NIDDM Is the Major Cause of Diabetic End-Stage Renal Disease

More Evidence From a Tri-Ethnic Community

Jacqueline A. Pugh, Rolando A. Medina, John C. Cornell, and Srabashi Basu

Diabetes is the single largest cause of end-stage renal disease (ESRD) in adults in the U.S. Insulin-dependent diabetes mellitus (IDDM) has been recognized for some time as an important cause of ESRD, but non-insulin-dependent diabetes mellitus (NIDDM) has been assumed, until recently, to rarely cause ESRD. The objective of this study is to determine the incidence of treatment of diabetic ESRD by diabetic type for three ethnic/racial groups: non-Hispanic whites, African-Americans, and Mexican-Americans. A population-based incidence cohort was assembled from all dialysis centers in Bexar (San Antonio) and Dallas counties in Texas. All patients with diabetic ESRD beginning dialysis between 1 December 1987 (Bexar) or 1 December 1988 (Dallas) and 31 July 1991 were identified. All non-Hispanic whites and African-Americans and a 1/2 random sample of Mexican-Americans were approached for enrollment. Individuals were confirmed to have diabetes using the World Health Organization criteria. Diabetes typing was done using a computerized historical algorithm. Age-specific and age-adjusted incidence rates were obtained by diabetic type and ethnic/racial group. NIDDM causes the majority of diabetic ESRD: 59.5% for non-Hispanic whites, 92.8% for Mexican-Americans, and 84.3% for African-Americans. Mexican-Americans and African-Americans, respectively, have 6.1 and 6.5 times higher incidence of treatment for diabetic ESRD than non-Hispanic whites. NIDDM results in more ESRD than does IDDM. Minorities (African-Americans and Mexican-Americans) are at increased risk, and programs aimed at prevention of NIDDM-related ESRD must focus on them. *Diabetes* 44:1375–1380, 1995

Diabetes is the single largest cause of end-stage renal disease (ESRD) in adults (1–3), accounting for 33.8% of all new cases in the U.S. between 1988 and 1991 (3). The incidence of both diabetic and hypertensive ESRD is rising at a much faster rate than all other etiologies: 14 and 13% per year, respectively, among whites, 15 and 10% among African-Americans, and 13 and 23% among Native Americans (3). In Colorado between 1982 and 1989, there was a 770% increase in rates of diabetic ESRD among Hispanics compared with a 440% increase among African-Americans and a 190% increase among non-Hispanic whites (4). In Texas, the percentage of Hispanics with ESRD from diabetes is rising, while it is stable for African-Americans and non-Hispanic whites; it rose from 60 to 65% between 1991 and 1994 as compared with a stable 35% for African-Americans and 39% for non-Hispanic whites over the same time frame (5).

A large portion of the continuing rise in diabetic ESRD is in the older age-groups, who are most likely to have non-insulin-dependent diabetes mellitus (NIDDM) (6). The reasons for the continuing rise are multifactorial. First, individuals who previously were not felt to be candidates for dialysis are now being offered dialysis, especially the elderly and those with diabetes. Second, individuals with diabetes are likely to have shared in the decline in coronary disease mortality observed in this country. The decline in coronary mortality translates into a longer life with diabetes and, therefore, increased risk of ESRD, i.e., a decline in competing risks. Third, the prevalence of diabetes increased between 1959 and 1980 and the incidence of diabetes increased between 1980 and 1984; we may be experiencing the remote effects of these previous rises (7).

Insulin-dependent diabetes mellitus (IDDM) has been recognized for some time as an important cause of ESRD. In contrast, NIDDM has been assumed until recently to cause ESRD only rarely. Two cohort studies from Europe from the 1970s identified very few individuals with NIDDM who developed renal failure, confirming the assumption (8,9). As the criteria for use of dialysis has liberalized in the last 20 years, especially for age and diabetes, mounting evidence has shown that NIDDM not only causes ESRD but likely contributes the majority of new cases of diabetic ESRD (6,10,11,12), especially in African-Americans (13,14) and Native Americans (15). Even though the incidence of ESRD among individuals with NIDDM is lower than among individuals with IDDM, NIDDM constitutes 95% of the diabetic population and therefore contributes a greater absolute number of ESRD cases. Further, in some populations, even the incidence of diabetic nephropathy among NIDDM and IDDM may be similar (15,16).

Minorities (African-Americans, Mexican-Americans, Native Americans) are at a higher risk of developing diabetic ESRD than non-Hispanic whites (13–15,17). For African-

From the University of Texas Health Science Center at San Antonio (J.A.P., R.A.M., J.C.C., S.B.), the Mexican American Medical Treatment Effectiveness Research Center (MERECE) (J.A.P., R.A.M., S.B.), the Audie L. Murphy Memorial Veterans Administration Hospital (J.A.P., J.C.C.), and the Geriatric Research, Education, and Clinical Center (J.C.C), San Antonio, Texas.

Address correspondence and reprint requests to Dr. Jacqueline A. Pugh, Ambulatory Care (11C6), Audie L. Murphy Memorial Veterans Hospital, 7400 Merton Minter Blvd., San Antonio, TX 78284.

Received for publication 10 April 1995 and accepted in revised form 10 August 1995.

BMI, body mass index; ESRD, end-stage renal disease; HANES, Health and Nutrition Examination Survey; IDDM, insulin-dependent diabetes mellitus; NIDDM, non-insulin-dependent diabetes mellitus.

Americans and Native Americans, this excess is from NIDDM (13–15). Since Mexican-Americans are known to have at least a twofold higher prevalence of NIDDM than non-Hispanic whites (18–19), we postulated that the observed excess of diabetic ESRD among Mexican-Americans is secondary to NIDDM and further that even after adjusting for the excess underlying prevalence of NIDDM, Mexican-Americans would still have higher rates of diabetic ESRD than non-Hispanic whites.

RESEARCH DESIGN AND METHODS

Hypotheses. *1*) The excess incidence of diabetic ESRD among Mexican-Americans is secondary to NIDDM. *2*) Even after adjusting for the higher underlying prevalence of NIDDM in Mexican-Americans, some excess will still remain.

Design. All new cases of ESRD in Bexar (San Antonio) county between 1 December 1987 and 31 July 1991 and in Dallas county between 1 December 1988 and 31 July 1991 were identified on a monthly basis by dialysis center personnel. All African-Americans and non-Hispanic whites and a 1/2 random sample of Mexican-Americans with diabetic ESRD were approached for enrollment. After informed consent was obtained, the patient was interviewed and signed a release for study personnel to review the patient's medical records. The protocol was approved by the University of Texas Health Science Center Institutional Review Board.

The interview contained questions regarding the subject's diabetic history. The medical record abstracting included both inpatient and outpatient records. We attempted to obtain outpatient records as far back as the diagnosis of diabetes. The abstracting included information about the diagnosis of both diabetes and ESRD.

Individuals were first confirmed to have diabetes using the World Health Organization Criteria (20) of either current or previous treatment with insulin or oral agents or a minimum of two fasting glucose measurements >140 mg/dl or two random glucose measurements >200 mg/dl. (Previous treatment was included because once ESRD develops, need for treatment may be reduced or eliminated by the prolonged half-life of insulin.) For diabetic typing, a modification of the historical algorithm first devised by Cowie et al. (13) was used. The algorithm was run on a microcomputer. Below is the hierarchical algorithm (each subsequent case applies only if all before it have failed). The number of individuals classified by each step is given in parentheses.

1. If the subject has taken insulin, has never been off insulin for 3 months or longer since the first 2 years after diagnosis of diabetes, and has had one or more hospitalizations for diabetic ketoacidosis, the subject has IDDM (n = 84).

2. If the subject has taken insulin, has never been off insulin for 3 months or longer since the first 2 years after diagnosis of diabetes, was <25 years of age at diagnosis, and has body mass index (BMI) <30, the subject has IDDM (n = 21).

3. If the subject has never taken insulin, type is NIDDM (n = 166).

4. If the subject has taken insulin but has been off insulin for 3 months or longer since the first 2 years after diagnosis of diabetes, the subject has NIDDM (n = 264).

5. If age at diagnosis of diabetes was ≥25 years, BMI is ≥30, and there have been no hospitalizations for ketoacidosis, the subject has NIDDM (n = 43).

6. If the subject meets all other criteria in number 5 above but has had one or more episodes of diabetic ketoacidosis secondary to infection, myocardial infarction, or other provocative factors (not including insulin withdrawal), then the subject has NIDDM (n = 1).

7. If the subject was noted to have secondary diabetes (for example from pancreatitis), then the diabetic type is changed to secondary diabetes (n = 0).

TABLE 1
Proportion of cases by diabetic type: San Antonio and Dallas, TX, 1987–1991

	NIDDM	IDDM	Unclassifiable
Whites	59.5 (110) (52.4–66.6)	39.5 (73) (32.4–46.6)	1.1 (2) (0.5–5)
Mexican-Americans	92.8 (246) (89.7–95.9)	4.2 (11) (1.8–6.6)	3.0 (8) (0.9–5)
African-Americans	84.3 (167) (79.2–89.4)	13.1 (26) (8.4–17.8)	2.5 (5) (1–6.5)
Total	80.7 (523)	17.0 (110)	2.3 (15)

Data are % (n) (95% confidence interval). Significance testing of proportions: Non-Hispanic white vs. Mexican-American: NIDDM, $P < 0.001$; IDDM, $P < 0.001$; unclassifiable, $P = 0.14$. Non-Hispanic white vs. African-American: NIDDM, $P < 0.001$; IDDM, $P < 0.001$; unclassifiable, $P = 0.30$. Mexican-American vs. African-American: NIDDM, $P < 0.005$; IDDM, $P = 0.001$; unclassifiable, $P = 0.74$.

If the subject failed the computer algorithm (n = 69), the pertinent facts of the diabetic history were abstracted without ethnic identifiers and reviewed by two endocrinologists and a general internist for typing. All three had to agree on a classification. Only 15 subjects were left unclassified after review.

The etiology of the ESRD was designated by the subject's nephrologist. No individuals with diabetic ESRD had undergone biopsies. Clinical markers for diabetic nephropathy were abstracted in an effort to confirm the diagnosis.

Analysis. Age-specific rates for the three ethnic groups were computed using the 1990 census data for Bexar and Dallas counties. The number of Mexican-American cases was doubled because of the original 1/2 random sample. The Mexican-American population denominator had to be derived from the 1990 census data. The census reports the age distribution of all Hispanics combined and the total number, without age distribution, of individual Hispanic subgroups. We applied the proportional age distribution of the total Hispanic group to the population total for Mexican-Americans. Since Mexican-Americans represent 86% of the Hispanic population in Dallas county and 92% in Bexar county (21), the age distribution of the total Hispanic population for these counties primarily represented the age distribution of the Mexican-American population. Direct age standardization was performed using the 1980 U.S. total population as standard so that rates could be compared with previously published data.

Direct age standardization was also performed using a total diabetic denominator, generating rates for total diabetic ESRD and NIDDM ESRD. The diabetic denominators were derived from the Health and Nutrition Examination Survey (HANES) II (22) and Hispanic HANES (19). Unfortunately, no adult type-specific diabetes prevalence data are available. Since 90–95% of adults with diabetes are estimated to have NIDDM, use of the total diabetic denominator with a NIDDM ESRD numerator yields a reasonable estimate of incidence of ESRD among NIDDM. No rates were calculated using an IDDM denominator because there are no adequate estimates of the prevalence of IDDM in adults. (Two epidemiological studies of diabetes among whites have shown IDDM prevalences of 0.4 and 0.2%, calculated from a total of 8 of 2,240 and 1 of 1,300 individuals each [23,24]. The proportion of the total diabetic cases represented by IDDM was 1% for the Rancho Bernardo Study, but this was limited to older adults, ages 50–89 [25]. One study showed that IDDM represented 13% of the prevalent cases of diabetes in a white community [26]). The method of Kahn (27) for testing statistical significance between age-adjusted rates was used to produce summary z statistics for race/ethnic group rate comparisons.

RESULTS

Table 1 displays the proportion of cases of diabetic ESRD by diabetes type and racial/ethnic group. The majority of diabetic ESRD in all racial/ethnic groups is from NIDDM. The proportion represented by NIDDM is significantly higher for Mexican-Americans and African-Americans as compared with that for non-Hispanic whites.

Figure 1 displays the age-specific rates of treated diabetic ESRD, both types of diabetes combined, by ethnicity. In the

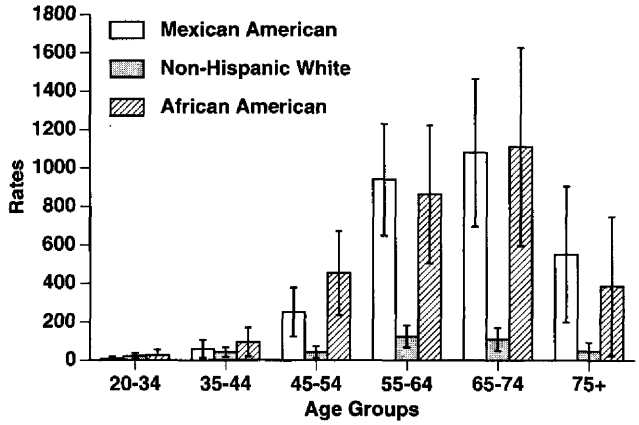

FIG. 1. Age-specific annual incidence of treatment of total diabetic ESRD per 1 million general population: San Antonio and Dallas, TX, 1987–1991.

younger age-groups, rates are similar, but they diverge significantly for African-Americans and Mexican-Americans compared with those for non-Hispanic whites in the older age-groups. Figures 2*A* and 2*B* display the age-specific rates of treated NIDDM- and IDDM-related ESRD, respectively. NIDDM rates show the same pattern as for total diabetic ESRD, with African-Americans and Mexican-Americans having higher rates in the older age-groups. IDDM rates are lower for Mexican-Americans at 45–64 years of age. African-Americans have slightly higher rates than either non-Hispanic whites or Mexican-Americans in the 35- to 44-year-old age-group, and they have markedly higher rates in the 45- to 54-year-old age-group.

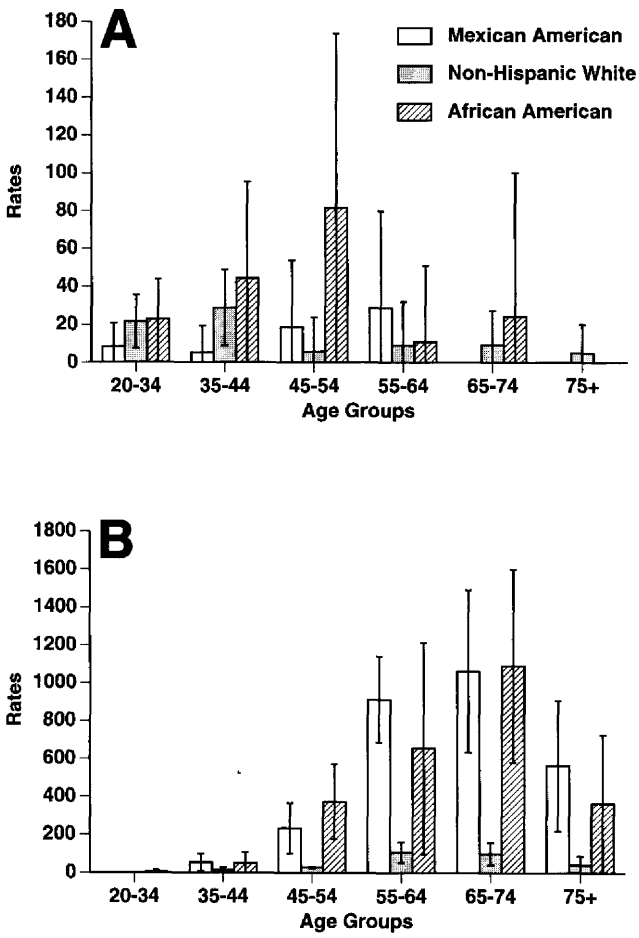

FIG. 2. Age-specific annual incidence of treatment of IDDM (*A*) and NIDDM (*B*) ESRD per 1 million general population: San Antonio and Dallas, TX, 1987–1991.

TABLE 2
Age-adjusted annual incidence of treatment of diabetic ESRD (per 1 million general population): San Antonio and Dallas, TX, 1987–1991

	Rate (95% CI)	Relative risk (95% CI)
Total diabetic ESRD		
Non-Hispanic whites	53.6 (40.2–67.1)	—
African-Americans	350.9 (266.2–435.5)*	6.5 (4.6–9.3)
Mexican-Americans	327.9 (263.2–392.6)*	6.1 (4.4–8.4)
NIDDM ESRD		
Non-Hispanic whites	34.3 (23.4–45.3)	—
African-Americans	318.7 (236.7–400.6)*	9.3 (6.2–14.0)
Mexican-Americans	316.8 (252.9–380.7)*	9.2 (6.3–13.5)
IDDM ESRD		
Non-Hispanic whites	19.3 (11.5–27.1)	—
African-Americans	32.2 (11.0–53.4)	1.7 (0.8–3.6)
Mexican-Americans	11.1 (0.9–21.3)	0.6 (0.2–1.6)

Rate and relative risk for African-Americans and Mexican-Americans are comparisons with non-Hispanic whites. CI, confidence interval. *$P < 0.00001$.

Table 2 displays the annual age-adjusted incidence of all diabetic ESRD as well as NIDDM- and IDDM-related ESRD by racial/ethnic group with a general population denominator. Age adjustment does not diminish the ethnic/racial differences. However, both of these ethnic groups have higher underlying prevalences of NIDDM. In order to adjust for these higher underlying prevalences of NIDDM, incidence rates using a diabetic population denominator were also calculated.

The age-adjusted NIDDM-related ESRD incidence using the diabetic population denominator continues to show a significant excess for both Mexican-Americans ($P = 0.001$) and African-Americans ($P = 0.0001$) compared with non-Hispanic whites, despite the adjustment for the increased underlying NIDDM prevalence (Table 3). The risk ratios for total diabetic ESRD are 0.9 for Mexican-Americans and 1.5 for African-Americans and for NIDDM ESRD are 2.5 for Mexican-Americans and 3.8 for African-Americans.

Since none of the diabetic ESRD patients identified in this

TABLE 3
Age-adjusted annual incidence of treatment of diabetic ESRD (per 100,000 diabetic population): San Antonio and Dallas, TX, 1987–1991

	Rate (95% CI)	Relative risk (95% CI)
Total diabetic ESRD		
Non-Hispanic whites	125.5 (83.8–167.2)	—
African-Americans	193.7 (141.2–246.2)*	1.5 (1.0–2.4)
Mexican-Americans	117.8 (87.8–147.8)	0.9 (0.6–1.4)
NIDDM ESRD		
Non-Hispanic whites	41.5 (22.6–60.4)	—
African-Americans	155.7 (114.5–196.9)‡	3.8 (2.2–6.4)
Mexican-Americans	103.8 (77.3–130.4)†	2.5 (1.5–4.2)
IDDM ESRD		
Non-Hispanic whites	84.0 (46.8–121.2)	—
African-Americans	38.0 (5.45–70.6)	0.45 (0.2–1.2)
Mexican-Americans	14.0 (0–28.0)†	0.2 (0.1–0.5)

Rate and relative risk for African-Americans and Mexican-Americans are comparisons with non-Hispanic whites. CI, confidence interval. *$P < 0.05$; †$P < 0.001$; ‡$P < 0.0001$.

TABLE 4
Diagnostic parameters for diabetic nephropathy: San Antonio and Dallas, TX, 1987–1991

	IDDM			NIDDM		
	Non-Hispanic whites	Mexican-Americans	African-Americans	Non-Hispanic whites	Mexican-Americans	African-Americans
Diabetes duration ≥10 years	100.00 (72/72)*	100.00 (10/10)	92.31 (24/26)	85.32 (93/109)	91.74 (222/242)	82.53 (137/166)
Retinopathy	95.77 (68/71)	100.00 (10/10)	96.15 (25/26)	82.41 (89/108)†	94.96 (226/238)	74.38 (119/160)‡
Proteinuria	75.00 (51/68)	70.00 (7/10)	91.67 (22/24)	80.73 (88/109)	73.86 (178/241)	77.30 (126/163)
Kidney size ≥9 cm	65.28 (47/72)	90.00 (9/10)	73.08 (19/26)	67.89 (74/109)	66.53 (161/242)	74.70 (124/166)
Slow decline of renal function	93.05 (67/72)	100.00 (10/10)	100.00 (26/26)	94.50 (103/109)	92.50 (224/242)	96.39 (160/166)
Absence of hematuria	61.19 (41/67)	50.00 (5/10)	70.83 (17/24)	62.39 (68/109)	60.67 (145/239)	58.39 (94/161)
Absence of renal cell casts	82.35 (56/68)*	90.00 (9/10)	100.00 (24/24)	83.48 (91/109)	79.92 (191/239)*	87.58 (141/161)

Data are % (n). Retinopathy includes coexistent retinopathy of any degree. Proteinuria includes ≥G/24 h or two urinalyses with 1+ or greater protein, in the absence of hematuria, red blood cell casts, or renal cell casts. Slow decline of renal function is no serum creatinine ≤132 μmol/l within 1 year before the date of first chronic dialysis. *$P < 0.05$ compared with African-Americans. †$P < 0.01$ compared with Mexican-Americans. ‡$P < 0.001$ compared with Mexican-Americans.

study had a renal biopsy, other parameters were used to assess how certain we were that they did indeed have diabetic ESRD. Table 4 displays the percentage of both the IDDM and the NIDDM groups that have parameters felt to be consistent with a diagnosis of diabetic nephropathy. These parameters include duration of diabetes of ≥10 years, coexistent retinopathy of any degree, proteinuria of ≥2,000 mg/24 h or two urinalyses with 1+ or greater protein, kidney size ≥9 cm, slow rate of renal function decline (no creatinine ≤1.5 within 1 year before the first date of chronic dialysis), and absence of hematuria or renal cell casts. These criteria were chosen from texts and literature as representing criteria that should be met to make the clinical diagnosis of diabetic nephropathy. For IDDM patients, duration of diabetes was met by virtually everyone, whereas for NIDDM patients, 9–17% had a duration <10 years. This is not surprising given the average estimated 7 years of diabetes duration before the diagnosis in NIDDM (28). Similarly, >95% of IDDM patients had some retinopathy, while 6–25% of NIDDM patients did not have retinopathy. Interestingly, the percentage of NIDDM patients with retinopathy varied between ethnic groups, with only 6% of non-Hispanic whites, compared with 25% of African-Americans, lacking retinopathy. The proteinuria and kidney-size criteria were less consistently met by both IDDM and NIDDM patients. The slow decline in renal function was met by the vast majority of both IDDM and NIDDM patients. Of IDDM and NIDDM patients, ~40% had hematuria. Absence of renal casts was met by the majority of NIDDM and IDDM patients. Few patients met all criteria. Without biopsy, we can only say that the majority of the patients identified had ESRD associated with long duration of diabetes, meeting some but not all of the sought after clinical criteria, and that NIDDM patients were less likely to meet the criteria than IDDM patients.

DISCUSSION

The data presented here add to a growing body of evidence documenting that NIDDM is the dominant cause of diabetic ESRD (10–15). A higher percentage of patients with IDDM may develop diabetic ESRD, but because the NIDDM population is so much larger than the IDDM population, it contributes a larger percentage of the total cases of diabetic ESRD. Further, in some ethnic groups, the rates among NIDDM patients may be as high as the rates observed among IDDM patients in other ethnic groups (15,16). The importance of these findings to clinical practice is enormous; NIDDM is not a benign disease to the kidneys, and prevention strategies must be formulated. Glycemic goals for NIDDM patients may not currently be low enough for prevention of microvascular complications, especially if the results of the Diabetes Complications and Control Trial are extended to NIDDM.

The continuing rise in the incidence of treatment of diabetic ESRD is multifactorial. Explanations include *1*) a more liberalized policy for admission to dialysis treatment than that of 15 years ago, especially for elderly individuals and individuals with diabetes; *2*) a decline in coronary artery disease mortality, thereby decreasing competing risks and increasing the duration of diabetes; *3*) a previous increase in the prevalence of diabetes itself now reflected in increased ESRD; *4*) high population growth rate in high-risk populations, such as Mexican-Americans; or *5*) a relative increase in minorities being treated for ESRD. Both primary (diabetes itself) and secondary (renal disease) prevention will be required to slow this rate of increase.

Despite different genetic backgrounds, minorities (Mexican-Americans, African-Americans, and Native Americans) in the U.S. share an increased propensity to develop diabetic ESRD. What is the link? One possible explanation is similar social and health care situations that lead to either poorer compliance or poorer accessibility of adequate treatment for diabetes and hypertension (29–32). Further study is necessary to tease out the contributions of genetic versus environmental, especially social, risks for diabetic ESRD.

The rates presented here rely on the attending nephrologist to assign etiology of renal disease. The amount of misclassification of etiology of ESRD is not known. Once ESRD occurs, there is little motivation from the clinical viewpoint to establish the etiology of ESRD with a high degree of certainty. Etiology will not change treatment at that point. Not only were biopsies not available, but few diagnostic tests (renal ultrasound, 24-h urine, etc.) were performed. Further, the diagnostic tests, short of biopsy, chosen by us to study misclassification were not as helpful as first thought. For example, a large proportion of the hypertensive patients had significant levels of proteinuria and the overlap of kidney sizes between hypertensive and diabetic ESRD was considerable. In fact, if a biopsy study were performed, one might find that NIDDM patients are likely to have a mixed hypertensive/diabetic lesion rather than a diabetic lesion alone.

Finally, our study looks only at the incidence of treated

ESRD, not total ESRD. This is also true of the vast majority of studies of ESRD because community surveillance for cases of untreated ESRD would be very expensive and time-consuming. We were not able to address whether there was differential treatment of minorities versus non-Hispanic whites but similar underlying rates of ESRD (treated plus untreated). Age, co-morbidities, availability of treatment facilities and personnel, and patient and family choice all contribute to the decision to start dialysis. It is possible that total ESRD, treated and untreated combined, may be the same in both minorities and non-Hispanic whites but that minority individuals are more likely to receive treatment for their diabetic ESRD than non-Hispanic whites. We doubt this to be the case because there are few other medical treatments (of which we are aware) in which minorities have been shown to have higher rates of treatment than whites (33). In fact, African-Americans have been shown to receive less treatment for such conditions as coronary artery disease (34–35) and Hispanics have been shown to receive less treatment for pain (36). We also doubt that the differences in rates could all be explained by patient choice, with large numbers of non-Hispanic whites choosing not to be dialyzed.

In summary, NIDDM is the major cause of diabetic ESRD. Minorities are at increased risk for NIDDM-related ESRD, and more work needs to be done to elucidate the mechanisms for this higher risk.

ACKNOWLEDGMENTS

This work was supported by grants from the RGK Foundation, National Institute of Diabetes and Digestive and Kidney Diseases (RO1-DK38392), and Agency for Health Care Policy and Research (UO1-HS07397). R.A.M. was supported by a minority faculty supplement (R01-DK38392–05S1). J.C.C. is supported by the Geriatric Research, Education, and Clinical Center at the Audie L. Murphy Memorial Veterans Hospital.

We are indebted to Drs. Marcelo Perez-Montes, Manuel Ramirez III, and Nauman Jameel for their assistance in compiling the data.

APPENDIX

We gratefully acknowledge the participation of the following dialysis centers and institutions in the identification of incident cases of ESRD: In Bexar county: Audie L. Murphy Memorial Veterans Administration Hospital, Bexar County Hospital District Medical Center Hospital, Santa Rosa Medical Center, Kidney Disease Clinic of San Antonio, San Antonio Kidney Disease Center, Southside Kidney Disease Center, Northeast Kidney Disease Center, Maldonado Eastside Kidney Center, Southwest Dialysis Center, Kidney Disease Clinic of Central San Antonio, Community Dialysis of San Antonio, Community Dialysis Southwest San Antonio, Rosedale Kidney Disease Center, Kidney Treatment Center, Miller Kidney Health Center, and Northwest Kidney Disease Center. In Dallas county: Dallas Nephrology Associates, Irving Dialysis Center, Mid-Cities Dialysis Center, Parkland Memorial Hospital, Veterans Administration Hospital, Metro South Dialysis Center, Oak Cliff Dialysis Center, Dallas North Dialysis Center, Southwestern Dialysis Center, Dallas Kidney Disease Center, Mesquite Dialysis Center, Plano Dialysis Center, Garland Dialysis Center, Arlington Dialysis Center, Lewisville Kidney Health Care, Quality Care Dialysis Center, and Northwest Dallas Dialysis Center.

REFERENCES

1. Rosansky SJ, Eggers PW: Trends in the U.S. end-stage renal disease population: 1973–1983. *Am J Kidney Dis* 9:91–97, 1987
2. Brunner FP, Brynger H, Challah S, Fassbinder W, Geerlings W, Selwood NH, Tufveson G, Wing AJ: Renal replacement therapy in patients with diabetic nephropathy, 1980–1985. *Nephrol Dial Transplant* 3:585–595, 1988
3. U.S. Renal Data System: *U.S. Renal Data System 1994 Annual Report.* Bethesda, MD, National Institutes of Health, National Instutute of Diabetes and Digestive and Kidney Diseases, Division of Kidney, Urologic, and Hematologic Diseases, 1994
4. Hamman RF, Turak A, Stiles SK, Finucane FF, Micahel SL, Garrett CJ, Meng C, Gabella BA: Incidence of treatment for end-stage renal disease attributed to diabetes mellitus, by race/ethnicity-Colorado, 1982–1989. *Morb and Mortal Wkly Rev* 41:845–847, 1992
5. Texas Department of Health Kidney Health Program: *Annual Reports.* Austin, TX, Texas Department of Health, 1991, 1992, 1993, 1994
6. Eggers PW: Health care policies/economics of the geriatric renal population. *Am J Kidney Dis* 16:384–391, 1990
7. Geiss L (Ed.): *Diabetes Surveillance 1993.* Atlanta, GA, Centers for Disease Control and Prevention, 1993
8. Mogensen CE: Microalbuminuria predicts clinical proteinuria and early mortality in maturity-onset diabetes. *N Engl J Med* 310:356–360, 1984
9. Fabre J, Balant LP, Dayer PG, Fox HM, Vernet AT: The kidney in maturity onset diabetes mellitus: a clinical study of 510 patients. *Kidney Int* 21:730–738, 1982
10. Humphrey LL, Ballard DJ, Frohnert PP, Chu C-P, O'Fallon WM, Palumbo PH: Chronic renal failure in non-insulin dependent diabetes mellitus: a population-based study in Rochester, Minnesota. *Ann Intern Med* III: 788–796, 1989
11. Cordonnier D, Janbon B, Guiserix J, Ledoux F, Balducci F, Zmirou D: Important prevalence of type 2 diabetes mellitus in dialysed uremic patients in French departments and overseas territories (Letter). *Presse Med* 21:1913, 1992
12. Rettig B, Teutsch SM: The incidence of end-stage renal disease in type I and type II diabetes mellitus. *Diabetic Nephrop* 3:26–27, 1984
13. Cowie CC, Port FK, Wolfe RA, Savage PJ, Moll PP, Hawthorne VM: Disparities in incidence of diabetic end-stage renal disease according to race and type of diabetes. *N Engl J Med* 321:1074–1079, 1989
14. Stephens GW, Gillespy JA, Clyne D, Mejia A, Pollak VE: Racial differences in the incidence of end-stage renal disease in types I and II diabetes mellitus. *Am J Kidney Dis* 15:562–567, 1990
15. Nelson RG, Newman JM, Knowler WC: Incidence of end-stage renal disease in type 2 (non-insulin-dependent) diabetes mellitus in Pima Indians. *Diabetologia* 31:730–736, 1988
16. Hasslacher CH, Ritz E, Wahl P, Michael C: Similar risks of nephropathy in patients with type I or type II diabetes mellitus. *Nephrol Dial Transplant* 4:859–863, 1989
17. Pugh JA, Stern MP, Haffner SM, Eifler CW, Zapata M: Excess incidence of treatment of end-stage renal disease in Mexican Americans. *Am J Epidemiol* 127:135–144, 1988
18. Stern, MP, Gaskill SP, Allen CR, Garza V, Gonzales JL, Waldrop RH: Cardiovascular risk factors in Mexican Americans in Laredo, Texas. *Am J Epidemiol* 113:546–555, 1981
19. Flegal KM, Ezzah TM, Harris MI, Haynes SG, Juarez RZ, Knowler WC, Perez-Stable EJ, Stern MP: Prevalence of diabetes in Mexican-Americans, Cubans, and Puerto Ricans from the Hispanic Health and Nutrition Examination Survey 1982–1984. *Diabetes Care* 14:628–638, 1991
20. Harris MI: Classification and diagnostic criteria for diabetes mellitus and other categories of glucose intolerance. *Primary Care* 15:205–225, 1988
21. Texas State Data Center: *1990 Census Data for Dallas and Bexar Counties.* College Station, TX, Texas Department of Commerce and the Department of Rural Sociology, Texas A & M Univ., 1992
22. Harris MI: Prevalence of non-insulin-dependent diabetes and impaired glucose tolerance. In *Diabetes in America.* Bethesda, MD, National Institutes of Health, 1984 (NIH publ. no. 85-1468) p. VI1–VI31
23. French R, Boen JR, Martinez AM, Bushhouse SA, Sprafka M, Goetz FC: Population-based study of impaired glucose tolerance and type II Diabetes in Wadena, Minnesota. *Diabetes* 39:1131–1137, 1990
24. Mykkänen L, Laakso M, Uusitupa N, Pyörälä K: Prevalence of diabetes and impaired glucose tolerance in elderly subjects and their association with obesity and family history of diabetes. *Diabetes Care* 13:1099–1105, 1990
25. Wingard DL, Sinsheimer P, Barrett EL, McPhillips JB: Community-based study of prevalence of NIDDM in older adults. *Diabetes Care* 13:3–8, 1990
26. Ballard DJ: *Selection in Referral for Medical Care: Impact on Assessment of Clinical Spectrum and Survival in Diabetes Mellitus.* Doctoral Dissertation in Epidemiology. Chapel Hill, University of North Carolina-Chapel Hill, 1990
27. Kahn HA: *An Introduction to Epidemiologic Methods.* New York, Oxford Univ. Press, 1983, p. 64–71
28. Harris MI: Undiagnosed NIDDM: clinical and public health issues. *Diabetes Care* 16:642–652, 1993
29. Anderson R, Lewis SZ, Giachello AL, Aday LA, Chiu G: Access to medical care among the Hispanic population of the southwestern United States. *J Health Soc Behav* 22:78–79, 1981

30. Pugh JA, Tuley MR, Hazuda HP, Stern MP: The influence of outpatient insurance coverage on the microvascular complications of non-insulin-dependent diabetes in Mexican Americans. *J Diabetes and Its Complications* 6:236–241, 1992
31. Weisfeld VD (Ed.): *Access to Health Care in the United States: Results of a 1986 Survey*. Princeton, NJ, Robert Wood Johnson Foundation, 1987
32. Moore P: Utilization of ambulatory health care services by Hispanics. *NTIS Pub* PB385–243096/AS, 1986
33. Gibaldi M: Ethnic differences in the assessment and treatment of disease. *Pharmacotherapy* 13:170–176, 1993
34. Hannan EL, Kilburn H, O'Donnell JF, Lukacik G, Shields EP: Interracial access to selected cardiac procedures for patients hospitalized with coronary artery disease in New York State. *Medical Care* 29:430–441, 1991
35. Carlisle DM, Leake BD, Shapiro MF: Racial and ethnic differences in the use of invasive cardiac procedures among cardiac patients in Los Angeles County, 1986 through 1988. *Am J Public Health* 85:352–356, 1995
36. Todd KH, Samaroo N, Hoffman JR: Ethnicity as a risk factor for inadequate emergency department analgesia. *JAMA* 269:1537–1539, 1993