Medication Adherence and Racial Differences in A1C Control

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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 A1C among type 2 diabetic patients at a large multispecialty group practice. We identified 1,806 adult (aged ≥18 at diagnosis) patients (467 black and 1,339 white) with newly initiated oral hypoglycemic therapy between 1 December 1994 and 31 December 2000. Race was identified using an electronic medical record and patient self-report. Baseline was defined as the 13 months preceding and included 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 A1C values compared with whites (9.8 vs. 8.9, a difference of 0.88; $P < 0.0001$). Blacks had lower average medication adherence during the first year of therapy (72 vs. 78%; $P < 0.0001$). Although more frequent medication refills were associated with lower average A1C values, adjustment for adherence did not eliminate the black-white gap.

CONCLUSIONS — We found persistent racial differences in 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.

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Diabetes 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 U.S. (3). Poorer glycemic control among blacks may be a key driver of these disparities (4). One explanation proposed for racial differences in glycemic control is 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). 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 A1C 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 A1C over time for newly treated black and white type 2 diabetic patients in a multispecialty group practice. We then compared the relative contributions of specific factors (e.g., refill adherence) to the black-white gap in A1C after 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 in which variations in quality of care have been minimized (6).

RESEARCH DESIGN AND METHODS

The setting for this study was Harvard Vanguard Medical Associates, a multispecialty 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 documented previously (16). This data source includes all ambulatory and inpatient encounters (e.g., laboratory tests, laboratory test results, prescribing information, and pharmacy contacts) in a combination of coded and narrative fields.

This analysis focused on patients newly treated with oral medication therapy after their first observed diabetes diagnosis. Restricting our cohort to newly diagnosed and treated patients ensured a more homogeneous 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 and December 2001, we identified >16,000 patients who had diabetes, defined as one inpatient or two 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 ovary syn-

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drome and no diabetes and those who had gestational diabetes. We further restricted our sample to adult (aged ≥18 years) black and white patients (n = 9,999). Our analyses were restricted to black and white patients because of small sample sizes and our 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 after 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 1,806 patients: 467 black and 1,339 white patients.

Measures

Glycemic control. Using information from outpatient laboratory records, we calculated the average A1C value for each month in which a patient was tested. A1C values before 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 A1C over the 1-year period before initiation of therapy to assess baseline severity of illness.

Race and demographic measures. We obtained data on patient race from the electronic medical record, on the basis of clinician reports. These data were validated and supplemented for a subset of patients with self-reports, obtained from a written questionnaire administered to currently enrolled diabetic 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 on the basis of the date of birth and month of diagnosis. Information on patient sex 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, on the basis of 1990 U.S. Census block group data, including median household income, the percentage of residents without a high school education, and the percentage 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 a day's supply is equivalent to daily dose and, therefore, cannot distinguish between physician initiated changes in therapy and patient 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 copayment 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 (minimum $0.01, maximum $60) and median copay for whites $10 (minimum $0.01, maximum $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 after 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 (× 100) during the 3-month period before each laboratory A1C test result.

Medication adherence for patients who discontinued therapy for >60 days was indicated as 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 3 months before an A1C test, we ran the models including and then excluding test months that included a hospitalization. The results were highly consistent across models, which was probably 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. 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 dispensing (or over 60 days after 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 before each laboratory assessed A1C 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 A1C, we created a time-varying measure of intensification, defined as having an increase in dosing or augmentation with another oral hypoglycemic medication after a laboratory-assessed A1C test during the last 6 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 A1C and BMI (underweight to normal <25 kg/m^2, overweight 25–29.9 kg/m^2, and 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 nondiabetic medicines taken in the 3 months before a A1C test, using the first eight digits of the American Hospital Formulary Services code. Because of the high prevalence of both hypertension and hypercholesterolemia among diabetic patients, we further adjusted for any evidence of elevated systolic blood pressure (≥130 mmHg) and total cholesterol (5.18 mmol/l or ≥200 mg/dl) during each year of follow-up (2). Missing laboratory values for these measures were imputed as described below.

The number of physician visits during the 3-month period before each A1C test was also assessed. To control for possible differences in patterns of care, we also created indicators for whether patients were known to have received at

Table 1—*Baseline characteristics among type 2 diabetic patients by race*

	Black	White
n	467 (26)	1,339 (74)
Female sex (%)	**52**	**41**
Age at time of diagnosis (%)		
<45 years	**29**	**14**
45–64 years	**62**	**54**
>65 years	**9**	**32**
Mean A1C level (%)	% missing: 14	% missing: 13
	9.8 ± 2.4	**8.9 ± 2.1**
BMI (kg/m^2)	% missing: 28	% missing: 29
Underweight to normal (%)	11	9
Overweight	31	29
Obese	58	62
Any hospitalization (%)	9	12
Mean systolic blood pressure >130 mmHg (%)	% missing: 1	% missing: 2
	61	**67**
Mean total cholesterol >5.18 mmol/l or >200 mg/dl (%)	% missing: 34	% missing: 31
	74	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**

Data are *n* (%), %, or means ± SD. Bold indicates values statistically significant at the $P < 0.05$ level.

least 50% of their care in one of two settings with a higher proportion of black patients.

Statistical analysis

We assessed baseline (13 months before, including the month the patient initiated therapy) differences in demographic and clinical characteristics using *t* tests and χ^2 tests. We used nonparametric tests (Kolmogorov-Smirnov) (19) to assess racial differences in the number of physician visits and the number of A1C tests. Median months of follow-up were similar for black (median 51 [minimum 13, maximum 108) and white patients (52 [13, 108]).

Our goal for the longitudinal analysis was to examine the relationship between various baseline and time-varying factors and A1C values over time by race. All modeling, including multiple imputation methods, was performed using SAS statistical software (version 9.1.3; SAS Institute, Cary, NC). We used 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 A1C within each racial group. We also ran a combined model that included both races to assess whether the inclusion of medication adherence attenuated the black-white gap.

To account for missing or unrecorded values (i.e., baseline BMI, baseline A1C, 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 (SAS OnlineDocTM: version 8; SAS Institute). We imputed values for covariates but not for the primary predictors or the outcome of interest (A1C).

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 χ^2 statistic (21). We also tested for correlations between the outcomes and medication adherence and self-monitoring behaviors during the previous 3–6 months but found no evidence to justify the inclusion of lags in these variables of longer than 3 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 1,806 newly diagnosed, newly drug-treated patients (26% black and 74% white) met the study criteria. Black patients in our sample were more likely to be women (52 vs. 41%) and aged <45 years (29 vs. 14%). In the 13-month baseline period (including the first month of therapy), black patients had higher average A1C (9.8 vs. 8.9; $P < 0.001$), a difference of 0.88. Black and white patients had similar BMI, laboratory 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 6 and 12 months after initiation of oral therapy, black patients had lower average

Table 2—*Patterns of adherence at 6 and 12 months after initiation of oral hypoglycemic therapy*

	First 12 months after therapy initiation			
	6 months		12 months	
	Black	White	Black	White
Medication adherence	**72.7**	**78.3**	**71.7**	**77.6**
Self-monitoring of blood glucose	20.3	20.7	15.5	17.1
Increase in dose or augmentation	28.6	27.2	38.2	37.8

Data are %. Bold indicates values statistically significant at the $P < 0.0001$ level.

Table 3—*Adjusted A1C values among newly treated diabetic patients*

	Black patients	White patients	Combined model
n	467	1,339	1,806
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 sex	−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 >130 mmHg*	−0.12 (−0.29, 0.04)	0.13 (0.06, 0.20)	0.08 (0.01, 0.14)
Total cholesterol ≥200 mg/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 A1C level*	0.05 (−0.01, 0.11)	0.06 (0.03, 0.09)	0.06 (0.02, 0.08)
Baseline medication (reference = 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)
No. 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)

Data are median (minimum, maximum). Care site and neighborhood socioeconomic status were included but were not significant at the 0.05 level. *Systolic blood pressure (SBP) and total cholesterol were measured annually. Baseline includes the 12 months before and including the therapy initiation month. Physician visits, A1C tests, test strip use, and medication adherence are measured 3 months before each A1C test. Therapy intensification indicates augmentation or an increase in dose for oral diabetes medications after a laboratory assessed A1C test during the last 6 months.

medication adherence compared with whites (Table 2). However, there were no significant differences in either self-monitoring behavior or therapeutic intensification by race.

Correlates of A1C 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 A1C 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 A1C value among blacks (e.g., 8.55 vs. 8.50%) and 0.07% lower A1C among whites. More frequent physician visits were also associated with lower average A1C.

Among whites, other significant predictors of higher A1C included greater comorbidity, younger age, and time since initiation of therapy. Among white patients, higher baseline A1C, systolic blood pressure, and female sex were associated with higher average A1C, but these were not statistically significant among blacks. Among black patients, initiation with >1 oral therapy was also associated with lower A1C values. Receiving care at clinics serving a disproportionate number of black patients and neighborhood socioeconomic states were not statistically significantly associated with A1C levels among black or white patients (data not shown).

Association between medication adherence and the black-white gap in A1C. Results of the combined models 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. With controls for time only 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 addition 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 (column 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 suggests that racial differences in glycemic control cannot be fully explained by variations in the site or quality of care (6,22). The purpose of this study was to explore medication adherence and other self-care determinants of glycemic control among black and white diabetic patients with equal access to care. After adjustment for potential confounders, we found a persistent black-white gap in A1C 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 (22). Still, we were surprised at the modest association of medication refill adherence with A1C. 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 (23,24). In addition, our claims-based measure of medication adherence may underestimate the association between adherence and A1C 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 A1C is consistent with the findings of Schectman et al. (13), who found evidence of a racial gap in A1C after controlling for medication adherence to oral medications. 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 showed that differences in controller medication adherence did not explain racial differences in asthma control (25). Our finding that patients who self-monitor had lower A1C values, although not supported by clinical trial evidence, is consistent with a recent review of self-monitoring among patients with type 2 diabetes (26).

Unlike previous studies (5), our findings of a persistent racial gap in A1C 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 before each test, allowing us to more effectively estimate the temporal relationship between recent patterns of self-management behavior and A1C. Because this was an observational cohort receiving usual care, we could only assess A1C values when patients had a laboratory test. It is possible that the frequency of laboratory testing may have been related to A1C value; patients with higher average A1C levels may have been more or less likely to be tested. However, rates of A1C testing did not vary by race in this population, so frequency of laboratory tests is an unlikely explanation for differences in A1C.

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 such as timing of medication administration, waste, 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 because of the small sample sizes for other racial and ethnic groups. We could not control for several patient-level factors including potential genetic factors (27), environmental influences, patient level barriers (e.g., health literacy), or complementary treatments (e.g., diet and 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 (28). Lastly, these results come from a single large, multispecialty group practice and may not represent the experiences of diabetic 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 (23,24) among black and white patients. Specifically, increased medication adherence was associated with clinically significant reductions in A1C 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 (22). Further research is needed to explore these factors across settings and conditions for which disparities in outcomes have been identified previously. Also, confirmation of these findings from studies using actual observed adherence would lend additional credibility to our results.

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References

1. American Diabetes Association: Economic costs of diabetes in the U.S. in 2002. *Diabetes Care* 26:917–932, 2003
2. American Diabetes Association: Clinical practice recommendations 2006. *Diabetes Care* 29 (Suppl. 1):S1–S85, 2006
3. Wong MD, Shapiro MF, Boscardin J, Ettner SL: Contributions of major disease to disparities in mortality. *N Engl J Med* 347: 1585–1592, 2002
4. Kirk JK, D'Agostino RB, Bell RA, Passmore LV, Bonds DE, Karter AJ, Narayan KMV: Disparities in HbA1c levels between African-American and non-Hispanic white adults with diabetes. *Diabetes Care* 29: 2130–2136, 2006
5. Bach PB, Pham HH, Schrag D, Tate RC, Hargraves JL: Primary care physicians who treat blacks and whites. *N Engl J Med* 351:575–584, 2004
6. Sequist T, Adams AS, Zhang F, Ross-Degnan D, Ayanian JZ: The effect of quality improvement on racial disparities in diabetes care within a multispecialty group practice. *Arch Intern Med* 166:675–681, 2006
7. Duru OK, Mangione CM, Steers NW, Herman WH, Karter AJ, Kountz D, Marrero DG, Safford MM, Waitzfelder B, Gerzoff RB, Huh S, Brown AF, TRIAD Study Group: The association between clinical care strategies and the attenuation of racial/ethnic disparities in diabetes care: the Translating Research into Action for Diabetes (TRIAD) Study. *Med Care* 44:1121–1128, 2006
8. Trinacty CM, Adams AS, Soumerai SB, Zhang F, Meigs JB, Piette JD, Ross-Degnan D: Racial differences in long-term self-monitoring practice among newly drug-treated diabetes patients in an HMO. *J Gen Intern Med* 22:1506–1513, 2007
9. Kirk JK, Graves DE, Bell RA, Hildebrandt CA, Narayan KM: Racial and ethnic disparities in self-monitoring of blood glucose among US adults: a qualitative review. *Ethn Dis* 17(1):135–142, 2007
10. Nwasuruba C, Khan M, Egede LE: Racial/ethnic differences in multiple self-care behaviors in adults with diabetes. *J Gen Intern Med* 22:115–120, 2007
11. Karter AJ: Race and ethnicity: vital con-

structs for diabetes research. *Diabetes Care* 26:2189–2193, 2003
12. Pladevall M, Williams LK, Potts LA, Divine G: Xi H, Lafata JE: Clinical outcomes and adherence to medications measured by claims data in patients with diabetes. *Diabetes Care* 27:2800–2805, 2004
13. Schectman JM, Nadkarni MM, Voss JD: The association between diabetes metabolic control and drug adherence in an indigent population. *Diabetes Care* 25:1015–1021, 2002
14. Hills-Briggs F, Gary TL, Bone LR, Hill MN, Levine DM, Brancati FL: Medication adherence and diabetes control in urban African Americans with type 2 diabetes. *Health Psychol* 24:349–357, 2005
15. Adams AS, Zhang F, Mah C, Grant RW, Kleinman K, Meigs JB, Ross-Degnan D: Race differences in long-term diabetes management in an HMO. *Diabetes Care* 28:2844–2849, 2005
16. Chan KA, Davis RL, Gunter MJ, Gurwitz JH, Herrinton LJ, Nelson WW, Raebel MA, Roblin DW, Smith DH, Platt R: The HMO Research Network. In *Pharmacoepidemiology*. 4th ed. Strom BL, Ed. West Sussex, England, Wiley, 2005, p. 261–269
17. Andrade SE, Kahler KH, Frech F, Chang KA: Methods for evaluation of medication adherence and persistence using automated databases. *Pharmacoepidemiol Drug Saf* 15:565–574, 2006
18. Schneeweiss S, Seeger JD, Maclure M, Wang PS, Avorn J, Glynn RJ: Performance of comorbidity scores to control for confounding in epidemiologic studies using claims data. *Am J Epidemiol* 154:854–864, 2001
19. Sheskin DJ: *Handbook of Parametric and Nonparametric Statistical Procedures.* 2nd ed. New York, Chapman and Hall/CRC, 2000
20. Littell RC, Milliken GA, Stroup WW, Wolfinger RD: *SAS System for Mixed Models*. Cary, NC, SAS Institute, 1996
21. McCulloch CE, Searle SR. *Generalized, Linear, and Mixed Models.* New York, Wiley, 2001
22. Karter AJ, Ferrara A, Liu JY, Moffet HH, Ackerson LM, Selby JV: Ethnic disparities in diabetic complications in an insured population. *JAMA* 287:2519–2527, 2002
23. Grant RW, Buse JB, Meigs JB: Quality of diabetes care in US academic medical centers: low rates of medical regimen change. *Diabetes Care* 28:337–442, 2005
24. Karter AJ, Moffet HH, Liu J, Parker MM, Ahmed AT, Go AS, Selby JV: Glycemic response to newly initiated diabetes therapies. *Am J Manag Care* 13:598–606, 2007
25. Erickson SE, Iribarren C, Tolstykh IV, Blanc PD, Eisner MD: Effect of race on asthma management and outcomes in a large, integrated managed care organization. *Arch Intern Med* 167:1846–1852, 2007
26. McGeoch G, Derry S, Moore RA: Self-monitoring of blood glucose in type-2 diabetes: what is the evidence? *Diabetes Metab Res Rev* 23:423–440, 2007
27. Hart CB: Race Differences in long-term diabetes management in an HMO: Response to Adams et al. *Diabetes Care* 29: 1461–1462, 2006
28. Soobader M, LeClere FB, Hadden W, Maury B: Using aggregate geographic data to proxy individual socioeconomic status: does size matter? *Am J Public Health* 91: 632–636, 2001