# Race Differences in Long-Term Diabetes Management in an HMO

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**OBJECTIVE** — We examined race differences in diabetes outcomes over 4–8 years in a single HMO.

**RESEARCH DESIGN AND METHODS** — We identified black and white adult diabetic patients who were continuously enrolled (1992–2001) and in whom diabetes was *1*) diagnosed before 1994 ($n = 1,686$) or *2*) newly diagnosed in 1994–1997 ($n = 1,280$). We used hierarchical models to estimate the effect of race on average annual $HbA_{1c}$ (A1C) controlling for baseline A1C, BMI, and age, as well as annual measures of type of diabetes medications, diabetes-related hospitalization, time and the number of A1C tests, physician visits, and nondiabetes medications. Stratifying by sex accounted for significant interactions between sex and race.

**RESULTS** — At baseline, black and white patients had similar rates of A1C testing and physician visits, but blacks had higher unadjusted A1C values. In multivariate models, among patients with previously diagnosed diabetes, average A1C was nonsignificantly 0.11 higher (95% CI −0.12 to 0.34) in black than in white men but was 0.30 higher (0.14–0.46; $P = 0.0007$) in black than in white women. Among patients with newly diagnosed diabetes, the adjusted black-white gap was 0.49 among men (0.17–0.80; $P = 0.007$) and was 0.05 among women (−0.20 to −0.31), which was positive but not significant.

**CONCLUSIONS** — Factors other than the quality of care may explain persistent race differences in A1C in this setting. Future interventions should target normalization of A1C by identifying potential psychosocial barriers to therapy intensification among patients and clinicians and development of culturally appropriate interventions to aid patients in successful self-management.

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Diabetes is a focal point in efforts to reduce health disparities in the U.S. (1–5), where an estimated 8.5% of racial differences in mortality are attributable to diabetes alone (6). A high prevalence of poor glycemic control and outcomes among black diabetic patients (7–14) may be attributable in part to inequalities in insurance coverage (15,16) and lower quality of care within clinics serving predominantly black patients (17–19). Yet, racial differences in care have also been demonstrated within coordinated care systems, such as the Veterans Administration health system (20–23).

HMOs provide a system-level focus on standardization of care that may limit decision making based on nonclinical patient characteristics when these decisions conflict with quality-of-care guidelines (24–31). Evidence of racial differences in outcomes within these systems (10,28,32) suggests that the primary causes of racial disparities may transcend the health care setting (e.g., genetics, culture, home environment, and/or psychosocial barriers to medication and lifestyle adherence). Few studies have examined racial differences in diabetes outcomes among HMO patients using a longitudinal design (30,32).

In a setting in which patients have equal access to care, we examined whether racial differences in A1C existed and whether they diminished over time. We hypothesized that any observed racial differences in average A1C in patients with diabetes at cohort inception would diminish over time due to increasing uniformity of the quality of care in this setting. As a consequence of improved quality of care in the later years of our study, we further hypothesized that we would find few or no racial differences among patients with newly diagnosed diabetes.

## RESEARCH DESIGN AND METHODS

— Patients in this study were served by Harvard Vanguard Medical Associates (HVMA), a multispecialty group practice in Massachusetts, and insured by Harvard Pilgrim Health Care (HPHC), one of the largest HMOs in New England. The automated medical records system at HVMA captures data from all ambulatory encounters (including laboratory and pharmacy services) in a combination of both coded and narrative fields. The automated medical records also contained data on enrollment and race of the member. The reliability of these data systems was previously documented (33–35).

From the overall patient population of 300,000, we identified 21,000 patients who were continuously enrolled (enrollment gaps <45 days) for at least a 24-month period between 1 January 1992 and 31 December 2001 and who had a diabetes diagnosis (one inpatient or two outpatient) and/or had any diabetes-specific medication dispensed (sulfonylureas, insulin, or metformin) during that time. We further restricted our sample to patients 18 years of age at identification who had at least 6 months of subsequent follow-up. Patients with a diagnosis of gestational diabetes or polycystic ovarian syndrome without a corresponding diabetes diagnosis were excluded, yielding a sample size of 13,696.

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**Abbreviations:** HPHC, Harvard Pilgrim Health Care; HVMA, Harvard Vanguard Medical Associates.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

We identified two groups of continuously enrolled groups of black and white patients: *1*) patients with any evidence of diabetes (i.e., diagnosis or drug use) between 1 January 1992 and 31 December 1993 and *2*) patients who had no diabetes diagnosis or diabetes medication use in 1992 or 1993 but in whom diabetes was newly diagnosed according to the abovementioned criteria in any year between 1994 and 1997. Requiring continuous enrollment and restricting our sample to patients who could be identified as black or white race only reduced our final sample sizes for each of the study cohorts to 1,686 patients with known diabetes and 1,280 patients with newly diagnosed diabetes.

The baseline for patients with known diabetes was 1994, and follow-up covered 1995 through 2001. For the newly diagnosed diabetic patient cohort, the baseline year began on 1 January after their initial diagnosis, and each patient contributed 3 years of follow-up. The identification of two distinct patient cohorts allowed us to separate the effect of changes in the quality of care over time from possible changes in the patient population over time. Specifically, racial differences among patients with previously diagnosed diabetes may reflect prior differences in the quality of care or the duration of illness. Patients of both races with newly diagnosed diabetes are more likely to have had similar levels of exposure to high-quality diabetes care.

### Clinical and demographic measures

Using information from outpatient laboratory tests, we calculated the average annual A1C for each patient for each calendar year. Earlier A1C values were adjusted to account for changes in the laboratory method of A1C computation in later years. For clarity in the reporting of A1C results, we express A1C values in absolute units (e.g., 5.00, rather than 5.00% [of total hemoglobin]). Clinical measures to be included as control variables were BMI measured at baseline and annual measures of health services utilization, including type of medication use (none or oral insulin), number of physician visits, and number of A1C tests. Patients using both oral and insulin therapy were classified as insulin users. Using a previously validated method (36), we assessed comorbidity by counting the number of medicines (excluding diabetes-specific medications) taken by each patient using the first eight digits of the American Hospital Formulary Services code. We also included an annual indicator for whether the patient had a diabetes-related hospitalization (ICD-9 = 250.XX) as a control for severity of illness.

Clinician-reported patient race was available from the medical record for 70% of the sample. We compared medical record race data at HVMA with patient self-reports for a subset of the diabetic population. Among black and white patients, there was 96% agreement between self-reported and medical record data on race classification, indicating that our race measure was highly reliable.

Patient age and sex were available from HPHC membership files. Socioeconomic status, another potential predictor of glycemic control (37), was measured using block group census data on the percentage of low income (<$15,000/year) residents and the percentage of residents with a high school education. Patient addresses used in the assignment of census measures were available from membership files.

### Statistical analysis

Our primary goal was to assess racial differences in annual A1C over time among patients with previously and newly diagnosed diabetes. We first assessed baseline differences in demographic and clinical characteristics using *t* tests and $\chi^2$ tests. We used nonparametric tests (Kolmogorov-Smirnov) (38) to assess racial differences in the number of physician visits and the number of A1C tests.

To examine trends in racial differences in average A1C within the sexes, we used hierarchical models (39), which account for correlation between multiple measurements for a single individual over time. We included both individual- and race-specific intercepts and slopes using SAS PROC MIXED (40). Baseline measurements included age, A1C, medication use, and BMI. We also tested the inclusion of two measures of neighborhood socioeconomic status (percentage of residents with income <$15,000 and percentage with a high school education) based on the patient's home address in 1990. Time-varying covariates included number of physician visits, number of A1C tests, total number of unique chemical entities dispensed (excluding diabetes drugs), and an indicator for whether the patient had a diabetes-related hospitalization (i.e., ICD-9: 250) in each year.

All of the above variables, with the exception of the census variables, were included in the models regardless of significance to isolate the independent effect of race. Although the inclusion of the census variables did not affect the magnitude or significance of the race measure, a high level of correlation between these covariates and race led us to leave these variables out of the final model. We assessed the utility of various functional forms (e.g., quadratic terms) for our measures of time and baseline A1C as well as interactions between race and important covariates using the likelihood ratio test (41).

In bivariate analyses, we found significant baseline differences by sex in the percentage of black patients, BMI, age, and number of physician visits in both study cohorts. We evaluated the inclusion of interactions between sex and race in unstratified models using the likelihood ratio statistic. For both patient cohorts, the combined race and sex effect was at or near significance (known diabetes $P < 0.10$; newly diagnosed diabetes $P < 0.05$), although the individual race parameter did not lose significance, indicating that either interactions or stratification was appropriate. For ease of interpretation of the race effect, we present the results of the stratified analysis. All analyses were conducted using SAS version 8.2 (40), and statistical significance was defined as $P < 0.05$. The study was approved by the HPHC Institutional Review Board.

**RESULTS** — Selected baseline characteristics by race, sex, and cohort are presented in Table 1. The average baseline A1C was well >7.0 for all patients with previously diagnosed diabetes and near to or <7.0 for patients with newly diagnosed diabetes. Black patients with previously diagnosed diabetes tended to be younger at baseline, had a higher proportion of insulin use, and had higher A1C levels than white patients regardless of sex.

Results were similar in the newly diagnosed diabetes cohort except that there was no difference in initial therapy between black and white women. Within sex groups, baseline rates of physician visits and A1C laboratory testing did not differ by race either among patients with previously diagnosed or newly diagnosed diabetes. Below we present results of the unadjusted and multivariate analyses for the previously diagnosed and newly diagnosed diabetic cohorts, respectively.

**Table 1—*Baseline racial differences in demographic characteristics, management, and A1C level within sex among continuously enrolled adult diabetic patients by date of diagnosis***

| Baseline demographic characteristics | Previously diagnosed (diabetes in 1992 or 1993); baseline 1994 | | | | Newly diagnosed (no diabetes in 1992–1993); baseline 1st year after diagnosis | | | |
|---|---|---|---|---|---|---|---|---|
| | Men | | Women | | Men | | Women | |
| | Black | White | Black | White | Black | White | Black | White |
| *n* | 159 | 651 | 269 | 607 | 151 | 457 | 180 | 492 |
| Age (%) | | | | | | | | |
| 18–44 | 23.3 | 14.0* | 24.2 | 15.7* | 29.1 | 10.7* | 30.6 | 14.8* |
| 45–64 | 62.3 | 54.2 | 56.1 | 46.0 | 57.6 | 58.0 | 57.2 | 47.8 |
| 65+ | 14.5 | 31.8 | 19.7 | 38.4 | 13.3 | 31.3 | 12.2 | 37.4 |
| Age (years) | 53.3 ± 10.4 | 57.9 ± 11.6* | 53.7 ± 11.8 | 59.3 ± 13.2* | 51.5 ± 11.4 | 58.4 ± 11.3* | 51.4 ± 11.2 | 59.3 ± 13.2* |
| BMI (kg/m$^2$) | 30.5 ± 6.1 | 30.8 ± 5.6 | 33.0 ± 7.2 | 32.2 ± 7.3 | 30.5 ± 6.0 | 31.6 ± 5.8 | 33.8 ± 7.9 | 32.8 ± 6.6 |
| Drug therapy at baseline (%) | | | | | | | | |
| None | 17.0 | 29.3‡ | 27.1 | 28.5 | 44.4 | 58.0‡ | 64.4 | 69.1 |
| Oral medication | 40.3 | 40.6 | 31.2 | 37.7 | 44.4 | 37.4 | 30.6 | 26.6 |
| Any insulin | 42.8 | 30.1 | 41.6 | 33.8 | 11.3 | 4.6 | 5.0 | 4.3 |
| No. physician visits | 4.6 ± 3.2 | 4.9 ± 3.6 | 5.8 ± 4.2 | 5.7 ± 4.4 | 4.4 ± 3.8 | 4.3 ± 3.8 | 5.3 ± 4.4 | 5.2 ± 4.2 |
| No. A1C tests | 1.5 ± 1.2 | 1.6 ± 1.4 | 1.7 ± 1.4 | 1.8 ± 1.5 | 1.4 ± 1.2 | 1.5 ± 1.4 | 1.4 ± 1.3 | 1.4 ± 1.3 |
| A1C value (%) | 8.1 ± 2.0 | 7.6 ± 1.7† | 8.6 ± 2.0 | 8.0 ± 1.7* | 7.2 ± 1.8 | 6.6 ± 1.3‡ | 7.2 ± 1.9 | 6.8 ± 1.3† |

Data are frequencies (%) or means ± SD. *$P < 0.0001$; †$P < 0.01$; ‡$P < 0.001$.

**Previously diagnosed diabetic cohort**

**Trends in A1C.** For both women and men with previously diagnosed diabetes, higher average unadjusted A1C levels persisted over the study period (Fig. 1). The unadjusted A1C gap between black and white men was 0.50 units at baseline and almost 1.0 (8.92 [black] vs. 7.87 [white] units) in 2001. Black women had higher A1C at baseline (difference 0.68 units; 8.63 vs. 7.95 units), and the racial difference remained roughly constant throughout the study period.

**Effect of race on A1C over time.** The results of the hierarchical models are presented in Table 2. For all models, the race effect should be interpreted as the difference between two patients of different race but equal baseline A1C. Among men, black men had average A1C levels that were 0.11 unit higher than those for white men (95% CI −0.11 to 0.34, $P = 0.34$), but the difference was not significant.

Significant predictors of higher average A1C included baseline A1C, insulin or oral medication use, year squared, and higher baseline BMI. Older age (age ≥65 years), a higher number of A1C tests, year, and the total number of other medications dispensed per year were significant predictors of lower average A1C. The average rate of increase in A1C over time was the same for black and white men.

Among women with previously diagnosed diabetes, blacks had a 0.30 higher adjusted average A1C than whites (95% CI 0.14–0.46, $P = 0.0002$). Other significant predictors of higher average A1C included baseline A1C, younger age, use of oral hypoglycemic agents or insulin, and year squared. Higher comorbidity, diabetes-related hospitalization, and year were significant predictors of lower average A1C. As for men with previously diagnosed diabetes, the rate of increase in A1C did not differ by race.

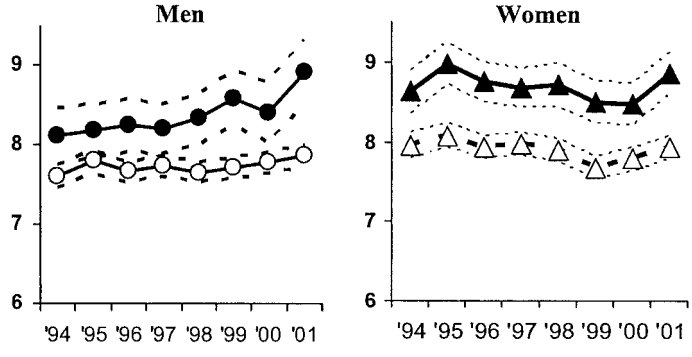


**Figure 1—*Racial differences in observed average A1C (1994–2001) with 95% CIs among continuously enrolled diabetic patients with evidence of diabetes before 1993. ●, black men; ○, white men; ▲, black women; △, white women. - - -, 95% CI. x-axis = years since diagnosis.***

**Newly diagnosed diabetic cohort**

**Trends in A1C.** Figure 2 shows the average unadjusted A1C over the first 4 years after diagnosis for new patients. The racial gap among men with newly diagnosed diabetes was 0.67 units (7.25 [black] vs. 6.58 [white] units) at baseline and 0.95 units (8.27 vs. 7.32 units) 4 years after diagnosis. Among women with newly diagnosed diabetes, the black-white gap in unadjusted A1C levels was 0.46 units (7.22 vs. 6.76 units) at baseline and 0.50 units (7.66 vs. 7.16 units) 4 years after diagnosis.

**Effect of race on A1C over time.** Among men with newly diagnosed diabetes, after covariate adjustment black race was associated with a significant 0.49 greater average A1C (95% CI 0.18–0.81, $P = 0.002$) (Table 2). Other significant predictors of higher A1C included baseline A1C, diabetes medication use, and year. The rate of increase in A1C was similar for blacks and whites.

Among women with newly diagnosed diabetes, although the direction of the ef-

**Table 2—*Estimated effect of black race on average annual A1C, hierarchical mixed model***

| | Previously diagnosed diabetes | | Newly diagnosed diabetes | |
|---|---|---|---|---|
| | Men | Women | Men | Women |
| Intercept | 3.31 ± 0.30 (2.71–3.91) | 3.76 ± 0.26 (3.24–4.27) | 2.34 ± 0.51 (1.34–3.34) | 2.35 ± 0.44 (1.49–3.21) |
| A1C* | 0.52 ± 0.02 (0.47–0.57) | 0.56 ± 0.02 (0.52–0.61) | 0.57 ± 0.04 (0.48–0.65) | 0.63 ± 0.04 (0.55–0.70) |
| Black race | 0.11 ± 0.12 (−0.12 to 0.34) | 0.30 ± 0.08 (0.14–0.46) | 0.49 ± 0.16 (0.17–0.80) | 0.05 ± 0.13 (−0.20 to 0.31) |
| Age <45† | — | — | — | — |
| Age 64* | −0.14 ± 0.12 (−0.38 to 0.09) | −0.40 ± 0.10 (−0.61 to −0.20 | −0.004 ± 0.19 (−0.37 to 0.36) | 0.06 ± 0.15 (−0.23 to 0.36) |
| Age 65+* | −0.40 ± 0.13 (−0.66 to −0.14) | −0.67 ± 0.11 (−0.89 to −0.45) | −0.22 ± 0.21 (−0.64 to 0.21) | −0.07 ± 0.18 (−0.42 to 0.28) |
| No treatment† | — | — | — | — |
| Oral* | 0.50 ± 0.08 (0.34–0.66) | 0.58 ± 0.08 (0.43–0.73) | 0.44 ± 0.10 (0.24–0.63) | 0.63 ± 0.11 (0.42–0.84) |
| Insulin* | 0.55 ± 0.09 (0.37–0.74) | 0.54 ± 0.09 (0.36–0.71) | 0.64 ± 0.22 (0.21–1.1) | 0.42 ± 0.20 (0.03–0.81) |
| BMI* | 0.02 ± 0.01 (0.003–0.03) | 0.005 ± 0.005 (−0.005 to 0.01) | 0.007 ± 0.01 (−0.01 to 0.03) | 0.01 ± 0.03 (−0.08 to 0.03) |
| No. physician visits | 0.004 ± 0.01 (−0.01 to 0.02) | 0.01 ± 0.005 (−0.001 to 0.02) | 0.02 ± 0.01 (0.01–0.04) | −0.001 ± 0.01 (−0.02 to 0.02) |
| No. A1C tests | −0.04 ± 0.02 (−0.01 to 0.006) | −0.002 ± 0.01 (−0.03 to 0.03) | −0.02 ± 0.03 (−0.09 to 0.04) | −0.01 ± 0.03 (−0.08 to 0.05) |
| Comorbidity‡ | −0.02 ± 0.01 (−0.04 to −0.01) | −0.02 ± 0.006 (−0.03 to −0.01) | −0.04 ± 0.01 (−0.07 to −0.02) | −0.004 ± 0.01 (−0.03 to 0.02) |
| Diabetes hospitalization | −0.04 ± 0.06 (−0.15 to 0.07) | −0.15 ± 0.05 (−0.25 to −0.04) | 0.11 ± 0.13 (−0.13 to 0.36) | −0.28 ± 0.13 (−0.53 to −0.03) |
| Year | −0.14 ± 0.04 (−0.21 to −0.07) | −0.24 ± 0.03 (−0.31 to −0.17) | 0.45 ± 0.20 (0.05–0.85) | −0.22 ± 0.23 (−0.67 to 0.23) |
| Year squared | 0.02 ± 0.004 (0.01–0.03) | 0.03 ± 0.004 (0.02–0.04) | −0.05 ± 0.05 (−0.15 to 0.05) | 0.07 ± 0.06 (−0.04 to 0.18) |

Data are coefficients ± SE (95% CI). *Measured at baseline. †Reference groups. ‡Number of non–diabetes-related medications.

fect remained positive, there were no significant differences in A1C by race (difference 0.05 units; 95% CI −0.20 to 0.31, $P = 0.69$). Other significant variables included A1C, type of drug therapy, and diabetes-related hospitalization. Time was not a significant predictor of A1C in this group.

**CONCLUSIONS** — In our longitudinal analysis of diabetes management in an HMO, we found consistently higher A1C levels in blacks than in whites. Although these differences were diminished after controlling for baseline A1C values for two of our groups, the race effect remained positive and significant for women with known diabetes and for men with newly diagnosed diabetes. Contrary to our initial hypothesis, racial differences in diabetes control did not diminish over time.

In accordance with previous studies, we found similar rates of annual physician visits and annual A1C testing by race, indicating that access and quality are not the primary drivers of differences in glycemic control. However, our findings of a significant effect of race after multivariate adjustment were in contrast to findings from Gary et al. (30), who reported that racial differences in diabetes control (A1C <8.0) declined over time and were not significant after adjustment for age and sex. Although the inclusion of baseline A1C diminished the effect of race in two of our groups, controlling for it and a number of other factors did not explain the race effect among women with known diabetes and men with newly diagnosed diabetes.

In a recent longitudinal study of race differences in diabetes-related complications, Karter et al. (32) postulated that persistent race differences may be explained by genetic or environmental factors rather than by differences in the quality of care. The findings of this study lend additional support to that hypothesis. In particular, the combined race and sex effect found in our study may reflect differences in the availability of social support and its relationship to self-management (42,43). Other researchers have found evidence of a combined race and sex effect in quality and outcomes (7,10,28,44,45).

On the other hand, our findings do not rule out the possibility of differential clinician response to elevated A1C by race and sex. Unlike initiation of therapy, intensification of therapy is difficult to reg-

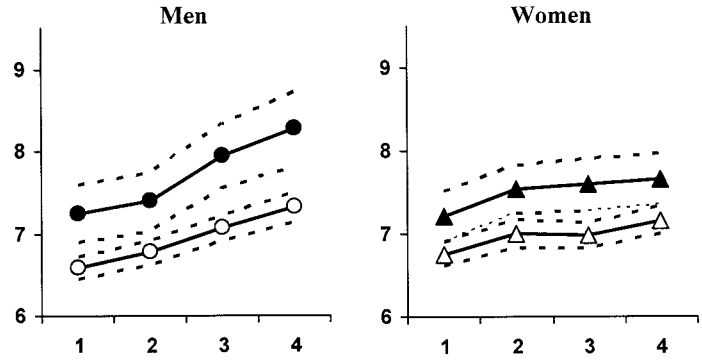


**Figure 2—***Racial differences in observed average A1C (1994–2001) with 95% CIs among continuously enrolled diabetic patients with no evidence of diabetes diagnosis before 1994. ●, black men; ○, white men; ▲, black women; △, white women. - - -, 95% CI.* x*-axis = years since diagnosis.*

ulate at the system level. Both patient concerns about moving to insulin therapy and clinician inertia may be influenced by the patient's race and sex.

The examination of multiple years of laboratory A1C values in a fully insured, HMO population sets this study apart from previous analyses of racial differences in diabetes control. However, there are limitations to this analysis that merit discussion. First, race data were unavailable for 30% of patients, and there may be racial differences in the recording of these data. Also, a number of socioeconomic factors that may be correlated with race were not controlled for in this analysis (2,37). Lastly, regression models did not control for nonmedical management of diabetes, medication adherence, intensity of drug therapy, out-of-pocket expenses, or duration of illness. It is possible that additional control for confounding would have further narrowed the black-white gap in diabetes control.

Our findings of persistent white-black differences in A1C among insured patients deserve further investigation. Future research should target the design of interventions to normalize A1C among black patients by identifying potential psychosocial barriers to therapy intensification among patients and clinicians and developing culturally appropriate interventions to aid patients in successful self-management. By focusing primarily on improving quality-of-care indicators (e.g., A1C testing), providers may miss significant opportunities to improve disparities in outcomes.

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