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Mary Rhee, Emory University
David Carleton Ziemer, Emory University
Jane M. Caudle, Emory University
Paul Kolm, Christiana Care Center for Outcomes Research
Lawrence S Phillips, Emory University

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Use of a Uniform Treatment Algorithm Abolishes Racial Disparities in Glycemic Control

Mary K. Rhee, MD, David C. Ziemer, MD, Jane Caudle, MLn, Paul Kolm, PhD, and Lawrence S. Phillips, MD

Division of Endocrinology and Metabolism, Department of Medicine, Emory University School of Medicine, Atlanta, Georgia (Dr Rhee, Dr Ziemer, Ms Caudle, Dr Phillips), and the Christiana Care Center for Outcomes Research, Newark, Delaware (Dr Kolm).

Abstract

Purpose—The purpose of this study is to compare glycemic control between blacks and whites in a setting where patient and provider behavior is assessed, and where a uniform treatment algorithm is used to guide care.

Methods—This observational cohort study was conducted in 3542 patients (3324 blacks, 218 whites) with type 2 diabetes with first and 1-year follow-up visits to a municipal diabetes clinic; a subset had 2-year follow-up. Patient adherence and provider management were determined. The primary endpoint was A1c.

Results—At presentation, A1c was higher in blacks than whites (8.9% vs 8.3%; \(P < .001\)), even after adjusting for demographic and clinical characteristics. During 1 year of follow-up, patient adherence to scheduled visits and medications was comparable in both groups, and providers intensified medications with comparable frequency and amount. After 1 year, A1c differences decreased but remained significant (7.7% vs 7.3%; \(P = .029\)), even in multivariable analysis (\(P = .003\)). However, after 2 years, A1c differences were no longer observed by univariate (7.6% vs 7.5%; \(P = .51\)) or multivariable analysis (\(P = .18\)).

Conclusions—Blacks have higher A1c than whites at presentation, but differences narrow after 1 year and disappear after 2 years of care in a setting where patient and provider behavior are comparable and that emphasizes uniform intensification of therapy. Presumably, racial disparities at presentation reflected prior inequalities in management. Use of uniform care algorithms nationwide should help to reduce disparities in diabetes outcomes.


Correspondence to Mary K. Rhee, MD, Division of Endocrinology and Metabolism, Emory University School of Medicine, 49 Jesse Hill Jr Drive SE, Atlanta, GA 30303 (E-mail: mrhee@emory.edu)

Portions of this work were presented at the Scientific Sessions of the American Diabetes Association in Philadelphia, PA (2001) and in Orlando, FL (2004).
Moreover, racial disparities in health have been recognized as early as the 1980s, but recent studies show that blacks continue to have lower rates of reperfusion therapy, coronary angiography, and higher in-hospital mortality after myocardial infarction (MI) compared to whites, and persistent differences in rates of major surgical procedures.\textsuperscript{9,10} An evaluation of trends in quality of care measures (Health plan Employer Data and Information Set [HEDIS]) found that racial differences in glycemic control and low-density lipoprotein (LDL)–lowering post-MI actually widened during a 5- to 6-year period.\textsuperscript{11} Racial disparities in health care have been attributed to differences in the clinical training and health resources of physicians who treat black versus white patients,\textsuperscript{12} trust,\textsuperscript{13} communication during visits,\textsuperscript{14} and interpersonal bias,\textsuperscript{13,15,16} prompting calls for new approaches to help close the gap in care.\textsuperscript{17}

The purpose of this study is to compare glycemic control between blacks and whites in a setting where patient and provider behavior is assessed, and where a uniform treatment algorithm is used to guide care.

**Research Design and Methods**

**Design**

In 1991, we began a prospective, observational, cohort study to determine whether the use of a uniform treatment algorithm would improve diabetes care over time. Given the improvements observed with our algorithm, we then conducted a retrospective analysis in order to determine the association between race (exposure of interest) and glycemic control during care under a uniform treatment algorithm (outcome of interest) during the same period of time. The advantage of cohort studies such as this is that they provide the strongest observational design for establishing clear temporal relationships between exposure and disease and generally allow minimization of bias. However, they can be limited by biases of differential loss to follow-up between exposure groups and cannot fully account for differences in confounders.

**Setting and Subjects**

The Diabetes Clinic of the Grady Health System in Atlanta, Georgia, serves a municipal, predominantly uninsured population and provides multidisciplinary diabetes management.\textsuperscript{18} Under a uniform care algorithm, nonpharmacologic approaches (diet and exercise) are emphasized for the first 2 months, after which pharmacologic therapy is increased progressively, aiming to achieve A1c < 7%.\textsuperscript{18} The algorithm\textsuperscript{19} recommends that pharmacologic treatment be initiated or intensified whenever A1c is $\geq 7.0\%$ and/or random glucose (RBG) levels are $\geq 150$ mg/dL (8.3 mmol/L) at the time of out-patient visits.\textsuperscript{20} The algorithm takes into consideration previous use of different therapies and recommends changes in therapy that are specific and scaled to glucose levels. Clinical and laboratory data from all visits are maintained in a relational database (Oracle, Redwood City, Calif).

From the Diabetes Clinic database, selected patients included those with type 2 diabetes, of black or white race, and a first visit to the clinic with an A1c between April 1, 1991, and July 1, 2005, with a follow-up visit 1 year after initial presentation (10-14 months after initial presentation; n = 3542). Among these patients, those with 2-year follow-up visits (22-26 months after initial presentation) were also evaluated. Race was self-reported (eg, white [non-Hispanic white], black [non-Hispanic black], etc), and type 2 diabetes was identified by typical clinical features.

A1c was measured using instrumentation from BioRad (high-performance liquid chromatography [HPLC]; DIA-MAT, Hercules, Calif) between 1991 and 1997 and Boehringer Mannheim (turbidimetric immuno-inhibition; Hitachi 717, Indianapolis, Ind) from 1997 to

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present (normal range, 3.5%-6.0%, both methods). Both methods are National Glycohemoglobin Standardization Program certified and were shown to give comparable results.

Methods

Three components of patient health were evaluated as potential contributors to differences in glycemic control:

a. Demographic/clinical factors included age, gender, body mass index (BMI), and disease duration.

b. Patient adherence to management recommendations was assessed as reported previously\textsuperscript{21} by use of medications and the number of scheduled follow-up appointments kept. Reported medication use at each incoming visit was compared with medications recommended at the previous visit; use of glucose-related medications that was identical to or greater than that recommended at the previous visit was assigned an adherence value of 1; use that was less than recommended was assigned a value of 0. For each patient, an adherence score was averaged for all visits. Appointment-keeping behavior was assessed by the number of visits per year; 7 visits are routinely scheduled in the first year after presentation, and 4 visits per year (approximately every 3 months) are usually scheduled in subsequent years.

c. Measures of management consisted of diabetes therapy, percentage of visits in which diabetes therapy was intensified when needed (“frequency”), and the amount of intensification compared to recommendations by the algorithm (see below). Such measures allow assessment of provider behavior, with lower levels of intensification reflecting greater “clinical inertia”—the failure of providers to intensify therapy when appropriate\textsuperscript{22}—which has been associated with worse glycemic control in patients with diabetes.\textsuperscript{23} The algorithm called for intensification whenever A1c was above 7% or RBG above 150 mg/dL (8.3 mmol/L). The frequency of intensification was defined as the number of visits in which providers intensified therapy divided by the total number of visits in which intensification was indicated, expressed as a percentage. The amount of intensification was determined as well. Data in the literature were used to estimate the amounts of sulfonylurea (SU), metformin, or insulin required to effect a 1% change in A1c, and each defined as a “step” of therapy. One step of therapy was designated as appropriate for each 1% A1c above 7% or each 50 mg/dL (2.8 mmol/L) random plasma glucose above 150 mg/dL (8.3 mmol/L).\textsuperscript{24} The amount of intensification was defined as the number of “steps done” divided by the number of “steps needed” (recommended by the algorithm for given antecedent therapies and presenting glucose levels), expressed as a percentage for each visit.

Statistical Analysis

Univariate analyses were performed using the Student t test, nonparametric testing, or the chi-square test as appropriate. Multivariable linear regression analysis was used to examine the contribution of race to A1c, adjusting for age, gender, BMI, diabetes duration, diabetes therapy, year of presentation, number of appointments kept, medication adherence and frequency, and amount of intensification by providers. Analyses evaluating factors associated with A1c at 1-year follow-up and at 2-year follow-up were also adjusted for initial A1c. Unpaired t tests were used to compare A1c values of blacks and whites at each time point. All statistical analyses were conducted using SPSS 13.0 (SPSS Inc, Chicago, Ill).
Results

This prospective, observational study included 3542 eligible patients, 3324 black and 218 white. At presentation, age, BMI, and diabetes duration were comparable between both groups, but blacks were more likely to be female and to be treated with insulin than whites (both \( P < .05 \)) (Table 1). Initial A1c was higher in blacks than whites (8.9% vs 8.3%; \( P < .001 \)) (Figure 1A). In multivariable regression analysis, adjusting for age, gender, BMI, diabetes duration, therapy, and year of presentation, black race had a strong, independent association with poorer glycemic control—a 0.61% higher A1c than whites (\( P < .001 \)) (Table 2).

After 1 year of follow-up, the difference in A1c between blacks and whites narrowed but remained significant (7.7% vs 7.3%; \( P = .029 \)), shown in Table 1. Patient adherence was comparable in blacks vs whites (adherence to medications [74% black vs 76% white], and the number of interim visits [5.5 black vs 5.6 white]; both \( P = \) not significant [NS]). Provider behavior was similar as well (“frequency of intensification” [44% of visits of blacks vs 43% of whites], and “amount” of intensification [50% in visits of blacks vs 48% of whites]; both \( P = \) NS). In multivariable regression analysis, better medication adherence and appointment keeping, along with higher amount of provider intensification, contributed to improved A1c levels at 1 year. After adjustment for all other factors, black race continued to confer a 0.48% higher A1c than whites after 1 year of care (\( P = .003 \)) (Table 2).

Because blacks presented with higher A1c levels (Figure 1A), and because patient and provider behaviors were similar between blacks and whites after 1 year of care, patients who returned for care after 2 years were further evaluated to determine whether the racial disparity in glycemic control dissipated with more time: more time for patients to have been taking appropriate medications and to have seen their providers, and more time for the providers to have intensified therapy as needed. The patients who returned for 2-year follow-up visits (\( n = 1795 \)) were older (56 vs 53 years), more likely to be female (66% vs 62%), and had a shorter duration of diabetes (4.5 vs 5.1 years) than those who did not return after 2 years (all \( P < .05 \)). Patients who returned for 2-year follow-up visits also had higher medication adherence (78% vs 71%) and better appointment-keeping behavior (5.9 vs 5.1 visits) after 1 year of follow-up (both \( P < .001 \)). However, provider behavior was somewhat less aggressive for patients who returned for 2-year follow-up versus those who did not (frequency of intensification 42% vs 45%, amount of intensification 47% vs 54%; both \( P < .001 \)). There were no differences in A1c at presentation or after 1 year of care between those who returned for 2-year follow-up versus those who did not.

Among the patients who returned for a 2-year follow-up visit (1681 black, 114 white), there were no racial differences in age, gender, BMI, duration of diabetes, or therapy (Table 1). Between year 1 and year 2, patient adherence was comparable in the 2 groups (adherence to medication [85% black vs 86% white], number of appointment kept [3.4 black vs 3.3 white]; both \( P = \) NS). There was also no difference in provider frequency of intensification (61% black vs 56% white; \( P = .28 \)) or amount of intensification in blacks compared to whites (73% vs 59%; \( P = .10 \)). A1c levels after 2 years of follow-up were no longer different between blacks and whites (7.6% vs 7.5%; \( P = .17 \)) (Figure 1B).

In multivariable regression analysis, both better patient adherence to medications and the amount of intensification conferred lower A1c values. However, black race was no longer a significant contributor to A1c after 2 years of follow-up (Table 2) even after adjusting for patient adherence and provider behavior. Similar results were found by analysis using linear mixed effects models for incomplete repeated measures data (not shown).
Conclusions

Blacks are disproportionately afflicted with diabetes and suffer higher rates of complications and mortality than whites. While the cause of these disparities is unclear, higher diabetes-associated morbidity and mortality are likely due largely to the combination of poor blood pressure, lipid, and glycemic control in blacks. Although racial disparities in health care are well recognized, and process measures have improved, differences in outcomes have shown little or no improvement. Indeed, a recent analysis of trends in glycemic control in a Medicare population showed that the gap between blacks and whites increased during the 5-year period, even after adjusting for socioeconomic factors. Moreover, in both a Medicare managed-care and a health maintenance organization (HMO) population, where blacks and whites have equal access to care, blacks continued to have higher A1c levels than whites. Our study sought to determine (a) whether racial differences in glycemic control can be attributed to differences in demographic/clinical characteristics, patient adherence measures, and/or measures of care by providers, and (b) whether use of a uniform algorithm for diabetes management can alleviate racial differences in glycemic control.

In our study, blacks had higher A1c levels than whites at presentation, but differences narrowed after 1 year of care, and after 2 years of follow-up care, differences in A1c levels disappeared. The racial differences observed at presentation could not be explained by other important clinical characteristics (ie, age, BMI, diabetes duration, etc), and the reduction of such differences after 1 and 2 years of care occurred in a setting where important patient and provider behaviors were similar between groups.

Previous studies have found that blacks have higher A1c levels than whites at presentation, but most were unable to determine whether differences could be attributed to potential confounders, such as gender, BMI, diabetes duration, diabetes therapy, patient adherence, and/or provider management. For example, the subanalysis of type 2 diabetes patients in the Insulin Resistance Atherosclerosis Study (IRAS) database excluded insulin-treated patients, the Health ABC study investigated only patients 70 to 79 years of age, and the National Health and Nutrition Examination Survey (NHANES) III survey (1988-1994) did not adjust for diabetes therapy, patient adherence, or provider behavior. Moreover, many of these studies were performed in different sites without adjustment for potential differences in providers and access to healthcare resources. In contrast, we were able to examine a large number of patients both at initial presentation, after 1 year of follow-up, and after 2 years of follow-up in a single healthcare unit where access to resources was uniform, where quality improvement efforts were routine, and where it was possible to assess the potential contributions of demographic and clinical characteristics, patient adherence, and the pattern of provider management. Similar to the other studies, we did find racial disparities in glycemic control at presentation, but these differences dissipated during follow-up.

Racial disparities in medical care and health outcomes have been attributed to patient-related factors such as genetic differences, poor compliance, low socioeconomic conditions, limited access to health care, negative attitudes toward health and providers, and personal preferences, and/or provider factors such as clinical training, poor communication, clinical inertia, and racial (interpersonal) bias, and limitations in systems support. Clinical inertia, the provider’s perception that patients are poorly compliant with therapy, and/or poor communication are potential problems underlying inadequate provider action. Our study shows that glycemic disparities at presentation may have been caused by racial differences in patient adherence and/or provider behavior prior to care at our clinic.
contrast, during follow-up in our clinic, patient adherence and provider behavior were similar for blacks versus whites. The differences in A1c levels at presentation disappeared after 2 years of care, which may reflect the time required for the effects of improved provider intensification (and possibly improved patient adherence) to take place. These results support our previous findings that both patient adherence and provider behavior are important for glycemic control and that use of interventions that help to overcome clinical inertia and improve provider behavior can enhance glycemic control.

There was no way to fully test the hypothesis that the observed racial difference in glycemic control was caused by differences in patient adherence and/or provider behavior prior to care at our clinic. However, it was possible to explore antecedent provider behavior according to the dosage of insulin in patients who were using this agent at presentation. In such patients (1405 blacks, 69 whites), average A1c at presentation was 9.5% in blacks versus 9.0% in whites, but average insulin dosage was 0.42 units/kg/d in blacks versus 0.53 units/kg/d in whites ($P = .016$)—evidence that care prior to presentation was less intensive in blacks despite higher A1c levels.

The findings of this study suggest that with appropriate implementation of a uniform algorithm for diabetes management, racial disparities may be minimized or even eliminated. Recently, the ADA and the European Association for the Study of Diabetes (EASD) published a joint consensus statement providing an algorithm for the management of hyperglycemia in type 2 diabetes. They recommend beginning treatment with both lifestyle intervention (weight loss and increased physical activity) and metformin therapy titrated to its maximally effective dose for 1 to 2 months. If after 2 to 3 months glycosylated hemoglobin is still not at the goal of <7%, additional treatment with a basal insulin, a SU, or a thiazolidinedione should be prescribed. Titration of insulin doses should be performed frequently, based on fasting glucose (fingerstick) measurements, to reach a goal of 70 to 130 mg/dL (3.9-7.2 mmol/L). Continued titration of doses and/or addition of medications, including those not listed as second-line agents, should ensue until optimal glycemic control has been obtained. However, different algorithms and guidelines have often not succeeded in bringing patients to optimal glycemic control because they have not provided recommendations that are individualized, timely, and specific. The impact of the ADA/EASD guidelines might also be limited, as they are based on A1c levels that may not be available at the time of the visit, and because they offer a variety of choices that may not be specific enough to aid clinical decision making.

In contrast, our algorithm is driven by point-of-care glucose measurements, facilitating immediate implementation, and provide specific instructions as to which medications to add and/or how to adjust dosages. It is possible that the use of this algorithm contributed significantly to the improvements in diabetes control we observed.

Our study has some limitations. Potential differences in socioeconomic status and healthcare access could not be accounted for, but there was no association between socioeconomic factors and glycemic control in the NHANES III database, and in a multicenter managed care population potential confounding by socioeconomic differences is unlikely in our municipal population (in which most patients are poor and uninsured); a recent study in our clinic failed to show racial differences in socioeconomic factors and healthcare access. Patient dropout between the 1- and 2-year follow-up visits may have led to selection bias, but the demographic/clinical characteristics of those who returned were similar in blacks and whites. It is also possible that the characteristics of our population (predominantly urban, uninsured, and low income) may limit generalizability of the findings.

The escalation in the prevalence, complications, and healthcare costs of diabetes poses a particular burden on blacks who suffer disparities in health. Recent studies demonstrating the
persistence of racial disparities in health care and outcomes underscore the critical need for healthcare workers not only to acknowledge such differences but also to develop strategies which ameliorate these differences. These findings suggest that differences in glycemic control between blacks and whites disappear under conditions where a uniform management protocol is used, helping to overcome any tendencies toward interpersonal bias and/or clinical inertia of the providers.

Acknowledgments

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References


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Figure 1.
A1c at presentation, 1-year and 2-year follow-up stratified by race. A, A1c at presentation and after 1 year of care (n = 3542). *P < .001 for A1c difference between blacks and whites; †P = .029 for A1c difference between blacks and whites. B, A1c at presentation, after 1 year of care, and after 2 years of care in patients who returned for 2-year follow-up visits (n = 1795). **P = .029 for A1c difference between blacks and whites.
## Table 1

### Patient Characteristics

<table>
<thead>
<tr>
<th>Patient Characteristic</th>
<th>Black Follow-up (n = 3542)</th>
<th>White Follow-up (n = 1795)</th>
<th>Black Follow-up (n = 1795)</th>
<th>White Follow-up (n = 1795)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Black (n = 3324)</td>
<td>White (n = 218)</td>
<td>Black (n = 1681)</td>
<td>White (n = 114)</td>
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<tr>
<td><strong>Presentation</strong></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Age, y</td>
<td>55 (0.21)</td>
<td>56 (0.73)</td>
<td>56 (0.28)</td>
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<td>54</td>
<td>67</td>
<td>60</td>
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<tr>
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<td>32 (0.50)</td>
<td>34 (1.80)</td>
<td>33 (0.76)</td>
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<td>Diabetes duration, y</td>
<td>4.8 (0.13)</td>
<td>5.1 (0.48)</td>
<td>4.5 (0.18)</td>
<td>4.7 (0.65)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet</td>
<td>16</td>
<td>19</td>
<td>17</td>
<td>22</td>
</tr>
<tr>
<td>Oral agents</td>
<td>42</td>
<td>49</td>
<td>43</td>
<td>46</td>
</tr>
<tr>
<td>Insulin</td>
<td>42</td>
<td>32</td>
<td>40</td>
<td>33</td>
</tr>
<tr>
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<td>8.9 (0.045)</td>
<td>8.3 (0.15)</td>
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<tr>
<td><strong>1-year follow-up</strong></td>
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<td></td>
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<tr>
<td>Diet</td>
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<td>17</td>
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<td>Oral agents</td>
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<td>44</td>
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<td>50</td>
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<tr>
<td>Insulin</td>
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<tr>
<td>A1c, %</td>
<td>7.7 (0.043)</td>
<td>7.3 (0.14)</td>
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<td>Number of visits</td>
<td>5.5 (0.036)</td>
<td>5.6 (0.14)</td>
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<tr>
<td>Medication adherence, %</td>
<td>74 (0.29)</td>
<td>76 (0.99)</td>
<td>78 (0.003)</td>
<td>78 (0.011)</td>
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<tr>
<td>Frequency of intensification, %</td>
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<td>43 (2.31)</td>
<td>34 (0.008)</td>
<td>33 (0.032)</td>
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<td>Amount of intensification, %</td>
<td>50 (1.0)</td>
<td>48 (3.57)</td>
<td>47 (0.013)</td>
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<td><strong>2-year follow-up</strong></td>
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<tr>
<td>Diet</td>
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<td>A1c, %</td>
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<tr>
<td>Number of visits</td>
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<tr>
<td>Medication adherence, %</td>
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<table>
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<tr>
<th>Patient Characteristic</th>
<th>Patients With 1-Year Follow-up (n = 3542)</th>
<th>Patients With 2-Year Follow-up (n = 1795)</th>
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<tr>
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<td>Frequency of intensification, %</td>
<td>—</td>
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<tr>
<td>Amount of intensification, %</td>
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<td>73 (2.11)</td>
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BMI, body mass index.

All values represent mean (SE) or percentages.
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<tr>
<th>Patient Characteristic</th>
<th>A1c at Presentation</th>
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<th>P Value</th>
<th>A1c at 1 Year</th>
<th>Coefficient</th>
<th>P Value</th>
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<td>&lt;.001</td>
<td>0.257</td>
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<td>-0.001</td>
<td>&lt;.001</td>
<td>-0.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Oral agents</td>
<td>0.709</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Insulin</td>
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<td>-0.130</td>
<td>&lt;.001</td>
<td>-0.130</td>
<td>&lt;.001</td>
<td>-0.130</td>
<td>&lt;.001</td>
<td>-0.130</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Year of presentation</td>
<td>0.709</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>A1c at presentation, %</td>
<td>0.709</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
<td>0.377</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Number of follow-up visits</td>
<td>-0.014</td>
<td>-0.014</td>
<td>&lt;.001</td>
<td>-0.014</td>
<td>&lt;.001</td>
<td>-0.014</td>
<td>&lt;.001</td>
<td>-0.014</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Medication adherence, %</td>
<td>0.932</td>
<td>0.932</td>
<td>&lt;.001</td>
<td>0.932</td>
<td>&lt;.001</td>
<td>0.932</td>
<td>&lt;.001</td>
<td>0.932</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

**Table 2**

Effect of A1c: Multivariable Linear Regression

BMI, body mass index.

a Proportion of patient visits with random plasma glucose ≥ 150 mg/dL (8.3 mmol/L) for which the provider intensified diabetes therapy at all (since preliminary studies of individuals with normal glucose tolerance revealed no individuals with random plasma glucose exceeding 140 mg/dL [7.8 mmol/L], intensification of therapy was considered indicated if random plasma glucose exceeded 150 mg/dL [8.3 mmol/L]).

b Average amount of intensification done for the visits during which intensification was indicated (random plasma glucose ≥ 150 mg/dL [8.3 mmol/L]).