Hypertension, Dyslipidemia, Body Mass Index, Diabetes and Smoking Status in Aboriginal Australians in a Remote Community

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Objectives: Study objectives were: 1) to describe the differences in the prevalence of CHD risk factors between Aboriginal people in a remote community and the general Australian population; and 2) to compare the predicted risks of CHD events between Aboriginal and non-Aboriginal Australians.

Design: A cross-sectional study.

Participants: 681 Aboriginal adults aged 25 to 74 years.

Results: Aboriginal young adults had substantially higher prevalence of diabetes compared to non-Aboriginal Australians. The prevalence ratios for diabetes were 12.5, 5.6, 3.2, 1.3, and 0.73 for 25-, 35-, 45-, 55-, and 65- to 74-year-old females, respectively. The corresponding values for males were 12.1, 2.7, 2.9, 0.69, and 0.42. Young females had a higher prevalence of obesity, overweight, and abnormal waist circumference, while males and females 45 years and older tended to have a lower prevalence of overweight and abnormal waist circumference. Compared to the general population, Aboriginal adults had a lower prevalence of abnormal total cholesterol but a higher prevalence of abnormal HDL, triglycerides, hypertension, and smoking. The risk ratios of abnormal total cholesterol for females ages 25–34, 35–44, 45–54, 55–64, and 65–75 years were 0.38, 0.53, 0.48, 0.48, and 0.41, respectively.

Conclusions: Aboriginal people in the remote community experienced different levels of CHD risk predictors from the general Australian population. They had a lower prevalence of abnormal total cholesterol and a higher prevalence of abnormal HDL, smoking, diabetes, and hypertension. (*Ethn Dis*. 2003;13: 324–330)

Key Words: Aboriginal Australians, Hypertension, Dyslipidemia, Body Mass Index, Diabetes, Smoking

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Introduction

Cardiovascular disease death rates are 6 times higher for Aboriginal males and 9 times higher for Aboriginal females 25 to 64 years of age, compared with other Australians in the same age group.[1] A striking aspect of Aboriginal mortality from coronary heart disease (CHD) is the high death rates among young and middle-aged Aboriginal adults aged 25–44 years, with rates more than 10 times those of other Australians.[2] The higher levels of mortality from CHD experienced by Aboriginal people are also reflected in hospitalization rates.[3,4] Rates of hospitalization for conditions such as CHD and stroke are 2 and 4 times higher among Aboriginal people.[5] Why do Aboriginal people experience poor cardiovascular health? It is suggested that the risk profile of Aboriginal Australians is one reason for the considerably higher morbidity and mortality that this population experiences.[5] Several risk factors for cardiovascular disease, such as high blood pressure, smoking and diabetes, are more prevalent among Aboriginal Australians than non-Aboriginal Australians.[2,6] However, it is not clear whether the observed differences in CHD incidence between Aboriginal and non-Aboriginal populations can be explained by differences in prevalence of established risk factors. While reports have clearly documented an excess mortality, there are few studies that systematically describe detailed differences in risk factor profiles between this population and the general Australian population.

Assessment of absolute CHD risk is the first step of a cost-effective intervention. The Framingham functions were developed to assess the relative importance of CHD risk factors and to quantify the absolute level of risk for individuals.[7,8] Concern exists as to the generalizability of these functions to other populations.[9–11] For example, the Framingham functions systematically overestimated the risk of 5 year CHD events among Japanese-American and Hispanic men and Native American women.[10] The New Zealand cardiovascular disease risk charts[12] have been endorsed by the Australian Lipid Management Guidelines 2001.[13] It is not known if formulas from other populations are generalizable to Aboriginal people. The objectives of this study were: 1) to describe the differences in the prevalence of CHD risk factors between Aboriginal people in a remote community and the general Australian population; and 2) to compare the predicted risks of CHD events between Aboriginal and non-Aboriginal Australians.

Methods

In 1992, a community-wide screening program was started in a remote Northern Territory Aboriginal community with high rates of premature death. Between 1992 and 1998, participants were offered testing at baseline. A total of 848 adults (more than 80% in the community) ages 25 to 74 years were included in this study. The project was approved by the Joint Institutional Ethics Committee of the Menzies School of Health Research and Territory Health Services, Darwin, Australia. One hundred sixty-seven (167) participants were excluded due to missing values. The 10-year predicted CHD risk was calculated based on 681 participants with a complete set of baseline measurements. The cross-sectional baseline data were used for the present analysis. Prevalence rates

A striking aspect of Aboriginal mortality from coronary heart disease (CHD) is the high death rates among young and middle-aged Aboriginal adults aged 25–44 years, with rates more than 10 times those of other Australians.

of Aboriginal people were compared with those reported in the Australian Diabetes, Obesity, and Lifestyle Study (AusDiab),[14] in which a representative sample of 11,247 adults was examined across Australia. The AusDiab study is the first national survey in Australia to determine the prevalence of diabetes and other cardiovascular risk factors, including obesity, hypertension, and dyslipidemia.

Measurements and Analysis

We used the same definitions for CHD risk factors as described in the AusDiab report.[14] Dyslipidemia was defined as ≥5.5 mmol/L for abnormal total cholesterol and <1.0 mmol/L for abnormal HDL cholesterol. Hypertension was determined as systolic ≥140 mm Hg or diastolic ≥90 mm Hg or if on treatment for hypertension. Diabetes was defined if participants had an existing diagnosis by medical record review (performed for each person) or were on treatment for diabetes. In addition, 389 participants had a fasting glucose test and 306 participants had an oral glucose tolerance test (OGTT). The presence of diabetes among them was diagnosed according to fasting glucose and OGTT cut-off ranges outlined by WHO.[15] Overweight was defined as BMI 25+ and an abnormal waist circumference as 80+ cm for females and 94+ cm for males.

We calculated the age-sex specific prevalence of dyslipidemia (total cholesterol and HDL cholesterol), hypertension, diabetes, and cigarette smoking. These prevalence rates were compared with corresponding values from the general Australian population collected from the AusDiab study.[14] The prevalence ratios and their 95% confidence intervals were estimated with the prevalence in AusDiab data as the reference value.

The 10-year risk of CHD was predicted for each individual according to his age, sex, smoking status, total and HDL cholesterol, levels of systolic blood pressure, and the presence of diabetes using the Framingham formula.[7] Age-sex specific mean risks were compared with corresponding values in AusDiab data. As in the AusDiab study, information on left ventricular hypertrophy, a predictor in Framingham formula, was not available, and so it was assumed absent in all cases. All calculations were performed using the Stata 7 program.

Results

Table 1 shows the means and standard deviations of CHD risk factors. The average levels of risk factors, except HDL, changed significantly with age. Systolic blood pressure monotonically increased with age while other factors tended to have higher values in the middle-age groups.

Prevalence and Prevalence Ratios of CHD Risk Factors

Table 2 shows the prevalence of CHD risk factors. The prevalence ratios are presented in Table 3. Young Aboriginal adults ages 25 to 54 years had a significantly higher prevalence of diabetes than their non-Aboriginal counterparts, especially in the younger age groups. Such a difference was not found in those older than 55 years. A similar trend was found for the prevalence of hypertension.

The proportion of overweight was higher for females ages 25 to 44 years, but lower for females ages 45 years or older, and males of all ages. Females also had a higher prevalence of abnormal waist circumference values while males had a significantly lower prevalence than their non-Aboriginal counterparts.

The prevalence of elevated total cholesterol levels for Aboriginal participants was significantly lower than that found in other Australians. Although prevalence ratio and its 95% confidence intervals for males in the 65- to 74-year group could not be calculated due to a small sample size and zero positive in this stratum, the Fisher's exact test was performed. The *P* value was .01, indicating even though the sample size was small in this group the prevalence of abnormal total cholesterol was lower and statistically significant in Aboriginal men.

Aboriginal participants had a higher prevalence of elevated triglycerides in all age and gender groups. The prevalence of abnormal HDL cholesterol (<1.0 mmol/L) was substantially higher in Aboriginal females, but only slightly higher in Aboriginal males than that of their non-Aboriginal counterparts.

Predicted 10-Year CHD Risk

According to the values of total and HDL cholesterols, systolic blood pressure, smoking, and diabetes status, the 10-year risk of CHD was predicted using the Framingham function.[7,16] Age-sex specific 10-year mean risks are presented in Figure 1. The predicted 10-year risks for males were higher than for females in all age strata. The increased risk was associated with increasing age. Although both males and females had a higher predicted risk than their non-Aboriginal counterparts, the difference was more striking for females. The ratios of predicted risk between Aboriginal and non-Aboriginal people were 3+, 3.0,

Table 1. Mean and standard deviations of cardiovascular disease risk factors in an Aboriginal community

Variable	Age Groups, Years					*P* Value*
	25–	35–	45–	55–	65–74	
Female						
Number	135	100	66	26	12	
Age, years	29.4 (3.0)	39.6 (2.9)	48.6 (2.9)	59.3 (2.6)	69.4 (3.2)	
BMI, kg/m^2	24.9 (6.1)	26.5 (5.4)	25.5 (5.3)	22.1 (5.0)	20.6 (2.8)	0.0002
Waist circ., cm	90.5 (15.1)	95.0 (15.3)	95.9 (13.2)	91.2 (13.7)	87.8 (10.8)	0.0386
Cholesterol, mmol/l	4.4 (1.0)	4.7 (1.2)	5.0 (1.0)	5.2 (0.8)	4.8 (1.0)	0.0001
HDL, mmol/l	1.1 (0.3)	1.1 (0.3)	1.1 (0.2)	1.1 (0.2)	1.2 (0.3)	0.5822
Triglycerides, mmol/l	1.9 (1.2)	2.4 (1.7)	2.3 (1.2)	2.3 (1.7)	1.8 (0.6)	0.0185
Systolic BP, mm Hg	112.4 (14.5)	120.8 (18.3)	125.9 (22.8)	127.3 (22.7)	139.0 (27.1)	0.0000
Diastolic BP, mm Hg	70.6 (11.4)	75.2 (12.5)	77.6 (14.1)	68.8 (14.3)	78.9 (16.0)	0.0003
Male						
Number	180	94	47	16	5	
Age, years	28.9 (2.8)	38.8 (2.7)	49.0 (2.7)	57.8 (2.8)	70.2 (3.3)	
BMI, kg/m^2	22.9 (4.0)	24.4 (5.1)	25.0 (4.9)	24.7 (4.2)	21.0 (2.1)	0.0043
Waist circ., cm	85.0 (11.1)	91.2 (13.3)	94.6 (11.8)	95.1 (12.2)	91.0 (7.3)	0.0000
Cholesterol, mmol/l	4.9 (1.1)	5.2 (0.9)	5.1 (1.2)	4.6 (0.8)	4.5 (0.3)	0.0879
HDL, mmol/l	1.2 (0.3)	1.1 (0.2)	1.1 (0.3)	1.0 (0.2)	1.1 (0.2)	0.1478
Triglycerides, mmol/l	2.0 (1.4)	2.4 (1.7)	2.7 (1.9)	2.4 (0.7)	2.1 (0.3)	0.0092
Systolic BP, mm Hg	122.7 (14.4)	127.0 (18.5)	135.1 (20.0)	137.4 (16.5)	148.8 (14.0)	0.0000
Diastolic BP, mm Hg	76.3 (11.0)	82.4 (13.6)	85.1 (14.7)	77.5 (14.0)	88.0 (29.0)	0.0000

* Analysis of variance test with 4 *df*.

2.2, 1.7, 1.4, 1.28, and 1.19 for 25-, 35-, 45-, 55-, and 65- to 74-year-old females, respectively, and the corresponding ratios for males were 1.4, 1.3, 1.4, 1.1, and 1.2.

DISCUSSION

The prevalence values of CHD risk factors in Aboriginal people living in a remote community were compared with the general Australian population from the AusDiab study. Aboriginal people had different distributions of CHD risk factors from the general Australian population. This was characterized by a sig-

Table 2. Prevalence (95% CI) of cardiovascular disease risk factors in an Aboriginal community

	Age Groups, Years				
	25–	35–	45–	55–	65–74
Females					
Diabetes	4.7 (2.2, 8.8)	13.1 (8.1, 19.7)	17.8 (11.0, 26.3)	13.0 (5.4, 24.9)	11.8 (1.5, 36.4)
BMI 25+, kg/m^2	48.4 (41.1, 55.8)	56.3 (47.8, 64.6)	48.6 (38.7, 58.5)	32.1 (19.9, 46.3)	12.5 (1.6, 38.3)
BMI 30+, kg/m^2	24.2 (18.3, 30.9)	28.9 (21.6, 37.1)	19.0 (12.0, 27.9)	11.3 (4.3, 23.0)	0.0 (0.0, 20.6)
Waist 80+, cm	71.4 (64.3, 77.9)	85.8 (78.9, 91.1)	86.0 (77.6, 92.1)	88.2 (76.1, 95.6)	80.0 (51.9, 95.7)
Waist 88+, cm	54.9 (47.4, 62.3)	66.0 (57.5, 73.7)	76.0 (66.4, 84.0)	76.5 (62.5, 87.2)	73.3 (44.9, 92.2)
Cholesterol 5.5+, mmol/l	11.8 (7.0, 18.2)	20.6 (13.4, 29.5)	26.3 (16.9, 37.7)	34.4 (18.6, 53.2)	30.8 (9.1, 61.4)
HDL <1.0, mmol/l	38.2 (30.0, 47.0)	36.6 (27.3, 46.8)	30.9 (20.2, 43.3)	30.8 (14.3, 51.8)	16.7 (2.1, 48.4)
Triglycerides >2.0, mmol/l	38.2 (30.2, 46.7)	51.4 (41.5, 61.2)	56.6 (44.7, 67.9)	43.8 (26.4, 62.3)	38.5 (13.9, 68.4)
Hypertension	13.8 (9.2, 19.5)	16.7 (11.0, 23.8)	29.2 (20.8, 38.9)	35.2 (22.7, 49.4)	58.8 (32.9, 81.6)
Smoking	58.6 (51.3, 65.7)	63.4 (55.1, 71.3)	67.3 (57.5, 76.0)	68.5 (54.4, 80.5)	76.5 (50.1, 93.2)
Males					
Diabetes	2.1 (0.4, 4.8)	6.9 (3.4, 12.4)	20.0 (11.4, 31.3)	11.1 (3.1, 26.1)	9.1 (0.2, 41.3)
BMI 25+, kg/m^2	28.2 (22.6, 34.4)	42.7 (34.4, 51.2)	46.4 (34.3, 58.8)	38.9 (23.1, 56.5)	9.1 (0.2, 41.3)
BMI 30+, kg/m^2	7.5 (4.5, 11.5)	12.6 (7.6, 19.2)	17.4 (9.3, 28.4)	8.3 (1.8, 22.5)	9.1 (0.2, 41.3)
Waist 94+, cm	20.8 (15.8, 26.5)	40.0 (31.8, 48.6)	51.5 (39.0, 63.8)	50.0 (32.9, 67.1)	45.5 (16.7, 76.6)
Waist 102+, cm	10.2 (6.6, 14.8)	19.3 (13.1, 26.8)	30.9 (20.2, 43.3)	22.2 (10.1, 39.2)	9.1 (0.2, 41.3)
Cholesterol 5.5+, mmol/l	29.4 (23.2, 36.2)	40.7 (31.4, 50.6)	38.2 (25.4, 52.3)	25.0 (8.7, 49.1)	0.0 (0.0, 45.9)
HDL <1.0, mmol/l	20.6 (14.9, 27.2)	20.0 (12.5, 29.5)	33.3 (20.4, 48.4)	35.3 (14.2, 61.7)	20.0 (0.5, 71.6)
Triglycerides 2.0+, mmol/l	41.3 (34.4, 48.4)	57.4 (47.5, 66.9)	63.6 (49.6, 76.2)	80.0 (56.3, 94.3)	83.3 (35.9, 99.6)
Hypertension	23.4 (18.2, 29.3)	41.3 (33.1, 49.8)	60.9 (48.4, 72.4)	57.1 (39.4, 73.7)	81.8 (48.2, 97.7)
Smoking	78.8 (73.1, 83.8)	71.5 (63.4, 78.7)	70.0 (57.9, 80.4)	66.7 (49.0, 81.4)	72.7 (39.0, 94.0)

Table 3. Prevalence ratios (95% CI) of cardiovascular disease risk factors: Aboriginal vs general Australian population

	Age Groups, Years				
	25–	**35–**	**45–**	**55–**	**65–74**
Females					
Diabetes	12.5 (3.4, 45.9)	5.6 (3.3, 9.6)	3.2 (2.0, 5.1)	1.3 (0.64, 2.7)	0.73 (0.20, 2.7)
Hypertension	3.5 (2.2, 5.8)	2.2 (1.5, 3.3)	1.3 (0.93, 1.7)	0.82 (0.57, 1.2)	0.88 (0.59, 1.3)
BMI 25+, kg/m^2	1.4 (1.2, 1.6)	1.3 (1.1, 1.5)	0.84 (0.68, 1.0)	0.47 (0.32, 0.70)	0.18 (0.05, 0.66)
BMI 30+, kg/m^2	2.0 (1.5, 2.7)	1.5 (1.1, 2.0)	0.73 (0.49, 1.1)	0.35 (0.17, 0.76)	0*
Waist 80+, cm	2.0 (1.7, 2.2)	1.8 (1.7, 2.0)	1.5 (1.3, 1.6)	1.2 (1.1, 1.4)	1.0 (0.78, 1.3)
Waist 88+, cm	3.2 (2.6, 3.9)	2.6 (2.2, 3.0)	2.0 (1.8, 2.3)	1.6 (1.4, 1.9)	1.4 (1.0, 1.9)
Cholesterol 5.5+, mmol/l	0.38 (0.24, 0.60)	0.53 (0.36, 0.77)	0.48 (0.33, 0.70)	0.48 (0.30, 0.78)	0.41 (0.18, 0.93)
HDL <1.0, mmol/l	6.1 (4.3, 8.6)	8.9 (6.2, 12.8)	5.4 (3.6, 8.1)	5.2 (2.8, 9.6)	2.4 (0.66, 8.7)
Triglycerides >2.0, mmol/l	3.9 (2.9, 5.2)	5.5 (4.3, 7.0)	3.3 (2.7, 4.2)	1.7 (1.2, 2.6)	1.3 (0.64, 2.6)
Smoking	1.6 (1.3, 1.8)	1.5 (1.3, 1.7)	1.9 (1.6, 2.2)	2.3 (1.9, 2.8)	2.4 (1.8, 3.2)
Males					
Diabetes	12.1 (1.4, 103)	2.7 (1.3, 5.5)	2.9 (1.8, 4.9)	0.69 (0.27, 1.8)	0.42 (0.06, 2.7)
Hypertension	3.3 (2.3, 4.9)	2.4 (1.9, 3.1)	2.0 (1.6, 2.4)	1.2 (0.91, 1.6)	1.2 (0.91, 1.6)
BMI 25+, kg/m^2	0.47 (0.38, 0.58)	0.66 (0.55, 0.81)	0.64 (0.50, 0.83)	0.53 (0.35, 0.79)	0.12 (0.02, 0.81)
BMI 30+, kg/m^2	0.44 (0.27, 0.71)	0.72 (0.46, 1.1)	0.85 (0.50, 1.4)	0.33 (0.11, 0.97)	0.44 (0.07, 2.9)
Waist 94+, cm	0.52 (0.40, 0.68)	0.78 (0.63, 0.96)	0.88 (0.70, 1.1)	0.75 (0.54, 1.0)	0.64 (0.33, 1.2)
Waist 102+, cm	0.74 (0.48, 1.1)	0.79 (0.55, 1.1)	1.1 (0.78, 1.6)	0.62 (0.34, 1.2)	0.22 (0.03, 1.4)
Cholesterol 5.5+, mmol/l	0.92 (0.72, 1.2)	0.74 (0.58, 0.93)	0.63 (0.45, 0.88)	0.41 (0.19, 0.87)	0*
HDL <1.0, mmol/l	1.1 (0.80, 1.6)	1.1 (0.73, 1.7)	1.6 (1.1, 2.5)	1.8 (0.95, 3.6)	1.1 (0.19, 6.6)
Triglycerides 2.0+, mmol/l	2.8 (2.2, 3.7)	2.2 (1.8, 2.6)	2.0 (1.6, 2.5)	2.7 (2.1, 3.4)	3.4 (2.3, 4.9)
Smoking	2.1 (1.8, 2.4)	1.4 (1.3, 1.6)	1.4 (1.2, 1.7)	1.2 (0.92, 1.5)	1.2 (0.84, 1.8)

* 95% CI were not calculated, Fish exact test *P* value<.01.

nificantly low prevalence of abnormal total cholesterol, and high prevalence of abnormal HDL cholesterol, hypertension, diabetes, and smoking. The differences between Aboriginal and other Australians in abnormal HDL cholesterol, hypertension, diabetes, and smoking were more striking in females and in young adults of both sexes. Such differences in CHD risk factors between Aboriginal and other Australians should be considered in planning intervention strategies at the community level, as well as for individual risk assessment and management in clinical settings.

Our findings of higher prevalence of hypertension, diabetes and smoking in Aboriginal people are consistent with previous studies.[6,17] Guest et al reported that Aboriginal adults in 2 country towns of southeastern Australia had a higher rates of diastolic blood pressure than Europeans from the same area.[6] The recent surveys have also provided evidence of higher levels of some CHD risk factors, such as smoking, diabetes, and hypertension, among Aboriginal people, compared with other Australians.[2] The low prevalence of abnormal total cholesterol among Aboriginal people is similar to that reported for Torres Strait Islander people.[18] Leonard et al compared age-adjusted prevalence of 592 Torres Strait Islander people with those from AusDiab study. They reported a 0.8 (95% CI 0.7–0.9) standardized ratio for abnormal total cholesterol.[18] Guest et al found the cholesterol levels were significantly lower in Aboriginal people than their European counterparts.[6] The present study adds detailed age-sex specific differences between Aboriginal and non-Aboriginal Australians.

The high prevalence of risk factors in young Aboriginal adults is a major concern in this community. Young adults not only experienced a relatively higher prevalence of several risk factors than their non-Aboriginal counterparts, they also had a higher prevalence than older age groups in the community. For example, the participants of 45–54 year age group had a higher prevalence of diabetes than those 55 years or older in both sexes. One possible explanation for older Aboriginal people having relatively lower prevalence of risk factors is a "survivor effect," ie, those with lower prevalence of risk factors have survived longer. Another possible explanation is a "cohort effect,"[19] ie, the younger generation is currently exposed to higher CVD risk than was the older generation at the same age. If this is the case, we might expect a substantial increase in CVD events in the next few decades. The fact that young Aboriginal people have a higher prevalence of risk factors underscores the importance of early identification of high-risk individuals and early prevention in this population. While lipid control is important for preventing CHD, prevention and management of smoking, diabetes, and hypertension should be prioritized in Aboriginal populations.

Use of the prediction models would be most appropriate for individuals who resemble the study sample. Framingham functions have been found acceptable in

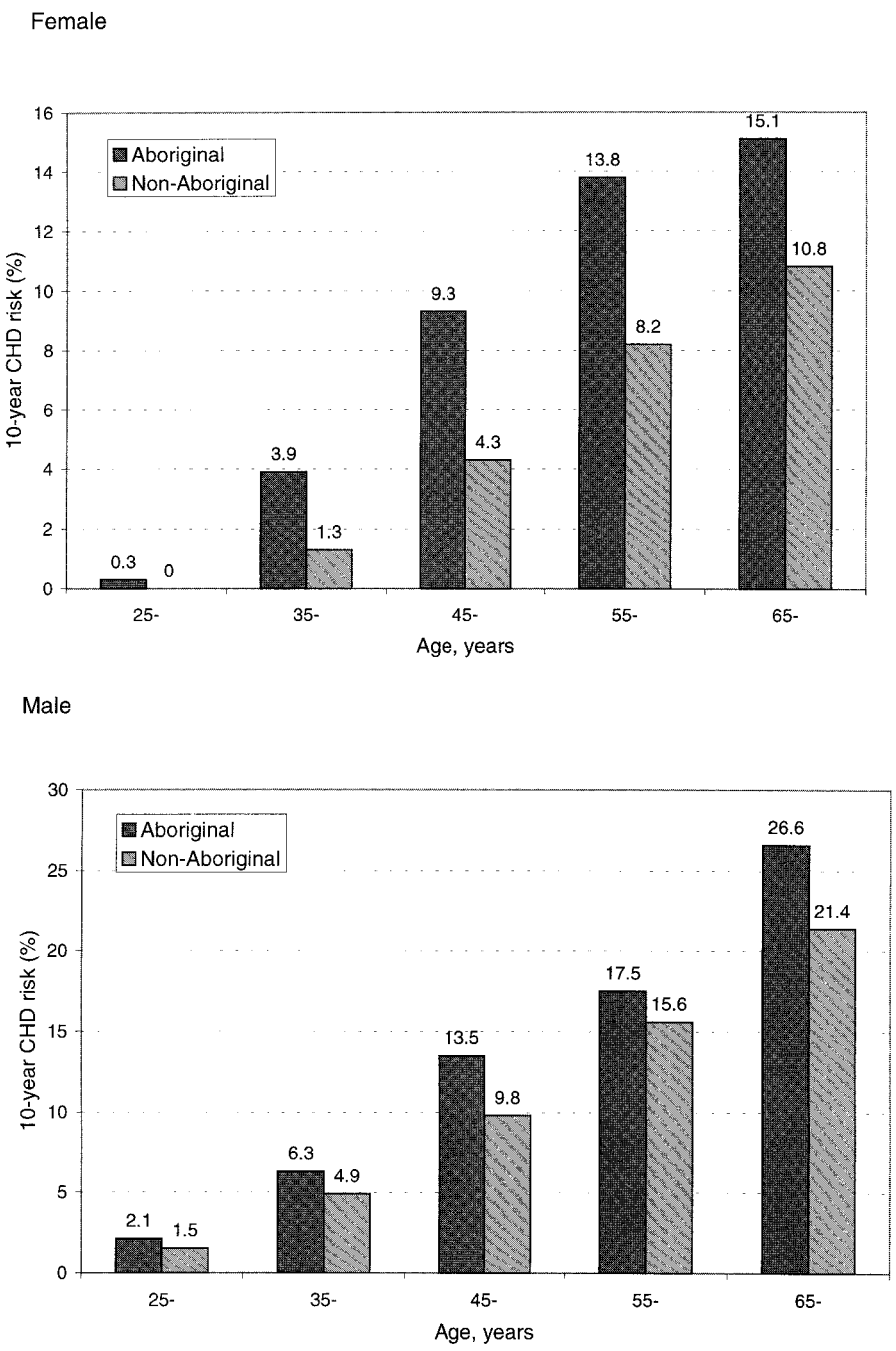

Fig 1. Mean 10-year risk (%) of coronary heart disease for those ages 25–74 years

The high prevalence of risk factors in young Aboriginal adults is a major concern in this community. Young adults not only experienced a relatively higher prevalence of several risk factors than their non-Aboriginal counterparts, they also had a higher prevalence than older age groups in the community.

some populations[9,10,20,21] but not in others.[10,11] Data from Menotti et al suggested that the Framingham function-based coronary risk chart overestimated absolute coronary risk in Italians who typically have a lower incidence of coronary events and should be used with caution.[11] Various absolute risk assessment tools have been developed.[12,22–24] The authors of the Australian Lipid Management Guidelines 2001[13] endorse the cardiovascular disease risk charts used in New Zealand for estimating absolute risk.[12] Aboriginal people were identified as a high CHD risk group in these Guidelines. The generalizability of tools developed from middle-class Americans to Aboriginal Australians has not been evaluated.

The predicted 10-year risks of CHD based on risk factors of total cholesterol, HDL cholesterol, systolic blood pressure, diabetes, and smoking status are higher in Aboriginal people, especially in females. This suggests that the high prevalence of established risk factors can partly explain the higher prevalence of CHD in the Aboriginal population. However, the magnitude of risk difference is far less than the extent observed in mortality and morbidity data. The mortality rates of CHD for the 25–54 age groups were reported to be more than 10 times higher in Aboriginal than other Australians,[1] whereas the estimated risk ratios were only about 1.4 times for males and 1.7 times for females in 45–54 age group. Although high CHD mortality rates in Aboriginal people could be the result of either high occurrence of CHD events or high case-fatality rate or both, it seems less likely that such high mortality ratios are due solely to high case-fatality in Aboriginal people. This suggests that some excess mortality in Aboriginal people may not be addressed through the management

of traditional risk factors. Our findings provide indirect evidence that the Framingham formula may underestimate the CHD risk in Aboriginal Australians. Direct evidence can be obtained through long-term follow-up data. Several longitudinal cohorts from Aboriginal communities have been established,[25–27] so it might be possible to obtain such direct evidence in future studies. Based on the results of this study, we propose that: 1) the predictive values of established risk factors in the Framingham formula should be assessed in Aboriginal populations; 2) other factors, which are not included in the original Framingham formula, may play important roles in high CHD risk in Aboriginal people.

Factors such as obesity or body mass index, family history of CHD, physical activity, albuminuria, fibrinogen, homocysteine Lp(a), small dense LDL, C reactive protein, clotting factors, antibodies to various infectious agents, genetic factors, and estrogen replacement therapy (ERT) have been considered in defining CHD risk.[28–30] Recent primary CHD models developed by D'Agostino et al[28] for assessing CHD risk contain risk factors such as triglycerides levels, alcohol use, and menopausal status. The contributions of those factors in Aboriginal people should be evaluated.

Several aspects of this study should be considered. The data were from a single community with a high rate of premature death. Therefore, the sample may not represent the whole Australian Aboriginal population. The risk factor variables were taken as dichotomous variables, and the detailed distribution for continuous variables, such as blood pressure, cholesterol, HDL, triglycerides levels, have not been compared with those from the general Australian population. The data was cross-sectional, and the occurrence of CHD events could not be obtained. Therefore, the validity of applying Framingham formula to Aboriginal community cannot be assessed directly. A cohort study is now being designed to address some of those issues.

Key Points and Policy Implications

• Aboriginal people living in a remote community had different levels of CHD risk factors from the general Australian population. They had a lower prevalence of abnormal total cholesterol and higher prevalence of abnormal HDL, smoking, diabetes, and hypertension.

• The differences in the prevalence of diabetes, hypertension, and smoking between Aboriginal and non-Aboriginal were more striking among young adults.

• Young females had a higher prevalence of overweight and obesity while males and females 45 years or older had a lower prevalence.

• The predicted 10-year risk of CHD risk is higher in Aboriginal participants, but the magnitude of risk difference is less than the extent reported in mortality data.

• The findings have implications in setting priorities and targets in prevention and management of CHD in Aboriginal communities.

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References

1. Mathur S, Gajanayake I. *Surveillance of Cardiovascular Mortality in Australia 1985–1996.* Canberra: Australian Institute of Health and Welfare; 1998.
2. Thomson N, Winter J, Pumprey M. *Review of the State of Knowledge of Cardiovascular Disease Among Aboriginal and Torres Strait Islander Populations.* Canberra: National Aboriginal and Torres Strait Islander Health Clearinghouse; 1999.
3. Australian Bureau of Statistics and Australian Institute of Health and Welfare. *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples 1999.* Canberra: Australian Bureau of Statistics and the Australian Institute of Health and Welfare; 1999.
4. Commonwealth Department of Health and Age Care and Australian Institute of Health and Welfare. *National Health Priority Areas Report: Cardiovascular Health: A Report on Heart, Stroke, and Vascular Disease.* Canberra: Australian Institute of Health and Welfare; 1999.
5. Australian Institute of Health and Welfare. *Heart, Stroke, and Vascular Diseases—Australian Facts 2001.* Canberra: AIHW, National Heart Foundation of Australia, National Stroke Foundation of Australia; 2001.
6. Guest CS, O'Dea K, Larkins RG. Blood pressure, lipids, and other risk factors for cardiovascular disease in Aborigines and persons of European descent of south-eastern Australia. *Aust J Public Health.* 1994;18:79–86.
7. Anderson KM, Odell PM, Wilson PW, et al. Cardiovascular disease risk profiles. *Am Heart J.* 1991;121:293–298.
8. Wilson PW, D'Agostino RB, Levy D, et al. Prediction of coronary heart disease using risk factor categories. *Circulation.* 1998;97:1837–1847.
9. Suka M, Sugimori H, Yoshida K. Application of the updated Framingham risk score to Japanese men. *Hypertens Res.* 2001;24:685–689.
10. D'Agostino RB Sr, Grundy S, Sullivan LM, et al. Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation. *JAMA.* 2001;286:180–187.
11. Menotti A, Puddu PE, Lanti M. Comparison of the Framingham risk function-based coronary chart with risk function from an Italian population study. *Eur Heart J.* 2000;21:365–370.
12. Jackson R. Guidelines on preventing cardiovascular disease in clinical practice. *BMJ.* 2000;320:659–661.
13. National Heart Foundation of Australia, The Cardiac Society of Australia and New Zealand. Lipid management guidelines—2001. *Med J Aust.* 2001;175:S57–S85.
14. The Australian Diabetes, Obesity, and Lifestyle Study (AusDiab). *Diabesity and Associated Disorders in Australia 2000: The Accelerating Epidemic.* Melbourne: The International Diabetes Institute; 2001.
15. World Health Organization. *Definition, Diagnosis, and Classification of Diabetes Mellitus and Its Complications; Part 1: Diagnosis and Classification of Diabetes Mellitus.* Geneva: Department of Noncommunicable Disease Surveillance, WHO; 1999.

16. Anderson KM, Wilson PW, Odell PM, et al. An updated coronary risk profile. A statement for health professionals. *Circulation.* 1991;83: 356–362.
17. Bastian P. Coronary heart disease in tribal Aborigines—the West Kimberley survey. *Aust N Z J Med.* 1979;9:284–292.
18. Leonard D, McDermott R, Odea K, et al. Obesity, diabetes, and associated cardiovascular risk factors among Torres Strait Islander people. *Aust N Z J Public Health.* 2002;26: 144–149.
19. Wang Z, Hoy W, McDonald S. Body mass index in Aboriginal Australians in remote communities. *Aust N Z J Public Health.* 2000; 24:570–575.
20. Haq IU, Ramsay LE, Yeo WW, et al. Is the Framingham risk function valid for northern European populations? A comparison of methods for estimating absolute coronary risk in high risk men. *Heart.* 1999;81:40–46.
21. Liao Y, McGee DL, Cooper RS, et al. How generalizable are coronary risk prediction models? Comparison of Framingham and two national cohorts. *Am Heart J.* 1999;137:837–845.
22. West of Scotland Coronary Preventive Study group. West of Scotland Coronary Preventive Study: identification of high-risk groups and comparison with other cardiovascular intervention trials. *Lancet.* 1996;348:1339–1342.
23. Pyorala K, De Backer G, Graham I, et al. Prevention of coronary heart disease in clinical practice. Recommendations of the Task Force of the European Society of Cardiology, European Atherosclerosis Society, and European Society of Hypertension. *Eur Heart J.* 1994;15:1300–1331.
24. Haq IU, Jackson PR, Yeo WW, et al. Sheffield risk and treatment table for cholesterol lowering for primary prevention of coronary heart disease. *Lancet.* 1995;346:1467–1471.
25. Hoy WE, Wang Z, VanBuynder P, et al. The natural history of renal disease in Australian Aborigines. Part 1. Changes in albuminuria and glomerular filtration rate over time. *Kidney Int.* 2001;60:243–248.
26. McDonald SP, Wang Z, Hoy WE. Physical and biochemical predictors of death in an Australian aboriginal cohort. *Clin Exp Pharmacol Physiol.* 1999;26:618–621.
27. Daniel M, Rowley KG, McDermott R, et al. Diabetes incidence in an Australian aboriginal population. An 8-year follow-up study. *Diabetes Care.* 1999;22:1993–1998.
28. D'Agostino RB, Russell MW, Huse DM, et al. Primary and subsequent coronary risk appraisal: new results from the Framingham study. *Am Heart J.* 2000;139:272–281.
29. Niccoli G, Iacoviello L, Cianflone D, et al. Coronary risk factors: new perspectives. *Int J Epidemiol.* 2001;30:S41–S47.
30. Kannel WB. The Framingham Study: its 50-year legacy and future promise. *J Atheroscler Thromb.* 2000;6:60–66.

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