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Albuminuria in Australian Aboriginal people: prevalence and associations with components of the metabolic syndrome

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Abstract

Aims/hypothesis. To examine the prevalence and associations with the metabolic syndrome of albuminuria among Australian Aboriginal people.
Methods. Early-morning urine specimens were collected as part of community-based risk factor surveys assessing the prevalence of diabetes and cardiovascular disease in eight remote communities, with a sample size of 1,075 people. Microalbuminuria was defined as urinary albumin : creatinine ratio 3.4–33.9 mg/mmol, macroalbuminuria as albumin : creatinine ratio equal to or greater than 34 mg/mmol.
Results. There were high prevalences of microalbuminuria (men 22.2%, women 26.9%) and of macroalbuminuria (men 10.4%, women 13.5%). There were highly statistically significant linear associations of microalbuminuria and macroalbuminuria with increasing number of coexisting components of the metabolic syndrome (hypertension, glucose intolerance, dyslipidaemia, insulin resistance, abdominal obesity): among people with zero, one, two and three to five of these conditions, respectively, prevalence of microalbuminuria was 16%, 20%, 36% and 32% ($p < 0.001$); prevalence of macroalbuminuria was 2%, 6%, 12% and 32% ($p < 0.001$). There were independent associations of microalbuminuria with hypertension (odds ratio, 95% confidence interval = 2.36, 1.63–3.42) and diabetes (2.10, 1.28–3.45): macroalbuminuria was independently associated with hypertension (6.39, 3.93–10.4), diabetes (3.49, 1.93–6.28) and abdominal obesity (4.56, 2.40–8.64) and had a weaker association with insulin resistance (1.99, 1.12–3.54). Dyslipidaemia and impaired glucose tolerance were neither independently associated with microalbuminuria or macroalbuminuria, nor was insulin resistance or abdominal obesity independently associated with microalbuminuria.
Conclusion/interpretation. There was a strong clustering of albuminuria with components of the metabolic syndrome. Diabetes, hypertension and abdominal obesity are major contributors to high rates of albuminuria among Australian Aboriginal people. [Diabetologia (2000) 43: 1397–1403]

Keywords Microalbuminuria, Australian Aboriginal people, epidemiology, metabolic syndrome, insulin resistance.

High prevalences of albuminuria and renal disease have been reported in several Australian Aboriginal populations [1, 2]. The incidence of end-stage renal disease in Northern Territory Aboriginal people was 925 per million in 1996 [3] and in a cohort of 374 central Australian Aboriginal people with diabetes followed from 1984 to 1991, renal disease was the most common cause of death [4]. Cardiovascular diseases remain the leading cause of death among Aboriginal and Torres Strait Islander people [5] and albuminuria is probably a risk factor for cardiovascular disease in this population as it is in others [6–8].

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Abbreviations: ACR, Albumin to creatinine ratio; HDL, high density lipoprotein; HOMA, homeostasis model assessment; OR, odds ratio; PAR %, population attributable risk per cent; UTI, urinary tract infection.

Among Aboriginal people there is a high prevalence of cardiovascular risk factors commonly associated with the metabolic syndrome [9]. Diabetes and hypertension strongly predict the presence of albuminuria in several population studies [10–14]. Some studies have indicated that abdominal obesity, insulin resistance and dyslipidaemia are also independently associated with albuminuria [2, 11, 14].

As part of diabetes and cardiovascular disease risk-factor surveys conducted in remote communities in northern and central Australia between 1995 and 1997, we examined urinary albumin excretion in community-based groups of over 1000 adult Aboriginal people. We report the prevalence of microalbuminuria and macroalbuminuria and have sought to determine the associations of albumin excretion with a range of conditions that constitute the metabolic syndrome, including insulin resistance and abdominal obesity.

Subjects and methods

The protocols described were approved by Deakin University Ethics Committee (the institution responsible at the time of the surveys), the Alice Springs Institutional Ethics Committee, the Ethics Committee of the Peninsula and Torres Strait Regional Health Authority and Cairns Base Hospital Institutional Ethics Committee.

Subject selection. Between 1995 and 1997 eight remote communities were screened for diabetes, albuminuria and other cardiovascular risk factors. These community-based surveys were carried out in three central Australian communities, one in the Kimberley region of Western Australia and four in Cape York, Queensland. All community members aged 15 and older were invited to participate. The response rates were 70% for central Australian Aboriginal people, 75% for Kimberley Aboriginal people and approximately 40% for Cape York Aboriginal people. The surveys in the Cape York area included a number of Torres Strait Islander people and these were excluded from this study. The survey sample was representative of the Australian indigenous population [15] for age and sex distribution. Further details of screening procedures and survey sample characteristics have been reported previously [16, 17]. Urine samples, complete anthropometric data (body weight and height, waist and hip circumferences), blood pressure measurements and an oral glucose tolerance test or a previously confirmed diagnosis of diabetes were available for 92% of this survey sample and these subjects are included in the study. Pregnant women ($n = 10$) were excluded.

Laboratory and anthropometric techniques. Because 24-h urine collections were not possible for cultural and logistical reasons, the albumin to creatinine ratio (ACR) in early-morning urine samples with which it correlates well was used as a surrogate marker of albumin excretion rate [18]. Samples were kept cool before being frozen at –20 °C or colder and were assayed within 2 months (usually less than 1 month) of collection. The stability of albumin during freeze-thawing has been questioned but the differences reported after freeze-thawing are generally small and occur in a non-differential manner [19]. Although field conditions require freezing of samples, this process did not greatly influence our conclusions. Urinary albumin concentration was measured using immunonephelometry [Kallestadt QM300 or Beckman 360 Array nephelometers; interassay coefficient of variance (CV) 3–5%]. Urinary creatinine concentration was measured using an alkaline picrate method (Olympus AU800 autoanalyser; interassay CV 2%). Microalbuminuria was defined as ACR in the range of 3.4 to less than 34 mg/mmol and macroalbuminuria was defined as an ACR of 34 mg/mmol or more, recommended by the United States National Kidney Foundation [20] and also appropriate for a population with a linear body build and a relatively low lean mass for a given BMI [21–24]. For samples collected in central Australia, suspected urinary tract infection (UTI) was diagnosed by urinalysis using reagent strips (N-Multistix SG, Bayer Diagnostics, Mulgrave, VIC, Australia) and these samples were sent for further microbiological examination to confirm the diagnosis. The presence of haematuria was also investigated using these reagent strips.

Plasma glucose, triglycerides and high density lipoprotein (HDL) cholesterol (after precipitation of other lipoproteins with 15% polyethylene glycol MW 6000) were measured using standard enzymatic techniques (Boehringer-Mannheim reagents; interassay CV < 7%). Insulin was measured by radioimmunoassay using a specific antibody (Linco Research, St. Charles Mo., USA; interassay CV 9–13%). Insulin resistance was estimated from fasting glucose and insulin concentrations using homeostasis model assessment (HOMA) [25]. Blood pressure was measured with a Dinamap automated blood pressure monitor (Critikon Inc., Tampa Fla., USA). Body weight was measured to 0.1 kg and height, waist, and hip circumferences to 0.1 cm. For categorising conditions that constitute the metabolic syndrome, dyslipidaemia was defined as the combination of increased fasting triglycerides (≥ 2.0 mmol/l) and low HDL cholesterol (< 0.91 mmol/l), hypertension was defined as systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg and/or current antihypertensive medication [26]. Subjects were categorised as "insulin resistant" if in the highest quartile of HOMA insulin resistance and cut-offs for high waist-to-hip ratio were 1.0 for men and 0.85 for women [27]. Diabetes and impaired glucose tolerance (IGT) were defined using World Health Organization (WHO) criteria based on a 75-g oral glucose tolerance test.

Statistical analyses. Differences in prevalence of microalbuminuria and macroalbuminuria across strata were tested using the chi-squared statistic for independence (χ^2_{indep}) or the Mantel-Haenzel chi-squared statistic for linear trend (χ^2_{trend}) as appropriate. Differences in mean levels of continuous variables across categories of ACR were tested by ANOVA using the General Linear Modelling function on SPSS 9.0 (SPSS Inc., Chicago Il., USA). Values for triglycerides and insulin resistance were log-transformed for these analyses and data are presented as geometric mean. Multinomial logistic regression analysis was used to determine odds ratios for categorical predictors of microalbuminuria and macroalbuminuria: blood pressure (hypertensive vs normotensive), glucose tolerance (non-diabetic versus diabetic), dyslipidaemia, BMI (<25, 25–70, > 30 kg/m^2), waist-to-hip ratio (normal vs high), insulin resistance (quartiles of HOMA insulin resistance), age (15–24 years, 25–34 years, 35–44 years and 45 years or more), sex and geographic location (central Australia, Kimberley, Cape York). Population attributable risk per cent was calculated using the formula: $100 \times P_e\,(OR - 1)/(P_e(OR\text{-}1) + 1)$, where P_e is the proportion of the population exposed to the predictor variable of interest [28]. differences were considered statistically significant at $p < 0.05$.

Table 1. Prevalence of elevated urinary ACR stratified by age, sex and geographic location ($n = 1075$)

	n	Prevalence (%) Microalbuminuria	Macro-albuminuria
Age group			
15–24 years	302	16.6	5.0
25–34 years	290	22.4	9.0
35–44 years	181	24.9	14.4
45 years +	302	35.4	20.9
p-value		< 0.001	< 0.001
Sex			
Male	473	22.2	10.4
Female	602	26.9	13.5
p-value		0.076	0.122
Location			
Central Australia	794	25.1	12.5
Kimberley	174	21.3	14.4
Cape York	107	29.0	5.6
p-value		0.334	0.075

Results

There was a strong linear trend towards increasing prevalence with increasing age for both microalbuminuria ($\chi^2_{trend} = 48.0$, $p < 0.001$) and macroalbuminuria ($\chi^2_{trend} = 39.4$, $p < 0.001$; Table 1). Men and women had similar prevalences of microalbuminuria ($\chi^2_{indep} = 3.2$, p not significant) and macroalbuminuria ($\chi^2_{indep} = 2.4$, p not significant; Table 1). There were no significant differences in the crude prevalence of microalbuminuria ($\chi^2_{indep} = 2.2$, p not significant) or macroalbuminuria ($\chi^2_{indep} = 5.2$, p not significant) according to geographic location (Table 1).

When continuous variables were stratified by categories of ACR, there were strong associations of systolic and diastolic blood pressure with increasing ACR (Table 2). The exclusion of patients currently prescribed antihypertensive medication ($n = 70$) did not substantially alter these associations (data not shown). Body mass index varied significantly across ACR categories: mean (95 % CI) BMI was 25.4 (24.9 – 26.0), 27.7 (26.9 – 28.5) and 28.5 (27.4 – 29.6) kg/m^2 for normal, microalbuminuric and macroalbuminuric people respectively ($p < 0.001$). Waist-to-hip ratio varied significantly across ACR categories (Table 2), with successive increases in mean value with greater degree of albuminuria. Fasting plasma triglycerides varied significantly across categories of ACR, with significantly elevated concentration in macroalbuminuric people compared to normal and microalbuminuric people (Table 2) but HDL cholesterol was only weakly associated with albuminuria. Insulin resistance estimated from HOMA was significantly greater with increasing ACR category (Table 2).

When categorical variables were stratified by categories of ACR, there was a statistically significant increase in the age-adjusted prevalence (using the overall survey sample as the reference population) of diabetes with worsening degrees of albuminuria ($\chi^2_{trend} = 74.2$, $p < 0.001$; Table 2). The prevalence of IGT did not vary according to ACR category ($\chi^2_{trend} = 0.4$, p not significant; Table 2). When glucose tolerance was defined according to fasting plasma glucose concentration using American Diabetes Association (ADA) criteria [29], the age-adjusted prevalence of impaired fasting glucose was 4.3 %, 5.1 % and 4.5 % in normal, microalbuminuric and macroalbuminuric people respectively ($\chi^2_{trend} = 0.03$, $p = 0.875$) and the prevalence of diabetes was 6.7 %, 17.9 % and 34.1 % in normal, microalbuminuric and macroalbuminuric people respectively ($\chi^2_{trend} = 76.1$, $p<0.001$). Among central Australian Aboriginal people, the presence of 'moderate' or greater degrees of haematuria (> 80 cells/μl) upon dipstick analysis was considered to indicate glomerulonephritis. The prevalence of haematuria was 8.5 %, 17.6 % and 17.2 % in normal, microalbuminuric and macroalbuminuric people respectively ($\chi^2_{trend} = 13.3$, $p < 0.001$). Stratification of the survey sample by the presence or absence of haematuria did not greatly alter the associations with the variables shown in Table 2 (data not shown). We identified 49 confirmed cases of UTI (2 men, 47 women) among central Australian Aboriginal people. The prevalence of microalbuminuria and macroalbuminuria was similar in those women with and without UTI (data not shown).

Table 2. Blood pressure, waist-to-hip ratio, plasma lipids, insulin resistance and glucose tolerance stratified by ACR category

	ACR category			*p*-value
	Normal	Microalbuminuria	Macroalbuminuria	
n	678	267	130	
Systolic BP, mmHg	124 (123–125)	129 (127–131)	140 (137–143)	< 0.001
Diastolic BP, mmHg	69 (68–69)	71 (70–73)	80 (78–82)	< 0.001
Waist-to-hip ratio	0.90 (0.89–0.90)	0.92 (0.91–0.93)	0.96 (0.94–0.97)	< 0.001
Triglycerides, mmol/l	1.7 (1.6–1.8)	2.0 (1.8–2.1)	2.5 (2.2–2.7)	< 0.001
HDL cholesterol, mmol/l	0.91 (0.89–0.93)	0.87 (0.84–0.90)	0.87 (0.83–0.91)	0.023
Insulin resistance (HOMA)	3.7 (3.4–3.9)	4.8 (4.4–5.3)	5.8 (5.1–6.6)	< 0.001
IGT %	10.0	9.5	12.7	0.510
Diabetes %	9.6	21.5	39.0	< 0.001

Data are estimated marginal mean (95 % confidence interval) adjusted for age, sex and geographic location except IGT and diabetes, which are age-adjusted prevalence

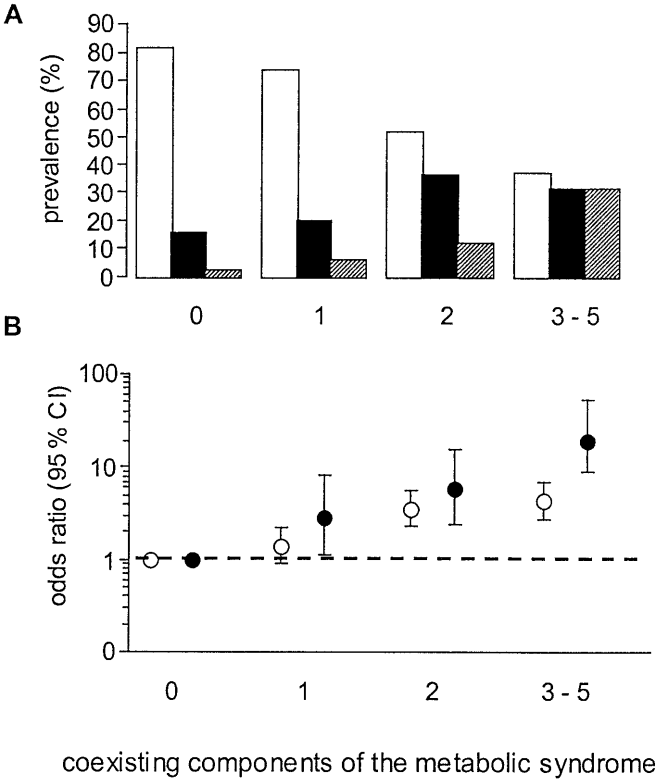

Fig. 1. **A** Prevalence of normal ACR (□), microalbuminuria (■) and macroalbuminuria (▨) stratified by the number of coexisting components of the metabolic syndrome (hypertension, abnormal glucose tolerance, dyslipidaemia, insulin resistance and abdominal obesity). For people with zero, one, two and three or more components respectively, $n = 312$, 292, 209 and 244; **B** odds ratios for microalbuminuria (○) and macroalbuminuria (●) by number of coexisting components of the metabolic syndrome

The prevalence of albuminuria was examined according to the number of coexisting components of the metabolic syndrome as defined above (Fig. 1). The proportion of the sample with zero, one, two and more than two of these conditions was 29.5 %, 27.6 %, 19.8 % and 23.0 % respectively. Among people with only one of these components, 18.8 % were hypertensive, 7.2 % were glucose intolerant (diabetes or IGT), 32.2 % were dyslipidaemic, 6.5 % were insulin resistant and 35.3 % had abdominal obesity. There was a progressive decrease in the proportion of people with normal ACR with increasing number of coexisting components (Fig. 1) and a corresponding increase in the prevalence of microalbuminuria ($\chi^2_{trend} = 64.1$, $p < 0.001$) and macroalbuminuria ($\chi^2_{trend} = 110$, $p < 0.001$). The risk of macroalbuminuria was significantly increased in the presence of one or more components of the metabolic syndrome, as indicated by odds ratios significantly greater than one (Fig. 1B). The risk of microalbuminuria was significantly increased in the presence of two or more components of the metabolic syndrome. Even when no components of the metabolic syndrome were present, there was however, a substantial prevalence of microalbuminuria.

Independent odds ratio (OR; derived from multinomial logistic regression as described above) and population attributable risk per cent (PAR %; see above) for components of the metabolic syndrome were calculated (Table 3). Hypertension was associated with a statistically significant increased risk of albuminuria ($p < 0.001$), with significantly increased OR for microalbuminuria and macroalbuminuria. Diabetes was associated with increased risk of albuminuria independently of other factors ($p < 0.001$), with greater risk of microalbuminuria and macroalbuminuria in diabetic people. Impaired glucose tolerance was not a statistically significant predictor of microalbuminuria or macroalbuminuria and was excluded from the final model. Dyslipidaemia was not independently associated with albuminuria ($p = 0.181$). Obesity defined by BMI criteria was not a significant risk factor and was omitted from the final model. High waist-to-hip ratio was independently associated with albuminuria ($p < 0.001$), with a significantly higher risk of macroalbuminuria in abdominally obese people ($p < 0.001$). The risk of microalbuminuria associated with abdominal obesity was not statistically significant ($p = 0.126$). Overall, insulin resistance did not reach statistical significance as an independent risk factor for albuminuria ($p = 0.073$). Results of multinomial logistic regression analyses remained substantially unchanged after stratification of the survey sample by diabetes or hypertension status. In terms of PAR %, hypertension, diabetes and ab-

Table 3. Independent odds ratio (OR; adjusted for all other factors) and population attributable risk per cent (PAR %) for components of the metabolic syndrome in relation to risk of microalbuminuria and macroalbuminuria

Condition	Microalbuminuria			Macroalbuminuria	
	Prevalence	OR (95 % CI)	PAR %	OR (95 % CI)	PAR %
Hypertension	25.6 %	2.4 (1.6–3.4)	26 (14–38)	6.3 (3.9–10.2)	58 (42–70)
Diabetes	16.9 %	2.0 (1.2–3.3)	15 (4–28)	3.8 (2.1–7.0)	32 (16–50)
Dyslipidaemia	35.6 %	1.1 (0.8–1.6)	4 (< 0–17)	1.6 (1.0–2.6)	17 (< 0–36)
Abdominal obesity	37.4 %	1.3 (0.9–1.9)	11 (< 0–25)	4.7 (2.5–8.8)	58 (35–75)
Insulin resistance[a]		1.8 (1.1–3.0)		1.2 (0.6–2.6)	

OR derived from multinomial logistic regression; PAR % calculated according to reference [28]
[a] Highest quartile of HOMA insulin resistance compared to lowest quartile

dominal obesity were major independent contributors to the prevalence of macroalbuminuria in this population. Hypertension accounted for about one quarter of the subjects with microalbuminuria, diabetes for less than 20 % and other conditions of the metabolic syndrome did not contribute significantly.

Among the central Australian group of people for whom the presence of UTI was determined (prevalence of UTI = 7.1 %), the presence of UTI was not statistically significantly associated with albuminuria and was omitted from the final model. Haematuria at levels indicative of glomerulonephritis (see above) was strongly and independently associated with both microalbuminuria (OR, 95 % CI = 3.3, 1.9 – 5.6) and macroalbuminuria (5.0, 2.4 – 10.6). The inclusion or exclusion of haematuric people from the analysis, however, did not substantially alter the risks associated with other variables (data not shown).

Discussion

Our results indicate that albuminuria among Aboriginal people in Central Australia, the Kimberley and Cape York occurs with a very high prevalence in non-diabetic and diabetic people. Albuminuria clustered strongly with characteristics of the metabolic syndrome: hypertension, glucose intolerance, dyslipidaemia, abdominal obesity (all of which were highly prevalent in this population) and insulin resistance. Moreover, microalbuminuria was highly prevalent even in the absence of any of these conditions. In multivariate analyses, microalbuminuria was independently associated only with hypertension and diabetes: macroalbuminuria showed highly statistically significant, independent associations with hypertension, diabetes and abdominal obesity but no statistically significant association with insulin resistance. Dyslipidaemia and IGT were not independently associated with albuminuria. The data are consistent with the high rates of albuminuria and renal disease in other Aboriginal populations in Australia [1, 2]. Our results are also quantitatively and qualitatively consistent with other population-based studies in the role of blood pressure, diabetes, IGT and glomerulonephritis as independent risk factors for albuminuria [2, 11, 14, 30].

A role for total and abdominal adiposity as a risk factor for albuminuria has been reported inconsistently among European, American and Australian populations [2, 8, 11, 14, 31–34]. In our survey sample, ACR was clearly associated with waist-to-hip ratio but only weakly or not at all with BMI in itself. Prevention of obesity would, however, clearly reduce the risk of albuminuria through concomitant prevention of hypertension, diabetes and other aspects of the metabolic syndrome.

Insulin resistance and hyperinsulinaemia have been linked previously with albuminuria. Fasting hyperinsulinaemia was independently associated with increased ACR in another Aboriginal population [2] and insulin resistance was statistically significantly associated with ACR in the non-diabetic population of the Insulin Resistance Atherosclerosis Study [11]. In other studies, albuminuria in patients with Type II (non-insulin-dependent) diabetes mellitus was associated with greater insulin resistance than in normalbuminuric diabetic patients [12], whereas in hypertensive patients, a positive association between insulin response to oral glucose and albumin excretion rate was observed [35]. In contrast to these studies, no independent association of fasting hyperinsulinaemia with albuminuria was found in a population-based study of European people [14] and the authors concluded that microalbuminuria was "a complication of hypertension and diabetes but not an integral part of the insulin resistance syndrome". In our study, insulin resistance was clearly higher with worsening degree of albuminuria. This trend was, however, largely explained by higher prevalences of diabetes among people with a greater ACR and multivariate analysis showed an independent relation between insulin resistance and albuminuria that was at best weak. Similar negative conclusions were reached in a study of several other indigenous populations [36].

Primary causes of renal disease leading to transplantation or dialysis among Aboriginal patients in the Northern Territory, Western Australia and Queensland from 1993 to 1997 (the period during which the present cross-sectional surveys were carried out) included diabetes (46 %), hypertension (5 %) and glomerulonephritis (29 %) [37]. Apart from the expense, treatment of end-stage renal disease with dialysis or transplantation has severe detrimental social effects on Aboriginal people [38, 39] and prevention of the initiation and progression of albuminuria is clearly a better method of dealing with this issue. Regardless of the exact causal associations between constituent components of the metabolic syndrome and albumin excretion, interventions to prevent obesity, diabetes and hypertension based on improved social conditions, diet and exercise are likely to contribute to the prevention of this major cause of premature morbidity and mortality among Aboriginal people.

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