RESEARCH

Growth and morbidity in children in the Aboriginal Birth Cohort Study: the urban–remote differential

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ALTHOUGH TWO-THIRDS of Indigenous people live in urban areas,[1] most research into the health status of Aboriginal Australians has been conducted in remote areas. It has focused on chronic diseases in adults[2] and poor growth and infectious disease in preschool-aged children.[3-5] Relatively few investigations have been done in the Indigenous school-aged population. The 1994 National Aboriginal and Torres Strait Islander Survey (NATSIS) measured height and weight, and found that rural children were shorter and lighter than their counterparts in capital cities.[6]

In this article, we report the prevalence of markers of growth, infection and chronic disease in a birth cohort of Aboriginal children living in the Darwin Health Region who were examined when aged 8–14 years (the Aboriginal Birth Cohort Study). We compared the prevalence of these markers between children living in the urban area of the region and those in remote Aboriginal communities.

ABSTRACT

Objectives: To describe the prevalence of markers of growth, chronic and infectious disease in peripubertal Aboriginal children living in the Darwin Health Region in the "Top End" of the Northern Territory, and to compare prevalence between children living in urban and remote areas.

Design: Cross-sectional survey nested in a prospective birth cohort.

Subjects: 482 children living in the region who were recruited at birth (Jan 1987 to Mar 1990) and were followed up between 1998 and 2001, when aged 8–14 years.

Main outcome measures: Selected parameters of growth and nutrition, infectious disease and potential markers of chronic adult disease were compared between children living at follow-up in suburban situations in Darwin–Palmerston (urban) and those living in rural communities with an Aboriginal council (remote).

Results: Remote children were shorter than urban children (mean height, 141.7 v 146.3 cm; $P < 0.001$), lighter (median weight, 30.3 v 37.1 kg; $P < 0.001$) and had lower body mass index (median, 15.3 v 17.9 kg/m^2; $P < 0.001$) and haemoglobin level (mean, 125.1 v 130.9 g/L; $P < 0.001$). Some potential markers of adult chronic disease were higher in urban than remote children: systolic blood pressure (mean, 109.6 v 106.2 mmHg; $P = 0.004$), and levels of total cholesterol (4.3 v 4.0 mmol/L; $P < 0.001$), high-density lipoprotein cholesterol (mean, 1.4 v 1.2 mmol/L; $P < 0.001$) and insulin (median, 7 v 4 mU/L; $P = 0.007$). Diastolic blood pressure, levels of red cell folate, serum glucose and low-density lipoprotein cholesterol, and urinary albumin–creatinine ratio did not differ by location. The prevalence of visible infections was also higher in remote than urban children ($P < 0.05$).

Conclusion: As some markers of health differ between peripubertal Aboriginal children living in urban areas and those in remote areas, results of surveys in remote areas cannot be generalised to urban Aboriginal populations.

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METHODS

Subjects

Liveborn singleton infants of mothers who identified as Aboriginal and were recorded as such in the delivery suite register of the Royal Darwin Hospital between January 1987 and March 1990 were eligible for enrolment into a cohort.[7] Births in the hospital include all routine deliveries of infants from the Darwin Health Region (which comprises 120 000 km^2 of the "Top End" of the Northern Territory) and high-risk deliveries referred from a larger area in northern Australia. Recruitment depended on the availability of the neonatal paediatrician (S M S) to see the mother.

The children were followed up between December 1998 and March 2001 in over 40 locations. They were traced using all possible contacts, including community councils, health centres and schools, and were identified by checking birthdate and the unique hospital record number used in hospital and clinic records and given at birth (and collected as part of the recruitment process) and by talking to the caregiver.

Assessment

Children were measured while wearing light clothing and no shoes. Weight was measured to the last complete 0.1 kg with a digital scale, and height to the nearest millimetre with a portable wall-mounted stadiometer. Mid-upper-arm and waist circumferences were measured to the nearest millimetre directly on the skin using a flexible tape (using the 1995 National Nutrition Survey definition of waist location).[8] Triceps and subscapular skinfolds were measured three times to the nearest millime-

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1: Demographic and growth characteristics, by location of residence, Aboriginal Birth Cohort Study, 1998–2001

	n	Total	n	Urban	n	Remote	P
Demographic characteristics							
Age (years) (mean, SD)	482	11.4 (1.1)	99	11.3 (1.2)	345	11.4 (1.1)	0.3
Age distribution	482		99		345		
8–10 years		35%		47%		30%	0.003
11 years		32%		20%		35%	
12 years		28%		24%		29%	
13–14 years		6%		9%		5%	
Male sex	482	51%	99	55%	345	49%	0.4
Commenced puberty	443	48%	94	53%	313	46%	0.2
Growth characteristics*							
Height (cm) (mean, SD)	481	143.0 (10.1)	99	146.3 (10.7)	344	141.7 (9.5)	< 0.001
Weight (kg) (median, IQR)	481	32.0 (27.2–40.2)	99	37.1 (30.6–48.2)	344	30.3 (26.5–36.9)	< 0.001
Height-for-age z score (mean, SD)	481	−0.5 (1.1)	99	−0.1 (1.1)	344	−0.7 (1.0)	< 0.001
Weight-for-age z score (mean, SD)	481	−0.6 (1.3)	99	−0.1 (1.4)	344	−0.8 (1.2)	< 0.001
Height-for-age z score < −2	481	7%	99	2%	344	8%	0.03
Weight-for-age z score < −2	481	10%	99	2%	344	13%	0.002
Body mass index (kg/m^2) (median, IQR)	481	15.9 (14.4–18.3)	99	17.9 (15.8–20.5)	344	15.3 (14.3–17.3)	< 0.001
Mid-upper arm circumference (cm) (mean, SD)	448	21.2 (3.9)	79	23.7 (4.5)	342	20.6 (3.5)	< 0.001
Waist circumference (cm) (mean, SD)	452	61.4 (57.7–67.7)	81	66.4 (60.3–73.7)	340	60.5 (57.4–64.8)	< 0.001
Subscapular skinfold (mm) (median, IQR)	475	8.2 (6.6–13.5)	97	10.5 (7.1–15.5)	340	7.9 (6.6–12.3)	0.02
Triceps skinfold (mm) (median, IQR)	476	8.6 (7.1–11.8)	98	11.4 (8.5–15.7)	340	8.2 (6.9–10.4)	< 0.001
Subscapular/triceps ratio (mean, SD)	475	1.1 (0.4)	97	1.0 (0.3)	340	1.1 (0.4)	< 0.001
Haemoglobin (g/L) (mean, SD)	442	126.4 (9.5)	88	130.9 (8.3)	319	125.1 (9.4)	< 0.001
Anaemia							
Paterson definition[13]	442	1%	88	1%	319	1%	0.9
WHO definition[14]	442	14%	88	2%	319	17%	< 0.01

n = sample size. SD = standard deviation. IQR = interquartile range.
*Unadjusted data and P values are shown for simplicity. However, adjustment of variables for age, sex and pubertal status using multiple linear regression or logistic regression did not substantially alter the size of the differences between urban and remote children. For example, one of the largest differences was for waist circumference, which differed by 5.9 cm before adjustment and 5.4 cm afterwards.

tre using a Harpenden calliper, and the measurements averaged. A trained observer (D E M) did 80% of the measurements, and interobserver agreement was close (average difference, −0.1 mm; 95% limit of agreement, −4.0 to 3.8 mm for subscapular skinfolds[9]).

A paediatrician examined the children for infections and assessed pubertal development.[10,11] Sitting blood pressure was measured using an automatic blood pressure monitoring unit. An overnight fasting venous blood sample was taken after applying local anaesthetic and transported in an insulated container with cold bricks. All laboratory measurements were made at Western Diagnostic Pathology, Darwin, except plasma insulin level, which was measured at the Western Australian Centre for Pathology and Medical Research, Perth, WA. Red cell folate was measured on an ACS180 automated chemiluminescence system (Bayer, US). Albumin–creatinine ratio (ACR) was measured on a random urinary specimen, with urinary blood and protein measured at the time of collection using dipsticks.

Analyses

This analysis was restricted to children who lived in the Darwin Health Region (as defined by the Northern Territory Department of Health and Community Services at the time of recruitment). Children were classified as "urban" (living in a suburban situation in the Darwin–Palmerston area), "remote" (living in a rural community with an Aboriginal council) or "other" (including those living in towns, such as Jabiru and Howard Springs, and non-suburban situations in Darwin–Palmerston — town camps and Aboriginal communities). As health at follow-up was the main subject of interest, location at time of follow-up was used. Because the "other" group was small and heterogeneous, subregion analyses compared only the urban and remote groups.

Weight-for-age and height-for-age z scores were calculated from sex-specific reference curves.[12] Body mass index (BMI) was categorised using the centiles from the 1985 Australian Health and Fitness Survey.[13,14] Pubertal status was dichotomised as prepubertal or commenced puberty.

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2: Distribution of body mass index in Indigenous children using the 1985 Australian Health and Fitness Survey as the standard (percentage of cohort)

Centile	Australian Health and Fitness Survey (1985)[*14] (*n* = 8492)	Aboriginal Birth Cohort Study			National Aboriginal and Torres Strait Islander Survey (1994)[†6] (*n* =2252)		
		Total (*n* = 481)	Urban (*n* = 99)	Remote (*n* = 344)	Capital city	Other urban	Rural
< 5	5	31	16	37	16	24.5	28
5–15	10	18	9	21			
15–85	70	37	49	33	55	45.5	47.5
85–95	10	8	11	6	29	30	24.5
> 95	5	7	15	4			

*Distribution by definition for children aged 7–15 years for the whole of Australia.
†Average for children aged 7–15 years (from Table 3.6 of Cunningham and Mackerras[6]).

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Serum triglyceride, glucose and insulin levels were included in analyses only for children who stated that they had fasted for eight hours or longer. Anaemia was defined in two ways: haemoglobin level <105 g/L, to allow comparison with a previous NT report;[15] and according to the World Health Organization definition (<115 g/L for children aged 5–11 years, and <120 g/L for those aged 12–13 years and non-pregnant women).[16] Eosinophilia was defined as an eosinophil count >1.0 x 10^9 cells/L, the level used by Royal Darwin Hospital to indicate need for anthelmintic treatment. Neutropenia was defined as a neutrophil count <1.8 x 10^9 cells/L, and neutrophilia as a count >8.0 x 10^9 cells/L.[17] Urinary results from the dipsticks were dichotomised with "NAD" and "trace" grouped as "none". Girls who had reached menarche were excluded from analyses of haematuria.

Numbers of children in the analyses varied, as some were seen by only part of the study team and others had disabilities that prevented some measurements (eg, could not stand) or refused some procedures. Small blood sample volume occasionally limited biochemical analysis.

Variables were compared between children in urban and remote areas using *t* tests (for normally distributed continuous variables) and χ^2 tests (for categorical variables). Non-normal continuous data were transformed to yield a normal distribution before *t* tests. Tests were repeated after adjustment for age, sex and pubertal status using multiple linear regression of the variables (or their transformation) or logistic regression using STATA 7.[18]

The study was approved by the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research. The Aboriginal subcommittee of this committee had veto power. Parents or carers gave written informed consent.

RESULTS

We recruited 686 children at birth, of whom 570 had mothers living in the Darwin Health Region. There was no significant difference between the 686 children recruited and the 552 born in the same period but not recruited in either mean birthweight (3020 g v 3068 g) or sex distribution (51% v 53% male).

We followed up 572 of the 686 children recruited (86%); 18 had died, 65 were traced but not seen (including one who refused), and 31 could not be found. Of the 572 children followed up, 482 lived in the Darwin Health Region at the time of follow-up and were included in the analysis.

At follow-up, 99 of the 482 were classified as "urban" (of whom 69 were also "urban" at birth), 345 as "remote" (of whom 334 were also "remote" at birth) and 38 as "other". Mean age of the 482 was 11.4 years; 51% were boys, and 48% had commenced puberty (Box 1).

Growth characteristics

All measured growth characteristics differed significantly between the urban and remote children (Box 1). Urban children were taller and heavier, and their average height-for-age and weight-for-age *z* scores put them almost on the 50th centile of the international growth chart.[12] In contrast, *z* scores for remote children were nearly one standard deviation below the reference median, with mean height on the 24th centile of the international growth chart, and mean weight on the 19th centile. Consequently, the proportion of children with *z* scores below −2 was greater among remote children. The urban–remote difference was apparent even among children aged under 11 years, with height-for-age *z* scores −0.2 (urban) v −0.6 (remote), and weight-for-age *z* scores −0.1 (urban) v −0.7 (remote).

BMI was higher among urban children than among remote children, and the distribution of BMIs differed markedly between the two groups (Box 2). When compared with results of the Australian Health and Fitness Survey (1985), the distribution in the urban Aboriginal group was trimodal, with an 11.2% excess of children with BMI below the 5th centile and a 10.2% excess above the 95th centile. In contrast, 37% of remote children fell below the 5th centile, and only 9% were above the 85th centile, compared with the expected 15%. Remote children in this survey were also lighter than rural children in the 1994 NATSIS.

Not surprisingly, the other measures of fatness — skinfold thickness and mid-upper-arm and waist circumferences — were lower in the remote group (Box 1). However, remote children had a higher subscapular/triceps ratio than urban children, indicating a body fat distribution with greater truncal fat.

There was little anaemia in urban children, regardless of definition, whereas nearly 20% of remote children were anaemic by the WHO definition (Box 1). Mean haemoglobin level in remote children was 127 g/L in boys (SD, 10) and 124 g/L in girls (SD, 8), while the prevalence of anaemia as defined by WHO was 14% in boys and 20% in girls (*P* = 0.1).

3: Markers of chronic adult disease, by location of residence, Aboriginal Birth Cohort Study, 1998–2001*

	n	Total	n	Urban	n	Remote	P
Systolic blood pressure (mm Hg) (mean, SD)	472	107.0 (10.3)	97	109.6 (9.9)	337	106.2 (10.1)	0.004
Diastolic blood pressure (mm Hg) (mean, SD)	472	67.9 (7.3)	97	68.8 (6.0)	337	67.6 (7.5)	0.2
Total cholesterol (mmol/L) (mean, SD)	448	4.1 (0.8)	90	4.3 (0.7)	321	4.0 (0.8)	<0.001
HDL cholesterol (mmol/L) (mean, SD)	447	1.2 (0.3)	90	1.4 (0.4)	320	1.2 (0.3)	<0.001
LDL cholesterol (mmol/L) (mean, SD)	446	2.3 (0.7)	90	2.4 (0.6)	319	2.3 (0.7)	0.09
Triglycerides[†] (mmol/L) (median, IQR)	276	1.0 (0.7–1.4)	86	0.9 (0.6–1.4)	163	1.0 (0.7–1.4)	0.3
Insulin[†] (mU/L) (median, IQR)	260	5.0 (3.0–10.0)	80	7.0 (4–12)	154	4.0 (3–9)	0.007
Glucose[†] (mmol/L) (median, IQR)	274	4.5 (4.3–4.8)	87	4.6 (4.3–4.8)	160	4.5 (4.2–4.7)	0.09
Red cell folate (ng/mL) (median, IQR)	430	223 (177–288)	87	224 (183–299)	310	220 (174–292)	0.2
Urinary ACR (median, IQR)	452	0.7 (0.5–1.0)	92	0.7 (0.5–1.2)	328	0.7 (0.5–1.0)	0.7

n = sample size. IQR = interquartile range. HDL = high-density lipoprotein. LDL = low-density lipoprotein. ACR = albumin–creatinine ratio. *Unadjusted data and P values are shown, but adjustment for age, sex and pubertal status using multiple linear regression or logistic regression did not substantially alter differences between urban and remote children. †Fasting results.

Disease markers

Some risk markers for chronic adult disease varied between urban and remote children (Box 3). Systolic, but not diastolic, blood pressure was significantly higher in urban children, as were total and HDL cholesterol levels. There were no significant differences in LDL cholesterol or triglyceride levels, ACRs, or the proportion of children with microalbuminuria (defined as ACR >3.4; 10.8% [urban] v 6.7% [remote]; P = 0.2). Although there was also no difference in mean serum folate levels, the proportion of children with levels below the reference range for our laboratory (145–1000 ng/mL) differed significantly (3.5% [urban] v 11.6% [remote]; P = 0.02). However, no macrocytosis was seen on blood films. Insulin, but not glucose, levels were higher in the urban children.

Urban children had little obvious infection, with the exception of infected skin sores and ear perforations, whereas remote children had a substantial burden of visible infection. Remote children also had a higher prevalence of eosinophilia and a different distribution of neutrophils from urban children. There was no association between eosinophilia and anaemia (P = 0.3).

DISCUSSION

This is the first study to measure a range of growth, infection and chronic disease markers in a large sample of peripubertal Aboriginal children living in both urban and remote areas. It showed that, while growth is poor in children in remote areas of the Darwin Health Region, it is appropriate in urban children. Additional analyses revealed that the urban–remote differences were not attributable to delayed puberty in children in remote areas, as differences in height- and weight-for-age z scores were also apparent in children aged under 11 years.

Although the children were not randomly selected, there was no significant difference in birthweight or sex distribution between those recruited for the study and those born in the same period but not recruited. Therefore, the group is likely to be representative of the region's peripubertal population, and can be used to estimate the prevalence of health conditions for the region. Although there was some migration between urban, other and remote subregions between birth and follow-up, this

4: Markers of infection and infestation, by location of residence, Aboriginal Birth Cohort Study, 1998–2001*

	Total		Urban		Remote		
	n	% With condition	n	% With condition	n	% With condition	P
Clinical examination							
Ear discharge	451	6%	96	2%	317	8%	0.04
Ear perforation	448	17%	96	5%	316	23%	0.001
Nose discharge	453	5%	97	0	318	7%	0.01
Infected skin sores	453	24%	97	10%	318	27%	0.001
Scabies	452	9%	96	1%	318	12%	0.001
Urinary analysis							
Presence of blood[†]	392	8%	60	10%	310	8%	0.7
Presence of leukocytes	408	14%	67	11%	318	15%	0.4
Serum analysis							
Eosinophilia	429	44%	88	24%	306	51%	<0.001
Neutrophils							
Neutropenia	429	0.9%	88	3%	306	0.3%	0.03
Normal levels		94%		93%		94%	
Neutrophilia		5%		3%		6%	

n = sample size. *Unadjusted data and P values are shown, but adjustment for age, sex and pubertal status using multiple linear regression or logistic regression did not substantially alter differences between urban and remote children. †Girls who had reached menarche were excluded from analysis of this variable.

would be expected to increase the similarities between groups. Thus, the differences reported here are likely to underestimate urban–remote differences.

In our analysis of BMI distribution, we used the 1985 rather than the 1995 Australian Health and Fitness Survey as the standard, as it is clear that Australian children became fatter between 1985 and 1995,[19] and as the 1985 survey was used to analyse data from the 1994 NATSIS.[6] The remote children in our study were underweight compared with children in the 1985 national survey, with 58% falling below the 15th centile. Although urban children in our study had higher BMIs than their remote counterparts, comparison with NATSIS data (Box 2) shows that they are more similar to Indigenous children in "other urban" locations than to those in capital cities. A notable feature was the trimodal BMI distribution in urban children. It is likely that this population includes subgroups with quite different needs.

Variation in chronic disease markers in our study did not always match geographic variation in BMI. Systolic blood pressure and insulin level, which are often associated with fatness, were higher in the urban group, but the same pattern was not seen for HDL and triglyceride levels. Future follow-up may reveal whether the differences become more or less pronounced after all children have passed through puberty, and which variables are most strongly related to future health.

Previously, Paterson and colleagues reported that 24% of children aged 3–18 years who participated in the school screening program in remote communities in the Northern Territory were anaemic.[15] Using their definition of anaemia, we found that fewer than 2% of our group were anaemic. This may be because our group contained no young children and because we used blood samples obtained by venepuncture rather than fingerprick (which may give lower haemoglobin levels[20]). Using the stricter WHO definition of anaemia, about one in five children in the remote area were anaemic. The difference between the sexes was not entirely explained by the 4% of girls who had reached menarche. The lack of association between eosinophilia and anaemia

was not surprising, as hookworm is now rare in the Top End.[21]

Folate levels have received recent attention because they affect homocysteine levels, a risk marker for heart disease.[22] However, lack of comparability of laboratory methods for measuring folate levels[23] means that we cannot compare our results with those of studies such as the health promotion intervention study in a community in the Darwin Health Region, which showed that improving store food quality and fruit and vegetable intake increased the mean red cell folate level of Aboriginal adults.[24]

In summary, health parameters differed between peripubertal Aboriginal children living in suburban situations in Darwin–Palmerston and those living in remote areas of the Darwin Health Region. The urban group included an excess of both overweight and underweight children, while the remote group included a large excess of underweight children. Potential markers of adult disease also varied by location. Peripubertal children in remote areas have a substantial burden of infectious disease. Although the results from the Darwin urban area do not necessarily reflect the health status of Aboriginal children in other Australian cities, they do indicate that it is inappropriate to generalise findings from remote areas to urban areas.

COMPETING INTERESTS

ACKNOWLEDGEMENTS

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