Quantifying the Excess Risk for Proteinuria, Hypertension and Diabetes in Australian Aborigines: Comparison of profiles in three remote communities in the Northern Territory with those in the AusDiab study.

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Abstract

**Objective:** To estimate the magnitude of excess risk for proteinuria, high blood pressure and diabetes in Australian Aboriginal adults in three remote communities by comparing them with nationwide Australian data.

**Methods:** Adult volunteers from three remote communities in the Northern Territory were screened for proteinuria (dipstick protein ≥1+), high blood pressure (BP ≥ 140/90 or antihypertensive treatment), and diabetes (WHO criteria) between 2000 and mid 2003. Rates for people age 25 to 74 years were compared with those from the nationwide AusDiab study conducted in 1999 and 2000.

**Results:** Compared to AusDiab, rates of these conditions were elevated in all Aboriginal communities, but differed among them. With adjustment for age and sex, rates of proteinuria were elevated 2.5 to 5.3 fold, rates of high blood pressure were elevated 3.1 to 8.1-fold and rates of diabetes were elevated 5.4 to 10-fold (p<0.001 for all). The risk of having any condition ranged from 3.0 to 8.7-fold and the risk of having 2 or more conditions ranged from 5.8 to 14.2-fold. The relative accentuation of risk for hypertension and diabetes was highest among young adults, while that for proteinuria was higher in those age 35 yr and above.

**Discussion:** The data are compatible with the excess morbidity and mortality from cardiovascular disease, diabetes and renal disease in these Aboriginal groups. They reflect the multitude of risk factors operating in these environments. They dictate urgent and systematic intervention to modify outcomes of established disease and to prevent their development. However, the resources required for effective secondary intervention will differ among communities according to the disease burden.

**Keywords**

Australian Aborigines, Hypertension, Diabetes Mellitus, Proteinuria, Chronic Disease
Introduction

Noncommunicable chronic diseases (NCDs) represent one of the major health issues of the 21st century. Increasing rates of hypertension, type 2 diabetes and chronic kidney disease are especially prominent in developing countries and in minority populations undergoing rapid epidemiologic and lifestyle transition. (1) Australian Aborigines are experiencing an accelerated and often brutal transition, and many are now living in deprived and marginalized circumstances, with poor access to health services. Prior to the 1980s, almost no NCDs were recognized, but they are now rife, with average rates of cardiovascular death more than 3 times those of non-Aboriginal Australians, diabetes deaths increased 8-fold and renal failure about 10-fold. (2, 3) Rates are much higher in some remote areas. (4, 5)

Awareness and management of this problem have improved in the last 10-15 years. Screening programs have been conducted in several regions. (6-15) Systematic management of affected people produces good results. McDermott et al showed that structured diabetes care reduces hospitalizations (16), and we showed that systematic treatment for hypertension and renal disease produces marked reductions in nonrenal and renal deaths (17), with dramatic cost-savings in dialysis avoided. We also showed that benefits are quickly lost when treatment falters. (18) Guidelines for chronic disease management have been developed and incorporated into standard care plans for Aboriginal adults in some regions. (19) However, serious deficiencies persist. The structures for Aboriginal primary health care, in which chronic disease programs should be nested, are a chaotic, inscrutable and ever changing mix. They are inadequately resourced, with a serious staffing shortage and average per capita spending estimated at less than one tenth of that for non-Aboriginal Australians. (20) In addition, there is a tacit assumption of a uniform disease burden across Aboriginal Australia, suggesting that one resourcing formula will suit all circumstances.

In this manuscript we describe the rates of proteinuria, high blood pressure and diabetes in adults in three remote Aboriginal communities in one region of Australia, and compared them with a nationally representative survey of participants from the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Our objective is to define the magnitude of the problem, the degree of homogeneity or variation, to speculate on causes and associations and make the case for systematic, needs-based health services planning.
Methods

The Aboriginal study communities (named 1 through to 3), were all from the ‘Top End’ of the Northern Territory, Australia (Figure 1). They all represent aggregations of people from several tribal groups in those regions. These communities have adult (25+ yr) Aboriginal populations ranging from 135 to 512.\(^{(21)}\)

Volunteers were tested in the context of programs to improve the awareness and management of chronic diseases, conducted between 2000 and 2003. Testing was generally done by Aboriginal health workers who had been trained by nurse coordinators. Results were recorded in hard copy and also entered in an electronic database. Testing included a brief history, medication review, anthropomorphic measurements, blood pressure, urine dipstick for protein and assessment of the presence of diabetes. The health profiles have been previously described.\(^{(9, 22)}\)

The AusDiab study was based on a stratified cluster sample of 11,247 consenting adults aged 25 and above, who participated in a nation-wide survey in Australia conducted between 1999 and 2000.\(^{(23)}\) Only 1% of the study participants in the AusDiab were of Aboriginal or Torres Strait Islander origin.

Data on people age 25 to <75 years of age were used to compare the Aboriginal and AusDiab findings. This lower limit was set by the lower age limit in the AusDiab study, and the upper limit was defined by the paucity of older people in the Aboriginal group (among screened Aboriginal people age 25+ years, only 2.5% were aged 75+ yr, compared with 7.2% for the AusDiab group). Proteinuria was defined as protein ≥ 1+ (30 mg/dl) by urine dipstick. High blood pressure was defined at two levels- as a blood pressure ≥ 140/90 mmHg, and as an inclusive definition of BP ≥ 140/90 and/or antihypertensive treatment for a past diagnosis of hypertension. Diabetes was defined by history, existing prescription of hypoglycaemic medication and/or by blood glucose measurement on testing. Aboriginal people not already known to be diabetic were screened by a fasting or random blood glucose measurement - those with values of ≥ 7.0 mmol/l or ≥ 11.1 mmol/l respectively were deemed diabetic. These were confirmed by repeat screening. AusDiab participants not already known to be diabetic were screened with an oral glucose tolerance test, and deemed diabetic if the fasting value was ≥ 7.0 mmol/l or the 2 hour level ≥ 11.1 mmol/l. The prevalence of each of these conditions was calculated. The prevalences of people with at least one condition and with more than one condition were also defined. Unadjusted proportions of people with these conditions were compared, and the odds ratios (95%
confidence intervals) of the Aboriginal groups relative to the AusDiab population were estimated using logistic regression, adjusted for age and sex. Statistical analyses were performed using Stata, version 9.1.\(^{(24)}\)

The Aboriginal data used for analyses in this report were collected in projects approved by the Ethics Committees of the Menzies School of Health Research and the Royal Darwin Hospital, and the University of Queensland. Comparison with AusDiab data was approved by the individual communities and by the Ethics Committee of the University of Queensland, and publication of the findings was approved by the individual communities. The AusDiab study was approved by the Ethics Committee of the International Diabetes Institute, and all participants gave written, informed consent.

**Results**

Data were compared on 10,434 people in the AusDiab study and 814 Aboriginal people in the specified age range. The Aboriginal participants represented approximately 71%, ≥100% and 64% of the age-eligible populations in Communities 1, 2 and 3 respectively. (Participation in Community 2 was greater than the census population of age-eligible adults). Females constituted 55.1% of the AusDiab group and 53.4% of the Aboriginal group (p=0.36). Table 1 shows that the mean age of the Aboriginal participants in each community was younger than the AusDiab population, which was due to higher proportions of Aboriginal people in the younger age group and lower proportions in the higher age groups. However, age was well matched within each age stratum (for age groups 25-<35, 35-<45, 45-<55 and 55-<75 respectively, AusDiab ages (means) were 29.9, 39.8, 49.5 and 63.6, and for Aboriginal were 29.7, 39.8, 49.0 and 63.0). Table 1 also shows a marked gradation of BMIs among the Aboriginal communities, with only those in Community 3 exceeding those of AusDiab.

Figure 2A shows the aggregate unadjusted rates of proteinuria, high blood pressure and diabetes. They were excessive in all the Aboriginal communities relative to AusDiab, but differed among them. The prevalence of proteinuria varied from 15% to 28%, of high blood pressure from 34% to 54%, and of diabetes from 17% to 30%. With adjustment for age and sex, as shown in Figure 2B, these corresponded to odds ratios for proteinuria ranging from 2.5 to 5.3, for high blood pressure from 3.1 to 8.1 and for diabetes, from 5.4 to 10.0. For all conditions the heterogeneity among Aboriginal communities was significant, with p<0.0001. As shown in table 2, the proportions of Aboriginal people with elevated blood pressure at
examination, while still higher than the AusDiab population, were relatively less elevated than the rates of hypertension by the expanded definition, probably reflecting some success in policies to control blood pressure in recent years in the Aboriginal environments.

Figure 3A shows that between 43% and 68% of people in the different Aboriginal communities had at least one of the three conditions, and 17% to 34% had more than one condition. As shown in Figure 3B, these correspond to odds ratios, adjusted for age and sex, varying from 3.0 to 8.7 for any condition and of 5.8 to 14.2 for multiple conditions. In both instances, the apparent heterogeneity among communities was significant (p<0.0001).

Figure 4A shows that the rates of these conditions were strongly correlated with age in the Aboriginal communities. In the AusDiab population, high blood pressure and diabetes also correlated strongly with age, but proteinuria did not. Rates in Aboriginal people were higher than those in the AusDiab population in every age group. Figure 4B shows an elevated risk for proteinuria in all age groups, but the risk accentuation was higher after age 35 yr. The figure shows that the risk accentuation for hypertension was highest in younger Aboriginal adults. Finally it shows a 19.5-fold exacerbation of risk for diabetes among the youngest Aboriginal adults, and although the relative risk fell somewhat with age, it remained very high throughout life.

Figure 5A shows that the probability of having at least one condition rose with age in both populations, but was always greater in the Aboriginal group. By age 35-44 yr, 59% of Aboriginal people had at least one condition and by age 55+ yr, 79% were affected. The probability of having multiple conditions was strikingly higher at all ages among the Aboriginal people, and reached 35% by middle age. Figure 5B shows that the adjusted risk of having any condition was more than four times that of AusDiab among the youngest adults, and peaked at 7.1-fold increase among those age 35-44 yr. The adjusted relative risk for multiple conditions was greatest among the younger adults, with an 18-fold increase.

Discussion

Rates of proteinuria, high blood pressure and diabetes were excessive in each of these Aboriginal communities relative to those in the AusDiab study. The accentuations of risk in Community 1, with a 2.5-fold increase in proteinuria, a 3.1-fold increase in hypertension and a 5.3-fold increase in diabetes, are serious indeed; the 5.3-fold increase in proteinuria, 8.1-fold increase in hypertension and a 10-fold increase in diabetes in Community 3 are truly
striking. The risk of having any condition was increased as much as 9-fold and the risk of having multiple conditions was increased 14-fold and more.

Several factors limit the precision of our findings, although they could not have caused a major distortion of the true relationships. The groups were tested by different teams, measurements of blood pressures might have varied with observers, and, while reported blood pressures were averaged from two or three readings in AusDiab participants, they represent a single reading in the Aboriginal people. The dipsticks for measuring proteinuria were not necessarily the same brand, and urine protein concentration could have been accentuated by the generally hot ambient temperature in the Aboriginal settings. The accentuation of risk for diabetes in Aboriginal people is probably understated, because a glucose tolerance test was not routinely performed on those without a known diagnosis of diabetes. Also, glucose screening might have missed some people with unsuspected IGT/IFG/Diabetes by accepting "normal range" fasting or random glucose screening. This will result in understatement by estimates of the risk exacerbation of the Aboriginal group compared with the AusDiab data. The possible effect of differential participation by age and gender among the communities is discussed below.

Striking as the risk accentuations are, the figures nonetheless markedly understate the real burden of these conditions in the Aboriginal communities. Participants in cross sectional studies are, by definition, survivors to that point: in the Aboriginal context this means they have so far escaped the greatly excessive risk for premature death carried by proteinuria, hypertension and diabetes.\(^{25, 26}\) According to nationwide estimates, about half the males and only a marginally lower proportion of females have already died before the age of 55 years, with the deaths heavily segregated among those with the morbidities under study. This must be contributing, at least in part, to the lower risk ratios in older Aboriginal participants.

The data are compatible with the >20-fold increase in renal failure and the three to six-fold increase in premature deaths in general, and cardiovascular deaths in particular, in these regions and with the nine-fold increase in deaths from ischemic heart disease and 28-fold increase in diabetes deaths in remote Aboriginal Australia more generally.\(^{3,4,5, 32}\) These are crisis-level statistics.

It is challenging to consider the mix and balance of risks factors that allows expression of disease at such levels. It is also important to consider the implications for health services policy and needs-based planning.
The very substantial overlap of conditions, described in more detail elsewhere, and demonstrated in the high rates of people with more than one condition, suggests that they are, in part, elements of a single vascular/renal/metabolic syndrome.\(^{(9, 13, 27-30)}\) Such integration suggests a shared base of risk factors. It also supports integrated rather than disease specific programs for surveillance and containment, and the need for multiple arms of management. With almost 60% of Aboriginal people having at least one morbidity by age 35-44 years, most people will need medical treatment by middle age, and many will need treatment with multiple drugs. Treatment will need to be sustained for many years if they are to aspire to a life span that approximates that of nonAboriginal Australians.

Some of the factors associated with these morbidities are being defined.\(^{(9-15, 27, 28, 31-34)}\) In addition to increasing age, they include higher levels of body fat, which is particularly flagged by higher waist measurements. A role for body fat is suggested here by the fact that the differential rates of morbidities in the Aboriginal groups follow the same pattern as their BMIs. Alone, however, they do not account for most of the excessive rates, for only in Community 3 are the BMIs greater than in the AusDiab population. A more detailed description of body habitus of the Aboriginal participants, its comparison with the AusDiab population, and the associations with the conditions described here are subjects of additional published and proposed reports (Dr. Kondalsamy-Chennakesavan, The University of Queensland).\(^{(9-11)}\) Other demonstrated risk factors include birthweight (inversely), skin infections, noncutaneous infections, inflammation, excessive alcohol use, for renal disease at least, and grand multiparity, while high rates of smoking (up to 80% in men, and up to 50% of women) and psychosocial stress are also probably instrumental.\(^{(9-11, 13, 35-39)}\) All the conditions are probably multideterminant, with the simultaneous or sequential operation of more than one risk factor amplifying effects of the others\(^{(10, 40)}\), a concept already demonstrated, as the “polyfactorial model” for ischemic heart disease.\(^{(41)}\) Such models help explain the massively higher rates of disease in environments with a high density of risk factors. We have also emphasized that the emergence of this epidemic is a consequence, in part, of prior health triumphs. The recent vast reduction in infant mortality since the 1950s, due to better hospital management of sick babies, has allowed cohorts of low birthweight babies to survive to adult life, where, as proposed by Barker and becoming manifest in the Aboriginal setting, they are at increased risk for chronic disease.\(^{(32-34, 42)}\) In addition, reduction in competing mortality, particularly due to fewer infectious deaths, has increased adult longevity and thus the opportunity for chronic disease expression.
The uniformly excessive rates support clinical impressions that all remote communities are afflicted by this epidemic of chronic disease. The variation in rates, however, shows that the magnitude of the situation cannot be generalized at any one particular time. Plausible reasons for the differences include obvious variations in body habitus, differences in diet, alcohol use and rates of smoking, differences in timing of the improved infant survival phenomenon, in the duration and intensity of lifestyle changes, in palpably different degrees of disadvantage at this point in time, and potentially, differences in genetic predisposition. Men constituted a somewhat larger proportion of the total screened population in community 1 than in the other sites (Table 1), and they tended to be somewhat younger. These phenomena were driven by an all-male screening team, and a paucity of males aged 45+ years in this community due to premature deaths, in large part associated with substance abuse and violence. However, the differences in rates of morbidities by community, and particularly the lowest rates in community 1, are very real. They applied for all the three conditions, except arguably for proteinuria, for any morbidity and for multiple morbidities in both sexes, and for all age groups below 55 years, after which small numbers sometimes became limiting.

The situation constitutes an emergency by any standards. With the clinical benefits and cost savings of effective treatment demonstrated, there can be no rational argument against early screening and intervention programs. There are also plenty of avenues for primary prevention, targeting exercise, nutrition, hygiene, smoking and infections, which can be implemented or strengthened, even while working to understand the pathophysiology more precisely. A problem of equal magnitude among nonindigenous Australians, adequately exposed, would excite a vigorous and systematic response.

The variation in rates by community, however, dictates needs-based health services planning. Primary prevention strategies might vary little across communities, but the level of resourcing for programs of screening and treatment will depend on the disease burden. Disease burden might influence the optimal frequency of regular testing throughout life, but will certainly influence the requirements of personnel, reagents, facilities, medicines, tests and documentation needed for proper treatment and intensified surveillance of people with morbidities. In fact, the capacity for a high treatment burden to overwhelm poorly resourced chronic disease intervention programs has already been demonstrated in such settings.

The governments of the Northern Territory and most other states have endorsed the need for good chronic disease surveillance and management, and protocols have been incorporated into standard care guidelines for Aboriginal people. Our observations in
these Outreach communities, and in an ongoing relationship with the Tiwi community, and in the radically different environments of two large and empowered Aboriginal Medical Services in Western Australia, combined with much anecdotal information more broadly, indicate that systematic application of these principles is constrained by limited resources.\(^{(18, 46)}\) Ironically, hospital services and renal replacement therapy offered to Aboriginal people are of good quality and essentially uncapped, with the latter now posing a fiscal crisis for health care budgets.\(^{(43)}\) The problem requires a rethinking of Aboriginal health services, adequate resourcing, strategies to combat the obstacles and hardships associated with remote locations and placements, and accountability at every level of the system.
Acknowledgements.

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### Table 1. Demographics of participants in this study

<table>
<thead>
<tr>
<th>Communities</th>
<th>n</th>
<th>Females</th>
<th>Age in years, Mean (SD)</th>
<th>BMI, Kg/m² Mean(SD)</th>
<th>Obese by BMI %</th>
</tr>
</thead>
<tbody>
<tr>
<td>AusDiab</td>
<td>10,434</td>
<td>55.1</td>
<td>49.4 (12.6)</td>
<td>27.0 (5.0)</td>
<td>22.9</td>
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<tr>
<td>Community 1</td>
<td>331</td>
<td>47.4</td>
<td>40.6 (12.0)</td>
<td>22.1 (4.7)</td>
<td>6.3</td>
</tr>
<tr>
<td>Community 2</td>
<td>157</td>
<td>56.7</td>
<td>41.9 (12.0)</td>
<td>25.7 (5.9)</td>
<td>23.4</td>
</tr>
<tr>
<td>Community 3</td>
<td>326</td>
<td>58.0</td>
<td>42.7 (13.2)</td>
<td>28.5 (6.2)</td>
<td>38.6</td>
</tr>
</tbody>
</table>
Table 2. Proportion of participants with high blood pressure using two definitions, and odds ratios relative to AusDiab, adjusted for age and sex

<table>
<thead>
<tr>
<th></th>
<th>BP ≥ 140/90</th>
<th>OR (95% CI)</th>
<th>BP ≥ 140/90 and or on medication</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AusDiab</td>
<td>23.0 %</td>
<td>Referent</td>
<td>29.0 %</td>
<td>Referent</td>
</tr>
<tr>
<td>Comm 1</td>
<td>24.6 %</td>
<td>2.3 (1.7-3.1)</td>
<td>33.7 %</td>
<td>3.1 (2.4-4.1)</td>
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<tr>
<td>Comm 2</td>
<td>32.7 %</td>
<td>3.6 (2.5-5.2)</td>
<td>47.1 %</td>
<td>6.0 (4.2-8.5)</td>
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<tr>
<td>Comm 3</td>
<td>35.4 %</td>
<td>3.7 (2.8-4.8)</td>
<td>54.4 %</td>
<td>8.1 (6.2-10.6)</td>
</tr>
</tbody>
</table>
References:


Figure 1. Aboriginal communities in the ‘Top End’ of the Northern Territory, Australia.

Estimated resident Indigenous population based on 2001 census (Aged 25-<75)

- Community 1: 468
- Community 2: 135
- Community 3: 512
Figure 2A. Prevalences of proteinuria, hypertension and diabetes, by community

Definitions:
Proteinuria: Urine dipstick ≥ 1+
Hypertension: BP ≥ 140/90 and or on antihypertensive medication
Diabetes: Fasting plasma glucose ≥ 7.0 or random or 2-hr plasma glucose ≥ 11.1 mmol/l or on hypoglycaemic medication.
Figure 2B. Odds ratios for proteinuria, hypertension and diabetes relative to AusDiab, by community (adjusted for age and sex)

Definitions:
Proteinuria: Urine dipstick ≥ 1+
Hypertension: BP ≥140/90 and or on antihypertensive medication
Diabetes: Fasting plasma glucose ≥ 7.0 or random or 2-hr plasma glucose ≥ 11.1 mmol/l or on hypoglycaemic medication.
Figure 3A. Prevalence of any and multiple conditions by community
Figure 3B. Odds ratio for any and multiple conditions relative to AusDiab, by community (adjusted for age and sex)
Figure 4A. Prevalences of proteinuria, hypertension and diabetes, by age group and Aboriginal status.

Definitions:
Proteinuria: Urine dipstick $\geq 1+$
Hypertension: BP $\geq 140/90$ and or on antihypertensive medication
Diabetes: Fasting plasma glucose $\geq 7.0$ or random or 2-hr plasma glucose $\geq 11.1$ mmol/l or on hypoglycaemic medication.
Figure 4B. Odds ratios for proteinuria, hypertension and diabetes among Aboriginals relative to AusDiab, by age group (adjusted for age and sex)

Definitions:
Proteinuria: Urine dipstick $\geq 1+$
Hypertension: BP $\geq 140/90$ or on antihypertensive medication
Diabetes: Fasting plasma glucose $\geq 7.0$ or random or 2-hr plasma glucose $\geq 11.1$ mmol/l or on hypoglycaemic medication.
Figure 5A. Prevalence of any and multiple conditions by age group and Aboriginal status.
Figure 5B. Odds ratios for any and multiple conditions relative to AusDiab, by age group (adjusted for age and sex).