Diabetic Nephropathy and Microalbuminuria in the Community

The South Auckland Diabetes Survey

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OBJECTIVE — To compare the clinical, anthropometric, and metabolic characteristics of New Zealand Europeans, Maori, and Pacific Islanders with non-insulin-dependent diabetes mellitus (NIDDM) with emphasis on risk factors for the development of diabetic nephropathy.

RESEARCH DESIGN AND METHODS — A cross-sectional survey of 555 (74% of 750 available) diabetic patients attending diabetes clinics and randomly selected primary care centers was conducted in Auckland, New Zealand.

RESULTS — Among those with NIDDM, Maori and Pacific Islanders were younger at diagnosis, more obese, and had poorer glucose control when compared with the Europeans (fructosamine in μmol/l: Maori 335 ± 78, Pacific Islanders 367 ± 90, Europeans 318 ± 55; overall $P < 0.001$). Systolic blood pressure (sBP) was higher in Maori (145 ± 31 mmHg) and lower in Pacific Islanders (135 ± 25 mmHg) when compared with Europeans (141 ± 25 mmHg; overall $P < 0.005$). Mean estimated daily urinary albumin excretion (UAE) was 18.2 (15.5–1.3) mg/day in Europeans, 94.8 (60.5–148.7) mg/day in Maori, and 44.2 (32.3–60.3) mg/day in Pacific Islanders. The prevalence of proteinuria and end-stage renal failure were also higher in Maori and Pacific Islanders. The excess prevalence of microalbuminuria and proteinuria in Maori was present within 5 years of diagnosis. Europeans with impaired renal function were least likely to have associated proteinuria or microalbuminuria. Microalbuminuria and nephropathy were not consistently associated with either higher blood pressure or worse glucose control.

CONCLUSIONS — NIDDM in Maori and Pacific Islanders is associated with a greater degree of proteinuria and end-stage renal failure than that in Europeans. This observation is not explained by conventional risk factors.

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NIDDM, non-insulin-dependent diabetes mellitus; SADC, South Auckland Diabetes Center; UAE, urinary albumin excretion; AER, albumin excretion rate; IDDM, insulin-dependent diabetes mellitus; MANOVA, multiple analysis of variance; CI, confidence interval; HDL, high-density lipoprotein; BMI, body mass index; WHR, waist-to-hip ratio; sBP, systolic blood pressure; dBP, diastolic blood pressure.

Auckland contains the largest urban Polynesian population in the world. It includes New Zealand Maori, Western Samoans, Tongans, Niueans, and Cook Islands Maori. The city is currently experiencing a 10% per annum increase in demand for treatment for end-stage renal failure, mainly due to Polynesians with non-insulin-dependent diabetes mellitus (NIDDM) needing dialysis (unpublished data). In comparison with New Zealand Europeans, Polynesians have an excess of NIDDM (1), more microalbuminuria and proteinuria with their NIDDM, assessed while attending a hospital clinic (2), more admissions for chronic renal failure associated with diabetes overall (3), and more reported end-stage renal failure due to all diabetes on death certification (4).

Our current study provides a cross-sectional comparison of microalbuminuria and diabetic nephropathy in European, Maori, and Pacific Islanders with NIDDM, from both hospital and community sources. The prevalence and risk factors for microalbuminuria, proteinuria, and end-stage renal failure in NIDDM are presented.

RESEARCH AND METHODS —

South Auckland has a population of 303,000 of whom 49,000 are Maori, 45,000 are of Pacific Island origin, and the remainder are predominantly Europeans (5). The area has high unemployment and population mobility (6). Diabetes care is provided by general practitioners who may refer patients to the Middlemore Hospital diabetes clinic (Whitiora), which provides a diabetes education program, or to a diabetes education and monitoring center run by diabetes nurse specialists and Pacific Island and Maori lay educators (South Auckland Diabetes Center, SADC).

All patients between 18 and 79 years old who attended Whitiora or SADC between June and November 1990 were invited into the study. The general practitioner sample was generated by ran-

domly selecting 62 of the 188 practitioners in South Auckland. The sampling frame was stratified according to the number of general practitioners within each of nine subdistricts. All patients visiting the general practitioners for a 17-week period between March and June 1991 were placed on a register and invited to participate in the study.

Interviews were conducted between 0700 and 1700 at local community venues (e.g., church halls, community houses). Subjects were asked to bring their medications and to continue with their established treatment regimen. A questionnaire was completed (with an interpreter when necessary). Questions included basic demography, medical, renal, and diabetic history. Ethnic group was assigned by ethnic self-identity; the method used by the New Zealand Government Department of Statistics for recent national censuses. Patients had their blood pressure taken with a random zero sphygmomanometer in the sitting position, according to a standard protocol, by one observer. Weight and height were taken in light clothes without shoes. Waist and hip circumferences were taken with waist defined as the minimum girth between the costal margin and iliac crests and the hip defined as that at the level of the greater trochanters. A casual blood sample was taken.

Before attending their review, subjects were asked to collect one urine sample as soon as they arose on the morning of the day of the interview. Urinary albumin excretion (UAE), using fresh samples, was measured by immunoturbidimetric assay (Cambridge Life Science, Cambridge, U.K.) on a Cobas Fara centrifugal analyzer (Roche, Berne, Switzerland). Urine results from those with significant infection were excluded from analyses. Serum fructosamine, glucose, creatinine and urate, and urinary creatinine concentrations were measured by commercial methods using a centrifugal analyzer. End-stage renal failure was considered to have occurred in those receiving dialysis, who had received a kidney transplant, or who had a serum creatinine concentration >0.3 mmol/l. Impaired renal function was considered to be present if the serum creatinine was ≥0.12 mmol/l in males and ≥0.10 mmol/l in females.

The UAE was initially expressed as the urinary albumin:creatinine ratio in mg/mmol (normal <2.5). An estimate of the daily albumin excretion rate (AER_{est}; mg/day) was made using predictions of daily urine creatinine excretion (mmol/day) based on the age, sex, and weight of the subject derived from the Cockcroft and Gault equations (7) as suggested by Ginsberg et al. (8) and Cundy et al. (9).

Predicted urine creatinine =

$$\frac{(140 - \text{age}) \times \text{weight}}{K}$$

where K = 665 for men and 782 for women and AER_{est} = predicted urine creatinine excretion × albumin:creatinine ratio. Those with an estimated daily UAE of 30–299 mg/day were considered to have microalbuminuria, and those at ≥300 mg/day were considered to have proteinuria. Insulin-dependent diabetes mellitus (IDDM) was considered to be present in those currently on insulin who had a history of ketoacidosis or who began taking insulin within 4 weeks of onset of severe symptoms (particularly weight loss) (10).

Data was analyzed using SPSS-PC (SPSS, Chicago, IL). All analyses were two-tailed, with a 5% level taken as significant. Continuous variables were compared using multiple analysis of variance (MANOVA) with visits to Whitiora as a covariate (patients were referred to Whitiora from the other two sample sources). Duncan's multiple range test was used as a post hoc–test if the analysis of variance showed significant ethnic differences. Discrete variables were adjusted for recruitment center by direct standardization. Simple comparisons of proportions were tested using the χ^2 test. Variables with a non-normal distribution (e.g., albumin:creatinine ratio, creatinine) were logarithmically transformed for analysis, and geometric mean and 95% confidence intervals (CIs) are shown. All variables (except ethnic group) were entered into a stepwise-multivariate regression to describe the proportion of variance accounted for by the entered variables. Dummy variables were entered as necessary and interaction terms were not included. Maori ethnicity and then Pacific Islander ethnicity were added to the regression to see if any further variance was accounted for. The study was approved by the University of Auckland Human Subjects Ethics Committee.

RESULTS— Overall, 932 patients were invited into the study, of whom 479 were European, 172 were Maori, 249 were of Pacific Islands origin, and 32 were of other origins (e.g., Chinese, Indians). Twelve patients died, 94 moved between identification and invitation, and 76 were never contacted after repeated attempts (including house visits). (11.3% Europeans [n = 54], 25.6% Maori [n = 44], 30.1% Pacific Islanders [n = 75] [χ^2 = 104.3, $P < 0.001$], and 28.1% others [n = 9].) Of the remaining 750 patients, 555 (74.0%) were interviewed, 147 (19.6%) refused to be involved, and the remainder did not attend. Although there were no significant sex or ethnic group differences in attendance among those contacted, in European and Pacific Island patients, those attending were older than those who were not seen.

IDDM was present in 7.3% (n = 34) of Europeans, 1.2% (n = 2) of Maori, but in no Pacific Islanders (χ^2 = 21.39, $P < 0.001$). These patients have been excluded from further analyses.

Patients with NIDDM from the different ethnic groups had different clinical profiles as shown in Table 1. Europeans were between 20 and 80 years old; Maori and Pacific Islanders were between 21 and 74 years old. The age at which diabetes was diagnosed was higher in Europeans (55 [95% CI 53–56] years) than in Pacific Islanders (49 [95% CI 47–51] years) and lowest in Maori (43 [95% CI

Table 1—*Characteristics of NIDDM subjects*

	European	Maori	Islander	*P* across all ethnic groups
n	297	84	123	
Age (years)	62 ± 10*	53 ± 9*	56 ± 9*	<0.001
Diabetes duration (years)	5 (0–47)	6 (0–34)	4 (0–32)	NS
BMI (kg/m^2)	30.5 ± 5.6*	33.3 ± 6.8	33.4 ± 5.8	<0.001
WHR	0.91 ± 0.09	0.90 ± 0.08	0.97 ± 0.08*	<0.001
Random glucose (mmol/l)	10.3 ± 5.0*	11.8 ± 4.8	11.6 ± 5.8	<0.05
Fructosamine (μmol/l)	318 ± 55*	335 ± 78	367 ± 90	<0.001
Total cholesterol (mmol/l)	6.0 ± 1.3	6.2 ± 1.5	5.8 ± 1.3	NS
HDL cholesterol (mmol/l)	1.1 ± 0.3*	1.0 ± 0.3	1.0 ± 0.2	<0.05
sBP (mmHg)	141 ± 25	145 ± 31*	135 ± 24	<0.005
dBP (mmHg)	81 ± 12	84 ± 13	80 ± 13	NS
Urate (mmol/l)	0.31 ± 0.08*	0.38 ± 0.10	0.39 ± 0.10	<0.001
Ever smoked (%)	56.8	72.5	46.1	<0.001

Data are means ± SD adjusted for recruitment center. BMI, WHR, random glucose, fructosamine, total cholesterol, sBP, dBP, and Urate are adjusted for age and sex. Ever smoked combines current and past smokers. *$P < 0.05$ vs. other two ethnic groups by Duncan's test.

41–45] years). There was no overall difference in duration of diagnosed diabetes between ethnic groups. Europeans were the most lean, with the lowest fructosamine, glucose, and urate, and highest high-density lipoprotein (HDL) concentrations.

Maori and Pacific Islanders had similar body mass index (BMI), urate, HDL cholesterol, fructosamine, and casual glucose concentrations. Pacific Islanders had a markedly higher waist-to-hip ratio (WHR) than the other ethnic groups. Maori had the highest current systolic blood pressure (sBP) and were most likely to have ever smoked. Of similar proportions within each ethnic group was a past history of renal disease (e.g., pyelonephritis, tuberculosis, malformations, nephrolithiasis: 13.8% Europeans; 17.6% Maori; 13.0% Pacific Islanders).

Major ethnic group differences in the prevalence and pattern of nephropathy were found. Table 2 shows that while mean creatinine concentrations were similar (after exclusion of those with end-stage renal failure), Maori and then Pacific Islanders had a higher estimated AER than Europeans. Table 3 shows that Maori and Pacific Islanders had more end-stage renal failure, proteinuria, and microalbuminuria than Europeans. In those with impaired renal function, proteinuria was significantly more likely among Maori (odds ratio 5.7 [95% CI 2.2–15.1]) and Pacific Islanders (3.1 [95% CI 1.1–8.8]) than Europeans. Of those with end-stage renal failure ($n = 9$), two were on no treatment, one was receiving hemodialysis, two had received transplants, and the remainder were on continuous ambulatory peritoneal dialysis.

Figure 1 shows that the overall prevalence of microalbuminuria and proteinuria was higher in Maori and Pacific Islanders within 5 years of diagnosis. Figures 2 and 3 show the higher prevalence of proteinuria in Maori and Pacific Islanders with time since diagnosis and with increasing sBP, respectively. Table 4 compares those with and without microalbuminuria, proteinuria, impaired renal function, or end-stage renal failure. In all ethnic groups, the WHR was higher (not significant in Maori, $P = 0.08$) and the sBP was higher (only significant in Europeans, $P < 0.05$) in those with microalbuminuria or nephropathy (not significant in Maori, $P = 0.08$). Among Europeans, those with microalbuminuria or nephropathy also had a higher BMI and were more likely to be current or ex-smokers. In Maori and Pacific Islanders, but not Europeans, those with microalbuminuria and nephropathy were diagnosed at a significantly younger age (and had a longer duration of diagnosed diabetes) than the others. Measures of glycemia were not higher in patients with microalbuminuria and nephropathy.

Age, age at diagnosis, log(duration of diabetes), sBP, diastolic blood pressure (dBP), fructosamine, total and HDL cholesterol, BMI, WHR, height, receipt of antihypertensive therapy, attendance at Whitiora, ever smoked, and sex categories were entered into a stepwise-multivariate regression. BMI, duration of

Table 2—*Renal characteristics of NIDDM subjects*

	European	Maori	Pacific Islander	*P* across all ethnic groups
Creatinine (mmol/l)	0.094 (0.093–0.097)	0.096 (0.091–0.101)	0.096 (0.092–0.100)	NS
Albumin:creatinine ratio (mg/mmol)	2.18 (1.87–2.55)	9.06 (5.85–14.04)	4.38 (3.21–5.98)	<0.001
Estimated albumin excretion (mg/day)	18.2 (15.5–21.3)	94.8 (60.5–148.7)	44.2 (32.3–60.3)	<0.001

Data are geometric mean (95% CI). Patients with end-stage renal failure excluded from creatinine measurement. All data adjusted for age, sex, and recruitment center.

Table 3—*Prevalence of microalbuminuria and nephropathy in NIDDM subjects*

	European	Maori	Islander	P
End-stage renal failure	0.3	4.7	3.3	<0.05
Proteinuria (overall)	5.4	30.2	13.0	<0.001
Microalbuminuria (overall)	22.1	26.7	33.3	
Impaired renal function	18.1	20.9	21.1	
With proteinuria	9.4	53.8	29.2	<0.001
With microalbuminuria	28.3	15.4	54.2	
No microalbuminuria	72.3	30.8	16.6	
No microalbuminuria and normal creatinine	60.9	31.4	45.5	

Data are % and are not age-adjusted. Those with impaired renal function include men with a serum creatinine ≥0.12 mmol/l and women with a serum creatinine ≥0.10 mmol/l.

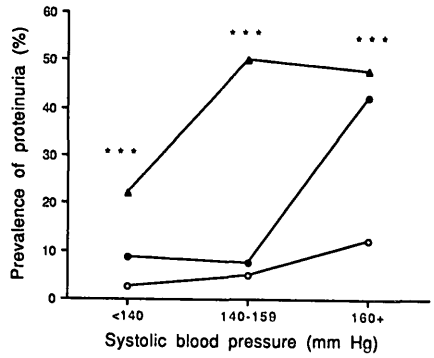

Figure 3—*Proportion with proteinuria by sBP and ethnic group. ○, Europeans; ▲, Maori; ●, Pacific Islanders. ***P < 0.001 across the three ethnic groups.*

diagnosed diabetes, WHR, ever smoked, age at diagnosis, and sBP explained 21.0% of the variance. On entering Maori ethnicity into the regression, a further 4.9% of variance was explained ($P < 0.001$). Pacific Islander ethnicity was then entered: this explained a further 2.7% of the variance ($P < 0.001$; total variance explained 28.6%). Only age at diagnosis dropped out of the regression on entry of ethnic group.

CONCLUSIONS— The high proportion of Maori and Pacific Islanders with NIDDM and end-stage renal failure is consistent with published New Zealand death certificate data (4). It is also consistent with the high incidence of end-stage renal failure in other ethnic groups at high risk of NIDDM (e.g., South Asians [11], Mexican-Americans [12], Pima Indians [13], and Black Americans [14]). The prevalence of end-stage renal failure among Europeans with NIDDM was similar in this study as found elsewhere (0.2%) (15). Ethnic differences in the incidence of proteinuria in NIDDM also exist. While 65% of Pima Indians develop nephropathy (16), only 14% of Europeans with NIDDM do so (15). In Europeans, nephropathy is more common in IDDM with its development in 30–40% of patients (17). This difference between IDDM and NIDDM in Europeans is reflected in the predictive nature of microalbuminuria for proteinuria in 87.5% of patients with IDDM (18), but in only 22% of NIDDM patients (19). In the latter, microalbuminuria is a predictor of cardiovascular mortality rather than end-stage renal failure (19). To an uncertain extent, this difference may be due to competing mortality.

One important new finding is the consistent difference in fat distribution between those with and without microalbuminuria or nephropathy; a phenomenon previously shown only in Indians (20). Central fat patterning occurs more commonly in those with a low weight at birth and is associated with a risk of ischemic heart disease, hypertension, and NIDDM (i.e., syndrome X) (21). This putative relationship between intrauterine starvation and the development of diabetes and heart disease (22) could, therefore, also include abnormal fetal renal development. Naturally, a genetic predisposition to nephropathy has not been excluded. Unfortunately, this cross-sectional study is not able to unravel these associations further. Similarly, it is not possible to explain the drop in prevalence of proteinuria after 20 years of diagnosed diabetes, although this is most likely to be a survivor effect.

The reasons for the ethnic differences described here are unknown. The independent entry of Maori ethnicity or Pacific Islander ethnicity, after entry of all other variables into a multivariate regres-

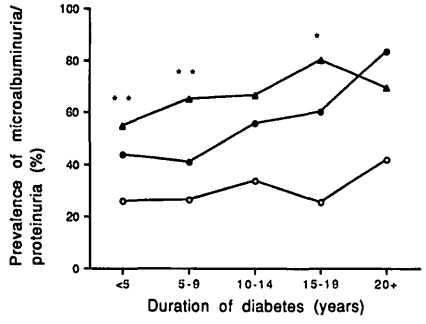

Figure 1—*Proportion with either microalbuminuria or proteinuria by duration of NIDDM and ethnic group. ○, Europeans; ▲, Maori; ●, Pacific Islanders. **P < 0.01; *P < 0.05 across the three ethnic groups.*

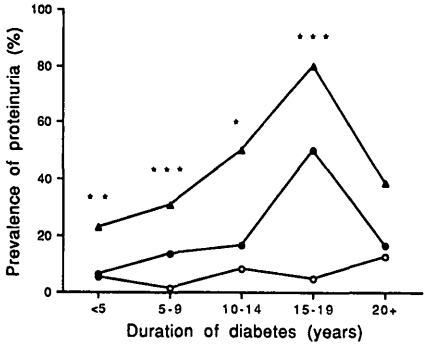

Figure 2—*Proportion with proteinuria by duration of NIDDM and ethnic group. ○, Europeans; ▲, Maori; ●, Pacific Islanders. ***P < 0.001; **P < 0.01; *P < 0.05 across the three ethnic groups.*

Table 4—*Comparison between NIDDM patients with and without microalbuminuria or nephropathy*

	European		Maori		Pacific Islander		Overall *P* of normal vs. the rest of MANOVA after ethnic group
	Normal	Nephropathy or microalbuminuria	Normal	Nephropathy or microalbuminuria	Normal	Nephropathy or microalbuminuria	
n	216	83	30	54	69	54	
Age (years)	61 ± 10	62 ± 10	53 ± 10	52 ± 9	55 ± 7	57 ± 9	NS
Age at diagnosis (years)	55 ± 12	53 ± 11	46 ± 10	41 ± 10*	51 ± 9	47 ± 11†	<0.001
BMI (kg/m^2)	28.9 ± 5.3	31.3 ± 6.1‡	32.2 ± 6.9	34.9 ± 6.4	33.0 ± 5.5	34.3 ± 6.2	<0.001
WHR	0.90 ± 0.10	0.94 ± 0.09‡	0.88 ± 0.07	0.91 ± 0.09	0.95 ± 0.08	1.00 ± 0.09†	<0.001
sBP (mmHg)	140 ± 25	147 ± 25*	138 ± 26	149 ± 32	136 ± 21	137 ± 28	<0.005
Ever smoked (%)	57.1	72.3	82.5	75.5	38.1	54.2	=0.05

Data are means ± SD. All continuous data are adjusted for age, sex, and referral center. No MANOVA had a statistically significant interactive term between ethnic group and albuminuria/nephropathy group. Post hoc analyses: * $P < 0.05$; † $P < 0.01$; ‡ $P < 0.001$.

sion, indicates that other factors are involved. The duration of time with diabetes before diagnosis is also unlikely to explain the ethnic differences found here. First, even after 20 years of diagnosed diabetes, the prevalence of microalbuminuria and nephropathy in Europeans does not exceed that of Maori and Pacific Islanders with diabetes known for up to 5 years duration. Second, significant microalbuminuria is found in Maori and Pacific Islanders without diabetes (23). There is no evidence that Europeans are diagnosed at an earlier stage in their diabetes than other ethnic groups, although this is probable.

It is possible that past blood pressure and glycemia were important in the development of nephropathy. In IDDM, high blood pressure is a major risk factor for diabetic nephropathy (24). In the NIDDM patients in the current study, the higher the current sBP, the higher the prevalence of nephropathy. However, differences in blood pressure did not explain the ethnic group differences. Furthermore, although sBP was highest in Maori, it was lowest in Pacific Islanders both overall and with nephropathy. This cross-sectional study is unable to determine whether high blood pressure was untreated for a longer time in non-Europeans, although this may have been the case.

Poor glucose control is another risk factor for diabetic nephropathy (25). The Maori and Pacific Islanders had higher measures of glycemia than Europeans. No significant differences in current glycemia were present between those with and without microalbuminuria and nephropathy. The kinetics of the glycated proteins contributing to the measured fructosamine could be altered with abnormal renal function and, hence, HbA_{1c} could have been a better discriminator. Alternatively, hyperglycemia could be a permissive factor for the development of nephropathy in those so predisposed. Although a cross-sectional study cannot predict past glycemic control, the higher casual glucose in those with nephropathy suggests that poor glycemic control could have contributed to the development of nephropathy.

In Europeans, those with nephropathy and microalbuminuria were more obese than those without. The occurrence of nephrotic syndrome has been reported in massive obesity (26,27). The relationship between microalbuminuria and BMI in Europeans, Maori, and Pacific Islanders without diabetes has also previously been shown (23). The importance of massive obesity in the initiation of microalbuminuria and proteinuria cannot be assessed here, because many of those with diabetes reported that they had lost weight since diagnosis. Smoking has previously been reported to be associated with progression of diabetic nephropathy in IDDM (28). The only other possible risk factor for nephropathy, protein intake (29), was not described, and Polynesians are recognized as having a high protein intake (P. Metcalf, unpublished data).

Proteinuria could indicate coexisting renal disease (30). In the current study, while only two patients had a significant urinary tract infection and the proportion reporting a past history of other diagnosed renal diseases was similar for the three ethnic groups, other coexisting renal disease had not been excluded. However, past renal biopsies in Maori and Pacific Islanders, where the diagnosis of diabetic nephropathy was in doubt, have consistently shown no other diagnosis (31). The greater urate concentration in Maori and Pacific Islanders is well known (32), but again, there was no evidence to suggest that this had contributed to their predisposition to renal disease via gouty nephropathy (a diagnosis open to question in any case [33]).

There are a number of caveats to the data presented here. Overestimation of the prevalence of nephropathy may have occurred by reviewing only those patients involved with either the general practitioner or the diabetes services. In-

deed, if the less symptomatic Polynesians attended for their diabetes care less frequently than less symptomatic Europeans, then an overestimation of the prevalence of nephropathy would be seen. We are undertaking a survey to assess this issue, and early results from a house-to-house survey suggest similar proportions (~10%) of those with no ongoing care in all three ethnic groups (unpublished data). This is unlikely to significantly impact upon the mean measures of nephropathy. Similarly, the greater number of Polynesians who had moved or were untraceable could have biased the results if those contacted were those with more tissue damage. The Polynesian population is very mobile and it is unlikely that a bias has occurred in this way. The use of one urine sample and an estimate of daily UAE are less likely to result in imprecision. Our findings were almost identical using the albumin:creatinine ratio. Furthermore, this method for calculating daily UAE has been thoroughly validated against 24-h urine collection and provides similar results and variance (9).

In conclusion, the excess of end-stage renal failure in Maori and Pacific Islanders remains unexplained by traditional risk factors. When compared with Europeans, Maori and Pacific Islanders with NIDDM have more microalbuminuria and nephropathy with impaired renal function at an early stage of their diabetes.

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THE AMERICAN DIABETES ASSOCIATION
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CONCURRENT WORKSHOPS

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