The relationships between continuous variables were initially studied by the use of Pearson's correlation analysis and the Kendall-Tau non-parametric test. Chi squared analysis was used for categorical variables.

Regression models (linear and logistic) were built by putting all variables thought to be related to the dependent variable into the model and then removing the least significant variables one at a time. If there was a change in the coefficient of another variable of greater than 10% this was taken as an indicator of possible confounding and the removed variable was reinstated in the model. The final model was checked for collinearity using a condition number greater than 30 in linear regression models and greater than 20 in logistic regression models to indicate collinearity (SPIDA). For linear regression models, predicted values were plotted against residuals to look for evidence of outliers or lack of fit.

The nominal value of p<0.05 was taken as a measure of statistical significance, while p<0.1 was termed marginal statistical significance.

Personal involvement.

I participated in the 1996 children's examinations, taking bloods, measuring heights and weights, blood pressures and explaining, checking and processing urine specimens. I also reviewed some of the medical records. I performed the statistical analysis under supervision.

I presented my findings at a clinic feedback session and a local health board meeting. This is discussed in the Community feedback section.
RESULTS.

Demographics.

The data was gathered from 621 screened people, for whom birth weights and early growth parameters were available. The years of birth from 1955-90, and proportions screened are shown compared with the 1990 census (figure 3). Records of weight at around six months were available for 498 people and weight at around 12 months for 532. Of these 59% were men with 363 men and 258 women. The poor attendance of the women was likely to be due to child caring, home and work duties.

Of this population, 395 were examined as children (less than eighteen years at screening). They were born between 1975-91. In this population there was also a majority of males, with 226 in comparison with 169 females. This might be due to a lower priority placed on school attendance for girls, who stay home for childcare duties or are pregnant.

Figure 3. Proportions in the Study

![Bar graph showing proportions in the study across different years of birth and comparison with census data.]
Birthweight, Infant and Childhood Growth.

Birth Weights.

Mean birthweights in the study community were very low in the 1960s but have improved over time, from 2.67 kg in 1956-70 to 2.93kg in 1981-90 (p=0.01) (Figure 4). These are still well below the WHO mean (Table 3). The proportion of babies who are low birthweight has decreased, from 38% in 1956-70 to 19% in 1981-90 (chi=20.09, p=0), but remains unacceptably high. In 1986-90 15% of babies were low birth weight (<2.5kg) and 5% of births were very low birth weight (<2kg).

Girls had lower mean birth weights and higher proportions of low birth weight, but had higher mean z scores than boys (Table 3). This demonstrates that while low birth weight is an arbitrary definition (<2.5kg), z scores allow for the expected sex differences in weights. The odds ratio for low birthweight in girls compared with boys was 1.50 (C.I. 1.04-2.16).

Birthweight predicted WAZ_6, WAZ_{12} and WAZ_4 (at screening) (table 2). This was confirmed in linear regression after controlling for sex and year of birth (Appendix).

<table>
<thead>
<tr>
<th>Bwt.</th>
<th>WAZ_6</th>
<th>WAZ_{12}</th>
<th>WAZ_4*</th>
<th>HAZ_4*</th>
<th>W_{std}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bwt.</td>
<td>1</td>
<td>0.46*</td>
<td>0.22*</td>
<td>0.31*</td>
<td>0.16*</td>
</tr>
<tr>
<td>WAZ_6</td>
<td>1</td>
<td>0.56*</td>
<td>0.26*</td>
<td>0.36*</td>
<td>0.18*</td>
</tr>
<tr>
<td>WAZ_{12}</td>
<td>1</td>
<td>0.42*</td>
<td>0.40*</td>
<td>0.23*</td>
<td></td>
</tr>
<tr>
<td>WAZ_{4}*</td>
<td>1</td>
<td>0.58*</td>
<td>0.57*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HAZ_{4}*</td>
<td>1</td>
<td>1</td>
<td></td>
<td>0.27*</td>
<td></td>
</tr>
<tr>
<td>W_{std}</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
</tbody>
</table>

Ad=in adulthood s=at time of screening in childhood. Ns=not significant. *p<0.05.
Table 3 Birth Weight & Infant weight by Year of Birth (Yob) and Sex

<table>
<thead>
<tr>
<th>Yob</th>
<th>Sex</th>
<th>n=</th>
<th>mean</th>
<th>%LBW</th>
<th>WAZ0</th>
<th>n=</th>
<th>mean</th>
<th>%&lt;-2sd</th>
<th>n=</th>
<th>mean</th>
<th>%&lt;-2sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>1956-70</td>
<td>M</td>
<td>125</td>
<td>2.67</td>
<td>34</td>
<td>-1.42</td>
<td>111</td>
<td>-0.52</td>
<td>5.4</td>
<td>121</td>
<td>-1.44</td>
<td>29.7</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>81</td>
<td>2.67</td>
<td>44</td>
<td>-1.15</td>
<td>74</td>
<td>-0.36</td>
<td>6.8</td>
<td>78</td>
<td>-1.47</td>
<td>20</td>
</tr>
<tr>
<td>1971-80</td>
<td>M</td>
<td>118</td>
<td>2.8</td>
<td>30</td>
<td>-1.18</td>
<td>85</td>
<td>-0.8</td>
<td>11.7</td>
<td>92</td>
<td>-1.7</td>
<td>43.4</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>82</td>
<td>2.75</td>
<td>22</td>
<td>-0.93</td>
<td>46</td>
<td>-0.56</td>
<td>13</td>
<td>60</td>
<td>-1.6</td>
<td>32</td>
</tr>
<tr>
<td>1981-90</td>
<td>M</td>
<td>112</td>
<td>2.95</td>
<td>16</td>
<td>-0.8</td>
<td>104</td>
<td>-0.73</td>
<td>12.5</td>
<td>106</td>
<td>-1.75</td>
<td>45.2</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>94</td>
<td>2.9</td>
<td>22</td>
<td>-0.63</td>
<td>85</td>
<td>-0.59</td>
<td>7</td>
<td>85</td>
<td>-1.55</td>
<td>35</td>
</tr>
<tr>
<td>Aggregate</td>
<td>M</td>
<td>355</td>
<td>2.81</td>
<td>25</td>
<td>-1.14</td>
<td>300</td>
<td>-0.68</td>
<td>10</td>
<td>319</td>
<td>45.2</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>257</td>
<td>2.78</td>
<td>32</td>
<td>-0.89</td>
<td>205</td>
<td>-0.51</td>
<td>8</td>
<td>223</td>
<td>35</td>
<td>29</td>
</tr>
</tbody>
</table>
Mean Birthweight Over Time

Figure 4
Study Community birthweights in 1986-90 were similar to those of the N.T. aboriginal aggregate, but lower than the Australian aboriginal aggregate, and much lower than the non-aboriginal aggregate for the N.T. and the Australian total.

Table 4 Community birthweights in 1986-90 vs. other Aboriginal and Australian total (1992,93).

<table>
<thead>
<tr>
<th></th>
<th>Mean Birthweight(kg)</th>
<th>Proportion LBW(&lt;2.5kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study Community</td>
<td>2.93</td>
<td>15.3%</td>
</tr>
<tr>
<td>N.T. Aboriginal*</td>
<td>3.005</td>
<td>17%</td>
</tr>
<tr>
<td>Aust. Aboriginal*</td>
<td>3.150</td>
<td>12.9%</td>
</tr>
<tr>
<td>N.T. non-Absoriginal*</td>
<td>3.308</td>
<td>6.2%</td>
</tr>
<tr>
<td>Australia*</td>
<td>3.356</td>
<td>6.3%</td>
</tr>
</tbody>
</table>

*Preinatal Statistics Series (Lancaster, Huang, Pedisch 1992)

**Weight at 6 months.**

The mean weight at 6 months was 7.01kg, 7.18kg in boys and 6.77kg in girls. The mean weight for age z scores were lower in boys than girls although this difference was not statistically significant (Table 3). Mean relative weight improved between birth and six months, reflected by the rise in mean z scores in both boys and girls.

Birthweight was correlated with weight at 6 months (Table 2). The proportion of children who were malnourished at 6 months (Table 5) was much higher in the low birthweight group and those with a low normal birthweight (2.5-2.99kg). The odds ratio for being malnourished at 6 months was 6.47 (C.I. 3.39-12.37) in low birth weight babies (<2.5kg).

Table 5 Distribution of WAZs among children categorised by birthweight.

<table>
<thead>
<tr>
<th>Birthwht</th>
<th>&gt;3kg</th>
<th>2.5-2.99kg</th>
<th>2.2-2.49kg</th>
<th>&lt;2kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>WAZ&lt;sub&gt;6&lt;/sub&gt;</td>
<td>n=173</td>
<td>n=188</td>
<td>n=113</td>
<td>n=31</td>
</tr>
<tr>
<td>&gt;0</td>
<td>46%</td>
<td>5%</td>
<td>10%</td>
<td>10%</td>
</tr>
<tr>
<td>-1-0</td>
<td>39%</td>
<td>36%</td>
<td>37%</td>
<td>16%</td>
</tr>
<tr>
<td>-2 to -1</td>
<td>12%</td>
<td>33%</td>
<td>35%</td>
<td>39%</td>
</tr>
<tr>
<td>-2*</td>
<td>3%</td>
<td>26%</td>
<td>18%</td>
<td>35%</td>
</tr>
</tbody>
</table>

\text{Chi}=106.926, \ p=0. \ *Reference \ rate \ of \ WAZ \leq -2 \ is \ 2.3%
Over time mean WAZ\textsubscript{b} has made little overall progress, unlike birthweight (see fig 5). WAZ\textsubscript{b} has actually fallen since the 1960s and has overall a negative association with year of birth in linear regression after controlling for WAZ\textsubscript{b}(p=0.001, coeff=-0.016).

**Weights at 1 year.**

The mean weight at 1 year was 8.26kg, 8.49kg in males and 7.93kg in females. Relative weights were lower in boys, with the proportions malnourished being significantly higher than girls (Table 3, chi=4.956, p=0.026).

The mean WAZ\textsubscript{12} was much lower than WAZ\textsubscript{b} (Table 3, figure 6). The standard deviations were similar, implying that the entire population’s weight was shifted downwards.

![Figure 6 Mean weight in Infancy](image)

The drop in relative weight was reflected in the huge rise in proportions of infants in the lowest decile of the reference population, from 25% at 6 months to 63% at 12 months (Fig 7). At 12 months 74% of girls and 93% of boys were below the WHO mean (the reference population’s rate would be 50%).
Weight at birth and six months was correlated with weight at 12 months (Table 2). Low birth weight babies have a poorer outcome at 12 months. 39% of low birth weight children are malnourished at 12 months of age compared with 26% of normal birth weight children (chi=7.942, p=0.005). Low birth weight babies made up 63% of children classified as malnourished at 12 months.

The odds ratio for malnutrition at 12 months increased with lower z scores at 6 months, demonstrating that the most at risk children are light already by 6 months.

<table>
<thead>
<tr>
<th>Table 6 Odds Ratios for Malnutrition at 12 months (n=488) by WAZ.&lt;br&gt;</th>
<th>Odds ratio</th>
<th>95% C.I.</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>waz&gt;0, n=131</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0&gt;waz&gt;-1, n=176</td>
<td>3.234</td>
<td>1.6-6.4</td>
<td>0.001</td>
</tr>
<tr>
<td>-1&gt;waz&gt;-2, n=134</td>
<td>14.28</td>
<td>7.1-28.4</td>
<td>0.000</td>
</tr>
<tr>
<td>z&lt;-2, n=47</td>
<td>21.40</td>
<td>8.9-51.1</td>
<td>0.000</td>
</tr>
</tbody>
</table>

WAZ<sub>12</sub> varied over time in a similar pattern to the WAZ<sub>6</sub> (see Fig 3). From 1966-70 to 1986-90 WAZ<sub>12</sub> fell from -1.21 (s.d 0.92) to -1.73 (s.d. 0.87). Linear regression confirmed this, with an overall negative association between year of birth and WAZ<sub>12</sub> (Coef -0.011, p=0.005) after controlling for WAZ<sub>6</sub> (WAZ<sub>6</sub> is not significant when WAZ<sub>6</sub> is included).
Birthweight and Infant Weight Over Time

figure 5
In 1986 the study community's mean weight in infancy (n=97) was comparable with that of Thailand, with rates of malnutrition close to those of Nigeria (table 7).

**Table 7 Infant weight means compared with Developing Countries.**

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Age group (yrs)</th>
<th>Mean Infant weight</th>
<th>S.D.</th>
<th>%&lt;2 S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study Community</td>
<td>1986</td>
<td>0.5-3</td>
<td>-1.17</td>
<td>1.09</td>
<td>23</td>
</tr>
<tr>
<td>Brazil</td>
<td>1986</td>
<td>0.5-3</td>
<td>-0.71</td>
<td>1.17</td>
<td>10.2</td>
</tr>
<tr>
<td>Burundi</td>
<td>1987</td>
<td>0.5-3</td>
<td>-1.53</td>
<td>1.21</td>
<td>32.5</td>
</tr>
<tr>
<td>Lebanon</td>
<td>1990</td>
<td>0.5-3</td>
<td>-0.61</td>
<td>1.04</td>
<td>7.9</td>
</tr>
<tr>
<td>Nigeria</td>
<td>1987</td>
<td>0.5-3</td>
<td>-1.34</td>
<td>1.10</td>
<td>26.9</td>
</tr>
<tr>
<td>Romania</td>
<td>1991</td>
<td>0-2</td>
<td>-0.49</td>
<td>1.04</td>
<td>6.2</td>
</tr>
<tr>
<td>Sri Lanka</td>
<td>1987</td>
<td>0.5-3</td>
<td>-1.65</td>
<td>1.03</td>
<td>38.3</td>
</tr>
<tr>
<td>Thailand</td>
<td>1987</td>
<td>0.5-3</td>
<td>-1.13</td>
<td>1.11</td>
<td>19</td>
</tr>
<tr>
<td>Trinidad/Tobago</td>
<td>1987</td>
<td>0.5-3</td>
<td>-0.43</td>
<td>1.22</td>
<td>6.6</td>
</tr>
</tbody>
</table>

*WHO figures

**Weights in childhood and adolescence.**

The mean weight for age z score at screening was -1.06 for boys (s.d. 1.02) and -0.74 (s.d. 1.11) for girls, boys being significantly worse (p=0.015) (Table 8). The means are still markedly lower than the reference, particularly in boys, who have rates of malnutrition at 11% in the 5 to 9 year age group.

**Table 8 Weights and Heights at Screening.**

| Age and gender | N=    | mean WAZ_{(sd)} | WAZ %<-2 | W-H-Z %<-2sd* | HAZ mean_{(sd)} | HAZ %<-2#
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>5-9 M</td>
<td>46</td>
<td>-0.87_{(0.78)}</td>
<td>10.8</td>
<td>-0.45_{(0.84)}</td>
<td>-0.76_{(0.04)}</td>
<td>8.7</td>
</tr>
<tr>
<td>5-9 F</td>
<td>59</td>
<td>-0.86_{(0.88)}</td>
<td>6.8</td>
<td>-0.61_{(0.8)}</td>
<td>-0.64_{(0.89)}</td>
<td>5.3</td>
</tr>
<tr>
<td>10-13M</td>
<td>103</td>
<td>-0.98_{(1.03)}</td>
<td>8.7</td>
<td>N.A.</td>
<td>N.A.</td>
<td>9.8</td>
</tr>
<tr>
<td>10-13 F</td>
<td>74</td>
<td>-0.66_{(1.13)}</td>
<td>6.8</td>
<td>N.A.</td>
<td>N.A.</td>
<td>18.8</td>
</tr>
<tr>
<td>14-17M</td>
<td>73</td>
<td>-1.15_{(1.27)}</td>
<td>19.1</td>
<td>N.A.</td>
<td>N.A.</td>
<td>12</td>
</tr>
<tr>
<td>14-17 F</td>
<td>35</td>
<td>-0.66_{(1.19)}</td>
<td>11.4</td>
<td>N.A.</td>
<td>N.A.</td>
<td>15</td>
</tr>
</tbody>
</table>

*The WHO definition of malnutrition. # WHO definition of stunting. +WHO definition of wasting

W-H-Z Weight for height in z scores. N.A. not available.

In linear regression, weight at screening was positively and significantly associated with HAZ_{0}, WAZ_{0} and WAZ_{12} after accounting for sex and age (Table 2 and appendix).
Height.

Height for age was also studied at the time of screening. Mean z scores were -0.82 for boys (s.d 0.96) and -0.93 for girls (s.d. 1.80). (Table 8).

In linear regression height at time of screening was significantly associated with WAZ₄, WAZ₀ WAZ₆ and WAZ₁₂, after accounting for sex, which was not statistically significant (see appendix). This implies that a poor nutritional state in infancy may lead to stunting in adolescence or delayed puberty, both causes of a lower height for age.

Weight for Height.

WHZ was calculated on children between 5-11 years. It showed few children to be wasted at these ages for their height, although the mean height was well below the reference mean. Figure 8 shows the majority of children to be light for their height, with 57% in the lowest three deciles of the reference population. Weight for height was unavailable on the older children, as the influence of puberty could not be accounted for.
Rates of Skin lesions, PSGN and Streptococcal Serology.

Skin Lesions.

68% of boys and 56% of girls had at least one skin sore at screening, (Table 9, chi=4.444, p=0.035). The rate of scabies infestation was 29% in children, 24% in boys and 33% in girls. The proportions of people with skin sores and scabies fell with increasing age.

In logistic regression analysis after controlling for age, the presence of sores was 4 times as likely in the presence of scabies (C.I. 2.29-8.40 p=0.00). After accounting for sex, age and sores, scabies had a negative association with weight for age z score at 12 months (O.R. 1.78, C.I.1.24-2.54, P=0.02), but sores did not.

Table 9 Prevalence of Scabies, Sores and PSGN.

<table>
<thead>
<tr>
<th></th>
<th>Sores (%)</th>
<th>Scabies (%)</th>
<th>PSGN (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;10</td>
<td>M(n=46)</td>
<td>73</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>F(n=58)</td>
<td>63</td>
<td>47</td>
</tr>
<tr>
<td>10-17</td>
<td>M(n=107)</td>
<td>66</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>F(n=89)</td>
<td>53</td>
<td>25</td>
</tr>
<tr>
<td>Adults</td>
<td>M(n=232)</td>
<td>24</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>F(n=176)</td>
<td>21</td>
<td>18</td>
</tr>
</tbody>
</table>

Streptococcal infection markers.

A large proportion of the streptococcal serology was above the range for normal (figure 9). The difference in titres between boys and girls was not significant. Girls had significantly higher proportions of abnormal antiDNAse titres than boys (chi=7.648, p=0.054).
ASOT titles were significantly associated with Anti-DNase titles and the presence of sores and scabies. Anti-DNase titles were also marginally associated with sores and scabies. It is of interest that asot titles were negatively associated with WAZ_{12} (coeff=-0.19, p<0.05).

Table 10 Kendall's Tau Correlations of Infectious Risk Factors.

<table>
<thead>
<tr>
<th></th>
<th>scabies</th>
<th>any sores</th>
<th>asot(cat)</th>
<th>antiDNase (cat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>scabies</td>
<td>1</td>
<td>0.32*</td>
<td>0.14*</td>
<td>0.10*</td>
</tr>
<tr>
<td>any sores</td>
<td>1</td>
<td>0.32*</td>
<td>0.14*</td>
<td>0.10*</td>
</tr>
<tr>
<td>asot(cat)</td>
<td>1</td>
<td>0.14*</td>
<td>0.17*</td>
<td></td>
</tr>
<tr>
<td>antiDNase(cat)</td>
<td>1</td>
<td></td>
<td>0.17*</td>
<td></td>
</tr>
</tbody>
</table>

*p<0.05, *p<0.1
Post-streptococcal Glomerulonephritis Epidemics.

A history of acute post-streptococcal glomerulonephritis (PSGN) was identified in 113 people, of these 106 were 4 years before the screening date.

The rates of people with a history of PSGN were very high, particularly in adults, 42% in females and 27% in males. PSGN was significantly more common in females (p=0.039). 86% of the people with a history of PSGN were below ten years at the time of infection.

Most of the cases were identified during epidemics; 36 from 1980, 43 from 1987 and 5 from 1994. The remaining 41 were sporadic infections, more of which were identified as time progressed. This is likely to be due to better identification of cases.

PSGN was not correlated with any parameter of infant or childhood weight. As most people had had the PSGN episode at least fours years before screening, the associations with screening results were deemed very weak and not included.

Prevalence of Albuminuria and Haematuria.

1) Albumin to Creatinine Ratio (ACR).

Table 11 shows ACR was above the normal range (0-1.1) in 22% of children, most of this in the “suspicious” category (1.1-3.390). Girls had significantly higher proportions of suspicious ACRs (OR 1.94, CI 1.19-3.19, p=0.01).
The rate of abnormal ACRs increased rapidly with age across the population. In adults only 30% of males and 38% of females had normal ACRs in the 28 to 38 year age group. Females sex, age and current weight were associated with log ACR in adults.

Table 11 Prevalence of Renal Markers by age group and sex.

<table>
<thead>
<tr>
<th>N=</th>
<th>ACR normal</th>
<th>ACR 1.1-3.35</th>
<th>ACR 3.4-33.9</th>
<th>ACR 34+</th>
<th>haematuria (&gt;trace)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5-9 M</td>
<td>48</td>
<td>79%</td>
<td>19%</td>
<td>2%</td>
<td>0%</td>
</tr>
<tr>
<td>5-9 F</td>
<td>59</td>
<td>78%</td>
<td>19%</td>
<td>2%</td>
<td>2%</td>
</tr>
<tr>
<td>10-17 M</td>
<td>174</td>
<td>83%</td>
<td>13%</td>
<td>3%</td>
<td>1%</td>
</tr>
<tr>
<td>10-17 F</td>
<td>107</td>
<td>68%</td>
<td>19%</td>
<td>11%</td>
<td>2%</td>
</tr>
<tr>
<td>18-27 M</td>
<td>98</td>
<td>54%</td>
<td>16%</td>
<td>27%</td>
<td>2%</td>
</tr>
<tr>
<td>18-27 F</td>
<td>64</td>
<td>33%</td>
<td>28%</td>
<td>20%</td>
<td>19%</td>
</tr>
<tr>
<td>28-38 M</td>
<td>70</td>
<td>30%</td>
<td>26%</td>
<td>28%</td>
<td>16%</td>
</tr>
<tr>
<td>28-38 F</td>
<td>51</td>
<td>38%</td>
<td>14%</td>
<td>32%</td>
<td>16%</td>
</tr>
</tbody>
</table>

2) Haematuria

Haematuria (defined as greater than trace of blood on dipstick) was found in 25% of the children. Girls had significantly higher rates, 33% compared with 18% in boys (OR 1.59, CI 0.99-2.55, p=0.055). Rates rose significantly with increasing age group in boys (p=0.002, chi=12.409) while girls maintained high levels throughout.

In adults haematuria was also significantly more common in females (chi=31.177, p=0).

Correlations between Birthweight, Infant Weights and Albuminuria.

The association between birthweight, WAZ6 and WAZ12 and ACR was examined and compared in children (aged <18 at screening) and adults (≥18 at screening).

1) In Children.

In linear regression, after accounting for age and sex, Log ACR was negatively associated with birthweight (expressed in weight for age z scores) in children(Table 12). However little of the variance was explained. There was also a negative correlation between logACR and WAZ6 and WAZ12 but this did not reach statistical significance.

Table 12 Association between lnACR and Birthweight in children.

<table>
<thead>
<tr>
<th></th>
<th>Coeff</th>
<th>p value</th>
<th>St.Err</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-0.933</td>
<td>0.00</td>
<td>0.161</td>
</tr>
<tr>
<td>sex f vs m</td>
<td>0.429</td>
<td>0.00</td>
<td>0.102</td>
</tr>
<tr>
<td>WAZ6</td>
<td>-0.085</td>
<td>0.05</td>
<td>0.043</td>
</tr>
</tbody>
</table>

R²=0.059
In children the rates of suspicious ACR and above were significantly and inversely associated with birthweight and WAZ\textsubscript{12} (table 13) in logistic regression. WAZ\textsubscript{6} failed to reach statistical significance.

WAZ at screening was also inversely associated with logACR and marginally (p=0.089) with suspicious ACR (table 13).

<table>
<thead>
<tr>
<th>Table 13  Logistic Regression Models for suspicious ACR (&gt;1.1) in Children.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Birthwt. (kg)</td>
</tr>
<tr>
<td>WAZ\textsubscript{12}</td>
</tr>
<tr>
<td>WAZ\textsubscript{6}</td>
</tr>
</tbody>
</table>

controlling for sex. NEG=Negative association.

Figure 10 demonstrates the similar inverse relationships of birthweight and infant weight with ACR≥1.1 in children, adjusted for sex.

Figure 10. Probability of ACR≥1.1 in children by birthweight and infant weight tertiles.

2) In Adults.

In adults, birthweight and infant weights carried equal strength and significance in association with logACR in linear regression (table 14). WAZ\textsubscript{6} and birthweight and WAZ\textsubscript{12} in categories were all significantly and negatively associated with logACR after controlling
for sex and current weight. Age was not significantly associated with ACR in these models (see appendix table for this and exact models).

<table>
<thead>
<tr>
<th>Model</th>
<th>Coeff</th>
<th>p value</th>
<th>St.Err.</th>
<th>Rsq.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1. Bwt. (cat)</td>
<td>-0.317</td>
<td>0.031</td>
<td>0.146</td>
<td>0.17</td>
</tr>
<tr>
<td>Model 2. WAZ6</td>
<td>-0.325</td>
<td>0.011</td>
<td>0.126</td>
<td>0.18</td>
</tr>
<tr>
<td>Model 3. WAZ12 (cat)</td>
<td>-0.283</td>
<td>0.046</td>
<td>0.141</td>
<td>0.17</td>
</tr>
</tbody>
</table>

(controlling for sex and current weight in same cohort, in adults).

In logistic regression, the odds of ACR 34 increased 3 times for each kg decrease in birthweight (CI 1.28-6.99, p=0.01). The odds for infants who were < 1 SD below the WHO median (Z<-1) was 3.79 times (CI 1.03-13 97, p=0.045) times that of those above the WHO median (z>0).

There was no correlation between early growth parameters and haematuria at the time of screening in adults or children.

**Correlations between Skin lesions, Streptococcal serology, PSGN and albuminuria and haematuria.**

**Skin lesions and renal markers.**

In adults, the presence of scabies was significantly associated with logACR in linear regression. In logistic regression the odds ratio for those with scabies to have ACR ≥3.4 was 1.85 (1.0-3.2) (table 15). The presence of skin sores was associated with haematuria, but not ACR.

In children in whom scabies and sores were more common, both were associated with suspicious ACR(>1.1), after controlling for sex and age. However when included in the
same model both failed to reach significance due to their high correlation. The odds ratio for ACR ≥ 1.1 and haematuria in children with a history of PSGN was 2.71 (0.9-7.7).

**Streptococcal Serology and renal markers.**

Streptococcal serology was not significantly associated with ACR or haematuria in logistic or linear regression after controlling for age and sex.

<table>
<thead>
<tr>
<th></th>
<th>Sores</th>
<th></th>
<th>Scabies</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Haematuria</td>
<td>27%</td>
<td>20.40%</td>
<td>29%</td>
<td>24%</td>
</tr>
<tr>
<td>ACR&gt;1.1</td>
<td>29%</td>
<td>18%</td>
<td>24%</td>
<td>17%</td>
</tr>
<tr>
<td>Children</td>
<td>OR 1.72</td>
<td>CI 1.0-2.9</td>
<td>O.R. 1.87</td>
<td>CI 1.0-3.4</td>
</tr>
<tr>
<td>ACR &gt;3.4</td>
<td>57%</td>
<td>50%</td>
<td>68%</td>
<td>53%</td>
</tr>
<tr>
<td>Adults</td>
<td>ns</td>
<td></td>
<td>O.R 1.85</td>
<td>CI 1.0-3.2</td>
</tr>
<tr>
<td>Haem &amp; suspACR Children</td>
<td>5%</td>
<td>3%</td>
<td>9%</td>
<td>4%</td>
</tr>
<tr>
<td></td>
<td>ns</td>
<td></td>
<td>O.R 2.71</td>
<td>CI 0.9-7.7</td>
</tr>
</tbody>
</table>

*ns = not statistically significant*

**PSGN.**

People with a history of PSGN were significantly more likely to have an elevated ACR, haematuria, or both, at screening (Table 16). The odds ratio was 3.9 (95% CI 2.1-7.2) in those whose history was more than four years previous to screening compared with those with no history of PSGN. This risk was amplified in children 10-18 years (n=68) with a history of PSGN where the odds ratio of haematuria and proteinuria together was 10.8 (C.I. 1.11-96.27, p=0.04) when compared with those with no history of PSGN.
When PSGN was modelled with ACR ≥ 3.4 in adults, 22% of the variance was explained and sex was much less significant. Scabies and PSGN were too highly correlated to be significantly modelled together.

<table>
<thead>
<tr>
<th>Table 16 Effect of PSGN on 3 Different Urinary Outcomes.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Outcome</strong></td>
</tr>
<tr>
<td>---------------</td>
</tr>
<tr>
<td>Elevated ACR(&gt;3.4)</td>
</tr>
<tr>
<td>adults+</td>
</tr>
<tr>
<td>children#</td>
</tr>
<tr>
<td>Haematuria</td>
</tr>
<tr>
<td>adults*</td>
</tr>
<tr>
<td>children*</td>
</tr>
<tr>
<td>Elevated ACR + haematuria</td>
</tr>
<tr>
<td>adults*</td>
</tr>
<tr>
<td>children*</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex and current weight #Adjusted for age, sex and WAZ. \(^*\)Adjusted for age and sex.

Figure 11 demonstrates people with a history of PSGN have a higher ACR in each age group.

**Figure 11. ACR and History of PSGN**

![Graph showing ACR and History of PSGN](image)

Figure 12 shows people with a history of PSGN are more likely to have an ACR in a higher category with increasing age. The probability of having an ACR 3.4\(^*\) is advanced by about 5 to 10 years by having a history of PSGN.
Figure 12. History of PSGN and ACR Category.

Probability of ACR 34+

Probability of ACR 3.4+

Probability of ACR 1.1+

There were inadequate numbers of people with values for infectious risk factors and infant weights to form a model for comparison of the strength of each variable independently.
DISCUSSION.

Growth patterns.

Birthweights and Infant Weights.

The study community had ongoing poor birthweights. The mean birthweights and proportions of low birthweight equate with the N.T. Aboriginal aggregate and are well below that of the Australian aggregate. Birthweights among survivors to screening have risen over time since 1956. This improvement is likely to be an underestimate as the survival of low birthweight babies has improved greatly over that time. However, the 1981-90 low birthweight rate of 16% is still unacceptable.

The well recognised pattern of deteriorating weight for age z scores associated with weaning and intercurrent infections between six months and one year was clearly demonstrated. Mean weights (expressed as z scores) rose from -1.04 at birth to -0.61 at 6 months and fell dramatically at 12 months to -1.73. At 12 months 39% of boys and 29% of girls were malnourished (weight for age <-2 z scores), with 63% of the population in the lowest decile. International Aid agencies agree that rates of malnutrition at 8% are nutritional emergency levels (Ruben and Walker 1994). Rates in the study community are way above this figure, close to those of African nations with extremely poor child nutrition, such as Nigeria.

Although mean birthweight has continued to rise since 1956, the mean WAZ and WAZ_t have fallen since the 1960s. Therefore the relative drop in z scores from birth to 12 months has increased over time confirming Dr. Alan Walker’s impression (Walker 1994). Many young mothers are not educated or attentive to the nutritional needs of weaned babies; not uncommonly infants go hungry or are
fed inappropriate food such as ice-cream or junk foods. This highlights the problems of the breakdown of community and social supports for young mothers and the transition from traditional lifestyle, to one with little bush tucker and a reliance on store bought foods. In addition, community demoralisation and dissipation of money on alcohol, cigarettes and gambling, socioeconomic disadvantage and lack of education and primary health care services are all contributing factors.

This study is the first for many years to measure rates of undernutrition in the N.T. at a community level. The rates of malnutrition at 12 months are much higher than N.T. hospital based estimates at 0.5-3 years (Walker and Ruben 1995) and those in aboriginal communities in other states (see lit review). While improved hospital based management has reduced hospital mortality rates, this fails to address the continued poor growth patterns in infancy.

Birthweight and infant weights were highly correlated. There was a progressive increment in rates of undernutrition at 12 months with lighter weights at 6 months and at birth. This reflects the vicious cycle of repeated infection and poor nutrition in the nutritionally disadvantaged children which reaches a peak after weaning at around 12 months. The predictive value of low birthweight for lower infant weights, as described by others, was demonstrated, with 22.3% being malnourished at 6 months and 39% at 12 months.

This study was limited in the estimate of growth in infancy as there were no measures of infant height, so the length of time of undernutrition could not be estimated.
Childhood Growth.

In childhood the mean weight for age at screening was substantially below the reference values, although less so than in infancy. The poor mean heights for age and high rate of stunting suggests that chronic undernutrition is a prevalent problem. However, low weight and height for age in adolescence can also be due to delayed puberty for which no information was available. Childhood weight and height were both correlated with infant weights. This suggests that poor infant weight may impart an ongoing growth disadvantage or highlight continuing poor nutritional environments.

Mean birthweight, infant weight and childhood weights and heights in z scores were all lower in males than females and rates of malnutrition were consistently higher in boys. This demonstrates the recognised phenomenon, for a similar nutritional environment, boys have a poorer outcome (Sue Sayers pers comm.)

**Prevalence of scabies, skin sores and PSGN.**

Rates of scabies and skin sores were both very high in the study population, particularly in the children. Streptococcal infection is prevalent in this community, with high antiDNAse titres almost universal, as Van Buynder's study also found (1992). High rates of streptococcal infection and scabies reflect overcrowding, the use of common bedding, clothing and towels, poor environmental and personal hygiene and possibly poor nutrition.

The association between skin sores and scabies, and ASOT and anti-DNAse titres demonstrates the process of excessive itching from scabies and a consequent breach
of the skin leading to sores and secondary infection with streptococci. The presence
of scabies, and ASOT titres were negatively associated with WAZ12. This suggests
that poor early growth may retard the immune system predisposing to infectious
disease, although the small numbers make this only a suspicion.

Rates of previous FSGN infection are astoundingly high. Rates reached 22% of boys
and 36% of girls between 10 and 18 years in whom the record were the most
reliable. It is of interest that FSGN was more common in females, as Boineau et al
(1989) state FSGN is more common in males, with the usual male to female ratio at
1.5:1. Rates of scabies and high ASOT and antiDNAse titres were also higher in
females, suggesting that all are part of the same risk factor group.

Prevalence of albuminuria and haematuria.

This is the first study of Aboriginal children using the ACR. Consistently high rates of
suspicious ACR (>95th centile in a normal population) were found, probably
marking the beginning of the renal disease common in the adults. Van Buynnder
(1992) found high rates of proteinuria (>50mg/mmol urinary protein to creatinine
ratio) in Aboriginal children which correlates with the ACR but is less sensitive.

The rates of elevated ACR rose across the age groups, showing the progresion of
renal disease with age at a population level. Rates of raised ACR (>3.4) were very
high (over 20%) in the young adult population and 55% of all adults had an ACR ≥ 3.4 (Hoy 1996 in press). ACRs were consistently higher in females.

Haematuria was also common, more so in females in keeping with the rates of
abnormal ACRs. The attempts to control menstrual contamination were probably not
entirely successful in eliminating it from the samples. This does not account for the
whole difference, however, as the discrepancy began before puberty and continued after menopause. While some haematuria was glomerular in origin, and associated with $ACR \geq 3.4$, the incompleteness of the sample makes this difficult to interpret. The significance of isolated microscopic hematuria, much of which is probably non-renal needs further evaluation.

*Association between early Infant Growth and Renal Disease.*

The inverse association between birth weight and renal disease, measured by the ACR, was found for the first time in children and was confirmed in adults. As discussed in the background, low birth weight was agreed to be an adequate substitute for intrauterine growth retardation in Aboriginal newborns (Villar et al 1982, Sayers 1993, 1996, Blair 1994).

An inverse association between renal disease and early infant nutritional status was demonstrated for the first time in both adults and children. In adults $WAZ_{0}$ and $WAZ_{12-24}$ were found to be equivalent to birthweight in their association with ACR. In children, both $WAZ_{12}$ and birthweight were negatively associated with $ACR \geq 1.1$. This is a subtle range of albuminuria which would be missed with less sensitive traditional protein assays.

These findings are compatible with Barker’s observation of infant weight (as well as birthweight) predicting chronic adult disease. The correlation of renal disease with birthweight and $WAZ_{12}$, even in young children suggest mechanisms that operate on a continuum over life. These may antedate or be independent of the influence of diabetes, insulin resistance and hypertension. Increasing age and weight and a variety of other infectious and hemodynamic/metabolic factors probably magnify this effect throughout life.
Both infant weights and birthweight are highly correlated so it is not surprising they are equivalent in their prediction of ACR. The independant influences of each in predicting renal disease could not be compared. However the association of both with ACR suggests the nephrogenesis and the maturation of the kidney continues through early infancy as well as in utero.

The current epidemic of adult disease may be a legacy of improved survival of lower birthweight newborns since the 1960s, combined with the many other risk factors common in transitional communities. With the improvement in birthweights, but continued poor infant weights, the strength of each parameter may become evident. However it is imperative that both birthweight and infant weights be raised to reduce childhood morbidity and mortality in the short-term, as well as chronic renal and other disease later in life.

**Association between Scabies, Skin Sores, PSGN and Renal Disease.**

Scabies was associated with elevated ACR in both children and adults. This is likely to be due to the direct entry of streptococcal infection through breaches of the skin exacerbated by scratching or carried by the scabies mite itself. The presence of skin sores was significantly associated with elevated ACR in children. No statistical relationship was found between serological markers of streptococcal infections and ACR levels; this might be due to the high prevalence of abnormal streptococcal titres, giving little discrimination of risk, and the limited numbers in the study with a streptococcal serology measurement.

PSGN was strongly associated with elevated ACR, haematuria and the combination of haematuria and proteinuria. The very high odds ratio associated with PSGN for
ACR ≥ 3.4 in children aged 10-14 could be due to the greater accuracy of PSGN identification in this latter group. The female predominance of PSGN did not account for the sex difference in rates of elevated ACR in adults.

The association between ACR and PSGN was maintained after a follow-up period of over four years. This suggests PSGN has a role in chronic renal disease, which is not surprising in a community with high rates of streptococcal infection, PSGN and rapidly changing serotypes (Gardiner et al.). The ongoing renal dysfunction after PSGN confirms the findings of many other reports discussed in the Background (p 29). This might represent a continued inflammatory process after a simple PSGN episode, or mark repeated episodes of renal injury associated with streptococcal infections in predisposed individuals.

**Birthweight, Infant weight, Infections and Renal Disease.**

Birthweight, infant weight, scabies, skin sores and PSGN were all significantly associated with ACR. Modelling in adults showed the additional influence of diabetes, insulin resistance, increasing blood pressure and excessive drinking on ACR profiles (Hoy in press). This presents a multidimensional model of renal disease in this high risk Aboriginal population, with interactions between early undernutrition, infectious insults and haemodynamic/metabolic factors all significantly contributing to ongoing renal damage.
Recommendations.

Improved maternal and child health education and primary care programs are essential. Particular focuses will be needed towards young mothers and the introduction of adequate and nutritious food at weaning. Closer monitoring of infant and children’s nutritional state is needed, particularly in those who are low birthweight and around the time of weaning. Improved community based interventions are also needed for children identified as being poorly nourished.

Prevention of scabies, streptococcal infections and further PSGN outbreaks is paramount for the prevention of renal disease. Improved housing and hygiene facilities and practises, intensive scabies management and prompt treatment of skin sores are the cornerstones.

Maternal and child health and housing are closely intertwined with the many interrelated social problems common in Aboriginal communities, such as poor education, unemployment and substance abuse. These must also be addressed for an improvement in the overall disease burden. (photo H.Smith)
Community Feedback

The results of this study were presented to the local health board meeting on the 12th of December and at a staff meeting at the clinic. The diagrams discussed are shown in the appendix. These were also distributed at the Alcoholics Anonymous centre and the Women’s centre.

The health workers were happy to see the improvement in the birthweights. They agreed there were many good mothers but some mothers did not feed their children enough, as too much of the money went to the club. They saw the solution as the older women teaching the younger mothers to give their children more bush tucker. They also wanted the “Strong Mothers, Strong Babies, Strong Culture” program, to be introduced into the community. This program uses local nutrition workers to educate and empower women to care for their babies so that the community fabric is strengthened.

A recent epidemic of PSGN was well controlled by the health worker’s early detection of cases and penicillin prophylaxis for children with skin sores in the school. The problems of primary prevention through skin sore and scabies management seemed much larger; it would require extensive skin sore and scabies treatment and the involvement of the council to achieve better housing and access to hygiene.

Maternal and child health interweaves with the complex social fabric of a community in transition. Public health programs will only be effective if they retain the picture of the whole community.
Appendix

Data collection forms used.

Figure 13 was used for the chart abstractions. Figure 15 is the form was used in the physical examination of the children.

Regression tables from results.

Table 17 Predictors of WAZ at Birth and in Infancy.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Predictor</th>
<th>Coef</th>
<th>St.Err</th>
<th>p</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waz0 n=612</td>
<td>const</td>
<td>-3.147</td>
<td>0.38</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>gender</td>
<td>0.231</td>
<td>0.09</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>year of birth</td>
<td>0.024</td>
<td>0</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Waz1</td>
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<td>0.355</td>
<td>0.003</td>
<td></td>
</tr>
<tr>
<td></td>
<td>year of birth</td>
<td>-0.016</td>
<td>0.005</td>
<td>0.001</td>
<td>R²=0.252</td>
</tr>
<tr>
<td></td>
<td>waz0</td>
<td>0.462</td>
<td>0.038</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Waz12</td>
<td>const</td>
<td>-0.334</td>
<td>0.305</td>
<td>0.276</td>
<td></td>
</tr>
<tr>
<td></td>
<td>year of birth</td>
<td>-0.011</td>
<td>0.004</td>
<td>0.005</td>
<td>R²=0.40</td>
</tr>
<tr>
<td></td>
<td>Waz6</td>
<td>0.564</td>
<td>0.034</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

Table 18 Log ACR and Infant weights adjusting for sex, and current weight in adults.

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
<th>Model 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>sex</td>
<td>0.850#</td>
<td>0.889#</td>
<td>0.970#</td>
<td>1.008#</td>
<td>0.882#</td>
<td>0.912#</td>
</tr>
<tr>
<td>current weight age</td>
<td>0.043#</td>
<td>0.045#</td>
<td>0.047#</td>
<td>0.049#</td>
<td>0.043#</td>
<td>0.044#</td>
</tr>
<tr>
<td>bwt(cat)</td>
<td>0.03</td>
<td>-0.304#</td>
<td>-0.317#</td>
<td>0.021</td>
<td>0.019</td>
<td></td>
</tr>
<tr>
<td>wt1-6 (waz)</td>
<td>-0.312#</td>
<td>-0.325#</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>wt1-2 (cat)</td>
<td></td>
<td></td>
<td>-0.262+</td>
<td>-0.283#</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rsq.</td>
<td>0.173</td>
<td>0.169</td>
<td>0.18</td>
<td>0.177</td>
<td>0.169</td>
<td>0.167</td>
</tr>
</tbody>
</table>

#p<0.05, +p<0.1
**TIWI KIDNEY DISEASE PROJECT**

**CHILDREN'S CHART ABSTRACTION FORM 1.1 WH 31/7/92**

<table>
<thead>
<tr>
<th>NAME:</th>
<th>Birth Date</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HRN:</th>
<th>Study No.</th>
<th>Exam date</th>
<th>Examiner</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**MOTHER**
**FATHER**

**SISTERS**
**BROTHERS**

**Growth:**
- birth weight (in kgs)
- failure to thrive (Y/N)
- percentile in first year
- percentile now

**Infections:** last year, total 0-12/12 1-5yrs 5-10yrs 10-14yrs

- Throat
- Skin
- Chest
- Ear
- G-E
- urine
- other (specify)

**Post infections**
- Acute GN Y/N
- Acute RF Y/N
- Rheum HD Y/N

**Renal History**

- Hematuria Y/N
- Proteinuria Y/N
- Hypertension Y/N level
- Evaluation Y/N by
- Family Hx Y/N DK

**Comments:**
**TIWI KIDNEY DISEASE PROJECT**

**BRIEF CHILDREN'S PHYSICAL EXAMINATION FORM**

**Children:** 10 - 17 years

**Exam Date:**

**Name:**

**ID Number**

**Birth date**

**HRN**

**Examiner:** WH BH JK NP

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**Height:** cm

**Weight:**

**Blood Pressure after sitting quietly for 2 mins:** /

**CHILD CUFF ONLY**

**SCABIES:** Yes / No

**SORES :** Yes / No

**SWAB TAKEN:** Yes / No

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<td>number wet</td>
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**BLOOD:** Red Cap Yes / No

**URINE:** Yes / No

**Urine Dipstick:** nitrite protein leukocytes glucose blood pH SG

**Urine Samples:** Clear Top Yes / No Yellow Top Yes / No

**Boric Acid:** Yes / No

**F & D:** Yes / No
Community Feedback.

The presentations which were used in the community feedback meetings and distributed around the community are included. Figure 15 describes proportions of low birthweight newborns over time. Low birthweight newborns were defined as <2.5 kg and were represented by the smaller, thinner babies. Figure 16 and 17 describe the proportions of malnourished babies at 6 and 12 months which were defined as < 2 standard deviations below the WHO median weight for age. These were represented by the smaller, thinner infants at both ages. Figure 18 shows the difference in ranges of infant weights between the Study community and non Aboriginal figures.

Figures 19 and 20 are flow diagrams were used to describe the influence of skin lesions and scabies and PSGN and kidney disease.
Weight at 6 months Over Time

1956 - 70

1986 - 90
Range of Weights at 1 year
Non-Aboriginal

Tiwi
Figure 19

- Not enough good food
- Skin Sores Scabies
- Kidney Problem
- Dirty, crowded houses
- Sick dogs
- Not enough washing
Submissions to Conferences.

The following abstracts were submitted to the International Society of Nephrology.


End stage renal disease (ESRD) rates in NT AA are >19 times of nonAA. With poor living conditions, high rates of scabies and skin sores, heavy colonisation with group A streptococci (GAS) of constantly changing serotypes, and endemic and epidemic acute post-streptococcal glomerulonephritis (APSGN), the role of GAS infection in chronic nephropathy needs careful evaluation.

We conducted a renal screen in one community with a 60-fold increase in ESRD. The community has had 4 epidemics of APSGN over 18 years, many sporadic cases, and repeated episodes in some individuals. From record review, 34% of female participants and 22% of males had a history of APSGN; 55% of adults had an ACR ≥3.4+; 35% of females and 15% of males had had microscopic hematuria; 19% had scabies, 62% of children and 29% of adults had skin sores, and grew GAS from 40% of cultures. ACR increased with age, female sex, weight, blood pressure, insulin resistance and diabetes, and with decreasing infant and birth weights. It also correlated with skin sores, with elevated ASOT in children (p=0.06), and with antibody to streptococcal M protein in adolescents and adults. A history of APSGN carried a 5-fold increase in risk for ACR 3.4+, and a 3.3-fold increase in ACR 3.4+ plus hematuria; APSGN >4 years prior was associated with 3.3 and 2.7-fold increases in these risks (p<.05 for all).

GAS infection contributes to chronic nephropathy in AA. Renal biopsies, which are avoided during APSGN, and are mostly from adults with longstanding proteinuria when specific changes have resolved, are insensitive to this fact. Only socioeconomic advancement and education, with improvements in housing, nutrition and hygiene will make a lasting change in this multi-dimensional problem.
FETAL AND INFANT MALNUTRITION AND CHRONIC RENAL DISEASE IN A HIGH RISK ABORIGINAL COMMUNITY: WE Hoy, KA Walker, DM McCredie, BD Hayhurst, JD Mathews. Menzies School of Health Research, Darwin, NT, Australia.

Low birth weight and infant malnutrition are common in Australian Aborigines. We screened one community with an incidence of renal failure of 2700 per million, using the urinary albumin/creatinine ratio (ACR, gm/mole) as the renal disease marker. Weights at birth (BW), and around 6 and 12 months were available on 482 people, ages 4-37 yr at screening. These were converted to weight-for-age Z scores (standard deviations from age-and sex-specific NCHS medians: WAZ0, WAZ6, WAZ12).

Mean BW was 2.79 kg, and WAZ0 -1.05. Mean WAZ6 was -0.63, but WAZ12 had fallen to -1.58. This pattern is associated with weaning and recurrent infections, and was more marked in the late 1980s than the 1960s (WAZ12 -1.74 vs -1.21, p=.012). Mean WAZ in children (<18yr) at screening was -0.88, but adults progressively gained weight, mostly as central fat, until their late 40s.

ACR levels correlated with age, female sex, weight, blood pressure, insulin resistance and diabetes, and evidence of streptococcal skin infections. LogACR also correlated with the somewhat interchangeable parameters of early malnutrition. In children, risk for ACR >1.1 (>95th percentile) increased 1.9-fold for each kg decrease in BW and 1.4-fold for each SD decrease in WAZ12. In adults, rates of ACR 3.4+ (microalbuminuria and above) increased 1.4-fold for each 500 mg decrease in BW, and for each SD decrease in both WAZ6 and WAZ12 (p <.05 for all).

Fetal and infant malnutrition are risk factors for chronic renal disease, possibly through impaired nephrogenesis. They compound the powerful accentuating effect of increasing weight on albuminuria in adolescents and adults. Birth weights are gradually improving, but the deterioration in infant nutrition since the 1960s is seriously disturbing. Maternal and child health programs must be improved, with promotion of healthy eating and exercise for entire communities.
The following abstracts were submitted to the Australian dieticians society conference.

LITTLE IMPROVEMENT SINCE 1960 IN WEIGHT-FOR-AGE IN INFANTS IN AN ABORIGINAL COMMUNITY.

Kate Walker, Wendy Hoy, Dorothy Mackerras
Menzies School of Health Research

Between 1992-4, most of members of an aboriginal community in the Top End aged ≥5 years were screened for markers of renal disease. Information about birthweight and weight between the ages of 1-24 months was extracted from the clinic records for 612 individuals born in 1956 and later. To permit comparisons across ages and sexes, weight-for-age was converted into a z-score using the NCHS reference curve through EpiInfo. For well nourished populations that are identical to the reference curve, the average z-score at all ages is 0.

From 1956-60 to 1987-89 mean birthweight rose from 2656g to 2936g and the low birthweight rate fell from 39% to 15%. By comparison, in 1992 the mean birthweight in Australia was 3356g with a low birthweight rate of 6.3%.

The mean z-score rose from -1.04 at birth to -0.61 at 6 months and fell dramatically at 12 months to -1.58, following a well recognised pattern associated with weaning and infection. In spite of increasing birthweights over time, there was a deterioration in z-scores at 12 months from 1966-70 to 1986-90 from -1.214 (s.d. 0.92) to -1.73 (s.d. 0.87) (see Figure). Between 1981-90, 45% of boys and 35% of girls met the definition of malnutrition (z-score < -2) at 12 months.

Thus improving birthweights have not lead to improved weight-for-age in infants over time. In fact z scores at 1 year have fallen. Although birthweight is the only anthropometric measurement collected on all children in Australia we cannot assume that birthweight trends can be used as a surrogate indicator for toddler’s nutritional status.

The prevalence of malnutrition in this and other communities is disturbing. Transition from traditional lifestyle and socioeconomic disadvantage and disempowerment, with inadequate health education and primary care services are all likely to be contributory factors. Concern about overweight in 'mainstream' Australia should not divert attention from development of programs to address this neglected situation.
THE BARKER HYPOTHESIS AND CHRONIC RENAL DISEASE: SUPPORTIVE DATA

K Walker, W Hoy, D McCreadie, B Hayhurst, JD Mathews
Menzies School of Health Research, Darwin, NT

Barker has hypothesised that the so-called diseases of affluence are really diseases of change and occur more frequently in populations characterised by early malnutrition (intra-uterine growth retardation) and adult obesity. He hypothesises that diseases often referred to as being due to 'Syndrome X' are actually due to this cause. Evidence to date has mainly related to coronary disease and diabetes. Chronic renal failure in Aborigines exhibits similar epidemiological patterns to the other Syndrome X diseases. Others have shown that a substantial proportion of low birthweight in Aborigines in the Top End is due to intra-uterine growth retardation, not pre-term birth. In this study we examine the early nutritional status and later renal disease patterns in a community that has the highest rate of renal failure in the world (2700 per million).

Methods

A spot urine obtained to determine the albumin/creatinine ratio (ACR) and a blood sample to measure insulin resistance from virtually all community residents aged 5 and over. Blood pressure, weight and the presence of streptococcal skin infections and diabetes were ascertained. Birthweight and weight-for-age during the first two years of life were extracted from clinic records for all those born in 1956 or later. ARC was normalised using a log_{10} transformation. Malnutrition in early childhood was examined in two periods: 1-6 months of age and 12-24 months of age. The weight closest to 6 or 12 months of age was extracted. To correct for differences in age, weight-for-age for these two periods was converted to z-scores using the NCHS reference curve (WAZ_{6} and WAZ_{12}).

In those under 18, presence of renal disease was defined as logACR greater than the 95\% centile in the population; in adults it was defined as greater than 3.4 (microalbuminuria). Logistic regression was done to examine the strength of the association of other factors on the risk of renal disease.

Results

In children, birthweight and WAZ_{12} were both associated with the risk of renal disease. There was a 1.9-fold increase in risk for each kg decrease in birthweight and a 1.4-fold increase in risk per decline of 1 unit in WAZ_{12}, whereas there was no association with WAZ_{6}.

In adults, risk of renal disease was independently and inversely associated with weight at all three ages: there was a 1.4-fold increase in risk for decline of 500g in birthweight, 1 unit in WAZ_{1} and 1 unit in WAZ_{12} after adjustment for other factors, including current weight.

Discussion

Improving birthweight and infant weights are important because this lowers perinatal and infant mortality and morbidity. Increasing evidence shows that it may also reduce the incidence of chronic disease in adults increases the urgency to improve the situation. Maternal and child health programs need to be improved, as do diet and exercise levels.
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