HbA_{1c} Levels Among American Indian/Alaska Native Adults

SUSAN S. GILLILAND, PHD[1]
JANETTE S. CARTER, MD[2,3]
BETTY SKIPPER, PHD[4]
KELLY J. ACTON, MD[5]

OBJECTIVE — Type 2 diabetes is a major public health problem among many American Indian/Alaska Native communities. Elevated levels of HbA_{1c} have been observed in younger American Indian/Alaska Native adults. The objectives of this study were: *1*) to determine whether HbA_{1c} levels were elevated among younger American Indian/Alaska Native adults nationally and, if so, *2*) to determine the relationship between HbA_{1c} levels and age due to treatment type, BMI, renal disease, duration of diabetes, survival, or a poor diabetes health care index.

RESEARCH DESIGN AND METHODS — The national Indian Health Service Diabetes Care and Outcomes Audit was completed for a total of 11,419 American Indian/Alaska Native adults with type 2 diabetes from tribes across the U.S. in 1998. Glucose control was assessed by HbA_{1c}. BMI, diabetes duration, treatment type, and proteinuria were assessed from the Diabetes Care and Outcomes Audit data. To assess diabetes quality of care, an index was developed from six standard of care Diabetes Care and Outcomes Audit variables.

RESULTS — We found HbA_{1c} level decreased with increasing age. HbA_{1c} levels were 9.2, 8.9, 8.8, 8.3, and 7.8 for ages 18–39, 40–49, 50–59, 60–69, and ≥70 years, respectively ($P < 0.0001$). This inverse relationship was not accounted for by differences in BMI, diabetes duration, treatment type, proteinuria, or health care index.

CONCLUSIONS — Among American Indian/Alaska Native adults, HbA_{1c} levels were highest in the youngest age-group. With increasing numbers of young American Indian/Alaska Native adults with diabetes, poorer glucose control is expected to bring concomitant increased morbidity and mortality unless more effective and efficient interventions are developed to improve glucose control among young American Indian/Alaska Native adults.

Diabetes Care 25:2178–2183, 2002

Type 2 diabetes is a leading health problem for many American Indian/Alaska Native communities, with high and increasing prevalence, morbidity, and mortality (1–3). The expanding burden of this disease is shown by a rise in morbidity, including a high prevalence of end-stage renal disease (4), and a rapid increase in diabetes-related mortality (3) among American Indian/Alaska Native people. With the increasing prevalence and shift in the onset of diabetes to younger ages (5,6), the morbidity and mortality associated with type 2 diabetes can be expected to continue to escalate.

Decreased morbidity and mortality associated with type 2 diabetes can be achieved with tight glycemic control (7). Although the Indian Health Service (IHS) has reported strides made toward improving glycemic control over the past decade, recent studies reveal a worrisome direction of higher HbA_{1c} levels in younger American Indian/Alaska Native adults, particularly among those residing in the Southwestern region of the U.S. (8,9). The Strong Heart Study, a longitudinal study of cardiovascular disease involving 15 tribes, found that younger individuals, women, and those taking insulin or oral hypoglycemic agents had the worst glucose control (9). The Native American Diabetes Project, a study of eight tribes in New Mexico, found that participants aged <55 years had the highest adjusted HbA_{1c} levels (9.5%), whereas those aged ≥65 years had the lowest levels (7.8%) (8).

Higher HbA_{1c} levels suggest an increased risk for the development of complications in the younger age-groups. However, it is possible that the higher HbA_{1c} levels observed among the younger age-groups in these studies can be explained by a survival bias because older people with poorly controlled diabetes may die at younger ages. The higher HbA_{1c} levels observed in younger American Indian/Alaska Native adults may be the result of differences among age-groups in treatment strategies or visit frequency. The presence of renal disease and, consequently, increased circulating insulin levels could contribute to better glucose control in the older age-groups. It has also been suggested that younger American Indian/Alaska Native adults are less compliant with treatment and therefore have higher HbA_{1c} levels (9). Curtin et al. (10) have reported that conformity to medical care improves with age; however, no studies have been conducted on the frequency of conformity or health system factors that influence medical care, HbA_{1c} levels, and age in American Indian/Alaska Native populations.

The purpose of this cross-sectional study was to determine whether younger American Indian/Alaska Native adults with type 2 diabetes exhibit higher HbA_{1c}

From the [1]University of Southern California, Department of Preventive Medicine, Los Angeles, California; the [2]New Mexico Veterans Health Care System, Albuquerque, New Mexico; the [3]Department of Internal Medicine, University of New Mexico School of Medicine, Albuquerque, New Mexico; the [4]Department of Family and Community Medicine, University of New Mexico School of Medicine, Albuquerque, New Mexico; and the [5]Indian Health Service National Diabetes Programs, Albuquerque, New Mexico.

Address correspondence and reprint requests to Susan S. Gilliland, PhD, University of Southern California, Keck School of Medicine, Department of Preventive Medicine, 1540 Alcazar St., CHP 218, Los Angeles, CA 90033-9010. E-mail: sgillila@usc.edu.

Received for publication 1 January 2002 and accepted in revised form 2 September 2002.

J.S.C. is deceased.

Abbreviations: IHS, Indian Health Service.

The opinions expressed in this article are those of the authors and do not necessarily reflect the views of the Indian Health Service.

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

levels nationally and, if so, to determine whether HbA_{1c} levels are due to BMI, duration of diabetes, treatment type, proteinuria status, or poorer diabetes health care index.

RESEARCH DESIGN AND METHODS

Data collection

The IHS Diabetes Care and Outcomes Audit is conducted yearly through medical chart reviews. Methods for the Diabetes Care and Outcomes Audit have been described previously (11–13). Briefly, IHS and tribal clinic facilities are encouraged to maintain diabetes registries of all individuals with diabetes. Selected clinical variables and interventions are measured annually using a systematic random sample of charts at each facility. The methods for randomization and sample size collection have been reported elsewhere (13). In short, a sample size is chosen for each facility that is sufficient to provide an estimate within ±10% of the true rate of adherence for each facility, with a confidence level of ≥90% (14). The regional diabetes coordinators and professional staff members, trained by the coordinators, perform the Diabetes Care and Outcomes Audit. A uniform set of definitions is used throughout the U.S. The abstracted data are then entered into a general-purpose microcomputer-based software program. Data from participating sites were combined regionally and then aggregated to determine national rates. Data were collected following standardized protocols, using fixed definitions and data collection forms (13). Although no validation studies of the audit have been conducted, some areas have performed additional audits using different reviewers to both audit the same charts and assure accuracy in data entry procedures. Also, a comparison study has shown good observed agreement between the manual and electronic audits (0.78–1.00%) at a particular site.

HbA_{1c} level

We assessed glucose control by HbA_{1c} or by using the mean of the last three blood glucose readings. Because different laboratories are used throughout the nation, results were converted into a standardized HbA_{1c} value (16–19). If no HbA_{1c} was available, the mean of the last three blood glucose readings was used to calculate an "estimated HbA_{1c} value." The following formula was used: HbA_{1c} = (60.16 + mean glucose)/30.9 (19).

Table 1—*Selected demographic, clinical, and standards of care variables among American Indian/Alaska Native people with type 2 diabetes (IHS, 1998)*

Age at audit (years)	8,860 (54.9 ± 13.4)
Age at diagnosis (years)	8,860 (46.1 ± 13.2)
Diabetes duration (years)	8,860 (8.8 ± 7.4)
BMI	8,860 (32.9 ± 7.1)
HbA_{1c}	8,860 (8.6 ± 2.3)
Sex	
Male	3,532 (39.9)
Female	5,328 (60.1)
Proteinuria	
Yes	2,196 (24.8)
No	5,342 (60.3)
Unknown	1,322 (14.9)
Treatment type	
Diet alone	1,068 (12.1)
Oral agents	5,043 (56.9)
Insulin (with or without oral agents)	2,616 (29.5)
Unknown	133 (1.5)
Diabetes health care index factors	
Foot exam	
Yes	5,563 (62.8)
No	3,297 (37.2)
Eye exam	
Yes	5,004 (56.5)
No	3,856 (43.5)
Diet instruction	
Yes	5,858 (66.1)
No	3,002 (33.9)
Exercise instruction	
Yes	5,070 (57.2)
No	3,790 (42.8)
General diabetes education	
Yes	6,197 (69.9)
No	2,663 (30.1)
Influenza vaccination	
Yes	4,922 (55.6)
No	3,938 (44.4)

Data are *n* (mean ± SD) or *n* (%).

Covariates

Demographic, clinical, and standard of care variables were collected through the Diabetes Care and Outcomes Audit and included sex, age, height, weight, duration of diabetes, treatment type, and proteinuria status. Age was calculated as the date of audit minus the date of birth. BMI was calculated from recorded height and weight as follows: (703 × weight in pounds)/(height in inches × height in inches). Duration of diabetes was recorded as time since diagnosis in years. Treatment type was recorded as diet alone (no medication), oral agent, insulin, and oral agent plus insulin (combined with the insulin group for purposes of analyses). Proteinuria was defined as having ≥1+ (≥30 mg/dl) protein in a urine dipstick in the past year and did not include microalbuminuria.

"Health care index"

An index for quality of care was developed using six of the standard of care variables from the Diabetes Care and Outcomes Audit: foot examination, eye examination, diabetes diet instruction, exercise instruction, general diabetes education, and influenza vaccination. One point was assigned for each of the six items that were recorded as completed

Table 2—*Mean HbA_{1c} level by age and treatment among American Indian/Alaska Native people with diabetes (IHS, 1998)*

	Age category (years)					
	18–39	40–49	50–59	60–69	≥70	*P*
Overall						<0.0001
n	1,219	1,833	2,425	2,139	1,244	
Mean HbA_{1c} (%)	9.2	8.9	8.8	8.3	7.8	
Diet						<0.0001
n	164	214	243	250	197	
Mean HbA_{1c} (%)	7.3	7.1	7.0	6.8	6.4	
Oral agent						<0.0001
n	640	1,096	1,426	1,208	673	
Mean HbA_{1c} (%)	9.3	8.9	8.8	8.3	7.9	
Insulin						<0.0001
n	387	489	723	663	354	
Mean HbA_{1c} (%)	9.9	9.6	9.4	8.8	8.5	
Unknown						0.45
n	28	34	33	18	20	
Mean HbA_{1c} (%)	9.3	8.5	8.7	8.4	7.1	

within the year before the audit. For example, if all the standards of care measures were documented as having been completed within the year before the Diabetes Care and Outcomes Audit, the diabetes care health care index was 6, whereas if no measurements of the variables were documented as having been completed, the health care index was 0. If an activity was missing, it was recorded as "not done" and assigned a score of 0.

Data analyses

The analysis of the primary study end point, HbA_{1c}, was conducted for all people with measured and/or calculated HbA_{1c}. To test whether there were differences in HbA_{1c} by age, we used ANCOVA. ANOVA was used to test for differences in HbA_{1c} levels by age and treatment and for HbA_{1c} levels by diabetes health care index by age. The covariates for adjustment included BMI, duration of diabetes, treatment type, and the diabetes health care index. All statistical analyses were performed on SAS software (Cary, NC) (20).

RESULTS — Of the 11,419 potential participants aged ≥18 years who were identified from the 1998 National Indian Health Care and Outcomes Audit, 9,622 had measured HbA_{1c} and 1,158 had calculated HbA_{1c} levels. Data on duration of diabetes or BMI were missing for 1,920 participants, and these participants were excluded from the analyses. A total of 8,860 participants, 78% of the total sample, was used for analysis purposes.

Table 1 summarizes the demographic and selected clinical and measurements of care data. Consistent with sex differences in the prevalence of diabetes in American Indian/Alaska Native adults, more women than men were included in the sample. The mean age was 54.9 years, the mean duration of diabetes was 8.8 years, and the mean BMI was 32.9. The overall mean HbA_{1c} level was 8.6%. Of the patients, 25% had proteinuria, and most participants were treated with oral agents alone (57%). Diabetes health care factors ranged from 56% for influenza vaccine to 70% for general diabetes education.

HbA_{1c} was significantly different by age-group (Table 2). The youngest adults had the highest HbA_{1c} levels ($P < 0.0001$). In addition, BMI was highest in the youngest age-group, and it decreased with increasing age ($P < 0.0001$, data not shown). For the 1,920 patients who had missing data on duration of diabetes or BMI, a similar difference in HbA_{1c} levels by age-group was observed ($P < 0.0001$, data not shown). There was a statistically significant difference between age and all treatment types. The highest HbA_{1c} levels were found in the youngest age categories within each treatment regimen. We examined HbA_{1c} and BMI, using BMI categories of <25, 25–29.9, and ≥30, and we found no statistically significant association ($P = 0.36$).

Figure 1 shows HbA_{1c} levels by age-group and duration of diabetes. Within each duration category (0–4, 5–9, and ≥10 years), we observed an inverse relationship between age and HbA_{1c}. Table 3 presents HbA_{1c} level by health care index categories, with a higher index representing more health care factors. For each age-group, a higher health care index was significantly associated with improved HbA_{1c} levels, but it was not statistically significant for the group aged 18–39 years ($P = 0.06$). Within each health care index category, a statistically significant decrease in HbA_{1c} level was observed. Table 4 summarizes the adjusted mean HbA_{1c} by age-group for those with and without proteinuria and for those with unknown proteinuria status. An inverse relationship between age category and HbA_{1c} level for each category of proteinuria status was observed when adjusted for BMI, duration, treatment type, and health care index ($P < 0.0001$).

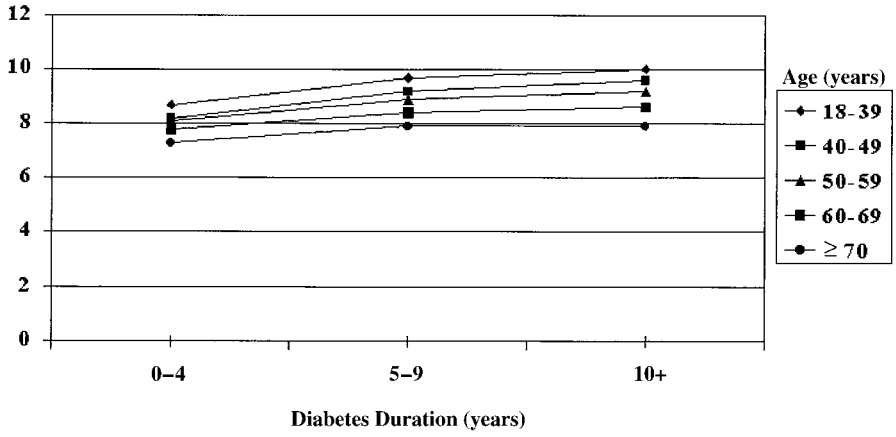

Figure 1—*HbA_{1c} by age and diabetes duration.*

Table 3—*Mean HbA_{1c} by health care index by age among American Indian/Alaska Native people with diabetes (IHS, 1998)*

	Age category (years)						
Health care index*	Overall†	18–39	40–49	50–59	60–69	≥70	*P*
0							0.007
n	485	115	112	122	82	54	
Mean HbA_{1c} (%)	9.0	9.5	9.3	9.0	8.8	8.1	
1–3							<0.0001
n	3,141	458	673	859	711	440	
Mean HbA_{1c} (%)	8.8	9.4	8.9	9.0	8.5	7.8	
4–5							0.0001
n	3,756	512	772	1,046	933	493	
Mean HbA_{1c} (%)	8.6	9.1	8.8	8.7	8.3	8.0	
6							0.0001
n	1,478	134	276	398	413	257	
Mean HbA_{1c} (%)	8.2	8.9	8.6	8.5	8.0	7.5	
P		0.06	0.02	0.005	0.003	0.005	

*One point was assigned for each of the following completed the year prior to the audit: foot exam, eye exam, diet instruction, exercise instruction, diabetes education, and influenza vaccine; †for overall, $P < 0.001$ across health care index categories.

CONCLUSIONS — An inverse relationship of HbA_{1c} level and age was found among American Indian/Alaska Native adults nationally after adjusting for BMI, diabetes duration, treatment type, proteinuria, and health care index. Although these findings are cross-sectional and are limited by our measure of health care index, they lend support to prior published findings that younger American Indian/Alaska Native people with type 2 diabetes have worse diabetes control (8,9). The factors underlying the elevated HbA_{1c} levels among younger American Indian/Alaska Native adults are not fully understood, but they do not appear to be explained by BMI, diabetes duration, treatment type, proteinuria, or diabetes health care index. We cannot directly measure whether these results are caused by a cohort effect (different age-groups represent different birth cohorts, and the same differences will continue to exist as the cohorts age) or represent developmental effects (as the cohorts age, their glycemic control will improve). However, because glycemic control generally deteriorates with time (7), it is likely a cohort effect. A longitudinal follow-up study would help to assess this question. There is an urgent need to understand the factors related to the elevated HbA_{1c} levels because the prevalence of diabetes among young American Indian/Alaska Native people is on the rise (5,6), and it is therefore likely that we may observe an increase in diabetes-related morbidity and mortality unless more effective and efficient interventions are developed to improve glycemic control in young American Indian/Alaska Native adults and thereby prevent complications of diabetes.

The higher HbA_{1c} levels seen in the younger age-groups may be related to rapid changes in lifestyle that have occurred in many American Indian/Alaska Native communities. In a study in New Mexico, HbA_{1c} levels were highest for those who reported consuming the most fat and sugar (8). Moreover, high consumption of fat and sugar was most closely associated with elevations in HbA_{1c} levels among those aged <55 years (8).

In this study, we found that BMI increased with younger age, which is consistent with national BMI trends (21). Studies have shown that American Indian/Alaska Native people are becoming more overweight and obese, an observation that may be associated with increased insulin resistance, and this increase in obesity may influence the ability to effectively treat diabetes because greater obesity and insulin resistance have been associated with poorer glucose control (22–27). However, higher BMI values in the younger age-groups did not account for the high HbA_{1c} levels observed among younger American Indian/Alaska Native adults. We lack data on insulin resistance in this study to assess the role of obesity and insulin resistance in the relationship between age and HbA_{1c}.

Environmental and socioeconomic factors play a role in diabetes outcomes, including glucose control, morbidity, and mortality (28). Access to care and medications may also influence health outcomes; however, these factors are unlikely to play a major role in the current study because health care and medications are generally provided free of charge for American Indian/Alaska Native people through IHS and tribal health programs, although in some situations, patients are charged a co-pay, and not all clinics carry all medications. However, younger adults may have difficulty attending clinic appointments and assuming self-care activities, given their work and child/family

Table 4—*Adjusted mean HbA_{1c} by age-group and by proteinuria status among American Indian/Alaska Native people with diabetes (IHS, 1998)*

		Age category (years)				
		<18–39	40–49	50–59	60–69	≥70
Proteinuria (n = 2,196)						
	Model 1*	9.8	9.3	9.1	8.4	7.6
	Model 2†	9.3	8.9	8.7	8.1	7.3
No proteinuria (n = 5,342)						
	Model 1*	9.4	9.0	8.8	8.3	7.8
	Model 2†	9.0	8.6	8.4	7.9	7.6
Unknown proteinuria (n = 1,322)						
	Model 1*	9.5	8.9	8.7	8.4	7.5
	Model 2†	9.3	8.8	8.6	8.4	7.7

Data are mean HbA_{1c} level. *Adjusted for duration, BMI, and health care index. $P < 0.0001$ for differences in HbA_{1c} level among age-groups. †Adjusted for duration, BMI, health care index, and treatment type. $P < 0.0001$ for differences in HbA_{1c} level among age-groups.

care activities. Prior studies have noted improved compliance with treatment type and clinic appointments with increasing age (10,29). In the current study, the diabetes care health care index was used to assess whether a poor health care index explained poorer glycemic control in young adults. Overall, each increase in the index was associated with an improvement in HbA_{1c}; however, higher HbA_{1c} values with decreasing age persisted within each health care index category. These results do not support the hypothesis that worse diabetes control in the younger age-group is caused by poorer health care indices for this population (9).

There are several limitations of this study. First, these data were collected as secondary data. Although the audit criteria are clearly set to include random and adequate sampling, standard definitions, and standard reporting protocol, it is possible that all reviewers did not always follow these standards. The large dataset and the many years of audit implementation should decrease the influence of deviation from the protocol by a minority of reviewers. No validation studies of the audit have been conducted. However, some areas have performed additional audits using different reviewers to both audit the same charts and assure accuracy in data entry procedures. A comparison study has shown good observed agreement between the manual and electronic audits (0.78–1.00%) at a particular site.

Cross-sectional studies are subject to survival bias. This is unlikely to have biased the current results, given the persistence of the relationship within each diabetes duration category. Selection bias is a possibility because not all Indian health care facilities participated in the audit. A larger percentage of women than men were included in the data, which is consistent with prior reports (11). The results do not appear to be biased by the presence of a higher percentage of elders with renal disease (with higher circulating insulin levels and therefore better glycemic control), given the persistence of the results when stratified by proteinuria status.

We cannot assume the health care index is reflective of patient self-care activities alone because it contains items that are influenced by both system as well as patient self-care factors. It would be helpful to have additional items reflective of system- and patient-centered factors in the future to address this issue.

The current study brings a major health challenge to health professionals in the Indian health care system: to understand the physiological, socioeconomic, and lifestyle factors influencing diabetes control in younger American Indian/Alaska Native adults and to develop and implement effective and culturally appropriate interventions in this high-risk group. Unless we are successful in these endeavors, the morbidity and mortality associated with diabetes in young American Indian/Alaska Native people can be expected to rise in the next decade.

Acknowledgments— This study was supported by the New Mexico Veterans Affairs Health Care System, Albuquerque, New Mexico, and by National Institutes of Health–National Institute of Diabetes and Digestive and Kidney Diseases Grant RO1-DK-47096-06.

This manuscript represents the results of a study that Dr. Janette S. Carter was actively involved in at the time of her untimely death. Dr. Carter dedicated her career to improving care of people with diabetes, especially in American Indian/Alaska Native communities. The diabetes community has suffered a huge loss with her passing.

References

1. Gohdes D, Bennett PH: Diabetes in American Indians and Alaska Natives. *Diabetes Care* 16:214–215, 1993
2. Carter J, Horowitz R, Wilson R, Sava S, Sinnock P, Gohdes D: Tribal differences in diabetes: prevalence among American Indians in New Mexico. *Pub Health Rep* 104:665–669, 1989
3. Gilliland FD, Owen C, Gilliland SS, Carter JS: Temporal trends in diabetes mortality among American Indians and Hispanics in New Mexico: birth cohort and period effects. *Am J Epidemiol* 145: 422–431, 1997
4. Nelson RG, Newman JM, Knowler WC, Sievers ML, Kunzelman CL, Pettitt DJ, Moffett CD, Teutsch SM, Bennett PH: Incidence of end-stage renal disease in type 2 (non-insulin-dependent) diabetes mellitus in Pima Indians. *Diabetologia* 31: 730–736, 1988
5. Dabelea D, Hanson RL, Bennett PH, Roumain J, Knowler WC, Pettitt DJ: Increasing prevalence of type II diabetes in American Indian children. *Diabetologia* 41:904–910, 1998
6. Fagot-Campagna A, Pettitt DJ, Engelgau MM, Burrows NR, Geiss LS, Valdez R, Beckles GL, Saaddine J, Gregg EW, Williamson DF, Narayan KM: Type 2 diabetes among North American children and adolescents: an epidemiologic review and a public health perspective. *J Pediatr* 136: 664–672, 2000
7. UKPDS: Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes. *Lancet* 352:837–853, 1998
8. Carter J, Gilliland S, Perez G, Skipper B, Gilliland F: Public health and clinical implications of high hemoglobin A1c levels and weight in younger adult Native American people with diabetes. *Arch Intern Med* 160:3471–3476, 2000
9. Hu D, Henderson JA, Welty TK, Lee ET, Jablonski KA, Magee MF, Robbins DC, Howard BV: Glycemic control in diabetic American Indians. *Diabetes Care* 22: 1802–1807, 1999
10. Curtin RB, Svarstad BL, Andress D, Keller T, Sacksteder P: Differences in older versus younger hemodialysis patients' noncompliance with oral medications. *Geriatr Nephrol Urol* 7:35–44, 1997
11. Mayfield JA, Rith-Najarian SJ, Acton KJ, Schraer CD, Stahn RM, Johnson MH, Gohdes D: Assessment of diabetes care by medical record review: the Indian Health Service model. *Diabetes Care* 17: 918–923, 1994
12. Acton K, Valway S, Helgerson S, Huy JB, Smith K, Chapman V, Gohdes D: Improving diabetes care for American Indians. *Diabetes Care* 16:372–375, 1993
13. Indian Health Services: *Indian Health Diabetes Chart Audit for Quality Assurance and Quality Improvement*. Albuquerque, NM, Indian Health Services Diabetes Programs, 2000, p. 1–18
14. Rosner B: *Fundamentals of Biostatistics*. Boston, MA, Duxbury Press, 1982
16. Gohdes D, Rith-Najarian S, Acton K, Shields R: Improving diabetes care in the primary health setting. *Ann Intern Med* 1996:149–152
17. Goldstein DE, Wiedmeyer H-M, Little RR, Vargas V, Nair SS, Reid J: Relationship between glycohemoglobin (GHB) and mean blood glucose (MBG) in the Diabetes Control and Complications Trial (DCCT) (Abstract). *Diabetes* 46 (Suppl. 1):8A, 1997
18. Nathan DM, Singer DE, Hurxthal K, Goodson JD: The clinical information value of gylcosolated hemoglobin assay. *N Engl J Med* 310:341–346, 1984
19. Little R, Wiedmeyer H, England J, Naito H, Goldstein D: Interlaboratory comparison of glycohemoglobin results: College of American Pathologists survey data. *Clin Chem* 37:1725–1729, 1991
20. *SAS/STAT Users Guide: Version 6:12*. Durham, NC, SAS Inst., 1990
21. Mokdad A, Bowman B, Ford E, Vinicor F,

Marks J, Koplan J: The continuing epidemics of obesity and diabetes in the United States. *JAMA* 286:1195–1200, 2001
22. Gilbert TJ, Percy CA, Sugarman JR, Benson L, Percy C: Obesity among Navajo adolescents: relationship to dietary intake and blood pressure [see comments]. *Am J Dis Child* 146:289–295, 1992
23. Knowler WC, Pettitt DJ, Saad MF, Charles MA, Nelson RG, Howard BV, Bogardus C, Bennett PH: Obesity in the Pima Indians: its magnitude and relationship with diabetes. *Am J Clin Nutr* 53 (Suppl. 6): 1543S–1551S, 1991
24. Broussard BA, Johnson A, Himes JH, Story M, Fichtner R, Hauck F, Bachman-Carter K, Hayes J, Frohlich K, Gray N, et al.: Prevalence of obesity in American Indians and Alaska Natives. *Am J Clin Nutr* 53 (Suppl. 6):1535S–1542S, 1991
25. Broussard BA, Sugarman JR, Bachman-Carter K, Booth K, Stephenson L, Strauss K, Gohdes D: Toward comprehensive obesity prevention programs in Native American communities. *Obes Res* 3 (Suppl. 2):289s–297s, 1995
26. Charles MA, Pettitt DJ, Saad MF, Nelson RG, Bennett PH, Knowler WC: Development of impaired glucose tolerance with or without weight gain. *Diabetes Care* 16: 593–596, 1993
27. Serrano Rios M: Relationship between obesity and the increased risk of major complications in non-insulin-dependent diabetes mellitus. *Eur J Clin Invest* 28 (Suppl. 2):14–17, 1998 [discussion p. 17–18]
28. Weng C, Coppini D, Sonksen P: Geographic and social factors are related to increased morbidity and mortality rates in diabetic patients. *Diabet Med* 17:612–617, 2000
29. O'Connor PJ, Fragneto R, Coulehan J, Crabtree BF: Metabolic control in non-insulin dependent diabetes mellitus: factors associated with patient outcomes. *Diabetes Care* 10:697–701, 1987