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

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Objective: Identifying modifiable covariables that reduce demographic disparities in controlling type 2 diabetes could inform efforts to improve health equity.

Research Design and Methods: This retrospective study utilized electronic health record data on 22,285 adults with type 2 diabetes seen at 110 outpatient clinics in the Southeast U.S. from 2004–2008. Demographic differences in diabetes control and modifiable covariables which reduce those disparities were quantified using descriptive and logistic regression analysis.

Results: Patients were 55.8±14.6 (SD) years old, 57.5% women, 61.0% White:39.0% Black and had baseline body mass index 34.0±9.3 kg/m^2 and HbA1c 7.6±1.9%. The percentage with HbA1c <7% was higher in Whites than Blacks (55.6% vs. 44.7%, $P<.0001$) and rose with age in all patients from 45.3% at <50, to 50.0% at 50–64, and 59.6% at ≥65 years, $P<.001$. White vs. Black race (odds ratio [OR] 1.59, 95% confidence interval [CI] 1.51–1.68) and age/10 years (OR 1.20/10 years, 95% CI 1.17–1.22) were predictors of HbA1c <7% in univariable logistic regression. In multivariable analysis, three modifiable covariables (initial HbA1c, therapeutic inertia, visit frequency) accounted for 47.9% of variance in diabetes control. When accounting for these modifiable covariables, the independent impact of race/ethnicity (OR 1.21, 95% CI 1.13–1.30) and age (OR 1.13, 95% CI 1.11–1.16) on HbA1c control declined.

Conclusions: Race and age-related difference in diabetes control declined significantly when modifiable covariates were considered. Greater attention to early diagnosis and treatment, ensuring regular healthcare visits and overcoming therapeutic inertia could improve diabetes control and health equity. (*Ethn Dis*. 2012;22:29-37)

Key Words: Diabetes, health disparities, glycosylated hemoglobin, anti-hyperglycemic therapy

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INTRODUCTION

The US population is becoming older and more ethnically diverse with a high prevalence of overweight and obesity.[1,2] Diabetes is more prevalent with increasing age and in non-White ethnic minorities and is often associated with obesity.[3,4] Thus, the prevalence of type 2 diabetes in the United States continues to rise along with costs, estimated at $174 billion US in 2007.[5]

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Diabetes is the single largest contributor to end-stage renal disease.[5–7] Better diabetes control, assessed by glycosylated hemoglobin (HbA1c), improves microvascular outcomes including nephropathy.[8] 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[9] although macrovascular benefits may be evident after longer follow-up periods than most clinical trials.[10]

Disparities between outcomes of Black and White patients with type 2 diabetes are well documented[11] and especially prevalent in the Southeast United States.[12] Moreover, while control of cardiovascular risk factors including diabetes improved across race/ethnicity groups, disparities persist.[11] 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.[12,13] 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.[14]

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

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 erthyrocyte 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 Improvement Network (OQUIN).[27,28] Each clinic signed a Business Associate Agreement approved by the institutional review board at the Medical University of South Carolina, which authorized research use of de-identified data.

Study Database

There were 169 practice sites in the Southeast United States (South Carolina, Tennessee, Georgia, Alabama, North Carolina, Virginia) contributing information for 2004–2008 on 1,276,169 adult patients including 155,411 with type 2 diabetes. Participating sites included private and hospital-managed community-based practices, Federally Qualified Health Centers, hospital based clinics including academic and residency-training clinics. Patients at 59 Veterans Affairs clinics were excluded, given smaller racial disparities in hypertension and diabetes control at these sites.[20,27]

Data were obtained from electronic medical/health record systems used at participating practices. Data included: age, race, sex, insurance status and insurer, height, weight, visit dates, anti-diabetic prescription medications, glycosylated hemoglobin, LDL-cholesterol, and serum creatinine; diagnoses including type 2 diabetes, hyperlipidemia/cholesterolemia, hypertension, coronary heart disease (ICD9 410–414), cerebrovascular disease (ICD9 431–438), chronic kidney disease (ICD9 403, 585, 586), peripheral arterial disease (ICD9 250.7, 440.2, 443.9) and depression (ICD9 296.2, 296.3, 298.0, 300.4, 311).

Subjects

Study patients had type 2 diabetes and were ≥18 years old, men or women, and Caucasian or African American. Inclusion criteria included ≥2 clinic visits and ≥1 HbA1c, LDL-cholesterol value and prescription for any medical condition during 2004–2008.

Study objectives

Primary objectives included defining differences by age, race, and sex in (a) HbA1c control to <7% and (b) anti-diabetic prescription medications by class and dose equivalents (daily dose prescribed/FDA maximum approved daily dose). Secondary objectives included: (a) defining demographic differences in frequency of visits, key laboratory tests, therapeutic inertia, and guideline-recommended prescriptions for statins, β-blockers, and renin-angiotensin system blockers for comorbid cardiovascular and chronic kidney disease and (b) identifying clinically modifiable factors that could explain disparities in diabetes control.

Therapeutic inertia for diabetes was defined as no change in the number or dose of anti-diabetic medications when the most recent HbA1c value preceding or coincident with the visit was ≥7%. Therapeutic inertia was calculated by dividing the number of visits with HbA1c ≥7% when therapy was not intensified by the number of visits with HbA1c ≥7%.

Data Reporting and Analysis

Baseline descriptive data are presented as mean and standard deviation. Comparative data are provided as mean and 95% confidence intervals. Chi square and Wilcoxon rank sum tests were used as appropriate to obtain p-values. Univariable and multivariable logistic regression analyses were performed to quantify covariate impact on diabetes control (HbA1c <7%). Multivariable regression was conducted with a pre-specified list of factors including visit frequency, body mass index, anti-diabetic medications (number and class), and comorbidities (ICD9 codes listed above). The multivariable analyses were conducted with and without insurance status, as these data were missing on ~50% of patients. The deviance and Pearson goodness-of-fit statistics were used to determine adequacy of model fit. The likelihood based

Table 1. Demographic, cardiovascular risk factor, and insurance data for diabetic patients by age, race and sex subgroups

	Age, years			Race		Sex	
	<50	**50–64**	**≥65**	**Black**	**White**	**Male**	**Female**
Number of pts	*n*=6996	*n*=8801	*n*=6489	*n*=8696	*n*=13590	*n*=9450	*n*=12809
Age, yrs	38.6	57.1[c]	72.6 [c]	54.2	56.8[i]	55.8	55.8
	38.4–38.8	57.1–57.2	72.4–72.7	53.9–54.5	56.6–57.1	55.5–56.0	55.6–56.1
% <50, 50–64, ≥65	100/0/0	0/100/0	0/0/100	34.1/41.4/24.6	29.7/38.3/32.0 [i]	31.0/40.8/28.2	31.7/38.5/29.8[h]
% Black / White	42.4/57.7	40.9/59.2	32.9/67.1[c]	100/0	0/100	28.2/71.8	47.0/53.0 [i]
% Male / Female	41.9/58.1	43.9/56.1[a]	41.1/58.9[f]	30.7/69.3	49.9/50.1[i]	100/0	0/100
BMI, kg/m^2	35.0	34.6[b]	32.0 [c,f]	34.5	33.7[i]	32.6	35.0 [i]
	34.8–35.3	34.3 – 34.8	31.7–32.2	34.3–34.8	33.5–33.9	32.3–32.8	34.8–35.2
LDL-C, mg/dL	112.6	109.5[c]	102.0 [c,f]	114.6	104.3 [i]	102.2	112.7[i]
	111.7–113.6	108.6–110.3	101.1–102.9	113.7–115.4	103.6–104.9	101.5–103.0	112.0–113.4
LDL <100, %	39.8	43.8 [c]	53.7[c]	39.0	49.5% [i]	51.50%	41.0% [i]
SBP, mmHg	134.6	139.9 [c]	140.1[c,f]	140.7	136.7 [i]	138.2	138.4[h]
	134.0–135.2	139.4–140.4	139.5–140.6[f]	140.2–141.2	136.3–137.2	137.7–138.8	138.0–138.8
DBP, mmHg	81.7	81.3[c]	76.7 [c,d]	81.0	79.5 [i]	81.1	79.3 [i]
	81.4–82.0	81.0–81.6	76.4–77.1	80.7–81.3	79.3–79.8	80.8–81.4	79.1–79.6
BP <140/<90, %	59.5	51.0 [c]	50.1[c]	48	56.8 [i]	54.2	52.7[g]
BP <130/<80, %	28.5	23.3 [c]	25.0 [c,e]	22.6	27.3 [i]	25.2	25.6
HbA1c, % (initial)	8.0	7.6 [c]	7.1 [c,f]	8.0	7.3 [i]	7.6	7.6
	7.9–8.0	7.6–7.7	7.1–7.2[f]	8.0–8.1	7.3–7.3	7.5–7.6	7.6–7.6
HbA1c <7%, % (initial)	42.4	46.4 [c]	55.3 [c,f]	38.9	53.4 [i]	47.5	47.9
HbA1c, % (final)	7.7	7.4 [c]	7.0 [c,f]	7.7	7.1 [i]	7.4	7.4 [h]
	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<7%, % (final)	45.3	50.0[c]	59.6 [c,f]	44.7	55.6 [i]	50.0	52.1[h]
Insurance status							
Commercial,%	58.6	50.3 [c]	39.0 [c,f]	40	55.8 [i]	53.7	46.3 [i]
Medicaid, %	19.8	10.7 [c]	6.2 [c,f]	17	9.2 [i]	7.9	15.5 [i]
Medicare, %	22.4	27.7 [c]	67.6 [c,f]	31.9	41.3 [i]	38.4	37.1[g]
Uninsured, %	17.9	9.6 [c]	2.9 [c,f]	14.9	7.2 [i]	9.0	11.2 [i]

Data are presented as mean and 95% CI or percentages of patients 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] $<.001$.

(pseudo) Max-rescaled R-squared[29] 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/m^2. 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

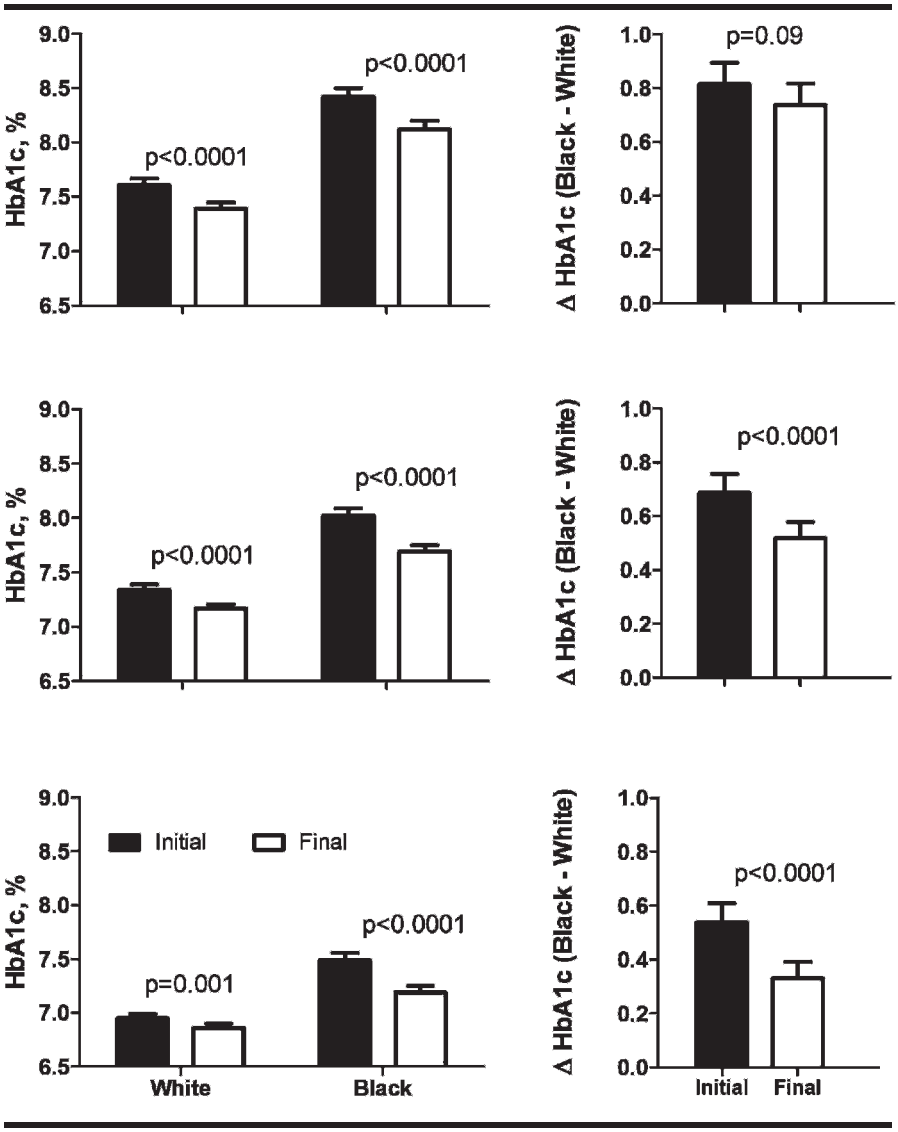

Fig 1. Initial and final (first and last) glycosylated hemoglobin (HbA1c) values are displayed (mean and 95% confidence intervals) for Black and White patients (left side). Values are displayed for patients <50 (top panel), 50–64 years (middle panel) and ≥65 years old (bottom panel). The difference in HbA1c between Black and White patients at the initial visit (solid bar) and last visit (open bar) are also displayed. All of the differences between Black and White patients at the initial and final visits are significant at *P*<.0001. The p-value provided over the change in HbA1c bars compares racial differences between the first and last visits. The racial differences in HbA1c also declined with increasing age on initial and final values (both *P*<.0001).

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 of renin-angiotensin system blockers and statins increased with age, was greater in Black than White patients, and 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".[29] Information gain is analogous but not identical to variance explained or true R^2 in linear regression. The more commonly used analagous term variance explained is used henceforth. The initial multivariable analyses (Multivariable–1) accounted for 43% of variance in HbA1c control to <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

	Age, years			Race		Sex	
	<50	**50–64**	**≥65**	**Black**	**White**	**Male**	**Female**
Number of Pts	*n*=6996	*n*=8801	*n*=6489	*n*=8696	*n*=13590	*n*=9450	*n*=12809
Process of Care							
Visits/yr Number	3.8	3.5	3.7	3.8	3.6	3.3	3.9
	3.7–3.9	3.4–3.6[c]	3.6–3.7[c]	3.7–3.9	3.5–3.6[h]	3.3–3.4	3.8–3.9[i]
Visit prior 6 mo	77.60%	79.0% [c]	80.8%[c,f]	75.50%	81.4%[i]	78.80%	79.30%
Visit w/i 3 mo elevated HbA1c	59.60%	56.4% [c]	48.0% [c,f]	62.10%	50.4%[i]	55.20%	54.80%
Therapeutic Inertia, %	51.1	48.7	44.4	52.9	45.1	49.1	47.5
	50.1–52.1	47.8–49.5 [c]	43.4–45.5[c,f]	52.1–53.8	44.4–45.9[i]	48.2–49.9	46.8–48.3[h]
Therapeutic Inertia (HbA1c≥7%), %	73.1	73.4	77.1	71.8	76.1	73.8	74.4
	72.2–74.0	72.5–74.2	76.1–78.1[c,f]	71.0–72.6	75.3–76.8[i]	73.0–74.7	73.7–75.1
[1]Therapeutic Inertia >50%, %	53.6	51.0 [c]	46.6[c,f]	56.0	47.1[h]	50.9	50.3
EB prescribing, %							
RAS blocker, %: CKD, CHF, or Post-MI	80.70%	73.1%[b]	69.6%[c]	79.50%	65.1%[i]	69.20%	74.50%
	n=233	*n*=750	*n*=945	*n*=962	*n*=966[i]	*n*=780	*n*=1147
β-blocker, %	55.10%	55.3% [c]	54.1% [c,f]	60.60%	51.6%[i]	54.10%	55.40%
CHD or CHF	*n*=385	*n*=1154	*n*=1413	*n*=1025	*n*=1927[i]	*n*=1436	*n*=1514[i]
Statin							
Hyperlipidemia or CVD Dx, LDL ≥100;	61.0%	69.6%[c]	67.3% [c,f]	68.10%	65.2%[i]	66.70%	66.0%
>40 yr & ≥1 CV risk factor	*n*=6587	*n*=8684	*n*=6400	*n*=8425	*n*=13246	*n*=9182	*n*=12463

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

	Age (in years)			Race		Sex	
	<50	**50–64**	**≥65**	**Black**	**White**	**Male**	**Female**
Number of pts	*n*=6990	*n*=8801	*n*=6489	*n*=8696	*n*=13590	*n*=9450	*n*=12836
Any anti-DM med, %	79.70%	78.60%	73.9% [b,d]	82.0%	74.7%[f]	77.20%	77.80%
Anti-DM meds, *N*	1.5	1.4	1.3	1.5	1.4	1.4	1.4
95% CI	1.5–1.5	1.4–1.5	1.3–1.3[b,d]	1.5–1.5	1.3–1.4[f]	1.4–1.4	1.4–1.4
Anti-diabetic med class							
α-glucosidase inhibitors, %	1.70	0.5[b]	0.5[b]	1.10	0.7[e]	0.8	0.9
Dose equivalents[a]	0.26	0.32	0.35	0.36	0.32	0.35	0.31
Biguanides, %	61.20	58.3 [b]	47.3[b,c]	58.70	54.3[f]	54.0	57.5[f]
Dose equivalents[a]	0.44	0.48 [b]	0.46 [b,c]	0.48	0.45[f]	0.49	0.45 [f]
DPP-4 Inhibitor, %	7.70	8.30	6.7 [b,c]	6.20	8.7[f]	8.30	7.2[e]
Dose equivalents[a]	1.03	1.03	1.01[b,c]	0.93	1.10[f]	1.03	1.00
Meglitinides, %	0.9	1.0	1.6 [b,c]	1.40	0.9[e]	1.0	1.20
Dose equivalents[a]	0.17	0.25 [b]	0.21[b,c]	0.22	0.23	0.23	0.19[e]
Sulfonylureas, %	36.10	39.8 [b]	42.1[b,c]	42.50	37.2[f]	40.2	38.5[d]
Dose equivalents[a]	0.33	0.31 [b]	0.32[b,c]	0.31	0.33	0.34	0.31[f]
Thiazolidinedione, %	21.30	24.6[b]	21.8[c]	22.70	22.80	24.60	21.3[f]
Dose equivalents[a]	0.62	0.63[b]	0.58[c]	0.56	0.66[f]	0.60	0.64[f]
Insulin, %	19.10	16.3[b]	13.0 [b,c]	24.10	11.2[f]	14.10	17.7[f]

DM = diabetes mellitus;
[a] proportion of FDA approved maximum daily dose;
[b] $P<.01$ between age groups 1 and 2 or 1 and 3; [c] $P<.01$ between age groups 2 and 3; [d] $P<.05$, [e] $P<.001$, [f] $P<.0001$ between the race and sex groups.

Table 4. Results from univariate and multivariate logistic regression models predicting HbA1c <7%

	Univariable[e]	Multivariable 1	Multivariable 2	Multivariable[e] 3
Variance in HbA1c control explained		0.43	0.47	0.49
Age (10 yr increase)	1.20[d]	1.20[d]	1.13[d]	1.12[d]
	1.17–1.22	1.17–1.24	1.10–1.16	1.09–1.15
White race	1.59[d]	1.35[d]	1.18[c]	1.12[a]
	1.51–1.68	1.24–1.48	1.07–1.29	1.02–1.23
Male	0.92[b]	0.93	0.95	0.94
	0.87–0.97	0.86–1.01	0.87–1.03	0.86–1.02
BMI (5 kg/m^2 increase)	0.97[c]	0.98	0.98	0.98
	0.95–0.99	0.96–1.03	0.96–1.00	0.96–1.00
Hypertension	1.58[d]	1.18[a]	1.17[a]	1.24[b]
	1.45–1.72	1.03–1.34	1.02–1.35	1.07–1.42
Hyperlipidemia	1.43[d]	0.92	0.95	1.00
	1.31–1.55	0.81–1.05	0.83–1.09	0.87–1.15
Depression	1.29[d]	1.05	1.04	1.04
	1.17–1.41	0.91–1.20	0.90–1.20	0.90–1.20
CKD	1.30[c]	1.21	1.19	1.23
	1.13–1.50	0.98–1.50	0.96–1.49	0.98–1.53
CVD[f]	1.34[d]	0.97	0.99	1.02
	1.24–1.44	0.86–1.10	0.87–1.12	0.90–1.16
Visits / yr	1.03[d]	1.11[d]	1.09[d]	1.13[d]
	1.02–1.04	1.10–1.12	1.08–1.12	1.12–1.15
Therapeutic inertia	0.73[d]	0.71[d]	0.75[d]	0.73[d]
	0.72–0.73	0.70–0.72	0.74–0.75	0.72–0.74
FQHC vs Academic	1.24[d]	1.12	1.06	0.95
	1.17–1.32	1.02–1.24	0.96–1.17	0.86–1.05
Private vs Academic	1.65[d]	1.06	0.92	0.83[b]
	1.54–1.77	0.95–1.19	0.82–1.04	0.74–0.94
Initial HbA1c	0.51[d]		0.71[d]	0.74[d]
	0.50–0.52		0.69–0.73	0.72–0.76
Diabetes Meds, N	0.75[d]			0.95[d]
	0.73–0.77			0.94–0.95

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.

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 antidiabetic 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.

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.

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 diagnosed[25] 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 intensification 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.[39] 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 therapeutic inertia. 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.

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