Coronary heart disease events in Aboriginal Australians: incidence in an urban population

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ABSTRACT

Objective: To determine the incidence of coronary heart disease (CHD) events in an urban Aboriginal population.

Design, setting and participants: Cohort study of 906 Aboriginal people without CHD from 998 who had undergone risk-factor assessment in the Perth Aboriginal Atherosclerosis Risk Study (PAARS) in 1998–1999. PAARS cohort data were electronically linked to a range of databases that included Western Australian hospital morbidity data and death registry data. We analysed data from January 1980 to December 2006 to identify previous admissions for CHD from 1980 to baseline (1998–1999) and new events from baseline to 2006.

Main outcome measure: First CHD event (hospital admission or death).

Results: There were 891 linked records for the 906 participants without previous CHD. The event rate was 12.6/1000 person-years (95% CI, 10.2–15.6/1000 person-years). Annual CHD event rates ranged from 8 to 18/1000 person-years. After adjustment for age (sex was not associated with the risk factors assessed), factors associated with risk of a CHD event in the PAARS cohort were a history of diabetes, overweight or obesity (indicated by body mass index), smoking, and hypertension, but not waist circumference. People with these risk factors were 1.9–2.7 times more likely to experience a CHD event. Compared with previously published information from a remote Aboriginal community in the Northern Territory, the incidence of CHD events among urban-dwelling Aboriginal people was not significantly different ($P > 0.05$ overall and for subgroups defined by age and sex).

Conclusions: City-dwelling Aboriginal Australians have an incidence of CHD events comparable to that of Aboriginal people living in remote northern Australia.

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Cardiovascular disease (CVD) is the major cause of premature death among adult Aboriginal Australians, and contributes to a reduced life expectancy.[1] Age-specific death rates from CVD in Aboriginal people are estimated to be four to seven times the rates in the general population.[2] Despite these statistics, there is almost no information on the incidence of CVD among Aboriginal people, as there have been few opportunities to follow cohorts in longitudinal studies, especially among urban populations.[3]

The only sizeable study to report the incidence of coronary heart disease (CHD) in Aboriginal people comes from a remote community in the Northern Territory, where the rate of CHD events was 11.0 per 1000 person-years among participants for whom a Framingham score could be calculated.[4] To investigate whether those findings were applicable to other Aboriginal populations, we measured cardiovascular events in the Perth Aboriginal Atherosclerosis Risk Study (PAARS) cohort, an urban-dwelling population.[5] Our aim was to determine the incidence of cardiovascular events in a cohort of urban adult Aboriginal people without known CHD.

METHODS

The PAARS has been described in detail previously.[5] Briefly, an atherosclerosis risk factor study was conducted in 1998–1999 among 998 residents of Perth, Western Australia, who identified themselves as Aboriginal Australians. The mean age of participants was 40 years (range, 18–80 years).

Perth is a city of 1.6 million people, of whom less than 2% are Aboriginal.[6] At the time of initial assessment (1998–1999), the PAARS cohort was representative of the broader adult Aboriginal population of Perth in age structure, but with a higher proportion of employed people.

Data linkage

Outcome data were obtained through record linkage to population-based administrative databases, facilitated by the WA Data Linkage System. The external dataset (the PAARS cohort data) was electronically linked to a range of databases that included hospital morbidity data for public and private hospitals in WA (1980–2006) and deaths recorded in the WA Registry of Births, Deaths and Marriages (1998–2006). Probabilistic matching of records was based on name, date of birth, sex, and last known residential address. The validity of the matching for hospital morbidity in the general population has been assessed, with false positives and false negatives each estimated to be 0.11%.[7]

We analysed data from January 1980 to December 2006 to identify previous admissions for CHD from 1980 to baseline (1998–1999) and new events from baseline to 2006.

Outcome variable

The primary outcome was a CHD event, defined as a first hospital admission for ischaemic heart disease, a coronary revascularisation procedure, or a cardiac-related death. Events were identified using the International classification of diseases ICD-9-CM (ninth revision, clinical modification) and ICD-10-AM (tenth revision, Australian modification) codes, including those for acute myocardial infarction (410, I21), angina pectoris (411, I20) and other ischaemic heart disease (413–414, I22–I25), together with procedure codes for coronary artery bypass grafting and percutaneous coronary intervention. Outcome events from baseline assessment to 2006 were captured from the principal diagnosis field, while a "previous CHD" diagnosis was identified from up to 21 diagnosis fields recorded during any admission from 1980 and prior to the baseline assessment in 1998–1999.

The time from baseline assessment to the first CHD event was calculated for each participant who reached an event; otherwise, follow-up was censored at the date of death from a non-cardiac cause or 31 December 2006.

Definitions

Hypertension was defined by blood pressure ⩾ 160/95 mmHg or current use of antihypertensive medication.

Overweight and obesity were defined in two different ways:

- By *body mass index* (BMI): BMI 25.0–29.9 kg/m^2 was defined as overweight and BMI ⩾ 30.0 kg/m^2 as obese; or
- By *waist circumference*: waist circumference 94.0–101.9 cm (men) and 80.0–87.9 cm (women) was defined as overweight, and waist circumference ⩾ 102.0 cm (men) and ⩾ 88.0 cm (women) as obese.[8]

Analysis

After excluding the records of unmatched participants, we divided the cohort into those with and without a history of CHD. Using the Kaplan–Meier method, the cumulative incidence of CHD to 9 years was estimated for participants with no history of CHD. The cohort was stratified by age at baseline into four groups (< 35, 35–44, 45–54 and ⩾ 55 years) for comparison with the event rate reported from a remote community.[4] The incidence of CHD events per 1000 person-years was calculated, with follow-up time assigned proportionally for those who were in two age groups during the study period. (For instance, if a person followed up for 7 years was 43 years of age at baseline, they would contribute 2 person-years to the 35–44-year age group and 5 person-years to the 45–54-year age group, if they did not have a CHD event.) Sex-specific rates were also calculated.

The incidence of CHD events was also calculated for groups defined by the presence or absence of common risk factors at baseline: reported diabetes, hypertension and obesity. Incident rate ratios were calculated for these groups. The joint effects of these variables were assessed in a Cox proportional hazards model in which age and sex were also included.

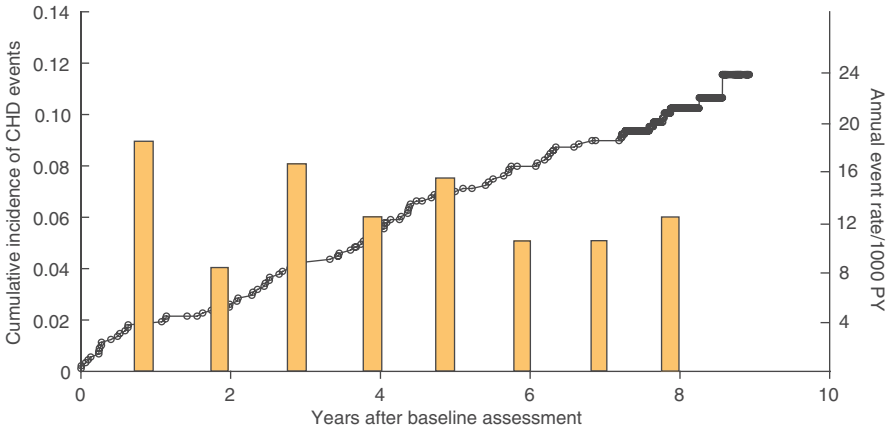
Finally, the age-group and sex-specific rates were compared with summary data published by Wang and Hoy from their investigations in a remote NT Aboriginal community in 1992–1995.[4] The study participants from the community were followed to December 2003, with first CHD events (fatal or non-fatal) identified from hospital and death records.

Analyses were conducted using Stata software, version 10 (StataCorp, College Station, Tex, USA) and SPSS software, version 15 (SPSS Inc, Chicago, Ill, USA).

Ethical issues

Perth lies within the traditional lands of the Nyoongar people of south-western WA. The PAARS was conducted in collaboration with the Derbarl Yerrigan Health Service Inc (formerly Perth Aboriginal Medical Services) and in accordance with National Health and Medical Research Council (NHMRC) criteria for health and medical research among Indigenous Australians.[9] All participants in

1 Cumulative incidence of CHD events since baseline assessment (line) and annual event rate per 1000 person-years (PY) (bars) for the PAARS cohort



CHD = coronary heart disease. PAARS = Perth Aboriginal Atherosclerosis Risk Study. ◆

2 Incidence of CHD events in the PAARS cohort compared with incidence in a remote Aboriginal community in the Northern Territory, by age group and sex*

	PAARS cohort without previous CHD (n = 891)			Remote community (n = 687)[4]	
Age group (years)	Person-years	Events	Rate/1000 person-years (95% CI)	Person-years	Rate/1000 person-years (95% CI)
All					
< 35	2702	8	3.0 (1.5–6.0)	2822	3.2 (1.7–6.1)
35–44	2221	35	15.8 (11.3–22.0)	1827	9.8 (6.2–15.5)
45–54	1452	29	20.0 (13.9–28.7)	949	19.7 (12.6–30.9)
⩾ 55	456	14	30.7 (18.2–51.9)	562	39.2 (25.8–59.5)
Total	6830	86	12.6 (10.2–15.6)	6188	11.0 (8.7–13.9)
Women					
< 35	1420	3	2.1 (0.7–6.6)	1197	3.3 (1.3–8.9)
35–44	1198	17	14.2 (8.8–22.8)	858	10.5 (5.5–20.2)
45–54	844	17	20.1 (12.5–32.4)	553	18.1 (9.7–33.6)
⩾ 55	273	9	33.0 (17.1–63.3)	329	45.6 (27.5–75.6)
Total	3735	46	12.3 (9.3–16.6)	2936	12.9 (9.4–17.8)
Men					
< 35	1282	5	3.9 (1.6–9.4)	1625	3.1 (1.3–7.4)
35–44	1023	18	17.6 (11.1–27.9)	983	9.2 (4.8–17.6)
45–54	608	12	19.8 (11.2–34.8)	411	21.9 (11.4–42.1)
⩾ 55	182	5	24.4 (11.4–65.9)	232	30.1 (14.4–42.1)
Total	3095	40	12.9 (9.8–18.1)	3252	9.2 (6.5–13.2)

CHD = coronary heart disease. PAARS = Perth Aboriginal Atherosclerosis Risk Study. * P values were > 0.05 for all comparisons between the two cohorts. ◆

3 Comparison of CHD event rates per 1000 person-years for PAARS participants with and without common risk factors for CHD

	Number of people	Events	Person-years	Event rate*	Incidence rate ratio (95% CI)	Adjusted incident rate ratio† (95% CI)	*P*
Reported diabetes							
No	722	46	5552	8.3	1.0	1.0	
Yes	169	40	1157	34.6	4.1 (2.7–6.3)	2.4 (1.4–3.8)	0.001
Waist circumference‡							
Not overweight	246	16	1879	8.5	1.0	1.0	
Overweight	163	10	1259	7.8	0.9 (0.4–2.1)	0.4 (0.2–1.0)	0.054
Obese	442	57	3279	17.4	2.0 (1.2–3.5)	0.66 (0.3–1.4)	0.30
BMI (kg/m^2)§							
< 25.0	196	8	1508	5.3	1.0	1.0	
25.0–29.9	266	28	1983	14.1	2.5 (1.1–5.5)	2.7 (1.2–6.4)	0.023
⩾ 30.0	381	47	2864	16.4	2.9 (1.4–6.2)	2.7 (1.0–7.2)	0.042
Current cigarette smoker							
No	472	37	3619	10.2	1.0	1.0	
Yes	417	49	3074	15.9	1.6 (1.0–2.4)	2.7 (1.7–4.2)	< 0.001
Hypertension¶							
No	716	52	5480	9.5	1.0	1.0	
Yes	175	34	1228	27.7	2.9 (1.9–4.7)	1.9 (1.2–3.0)	0.007

BMI = body mass index. CHD = coronary heart disease. PAARS = Perth Aboriginal Atherosclerosis Risk Study. * Incidence of CHD events per 1000 person-years. † Adjusted for age, sex, diabetes status, waist circumference, BMI, smoking status and hypertension. ‡ Overweight: waist circumference 94.0–101.9 cm (men) and 80.0–87.9 cm (women); obese: waist circumference ⩾ 102.0 cm (men) and ⩾ 88.0 cm (women). § Overweight: BMI 25.0–29.9 kg/m^2; obese: BMI ⩾ 30.0 kg/m^2. ¶ Hypertension: blood pressure ⩾ 160/95 mmHg or current use of antihypertensive medication. ◆

the PAARS study provided written informed consent using a form of consent developed in consultation with community representatives.

Ethical approval for the study was obtained from the WA Aboriginal Health Information and Ethics Committee, the Human Research Ethics Committee of the University of Western Australia and the WA Confidentiality of Health Information Committee.

RESULTS

There was a minimum of 7 years' follow-up for all participants, providing more than 6830 person-years of data. Record linkage was possible for 983 of the 998 PAARS participants (98.5%). Of the 983 with linked data, 931 had a record of at least one hospitalisation (for any cause), or a death record. There were 14 503 hospital admissions, with 86 first CHD events identified for people with no history of CHD. There were 53 deaths during the follow-up period for the whole cohort. Of those, 15 were due to CHD, the most common cause. Among the 891 with a linked record and no history of CHD, there were 28 deaths, of which six were due to CHD.

The annual CHD event rate was highest in the first year after assessment (18/1000 person-years) and varied between 8 and 16 per 1000 person-years in subsequent years (Box 1). The cumulative hazard estimate for a first CHD event to 9 years was 12.6/1000 person-years in the PAARS cohort, compared with 11.0/1000 person-years in the remote community (Box 2). The incidence of CHD increased with age, and was little different for men and women. Analysis by age group showed a tendency towards a higher CHD event rate in the PAARS cohort than the remote community in the 35–44-year age group (especially for men), but the opposite pattern in the ⩾ 55-year age group. However, the confidence intervals were large and the differences not statistically significant (Box 2).

After adjustment for age (sex was not associated with the risk factors assessed), factors associated with risk of a cardiac event in the PAARS cohort were a history of diabetes, overweight or obesity (indicated by BMI), smoking, and hypertension, but not waist circumference (Box 3). PAARS cohort participants with no history of CHD at baseline (which includes those who either did or did not experience a CHD event during follow-up) were characterised by a high proportion of overweight or obesity, with more than three-quarters having a BMI > 25 kg/m^2. Compared with Aboriginal people from the remote community, they were older and had a higher proportion of reported diabetes, as well as higher mean blood pressure (especially among the women). However, rates of current smoking were significantly lower in the PAARS cohort than the remote community. Over 35% of men and 40% of women in the PAARS cohort had never smoked. The main risk factors present at baseline are shown in Box 4.

DISCUSSION

Our results showed that the CHD event rate was similar in PAARS participants (12.6/1000 person-years) to the rate in the remote NT population (11.0/1000 person-years). However, the overall rate for those living in the remote community would be higher than this figure, as it did not include the 202 participants (23%) who had insufficient data to calculate a Framingham risk score. For those excluded, the rate was estimated to be 17.8/1000 person-years (95% CI, 11.9–26.6/1000 person-years).[4]

Although differences in CHD event rates between PAARS participants and the remote population appeared to be considerable in some age groups, the differences were not statistically significant and no firm conclusions can be drawn about differences in age-related rates of CHD for Aboriginal people living in different environments.

Risk factors for cardiovascular disease (especially smoking, diabetes and obesity) were highly prevalent in both cohorts. Although some studies have found waist circumference to be a better predictor of diabetes among Aboriginal people than BMI,[10] our results showed that higher BMI, but not waist circumference, was associated with a higher incidence of CHD events.

Although the city-dwellers were less likely to be current smokers, blood pressure, prevalence of diabetes and measures of obesity were higher in this group. The

4 CHD risk factors at baseline (1998–1999) for PAARS participants compared with those reported for a remote Aboriginal community in the Northern Territory*

	PAARS cohort without CHD		Remote community[4]	
Risk factor	Men ($n = 400$)	Women ($n = 491$)	Men ($n = 356$)	Women ($n = 331$)
Age (years)	38.3 (10.4)	39.2 (10.8)	32.8	36.1
Systolic BP (mmHg)	126.7 (14.6)	123.5 (16.8)	125.5	116.4
Diastolic BP (mmHg)	82.2 (10.6)	79.2 (11.2)	77.7	71.5
Total cholesterol (mmol/L)†	5.5 (1.0)	5.2 (1.1)	4.9	4.5
HDL cholesterol (mmol/L)†	1.1 (0.26)	1.3 (0.32)	1.1	1.0
Body mass index (kg/m^2)	29.1 (5.1)	30.6 (7.2)	23.2	24.1
Waist circumference (cm)	97.7 (13.2)	93.2 (15.9)	86.9	91.2
Reported diabetes (%)	18%	20%	9%	16%
Current cigarette smoker (%)‡	48%	46%	84%	71%
Overweight (%)	39%	25%	nd	nd
Obese (%)	40%	51%	nd	nd
Hypertension (%)§	20%	19%	nd	nd

BP = blood pressure. CHD = coronary heart disease. HDL = high-density lipoprotein. nd = no data. PAARS = Perth Aboriginal Atherosclerosis Risk Study. * Values are mean (SD) unless otherwise specified. † 752 PAARS participants had lipid measurements (350 men, 402 women). ‡ Current smoker or quit within previous 12 months. § BP ⩾ 160/95 mmHg or current use of antihypertensive medication. ◆

higher blood pressure and greater prevalence of obesity may reflect the higher mean age of the PAARS cohort. Lower smoking rates in the PAARS cohort may be related to several factors, including greater exposure to anti-smoking messages in the city, legislation precluding smoking in the workplace (the PAARS cohort contained a higher proportion of employed people than the broader Perth Aboriginal population), and higher levels of education. It is possible that a sedentary lifestyle and easier access to a greater variety of food (including take-away food) are related to the greater prevalence of obesity among city-dwellers, although the higher average age of the PAARS participants may be sufficient explanation. Also, there were some years between the baseline data collections: the data reported by Wang and Hoy were collected between 1992 and 1995[4] and those for the PAARS cohort in 1998–1999. The 3–7-year gap may have encompassed social changes that influenced health behaviours such as smoking habits, diet and exercise in one or both settings. The risk factors for the PAARS cohort were measured only at baseline, and so there is no information on their progression, regression or treatment over the intervening 7 years.

CONCLUSION

The incidence of CHD events in city-dwelling Aboriginal Australians is comparable with the incidence in Aboriginal people living in remote northern Australia. Neither environment provides protection from the hazards of risk factors such as smoking and obesity, with the associated increased risk of developing diabetes and hypertension. Efforts to reduce risk factor prevalence among Aboriginal people should be intensified at all levels, bearing in mind that those living in cities are at just as great a risk for CHD as those living in remote communities.

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COMPETING INTERESTS

None identified.

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