Continuing Disparities in Cardiovascular Risk Factors and Complications Between Aboriginal and Anglo-Celt Australians With Type 2 Diabetes

The Fremantle Diabetes Study

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OBJECTIVE—To determine whether disparities in the nature and management of type 2 diabetes persist between Aboriginal and the majority Anglo-Celt patients in an urban Australian community.

RESEARCH DESIGN AND METHODS—Baseline data from the observational Fremantle Diabetes Study collected from 1993 to 1996 (phase I) and from 2008 to 2011 (phase II) were analyzed. Patients characterized as Aboriginal or Anglo-Celt by self-report and supporting data underwent comprehensive assessment, including questionnaires, examination, and biochemical testing in a single laboratory. Generalized linear modeling with age/sex adjustment was used to examine differences in changes in variables in the two groups between phases I and II.

RESULTS—The indigenous participants were younger at entry and at diabetes diagnosis than the Anglo-Celt participants in both phases. They were also less likely to be educated beyond primary level and were more likely to be smokers. HbA_{1c} decreased in both groups over time (Aboriginal median 9.6% [interquartile range 7.8–10.7%] to 8.4% [6.6–10.6%] vs. Anglo-Celt median 7.1% [6.2–8.4%] to 6.7% [6.2–7.5%]), but the gap persisted ($P = 0.65$ for difference between phases I and II by ethnic group). Aboriginal patients were more likely to have microvascular disease in both phases. The prevalence of peripheral arterial disease (ankle-brachial index ≤0.90 or lower-extremity amputation) increased in Aboriginal but decreased in Anglo-Celt participants (15.8–29.7 vs. 30.7–21.5%; $P = 0.055$).

CONCLUSIONS—Diabetes management has improved for Aboriginal and Anglo-Celt Australian patients, but disparities in cardiovascular risk factors and complications persist.

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Diabetes is more common and diagnosed at a younger age in indigenous Australian Aboriginal or Torres Strait Islander people relative to other ethnic and racial groups in Australia (1–3). In addition, metabolic control is comparatively poor (4,5), and complications, especially nephropathy (6), are more frequent (7–9). Although most of the data characterizing diabetes in indigenous Australians have been collected from remote and rural settings, phase I of the Fremantle Diabetes Study (FDS), which was conducted between 1993 and 2001, confirmed that diabetes is common among Aboriginal people in an urban Australian community and that it presents at a relatively young age (10). Aboriginal patients with diabetes in FDS phase I had worse glycemic control, a higher prevalence of smoking, and a higher urinary albumin-creatinine ratio (ACR) than the majority Anglo-Celt group. The Aboriginal patients died an average of 18 years younger than their Anglo-Celt counterparts (10).

There is evidence that diabetes care, control, and complications have changed over the last 10–20 years in developed countries such as the U.S. More intensive blood glucose–lowering therapies are being used (11), and there are increasing efforts to control nonglycemic vascular risk factors (12,13), which consequently improves prognosis (14,15). However, disparities in diabetes management and outcome between racial and ethnic groups persist (12,16). In Australia, recent health initiatives such as government-subsidized diabetes-specific care plans and improved delivery of primary care services to diabetic patients have been designed to improve outcomes and reduce racial/ethnic inequalities (17–20). The aim of the current study was, therefore, to use baseline data from FDS phase I and the more recent phase II, which recruited patients between 2008 and 2011, to determine whether gaps in the nature and management of type 2 diabetes remain between Aboriginal and Anglo-Celt Australians.

RESEARCH DESIGN AND METHODS

Patients

Both FDS phases are longitudinal observational studies carried out in the same zip code–defined geographical area surrounding the port city of Fremantle in the state of Western Australia. Details of FDS phase I recruitment procedures and sample characteristics, including classification of diabetes type and nonrecruited

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patients, have been published elsewhere (21). In brief, any patient resident in the study catchment area with a clinician-verified diagnosis of diabetes was eligible for recruitment. Sources of identification and/or diagnostic data included public hospital inpatient/outpatient clinic lists and laboratory databases and notifications by local primary care/specialist physicians and allied health services, including diabetes education, dietetics and podiatry, advertisements in pharmacies and local media, and word of mouth. The protocol was approved by the Human Rights Committee of Fremantle Hospital, and all subjects gave informed consent.

We identified 2,258 eligible phase I subjects between 1993 and 1996 in the local population of 120,000 (crude diabetes prevalence, 1.9%) and recruited 1,426 (63%). Of 1,444 self-identified Aboriginal people living in the study area (22), 57 had diabetes (crude prevalence, 4.0%), of whom 19 (33%) were recruited to the FDS (10). Eighteen (95%) had clinically defined type 2 diabetes compared with 1,294 (91%) of the total cohort. The 819 type 2 diabetic subjects of Anglo-Celt ethnicity, based on self-identification as well as country of birth, country of father's/mother's birth, and language spoken at home, represented the largest FDS ethnic group (63% of the phase I type 2 diabetic patients) and were selected as the comparator (10).

FDS phase II was approved by the Human Research Ethics Committee of the Southern Metropolitan Area Health Service and used the same recruitment procedures as phase I. However, because of the relatively low numbers in phase I, an Aboriginal health worker (K.H.) found and recruited indigenous patients in the local community. Of 4,793 diabetic patients identified between 2008 and 2011 in the local population of 153,000 (crude diabetes prevalence, 3.1%), 1,668 (35%) were recruited. Of 2,048 self-identified Aboriginal people living in the study area (23), 237 had diabetes (crude prevalence, 11.6%), of whom 112 (47.3%) were recruited. One hundred and six (94.6%) had clinically defined type 2 diabetes compared with 1,509 (90.5%) of the total cohort. There were 796 type 2 diabetic Anglo-Celt subjects recruited from the local population (52.6% of the phase II type 2 diabetic patients). Five of the 106(4.7%) Aboriginal and 149 of the 796 (18.7%) Anglo-Celt phase II type 2 diabetic patients had also participated in phase I.

Methods

Each FDS phase I participant was assessed at baseline and invited to attend annual reviews for ≥5 years. For phase II, face-to-face assessments are biennial with questionnaire follow-up in alternate years. Because longitudinal data collection in phase II is at an early stage, we included only baseline data from both phases in the present analyses. All FDS face-to-face assessments comprise a comprehensive questionnaire, physical examination, and standard fasting biochemical tests (21). For both phases, diabetes type was assessed from diabetes treatment history, BMI, age at diagnosis, nature of first presentation, and/or self-identification. Noninsulin-treated patients and those ≥60 years of age at diagnosis were usually considered to have type 2 diabetes, as were patients <60 years of age at diagnosis and taking insulin at the time of study entry but whose first treatment was not insulin. In these and other cases, case records were consulted for evidence of ketonemia, as well as islet cell antibody, GAD antibodies, serum insulin, and C-peptide levels, if available.

Ethnic background in the FDS was based on self-selection, country/countries of birth and parents' birth, language(s) spoken at home, and, for phase II, country of grandparents' birth. In line with Australian legal rulings and a range of other studies of indigenous Australians with diabetes (1,2,4,7), we used self-identification and acceptance by the local community as the primary criteria for Aboriginality. There were no participants who identified themselves as from a Torres Strait Islander ethnic background in either phase of the FDS.

Complications were ascertained using standard criteria (24). Micro- and macroalbuminuria were defined as a urinary ACR ≥3.0 and ≥30.0 mg/mmol, respectively, on an early morning urine sample, neuropathy as a score of >2/8 on the Michigan Neuropathy Screening Instrument clinical portion, and retinopathy as any grade in either/both eyes on direct and/or indirect ophthalmoscopy and/or detailed specialist assessment (phase I) or retinal photography using a nonmydriatic camera (phase II). The estimated glomerular filtration rate (eGFR) was calculated from the serum creatinine (25). Peripheral vascular disease (PAD) was defined as an ankle-brachial index ≤0.90 or a diabetes-related lower-extremity amputation. In phase I, prior hospitalizations accessed through the Western Australian Data Linkage System (26) provided important supplementary data for ascertainment of coronary heart disease and cerebrovascular disease, but this source is not yet available for phase II subjects and so these complications have not been included in the current study.

Biochemical testing in both phases of the FDS was carried out in the same nationally accredited diagnostic biochemistry laboratory. The analytical systems used are detailed in the Supplementary Data. Between-run imprecision for all methods was <3.5%, except for urine albumin and serum HDL-cholesterol in phase II, for which it was <5.0%. Serum LDL-cholesterol was estimated using the Friedewald equation. For assays that had changed between 1993 and the present, calibration equations were applied to standardize all concentrations to current assays used for phase II (Supplementary Data).

Statistical analysis

The computer package IBM SPSS Statistics 19 (IBM Corporation, Armonk, NY) was used for statistical analysis. Data are presented as proportions, mean ± SD, geometric mean (SD range), or, in the case of variables that did not conform to a normal or log-normal distribution, median and interquartile range (IQR). For independent samples, two-way comparisons for proportions were performed by Fisher exact test, normally distributed variables by Student *t* test, and nonnormally distributed variables by Mann-Whitney *U* test. Since age and sex distributions were different between the Aboriginal and Anglo-Celt groups, generalized linear modeling with adjustment for age and sex was used to determine whether differences in prevalent diabetes complications and their risk factors had changed between phases I and II. A two-tailed significance level of $P < 0.05$ was used throughout.

RESULTS—Demographic and anthropometric details of the Aboriginal and Anglo-Celt patients recruited to phases I and II are summarized in Table 1. For phase I, these data have been published previously (10). As a result of the incorporation of the country/countries of grandparents' birth in the categorization of non-Aboriginal ethnicity and the availability of an Aboriginal health worker to assist with detailed data collection from Aboriginal participants in phase II, the ethnicity of one of the original 18 self-identified

Aboriginal type 2 diabetic patients in phase I changed when she was recruited to phase II, and two others who had not been identified as Aboriginal changed to Aboriginal in phase II. There were, therefore, 19 type 2 diabetic Aboriginal patients in phase I. In the case of the Anglo-Celt group, the 819 in the original report became 796 with additional data collected as part of phase II. The significant differences in phase I baseline characteristics reported originally (10) did not change as a result of this reclassification (data not shown).

As in phase I, Aboriginal participants in phase II were significantly younger than the Anglo-Celt participants, both at diagnosis and recruitment. They were also less likely to be educated beyond primary level and were much more likely to be current smokers. The proportion of Aboriginal males was lower than in the Anglo-Celt group in both phases, but this difference was significant with the larger sample size in phase II. Phase II Aboriginal subjects were less likely to be married or in a de facto relationship than the Anglo-Celt subjects. There was a substantial (almost 30%) increase in the proportion of Aboriginal subjects being educated beyond primary level between phases I and II, but this measure of educational attainment also increased in the Anglo-Celt group.

There was an improvement in HbA_{1c} in both groups over time (Table 2). However, consistent with similar blood glucose–lowering treatment intensification between phases I and II in the two ethnic groups, the gap persisted, and 70.6% of the Aboriginal subjects in phase II had an HbA_{1c} >7.0% at baseline compared with only 37.7% of the Anglo-Celt subjects. These findings applied to males and females considered separately (data not shown). A greater proportion of Anglo-Celt subjects in phase II were receiving antihypertensive treatment compared with phase I, but there was no change in the Aboriginal group. Despite a significantly lower percentage of Aboriginal patients being treated with antihypertensive therapy in phase II, there was no difference in blood pressure compared with the Anglo-Celt group. There was a dramatic increase in the percentage of patients taking lipid-modifying treatment (largely statins) between phases I and II in both ethnic groups, but fewer Aboriginal patients were receiving this form of treatment in both phases despite adverse lipid profiles, especially relatively high serum triglycerides and low serum HDL-cholesterol in phase II.

Table 1—*Demographic and anthropometric variables in Aboriginal and Anglo-Celt participants of phases I and II*

	Phase I			Phase II			Difference (95% CI)		
	Aboriginal	Anglo-Celt	*P* value	Aboriginal	Anglo-Celt	*P* value*	Aboriginal	Anglo-Celt	*P* value*
n	19	796		106	796				
Age (years)	51.2 ± 11.4	64.9 ± 11.4	<0.001	54.3 ± 11.9	67.2 ± 10.6	<0.001	3.0 (−2.8 to 8.9)	2.3 (1.2–3.4)	0.80
Sex (% male)	36.8	48.9	0.36	34.9	50.9	0.003	−1.9 (−25.7 to 21.8)	2.0 (−2.9 to 6.9)	0.76
Age at diabetes diagnosis (years)	45.6 ± 13.1	59.2 ± 11.6	<0.001	43.5 ± 14.4	57.7 ± 11.2	<0.001	−2.1 (−9.1 to 4.9)	−1.5 (−2.6 to −0.4)	0.50
Duration of diabetes (years)	4.7 [2.0–10.0]	4.0 [1.0–8.0]	0.44	8.0 [3.0–16.9]	8.0 [2.3–15.0]	0.17	5.1 (2.4–7.8)	3.8 (3.0–4.5)	0.50
Education beyond									
primary level (%)	55.6	85.5	0.003	84.7	93.6	0.007	29.2 (2.8–55.5)	8.1 (5.1–11.1)	0.19
Paid employment (%)	5.3	17.5	0.23	22.2	28.9	0.22	17.0 (3.2–30.8)	11.4 (7.3–15.5)	0.30
Married/de facto relationship (%)	52.6	61.8	0.48	39.6	61.6	<0.001	−13.0 (−37.4 to 11.4)	−0.3 (−5.1 to 4.5)	0.34
Alcohol use (standard drinks/day)	0 [0–1.2]	0 [0–0.3]	0.49	0.1 [0–1.4]	0.1 [0–1.2]	0.60	−0.5 (−2.2 to 1.1)	0.2 (0.1–0.4)	0.13
Smoking status									
Never smoked (%)	36.8	41.3		24.0	45.5		−13.3 (−35.0 to 8.3)	4.2 (−0.6 to 9.1)	0.10
Ex-smoker (%)	21.1	43.3	0.009	32.0	46.3	<0.001	10.9 (−11.0 to 32.9)	3.0 (−1.9 to 7.9)	0.42
Current smoker (%)	42.1	15.4		44.0	8.2		1.9 (−22.9 to 26.7)	−7.2 (−10.4 to −4.0)	0.13
BMI (kg/m^2)	30.5 ± 5.7	29.6 ± 5.6	0.49	32.0 ± 7.3	31.3 ± 6.2	0.32	1.6 (−1.9 to 5.1)	1.7 (1.1–2.3)	0.93
Obese by waist (%)†	68.4	64.4	0.81	77.6	72.1	0.28	9.1 (−12.2 to 30.4)	7.7 (3.1–12.3)	0.87
Overweight/obese by									
waist-to-hip ratio (%)‡	84.2	71.8	0.31	90.7	83.6	0.08	6.5 (−8.7 to 21.7)	11.8 (7.8–15.9)	0.81

Data are proportions, mean ± SD, median [IQR], or mean difference (95% CI). *Age and sex adjusted for the interaction between phase and ethnic group. †≥102.0 cm for males and ≥88.0 cm for females. ‡≥0.95 for males and ≥0.80 for females.

Table 2—*Baseline complications and their risk factors and management in Aboriginal and Anglo-Celt participants of phases I and II*

	Phase I			Phase II			Difference (95% CI)		
	Aboriginal	Anglo-Celt	*P* value	Aboriginal	Anglo-Celt	*P* value	Aboriginal	Anglo-Celt	*P* value*
Fasting serum glucose (mmol/L)	11.1 [7.2–14.6]	7.9 [6.3–10.1]	0.013	8.4 [6.4–12.1]	7.0 [6.2–8.5]	<0.001	−1.1 (−3.4 to 1.1)	−1.0 (−1.3 to −0.7)	0.90
HbA_{1c} (%)	9.6 [7.8–10.7]	7.1 [6.2–8.4]	<0.001	8.4 [6.6–10.6]	6.7 [6.2–7.5]	<0.001	−0.3 (−1.5 to 0.9)	−0.5 (−0.6 to −0.3)	0.65
HbA_{1c} >7.0% (%)	84.2	50.5	0.004	70.6	37.7	<0.001	−13.6 (−33.5 to 6.3)	−12.8 (−17.6 to −7.9)	0.70
Diabetes treatment									
Diet (%)	26.3	34.4		16.2	28.3		−10.1 (−29.0 to 8.8)	−6.2 (−10.7 to −1.6)	0.57
Oral agents (%)	57.9	55.2	0.55	61.0	51.8	0.025	3.1 (−21.3 to 27.4)	−3.7 (−8.6 to 1.2)	0.58
Insulin ± oral agents (%)	15.8	10.3		22.9	20.0		7.1 (−13.4 to 27.6)	9.9 (6.4–13.4)	0.62
Systolic blood pressure (mmHg)	138 ± 24	152 ± 23	0.012	141 ± 28	146 ± 22	0.11	3 (−11 to 17)	−6 (−8 to −4)	0.11
Diastolic blood pressure (mmHg)	79 ± 11	81 ± 11	0.57	81 ± 15	79 ± 12	0.25	2 (−5 to 9)	−1 (−3 to 0)	0.24
Systolic >130 mmHg or diastolic >80 mmHg (%)	63.2	80.2	0.08	71.2	79.8	0.06	8.0 (−14.8 to 30.8)	−0.4 (−4.4 to 3.5)	0.45
On antihypertensive therapy (%)	57.9	53.4	0.82	52.8	75.9	<0.001	−5.1 (−29.8 to 19.7)	22.5 (17.9–27.1)	0.015
On renin-angiotensin blockers (%)	26.3	22.2	0.59	47.2	66.0	<0.001	20.9 (−2.7 to 44.4)	43.7 (39.3–48.1)	0.08
Total serum cholesterol (mmol/L)	5.9 ± 0.8	5.5 ± 1.2	0.19	4.6 ± 1.4	4.3 ± 1.1	0.06	−1.3 (−1.7 to −0.8)	−1.2 (−1.3 to −1.1)	0.75
Serum HDL-cholesterol (mmol/L)	1.03 ± 0.25	1.06 ± 0.34	0.67	1.12 ± 0.29	1.27 ± 0.35	<0.001	0.09 (−0.05 to 0.23)	0.21 (0.17–0.24)	0.12
Serum LDL-cholesterol (mmol/L)	3.6 ± 1.0	3.3 ± 0.9	0.29	2.5 ± 1.1	2.3 ± 0.9	0.12	−1.1 (−1.7 to −0.5)	−1.0 (−1.1 to −1.0)	0.74
Serum LDL >2.5 mmol/L (%)	93.8	83.0	0.49	41.5	34.2	0.17	−52.3 (−68.6 to −35.9)	−48.8 (−53.1 to −44.4)	0.42
Serum triglycerides (mmol/L)	2.8 (1.8–4.4)	2.2 (1.2–4.1)	0.09	1.9 (1.1–3.3)	1.5 (0.9–2.4)	<0.001	−0.9 (−1.5 to −0.2)	−1.1 (−1.3 to −0.8)	0.70
On lipid-modifying treatment (%)	5.3	9.7	1.00	51.9	70.1	<0.001	46.6 (32.2–61.0)	60.5 (56.7–64.3)	0.93
Taking aspirin (%)	21.1	22.5	1.00	27.4	39.9	0.014	6.3 (−15.6 to 28.2)	17.5 (13.0–21.9)	0.43
Urine ACR (mg/mmol)	12.7 (1.7–95)	5.0 (1.5–17)	0.07	4.0 (0.1–289)	2.0 (0.1–34)	0.12	287 (−1,070 to 1,645)	−2.7 (−10.6 to 5.1)	0.11
Microalbuminuria (%)	61.1	57.1	0.81	61.7	38.0	<0.001	0.6 (−24.4 to 25.6)	−19.0 (−23.9 to −14.2)	0.12
Serum creatinine (μmol/L)	85 (45–162)	81 (60–110)	0.76	83 (47–146)	78 (57–106)	0.21	−14 (−80 to 53)	−4 (−8 to 0)	0.49
eGFR <60 mL/min/1.73 m^2 (%)	10.5	22.7	0.27	23.8	18.8	0.24	13.3 (−3.7 to 30.3)	−3.9 (−7.9 to 0.1)	0.13
Any retinopathy (%)	27.8	13.5	0.09	33.0	19.2	0.004	5.2 (−18.9 to 29.3)	5.7 (1.9–9.4)	0.77
Peripheral sensory neuropathy (%)	38.9	33.6	0.62	48.5	63.3	0.005	9.6 (−16.9 to 36.1)	29.6 (24.9–34.4)	0.09
Peripheral arterial disease (%)	15.8	29.7	0.31	30.7	21.5	0.043	14.9 (−5.0 to 34.8)	−8.3 (−12.6 to −4.0)	0.055

Data are proportions, mean ± SD, geometric mean (SD range), median [IQR], or mean difference (95% CI). *Age and sex adjusted for the interaction between phase and ethnic group.

In relation to complications, Aboriginal patients were more likely to have retinopathy than Anglo-Celt patients in both phases (Table 2). The improvement in HbA_{1c} with time was not associated with a reduction in the prevalence of retinopathy in either group, and the same was true of peripheral sensory neuropathy, which increased in both groups, especially the Anglo-Celt patients. Microalbuminuria was more prevalent in the Aboriginal patients in phase II. The prevalence of PAD increased in Aboriginal but decreased in Anglo-Celt participants.

CONCLUSIONS—In both phases I and II of the FDS, the Aboriginal participants were younger at recruitment and diagnosed with type 2 diabetes at a younger age than those of Anglo-Celt ethnicity. They had worse glycemic control and were more likely to have microvascular complications, especially in the larger sample that participated in phase II. The Aboriginal subjects were also more likely to smoke and, in phase II, to have diabetic dyslipidemia (high serum triglycerides and low HDL-cholesterol) and PAD. Thus, despite attempts to improve diabetes-related outcomes and reduce racial/ethnic inequalities in primary care over the last 15–20 years (17–20), Aboriginal Australians with type 2 diabetes remain substantially worse off than their Anglo-Celt counterparts.

There have been improvements in the 15 years between the phase I and II registration periods. Glycemic control improved in the two ethnic groups. However, although the majority of Anglo-Celt subjects had achieved the nationally recommended target HbA_{1c} of <7.0% (27) by phase II, over two-thirds of the Aboriginal patients had not. There was evidence that Aboriginal patients in phase II were more likely to be treated with oral blood glucose–lowering therapies or insulin than the Anglo-Celt patients. However, the HbA_{1c} data suggest that Aboriginal patients are being treated less intensively than they should, especially given their relatively young age and increased prevalence of established microvascular complications. This disparity could be explained by primary care and hospital-based, diabetes-specific facilities that do not attract and retain Aboriginal patients because of a failure to recognize important cultural sensitivities in the provision of care. Alternatively, adherence with treatment might be lower in Aboriginal patients, perhaps reflecting ethnicity-specific differences in disposable income, prioritization of health behaviors, and understanding of the long-term benefits of optimal treatment.

Smoking rates among the Anglo-Celt patients had halved over the 15 years to only 8.2%, but almost half of the Aboriginal patients were current smokers in both phases of the FDS. A similar high prevalence of smoking has been found in remote Aboriginal communities (28). Although the percentages of Aboriginal type 2 diabetic patients who were current smokers did not change over time, there was the suggestion, albeit not significant, that more Aboriginal type 2 diabetic patients had started smoking and more had quit in phase II compared with phase I. Although requiring confirmation, this suggests that contemporary antismoking programs are relatively ineffective in indigenous patients contemplating smoking for the first time.

The Aboriginal participants did not have higher blood pressures than the Anglo-Celt participants in either phase, as found in other studies (5). There was, however, significantly greater use of antihypertensive medications by the Anglo-Celt subjects in phase II, especially ACE inhibitors and angiotensin receptor blockers. This may help explain their lower prevalence of microalbuminuria, which is itself a strong cardiovascular risk factor in type 2 diabetes (29). Indeed, a lower percentage of Aboriginal patients were taking renin-angiotensin system blocking agents than had microalbuminuria in both phases, suggesting that these drugs were underutilized in this group. There was also a trend towards a slower uptake in use of renin-angiotensin system blockers by Aboriginal patients relative to the Anglo-Celt group between phases I and II.

The substantial increase in use of lipid-modifying therapy by both groups reflects changes in practice resulting from major trials such as the Heart Protection Study (30) and Collaborative Atorvastatin Diabetes Study (31) published between the two FDS phases. However, significantly fewer phase II Aboriginal patients were taking this form of treatment than Anglo-Celt patients despite >40% of them having a serum LDL-cholesterol above the Australian nationally recommended target range of <2.5 mmol/L (32).

The higher prevalence of microvascular disease in the Aboriginal group, especially in phase II, is likely to largely reflect their worse glycemic control. However, the fact that the prevalence of both retinopathy and neuropathy did not decrease between phases I and II despite an improved median HbA_{1c} in both groups may be, in part, a function of the longer diabetes duration and older age of phase II subjects (33,34). The more systematic collection of retinopathy data by retinal photography in phase II may also have contributed. Despite a higher prevalence of microalbuminuria in the Aboriginal group, especially in phase II, serum creatinine and eGFR did not differ by ethnicity in either phase. This finding reflects previous research suggesting that albuminuria is a better prognostic marker than eGFR for chronic kidney disease in indigenous patients with type 2 diabetes (35).

The prevalence of PAD increased in the Aboriginal group between phases I and II and decreased in the Anglo-Celt group. Since smoking is a strong risk factor for this macrovascular complication (24), it is likely that the continued high rates of current smoking among the Aboriginal patients and decreased rates in the Anglo-Celt group were primarily responsible. Smoking has also been shown to correlate significantly with carotid intima-media thickness (36) and measures of arterial stiffness (37) in Aboriginal patients. Diabetic dyslipidemia, as observed more frequently in phase II Aboriginal participants than the Anglo-Celt participants, may also be contributory, as has been found in other studies of arterial structure and function in Aboriginal Australians (38). PAD is independently associated with an increased risk of cardiac death (24), and the development of this complication might help explain the relatively high rate of premature deaths in Aboriginal patients with type 2 diabetes in phase I (10). Indeed, despite the limited phase II follow-up period, 7.1% of the Aboriginal phase II patients had died by the end of 2011 compared with only 3.9% of the remainder.

Our study had limitations. The number of Aboriginal participants in phase I was small and they may not have been representative of the local indigenous population at the time. Some variables in this group and the magnitude of their change between phases I and II had relatively wide confidence intervals, and there may have been additional significant differences both between groups and across time had there been more phase I indigenous participants. However, as noted in the original publication (10), the main findings in the Aboriginal group in phase I were consistent with those of a

range of other studies. This includes a greater representation of women than men (39), which was also evident in phase II and does not appear to be due to a survivor effect given the higher risk of coronary heart disease among Aboriginal women (8). We did not have sufficient longitudinal data from phase II to compare with those from phase I but, because of the procedural improvements in phase II, it is likely that there will be more complete follow-up especially for the Aboriginal participants. Additional data relating to social, economic, and psychological factors that could influence metabolic control and complications have been collected and are being validated for special groups such as Aboriginal patients, but their inclusion in the analyses was beyond the scope of the current study.

The present data parallel those from the National Health and Nutrition Examination Survey (NHANES) between 1999 and 2008, which show improvement in glycemic control and nonglycemic risk factors but continuing disparities between ethnic groups, with whites faring better than blacks and Hispanics (12,16). Smoking rates, which have remained stable in NHANES (12), have also proved difficult to change in Australian Aboriginal patients provided with intensified diabetes management in both urban (5) and remote (40) settings. However, the reduction in smoking observed in our Anglo-Celt patients gives some cause for hope. There is a continuing need for effective, culturally specific diabetes prevention and management programs for type 2 diabetes in Australian Aborigines that should target glycemic control, lipid management, and smoking in particular, perhaps supplemented by other initiatives, including free or heavily subsidized medications for such high-risk groups. The significant increase in educational attainment in Aboriginal patients between the two phases of the FDS could mean that new initiatives are adopted more readily in future.

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T.M.E.D. is principal investigator of the FDS and wrote the manuscript. K.H. coordinated data collection from the Aboriginal patients and reviewed and edited the manuscript. D.M. and D.G.B. assisted with study design and reviewed and edited the manuscript. S.A.P.C. coordinated laboratory analyses and reviewed and edited the manuscript. B.A.S. assisted with data collection and analysis and reviewed and edited the manuscript. W.A.D. researched data, performed all statistical analyses, and reviewed and edited the manuscript. T.M.E.D. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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References

1. Minges KE, Zimmet P, Magliano DJ, Dunstan DW, Brown A, Shaw JE. Diabetes prevalence and determinants in Indigenous Australian populations: a systematic review. Diabetes Res Clin Pract 2011;93:139–149
2. McDermott RA, Li M, Campbell SK. Incidence of type 2 diabetes in two indigenous Australian populations: a 6-year follow-up study. Med J Aust 2010;192:562–565
3. Maple-Brown LJ, Sinha AK, Davis EA. Type 2 diabetes in indigenous Australian children and adolescents. J Paediatr Child Health 2010;46:487–490
4. O'Neal DN, Piers LS, Iser DM, et al. Australian Aboriginal people and Torres Strait Islanders have an atherogenic lipid profile that is characterised by low HDL-cholesterol level and small LDL particles. Atherosclerosis 2008;201:368–377
5. Thomas M, Weekes AJ, Thomas MC. The management of diabetes in indigenous Australians from primary care. BMC Public Health 2007;7:303
6. Weil EJ, Nelson RG. Kidney disease among the indigenous peoples of Oceania. Ethn Dis 2006;16(Suppl. 2):24–30
7. Landers J, Henderson T, Craig J. Prevalence and associations of cataract in indigenous Australians within central Australia: the Central Australian Ocular Health Study. Clin Experiment Ophthalmol 2010;38:387–392
8. Wang Z, Hoy WE. Association between diabetes and coronary heart disease in Aboriginal people: are women disadvantaged? Med J Aust 2004;180:508–511
9. Ewald D, Patel M, Hall G. Hospital separations indicate increasing need for prevention of diabetic foot complications in central Australia. Aust J Rural Health 2001;9:275–279
10. Davis TM, McAullay D, Davis WA, Bruce DG. Characteristics and outcome of type 2 diabetes in urban Aboriginal people: the Fremantle Diabetes Study. Intern Med J 2007;37:59–63
11. Dodd AH, Colby MS, Boye KS, Fahlman C, Kim S, Briefel RR. Treatment approach and HbA1c control among US adults with type 2 diabetes: NHANES 1999-2004. Curr Med Res Opin 2009;25:1605–1613
12. Ford ES. Trends in the control of risk factors for cardiovascular disease among adults with diagnosed diabetes: findings from the National Health and Nutrition Examination Survey 1999-2008. J Diabetes 2011;3:337–347
13. Kuznik A, Mardekian J. Trends in utilization of lipid- and blood pressure-lowering agents and goal attainment among the U.S. diabetic population, 1999-2008. Cardiovasc Diabetol 2011;10:31
14. Ford ES. Trends in the risk for coronary heart disease among adults with diagnosed diabetes in the U.S.: findings from the National Health and Nutrition Examination Survey, 1999-2008. Diabetes Care 2011;34:1337–1343
15. Hoerger TJ, Zhang P, Segel JE, Gregg EW, Narayan KM, Hicks KA. Improvements in risk factor control among persons with diabetes in the United States: evidence and implications for remaining life expectancy. Diabetes Res Clin Pract 2009;86:225–232
16. Chatterji P, Joo H, Lahiri K. Racial/ethnic- and education-related disparities in the control of risk factors for cardiovascular disease among individuals with diabetes. Diabetes Care 2012;35:305–312
17. McDermott RA, McCulloch BG, Campbell SK, Young DM. Diabetes in the Torres Strait Islands of Australia: better clinical systems but significant increase in weight and other risk conditions among adults, 1999-2005. Med J Aust 2007;186:505–508
18. O'Dea K, Rowley KG, Brown A. Diabetes in indigenous Australians: possible ways forward. Med J Aust 2007;186:494–495
19. Longstreet DA, Griffiths MM, Heath D, et al. Improving diabetes care in an urban Aboriginal medical centre. Aust J Prim Health 2005;11:25–31
20. Shortus TD, McKenzie SH, Kemp LA, Proudfoot JG, Harris MF. Multidisciplinary care plans for diabetes: how are they used? Med J Aust 2007;187:78–81
21. Davis TM, Zimmet P, Davis WA, Bruce DG, Fida S, Mackay IR. Autoantibodies to glutamic acid decarboxylase in diabetic patients from a multi-ethnic Australian community: the Fremantle Diabetes Study. Diabet Med 2000;17:667–674
22. Australian Bureau of Statistics. *1991 Census of Population and Housing. Basic Community*

Profile. Report No. 2722.0. Canberra, Commonwealth Government of Australia, 1997
23. Australian Bureau of Statistics. Census [Internet], 2006. Canberra, Commonwealth Government of Australia. Available from http://www.abs.gov.au/websitedbs/censushome.nsf/home/Census. Accessed January 2012
24. Norman PE, Davis WA, Bruce DG, Davis TM. Peripheral arterial disease and risk of cardiac death in type 2 diabetes: the Fremantle Diabetes Study. Diabetes Care 2006;29:575–580
25. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D; Modification of Diet in Renal Disease Study Group. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Ann Intern Med 1999;130:461–470
26. Holman CD, Bass AJ, Rouse IL, Hobbs MST. Population-based linkage of health records in Western Australia: development of a health services research linked database. Aust NZ J Public Health 1999; 23:453–459
27. Colagiuri S, Dickinson S, Girgis S, Colagiuri R. *National Evidence Based Guideline for Blood Glucose Control in Type 2 Diabetes*. Canberra, Diabetes Australia and the National Health and Medical Rsearch Council, 2009
28. McCulloch B, McDermott R, Miller G, Leonard D, Elwell M, Muller R. Self-reported diabetes and health behaviors in remote indigenous communities in Northern Queensland, Australia. Diabetes Care 2003; 26:397–403
29. Heart Outcomes Prevention Evaluation Study Investigators. Effects of ramipril on cardiovascular and microvascular outcomes in people with diabetes mellitus: results of the HOPE study and MICRO-HOPE substudy. Lancet 2000;355:253–259
30. Collins R, Armitage J, Parish S, Sleigh P, Peto R; Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol-lowering with simvastatin in 5963 people with diabetes: a randomised placebo-controlled trial. Lancet 2003;361:2005–2016
31. Colhoun HM, Betteridge DJ, Durrington PN, et al.; CARDS investigators. Primary prevention of cardiovascular disease with atorvastatin in type 2 diabetes in the Collaborative Atorvastatin Diabetes Study (CARDS): multicentre randomised placebo-controlled trial. Lancet 2004;364:685–696
32. Australian Centre for Diabetes Strategies for the Diabetes Australia Guideline Development Consortium. *National Evidence Based Guidelines for the Management of Type 2 Diabetes Mellitus. Part 7. Lipid Control in Type 2 Diabetes*. Canberra, National Health and Medical Research Council, 2004
33. Klein R, Klein BE, Moss SE, Davis MD, DeMets DL. The Wisconsin epidemiologic study of diabetic retinopathy. III. Prevalence and risk of diabetic retinopathy when age at diagnosis is 30 or more years. Arch Ophthalmol 1984;102:527–532
34. Tesfaye S, Chaturvedi N, Eaton SE, et al.; EURODIAB Prospective Complications Study Group. Vascular risk factors and diabetic neuropathy. N Engl J Med 2005; 352:341–350
35. Maple-Brown LJ, Cunningham J, Hodge AM, et al. High rates of albuminuria but not of low eGFR in urban indigenous Australians: the DRUID study. BMC Public Health 2011;11:346
36. Haluska BA, Chan L, Jeffriess L, Shaw AA, Shaw J, Marwick TH. Correlates of preclinical cardiovascular disease in indigenous and non-indigenous Australians: a case control study. Cardiovasc Ultrasound 2008;6:36
37. Maple-Brown LJ, Piers LS, O'Rourke MF, Celermajer DS, O'Dea K. Increased arterial stiffness in remote indigenous Australians with high risk of cardiovascular disease. J Hypertens 2007;25:585–591
38. Maple-Brown LJ, Cunningham J, Barry RE, et al. Impact of dyslipidaemia on arterial structure and function in urban indigenous Australians. Atherosclerosis 2009;202:248–254
39. Wang Z, Hoy WE, Si D. Incidence of type 2 diabetes in Aboriginal Australians: an 11-year prospective cohort study. BMC Public Health 2010;10:487
40. Simmons D. Impact of an integrated approach to diabetes care at the Rumbalara Aboriginal Health Service. Intern Med J 2003;33:581–585