The multidimensional nature of renal disease: Rates and associations of albuminuria in an Australian Aboriginal community

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The multidimensional nature of renal disease: Rates and associations of albuminuria in an Australian Aboriginal community.

Background. An epidemic of end-stage renal disease (ESRD) is accompanying the rising rates of hypertension, type 2 diabetes and cardiovascular disease among Aborigines in the Northern Territory of Australia. Incidence rates are now 21 times those of nonAboriginal Australians and are doubling every four years. We describe the rates and associations of renal disease in one remote community, which has a current ESRD incidence of 2700 per million, and cardiovascular mortality among the highest in Australia.

Methods. Between 1992 and 1995 a community-wide screening program was conducted, in which the urinary albumin/creatinine ratio (ACR) was used as the chief renal disease marker. More than 90% of the population ages five and older participated.

Results. Albuminuria was evident in early childhood and increased dramatically with age; 26% of adults had microalbuminuria and 24% had overt albuminuria. All renal failure developed out of a background of overt albuminuria. ACR was significantly correlated with the presence of scabies at screening, with a history of poststreptococcal glomerulonephritis, which is epidemic and endemic in the community, with increasing body wt, blood pressure, glucose, insulin and lipid levels, and with evidence of heavy drinking. ACR was also significantly and inversely correlated with birth weight. As a result of its association with deteriorating hemodynamic and metabolic profiles, increasing ACR was also correlated with increasing cardiovascular risk score. Direct observations showed, and multivariate models predicted, progressive amplification of ACR when multiple risk factors were present simultaneously. Albuminuria also clustered in families.

Conclusion. Renal disease in this population is multifactorial, with risk factors related to whole-of-life nutrition, metabolic and hemodynamic profiles, infections, health behaviors, and possibly a family predisposition. Its relationship to low birth weight, and its associations with deteriorating metabolic and hemodynamic profiles, suggest that renal disease is, in part, a component of Syndrome X, which explains the simultaneous increase in metabolic, cardiovascular and renal disease in Aboriginal people. The family clustering might have both environmental and genetic causes, and is under further investigation. Most of the identified risk factors arise out of poverty, disadvantage and accelerated lifestyle change, and the current epidemic can be explained by the confluence of many risk factors in the last few decades. The introduction of effective and sustained programs to address social, economic and educational inequities in all Aboriginal communities, and of screening and renal- and cardiovascular-protective treatment programs for those already afflicted are matters of great urgency.

The health of Aboriginal people in the Northern Territory (NT) of Australia is among the worst in the world, with persisting low birth weights and serious infant malnutrition, repeated and persistent infections, and changes in diet, exercise and health behaviors related to “Westernization.” Body weight, hypertension, type 2 diabetes, cardiovascular disease and renal disease are increasing rapidly. Between 1978 and 1991 standardized mortality rates (SMRs) for Aboriginal adults from cardiovascular disease, diabetes and renal disease were respectively about 5, 12 and 20 times those of non-Aboriginal people. Over the 1980s, SMRs for Aboriginal males increased, while deaths from ischemic heart disease and diabetes in females actually tripled. The aggregate incidence of treated end-stage renal disease (ESRD) among Aboriginal people in the “Top End” of the Northern Territory rose from 167 per million (pm) between 1985 to 1988, to 925 pm in 1996, and is doubling every four years [1–5]. Rates vary among different groups, but all show the same trends, and the incidence currently exceeds 1000 pm in at least five different regions [1–5].

We investigated the rates and associations of the renal disease underlying this epidemic of renal failure in one remote Aboriginal community.

METHODS

Between 1992 and 1995 we screened volunteers from an isolated Northern Territory (NT) Aboriginal island community. These people have a current ESRD incidence rate.
of 2700 per million and share with another NT Aboriginal group the highest adult mortality rates in Australia [1, 2, 6]. The 1991 census counted 957 people in this community, with 50% less than 20 years of age [7]. In the screen, we measured weight and height in children, examined skin for sores and scabies, and, for people aged 10+ years, measured blood pressure and serum creatinine. The adult screen also included questions about smoking and drinking, measurement of fasting glucose, insulin (total immunoreactive insulin, mU/liter; Pharmacia Assay Kit, Ryde, NSW, Australia), lipids and serum gamma glutamyl transferase (γGT) levels. Glucose tolerance, defined by WHO standards [8], was evaluated by a 75 g oral glucose challenge in persons not known to be diabetic. In all participants, a random urine specimen, remote from menstrual periods in females, was tested by dipstick, avoiding samples positive for leukocytes or nitrates until urinary infections or sexually transmitted diseases had been excluded. The albumin/creatinine ratio (ACR) on a single random urine specimen was used as the renal disease marker, with albumin levels measured by the Beckman immunoassay (Beckman Instruments, Brea, CA, USA), and adjusted for simultaneous creatinine concentrations to control for variations in urinary concentration and dilution [9–15]. The following ACR categories in g/mol, (and their US equivalents in mg/gm) were employed: (a) ACR1, < 1.1, (95th percentile by lab standards, or <12 mg/g) normal; (b) ACR2, 1.1 to 3.3 (12 to 29 mg/g), suspicious; (c) ACR3, 3.4 to 33, (30 to 299 mg/g) microalbuminuria, and (d) ACR4, ≥ 34 (≥300 mg/g), overt albuminuria [16–19]. Results of women screened during pregnancy or within six months of delivery were excluded from the analysis. The 18 people living away from the community for chronic dialysis treatment could not be included in the survey.

Birth weights of participants, recorded in clinic charts sporadically after 1958, and consistently by the mid 1960s, were noted. Records were also reviewed for a history of poststreptococcal glomerulonephritis (PSGN) for persons under 30 years at screening; records on older persons were less complete. A positive PSGN history ultimately relied on a diagnosis by the health care provider at the time, but always involved a combination of hematuria and proteinuria with recent skin sores, often with edema and increased blood pressures; anti-DNAse titers were high in most, and complement levels, when tested, were low.

RESULTS

Results were obtained on 382 children aged 5 to 17 years and on 487 adults (18+ years old), representing 100% and 89% of the respective census populations. Their general health was poor. The average children's weight was at the 25th percentile, 67% had skin sores, and 33% had scabies; 80% had chronic middle ear disease, many had mucopurulent nasal discharge and 10% had a productive cough. As reported elsewhere [23], 75% of adults smoked, and 25% of women and 85% of men drank, with most males drinking to excess. Twenty percent of adults had skin sores and 15% had scabies; 33% were overweight (BMI >25 kg/m²), 24% had hypertension (SBP ≥140 or DBP ≥90), 19% had diabetes and 12% had impaired glucose tolerance, and 26% had a high γGT (>40 U/liter for women, >60 U/liter for men), which segregated strongly (4-fold) among drinkers. Fungal skin infections, otitis media and externa, productive cough, chronic obstructive lung disease, gingivitis and loss of teeth were all very common.

Episodes of PSGN had been recorded for 120 of 423 (28.4%) people less than 30 years old. Eighty-six percent of PSGN episodes had occurred before 10 years of age, and 108 episodes (90%) had occurred more than four years before screening. Seventy-five of these episodes occurred during four epidemics, and 45 cases were sporadic.

Birth weights were available for 618 people who were 5 to 37 years old at screening. Mean birth weight was 2.795 ± 0.48 kg; it rose from 2.64 ± 0.49 kg for those born in 1955 to 1964, to 2.94 ± 0.48 kg for those born in 1985 to 1988. Overall 27.6% had been low birth weight babies (<2.5 kg). Gestational age could not be accurately estimated for most;
however, as is in other developing societies, most underweight Aboriginal babies in the NT, and 70% from a recent study, are “small for dates” [24–26].

Table 1 shows the dramatic increase in ACR with increasing age. More than 20% of the youngest children had elevated ACR, and half the adults, and 84% of those of 50+ years had either micro- or overt albuminuria. ACRs were slightly higher in females up to age 30 years, and higher in males after 30 years of age.

Figure 1 shows that hematuria, detected in 25.5% of participants overall, was more common in those with micro- or overt albuminuria, but was also present in a substantial proportion of persons with normal ACRs. Rates of hypertension rose with increasing ACR category. Finally, people with elevated serum creatinine levels were segregated powerfully in the highest ACR category.

As recognized previously [27], some families are at special risk for renal disease and albuminuria. Figure 2
Table 2. Clinical features that change with increasing albumin/creatinine ratio (ACR) category

<table>
<thead>
<tr>
<th>Children 5–17 years old</th>
<th>ACR1</th>
<th>ACR2+</th>
</tr>
</thead>
<tbody>
<tr>
<td>With scabies (of 298 person)</td>
<td>26%</td>
<td>39%</td>
</tr>
<tr>
<td>Birth weight, kg (of 320)</td>
<td>2.91 ± 0.51</td>
<td>2.73 ± 0.49</td>
</tr>
<tr>
<td>Birth weight &lt;2.5 kg</td>
<td>21%</td>
<td>28%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Persons 5–29 years old</th>
<th>ACR1</th>
<th>ACR2</th>
<th>ACR3</th>
<th>ACR4</th>
</tr>
</thead>
<tbody>
<tr>
<td>With a history of PSGN (of 423 persons)</td>
<td>25%</td>
<td>20%</td>
<td>58%</td>
<td>54%</td>
</tr>
<tr>
<td>Adults 18+ years old</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With scabies (of 373 persons)</td>
<td>7%</td>
<td>14%</td>
<td>12%</td>
<td>31%</td>
</tr>
<tr>
<td>Birth weight, kg persons &lt;38 years (of 250)</td>
<td>2.71 ± 0.47</td>
<td>2.75 ± 0.48</td>
<td>2.73 ± 0.48</td>
<td>2.54 ± 0.46</td>
</tr>
<tr>
<td>Birth weight &lt;2.5 kg, persons &lt;38 years</td>
<td>31%</td>
<td>34%</td>
<td>31%</td>
<td>52%</td>
</tr>
<tr>
<td>Weight, kg (of 488)</td>
<td>60 ± 14</td>
<td>63 ± 14</td>
<td>68 ± 15</td>
<td>71 ± 18</td>
</tr>
<tr>
<td>Waist, cm (of 486)</td>
<td>82 ± 12</td>
<td>87 ± 13</td>
<td>91 ± 13</td>
<td>97 ± 13</td>
</tr>
<tr>
<td>Hips, cm (of 486)</td>
<td>87 ± 13</td>
<td>90 ± 13</td>
<td>95 ± 13</td>
<td>98 ± 14</td>
</tr>
<tr>
<td>BMI, kg/m² (of 488)</td>
<td>22 ± 5</td>
<td>23 ± 5</td>
<td>24 ± 5</td>
<td>26 ± 5</td>
</tr>
<tr>
<td>Systolic BP, mm Hg (of 484)</td>
<td>117 ± 15</td>
<td>118 ± 18</td>
<td>125 ± 16</td>
<td>131 ± 20</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg (of 484)</td>
<td>69 ± 12</td>
<td>73 ± 12</td>
<td>76 ± 13</td>
<td>83 ± 16</td>
</tr>
<tr>
<td>Fasting triglycerides, mmol/liter (of 407)</td>
<td>1.7 ± 1.0</td>
<td>1.8 ± 1.4</td>
<td>2.1 ± 1.4</td>
<td>3.2 ± 3.3</td>
</tr>
<tr>
<td>Fasting LDL cholesterol, mmol/liter (of 409)</td>
<td>3.2 ± 0.9</td>
<td>3.6 ± 1.2</td>
<td>3.6 ± 1.1</td>
<td>4.3 ± 1.3</td>
</tr>
<tr>
<td>Gamma glutamyl transferase, U/liter (of 468)</td>
<td>35 ± 27</td>
<td>43 ± 39</td>
<td>54 ± 50</td>
<td>60 ± 63</td>
</tr>
<tr>
<td>Fasting insulin, μU/liter (of 379)</td>
<td>6.2 (5.1–7.0)</td>
<td>6.7 (5.5–8.2)</td>
<td>8.6 (7.3–9.8)</td>
<td>9.4 (7.7–10.7)</td>
</tr>
<tr>
<td>Fasting glucose, mmol/liter (of 428)</td>
<td>4.5 ± 0.7</td>
<td>4.8 ± 1.2</td>
<td>5.5 ± 2.5</td>
<td>6.3 ± 3.1</td>
</tr>
<tr>
<td>2 hour insulin, μU/liter (of 336)*</td>
<td>26 (22–31)</td>
<td>28 (24–37)</td>
<td>42 (31–50)</td>
<td>44 (34–55)</td>
</tr>
<tr>
<td>2 hour glucose, mmol/liter (of 358)*</td>
<td>5.8 ± 1.5</td>
<td>6.2 ± 2.1</td>
<td>7.1 ± 3.5</td>
<td>7.7 ± 3.6</td>
</tr>
<tr>
<td>Diabetes (of 404)</td>
<td>4%</td>
<td>9%</td>
<td>17%</td>
<td>43%</td>
</tr>
<tr>
<td>Cardiovascular risk score ≥3 (of 402)</td>
<td>10%</td>
<td>13%</td>
<td>30%</td>
<td>52%</td>
</tr>
</tbody>
</table>

Data are mean ± sd, or median (95% CI), or percentages.

*Excludes known diabetics

shows one (of many) such families. However, every community member had a first or second degree relative with some degree of renal disease. ACR also correlated significantly with several factors evaluated in the health screen. In children of 5 to 17 years ACR correlated significantly with scabies at time of screening, with a history of PSGN, and inversely with birth weight. In adults (18+ years), ACR was significantly correlated with the presence of scabies at screening, and with a history of PSGN. It was also correlated with weight, BMI, waist and hip measurements, with increasing blood pressure, with fasting levels of cholesterol, triglycerides, glucose, insulin and γGT, and with rates of diabetes. ACR in adults was also correlated inversely and significantly with birth weight. There was no increase in albuminuria among smokers. Table 2 shows how these characteristics changed with increasing ACR category. As a result of its correlation with deteriorating blood pressure and metabolic profiles, increasing ACR category was also correlated with increasing cardiovascular risk score.

Table 3 shows the estimated strength of some of these associations. In children the likelihood of albuminuria was almost doubled in the presence of scabies; it was also increased in low birth weight children, but the association did not achieve significance. A PSGN history was associated with a fourfold increase in the likelihood of microalbuminuria in persons of 10 to 29 years. In adults, the risk enhancement associated with all the cited factors was strong, and weight, hypertension, high cholesterol levels, high triglyceride levels, scabies, and diabetes, retained independent significance in the final model.

Figures 3 to 7 demonstrate some of these correlations. Figure 3 shows the increased frequency of overt albuminuria in adults of low birth weight; this effect is described in more detail elsewhere [28]. Figure 4 shows the pronounced effect of a history of PSGN on microalbuminuria. Figure 5 shows the higher rates of albuminuria in adults with scabies present at time of screening. Figure 6 shows the correlation of hypertriglyceridemia with albuminuria. This correlation was observed in drinkers and nondrinkers, and in persons with normal glucose tolerance as well as the aggregate group. Figure 7 shows the marked increase in overt albuminuria at every age in persons with diabetes.

Figure 8 to 10 illustrate the successive amplification of albuminuria in the presence of more than one risk factor. Figure 8 demonstrates the combined effects of BMI and birth weight in adults. Those with higher BMIs (above the adult median) had higher rates of albuminuria than those of lower BMIs, and low birth weight amplified risk in both categories. Overt albuminuria was least frequent in persons of normal birth weights and lower BMIs, and most common in low birth weight persons of higher BMIs. Figure 9 shows
the additive effects of higher BMIs and scabies on albuminuria in adults. Finally, Figure 10 shows the predicted amplification of albuminuria produced by the simultaneous operation of risk factors that retained significance in the final adult multivariate model.

### DISCUSSION

The extraordinary prevalence of albuminuria in this community is compatible with their high rates of serious renal disease and renal failure. Nonetheless it underestimates the full impact of renal disease on the community, due to a survival bias against persons with albuminuria and inability to include dialysis patients with terminal renal disease in the study. The presence of albuminuria, although subtle, in the youngest children, and its increase with age, provide strong circumstantial evidence for the progression of renal disease from early in life. Hematuria, however, is probably of nonrenal as well as renal origin, and in the absence of significant albuminuria is unlikely to mark serious renal disease [29, 30].

Albuminuria is associated with skin infections and their sequelae, with low birth weight, with increasing adolescent and adult weight, increasing blood pressure, dyslipidemia, insulin resistance and glycemia, of which diabetes constitutes only a variable and advanced manifestation, and probably with excessive drinking. Thus, renal disease in this community, and in individuals within the community, is multidimensional. The extraordinary rates of disease are potentially explained by the multitude of risk factors, their high prevalence, and the amplification of renal damage through their interaction. A confluence of crucial events in the last five decades has probably produced the current epidemic, such as abandonment of hunter-gatherer/no-madic ways, settlement in crowded, substandard and dirty houses, poor hygiene, increases in body fat due to a Westernized diet of poor quality and lack of exercise, abuse of alcohol (legalized in this community in 1967), and, as outlined below, improved survival of low birth weight infants. Renal disease in other Aboriginal people probably has similar causes [14, 13]. The pattern and basis of family clustering is still to be illuminated; it might be based on environmental and/or genetic factors, which are matters for further analysis. However, with albuminuria present in most adults in the community, and in nearly all those who

### Table 3. Enhancement of risk for elevated albumin/creatinine ratio (ACR)

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Odds ratio (95% CI) for ACR 2+ vs. ACR</th>
<th>Adjusted for age and sex</th>
<th>Adjusted for all confounders a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children 5–17 years</td>
<td>Birth weight, per kg reduction 1.9 (1.2–3.2)</td>
<td>1.8 (1.1–3.1)</td>
<td>1.7 (0.9–2.8)</td>
</tr>
<tr>
<td></td>
<td>Scabies 1.7 (0.9–2.8)</td>
<td>1.8 (1.1–3.1)</td>
<td>1.6 (0.9–2.5)</td>
</tr>
<tr>
<td>Adults 18–29 years</td>
<td>PSGN history 5.5 (2.4–12.6)</td>
<td>4.5 (1.9–10.5)</td>
<td>4.8 (2.0–11.5)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Odds ratio (95% CI) for ACR 3+ vs. lower ACR</th>
<th>Adjusted for age and sex</th>
<th>Adjusted for all confounders b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children 5–17 years</td>
<td>PSGN history 3.3 (1.8–9.2)</td>
<td>3.5 (1.1–11.5)</td>
<td>3.1 (1.0–10.4)</td>
</tr>
<tr>
<td>Adults 18–37 years</td>
<td>Birth weight, per kg reduction 1.8 (0.5–4.0)</td>
<td>1.6 (0.8–3.6)</td>
<td>3.0 (1.2–7.2)</td>
</tr>
<tr>
<td></td>
<td>Scabies 3.2 (1.8–5.8)</td>
<td>2.8 (1.5–4.4)</td>
<td>2.2 (1.1–4.6)</td>
</tr>
<tr>
<td></td>
<td>BMI &gt;25 kg/m² 2.7 (1.7–4.1)</td>
<td>2.8 (1.7–4.4)</td>
<td>1.8 (1.0–3.0)</td>
</tr>
<tr>
<td></td>
<td>SBP ≥ 140 or DBP ≥ 90 mm Hg 5.2 (3.2–8.2)</td>
<td>4.7 (2.8–8.0)</td>
<td>3.5 (1.9–6.2)</td>
</tr>
<tr>
<td></td>
<td>Cholesterol &gt;5.5 mmol/liter 3.6 (2.1–6.0)</td>
<td>3.1 (1.8–5.5)</td>
<td>2.2 (1.2–4.2)</td>
</tr>
<tr>
<td></td>
<td>Triglycerides &gt;1.8 mmol/liter 3.5 (2.2–5.7)</td>
<td>2.7 (1.7–4.5)</td>
<td>1.7 (1.0–3.0)</td>
</tr>
<tr>
<td></td>
<td>Diabetes 6.0 (3.5–10.4)</td>
<td>3.8 (2.1–6.9)</td>
<td>2.1 (1.1–4.0)</td>
</tr>
<tr>
<td></td>
<td>Fasting insulin &gt;10.1 μU/liter 1.8 (1.1–3.0)</td>
<td>1.7 (1.0–3.0)</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Fasting glucose &gt;5.4 mmol/liter 4.5 (2.6–7.7)</td>
<td>2.8 (1.5–5.0)</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>High gamma GT d 2.2 (1.4–3.5)</td>
<td>1.8 (1.1–3.0)</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Impaired glucose tolerance 2.5 (1.3–5.0)</td>
<td>1.7 (0.8–3.5)</td>
<td>—</td>
</tr>
</tbody>
</table>

Abbreviations are in the Appendix.

a Adjusted for age, sex and weight for age
b Adjusted for age, sex, weight, birth weight, and, in adults, blood pressure.
c Adjusted for age, sex, weight, blood pressure for birth weight cohort, and adjusted for age, sex, and factors that retained significance in the final model in the aggregate adult population
d >40 μ/liter for women, >60 U/liter for men
survive to middle age, any family predisposition is clearly a matter of degree.

Decreasing birth weight correlates with albuminuria from childhood, and its link with overt albuminuria by early adult life confirms its ultimate relationship to renal failure [31]. Thus, intrauterine malnutrition and/or other causes of low birth weight confer susceptibility to progressive renal disease. This complements the relationship of low birth weight to cardiovascular disease, hypertension, type 2 diabetes and dyslipidemia, collectively termed Syndrome X, reported in other populations [32, 33]. The predisposition to renal disease might be mediated through reduced
nephron endowment related to intrauterine malnutrition [34, 35], which accelerates renal obsolescence that accompanies ageing and the multiple renal insults encountered throughout postnatal life. In our young adults we could find no association between low birth weight and increased blood pressure, impaired glucose tolerance or diabetes to explain its relationship to renal disease indirectly. Ironically, the current chronic disease epidemic is probably the legacy, in part, of the dramatic fall in infant mortality between the late 1950s and late 1970s (from perhaps 140 to 160/1000 to 30/1000) due to better hospital management of sick babies [36]. With most of the prior excess mortality and subsequent retrieval among low birth weight infants, a large cohort of persons has now survived to adult life at high risk.
for renal and other “lifestyle” diseases. The gradual increase in birth weights in this community that continues to the present is grounds for some optimism.

The correlations of albuminuria with the features of Syndrome X, and their shared links with low birth weight suggest that renal disease is, at least in part, a component of that syndrome. This explains the simultaneous rise in cardiovascular disease and renal disease in Aboriginal people, and the link between albuminuria and cardiovascular disease documented in many populations [37–43].

The extraordinary rates of PSGN in this community are compatible with the high rates of skin disease and heavy colonization with group A streptococci with constantly changing sero-types [44, 45]. A (mostly remote) history of PSGN is, in turn, associated with albuminuria. Other reports of subtle evidence of ongoing disease in persons remote from their PSGN episodes support this association [46–48]. High rates of albuminuria have also been noted in persons with scabies and skin sores in other populations [49]; these lesions might mark persons likely to develop PSGN, but, like other persistent infections, they might also mediate renal injury more directly through inflammatory mediators and growth factors [50–52].

Renal biopsies in this community (N = 88) show the usual spectrum of changes, and incidentally with features of diabetic nephropathy in only 28% of diabetics. The single consistent finding, in isolation, or in conjunction with other changes, is marked glomerular enlargement, with variable degrees of segmental and global sclerosis [53–55]. Trophic effects of Syndrome X, chronic infection, and compensatory hypertrophy due to reduced nephron endowment might contribute to such glomerulomegaly, which has also been noted in other Australian Aborigines, US Blacks, American Indians and Hispanic people, all at high risk for renal disease [55–57].

Current renal disease classifications do not accommodate a multidimensional perspective. More practical systems might incorporate epidemiologic observations, acknowledge the limitations of morphology in defining etiology, recognize the operation and interaction of pathophysiological factors over a continuum, and further explore relationships between renal disease and other common conditions.

All the environmental and clinical risk factors identified in this study are features of poverty, disadvantage, and accelerated lifestyle change [58], and all are amenable to modification and prevention. The introduction of coherent, effective and sustained programs into all Aboriginal communities to address these basic issues is of immediate urgency. We must also implement systematic screening and treatment programs to identify people with early renal disease and its risk factors, and prevent or arrest its progression to renal insufficiency [59]. Morbidity and mortality from diabetes and cardiovascular disease will thereby be reduced as well.

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APPENDIX

Abbreviations used in this article are: ACR, albumin/creatinine ratio; BMI, body mass index; CI, confidence intervals; DBP, diastolic blood pressure; ESRD, end-stage renal disease; γGT, gamma glutamyl transferase; HDL, high density lipoprotein; NT, Northern Territory; PSGN, poststreptococcal glomerulonephritis; SBP, systolic blood pressure.

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