Microvascular Complications of NIDDM in Hispanics and Non-Hispanic Whites

San Luis Valley Diabetes Study

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The goal of this article was to examine the differences in the rates of microvascular complications of non-insulin-dependent diabetes mellitus (NIDDM) in Hispanic and non-Hispanic white subjects. This was a geographically based case-control study where prevalent cases of NIDDM were identified in medical records. Subjects attended a 4-h clinic to confirm NIDDM diagnosis and assess complication end points. Retinopathy was defined by stereofundus photographs. Distal symmetric neuropathy was determined by standardized clinical examination. Nephropathy was indicated by serum creatinine level, urine protein–creatinine ratio, and urine albumin concentration. This study consisted of 279 NIDDM subjects confirmed by oral glucose tolerance test and World Health Organization criteria aged 20–74 yr (187 Hispanic and 92 non-Hispanic white subjects). Duration-adjusted prevalence of retinopathy was significantly higher in non-Hispanic white subjects (54.1 per 100, 95% confidence interval [CI] 44.4–63.7) than in Hispanics (41.8 per 100, 95% CI 34.8–48.8). This excess occurred only in non-Hispanic white subjects with background retinopathy but not in those with more severe retinopathy. Hispanics and non-Hispanic white subjects did not differ significantly for the prevalence of neuropathy (31.6 per 100 in non-Hispanic white subjects and 26.3 per 100 in Hispanics) or nephropathy by any measure. There were no significant differences in duration of diabetes or mean glycohemoglobin levels between ethnic groups. Microvascular complications of NIDDM are not in excess among Colorado Hispanics, and retinopathy may be somewhat more common in non-Hispanic white people. *Diabetes Care* 14 (Suppl. 3):655–64, 1991

The principal diabetic microvascular disease complications include retinopathy, neuropathy, and nephropathy (1–4). These complications, although common in diabetic subjects after several years of disease, until recently have not been examined in geographically based studies that represent all people with diabetes. Furthermore, these complications have rarely been examined together as comorbid conditions. Clinic-based studies of complications may suffer from serious referral bias (5), i.e., the tendency for more serious or complicated cases to be cared for at tertiary-care centers where journal publications originate. Thus, a description of microvascular complications in a representative group of prevalent diabetic subjects provides a clearer view of the frequency of these complications in the community. Older studies (6,7) of complications have not differentiated diabetic subjects by type (insulin-dependent diabetes mellitus [IDDM] or non-insulin-dependent diabetes mellitus [NIDDM]; 8,9). Whether these types of diabetes have the same or different complication patterns may provide additional information about the causes of these complications. Complications prevalence must also be compared with standardized reproducible measurement techniques before meaningful conclusions about intergroup differences can be made with any certainty.

We were especially interested to learn whether Hispanics had an excess of any or all of these microvascular complications when compared with non-Hispanic

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whites living in the same area and receiving the same medical care. If different rates of complications occur by ethnic group, it could provide useful leads to the causes of microvascular complications. Hispanics are an important group to study, because Hispanic men had 2.1 times and Hispanic women 4.8 times the prevalence of NIDDM compared with non-Hispanic whites in Colorado (10). Rates reported from Texas are of similar or greater magnitude (11). This article presents the prevalence of these complications with objective measures in Hispanics and non-Hispanic whites at the baseline visit in the San Luis Valley Diabetes Study, a geographically based study of NIDDM prevalence, complications, and risk factors in two southern Colorado counties. Two reports have been published previously from portions of this study, which indicate that Hispanics do not show an excess prevalence of retinopathy or neuropathy compared with non-Hispanic whites (12,13). This analysis extends those findings by including nephropathy and examining these complications in conjunction with each other.

RESEARCH DESIGN AND METHODS

Detailed methods have been reported elsewhere (10) and are briefly summarized herein. The study was designed as a geographically based case-control study in Alamosa and Conejos counties, in the San Luis Valley of southern Colorado. All procedures were approved by the University of Colorado Health Science Center Human Subjects Review Committee, and informed consent for participation was obtained from each subject.

Diabetic subjects were ascertained by review of medical records in all health-care facilities in the study area and by local publicity. Clinic-eligible subjects with a medical diagnosis or self-report of diabetes (either IDDM or NIDDM) were 20–74 yr of age, noninstitutional residents of the study area, mentally competent, and spoke either English or Spanish. Of the 420 eligible people with diabetes, 343 attended the clinic (81.7%). The 279 people with diabetes in this report include all those confirmed with NIDDM, according to the criteria noted below. Sixty-four of the 343 identified diabetic subjects were either classified as IDDM (n = 25) or had a glucose tolerance test result of nondiabetic (n = 39). Respondents were similar to those who did not attend clinic on important diabetes-related factors such as disease duration, type of hypoglycemic medication, history of hypertension, and smoking, among others (10).

Data for nondiabetic control subjects are reported only for the neuropathy analyses. Control subjects were selected with a geographically based two-stage sampling procedure. Residential structures were identified, sampled, and a 5- to 10-min home interview was completed that enumerated the occupants (96.6% response rate). Enumerated subjects were randomly selected within age, sex, ethnic group, and county strata to reflect the Hispanic diabetic age structure, and asked to attend clinic. Of the 887 people who were eligible for clinic as a control subject, 607 attended (68.4%).

Subjects attended a 4-h clinic visit in the morning after an overnight fast. At the start of each visit, subjects were examined, interviewed, and blood was drawn for various studies while fasting and at 1 and 2 h after a 75-g oral glucose load. The variables used in this analysis were defined as follows:

Oral glucose tolerance test (OGTT). Subjects who reported at least an 8-h fast were given 75 g of glucose (Koladex, Orangedex, Custom, Baltimore, MD). Ninety-eight percent of subjects had fasted for a minimum of 10 h. Glucose was measured with the glucose oxidase method on venous plasma.

Diabetes mellitus. People reported herein with previously diagnosed diabetes were confirmed by OGTT according to the World Health Organization criteria (9). Subjects on insulin or oral hypoglycemics were also classified as diabetic, regardless of their OGTT result. Subjects selected as control subjects were not on any hypoglycemic medication and had impaired glucose tolerance or normal glucose tolerance by OGTT. Control subjects who were diagnosed with NIDDM for the first time at the clinic visit are not included in this report.

Type of diabetes. Twenty-five subjects who met the World Health Organization criteria for diabetes were classified as IDDM based on C-peptide levels <0.1 pM at fasting (n = 23) or by age of onset <18 yr and insulin use for all years of duration if C-peptide levels were missing (n = 2; 10). These subjects were removed from the case group. The remaining subjects were considered to have NIDDM.

Duration of diabetes. Age at diagnosis as reported by the subject was used to calculate duration.

Glycohemoglobin. A microcolumn method was used to measure total glycosylated hemoglobin (14).

Ethnic identification. Hispanic ethnicity was defined by the self-report of subjects to the 1980 U.S. Census questions about Hispanic identification (15), excluding subjects who reported Asian or black race. Our use of Hispanic here is consistent with other reports of Mexican Americans (16).

Hypertension. Antihypertensive treatment or a diastolic blood pressure level ≥90 mmHg on the average of the last two of three repeated measurements at the clinic visit was used to classify persons as hypertensive.

Retinopathy. Stereofundus photographs were taken of three fields (I, II, IV) through dilated pupils (17). These were graded by the University of Wisconsin Fundus Photograph Reading Center with modified Airlie House criteria (18,19). For this analysis, findings were grouped into no retinopathy (codes 10–12), background (codes 15–40), preproliferative (code 50), and proliferative disease (code 60–80), with the worse eye used for level of severity.

Neuropathy. Subjects were considered to have distal

symmetrical neuropathy if any two of the three following symptoms or signs were present: *1*) positive history of pain or discomfort in both legs or both feet, such as numbness, burning, or tingling when you are not walking in the past 6 mo, *2*) absent deep tendon reflexes in knees or ankles, and *3*) absent temperature sensation in feet (to an iced tuning fork) based on an examination by a nurse practitioner standardized to a neurologist (13).

Nephropathy. Three indications of kidney disease were used: *1*) serum creatinine >132.6 μM, *2*) urine total protein-creatinine ratio >1 (20), and *3*) urine albumin concentration >25.5 μg/ml by radioimmunoassay (21), which is roughly equivalent to 30 μg/min excretion (22). Subjects voided on arrival at clinic, and urine samples were collected after 2 h in the clinic. The cut point for the distribution of albumin concentration was selected as roughly equivalent to excretion rates reported in the literature (22,23), and the 25.5-μg/ml cut point was approximately the upper 95th percentile for people in this study with normal glucose tolerance. Thus, this cut point identifies people above a conventional normal range for nondiabetic subjects with our method. A dipstick method to detect asymptomatic bacteriuria was also used (Chemstrip LN, Boehringer Mannheim, Indianapolis, IN). Nitrate positivity was used to indicate possible infection (24). Distributions of albumin concentration were examined for subjects positive and negative for nitrate and no significant differences were identified; therefore, the nine subjects with nitrate positivity were not excluded from these analyses.

Statistical methods. Duration adjustment by ethnicity used the direct method (25), with the duration distribution of all subjects as the standard. Ninety-five percent confidence limits on the duration-adjusted prevalence were calculated with the normal approximation (26,27). Logistic regression was conducted with GLIM (28).

RESULTS

There were 187 Hispanic and 92 non-Hispanic white people with NIDDM who were included in this analysis (Table 1). On average, Hispanic subjects were 60 yr old and non-Hispanic white subjects were 59 yr old, with the average onset of diabetes reported ~10 yr earlier for both ethnic groups. Mean glycohemoglobin levels were 10.6 and 10.3% in Hispanics and non-Hispanic whites, respectively (normal range 6.8–8.2%). Hispanic subjects were more likely to be treated with insulin and less likely to be treated with oral agents than non-Hispanic white subjects. None of these differences reached conventional levels of statistical significance.

Because previous reports have shown the strong dependence of complication frequency on disease duration, we examined duration for each end point before calculating prevalence by ethnicity. Average diabetes duration was similar for Hispanics and non-Hispanic whites. Figure 1 shows that retinopathy, distal symmetric neuropathy, serum creatinine level, and albumin levels >25.5 μg/ml were all duration dependent for both ethnic groups. All subsequent data tables were duration adjusted to remove any minor differences in duration distribution between ethnic groups.

TABLE 1
Number of subjects with confirmed non-insulin-dependent diabetes mellitus (NIDDM) and summary of characteristics by ethnicity (San Luis Valley, Colorado, 1984–1986)

Characteristic	Ethnicity		*P*
	Hispanic	Non-Hispanic whites	
n with NIDDM*	187	92	
Age at clinic visit (yr)	59.7 ± 0.7	58.8 ± 1.1	0.51
Age at reported NIDDM diagnosis (yr)	49.3 ± 0.8	49.7 ± 1.3	0.75
Duration of NIDDM (yr)	10.4 ± 0.6	9.1 ± 0.8	0.19
Glycohemoglobin (%)	10.6 ± 0.2	10.3 ± 0.3	0.70
Treatment (%)			
Insulin	52.7	41.8	0.06
Oral	22.0	38.4	
Diet only	5.9	6.6	
Insulin + oral	2.7	1.1	
None reported	16.7	12.1	

Values are means ± SE.
*Previously undiagnosed subjects are excluded.

As previously reported, diabetic retinopathy was diagnosed by fundus photographs in 71 of 166 Hispanics with photographs (42.8%) and in 44 of 85 non-Hispanic whites (51.8%; 12). Table 2 shows the duration-adjusted prevalence per 100 by severity of retinopathy. There was significantly less retinopathy among Hispanics (41.8 per 100, 95% confidence interval [CI] 34.8–48.8) compared with non-Hispanic whites with NIDDM (54.1 per 100, 95% CI 44.4–63.7, $P = 0.04$). This deficit among Hispanics was concentrated in background retinopathy (23.3 vs. 32.8 per 100 for Hispanics and non-Hispanic whites, respectively), whereas more severe retinopathy was similar between the two ethnic groups. Review of medical record data from subjects without photographs (7 non-Hispanic whites, 21 Hispanics) did not identify any excess of retinopathy or major differences in risk factors for either ethnic group (data not shown).

The prevalence of distal symmetric neuropathy in San Luis Valley Diabetes Study subjects has been published elsewhere (13) and crude prevalence rates ranged from 3.5 per 100 in nondiabetic subjects to 27.8 per 100 in diabetic subjects. Neuropathy prevalence by ethnicity and glucose tolerance category is shown in Table 3. Because the definitions of neuropathy used in this study are consistent with clinical criteria but not widely used, results for subjects with normal and impaired glucose

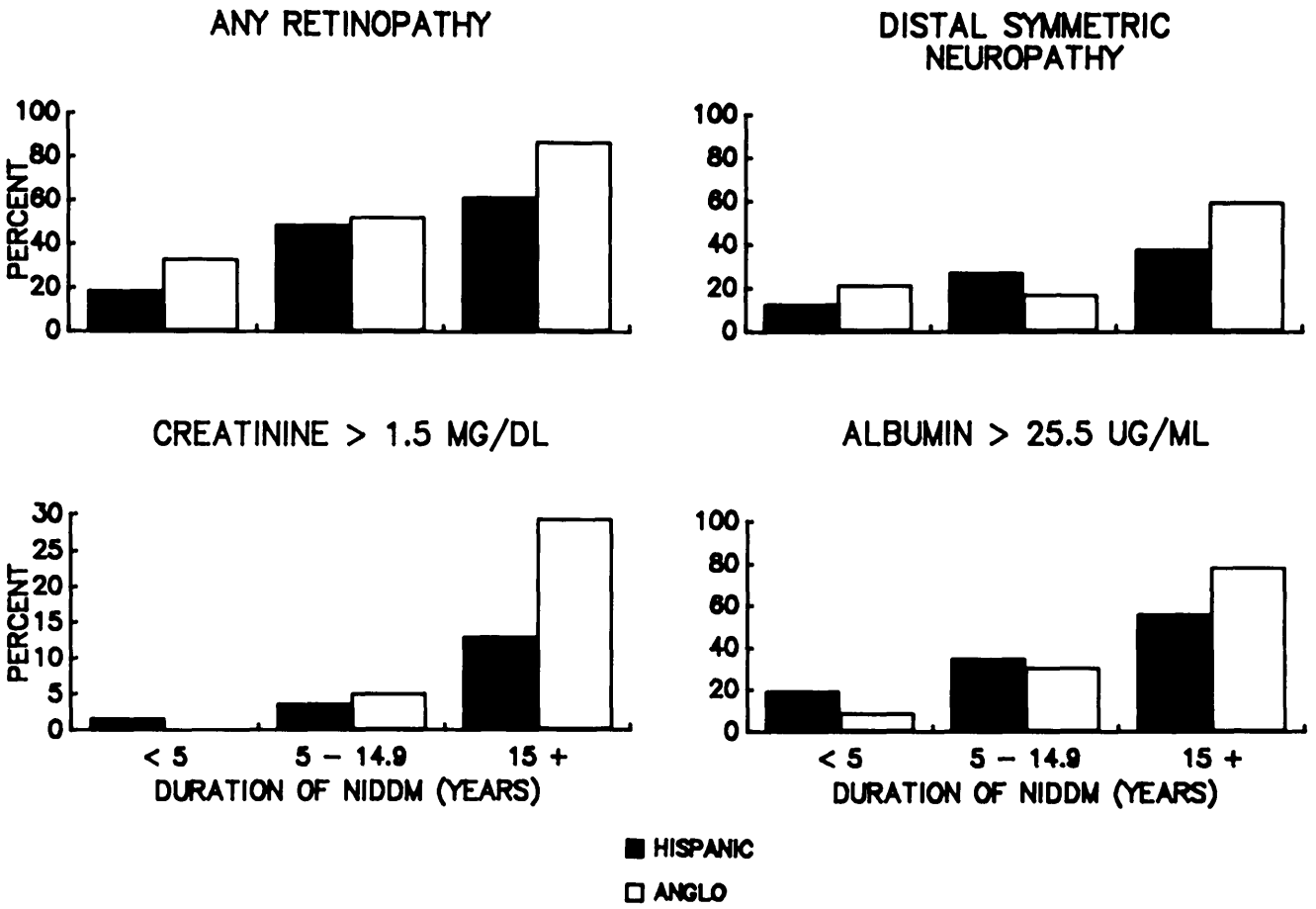

FIG. 1. Prevalence of complications by reported duration of non-insulin-dependent diabetes in Hispanics and non-Hispanic whites.

tolerance are also shown for comparison. The crude prevalence of distal symmetric neuropathy was 2.7 and 4.8 per 100 among non-Hispanic whites and Hispanic people with normal glucose tolerance, was 9.8 and 12.5 per 100 for non-Hispanic whites and Hispanic people with impaired glucose tolerance, and among subjects with NIDDM was 29.7 and 26.9 per 100 in non-Hispanic whites and Hispanics, respectively. After duration adjustment among subjects with NIDDM, Hispanics had nonsignificantly less distal symmetric neuropathy (26.3

TABLE 2
Duration-adjusted prevalence (per 100) of retinopathy by severity and ethnicity among people with confirmed non-insulin-dependent diabetes (San Luis Valley, Colorado, 1984–1986)

	Hispanic			Non-Hispanic whites		
Retinopathy severity	*n*	Adjusted prevalence per 100*	95% confidence interval	*n*	Adjusted prevalence per 100*	95% confidence interval
None	95 of 166	58.2	(51.2–65.2)	41 of 85	45.9	(36.3–55.6)
Background (codes 15–30)	39 of 166	23.3	(17.1–29.6)	28 of 85	32.8	(22.8–42.8)
Preproliferative (code 40)	21 of 166	12.1	(7.4–16.9)	12 of 85	15.6	(8.0–23.1)
Proliferative (codes 60–80)	11 of 166	6.4	(2.8–10.0)	4 of 85	5.7	(0.9–10.6)
Any retinopathy (codes 15–80)	71 of 166	41.8	(34.8–48.8)	44 of 85	54.1	(44.4–63.7)

From Hamman et al. (12).
*Directly adjusted with duration distribution of all subjects as standard. Excludes 21 Hispanics (11.2%) and 7 non-Hispanic whites (7.6%) with no photographs.

TABLE 3
Prevalence (per 100) of distal symmetrical sensory neuropathy by ethnicity and glucose tolerance status (San Luis Valley, Colorado, 1984–1986)

Glucose tolerance status	Hispanic		Non-Hispanic whites	
	n	Crude prevalence per 100	*n*	Crude prevalence per 100
Nondiabetic	9 of 188	4.8	8 of 298	2.7
Impaired glucose tolerance	6 of 48	12.5	4 of 41	9.8
Non-insulin-dependent diabetes	50 of 186	26.9	27 of 91	29.7
Non-insulin-dependent diabetes duration-adjusted prevalence (95% confidence interval)*	26.3	20.2–32.4	31.6	23.0–40.2

*Directly adjusted with duration distribution of all subjects as standard.

per 100, 95% CI 20.2–32.4) compared with non-Hispanic whites (31.6 per 100, 95% CI 23.0–40.2, $P = 0.32$).

Several indicators of possible diabetic kidney disease were examined (Table 4). Poor kidney function, marked by elevated serum creatinine of >132.6 μM, was noted in 5.2 per 100 Hispanic diabetic subjects and in 8.9 per 100 non-Hispanic whites adjusted for duration of diabetes ($P = 0.23$).

A ratio of total urinary protein concentration divided by urine creatinine concentration >1 has been suggested as a quantitative marker of significant proteinuria from diabetic nephropathy (20). A somewhat higher prevalence of this end point was found in both Hispanics and non-Hispanic whites than for serum creatinine. Among Hispanics, 9.2 per 100 had elevated protein-creatinine ratios compared with 11.4 per 100 in non-Hispanic whites, a difference that was not statistically significant ($P = 0.56$).

The most sensitive marker of early kidney damage from diabetes is urinary albumin measured by sensitive radioimmunoassay (29). In this field study, it was not possible to do overnight timed collections; therefore, 2-h spot urine samples were analyzed for both albumin and creatinine. Ratios of urinary albumin to creatinine were also analyzed but the results were identical to those shown for albumin concentration, so they are not reported here. The prevalence of elevated albumin levels >25.5 μg/ml was virtually identical in Hispanics and non-Hispanic whites, after adjustment for duration. Figure 2 shows the distribution of albumin levels by ethnic group at arbitrary cut points. No important differences between ethnic groups were seen.

After examining each complication separately, it was important to determine whether there was a difference in the pattern of complications by ethnicity. That is, were Hispanics more or less likely to have only one complication or a specific cluster of complications? Table 5 addresses this question with the use of subjects with data to classify all three end points. There was a lower prevalence of retinopathy as a single complication among Hispanics, but most other complication patterns were similar between the two ethnic groups, given the relatively small sample size.

It is possible that the lack of Hispanic excess in the duration-adjusted results (Tables 2–5) is due to a different distribution of major risk factors for microvascular complications in the two ethnic groups, although this appears unlikely from the mean age, duration, and glycohemoglobin for each ethnic group presented in Table

TABLE 4
Duration-adjusted prevalence (per 100) of various markers of nephropathy among people with confirmed non-insulin-dependent diabetes by ethnicity (San Luis Valley, Colorado, 1984–1986)

Nephropathy marker	Hispanic			Non-Hispanic whites		
	n	Adjusted prevalence per 100*	95% confidence interval	*n*	Adjusted prevalence per 100*	95% confidence interval
Serum creatinine >132.6 μM	10 of 184	5.2	(2.1–8.3)	7 of 92	8.9	(3.2–14.7)
Urine protein-creatinine ratio >1	18 of 187	9.2	(5.4–13.1)	9 of 91	11.4	(4.8–18.0)
Urinary albumin >25.5 μg/ml†	53 of 145	34.5	(27.1–41.9)	21 of 62	34.3	(24.3–44.3)

*Directly adjusted with use of duration distribution of all subjects as standard.
†Roughly equal to 30 μg/min excretion rate.

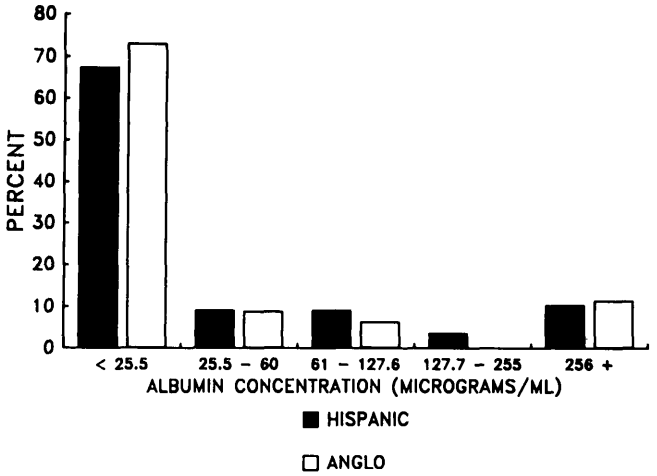

FIG. 2. Distribution of urine albumin concentration by Hispanic and non-Hispanic whites.

1. Table 6 summarizes the Hispanic to non-Hispanic white odds ratios for each complication, adjusted for potential confounding factors, including level of blood glucose control (as glycohemoglobin percent), age at onset, duration (in three groups), use of insulin, and presence of hypertension. The results of these multiply adjusted analyses are similar to those adjusted only for duration. Hispanics had a significant deficit of any retinopathy, a nonsignificant deficit of distal symmetric neuropathy, and somewhat higher levels of moderate albuminuria (>25.5 μg/ml) compared with non-Hispanic whites. Hispanics also had a deficit of people with urine albumin concentration >255 μg/ml, although this was not significant. Only for retinopathy was there a statistically significant deficit among Hispanics. There was no evidence of any difference in neuropathy or nephropathy prevalence between the two ethnic groups. Thus, the lack of excess Hispanic complications is not due to altered patterns of risk factors. Although other factors not included in this analysis (smoking, height) may be important in defining the actual risk of specific complications, the factors included herein contribute to risk and would be the ones likely to make major differences in population prevalence.

TABLE 5
Duration-adjusted prevalence (per 100) of microvascular complications among people with confirmed non-insulin-dependent diabetes mellitus (NIDDM) by ethnicity (San Luis Valley, Colorado, 1984–1986)

Complications	Hispanic		Non-Hispanic whites	
	n	Adjusted prevalence per 100*	*n*	Adjusted prevalence per 100*
None	54	39.00	22	29.33
Retinopathy only†	20	13.83	19	26.08
Nephropathy only‡	11	7.51	2	2.87
Neuropathy only§	12	8.41	7	9.92
Retinopathy + nephropathy	18	12.03	5	7.77
Retinopathy + neuropathy	13	8.90	5	7.03
Neuropathy + nephropathy	3	1.98	2	2.70
All three complications	13	8.36	9	14.30
Total with NIDDM‖	144		71	

*Directly adjusted with use of duration distribution of all subjects as standard.
†Any retinopathy.
‡Albumin >25.5 μg/ml (~30 μg/min).
§Any definite distal symmetric neuropathy.
‖43 Hispanics and 21 non-Hispanic whites excluded due to missing data on one or more end points.

TABLE 6
Effect of Hispanic ethnicity compared with non-Hispanic whites for microvascular complications adjusted for potential confounding variables with logistic regression (San Luis Valley, Colorado, 1984–1986)

Complication end point	Hispanic vs. non-Hispanic whites odds ratio	95% confidence interval
Any retinpathy vs. none	0.47	(0.24–0.94)
Distal symmetric neuropathy vs. none	0.59	(0.29–1.20)
Albuminuria >25.5 vs. ≤25.5 μg/ml*†	1.11	(0.54–2.27)
Albuminuria >255 vs. ≤25.5 μg/ml‡§	0.72	(0.25–2.01)

Each complication end point was dependent variable in separate logistic model that also included sex, age at onset of diabetes (in years), duration of diabetes (<5, 5–14, ≥15 yr), glycohemoglobin (%), use of insulin (yes/no), and hypertension (yes/no).
*Roughly equal to 30 μg/min excretion rate.
†Comparison of all subjects with albuminuria >25.5 μg/ml vs. subjects ≤25.5 μg/ml.
‡Comparison of subjects >255 μg/ml vs. subjects ≤25.5 μg/ml.
§Roughly equivalent to 300 μg/min excretion rate.

CONCLUSIONS

Microvascular complications were examined for Hispanics and non-Hispanic whites with standardized objective clinically relevant end points. Non-Hispanic whites had significantly more background retinopathy. Distal symmetric neuropathy prevalence and markers of nephropathy (except moderate microalbuminuria) were also elevated in non-Hispanic whites, although not significantly. Therefore, we found no excess of microvascular complications in Hispanics, a finding that differs from the San Antonio Heart Study (SAHS), at least for retinopathy (30) and nephropathy (31).

The limitations of this study must be kept in mind.

Because this was a cross-sectional review of prevalent diabetic subjects, it is possible that differential survivorship may have altered the findings (32). Because subjects who were examined were survivors, and if Hispanic mortality from NIDDM differs from the mortality of non-Hispanic whites, there could be different patterns of complications in all Hispanic diabetic subjects that went undetected. We are not aware of case-fatality data for Hispanics with diabetes, however, among younger blacks with IDDM, excess mortality has been reported when compared with whites (33). In our study subjects, physician visits (5.8/person/yr for Hispanics and 4.9/person/yr for non-Hispanic whites, $P = 0.09$) and hospitalization frequency (1.7 and 1.5% for Hispanics and non-Hispanic whites, respectively, $P = 0.48$) did not differ significantly by ethnicity, suggesting that differential medical care access is not a problem. This differs from national data where Mexican Americans are reported to have less medical care utilization (34). In addition, we found no evidence of severity bias for the participants in this study when compared with the nonresponders (10). Thus, survivorship or selection biases seem unlikely to explain these results, although prospective follow-up is the only solution for these potential biases. We also had relatively small numbers of subjects for comparison of clusters of complications. Additional subjects are now under study to alleviate this problem.

There have been several reviews of the pathophysiology and natural history of microvascular complications in people with diabetes (1–3,35) and several reports of complications in people with NIDDM (7,36–46). However, the question of whether there are quantitative differences in complication prevalence between ethnic subpopulations requires the use of similar methods before comparisons can be usefully made. For retinopathy, we are aware of only two population-based studies that have used retinal photographs with which direct comparison may be made: the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR; 47) and the SAHS (30).

WESDR surveyed 10,135 diabetic subjects in 11 central Wisconsin counties and identified a subsample of 1370 people with onset of diabetes after age 29 yr who are similar to those with NIDDM in this study. These subjects were largely non-Hispanic whites. The SAHS identified 206 Hispanic people with previously diagnosed NIDDM from three residential areas in San Antonio; a low-income barrio, a suburban area, and a transitional neighborhood, which was intermediate in sociodemographic characteristics between the other two (48). There were 49 previously diagnosed non-Hispanic whites identified only from the transitional and suburban areas.

The crude prevalence of retinopathy by severity category is summarized in Table 7 for previously diagnosed diabetic subjects from both studies along with our results. The differences in prevalence by severity between Colorado and Wisconsin are small. Colorado's non-Hispanic whites had slight excesses of background retinopathy and deficits of preproliferative and proliferative retinopathy compared to the rates from the Wisconsin study. In contrast, a substantial deficit of preproliferative retinopathy was found in San Antonio's non-Hispanic whites compared to Wisconsin, possibly due to chance because of the small number of non-Hispanic white subjects in the SAHS. There was a clear excess of preproliferative disease in San Antonio Mexican Americans compared with the Wisconsin study and both ethnic groups from the Colorado study.

Direct comparisons must be made with caution, because these data are not duration adjusted and because the definitions of NIDDM differ between the populations. These differences in definition may be important. The crude prevalence ratio is 1.2 for severe retinopathy (preproliferative plus proliferative) among San Antonio Mexican Americans compared with Wisconsin whites, as calculated from Table 7. However, data reported by Haffner et al. (30), based on a detailed comparison of the San Antonio and Wisconsin data, which excluded 474 WESDR subjects who did not meet the SAHS definition of NIDDM, showed the prevalence ratio for the same comparison to be 3.2. Differences between the

TABLE 7
Crude prevalence (per 100) of retinopathy by severity among previously diagnosed people with non-insulin-dependent diabetes mellitus in three studies of diabetic retinopathy

Population	*n*	Retinopathy level			
		None	Background	Preproliferative	Proliferative
Non-Hispanic white					
Wisconsin (47)	1370	45.6	28.7	16.9	8.5
San Antonio (30)	49	65.3	20.4	8.2	6.1
Colorado	85	48.2	32.9	14.1	4.7
Hispanic (Mexican American)					
San Antonio	206	48.1	21.3	23.3	7.3
Colorado	166	57.2	23.5	12.7	6.6

References are in parentheses.

results could be attributable to operational redefinition of NIDDM, so direct comparison must be done only with careful attention to these definitions. We reanalyzed our data with the Wisconsin definition of onset after 29 yr of age (data not shown). Only slight differences from the results shown in Table 7 occurred without a change in the conclusions. In addition, duration-adjusted analysis published previously showed that Hispanics in the San Luis Valley had a significant deficit of retinopathy compared with WESDR subjects but no significant difference was seen between non-Hispanic whites and WESDR subjects (12). Next, with the San Antonio definition of NIDDM (49), we excluded 134 subjects with body mass index of <30 who were on insulin. The prevalence of retinopathy in our study was lower for both Hispanics and non-Hispanic whites with this definition; however, there was still no excess of retinopathy among Hispanics compared with non-Hispanic white subjects in Colorado. Thus, it appears that the use of different definitions of NIDDM is not responsible for the differences in ethnic comparisons between studies. Given the relatively small sample sizes, it would seem prudent to conclude that, although Hispanics in San Antonio may have some excess retinopathy prevalence, this phenomenon is not found in Hispanics in Colorado.

Excess end-stage renal disease has been reported in Mexican Americans from Texas (50), but a report from California has observed a deficit of end-stage renal disease in Hispanics compared with whites (51). Comparable data are not available on end-stage renal disease in other Hispanic populations. Few studies have been published on earlier stages of renal disease, and the number of population-based studies that used measures of kidney disease comparable with those used in the San Luis Valley Diabetes Study is limited. The prevalence of a protein-creatinine ratio >1 in Pima Indians, the population with the highest prevalence of NIDDM in the world (52), ranged from 9.1% in subjects with <5 yr duration to 34.8% for durations of >15 yr (53). Subjects in the San Luis Valley Diabetes Study had a somewhat lower duration-specific prevalence of elevated protein-creatinine ratio (data not shown), although in the longest duration subjects, 26.1% of Hispanics and 31.3% of non-Hispanic whites were affected. Elevated serum creatinine levels were comparable between Hispanics (13%) and Pima Indians (13.8%) of long duration, although Colorado non-Hispanic whites had the highest duration-specific prevalence of elevated creatinine levels (29.4%). In San Antonio, Mexican Americans had more dipstick-positive proteinuria (54) and a higher prevalence of microalbuminuria (>30 mg/L; 31) than non-Hispanic whites. The odds ratio for the prevalence of microalbuminuria for Hispanics versus non-Hispanic whites was higher in San Antonio (odds ratio = 3.54, 95% CI 1.28–9.81) than in Colorado (odds ratio = 1.11, 95% CI 0.54–2.27), and the estimate from San Antonio is outside of the upper confidence limit from Colorado. Because the confidence intervals did overlap, there is a range of elevated risk in Hispanics from 1.28 to 2.27 that could be consistent in both studies. Thus, the difference seen between the two studies may be due to small numbers of diabetic subjects with renal complications in each study. Nonetheless, the estimate from San Antonio is unlikely to be observed in the San Luis Valley population because it is above the upper confidence limit, suggesting that the magnitude of excess microalbuminuria seen in Hispanics in San Antonio is not universally found in all populations of Hispanics. In addition, it is unlikely that the slightly different cut points of albumin concentration (30 vs. 25.5 μg/ml for San Antonio and Colorado, respectively) would be sufficient to account for this difference in the risk of microalbuminuria because there were no consistent ethnic differences at any cut point in the San Luis Valley (Fig. 2). Although the rates of kidney disease in Hispanic diabetic subjects in Colorado are not consistent with the results from Texas, the lack of differences in these parameters among Colorado Hispanics and non-Hispanic whites reported herein and the deficit of end-stage renal disease seen in California Hispanics suggests again that all Hispanics may not be predisposed to more severe complications.

Other studies of kidney function in diabetic populations have been reported (7,41,43,44,55,56); however, the methods for urinary protein measurement have differed from those used in this study. Because of this and differences in patient selection criteria, these studies add little to answer the question of whether Hispanics have similar or excess amounts of diabetic renal involvement.

We are unable to compare our prevalence rates of distal symmetric neuropathy in NIDDM subjects to other studies because no other studies have used our approach to define neuropathy. Only limited data are available on microvascular complications that use comparable methods for direct comparison between Hispanics and other populations. We found no excess of microvascular complications in Hispanic people with NIDDM compared with non-Hispanic whites. Despite agreement between studies that an excess of NIDDM exists in Hispanic compared with non-Hispanic white people, there have been inconsistencies regarding the prevalence of microvascular complications by ethnicity. Given the excess prevalence of complications reported in Mexican Americans in Texas, but no evidence of such an excess in Colorado Hispanics, it seems unlikely that excesses found in Texas could be due to any universal Hispanic genetic predisposition to more severe complications. Rather this may reflect differences in subpopulations that are either associated with medical care (degree of hyperglycemia, blood pressure control) or a function of differences in sample selection, classification, survival, and/or size. If true, this is excellent news for Hispanic people with NIDDM, because it suggests that variations in medical care or other environmental characteristics are important in the determination of the risk of complications, which may be amenable to intervention.

ACKNOWLEDGMENTS

This research was funded by National Institutes of Health Grants DK-30747 and CRC-RR00051.

We thank the residents of Alamosa and Conejos counties, the San Luis Valley Diabetes Study (SLVDS) Advisory Council, medical personnel and staff of the area, the San Luis Valley Area Health Education Center, and the core staff of the SLVDS (Susan Baldwin, Ronna Cochran, Susan Feldman, William Hildebrandt, Olivia Martinez, Rose Romero, Rodney Sandoval, and Joyce Apodaca). The laboratory of the University of Colorado Health Sciences Center General Clinical Research Center conducted most of the blood chemistry analyses. C-peptide levels were determined in the laboratory of Dr. Arthur Rubenstein, University of Chicago. Secretarial help was provided by Gequinn Curry and Pat Cuddy. Programming support was provided by Jeff Amirani, Vince Ulibarri, and Jolene Fukuhara.

This article was presented in abstract form at the National Institutes of Health Workshop on Diabetes in Hispanic Americans, Bethesda, Maryland, May 1988.

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