

Diabetes Care in Black and White Veterans in the Southeastern U.S.

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OBJECTIVE — Eliminating health disparities is a national priority, but progress has been difficult because of racial/ethnic differences in insurance coverage and access to health care. We investigated whether there were differences in diabetes care in the Veterans Administration (VA), where health care access should be relatively uniform.

RESEARCH DESIGN AND METHODS — A1C and plasma glucose were compared before/after diagnosis of diabetes.

RESULTS — Data were available for 1,456 black and 2,624 white veterans who met criteria for consistent primary care. Over 4–5 years before and after diagnosis, blacks had similar glucose and ~0.2% higher A1C levels than whites, and A1C differences could be attributed to glucose-independent associations between race and A1C. Blacks and whites also had comparable intervals between diagnostic-level hyperglycemia and diagnosis and between diagnosis and drug initiation. However, A1C was higher in blacks at the time of diagnosis (7.8 vs. 7.1%) and at initiation of pharmacotherapy (8.5 vs. 7.8%) (both $P < 0.001$). Differences in A1C at diagnosis and drug initiation were too large to be explained by differences in age, sex, BMI, and glucose-independent associations between race and A1C.

CONCLUSIONS — In the VA, glucose levels are generally comparable in blacks and whites except at the times of diagnosis and initiation of pharmacotherapy, when glucose levels are higher in blacks. While understanding the basis for such residual disparities may be important to improve the health of racial/ethnic minorities in the U.S., a health care system with structure and organization similar to that in the VA may also contribute importantly to relieving disparities in health.

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Racial/ethnic minorities in the U.S. suffer from disparities in diabetes-related health. Minority groups have a high prevalence of type 2 diabetes (1), and the public health impact of the increased prevalence of diabetes in minorities is exacerbated by related morbidity and mortality that are higher than in whites (2). During the 1980s, diabetes-

related mortality for white men and women decreased 1.6 and 4.5%, respectively, yet mortality increased among blacks 11 and 5.5%, respectively. Among blacks with diabetes, hypertension, peripheral vascular disease, and retinopathy are all more common than in whites. Similarly, end-stage renal disease (3), lower-extremity amputations related to diabetes

(4), and rates of hospitalization and disability due to diabetes for blacks are nearly double those for whites; increased diabetes morbidity remains after adjustment for prevalence of hypertension (3).

Such increased morbidity appears to be due at least in some part to poor metabolic control (5). Blacks in the National Health and Nutrition Examination Surveys (NHANES) 1988–1994 and 1999–2002 had the highest prevalence of A1C $>8\%$ and highest average A1C compared with other ethnic groups (6), and a similar disparity was found in a Kaiser Permanente population (7). Such disparities are thought to reflect differences in socioeconomic status (8) and insurance coverage and related access to care (9). If such factors are the dominant basis for disparities in health, then disparities should not be present in settings where access to care is relatively uniform. We tested this hypothesis by comparing glucose and A1C levels in veterans receiving consistent follow-up care at medical centers in the southeastern U.S.

RESEARCH DESIGN AND METHODS

This study was approved by the Emory University Institutional Review Board. The sample population was a retrospective cohort of patients identified in the Corporate Data Warehouse for the Veterans Integrated Service Network (VISN) 7 (Veterans Administration [VA] medical centers in South Carolina, Georgia, and Alabama). We selected patients who had 1) diabetes diagnosed on 1 October 2002 or later, 2) consistent primary care (three or more visits over ≥ 2 years before the date of diagnosis, and four or more visits over ≥ 3 years after the diagnosis, including the “diagnostic” visit as one of the visits), 3) race information available, and 4) A1C and random plasma glucose values available within 3 months before and 6 weeks after the index date; the relatively modest requirement for consistent primary care (essentially one visit a year) is necessary to ensure that providers have had some opportunity to interact with the patients. In this VISN, 269,434 veterans had a primary care visit in 2008, and 44,806 had ≥ 7 consecutive years of follow-up with one or more primary care visits a year and

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two or more outpatient glucose measurements each year; 5,436 met our diabetes selection criteria, and 4,080 of these had race information available. A smaller number also had A1C and random plasma glucose values available at different time points (see text and the figure and table legends).

The date of hyperglycemia in the diabetes range was assigned according to the date of having any value twice or any two of outpatient random plasma glucose values ≥ 126 mg/dl before 10:00 A.M., random plasma glucose ≥ 200 mg/dl at a later time, or A1C $\geq 6.5\%$ (random plasma glucose > 125 mg/dl or A1C $\geq 6.5\%$ confers a high likelihood of having diabetes [10,11], and VA health care providers frequently check A1C levels in patients not known to have diabetes). The date of diagnosis was assigned according to initial use of the diabetes ICD-9 code 250.xx in primary care, use of the code twice in any setting, or prescription of a diabetes drug (whichever came first)—criteria establishing the disease (12). The use of medications was assessed during the period between 12 and 24 months after the index date. Medications were categorized as metformin, sulfonylureas, thiazolidinediones, insulin, and “other;” patients could be using more than one drug at a time.

Glucose and A1C values were assessed from 3 months before to 6 weeks after the dates of 1) diabetes-range hyperglycemia, 2) diagnosis, and 3) initial prescription of a diabetes drug. Values were also averaged over each of 1–4 years before diagnosis and 1–5 years after diagnosis. We included all A1C measurements, but to minimize potential confounding due to the stress of acute illnesses, we excluded glucose values obtained during hospitalizations. Glucose measurements were relatively common before diagnosis (a minimum of 371 measurements in blacks at year –4), whereas A1C measurements were less frequent (57 measurements in blacks at that time), but A1C measurements were more common after the index date (a minimum of 201 glucose and 333 A1C measurements in blacks at year +5).

Glucose and A1C were measured in VA clinical chemistry laboratories. Glucose was assessed with U.S. Food and Drug Administration–approved platforms such as the Beckman DXC or LX20 (Beckman Coulter, Fullerton, CA) or Roche P or COBAS (Roche Diagnostics, Indianapolis, IN), and A1C with National

Table 1—Patient characteristics at time of diagnosis of diabetes

	Black	White	P
n	1,456	2,624	
Age (years)	57.8 (57.3–58.3)	64.9 (64.5–65.3)	<0.001
Sex (% female)	4.9 (3.8–6.0)	1.6 (1.1–2.1)	<0.001
BMI (kg/m ²)	30.3 (30.0–30.6)	30.0 (29.8–30.2)	0.18
Date of diagnosis (month/day/year)	10/16/03	10/27/03	0.86
Days hyperglycemia to diagnosis			
Mean (95% CI)	479 (454–504)	504 (485–523)	0.22
Median (interquartile range)	333 (31–770)	356 (62–820)	0.12
Days diagnosis to initial drug Rx			
Mean (95% CI)	290 (265–315)	328 (309–347)	0.04
Median (interquartile range)	3 (0–427)	5 (0–537)	0.03

Data are percent and are either means (95% CI), with differences tested by *t* test, or median (interquartile range), with differences tested by Mann-Whitney *U* test.

Glycohemoglobin Standardization Program–approved methods, mainly high-performance liquid chromatography such as the Tosoh G7 (Tosoh Bioscience, South San Francisco, CA), but also immunological methods (from both Beckman Coulter and Roche Diagnostics).

For descriptive statistics, continuous variables were analyzed by *t* tests and categorical variables by χ^2 or Mann-Whitney *U* tests. Multiple linear regression models were used to assess the relationship between A1C values and race, adjusting for potential confounders including glucose. All analyses used SAS version 9.2 (SAS Institute, Cary, NC).

RESULTS— The 4,080 study patients were younger and included more women than the 1,356 patients who lacked race information: age 62 vs. 66 years ($P < 0.0001$), 2.8 vs. 1.2% female ($P < 0.0001$), and BMI 30.1 vs. 30.1 kg/m² ($P = 0.97$), respectively. Among the study patients (Table 1), blacks were younger than whites (58 vs. 65 years) and more likely to be female (4.9 vs. 1.6%) (both $P < 0.001$), but had comparable BMI (30.3 vs. 30.0 kg/m², $P = \text{NS}$). The average date of initial diabetes diagnosis was 16 October 2003 for blacks versus 27 October 2003 for whites ($P = \text{NS}$), and the period between initial diabetes-level hyperglycemia and diagnosis was comparable in blacks versus whites (479 vs. 504 days, $P = \text{NS}$); medications were initiated slightly earlier after the date of diagnosis in blacks (290 vs. 328 days, $P = 0.04$). Between 12 and 24 months after the index date, medications were used slightly but significantly more often in blacks (91 vs. 88%, $P = 0.022$), including metformin (34 vs. 29%, $P = 0.002$), sulfonylureas

(29 vs. 26%, $P = 0.048$), and insulin (5.4 vs. 3.6%, $P = 0.005$); there were no significant differences in use of thiazolidinediones (7.0 vs. 7.3%) or other drugs (0.3 vs. 0.7%).

Figure 1 shows A1C and random plasma glucose levels for blacks and whites at each of years 1–4 before diagnosis, at diagnosis, and years 1–5 after diagnosis. Both at years 1–4 before and years 1–5 after the date of diagnosis, random plasma glucose levels in blacks were generally slightly but not significantly lower than values in whites. Including both blacks and whites, A1C averaged 6.4% at years 1–4 before diagnosis and 6.9% at years 1–5 after diagnosis. Despite the similarity in random glucose levels, at both years 1–4 before and years 1–5 after the date of diagnosis, A1C levels in blacks were slightly but significantly higher than values in whites (average difference 0.11% before diagnosis and 0.24% after diagnosis, all $P < 0.005$ for blacks versus whites except for years –3 and –1 before diagnosis). After adjusting for age, BMI, and medical center, blacks had A1C values that were 0.20 higher than in whites 2 years before diagnosis and 0.22 higher 2 years after diagnosis. These small differences were generally within the range attributable to the glucose-independent association between race and A1C (see below).

In contrast, blacks had significantly higher levels of both plasma glucose and A1C at both the date of diagnosis (154 vs. 148 mg/dl and 7.77 vs. 7.11%) and when a glucose-lowering medication was first prescribed (176 vs. 169 mg/dl and 8.53 vs. 7.84%) (all $P < 0.05$) (Table 2). The differences in A1C at the times of diagnosis (0.66%) and first medication prescrip-

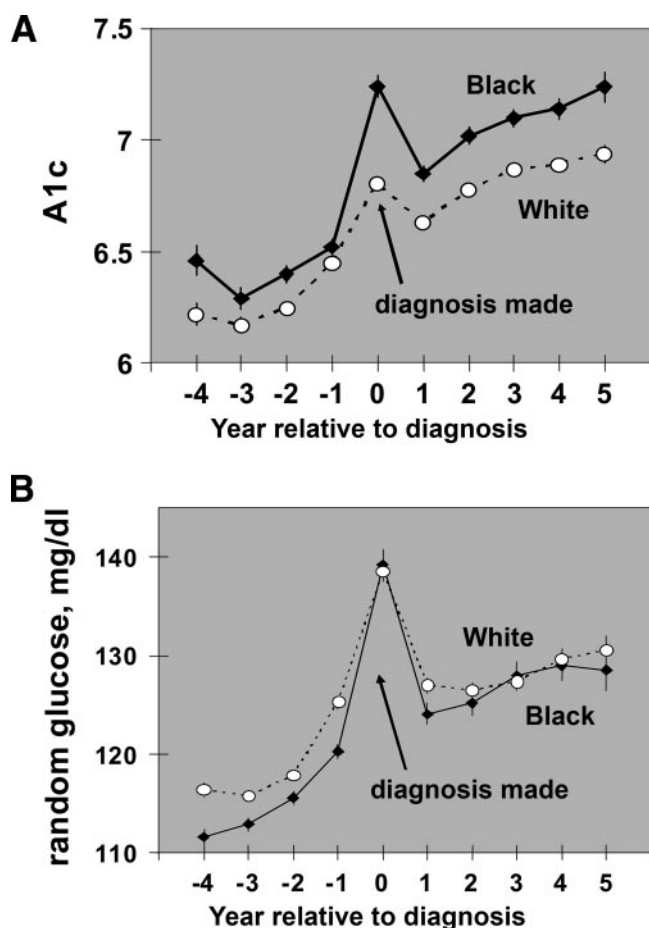


Figure 1—Levels of A1C (A) and random plasma glucose (B) in black and white veterans before and after the diagnosis of diabetes. Years before and after the date of diagnosis are shown and measures averaged for each patient within 6 months before and 6 months after the date of diagnosis and each year before and after the date of diagnosis. Data are means \pm SEM. Numbers of black and white veterans, respectively, contributing to data points were as follows: year -4 , 126 and 244; year -3 , 217 and 434; year -2 , 389 and 742; year -1 , 570 and 1,047; year 0, 1,258 and 2,166; year $+1$, 1,218 and 2,183; year $+2$, 1,199 and 2,198; year $+3$, 1,240 and 2,207; year $+4$, 1,063 and 1,846; year $+5$, 571 and 953.

tion (0.69%) were too large to be attributable to the glucose-independent association between race and A1C. As

shown by the multivariate analyses in Table 3, at both of these times, A1C levels were not influenced by the date of mea-

Table 2—Random plasma glucose and A1C in black and white veterans within 3 months before and 6 weeks after the date of diagnosis of diabetes and the date of initial prescription of a diabetes medication

	Blacks	Whites	P
Random plasma glucose			
At date of diabetes diagnosis (mg/dl)	154 \pm 79	148 \pm 61	0.035
n	1,054	1,924	
At start of diabetes medication (mg/dl)	176 \pm 88	169 \pm 69	0.041
n	730	1,321	
A1C			
At date of diabetes diagnosis (%)	7.77 \pm 2.37	7.11 \pm 1.60	<0.001
n	993	1,635	
At start of diabetes medication (%)	8.53 \pm 2.46	7.84 \pm 1.74	<0.001
n	798	1,321	

Data are numbers of subjects with measurements available and are means \pm SD. More patients had either glucose or A1C available than had both glucose and A1C available.

surement and were slightly higher with greater BMI and either slightly lower or not influenced by greater age. A1C was more strongly affected by the level of random plasma glucose and by the time of day glucose was measured (likely because of the Staub-Traugott effect [13], glucose tolerance is better after meals [and later in the day], so the same glucose value obtained later in the day reflects a higher A1C level). After adjusting for such confounders, the glucose-independent association between black race and A1C was 0.24% (units) at the time of initial diabetes-level hyperglycemia, 0.38% at diagnosis, and 0.40% at the first prescription of a diabetes drug (all $P < 0.001$). Although sex was not included as a covariate because of small numbers, the findings were changed only in the second decimal in analyses which included only men (not shown).

The differences in A1C between blacks and whites at diagnosis and initial pharmacotherapy were not associated with differences in numbers of visits or frequency of measurement of glucose or A1C levels. Between the dates of diabetes-level hyperglycemia and diagnosis, blacks and whites averaged 6.9 vs. 7.5 outpatient visits, 5.2 vs. 5.1 random plasma glucose measurements, and 2.2 vs. 2.3 A1C measurements (all $P = NS$). Between diagnosis and the first prescription of a diabetes drug, blacks and whites averaged 5.7 vs. 6.0 outpatient visits, 3.8 vs. 3.5 random plasma glucose measurements, and 3.2 vs. 3.3 A1C measurements (all $P = NS$).

CONCLUSIONS— Our results demonstrate that race is not a significant determinant of good metabolic control of diabetes in veterans with adequate follow-up. Among patients receiving consistent primary care in VA medical centers in South Carolina, Georgia, and Alabama, blacks and whites diagnosed with diabetes generally had fairly good metabolic control, with A1C levels at \sim 6.4% at years 1–4 before and 6.9% at years 1–5 after diagnosis. At these times, blacks had similar glucose levels and \sim 0.2% higher A1C levels than whites, and these differences in A1C were in the range attributable to the glucose-independent association between race and A1C. However, blacks had higher A1C and glucose levels than whites when the diagnosis was made and when the first diabetes drug was prescribed. Moreover, the disparities in A1C at these times (0.6–0.7%) were

Table 3—Multivariate regression analysis of factors influencing A1C

	Coefficient	SEM	P
At time of diabetes-level hyperglycemia (n = 1,335)			
Random plasma glucose (mg/dl)	0.018	0.0005	<0.0001
Time of day of random glucose (hours since midnight)	0.102	0.016	<0.0001
Actual date (days since 10/1/2002)	0.00002	0.00013	0.8685
Age (years)	-0.0049	0.0033	0.1381
BMI (kg/m ²)	0.00297	0.00658	0.6516
Black race	0.245	0.069	0.0004
At time of diagnosis of diabetes (n = 2,144)			
Random plasma glucose	0.0184	0.0004	<0.0001
Time of day of random glucose	0.032	0.013	0.0166
Actual date	-0.00001	0.00012	0.9273
Age	-0.0077	0.0031	0.0113
BMI	0.01780	0.00597	0.0029
Black race	0.383	0.065	<0.0001
At time of initial prescription of diabetes medication (n = 1,686)			
Random plasma glucose	0.0166	0.0005	<0.0001
Time of day of random glucose	0.027	0.018	0.1244
Actual date	-0.00006	0.00008	0.4150
Age	-0.0071	0.0040	0.0801
BMI	0.01639	0.00752	0.0295
Black race	0.405	0.082	<0.0001

Adjustment also included medical center (not shown). A1C is the dependent variable in the model; white race was the racial reference value. Positive estimate values show the extent to which unit changes in the factor contribute to higher A1C values, and negative estimate values show the converse.

too large to attribute to the glucose-independent association between race and A1C and were not associated with having fewer visits or glucose or A1C measurements.

The pattern of a gradual rise in glucose and A1C levels over years 1–4 before diagnosis, followed by a more rapid rise in glucose and A1C during the year prior to diagnosis, is consistent with previous reports (14,15); we also found a further increase in glycemia before initiation of pharmacotherapy. The factors contributing to the rapid rise are not known but might include a “vicious cycle” of effects of hyperglycemia-induced increases in inflammation and oxidative stress on susceptible β -cells. Our findings of a glucose-independent association between race and A1C are also consistent with previous reports (16); the basis for glucose-independent higher A1C in blacks is also not known.

Despite patient characteristics that predispose to poorer health, direct comparisons have generally shown better diabetes care in VA settings than non-VA settings. Veterans tend to be nonwhite, be unemployed, and have lower income, lower health status, and higher illness

burden than nonveterans (17), factors that increase the likelihood of poor-quality health care (18). However, patients managed in VA settings receive better diabetes-related processes of care (19) and exhibit lower A1C levels (20). Good VA care has been attributed to a combination of structural organization, use of electronic medical records and monitored “quality indicators,” and emphasis on evidence-based practice guidelines (21). But even within the VA, blacks are somewhat less likely than whites to have A1C <7% (22).

A meta-analysis in 2006 found A1C levels to average 0.65% higher in blacks than in whites (5). In the NHANES nationally representative samples of the U.S. population, A1C levels were 8.21% in blacks with diagnosed diabetes compared with 7.60% in whites in 1988–1994 and 8.02 vs. 7.30% in 1999–2002 (23), improving in both groups, but persistently worse in blacks. In NHANES 1999–2002, the likelihood of A1C <7% in blacks versus whites was unaffected by adjustment for age, sex, level of education, poverty level, and insurance coverage (6), but such adjustments may not fully account for the contributions of so-

cio-ecologic factors. In the Translating Research Into Action for Diabetes (TRIAD) Study of an insured population, where A1C measurement was less frequent and A1C levels were 0.2% higher in blacks than in whites (24), cost-related underuse of medications was also significantly higher in blacks than in whites (8), due in part to lower income and higher out-of-pocket drug costs.

In contrast, our analysis shows that in the VA setting, glucose levels are comparable or lower in blacks than in whites, and A1C levels are generally only modestly higher in blacks than in whites (and within the range attributable to the glucose-independent association between race and A1C). These findings are consistent with our previous observation in a municipal hospital population that racial disparities in glycemic control are largely abolished by use of a uniform treatment algorithm, under which rates of provider intensification of therapy and patient medication adherence were comparable (25). Presumably, the VA “system” of access to care, low cost for medications, use of quality indicators, and guideline-based management (above) helps to minimize potential disparities. However, these approaches may apply less well to the more dynamic processes of making the diagnosis and initiating pharmacotherapy; further studies will be required to explain why A1C is higher in blacks than in whites in the 1- to 2-year period around the times of diagnosis and initiation of diabetes medications.

The strengths of our study include a large number of patients, and availability of longitudinal information from multiple medical centers across three states, permitting us to compare groups of patients who had consistent primary care follow-up before and after the diagnosis of diabetes was made. Limitations include the lack of a national sample, patients who were largely male, lack of information on race in a substantial number of patients, and non-uniform measurement of A1C and glucose levels; the last reflects real-world as opposed to clinical trial conditions. Our findings apply to patients who have consistent follow-up care similar to patients in the present study; whether the consistency of follow-up care differs according to race is beyond the scope of our analysis. While further studies would be required to determine whether the findings would be similar in patients who had less than one primary care visit a year, it could also be argued

that findings in patients with less than one visit a year would not constitute a meaningful measure of primary care practice in any health care setting. It should also be recognized that care in the VA is not necessarily “free,” since some categories of eligibility require a copayment for pharmaceuticals or services received. However, such requirements are stratified according to service connection, income, and insurance coverage, which should minimize the impact of differences in financial status (8).

In conclusion, we found that differences in A1C between black and white veterans were generally small and within the range associated with race, per se, without a difference in underlying glucose levels. However, even in the VA, blacks had higher A1C levels than whites when the diagnosis was made and when drug treatment was initiated, and at these times, the differences in A1C reflected underlying differences in glycemia as well. While understanding the basis for such residual disparities may be important to improving the health of racial/ethnic minorities in the U.S., our overall finding may indicate that racial disparities in health care systems that provide features of structure and organization comparable to those of the VA.

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