Is Glycemic Control Improving in U.S. Adults?

THOMAS J. HOERGER, PHD¹
JOEL E. SEGEL, BA¹

EDWARD W. GREGG, PHD² JINAN B. SAADDINE, MD²

OBJECTIVE— The purpose of this study was to examine whether glycemic control has improved in recent years among individuals with diagnosed diabetes.

RESEARCH DESIGN AND METHODS — We examined trends in A1C levels for adults with diagnosed diabetes using three consecutive waves of the National Health and Nutrition Examination Survey (NHANES): 1999-2000, 2001-2002, and 2003-2004. We estimated mean A1C levels and the proportion with A1C <7.0, <8.0, and <9.0%. We used multivariate regression to test whether A1C levels differed by NHANES wave after controlling for other factors. Multivariate dichotomous logistic regression and predictive margins were used to test whether the percentages of individuals with diabetes in selected A1C intervals differed by NHANES wave.

RESULTS — Mean A1C levels among individuals with diagnosed diabetes declined from 7.82% in 1999–2000 to 7.47 and 7.18% in 2001–2002 and 2003–2004, respectively. After controlling for demographics and diabetes duration, A1C levels were 0.308 (P=0.20) and 0.511 (P=0.03) percentage points lower in 2001–2002 and 2003–2004, respectively, than in 1999–2000. The logistic results indicated corresponding improvements over time: the predictive margin for having A1C <7.0% increased from 37.0% in 1999–2000 to 49.7% in 2001–2002 and to 55.7% in 2003–2004.

CONCLUSIONS — Glycemic control improved between 1999 and 2004. This trend may represent an important improvement in diabetes care and is encouraging for future reduction of diabetes-related complications.

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ajor randomized controlled trials conducted during the 1990s demonstrated the importance of tightly and consistently managing A1C levels among type 1 and type 2 diabetic patients (1,2). A follow-up of the Diabetes Control and Complications Trial (DCCT) reaffirmed the value of glycemic control because microvascular complications remained lower among intervention participants 7 years after the trial concluded (3). These and other studies highlighted the central role of glycemic control in the management of diabetes. Accordingly,

professional associations, including the American Association of Clinical Endocrinologists (AACE), the American Diabetes Association (ADA), and the European Association for the Study of Diabetes (EASD), established clinical guidelines in the range of 6.5–7.0% to motivate health professionals and patients to consistently manage blood glucose levels (4,5). The National Diabetes Quality Improvement Alliance, a private-public entity, developed national performance measures to monitor glycemic control and other diabetes care processes and outcomes.

From ¹RTI International, Research Triangle Park, North Carolina; and the ²Centers for Disease Control and Prevention, Atlanta, Georgia.

Address correspondence and reprint requests to Thomas J. Hoerger, Director, RTI-UNC Center of Excellence in Health Promotion Economics, RTI International, 3040 Cornwallis Rd., P.O. Box 12194, Research Triangle Park, NC 27709. E-mail: tjh@rti.org.

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Abbreviations: DCCT, Diabetes Control and Complications Trial; NHANES, National Health and Nutrition Examination Survey; UKPDS, UK Prospective Diabetes Study.

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

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Despite the emphasis on glycemic control, evidence of improvement in A1C control over time remains scant. In a recent study comparing nationally representative data from 1999-2002 with comparable data from 1988–1994, the mean A1C level declined from 7.8 to 7.7% (not significant at the 5% level) among adults with self-reported diabetes (6). The study found some evidence suggesting an improvement in poorly controlled A1C; the percentage of patients with A1C >9.0% decreased from 24.5 to 20.6%, but this decline was not significant at the 5% level. Other studies have also shown little or no time trend in A1C levels (7,8); one study (8) reported that the rate of glycemic control (A1C <7.0%) fell from 44.5% in 1988–1994 to 35.8% in 1999-2000. Given the attention on glycemic control in clinical disease management and public health programs during the past decade, these observations are both concerning and surprising. In this study, we examined recent national trends in glycemic control among adults with diagnosed diabetes.

RESEARCH DESIGN AND

METHODS — We examined data from the National Health and Nutrition Examination Survey (NHANES), the same data source used in the previous studies (6–8). NHANES is a nationally representative, population-based survey designed to collect information on the health and nutrition of the U.S. civilian noninstitutionalized population. NHANES is now an ongoing survey, with results released in 2-year waves. Survey details are described elsewhere (9–11). Participants answer a household interview, and most complete clinical examinations in a mobile examination center.

We focused on the 1999–2000, 2001–2002, and 2003–2004 NHANES waves. This focus allowed us to use the most recent data to examine recent trends in glycemic control. We limited our analysis to adults (aged ≥18 years) who reported a previous diagnosis of diabetes by a health care professional and who received an A1C measurement.

Blood specimens were processed, stored, and shipped to the Diabetes Diagnostic Laboratory at the University of Mis-

Table 1—Characteristics among individuals who have been told they have diabetes and who received an A1C measurement by NHANES wave

	NHANES 1999–2000	NHANES 2001–2002	NHANES 2003–2004
n	403	448	482
Age (years)	59.0 ± 0.8	57.4 ± 1.2	59.7 ± 0.8
18–44 (%)	15.6 ± 2.7	22.1 ± 3.4	13.2 ± 1.6
45–64 (%)	45.8 ± 3.3	44.2 ± 3.4	44.9 ± 2.9
65+(%)	38.6 ± 2.1	33.8 ± 3.4	41.9 ± 3.3
Sex (%)	30.0 = 2.1	33.0 = 3.1	11.7 = 3.3
Female	48.4 ± 3.7	49.7 ± 2.0	52.4 ± 1.4
Male	51.6 ± 3.7	50.3 ± 2.0	47.6 ± 1.4
Race/ethnicity (%)	31.0 = 3	30.3 = 2.0	11.0 = 1.1
White non-Hispanic	61.2 ± 6.2	63.4 ± 3.9	70.8 ± 5.4
Black non-Hispanic	15.8 ± 3.8	14.4 ± 3.0	14.0 ± 2.9
Hispanic	17.0 ± 6.5	15.1 ± 4.5	11.3 ± 3.6
Other	5.9 ± 3.3	7.0 ± 2.4	3.8 ± 1.2
Education (%)	0.7 — 0.0	,	0.0 - 2.2
<high school<="" td=""><td>43.2 ± 3.9</td><td>30.5 ± 2.7</td><td>29.8 ± 2.3</td></high>	43.2 ± 3.9	30.5 ± 2.7	29.8 ± 2.3
High school	25.7 ± 4.5	24.7 ± 2.2	24.3 ± 2.2
>High school	31.1 ± 4.7	44.8 ± 3.2	45.9 ± 2.7
BMI (kg/m ²)	32.3 ± 0.6	31.5 ± 0.5	32.2 ± 0.7
<30 (%)	45.8 ± 4.4	48.7 ± 4.1	42.8 ± 4.0
≥30 (%)	54.2 ± 4.4	51.3 ± 4.1	57.2 ± 4.0
Income (%)			
<\$20,000	36.3 ± 4.1	28.8 ± 3.4	28.7 ± 2.8
\$20,000+	63.7 ± 4.1	71.2 ± 3.4	71.3 ± 2.8
Insurance (%)	87.0 ± 2.9	91.3 ± 1.8	89.7 ± 1.5
Duration (%)			
<5 years	34.3 ± 3.6	37.5 ± 3.1	33.7 ± 2.8
5–14 years	33.1 ± 3.0	35.0 ± 3.0	39.1 ± 2.5
15+ years	32.6 ± 3.0	27.3 ± 2.4	27.0 ± 2.8
Smoker (%)			
Current	16.4 ± 2.6	22.6 ± 2.0	20.4 ± 2.4
Former	33.2 ± 3.7	31.4 ± 2.1	34.6 ± 2.4
Never	50.5 ± 4.9	46.0 ± 2.4	45.0 ± 2.9
Diabetes treatment (%)			
Diabetes pills only	53.2 ± 3.6	56.6 ± 2.4	58.9 ± 3.6
Insulin only	16.2 ± 2.2	16.4 ± 3.5	13.6 ± 2.0
Pills and insulin	10.7 ± 3.5	9.5 ± 1.8	9.2 ± 1.9
Diet only	19.9 ± 3.7	17.5 ± 2.7	18.3 ± 2.8

Data are means ± SEM.

souri–Columbia for analysis of A1C, using Primus CLC330 and Primus CLC385. The system was standardized to the reference method used in the DCCT. We included data from 403, 448, and 482 individuals in, respectively, the 1999–2000, 2001–2002, and 2003–2004 waves.

We estimated mean A1C levels for individuals reporting diagnosed diabetes for each of three NHANES data waves, as well as the percentages of the population with A1C <7.0, <8.0, and <9.0%. We applied multivariate regression to test whether A1C levels differed among NHANES waves after controlling for age,

sex, race/ethnicity, education, BMI, insurance status, income, and duration of diabetes. We estimated a second specification that also included treatment variables (diabetes pills only, insulin only, and both pills and insulin; the omitted treatment variable was diet only).

We used multivariate dichotomous logistic regression to test whether the percentage of individuals with A1C <7.0, <8.0, and <9.0% differed significantly in the 2001–2002 and 2003–2004 waves relative to the 1999–2000 wave. These cutoffs can be interpreted as different benchmarks for glycemic control. We also used multivariate logistic regression

analyses to compute predictive margins (12). The predictive margin for a wave represents the average predicted response if the entire sample (i.e., everyone in the 1999–2000, 2001–2002, and 2003–2004 waves) had been in that wave. Results were judged to be significant if P < 0.05. We conducted statistical analyses using Stata 9.2 and SAS-callable SUDAAN (version 9.0.1) in Linux (version 9.1.3) to produce point estimates and standard errors that used sampling weights to account for the complex survey sampling design.

RESULTS — The characteristics of individuals reporting diabetes and receiving an A1C measurement were relatively consistent among the NHANES waves (Table 1). Education was the only characteristic with significant changes between 1999–2000 and the last two waves.

Mean A1C levels among study participants with diagnosed diabetes were 7.82% in 1999–2000, 7.47% in 2001– 2002, and 7.18% in 2003-2004 (Table 2). Improvements in mean A1C levels were observed for most subgroups of the population during the later waves, with significant univariate reductions for individuals aged ≥65 years, women, all racial/ethnic groups except Hispanic, those with more than a high school education, those with BMI \leq 30 and BMI \geq 30 kg/m², those of both income levels, those with insurance, those with diabetes duration < 5 years and between 5 and 14 years, those who formerly or never smoked, and those treated with diabetes pills only or diet only. Only Hispanics had a higher mean A1C in 2003-2004 than in 1999-2000.

The A1C interval data show a corresponding shift to the left (Table 2 and Fig. 1). The percentage of the overall population with A1C <7.0% increased from 36.9% in 1999-2000 to 49.4% in 2001-2002 and to 56.8% in 2003-2004. Improvements were observed for every subgroup of the population shown in Table 2; many but not all of the subgroup improvements reached statistical significance in 2003–2004. The distribution by A1C interval in Fig. 1 shows that the percentage of individuals with A1C <6% increased from 12.9% in 1999-2000 to 21.8% in 2003-2004. In addition, poor glycemic control (A1C > 9.0%) was lower in 2003–2004 than in previous waves (12.4 vs. 21.0% in 1999-2000 and 17.8% in 2001–2002).

The multivariate regression results in-

Table 2—Mean A1C and the proportion of individuals with A1C <7.0% by NHANES wave

	A1C		A1C < 7%			
	NHANES 1999–2000	NHANES 2001–2002	NHANES 2003–2004	NHANES 1999–2000	NHANES 2001–2002	NHANES 2003–2004
n	403	448	482	403	448	482
Overall	7.82 ± 0.17	7.47 ± 0.16	7.18 ± 0.11 *	$36.9\% \pm 4.4$	49.4 ± 3.6*	$56.8 \pm 3.5*$
Age						
18–44 years	8.71 ± 0.82	7.68 ± 0.31	7.22 ± 0.24	29.7 ± 13.0	42.9 ± 9.1	48.5 ± 5.3
45–64 years	7.81 ± 0.19	7.68 ± 0.25	7.59 ± 0.18	40.0 ± 6.6	47.5 ± 4.2	48.5 ± 5.8
65+ years	7.49 ± 0.16	$7.04 \pm 0.12*$	$6.72 \pm 0.07*$	36.2 ± 6.0	$56.3 \pm 4.4*$	$68.3 \pm 2.7*$
Sex						
Female	7.91 ± 0.28	7.36 ± 0.16	$7.04 \pm 0.10*$	37.1 ± 5.9	$52.3 \pm 4.0*$	$58.2 \pm 4.6*$
Male	7.75 ± 0.14	7.57 ± 0.21	7.33 ± 0.23	36.7 ± 4.7	46.6 ± 4.4	$55.2 \pm 4.5*$
Race/ethnicity						
White non-Hispanic	7.52 ± 0.25	7.23 ± 0.14	6.90 ± 0.11 *	41.5 ± 6.7	53.3 ± 4.2	$63.3 \pm 4.3*$
Black non-Hispanic	8.26 ± 0.21	7.82 ± 0.22	7.61 ± 0.21 *	28.5 ± 4.0	$44.2 \pm 4.2*$	44.4 ± 5.0 *
Hispanic	8.10 ± 0.17	7.71 ± 0.36	8.36 ± 0.34	35.2 ± 5.9	47.1 ± 5.1	37.2 ± 6.2
Other	9.01 ± 0.25	8.40 ± 0.65	$7.15 \pm 0.18*$	16.9 ± 7.8	30.3 ± 15.0	40.6 ± 12.8
Education						
<high school<="" td=""><td>7.96 ± 0.13</td><td>7.57 ± 0.27</td><td>7.57 ± 0.29</td><td>30.5 ± 3.9</td><td>$51.5 \pm 5.2*$</td><td>43.7 ± 5.5</td></high>	7.96 ± 0.13	7.57 ± 0.27	7.57 ± 0.29	30.5 ± 3.9	$51.5 \pm 5.2*$	43.7 ± 5.5
High school	7.32 ± 0.27	7.47 ± 0.16	7.01 ± 0.14	51.6 ± 7.0	47.2 ± 5.2	58.8 ± 5.4
>High school	8.05 ± 0.41	7.40 ± 0.22	$7.01 \pm 0.12*$	33.9 ± 7.4	49.2 ± 4.3	$64.2 \pm 4.7*$
BMI						
$BMI < 30 \text{ kg/m}^2$	8.13 ± 0.31	7.48 ± 0.28	7.34 ± 0.15 *	32.8 ± 7.7	$53.9 \pm 5.0*$	50.6 ± 5.1
BMI \geq 30 kg/m ²	7.62 ± 0.11	7.46 ± 0.13	7.06 ± 0.15 *	38.8 ± 4.9	45.8 ± 4.7	$61.3 \pm 4.5*$
Income						
<\$20,000	8.18 ± 0.31	$7.22 \pm 0.20*$	$7.23 \pm 0.19*$	33.7 ± 4.9	$54.8 \pm 4.3*$	$55.4 \pm 5.9*$
≥\$20,000	7.66 ± 0.17	7.55 ± 0.19	$7.16 \pm 0.12*$	38.4 ± 6.4	47.6 ± 4.1	$57.3 \pm 3.8*$
Insurance						
Uninsured	8.55 ± 0.70	8.44 ± 0.51	7.54 ± 0.38	44.3 ± 9.6	37.8 ± 6.8	46.1 ± 12.2
Insured	7.70 ± 0.15	7.35 ± 0.15	$7.13 \pm 0.12*$	35.6 ± 5.0	$50.8 \pm 3.7*$	$58.1 \pm 3.8*$
Duration						
<5 years	7.94 ± 0.49	7.09 ± 0.17	$6.74 \pm 0.20*$	40.7 ± 7.1	57.8 ± 5.0 *	$74.2 \pm 5.3*$
5–14 years	7.94 ± 0.13	7.88 ± 0.32	7.49 ± 0.15 *	34.2 ± 4.2	41.3 ± 5.9	50.0 ± 4.5 *
15+ years	7.58 ± 0.19	7.46 ± 0.22	7.26 ± 0.15	35.7 ± 7.0	48.2 ± 5.5	44.6 ± 6.5
Smoker						
Current	7.99 ± 0.33	7.49 ± 0.31	7.48 ± 0.32	28.9 ± 10.5	50.0 ± 8.0	50.2 ± 6.1
Former	7.47 ± 0.14	7.18 ± 0.26	7.00 ± 0.13 *	46.6 ± 4.0	55.7 ± 8.0	60.3 ± 5.4 *
Never	8.00 ± 0.28	7.65 ± 0.20	7.17 ± 0.14 *	33.2 ± 5.0	44.6 ± 4.9	57.3 ± 4.8 *
Diabetes treatment						
Diabetes pills only	7.80 ± 0.18	7.37 ± 0.17	$7.26 \pm 0.07*$	34.1 ± 4.9	$50.3 \pm 4.1*$	55.3 ± 3.8 *
Insulin only	8.70 ± 0.40	8.15 ± 0.31	7.79 ± 0.25	24.2 ± 10.4	30.4 ± 6.7	33.2 ± 9.6
Pills and insulin	8.32 ± 0.20	8.22 ± 0.45	7.92 ± 0.16	13.7 ± 6.9	26.1 ± 12.2	$36.3 \pm 7.0^{*}$
Diet only	7.04 ± 0.31	6.73 ± 0.43	$6.07 \pm 0.18*$	62.6 ± 9.8	77.2 ± 7.1	89.7 ± 4.8*

Data are means \pm SEM. *Significantly different from NHANES 1999–2000 at P < 0.05.

dicate that mean A1C levels were 0.308 percentage points (P=0.20) lower in 2001–2002 and 0.511 percentage points (P=0.03) lower in 2003–2004 than in 1999–2000 (Table 3; no treatment variables included). After controls for other factors, the differences in waves were less than those in univariate means. However, the mean A1C level for the 2003–2004 wave was still significantly lower than that for the 1999–2000 waves. The coefficients for 2001–2002 and 2003–2004 were virtually unchanged when treatment

variables were included in the regression. The treatment effects were associated with higher A1C levels, probably because the treatments were prescribed when A1C was not controlled by diet only.

The multivariate logistic results indicate corresponding improvements over time in the benchmarks for glycemic control (Table 4). The odds ratios (ORs) for 2001–2002 and 2003–2004 were always >1.0, and both the difference between the 2001–2002 and 1999–2000 waves (P=0.04) and the difference between the

2003–2004 and 1999–2000 waves (P=0.002) were significant for A1C <7.0%. The difference between the 2003–2004 wave and the 1999–2000 wave was also significant for A1C <8.0% (P=0.004). The OR for the 2003–2004 wave for A1C <9.0% was not significant (P=0.12), possibly because the 1999–2000 predictive margin was already high. The predictive margins convey the scale of the changes across NHANES waves; for all individuals in the sample and with no treatment variables included, the predictive

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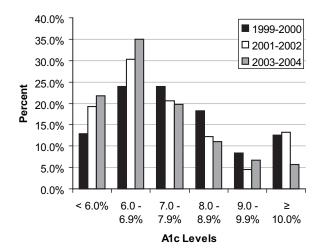


Figure 1—A1C distribution by NHANES wave.

margin for having A1C <7.0% increased from 37.0% in 1999–2000 to 49.7% in 2001–2002 and to 55.7% in 2003–2004. Including treatment variables had little effect on the ORs and predictive margins.

CONCLUSIONS — A previous report using NHANES data indicated that processes of diabetes care improved between 1988–1994 and 1999–2002 (6). Much smaller changes in intermediate outcomes, such as A1C and blood pressure, accompanied these improvements; reported changes in A1C were small and not significant. Harris et al. (13) reported

a mean A1C of 7.6% for NHANES III (1988–1994), and Saaddine et al. (6), using a slightly different data cut, reported that mean A1C fell from 7.8% in NHANES III to 7.7% in NHANES 1999–2002. Another study reported that the percentage of individuals achieving optimal control fell between NHANES III and NHANES 1999–2000 (8). From a public health and clinical perspective, these findings were disappointing because glycemic control had long been emphasized as a key tenet in diabetes care.

In this context, our finding that A1C levels decreased significantly between

1999-2000 and 2003-2004 may indicate that diabetes care improved dramatically between 1999 and 2004. Several factors could explain the decrease in A1C levels in recent years. After publication of the DCCT and UK Prospective Diabetes Study (UKPDS) results, clinical guidelines for diabetes care were strengthened, with adoption of lower goals for A1C. Health plans embraced multifaceted disease management programs to improve care and reduce A1C levels. These plans were targeted to health systems and providers as well as to patients with diabetes. National public health awareness programs, including those promoted by the National Diabetes Education Program, the National Diabetes Quality Improvement Alliance, and several professional organizations (ADA, EASD, and AACE), emphasized improved glycemic control along with cardiovascular risk factor control. Improved measurement and reporting of diabetes care in health plans may have further motivated glycemic control efforts. At the same time, new drugs became available in the U.S., including metformin (available by brand name since 1995 and as a generic drug since 2002) and the thiazolidinediones (available in current brands since 1999). Patient adherence to therapy may have increased, possibly due to improved diabetes education and/or fewer side effects from medi-

Table 3—Multivariate regression results: effects of demographic factors and NHANES wave on A1C

	No treatment	variables included	Treatment variables included		
	Coefficient (SE)	95% CI	Coefficient (SE)	95% CI	
Age	-0.024 (0.008)*	-0.039 to -0.008	-0.022 (0.007)*	-0.036 to -0.008	
Black non-Hispanic	0.553 (0.188)*	0.175 to 0.931	0.495 (0.171)*	0.152 to 0.839	
Hispanic	0.424 (0.267)	-0.114 to 0.963	0.500 (0.264)	-0.032 to 1.032	
Other	0.660 (0.284)*	0.087 to 1.232	0.575 (0.263)*	0.044 to 1.105	
Female	-0.168(0.150)	-0.471 to 0.135	-0.156(0.137)	-0.431 to 0.119	
<high school<="" td=""><td>0.263 (0.213)</td><td>-0.165 to 0.692</td><td>0.208 (0.194)</td><td>-0.182 to 0.599</td></high>	0.263 (0.213)	-0.165 to 0.692	0.208 (0.194)	-0.182 to 0.599	
High school	0.041 (0.188)	-0.338 to 0.421	0.049 (0.169)	-0.292 to 0.39	
BMI	-0.020 (0.013)	-0.046 to 0.005	-0.026 (0.011)*	-0.049 to -0.003	
Insured	-0.519(0.343)	-1.21 to 0.171	-0.624(0.329)	-1.286 to 0.038	
Income <\$20K	0.075 (0.158)	-0.244 to 0.394	0.079 (0.147)	-0.217 to 0.375	
Duration 5–14 years	0.626 (0.205)*	0.213 to 1.04	0.428 (0.205)*	0.015 to 0.84	
Duration 15+ years	0.472 (0.216)*	0.036 to 0.908	0.099 (0.229)	-0.363 to 0.561	
Diabetes pills only		_	0.949 (0.182)*	0.582 to 1.317	
Insulin only		_	1.658 (0.237)*	1.18 to 2.137	
Diabetes pills and insulin		_	1.564 (0.263)*	1.033 to 2.095	
NHANES 2001–2002	-0.308 (0.234)	-0.78 to 0.164	-0.317(0.194)	-0.708 to 0.073	
NHANES 2003-2004	-0.511 (0.222)*	-0.959 to -0.063	-0.500 (0.164)*	-0.830 to -0.169	
Constant	9.662 (1.042)*	7.563 to 11.761	9.123 (0.902)*	7.305 to 10.941	
n	1	,132		1,129	
R^2	C	0.118	(0.196	

^{*}Significant at P < 0.05.

Table 4—Multivariate logistic results: ORs and predictive margins by NHANES wave

	No treatment variables included		Treatment variables included	
	ORs	Predictive margin (%)	ORs	Predictive margin (%)
A1C <7.0%				
1999-2000	1.00	37.0 (4.6)	1.00	36.8 (3.6)
2001-2002	1.75 (0.45)*	49.7 (3.2)	1.91 (0.43)*	50.0 (3.1)
2003-2004	2.27 (0.58)*	55.7 (3.4)	2.52 (0.57)*	55.8 (3.4)
A1C <8.0%				
1999-2000	1.00	61.9 (3.8)	1.00	62.2 (3.2)
2001-2002	1.60 (0.40)	71.4 (2.9)	1.61 (0.37)*	71.3 (2.9)
2003-2004	2.00 (0.46)*	75.4 (2.3)	2.04 (0.40)*	75.3 (2.1)
A1C < 9.0%				
1999-2000	1.00	81.3 (3.4)	1.00	81.4 (3.0)
2001-2002	1.11 (0.37)	82.6 (2.7)	1.09 (0.35)	82.5 (2.8)
2003-2004	1.61 (0.48)	86.9 (1.5)	1.64 (0.44)	87.0 (1.5)

Data are ORs (SE) or predictive margin (SE). Results control for age, sex, race/ethnicity, education, BMI, insurance, income, and duration of diabetes. Treatment variables include use of diabetes pills only, insulin only, and insulin and diabetes pills; the treatment variable omitted is diet only. *ORs are significantly different from 1999–2000 at P < 0.05.

cation. Medicare also increased coverage for diabetes-related supplies during this period. Whether these or other factors can explain the decrease in mean A1C levels is a topic for future research.

Our findings will be strengthened if they are confirmed by analyses of other datasets and future NHANES waves. Nonetheless, our analysis has certain strengths. The NHANES sample is nationally representative and has a sufficient sample size to detect differences between time periods. Uniform methods were used for the diabetes section of NHANES, and A1C values were assessed by a single laboratory with close attention to quality of control and measurement. Our results are significant even after we controlled for demographic variables in the multivariate analysis. The fact that mean A1C was lower in 2001-2002 than in 1999-2000 and then lower still in 2003-2004, both for the entire sample and for most demographic groups (Table 2), further suggests that there was a real trend in A1C levels.

Our analysis has at least two potential limitations. First, diabetes status was self-reported and, in the absence of a clinical patient history, some individuals in the sample might not have had diabetes and would thus be expected to have A1C levels near normal. However, previous studies have suggested that the self-reported diabetes variable has high reproducibility and validity (14,15), and there is little reason to believe that errors in self-reporting would increase over time. Second, although respondents reported time since diagnosis of diabetes, information on di-

abetes onset is not available. It is possible that earlier detection of diabetes in recent years could bias the results toward lower mean A1C. On the other hand, we found a time trend toward lower A1C levels for all categories of years of diagnosed duration, and the percentage of individuals with duration of diagnosed diabetes <5 years changed little among NHANES waves. We also controlled for time since diagnosis in the regression analyses. However, these results do not fully resolve the issue of whether diagnosis has been occurring closer to onset.

Our findings are encouraging for the long-term outcomes of patients with diabetes. Hazard model estimates based on 10-year outcomes observed in UKPDS data suggest that a mean 0.511 percentage point decrease in glycemic level would, if maintained, produce a 10.7% reduction in diabetes complications (16) and an important improvement in diabetes morbidity with accompanying reductions in complication costs. However, these results should not breed complacency about the need for further efforts to achieve glycemic control. Although averages are improving, many individuals still are not meeting the optimal control targets, and A1C levels and rates of suboptimal control are especially high in some demographic groups. Moreover, our estimated reduction in A1C is only about half of the average 0.9 percentage point reduction achieved by intensive glycemic control in the UKPDS relative to conventional control (2), suggesting that more improvement is possible. Perhaps the best

response to our results is a continuing effort to make even more progress in the future both for A1C and for other risk factors for diabetes complications.

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References

- 1. The Diabetes Control and Complications Trial Research Group: The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 329:977–986, 1993
- UK Prospective Diabetes Study (UKPDS)
 Group: Intensive blood-glucose control
 with sulphonylureas or insulin compared
 with conventional treatment and risk of
 complications in patients with type 2 di abetes (UKPDS 33). Lancet 352:837–853,
 1998
- 3. The Writing Team for the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Research Group: Effect of intensive therapy on the microvascular complications of type 1 diabetes. *JAMA* 287:2563–2569, 2002
- American Association of Clinical Endocrinologists: American College of Endocrinology consensus statement on guidelines for glycemic control. *Endocr Pract* 8 (Suppl. 1):5–11, 2002
- 5. Nathan DM, Buse JB, Davidson MB, Heine RJ, Holman RR, Sherwin R, Zinman B: Management of hyperglycemia in type 2 diabetes: a consensus algorithm for the initiation and adjustment of therapy: a consensus statement from the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care 29:1963–1972, 2006
- Saaddine JB, Cadwell B, Gregg EW, Engelgau MM, Vinicor F, Imperatore G, Venkat Narayan KM: Improvements in diabetes processes of care and intermediate outcomes: United States, 1988–2002. Ann Intern Med 144:465–474, 2006
- Saydah SH, Fradkin J, Cowie CC: Poor control of risk factors for vascular disease among adults with previously diagnosed diabetes. *JAMA* 291:335–342, 2004
- 8. Koro CE, Bowlin SJ, Bourgeois N, Fedder DO: Glycemic control from 1988 to 2000 among U.S. adults with diagnosed type 2 diabetes. *Diabetes Care* 27:17–20, 2004
- 9. National Center for Health Statistics: *NHANES* 1999–2000. Hyattsville, MD, U.S. Department of Health and Human Service, Centers for Disease Control and Prevention, National Center for Health Statistics. Available from www.cdc.gov/

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- nchs/about/major/nhanes/nhanes 99_00.htm. Accessed 17 April 2007
- 10. National Center for Health Statistics: *NHANES* 2001–2002. Hyattsville, MD, U.S. Department of Health and Human Service, Centers for Disease Control and Prevention, National Center for Health Statistics. Available from www.cdc.gov/nchs/about/major/nhanes/nhanes01_02. htm. Accessed 17 April 2007
- 11. National Center for Health Statistics. *NHANES* 2003–2004. Hyattsville, MD, U.S. Department of Health and Human Service, Centers for Disease Control and Prevention, National Center for Health
- Statistics. Available from www.cdc.gov/nchs/about/major/nhanes/nhanes03_04.htm. Accessed on 17 April 2007
- 12. Graubard BI, Korn EL: Predictive margins. *Biometrics* 55:652–659, 1999
- 13. Harris MI, Eastman RC, Cowie CC, Flegal KM, Eberhardt MS: Racial and ethnic differences in glycemic control of adults with type 2 diabetes. *Diabetes Care* 22:403–408, 1999
- 14. Stein AD, Courval JM, Lederman RI, Shea S: Reproducibility of responses to telephone interviews: demographic predictors of discordance in risk factor sta-

- tus. Am J Epidemiol 141:1097-1105, 1995
- 15. Bowlin SJ, Morrill BD, Nazfiger AN, Lewis C, Pearson TA: Reliability and changes in validity of self-reported cardiovascular disease risk factors using dual response: the behavioral risk factor survey. *J Clin Epidemiol* 49:511–517, 1996
- Stratton IM, Cull CA, Adler AI, Matthews DR, Neil HA, Holman RR: Additive effects of glycaemia and blood pressure exposure on risk of complications in type 2 diabetes: a prospective observational study (UKPDS 75). Diabetologia 49:1761–1769, 2006