidence, the American Diabetes Association guidelines continued to recommend an HbA1c goal of <7% for diabetic patients of all ages but note less stringent goals may be appropriate for patients with advanced clinical disease uncontrolled with usual management strategies.

Given these reports, race (Black–White), age, and sex-related differences in diabetes treatment and control were examined among adults with type 2 diabetes mellitus receiving care at civilian outpatient clinics in the Southeast United States. An attempt was made to quantify clinically modifiable factors that could potentially attenuate...
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demographic differences in diabetes control.

In our report, glycosylated hemoglobin (HbA1c) served as the measure for comparing glucose control in Blacks and Whites, yet differences in HbA1c between Blacks and Whites may partially reflect glucose-independent factors.15,16 Glucose-independent Black-White differences in HbA1c were reported in subjects with normal glucose metabolism, pre-diabetes, and diabetes and these differences increased as glucose tolerance decreased.17 Concerns were raised that glucose sampling in various studies was insufficient to definitively establish race/ethnicity differences in the glucose-independent contribution to HbA1c.18,19 Previous studies suggesting that racial differences in hemoglobin glycation and erythrocyte turnover account for glucose-independent variation in HbA1c were recently challenged with a recommendation to focus more careful attention on racial differences in non-fasting glucose.19 Until those studies are completed, there may or may not be a ∼0.2%–0.5% glucose-independent elevation of HbA1c among Black relative to White patients with diabetes.17,19,20

Multiple risk factor control and evidence-based prescribing are important for reducing both microvascular and macrovascular complications of diabetes.11,13,14,21–23 Race and sex-related disparities among diabetics in clinical outcomes including coronary heart disease are partially explained by variations in hyperlipidemia and hypertension management.24–26 Demographic differences in these modifiable risk factors were examined as secondary outcomes.

METHODS

This retrospective study used data on patients seen from January 2004 through December 2008 at clinical sites in the Outpatient QUality Im-
Table 1. Demographic, cardiovascular risk factor, and insurance data for diabetic patients by age, race and sex subgroups

<table>
<thead>
<tr>
<th>Age, years</th>
<th>Race</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50</td>
<td>Black</td>
<td>Male</td>
</tr>
<tr>
<td>38.6 43.8</td>
<td>38.4–57.1</td>
<td>100/0 0/0</td>
</tr>
<tr>
<td>50–64</td>
<td>White</td>
<td>Female</td>
</tr>
<tr>
<td>35.0 34.6</td>
<td>34.3–34.8</td>
<td>102.2 112.7</td>
</tr>
<tr>
<td>≥65</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>34.8–35.3</td>
<td>31.7–32.2</td>
<td>114.6 104.3</td>
</tr>
</tbody>
</table>

LDL-C, mg/dL 112.6 109.5 111.7–113.6 108.6–110.3 101.0–102.9 113.7–115.4 103.6–104.9 102.2 112.7

LDL <100, % 39.8 43.6 53.7 39.0 49.5 140.7 136.7 138.2 138.4

SBP, mmHg 134.6 139.9 140.1 140.2–141.2 136.3–137.2 137.7–138.8 138.0–138.8

DBP, mmHg 81.7 81.3 76.7 80.7–81.3 79.3–79.8 80.8–81.4 79.1–79.6

BP <140/<90, % 59.5 51.0 50.1 48 56.8 54.2 52.7

BP <130/<80, % 28.5 23.3 25.0 22.6 27.3 25.2 25.6

HbA1c, % (initial) 8.0 7.6 7.1 8.0 7.3 7.6 7.6

HbA1c <7%, % (initial) 42.4 46.4 55.3 38.9 53.4 47.5 47.9

HbA1c <7%, % (final) 7.7 7.4 7.0 7.7 7.1 7.4 7.4

HbA1c <7%, % (final) 7.6–7.7 7.3–7.4 6.9–7.0 7.7–7.8 7.1–7.2 7.3–7.4 7.3–7.4

HbA1c % 45.3 50.0 59.6 44.7 55.6 50.0 52.1

Data are presented as mean and 95% CI or percentages of patients only.

For age: 1 vs 2 and 1 vs 3: P<.05; ^P<.01; ‡P<.001; 2 vs 3: 8P<.05; 9P<.01 1P<.001.

For race and sex: P<.05; 6P<.01; 7P<.001.

(pseudo) Max-rescaled R-squared was selected to quantify information gain, when including predictors in comparison to the null (“intercept only”) model. SAS Version 9.2 was used for all analyses. Two-sided P values <.05 were accepted as statistically significant.

RESULTS

The 22,285 adults with type 2 diabetes were selected from 155,411 affected patients seen in network practices 2004–2008. Patient exclusions included Veterans Affairs (N=61,665), missing race (N=49,082), <2 visits (N=18,998), no values for HbA1c (N=14,630) or LDL-cholesterol (N=6,316), and no prescription medications in the electronic health records system (N=3,588), ineligible age (N=1,056), and missing sex (N=76).

Mean age was 55.8±14.6 (SD) years with 61% White:39% Black, 42.5% women, body mass index 34.0±9.3 kg/m². Most patients had hypertension (88.4%) and hyperlipidemia (89.4%); 20.3% had chronic kidney disease, 9.5% coronary heart disease, 9.7% depression, 7.5% stroke/transient ischemic attack, and 5.4% peripheral arterial disease.

HbA1c fell with age, was higher in Black than White patients, and similar in men and women (Table 1). Diabetes control improved more between initial and final visits in Black than White diabetics (5.8% vs 2.2%, P<.0001) with a commensurate decrease in absolute racial difference (14.5% vs 10.9%, P<.0001). White and male patients had commercial and Medicare insurance more often but Medicaid less often than Black and female patients. Differences in HbA1c between initial and final values for race groups by age are depicted in Figure 1.

The proportion of patients with a visit in the last 6 months rose with age, was greater in White than Black patients but similar in men and women (Table 2). Therapeutic inertia in all patients declined with age, was greater in...
Black than White patients and men than women. Therapeutic inertia calculated for patients with HbA1c ≥7% rose with age, was lower in Black than White patients but similar in men and women. Evidence-based prescribing for β-blockers increased with age, was greater in Black than White patients and men than women. The number, class, and dose equivalents of anti-diabetic medications prescribed are provided (Table 3). The proportion of patients prescribed biguanides and insulin decreased with age, was higher in Black than White patients and women than men. The percentage prescribed sulfonylureas increased with age, was higher in Black than White patients and men than women. Thiazolidinediones prescriptions were highest in patients aged 50–64, similar in Blacks and Whites, and higher in women than men. Dipeptidyl peptidase (DPP)-4 inhibitor prescriptions were highest among patients aged 50–64 and more frequent in Whites than Blacks and men than women.

Statistically significant variables are shown in univariable analysis and carried into multivariable analyses (Table 4). The model fit between values for deviance and Pearson goodness-of-fit statistics and a chi-square distribution with appropriate degrees of freedom was adequate in all cases. The pseudo-$R^2$ output from this model is generally reported as “percent of information gain”. Information gain is analogous but not identical to variance explained or true $R^2$ in linear regression. The more commonly used analogous term variance explained is used henceforth. The initial multivariable analyses (Multivariable–1) accounted for 43% of variance in HbA1c control to $\leq 7\%$. Uninsured status was significant when added in Multivariable–1, (OR 0.82, 95% CI 0.75–0.89), yet variance explained remained at 43%. When initial HbA1c was included (Multivariable–2), the model explained 47% of variance in HbA1c control. When insurance status was added, the variance explained in HbA1c control remained 47%.

Adding anti-diabetic medications (Multivariable–3) raised variance explained in HbA1c control to 49%. The five major classes of anti-diabetic medications including insulin were associated with poorer HbA1c control (not shown) as patients with higher HbA1c received more anti-diabetic medications. Uninsured status remained predictive of lower diabetes control (OR 0.84, 95% CI 0.77–0.92), yet variance explained remained 49%. Diabetes
**Table 2.** Selected processes of care including visit frequency and therapeutic inertia

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Number of Pts</th>
<th>Visit/yr Number</th>
<th>Therapeutic Inertia, %</th>
<th>Therapeutic Inertia (HbA1c ≥7%), %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=6996</td>
<td>3.8</td>
<td>51.1</td>
<td>72.74–74.0</td>
</tr>
<tr>
<td>&lt;50</td>
<td>n=8801</td>
<td>3.5</td>
<td>50.1–52.1</td>
<td></td>
</tr>
<tr>
<td>50–64</td>
<td>n=6489</td>
<td>3.7</td>
<td>48.7</td>
<td></td>
</tr>
<tr>
<td>≥65</td>
<td>n=8696</td>
<td>3.8</td>
<td>44.4</td>
<td></td>
</tr>
</tbody>
</table>

**Process of Care**

- **Visits/yr Number**: 3.8, 3.5, 3.7
- **Visit prior 6 mo**: 77.60%
- **Visit w/ 3 mo elevated HbA1c**: 59.60%
- **Therapeutic Inertia, %**: 51.1
- **Therapeutic Inertia (HbA1c ≥7%), %**: 72.74–74.0

**EB prescribing, %**

- **RAS blocker, %**: 80.70%, 73.1%, 69.6%
- **β-blocker, %**: 55.10%, 55.3%, 54.1%
- **CHD or CHF**: 385, 1154, 1413
- **Hyperlipidemia or CVD Dx, LDL >40 yr & ≥1 CV risk factor**: 61.0%, 69.6%, 67.3%
- **Therapeutic Inertia >50%, %**: 53.6, 51.0, 46.6

**Table 3.** Classes of anti-diabetic medications and dose equivalents prescribed to diabetic patients by age, race/ethnicity, and sex

<table>
<thead>
<tr>
<th>Age (in years)</th>
<th>Number of pts</th>
<th>Any anti-DM med, %</th>
<th>Anti-DM meds, N</th>
<th>95% CI</th>
<th>Anti-diabetic med class</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50</td>
<td>n=6990</td>
<td>79.70%</td>
<td>1.5</td>
<td>1.5–1.5</td>
<td><strong>α-glucosidase inhibitors, %</strong></td>
</tr>
<tr>
<td>50–64</td>
<td>n=8801</td>
<td>78.60%</td>
<td>1.4</td>
<td>1.4–1.5</td>
<td>1.70, 0.5, 0.5</td>
</tr>
<tr>
<td>≥65</td>
<td>n=6489</td>
<td>73.9%</td>
<td>1.3</td>
<td>1.3–1.5</td>
<td>0.26, 0.32, 0.35</td>
</tr>
</tbody>
</table>

**Data are presented as mean and 95% confidence intervals or as percentages only.**

For age: 1 vs 2 and 1 vs 3: a P<.05; b P<.01; c P<.001; 2 vs 3: d P<.05; e P<.01 f P<.001.

For race and sex: g P<.05; h P<.01; i <.001.

EB=evidence-based; CKD=chronic kidney disease; CHF=chronic heart failure; MI=myocardial infarction; CVD=cardiovascular disease.

Percent of patients with therapeutic inertia scores >50%, i.e., anti-diabetic medications were intensified on fewer than 50% of visits when HbA1c was ≥7%.**
Table 4. Results from univariate and multivariate logistic regression models predicting HbA1c <7%

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Univariable</th>
<th>Multivariable 1</th>
<th>Multivariable 2</th>
<th>Multivariable 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variance in HbA1c control explained</td>
<td>0.43</td>
<td>0.47</td>
<td>0.49</td>
<td></td>
</tr>
<tr>
<td>Age (10 yr increase)</td>
<td>1.20(^b)</td>
<td>1.20(^d)</td>
<td>1.13(^d)</td>
<td>1.12(^d)</td>
</tr>
<tr>
<td>White race</td>
<td>1.17–1.22</td>
<td>1.17–1.24</td>
<td>1.10–1.16</td>
<td>1.09–1.15</td>
</tr>
<tr>
<td>Male</td>
<td>1.59(^d)</td>
<td>1.35(^d)</td>
<td>1.18(^b)</td>
<td>1.12(^d)</td>
</tr>
<tr>
<td>BMI (5 kg/m(^2) increase)</td>
<td>0.92(^b)</td>
<td>0.93</td>
<td>0.95</td>
<td>0.94</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.87–0.97</td>
<td>0.86–1.01</td>
<td>0.87–1.03</td>
<td>0.86–1.02</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>0.97(^c)</td>
<td>0.98</td>
<td>0.98</td>
<td>0.98</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.95–0.99</td>
<td>0.96–1.03</td>
<td>0.96–1.00</td>
<td>0.96–1.00</td>
</tr>
<tr>
<td>Male</td>
<td>1.58(^d)</td>
<td>1.18(^a)</td>
<td>1.17(^a)</td>
<td>1.24(^b)</td>
</tr>
<tr>
<td>Visits / yr</td>
<td>1.45–1.72</td>
<td>1.03–1.34</td>
<td>1.02–1.35</td>
<td>1.07–1.42</td>
</tr>
<tr>
<td>Therapeutic inertia</td>
<td>1.43(^d)</td>
<td>0.92</td>
<td>0.95</td>
<td>1.00</td>
</tr>
<tr>
<td>Depression</td>
<td>1.31–1.55</td>
<td>0.81–1.05</td>
<td>0.83–1.09</td>
<td>0.87–1.15</td>
</tr>
<tr>
<td>CKD</td>
<td>1.29(^d)</td>
<td>1.05</td>
<td>1.04</td>
<td>1.04</td>
</tr>
<tr>
<td>CVD(^f)</td>
<td>1.17–1.41</td>
<td>0.91–1.20</td>
<td>0.90–1.20</td>
<td>0.90–1.20</td>
</tr>
<tr>
<td>CVD(^f)</td>
<td>1.13–1.50</td>
<td>0.98–1.50</td>
<td>0.96–1.49</td>
<td>0.98–1.53</td>
</tr>
<tr>
<td>CVD(^f)</td>
<td>1.34(^d)</td>
<td>0.97</td>
<td>0.99</td>
<td>1.02</td>
</tr>
<tr>
<td>FQHC vs Academic</td>
<td>1.24–1.44</td>
<td>0.86–1.10</td>
<td>0.87–1.12</td>
<td>0.90–1.16</td>
</tr>
<tr>
<td>Private vs Academic</td>
<td>1.03(^d)</td>
<td>1.11(^d)</td>
<td>1.09(^d)</td>
<td>1.13(^d)</td>
</tr>
<tr>
<td>Initial HbA1c</td>
<td>1.02–1.04</td>
<td>1.10–1.12</td>
<td>1.08–1.12</td>
<td>1.12–1.15</td>
</tr>
<tr>
<td>Diabetes Meds, N</td>
<td>0.73(^d)</td>
<td>0.71(^d)</td>
<td>0.75(^d)</td>
<td>0.73(^d)</td>
</tr>
<tr>
<td>0.72–0.73</td>
<td>0.70–0.72</td>
<td>0.74–0.75</td>
<td>0.72–0.74</td>
<td></td>
</tr>
<tr>
<td>0.74–0.75</td>
<td>0.72–0.73</td>
<td>0.74–0.75</td>
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<tr>
<td>0.72–0.73</td>
<td>0.70–0.72</td>
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<td>0.74–0.75</td>
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<td>0.74–0.75</td>
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<tr>
<td>0.74–0.75</td>
<td>0.72–0.73</td>
<td>0.74–0.75</td>
<td>0.72–0.74</td>
<td></td>
</tr>
<tr>
<td>0.75–0.77</td>
<td>0.94–0.95</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data = odds ratio, 95% confidence intervals. \(^a\) P <.05, \(^b\) P <.01, \(^c\) P <.001, \(^d\) P <.0001.
\(^e\) The five most commonly prescribed classes of anti-diabetic medications were included in the univariable and multivariable–3 analyses (biguanides, dipeptidyl peptidase-4 inhibitors, insulin, sulfonylureas, and thiazolidinediones). In both analyses, all medication classes were associated with a significantly lower likelihood of HbA1c control to <7%.
\(^f\) CVD included coronary heart disease, cerebrovascular disease and peripheral arterial disease.

Race was the strongest demographic predictor of HbA1c <7% with Whites 59% (OR 1.59, 95% CI 1.51–1.68) more likely to obtain control than Blacks in univariable analysis.

control was better at academic than private practice sites and not different from federally qualified health centers. The changes from univariable associations coincided with higher initial HbA1c (mean 7.99 [95% CI 7.93–8.04], 7.58 [7.54–7.61], 7.19 [7.15–7.23]) and prescription of more anti-diabetic medications (1.67 [1.64–1.70], 1.53 [1.50–1.55], 1.02 [1.00–1.05]) at academic than FQHC or private practices, respectively.

Three modifiable variables (initial HbA1c, visit frequency, therapeutic inertia) accounted for 47.9% of variance in HbA1c control. These covariates attenuated the effect of race (OR 1.21, 95% CI 1.13–1.30) and age (OR 1.13, 95% CI 1.11–1.16) on diabetes control compared to univariable ORs, which indicates confounding. When BMI or insurance were added to the three modifiable covariates listed, the variances in HbA1c control explained were essentially unchanged at 46.7% and 48.0%, respectively.

**DISCUSSION**

This report focused on demographic differences in diabetes control among patients at civilian practices in the Southeast U.S. HbA1c values were higher and control lower in Blacks than Whites. HbA1c values declined and control to <7% increased with age (Table 1). Race was the strongest demographic predictor of HbA1c <7% with Whites 59% (OR 1.59, 95% CI 1.51–1.68) more likely to obtain control than Blacks in univariable analysis. Each 10-year increment in age raised the probability of control 20% (Table 4). Diabetes control was not significantly different between men and women.

A secondary study objective was to identify covariates that could explain demographic differences in diabetes control. The initial set of covariables, while significantly related to diabetes control in univariable analysis, (Table 4, Multivariable–1), did not alter the age effect and marginally attenuated the race effect on diabetes control. When initial HbA1c was added (Multivariable–2, Table 4), the contribution of race and age to diabetes control declined (no overlap univariable and multivariable-2 95% confidence intervals). These findings are consistent with data (Table 1, Figure 1) showing initial HbA1c values were lower in Whites than Blacks and declined with advancing age. Adding initial HbA1c increased the variance explained (pseudo R\(^2\) information gain) in diabetes control explained by the model from 43% to 47%.

Adding information on the number and class (not shown) of diabetic
medications did not alter the independent relationship of age and race or visit frequency and therapeutic inertia to diabetes control. Better diabetes control at private clinics and federally qualified health centers than academic clinics observed in univariable analysis was reversed for the former and eliminated for the latter in Multivariable–3. These findings coincide with higher initial HbA1c values and more anti-diabetic medications prescribed at academic than other clinical sites.

Three clinically modifiable variables including initial HbA1c, number of annual healthcare visits, and therapeutic inertia explained ~48% of variance in diabetes control. Together, these three modifiable factors attenuated the impact of race by nearly two-thirds (univariable OR 1.59, 95% CI 1.51–1.68 vs. multivariable OR 1.21, 95% CI 1.13–1.30) and age by about a third (univariable OR 1.20, 95% CI 1.17–1.22 vs. multivariable OR 1.13, 95% CI 1.11–1.16) on diabetes control. Previous reports indicate HbA1c values are higher in Blacks than Whites when diabetes is diagnosed25 and when treatment is initiated.20,30 These findings suggest that earlier diagnosis and treatment of diabetes in Black patients could reduce disparities in diabetes control. Black patients were less likely than White patients to be seen in the six months prior to the study end date but more likely to be seen within three months of elevated HbA1c (Table 2). Regular follow up, even when HbA1c is <7%, represents another factor that may reduce disparities in diabetes control.

Therapeutic inertia was greater in Black than White diabetic patients overall. Moreover, a greater percentage of Black than White patients had a therapeutic inertia score >50%, i.e., anti-diabetic medications increased on fewer than half of visits when HbA1c was above goal. When therapeutic inertia was restricted to patients with elevated HbA1c, Blacks were more likely to have anti-diabetic medications increased (Table 2). Adherence with anti-diabetic medications is lower in Black than White patients but does not appear to fully explain the racial disparity in diabetes control.30 The hypertension literature suggests that therapeutic inertia is a major factor in blood pressure control and that treatment intensification lowers blood pressure, even among patients assessed by their providers as less adherent.31,32 These findings are consistent with the observation that therapeutic intensified when HbA1c values are elevated improves diabetes control, especially among Black patients.33

The civilian clinics in our report attenuated the racial disparity in diabetes control when comparing initial and final HbA1c values (Table 2, Figure 1). Veterans Affairs Clinics reportedly eliminated the racial disparity in diabetes control between initial and final visits, adjusted for a glucose-independent 0.2% higher HbA1c in Black patients, which may reflect more equitable care.20 As noted earlier, the contribution of glucose-independent factors to Black-White differences in HbA1c have not been fully resolved.15–20 Even if confirmed, the glucose-independent factors are unlikely to fully explain the racial differences in HbA1c we observed, especially in patients <65 years old. More specifically, Black–White differences in diabetes control declined with aging (Figure 1), which may reflect greater equality of health care for Medicare age than younger individuals.11

HbA1c control was higher among patients ≥65 years than younger individuals (Table 1), although the National Center for Quality Assurance 2010 recognizes HbA1c <8% rather than <7% as the goal for patients 65 to 75 years old.12 However, the American Diabetes Association recommends goal HbA1c <7% for diabetic patients of all ages but note less stringent control may be appropriate for patients with multiple comorbidities and/or elevated values after applying usual management strategies.13 The three medically modifiable covariables including initial HbA1c, visit frequency and therapeutic inertia, also reduced age-related differences in diabetes control. Thus, greater attention to these factors could lead to better control in diabetic patients <65 and especially <50 years old.

Lipid lowering statin therapy was prescribed less often in diabetic patients <50 years than older patients, which could contribute to lower control rates of LDL-cholesterol to <100 mg/dL in younger diabetics (Table 1). Statins appear to be underutilized in at risk diabetic subjects <50 years old, which is consistent with an earlier report.34 However, Black patients received statin prescriptions more often than White patients but achieved lower control rates for LDL-cholesterol. Diabetic men and women were equally likely to have statins prescribed, yet men attained higher control rates. Our findings suggest greater attention to controlling hyperlipidemia, and not just to prescribing a statin, may be crucial in reducing cardiovascular risk in women and Blacks with diabetes.23,24,34,35 This study was not designed to explain discrepancies between statin prescriptions and LDL-cholesterol control.

Poorer hypertension control among patients on pharmacotherapy with aging and in Black than White patients (Table 1) is well described.26,36 Hypertension control decreases vascular complications in diabetes,37 which raises the importance of reducing disparities in blood pressure. Evidence-based prescribing for renin-angiotensin blockers was greater in Blacks than Whites, which confirms a report in heart failure.38 Blacks were also more likely than Whites to receive guideline-recommended prescriptions for β-blockers and statins.13,23 Our findings on statin therapy contrast with a previous report of lower statin use among Blacks than Whites who had at least one hospital admission for diabetes.25
Study limitations include reliance on data from diverse clinic types using various electronic health record systems with potential variations in data capture. Insurance information was often unavailable, limiting power of multivariate regression analyses including this variable. While insurance status did not increase the variance explained in diabetes control, it was an independent predictor of control and plays an important role in access to healthcare. Despite limitations, our findings are consistent with and extend previous reports that have been cited.

In summary, diabetes control was lower in Black than White patients as well as diabetics <50 and 50–64 than those who were ≥65 years old. These disparities were attenuated after accounting for differences in initial HbA1c, frequency of care and therapeutics. Our findings suggest greater attention to early diagnosis and treatment, ensuring regular health care visits, and reducing therapeutic inertia could improve diabetes control and health equity by reducing race and age-related disparities in control and potentially in outcomes.

ACKNOWLEDGMENTS
This research was supported primarily by a grant from Takeda Pharmaceuticals America, Inc., which also provided input on study design and editing of the paper. Dr. Keith Szymanski, an employee of Takeda Pharmaceuticals, is a co-author of the paper and participated in the design of the study and editing of the manuscript.

REFERENCES


