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Medication Adherence and Racial Differences in HbA1c Control

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Abstract

Objective—The purpose of this study was to examine medication adherence and other self-management practices as potential determinants of higher glycemic risk among black relative to white patients.

Research Design and Methods—We used a retrospective, longitudinal repeated measures design to model the contribution of medication adherence to black-white differences in HbA1c among Type 2 diabetes patients at a large multi-specialty group practice. We identified 1,806 adult (age 18+ at diagnosis) patients (black=467, white=1339) newly initiated on oral hypoglycemic therapy between 01/01/94 and 12/31/00. Race was identified using an electronic medical record and patient self-reports. Baseline was defined as the 13-months preceding and including the month of therapy initiation. All patients were required to have at least 12 months of follow up.

Results—At initiation of therapy, black patients had higher average hemoglobin A_{1c} values compared to whites 9.8 vs. 8.9, a difference of 0.88 ($p<0.0001$). Blacks had lower average medication adherence during the first year on therapy [72% vs. 78%; $p<0.0001$]. While more frequent medication refills were associated with lower average HbA1c values, adjustment for adherence did not eliminate the black-white gap.

Conclusions—We found persistent racial differences in hemoglobin A1c that were not explained by differences in medication adherence. Our findings suggest that targeting medication adherence alone is unlikely to reduce disparities in glycemic control in this setting. Further research is needed to explore possible genetic and environmental determinants of higher A1c among blacks at diagnosis, which may represent a critical period for more intensive intervention.

Diabetes mellitus is a highly prevalent and costly condition.(1) Adverse health events associated with diabetes include microvascular and macrovascular events. However, the risk of these and other complications of diabetes can be reduced through effective management including the use of efficacious prescription drugs.(2)

Diabetes is also a leading contributor to racial and ethnic disparities in health outcomes in the United States.(3) Poorer glycemic control among blacks may be a key driver of these disparities.(4) Explanations proposed for racial differences in glycemic control include lower quality of care within clinics serving predominantly black communities.(5) However, improving access and overall quality of care may not reduce disparities in outcomes.(6,7)

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Racial differences in medication adherence and other self-management practices (e.g., self-monitoring of blood glucose) have been identified in the literature.(8-10) A better understanding of how medication adherence and other modifiable factors influence disparities in glycemic risk is needed to design appropriate interventions.(11) To date, few studies have directly modeled the relationship between medication adherence and racial differences in hemoglobin A_{1c} values among insured populations with equal access to care.(12-14)

The primary objective of this study was to model the relationship between medication adherence and other modifiable behaviors and hemoglobin A1c over time for newly treated black and white Type 2 diabetes patients in a multi-specialty group practice. We then compared the relative contributions of specific factors (e.g., refill adherence) to the black-white gap in hemoglobin A_{1c} post adjustment. We hypothesized that racial differences in self-management practices would explain disparities in glycemic control previously identified in this insured population,(15) treated in a setting where variations in quality of care have been minimized.(6)

RESEARCH DESIGN AND METHODS

Setting and Study Population

The setting for this study was Harvard Vanguard Medical Associates, a multi-specialty group practice in Massachusetts with 14 clinic sites. All patients were insured by Harvard Pilgrim Health Care. The reliability of the automated medical records system at Harvard Vanguard Medical Associates, which captures data from all ambulatory encounters, has been previously documented.(16) This data source includes all ambulatory and inpatient encounters (e.g., lab tests, lab results, prescribing information, pharmacy contacts) in a combination of coded and narrative fields.

This analysis focused on patients newly treated on oral medication therapy following their first observed diabetes diagnosis. Restricting our cohort to newly diagnosed and treated patients ensured a more homogenous group of subjects in the initial phase of pharmacological management of hyperglycemia. Using a combination of electronic medical records and claims generated between January 1992 through December 2001, we identified more than 16,000 patients who had diabetes, defined as 1 inpatient or 2 outpatient diagnoses, at least one dispensing of a diabetes-specific medication (e.g., sulfonylureas), and/or at least one dispensing of test strips for home glucometers. We excluded patients with polycystic ovarian syndrome and no diabetes or who had gestational diabetes. We further restricted our sample to adult ($\geq$ 18 years) black and white patients (n=9,999). Our analyses were restricted to black and white patients due to small sample sizes and inability to reliably identify other racial and ethnic groups

Patients were considered to have a new diagnosis if they had no evidence of diabetes in the previous 12 months of continuous enrollment. We further restricted the cohort to individuals whose first recorded prescription for oral hypoglycemic therapy occurred at the time of or after their initial diagnosis and within 30 days of their first dispensing of this medication, and who were continuously enrolled for at least 12 months following their first dispensing (n=2,099). Because we did not have reliable prescribing data on daily dosing for insulin users, we excluded patients who had any insulin use during the study period. The analytic cohort included 1806 patients: 467 black and 1339 white patients.

Measures

Glycemic control—Using information from outpatient laboratory records, we calculated the average hemoglobin A_{1c} for each month in which a patient was tested. Hemoglobin A_{1c} values prior to September 1998 were adjusted to account for changes in the laboratory method of

computation in later years. Details on this procedure are described elsewhere.(15) We also calculated the average hemoglobin A_{1c} over the one year period prior to initiation of therapy to assess baseline severity of illness.

Race and demographic measures—We obtained data on patient race from the electronic medical record, based on clinician reports. These data were validated and supplemented for a subset of patients with patient self-reports, obtained from a written questionnaire administered to currently enrolled diabetes patients by the practice group. In the questionnaire, patients were asked to select the racial group that best represented their race; most patients selected only one race.

Patient age at diagnosis was calculated based on the date of birth and month of diagnosis. Information on patient gender and addresses were available from Harvard Pilgrim Health Care membership files. Socioeconomic status was not consistently recorded in the electronic medical record. Instead, we derived indicators of socioeconomic status, based on 1990 US Census block group data, including the median household income, the percent of residents without a high school education, and the percent who did not understand spoken English.

Medication adherence—We assessed refill-based medication adherence using both prescribing and dispensing data. Standard refill-based medication adherence measures assume that days supply is equivalent to daily dose and, therefore, cannot distinguish between physician initiated changes in therapy and patients noncompliance.(17) Our measure used prescribing notes to determine intended daily dose. In addition, patients had a strong financial incentive (i.e., smaller copay) to fill prescriptions within the health care system under study. Median co-payment levels for the most commonly used diabetes drugs were similar for blacks and whites during the study period (e.g., glyburide: median copay for blacks=$10 (min $0.01, max $60); median copay for whites=$10 (min $0.01; max $73).

Pharmacy records were used to calculate the amount dispensed, which was allocated in daily amounts according to the most recent prescription until the supply was exhausted (or over 60 days following the dispensing date if no subsequent dispensing occurred within that period). For each oral diabetes medication, a time-varying adherence measure was calculated as the milligrams dispensed divided by the amount prescribed per month to obtain a percentage of the prescribed amount that was available for use. For patients taking more than one oral medication, we calculated the combined average adherence per month. For the multivariate analysis, we calculated the average adherence (times 100) during the 3-month period prior to each laboratory hemoglobin A_{1c} test result.

Medication adherence for patients who discontinued therapy for more than 60 days was set to missing to limit the influence of these patients on the adherence measure. Because adherence could not be reliably established for patients who were hospitalized during the three months prior to an HbA1c test, we ran the models including and then excluding test months that included a hospitalization. The results were highly consistent across models, which was likely due to low rates of hospitalization among newly treated patients in this setting. The final model included all months, including those with hospitalization episodes.

Self-monitoring of blood glucose or glucose self-monitoring—Self-monitoring activity was measured as the average number of blood glucose test strips dispensed per month. As described in our previous work,(8) dispensed test strips were distributed evenly over the days between dispensings (or over 60 days following the dispensing date if no subsequent dispensing occurred within that period). The covariate included in the models was the average number of test strips dispensed per month during the 3-month period prior to each laboratory assessed hemoglobin A_{1c} value.

Medication type and intensification—We created dichotomous indicators of first prescribed therapy with glyburide as the reference group. Possible initial treatment included metformin, glipizide, other oral medications, or multiple oral medications.

For the unadjusted analysis, we defined therapy intensification as any evidence of an increase in dose or addition of a second oral agent during the study period. To model the relationship between therapy intensification and HbA1c, we created a time-varying measure of intensification, defined as having an increase in dosing or augmentation with another oral hypoglycemic medication following a laboratory assessed HbA1c test during the last six months. We excluded the current test month from this calculation to ensure that the intensification preceded the outcome of interest.

Clinical measures and health services use—Clinical covariates measured at baseline included hemoglobin A_{1c} and body mass index (underweight-normal=<25 kg/m^2; overweight=25-29.9 kg/m^2; obese>29.9 kg/m^2). We also included several time-varying measures of health status. Using a previously validated method,(18) we assessed comorbidity by counting the total number of non-diabetic medicines taken in the three months prior to an hemoglobin A_{1c} test, using the first eight digits of the American Hospital Formulary Services code. Because of the high prevalence of both hypertension and hypercholesterolemia among diabetes patients, we further adjusted for any evidence of elevated systolic blood pressure (systolic blood pressure ≥ 130 mmHg) and total cholesterol (5.18 mmol/L or ≥ 200mg/dL) during each year of follow-up.(2) Missing lab values for these measures were imputed as described below.

The number of physician visits during the 3-month period prior to each hemoglobin A_{1c} test were also assessed. To control for possible differences in patterns of care, we also created indicators for whether patients were known to have received at least 50% of their care in one of two settings with a higher proportion of black patients.

Statistical analysis—We assessed baseline (13 months prior to and including the month the patient initiated therapy) differences in demographic and clinical characteristics using t-tests and chi-square tests. We used nonparametric tests (Kolmogorov-Smirnov) (19) to assess racial differences in the number of physician visits and the number of hemoglobin A_{1c} tests. Median months of follow-up were similar for black (med: 51 (min:13, max: 108) and white patients (med: 52 (min: 13, max: 108)).

Our goal for the longitudinal analysis was to examine the relationship between various baseline and time varying factors and hemoglobin A_{1c} values over time by race. All modeling, including multiple imputation methods, was performed using SAS statistical software, Version 9.1.3 (SAS Institute Inc., Cary, North Carolina). We employed multilevel longitudinal (Proc Mixed) models, with random intercepts and slopes, and an unstructured covariance structure, to account for correlation within individuals over time.(20) We stratified these models by race to assess the relative importance of the specified covariates on hemoglobin A_{1c} within each racial group. We also ran a combined model that included both races in order to assess whether the inclusion of medication adherence attenuated the black-white gap.

To account for missing or unrecorded values (i.e., baseline body mass index, baseline hemoglobin A_{1c}, time-varying systolic blood pressure, time-varying total cholesterol, or baseline census-derived measures of socioeconomic status), we used a multiple-imputation method (Proc MI) to replace missing values with plausible values drawn from a conditional probability distribution that was a function of the observed values (Markov chain monte carlo method). We conducted 20 imputations, resulting in 20 estimates, which were then combined to obtain a single set of estimated coefficients with corresponding confidence intervals using

Proc MI and Proc MIAnalyze.(21) We imputed values for covariates, but not the primary predictors or the outcome of interest (hemoglobin A_{1c}).

Models that allowed for clustering of patients by health center site did not appear to fit the data better than models without this parameter as indicated by a modified X^2 statistic.(22) We also tested for correlations between the outcomes and medication adherence and self-monitoring behaviors during the previous three to six months, but found no evidence to justify the inclusion of lags in these variables of longer than three months.

The study was approved by the Harvard Pilgrim Health Care Institutional Review Board. The funders had no involvement in the design and conduct of the study; collection, management, analysis, and interpretation of the data; or preparation, review, or approval of the manuscript.

RESULTS

Baseline differences by race

A total of 1806 newly-diagnosed, newly drug-treated patients (26% black, 74% white) met the study criteria. Black patients in our sample were more likely to be female (52% vs. 41%) and under age 45 (29% vs. 14%). In the 13-month baseline period (including the first month of therapy), black patients had higher average hemoglobin A_{1c} (9.8 vs. 8.9; $p<0.001$); a differences of 0.88. Black and white patients had similar body mass index, lab tests, cholesterol levels and hospitalizations. The percentage of missing values was similar in black and white patients (Table 1).

Racial differences in medication adherence, self-monitoring and therapy intensification over time

During the first six and twelve months after initiation of oral therapy, black patients had lower average medication adherence compared to whites (Table 2). However, there were no significant differences in either self-monitoring behavior or therapeutic intensification by race.

Correlates of hemoglobin A_{1c} among black and white patients

Table 3 presents results of the stratified mixed models of the relationship between A1c, self-management practices and other factors after multiple imputation of covariates. More frequent medication refills and test strip refills were associated with lower average hemoglobin A_{1c} values among white and black patients. An increase in adherence of 25 percentage points (e.g., 50% vs. 85% of days covered during the month) was associated with a 0.05% lower HbA1c value among blacks (e.g., 8.55% vs. 8.50%) and 0.07% lower HbA1c among whites. More frequent physician visits were also associated with lower average hemoglobin A_{1c}.

Among whites, other significant predictors of higher A1c included greater comorbidity, younger age, and time since initiation of therapy. Among white patients, higher baseline hemoglobin A_{1c}, systolic blood pressure and female gender were associated with higher average hemoglobin A_{1c}, but these were not statistically significant among blacks. Among black patients, initiating on more than one oral therapy was also associated with lower hemoglobin A_{1c} values. Receiving care at clinics serving a disproportionate number of black patients and neighborhood SES were not statistically significantly associated with hemoglobin A_{1c} levels among black or white patients. [Data not shown]

Association between medication adherence and the black-white gap in HbA1c

The results of the combined model are presented in the last column of Table 3. The combined models provided an assessment of the degree of attenuation of the black-white gap after controlling for medication adherence. Controlling only for time since initiation of therapy, the

estimated black white gap was 0.80 ($p<0.0001$).[Data not shown] After controlling for all other covariates except medication adherence, this difference was attenuated to 0.48 ($p<0.0001$). [Data not shown] The additional of medication adherence to the models resulted in an additional attenuation of the black-white gap to 0.46% ($p<0.0001$) as indicated by the coefficient on black race in Table 3, col 4, row 3. An interaction term between race and medication adherence was not statistically significant at the 0.05 level, indicating insufficient evidence of a difference in the effect of adherence by race.

CONCLUSIONS

Evidence from well-insured, managed care populations suggest that racial differences in glycemic control cannot be fully explained by variations in the site or quality of care.(6,23) The purpose of this study was to explore medication adherence and other self-care determinants of glycemic control among black and white diabetes patients with equal access to care. After adjustment for potential confounders, we found a persistent black-white gap in hemoglobin A_{1c} levels over time, even among patients with high rates of refill adherence.

One explanation for these findings is that black patients have more severe diabetes at the time they initiate therapy and may require more intensive intervention. Unmeasured biological, cultural or environmental determinants may explain greater severity of illness among black patients.(23) Still, we were surprised at the modest association of medication refill adherence with hemoglobin A_{1c}. A possible explanation is that newly treated patients, even when adherent to prescribed therapy, are not receiving medication dosage sufficient to achieve maximum therapeutic benefit.(24,25) In addition, our claims-based measure of medication adherence may underestimate the association between adherence and HbA1c because it overestimates actual adherence among patients who pick up prescribed medicines, but do not take them as directed. (17)

Our estimate of a persistent racial gap in hemoglobin A_{1c} is consistent with findings of Schechtman et al who found evidence of a racial gap in A1c after controlling for medication adherence to oral medications.(13) Our findings differ from those of Pladevall et al,(12) who found no evidence of racial differences in glycemic control after controlling for adherence to metformin. Interestingly, a recent study of managed care patients with asthma also found that differences in controller medication adherence did not explain racial differences in asthma control.(26) Our finding that patients who self-monitor had lower HbA1c values, while not supported by clinical trial evidence, is consistent with a recent review of self-monitoring among patients with Type 2 diabetes.(27)

Unlike previous studies,(5) our findings of a persistent racial gap in hemoglobin A_{1c} did not appear to be driven by poorer quality of care clinics serving a disproportionate number of black patients. This difference may be due to the homogeneity of insurance benefits across individuals and clinics in this study.

Because of the retrospective cohort design, we cannot interpret the observed associations as causal relationships. However, unlike previous studies,(12,13) we assessed multiple behaviors during the time prior to each test, allowing us to more effectively estimate the temporal relationship between recent patterns of self-management behavior and hemoglobin A_{1c}. Because this was an observational cohort receiving usual care, we could only assess hemoglobin A_{1c} values when patients had a laboratory test. It is possible that the frequency of laboratory testing may have been related to hemoglobin A_{1c} value; patients with higher average hemoglobin A_{1c} levels may have been more or less likely to be tested. However, rates of hemoglobin A_{1c} testing did not vary by race in this population, so frequency of laboratory tests is an unlikely explanation for differences in hemoglobin A_{1c}.

Claims-based adherence measures can overestimate actual adherence among patients who pick up medicines, but who do not use them as directed.(17) There may be racial differences in factors like timing of medication administration, wastage or sharing of medicines, that could result in differential accuracy of these claims-based adherence measures by race. In general, claims-based measures have been shown to be highly sensitive measures of medication adherence in relation to other objective measures, and they are more practical for studies of real-world adherence behavior.(17)

Our analyses were restricted to black and white patients due to small sample sizes for other racial and ethnic groups. We could not control for several patient-level factors including potential genetic factors,(28) environmental influences, patient level barriers (e.g., health literacy) or complementary treatments (e.g., diet, exercise). Further, we could not measure important psychosocial factors that may correlated with medication adherence and rates of self-monitoring (e.g., readiness to change). We also could not measure individual level socioeconomic status and used block-level census measures as a proxy. In some cases, neighborhood socioeconomic status may capture important neighborhood effects not captured by individual measures.(29) Lastly, these results come from a single large, multi-specialty group practice and may not represent the experiences of diabetes patients in different geographic regions or systems of care with greater financial barriers to adherence.

Medication adherence is a key component of self-management for patients with diabetes, and our evidence supports the development of interventions to improve long-term medication adherence and intensification of therapy (24,25) among black and white patients. Specifically, increased medication adherence was associated with clinically significant reductions in HbA1c for both black and white patients, but was associated with only a modest reduction in the black-white gap in glycemic control. However, our findings suggest that improving medication adherence alone, is unlikely to reduce the black-white gap in glycemic control in this setting. Our findings of racial differences in glycemic control at the time of diagnosis are consistent with possible genetic or environmental drivers.(23) Further research is needed to explore these factors across settings and conditions for which disparities in outcomes have been previously identified. Also, confirmation of these findings from studies using actual observed adherence would lend additional credibility to our results.

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Table 1

Baseline characteristics among Type 2 diabetes patients by race*

	Black N=467 (26%)	White N=1339 (74%)
Female (%)	**52%**	**41%**
Age at Time of Diagnosis (%) <45 45-64 >65	**29%** **62%** **9%**	**14%** **54%** **32%**
Mean hemoglobin A_{1c} level (SD), %	***% Missing: 14%*** **9.8 (2.4)**	***% Missing: 13%*** **8.9 (2.1)**
Body Mass Index (%), kg/m^2 Underweight to Normal Overweight Obese	*% Missing: 28%* 11% 31% 58%	*% Missing: 29%* 9% 29% 62%
Any Hospitalization (%)	9%	12%
Mean Systolic Blood Pressure > 130 mmHg (%)	***% Missing: 1%*** **61%**	***% Missing: 2%*** **67%**
Mean Total Cholesterol > 5.18 mmol/L or >200 mg/dL (%)	*% Missing: 34%* 74%	*% Missing: 31%* 72%
Medication Use at Initiation of Therapy		
Glyburide	**83%**	**78%**
Metformin HCL	**13%**	**14%**
Glipizide	**3%**	**6%**
All Other (acarbose, rosiglitazone, tolazamide, tolbutamide)	**1%**	**2%**
>1 Oral Hypoglycemic Medication	**3%**	**2%**

* Bold=statistically significant at the p<0.05 level.

Table 2

Patterns of Adherence at 6 and 12 months post initiation of oral hypoglycemic therapy

	First 12 Months Following Therapy Initiation			
	6 Months		12 Months	
	Black	White	Black	White
Medication Adherence	**72.7%**	**78.3%**	**71.7%**	**77.6%**
SMBG	20.3%	20.7%	15.5%	17.1%
Increase in Dose or Augmentation	28.6%	27.2%	38.2%	37.8%

* Bold= statistically significant at the p<0.0001 level.

Table 3

Adjusted HbA1c values among newly treated diabetes patients*

	Black Patients (N=467)	White Patients (N=1339)	Combined Model (n=1806)
Time (months)	0.02 (0.02, 0.03)	0.02 (0.02, 0.02)	0.02 (0.02, 0.02)
Black race	--	--	0.46 (0.28, 0.63)
Age at diagnosis	-0.03 (-0.05, -0.02)	-0.02 (-0.03, -0.02)	-0.02 (-0.03, -0.02)
Male	-0.09 (-0.41, 0.23)	-0.20 (-0.35, -0.05)	-0.16 (-0.30, -0.03)
Comorbidity	0.04 (0.01, 0.07)	0.01 (-0.004, 0.02)	0.02 (0.003, 0.03)
SBP>130mmHg †‡	-0.12 (-0.29, 0.04)	0.13 (0.06, 0.20)	0.08 (0.01, 0.14)
Total Chol.>200mg/dL‡	0.01 (-0.15, 0.17)	0.06 (-0.01, 0.13)	0.05 (-0.01, 0.12)
Baseline BMI (kg/m^2) †‡	-0.003 (-0.02, 0.02)	0.003 (-0.01, 0.01)	0.001 (-0.01, 0.01)
Baseline HbA_{1c} level‡	0.05 (-0.01, 0.11)	0.06 (0.03, 0.09)	0.06 (0.02, 0.08)
Baseline Medication (ref=glyburide) ‡			
Metformin	-0.45 (-0.96, 0.06)	-0.12 (-0.34, 0.10)	-0.21 (-0.41, 0.003)
Glipizide	0.63 (-0.26, 1.52)	0.15 (-0.14, 0.45)	0.23 (-0.06, 0.53)
Multiple	-1.25 (-2.28, -0.23)	-0.01 (-0.56, 0.55)	-0.37 (-0.86, 0.12)
Other	-0.54 (-3.52, 2.43)	0.29 (-0.24, 0.82)	0.22 (-0.33, 0.77)
# of physician visits‡	-0.22 (-0.36, -0.08)	-0.14 (-0.20,-0.08)	-0.16 (-0.22, -0.11)
Test strip use‡	-0.01 (-0.01, -0.01)	-0.005 (-0.01, -0.004)	-0.01 (-0.01, -0.005)
Therapy intensification‡	-0.06 (-0.26, 0.14)	0.002 (-0.08, 0.09)	-0.02 (-0.10, 0.07)
Medication adherence‡	-0.002 (-0.004, -0.001)	-0.003 (-0.004, -0.002)	-0.003 (-0.004, -0.002)

Baseline includes the 12 months before and including the therapy initiation month; Physician visits, HbA1c tests, test strip use, and medication adherence are measured 3 months prior to each HbA1c test; Therapy intensification indicates augmentation or increase in dose for oral diabetes medications following a lab assessed HbA1c test during the last six months.

* Care site and neighborhood SES were included, but not significant at the 0.05 level.

† SBP=systolic blood pressure; BMI=body mass index

‡ SBP and total cholesterol were measured annually