Predictors of Glycemic Control in Insulin-Using Adults With Type 2 Diabetes

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OBJECTIVE — To determine the characteristics that influence glycemic control among insulin-using adults with type 2 diabetes.

RESEARCH DESIGN AND METHODS — We studied all 1,333 eligible members of a large not-for-profit health maintenance organization who responded to a 1997 survey. We tested associations among demographic, treatment, and psychometric variables with mean 1997 HbA_{1c} values. The Problem Areas in Diabetes (PAID) instrument was used to assess the emotional effect of living with diabetes, and the Short Form 12 Physical Function Scale was used to assess the effect of physical limitations on daily activities. Based on differences between and within treatment groups, we built models to predict glycemic control for subgroups of subjects who were using insulin alone and those who were using insulin in combination with an oral hypoglycemic agent.

RESULTS — Younger age, lower BMI, and increased emotional distress about diabetes (according to the PAID scale) were all significant predictors ($P < 0.05$) of worse glycemic control. However, except among individuals with an HbA_{1c} level of >8.0 who were receiving combination therapy, only ~10% of the variance in glycemic control could be predicted by demographic, treatment, or psychometric characteristics.

CONCLUSIONS — Personal characteristics explain little of the variation in glycemic control in insulin-using adults with type 2 diabetes. Possible explanations are that the reduced complexity of control in type 2 diabetes makes the disease less sensitive to personal factors than control in type 1 diabetes, that health-related behavior is less driven by personal and environmental characteristics among older individuals, or that, in populations exposed to aggressive glycemic control with oral hypoglycemic agents and nurse care managers, personal differences become largely irrelevant.

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Since the Diabetes Control and Complications Trial (DCCT) demonstrated that excellent glycemic control reduces microvascular complications resulting from diabetes (1), glycated hemoglobin has become an increasingly important measure of glycemic control. Although the DCCT studied only individuals with type 1 diabetes, prospective studies suggested (2,3), and the recent U.K. Prospective Diabetes Study (UKPDS) results confirmed (4), that improving glycemic control also reduces microvascular complications in type 2 diabetes. New standards of quality care hold providers accountable for periodic measurement of glycated hemoglobin in their diabetic patients (5–9) with the reasonable (although untested) presumption that intensified monitoring will intensify glucose management and thus glycemic control.

Despite the strong consensus that excellent glycemic control improves microvascular outcomes in type 2 diabetes (10), surprisingly little is known about which patient characteristics predict better glycemic control. Identifying demographic, treatment, and psychometric characteristics that predict poor control may improve outcomes by allowing better matching of patients to compensatory interventions.

Previous studies evaluating predictors of glycemic control have primarily been conducted in individuals with type 1 diabetes (11–13). Characteristics of disease, treatment, and the home environment explained an astounding 94% of the variance in HbA_{1c} level in a recent study of 2,579 French children with type 1 diabetes (11). Another recent study among 150 insulin-using adults, more than half of whom had type 1 diabetes (14), showed that family system variables such as cohesion and supportiveness failed to predict glycemic control but that age and duration of disease did predict glycemic control. We are not aware of any population-based studies that have assessed predictors of glycemic control in type 2 diabetes. Predictors may vary with the type of diabetes or the mode of therapy (15). We focused our study on insulin-using patients with type 2 diabetes who were members of a large group-model health maintenance organization (HMO). Our goals were to assess which characteristics of insulin-using subjects with type 2 diabetes were associated with poor glycemic control in a population-based sample and to propose a statistical model for predicting their glycemic control.

RESEARCH DESIGN AND METHODS

Research setting

Subjects were members of a long-established not-for-profit group-model HMO, Kaiser Permanente Northwest Division (KPNW). KPNW provides comprehensive prepaid

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Abbreviations: DCCT, Diabetes Control and Complications Trial; HMO, health maintenance organization; KPNW, Kaiser Permanente Northwest Division; PAID, Problem Areas in Diabetes; SF-12, Short Form 12; UKPDS, U.K. Prospective Diabetes Study.

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

coverage to about 20% of the Portland, Oregon, population (an average of 423,000 people at the time of this study). KPNW's subscribers are demographically similar to the area population as a whole (16).

The KPNW Diabetes Registry has been described in detail elsewhere (17). Briefly, we initiated the registry in 1987 by searching electronic records of pharmacy purchases and hospital discharge diagnoses. Ascertainment of new registrants now occurs in one of four ways: *1*) prescription of a glucose-lowering medication (sulfonylurea, metformin, acarbose, troglitazone, or insulin), *2*) two or more prescriptions for blood glucose testing supplies and subsequent review by an endocrinologist to confirm diabetes, *3*) enrollment in diabetes education classes or an outpatient visit to the medical office of a diabetes educator, or *4*) admission to an acute care hospital with any listed diagnosis indicating diabetes. After review by an endocrinologist (Harry S. Glauber, MD, Kaiser Permanente Center for Health Research), members may be removed from the registry when a clinician questions their inclusion or when a patient challenges the diagnosis.

In 1997, to learn additional information about KPNW members with diabetes that would not be accessible from databases linked to medical care, we mailed a 28-page comprehensive survey to 11,331 members of the registry, 6,623 (58.5%) of whom responded. Only members who completed and returned their surveys were included in this study. Consistent with other surveys conducted at this site, respondents were somewhat older than nonrespondents (aged 62.5 vs. 57.8 years, $P < 0.001$) and were more likely to be women than men (50.7 vs. 46.5%, $P < 0.001$).

Eligibility criteria

Our target population consisted of members with type 2 diabetes taking insulin alone or in combination with an oral hypoglycemic agent in 1997. Because most people diagnosed with diabetes after 45 years of age have type 2 diabetes (18), we classified subjects as having type 2 diabetes if age at diagnosis was >45 years. Individuals aged ≤45 years were also classified with type 2 diabetes if they did not purchase insulin during the 2 years after diagnosis. Additional inclusion criteria included responding to the 1997 survey, 12 full months of HMO eligibility in 1997, and an HbA_{1c} or fructosamine measurement in 1997. We eliminated <15% of the insulin-using population because they did not have one of these tests in 1997. Members who purchased three or more classes of antidiabetic drugs in 1997 were excluded from the study because we could not reliably assess their therapeutic regimen. A total of 1,333 members met the inclusion criteria. Incomplete data for 155 patients required that they be eliminated from the analyses, which resulted in a final study population of 1,178.

Measurements and calculations

All 1,178 subjects in this study had at least one HbA_{1c} or fructosamine test in 1997, and 77% (911) had two or more tests. The mean number of tests was 2.8 per subject. Glycemic control was defined as the mean of these test results. For this report, we converted fructosamine results (bG21 colorimetric assay; Boehringer Mannheim, Indianapolis, IN) to their HbA_{1c} equivalents (Diamat high-performance liquid chromatography assay; Bio-Rad, Hercules, CA) by using the following formula: fructosamine/40 = HbA_{1c}. This simple formula closely approximates the actual relationship found in our HMO based on regression analysis of 364 paired samples drawn simultaneously ($R^2 = 0.65$, $P < 0.001$ [S. Welch, PhD, unpublished observations]). Although the conversion performed best when fructosamine values were <400 µmol/l, <10% of the patients had values above that level. Mean HbA_{1c} (± SEM) was 7.9 ± 0.04% for the entire cohort.

Age and sex data were obtained from the HMO's membership data system. Weight was measured at clinic visits for 97% of the study subjects and was obtained by self-report from the 1997 survey for the remaining 3%. Time since diagnosis of diabetes was calculated either from time of entry into the registry or by self-report, whichever yielded a longer duration. Smokers were defined as subjects who reported smoking ≥20 cigarettes during the previous year on the 1997 survey.

Average daily units of insulin used were calculated by dividing the total number of units dispensed by the number of days supplied. This calculation should accurately represent average insulin dosages because ~97% of the members who responded to the survey question about pharmacy use reported obtaining all or most of their insulin and diabetes medications at pharmacies within the HMO (G.A.N., unpublished observations). Frequency of self-monitoring of blood glucose was based on self-report in the questionnaire.

To assess the ability of physical limitations, emotional distress, and lifestyle modifications to predict glycemic control, we used three scales derived from questionnaire responses. We used the Physical Functioning Scale from the Short Form 12 (SF-12) to assess health limitations on daily activities (19–21), and we used the Problem Areas in Diabetes (PAID) instrument to assess the adverse emotional effect of living with diabetes (22,23). Self-care was assessed with a third scale constructed with a summary of the yes/no responses to the following lifestyle modification question:

> Which of the following things did you do most of the time during the past 12 months: *1*) closely controlled the amount of calories I ate at most meals and snacks; *2*) at least three times a week, got moderate exercise such as walking, house cleaning, lawn mowing, or gardening; *3*) at least two times a week, got vigorous exercise such as aerobics, biking, swimming, running, or fast walking; *4*) kept the calories from fat in my food low (below 30%); *5*) ate four or more servings of fruit or vegetables each day; *6*) controlled the timing of my meals and snacks to match my insulin; and *7*) controlled the amount and timing of exercise I got to match my insulin.

The Physical Functioning Scale, the PAID, and the self-care scale demonstrated strong internal consistency/reliability (Cronbach's α of 0.84, 0.96, and 0.76, respectively).

Statistical analysis

Statistical analyses were performed by using SAS Version 6.12 (SAS Institute, Cary, NC). To make initial comparisons between subjects receiving insulin only and subjects receiving combination therapy, we used either a two-tailed Student's *t* test or Pearson's χ^2 test. We then built separate multiple linear regression models to predict glycemic control for patients receiving insulin only and for those receiving combination therapy. Our final models include those variables that were significantly associated ($P < 0.05$) with HbA_{1c} as well as those that the literature suggested should correlate with glycemic control.

RESULTS

— Subjects receiving insulin only ($n = 931$) differed from those receiving insulin in combination with oral hypoglycemic agents ($n = 247$) in several respects. Most differences were not clinically significant. Subjects receiving insulin alone were older (aged 65.9 vs. 64.3 years,

$P < 0.05$), had a longer duration of diabetes (16.5 vs. 13.5 years, $P < 0.001$), and were less obese (BMI 31.8 vs. 33.9, $P < 0.001$) than those receiving combination therapy. Subjects receiving insulin only tested their blood glucose levels at home slightly more frequently (9.9 vs. 9.0 times/week, $P < 0.05$) and were in slightly better glycemic control than subjects receiving combination therapy (7.8 vs. 8.1% HbA_{1c}, $P < 0.05$).

In the light of the differences between the two therapy subgroups, we built two separate multiple linear regression models of glycemic control: one for patients receiving insulin only and one for patients receiving combination therapy. Younger age, male sex, and lower BMI significantly predicted worse glycemic control (i.e., higher HbA_{1c} level) among subjects receiving insulin alone (Table 1, column 1). On a standardized basis, BMI was the strongest predictor (standard beta = −0.20) followed by age (standard beta = −0.15) and male sex (standard beta = −0.09). Increased worry about diabetes (according to the PAID scale) and less attention to self-care regarding diet and exercise also predicted worse glycemic control. Surprisingly, better physical functioning also predicted worse control. Overall, the model for subjects receiving insulin explained 9% of the variance in glycemic control.

The model for patients receiving combination therapy also explained ~9% of the variation in glycemic control (Table 1, column 2). In this group, lower BMI was again the strongest predictor of worse control (standard beta = −0.26) followed again by younger age, although the coefficient for age showed only borderline statistical significance (standard beta = −0.20, P = 0.086). Increased negative emotional effect of diabetes (according to the PAID scale) also repeated as a significant predictor of worse glycemic control. Sex, physical function, and self-care, however, disappeared as statistically significant predictors, and African-American race was significant in predicting worse control.

Two variables with known physiological effects on HbA_{1c} level (daily insulin dose per kilogram and duration of disease) were not statistically significant in either model. Other considered variables that were not found to be statistically significant in either model were dichotomous variables for Asian/Pacific Islander race, Native American race, frequency of home glucose monitoring, use of metformin among subjects receiving combination therapy, and smoking status.

Table 1—*Standardized β-coefficients of predictors of glycemic control*

	Insulin only	Combination therapy	Poor control, combination therapy
n	931	247	115
Age	−0.146*	−0.204	−0.933†
Years since diagnosis	0.137	−0.068	−1.848†
Age × duration	−0.025	0.114	1.913†
Female	−0.090*	−0.060	0.100
African-American	0.034	0.159*	0.132
BMI	−0.200†	−0.255†	−0.362†
Physical Functioning Scale (SF-12)	0.071*	−0.054	−0.208†
PAID	0.079*	0.137*	−0.076*
Self-care, diet, and exercise	−0.078*	−0.079	−0.090

*$P < 0.05$; †$P < 0.001$.

The proportion of the variance in explained HbA_{1c} was statistically significant but small in both models. To confirm that these results were not due to inadequate sample size, we calculated the power of our multivariate regression approach. The insulin-only model had 99% power to detect a small effect size (increase in variance explained = 0.02) in HbA_{1c} level. The smaller sample size of the combination therapy model had 80% power to detect a small effect size and 92% power to detect a medium effect size (increase in variance explained = 0.15) in HbA_{1c} level.

We also questioned whether our inability to predict more of the variance in glycemic control stemmed from the fact that most of the population was already in good or excellent control. With 83% of the population having HbA_{1c} levels <9% and 58% of the population having HbA_{1c} levels <8%, the explainable variance in this population may already have been minimized. We tested this hypothesis by modeling the subset of patients with an HbA_{1c} level >8% (n = 500). We again conducted these analyses separately for patients receiving insulin only (n = 385) and for patients receiving combination therapy (n = 115). The insulin-only model performed worse than the model for the entire insulin-only population and explained <4% of the variance in glycemic control. Only the variable for African-American race was significant (data not shown). Among subjects receiving combination therapy, however, the model limited to subjects who were poorly controlled explained 28% of the variation in HbA_{1c}.

The third column in Table 1 displays the results of the regression for 115 subjects receiving combination therapy with an average HbA_{1c} of ≥8%. Younger age, shorter duration of diagnosed diabetes, and the interaction of age and duration were all highly significant predictors of higher HbA_{1c} levels (i.e., worse control). Moreover, the standardized coefficients for these variables were much higher than those estimated in the full population models and ranged from −0.993 (age) to 1.913 (age × duration). Lower BMI again predicted worse glycemic control. The standardized β for BMI was larger than in the full-population models (−0.362) but was still much weaker than its fellow age and duration variables in the model for poorly controlled subjects. Poor physical function in this group was significantly associated with worse control rather than with better control as it was among patients receiving insulin only.

CONCLUSIONS — We regressed a measure of glycemic control (HbA_{1c}) against demographic, treatment, and psychometric variables in a relatively well-controlled population of 1,178 patients with type 2 diabetes who were using insulin. We constructed separate models for patients receiving insulin alone (n = 931), for patients receiving combination therapy (n = 247), and for subgroups with an HbA_{1c} level of >8%.

The dimensions of health represented by the questionnaire scales, as well as by demographic and treatment variables, generally explained much less of the variation in glycemic control than we expected. The models were statistically significant, adequately powered, and stable. Age, BMI, and the adverse emotional effect of diabetes (according to the PAID scale) were consistently significant predictors of glycemic con-

trol or, in the case of the PAID scale, lack of control. However, with one exception, ethnicity showed no association with HbA_{1c}, and sex was significant only among patients who used insulin exclusively. Perhaps more surprisingly, no aspect of therapy (intensity of insulin treatment, use of metformin, or intensity of self-monitoring of blood glucose) was significant in any model. The Physical Function Scale of the SF-12 survey, which is often an important correlate of medical behavior (24,25), performed enigmatically. Worse physical function predicted better control among subjects receiving insulin only, showed no predictive value among subjects receiving combination therapy taken as a whole, and predicted worse control among subjects receiving combination therapy who were poorly controlled. Although the correlations between physical functioning and other variables such as age and BMI were not high enough to suggest a multicollinearity problem (-0.19 and -0.23, respectively), we suspect that the shared variance among these variables contributed to the counterintuitive result. In our registry, male and especially younger members have substantially poorer glycemic control, presumably for behavioral reasons. The fact that removing physical functioning from the model did not greatly affect its overall performance supports this explanation.

These results are dramatically at odds with a recent study of French children with type 1 diabetes in which variables that were mostly nonsignificant in our model combined to explain 94% of the variance in HbA_{1c}. These variables included insulin dosage per kilogram, frequency of glucose monitoring, duration of disease, dietary compliance, and family support (11). More consistent with our results, an earlier cross-sectional study of 184 patients with type 2 diabetes found that psychosocial variables did not predict glycemic control (26). Recent data from the Third National Health and Nutrition Examination Survey have suggested that demographic and patient characteristics are not predictive in adults with HbA_{1c} values of $>8\%$ (27). In that study, measures of self-reported health status were not assessed (27).

The relative failure of these studies and our own to explain much of the variance in glycemic control suggests that psychosocial measures may have little effect on achieving glycemic control in type 2 diabetes. A contributing factor in our own study may have been the generally very good glycemic control of the study population. By 1997, this population had been exposed to a decade of concerted population-based quality improvement efforts aimed at improving glycemic control and overall diabetes care. These efforts included access to nurse care managers trained in promoting patient behavior changes via motivational interviewing, greatly expanded diabetes educational programs, and various initiatives designed to increase physician awareness of glycemic control. In fact, the average HbA_{1c} level decreased from 8.6% in 1988 to 7.75% in 1999 for the entire diabetes population (J.B.B., unpublished observations).

Support for the inability to explain the variance in glycemic control in a well-controlled population comes from the fact that, in the subgroup of subjects receiving combination therapy who were poorly controlled, patient characteristics explained a substantial proportion of HbA_{1c} variance (28%). In this subgroup, age, duration of diagnosed diabetes (shorter duration), and the interaction of duration and age strongly predicted poorer glycemic control. This suggests that excellent glycemic control may be much more difficult to achieve for some younger patients with a shorter duration of disease. Prospective studies are needed to evaluate whether individuals developing type 2 diabetes at an earlier age represent a different phenotype that requires more aggressive interventions to achieve glycemic control.

The strong significance of BMI in explaining glycemic control for all of the groups deserves comment. Members of our cohort exhibit a rate of obesity similar to that found in the U.S. as a whole (28). Higher BMI was by far the strongest and most consistent predictor of better glycemic control in the total population models and was similarly predictive among subjects who were poorly controlled. The most likely explanation is that improved control causes weight gain, not that weight gain improves control. Such an explanation is consistent with the UKPDS finding that intensive glycemic control caused a 2- to 5-kg weight gain compared with conventional control in insulin-using type 2 diabetic patients without hyperglycemic symptoms (4). The adverse effect of weight gain with improved glycemic control may worsen other physiological parameters such as hypertension and hypercholesterolemia, which are important risk factors for cardiovascular disease (29–32). This may help explain why studies of the effect of glycemic control on cardiovascular disease events have not found strong effects (4,33,34). An upcoming National Heart, Lung and Blood Institute multicenter trial aimed at evaluating the separate and combined effects of intensively managing glycemia, hypertension, and lipids should clarify this issue.

A limitation of the present study is that respondents and nonrespondents to the questionnaire differed in several respects. In addition to being somewhat older and more likely to be women, respondents were more likely to have had an HbA_{1c} or fructosamine measurement than nonrespondents (89.7 vs. 74.2%, $P < 0.001$), which was an inclusion criterion for the study. Furthermore, of those with a measurement, respondents had a lower HbA_{1c} value than nonrespondents (7.7 vs. 8.1%, $P < 0.001$). The predictors included in our total population models possibly may have explained more variance in HbA_{1c} if nonrespondents had responded to the survey.

In summary, demographic, treatment, and psychometric variables were not strong predictors of glycemic control for most subjects in a generally well-controlled population of insulin-using patients with type 2 diabetes. Whether the physicians and nurse care managers at this study site were particularly skilled at identifying and overcoming individual barriers to control, whether individual characteristics are now largely irrelevant in controlling late-stage type 2 diabetes, or whether aggressive pharmacologic treatment renders individual differences moot cannot be determined from our observational data. Further research on the relative contributions to diabetes outcomes of patient behavior modification versus more aggressive medical management may help optimize future resource use.

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