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In an Aboriginal birth cohort, only child size and not birth size, predicts insulin and glucose concentrations in childhood☆

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Abstract

The objectives were to describe cross-sectional growth in 279 Australian Aboriginal children aged 8–14 years in order to test the hypothesis that birth size interacts with child size to predict glucose and insulin metabolism. Cross-sectional growth outcomes were described using standard deviation scores or *z*-scores for height for age (HAZ) and weight for age (WAZ) calculated from CDC 2000 reference values in Epi Info 2000.

Interrelationships were examined using standard regression models with current height and weight and birth weight, ponderal index and birth weight below the 10th percentile for gestational age. All models were adjusted for gestational age, gender and chronological age. Growth outcomes were poor with negative mean *z* scores for height and weight. Children with a birth weight $<$10th percentile for gestational age were significantly smaller and lighter than those with a birth weight $\geq$ 10th percentile for gestational age, indicating post-natal catch-up growth of small babies was unlikely.

After adjustment for childhood size, there was no relationship between any birth measures and fasting glucose or insulin concentrations. Current child height and weight had positive relationships with both fasting insulin and glucose concentrations with a greater proportional change for insulin. For every increment of 1 cm in height or 1 kg in weight, insulin concentrations rose 2% whereas glucose increased by only 0.2%. In this indigenous Australian cohort with poor post-natal growth, only current child size is related to measures of glucose and insulin metabolism.

Keywords: Australian Aboriginal; Glucose and insulin; Birth size; Current child size

1. Introduction

The prevalence of type 2 diabetes in Aboriginal Australians has been estimated to lie between 10 and 30% [2] with younger Aboriginal children being increasingly diagnosed with this condition [3]. The death rates associated with diabetes among Aboriginal Australians has recently been estimated to be 12–17 times greater than those for the non-indigenous Australian population [4].

The "fetal origins hypothesis" proposes that adaptations to undernourishment in fetal life lead to permanent changes which increase the risk of chronic disease in adult life. This hypothesis has been tested

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by follow-up studies relating birth size to later glucose and insulin metabolism. Most of these studies are retrospective and report an association between low birth weight (LBW) and an increase risk of type 2 diabetes or glucose intolerance in adults from populations of developed countries [5]. However, there are also studies that relate an increased risk of diabetes in later life to high birth weight [6,7].

Only a proportion of LBW results from fetal under nutrition. Better surrogates of fetal growth relate birth size to gestational age, but gestational age information is frequently missing in retrospective studies. The few prospective studies confined to childhood have been inconsistent in their findings [8–11]. More recent speculation suggests that the fetal adaptations in response to intrauterine conditions may only become apparent in later life after modification by particular childhood growth patterns [12].

For some decades the rates of LBW have been high in the Australian Aboriginal population and even now LBW rates are over double those of non-indigenous Australians [13]. From the 1960s, the infant mortality rate has declined in Aboriginals, owing to improved health services and living conditions, leading to more LBW Aboriginal babies surviving into adult life. This raises the possibility that the current type 2 diabetes epidemic in the Aboriginal population may have its origins in events occurring during fetal life.

Using prospectively collected data in an Australian Aboriginal birth cohort, we describe child growth outcomes, and examine the childhood relationships of glucose and insulin metabolism with birth and child size.

2. Methods

The recruitment and follow-up of this birth cohort has been previously published in detail [14]. In brief, 686 out of 1238 Aboriginal children born at the Royal Darwin Hospital between January 1987 and March 1990 were recruited into the study. Although the babies were not randomly selected there were no significant differences in the mean birth weight, birth weight frequencies or sex ratio between those recruited and not recruited. Midwives measured the birth weights and crown-heel lengths within 2 h of delivery. Birth weights were recorded to the nearest gram using a balance scale. The crown-heel lengths were measured with a length board by the standard anthropometric technique [15]. A single neonatal pediatrician performed gestational age assessment on each study participant according to the Dubowitz Scoring System [16] within 4 days of birth.

At follow-up between December 1998 and March 2001, 572 children were examined, 18 had died and one parent refused permission for her child to be examined. There were 63 children who were traced but could not be physically accessed at the time of follow-up and 32 children were not found. The researchers examining the children were blind to their perinatal outcomes. Children were measured in light clothing while barefoot. Height was measured to the nearest millimeter with a portable stadiometer. Weight was measured to the last complete 0.1 kg with a digital electronic scale (Tanita model TBF-521).

Children were asked to fast from midnight and blood samples were taken in the morning after application of anaesthetic cream (EMLA) to the venepuncture site.

The Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research, including the Aboriginal Ethical Sub-committee which has the power of veto, approved the study. Written consent was obtained from the caregivers of all children.

Blood samples were collected into fluoride oxalate tubes then kept on ice or refrigerated until they were centrifuged. Most were centrifuged within 4 h and none later than 8 h. After being centrifuged they were again refrigerated and then transported to the laboratory packed in Styrofoam containers with cold bricks. Plasma glucose concentration was measured on a Hitachi 917 autoanalyzer using hexokinase assay at the local laboratory. Plasma insulin was measured at a distant (Perth) laboratory using a two-site immuno-enzymometric assay performed by the TOSOH AIA-600 immunoanalyzer using ASIA-PAK with no cross-reactivity with proinsulin. Serum hemoglobin was measured on a Coulter Max M.

Cross-sectional growth outcomes in childhood were described using the standard deviation scores, or *z*-scores, for height for age (HAZ) and weight for age (WAZ), calculated from CDC 2000 reference values in Epi Info 2000 [17]. Positive and negative *z*-scores

mean growth for age is above or below the reference median, respectively.

Children were defined as fasting if they stated that they had fasted for 8 h or more. Of the 306 children meeting this criterion (including 5 seen in the afternoon), 279 children had complete data for gestational age and both fasting insulin and glucose values. This sub-set was not significantly different from the non-fasting children in the measures of birth size, gestational age, sex ratio, current age, WAZ, HAZ or serum hemoglobin.

Fasting insulin (Io) and fasting glucose (Go) concentrations were normalized using $\log_{10}$-transformation. Insulin resistance was estimated from fasting insulin and glucose concentrations using the homeostasis model assessment (HOMA-IR) equation [18]. Birth size and shape measures were birth weight and ponderal index $((\text{birth weight}/\text{birth length}^3) \times 100)$ [19], studied as continuous variables and birth weight for gestational age dichotomized at the 10th percentile of weight for gestational age [20]. Child size was studied as continuous variables of height (cm) and weight (kg). The relationships of Io, Go and HOMA-IR to the birth measures and child size variables, were analyzed in a series of standard regression models [21] adjusting for gestational age, gender and chronological age using Stata 7 [22]. Table 1 shows the expected significant relationships between the outcome variables (Io, Go and HOMA-IR) and the explanatory variables of birth measures and child size if the fetal origins hypothesis is supported. Each of the outcome variables was tested in separate univariate models for each of the birth measures (Table 1, early). Because the best method of describing child size for this hypothesis is not clear, we ran separate sets of models adding either current childhood weight or height to the models (Table 1, combined). Then the interaction term between the birth measures and the child weight or height was included (Table 1, interaction). These interaction terms were also tested by the best likelihood ratio test. Lastly, each of the outcome variables was tested in a separate univariate model with the current child weight or height (Table 1, late).

These analyses assume that there is a linear relationship with birth size. However, there may be a U-shaped relationship with both small and large infants being at higher risk than those of intermediate size. In this case, no association may be evident under a linear assumption. To investigate this possibility, we examined the residuals from the regression models for evidence of patterns and also redid the models after dividing birth weight into quartiles and entering it as an indicator variable.

Because insulin and glucose were log-transformed, the expotentiated coefficients present the effect as a proportional change in insulin and glucose level per unit increment in birth or childhood measures.

Table 1
Standard regression models with expected direction of significant associations of fasting concentrations of insulin and glucose and HOMA-IR values with birth size and child size if fetal origins hypothesis supported [21]

Model	Birth outcome	Child outcome	Birth outcome × child outcome
Early	Negative		
Combined	Negative	Positive	
Interaction	Negative	Positive	Negative
Late		Positive	

3. Results

Of the 279 children with gestational age information and both fasting insulin and glucose values there were 143 boys and 136 girls aged 8.9–13.8 years. Table 2 shows their birth and child characteristics. At a mean age of 11.4 years, there were significant differences for BMI, HAZ and WAZ between children whose birth weight was <10th percentile for gestational age and sex (small for gestational age, SGA) and those with birth weight ≥ 10th percentile for gestational age (appropriate for gestational age, AGA) (Table 3).

There was no relationship between any of the birth measures examined and any of the current childhood fasting insulin or glucose concentrations and HOMA-IR values, even after adjustment for current height or weight, nor were there any significant interactions between birth measures and current child size (Tables 4 and 5, HOMA-IR interaction models not shown). However, there were significant positive relationships between both current height and weight and all of the outcomes in the univariate (late) models with the proportional changes being greatest for the insulin and HOMA-IR values and relatively trivial for glucose. For every kilogram increment in weight

Table 2
Birth and childhood characteristics of 279 fasting Aboriginal children

Characteristics	No.	Total	Boys (143)	Girls (136)
Birth data				
Birth weight (g)	279	3069 ± 615	3139 ± 648	2994 ± 570
Gestational age (weeks)	279	38.9 ± 1.7	38.9 ± 1.7	38.8 ± 1.5
Ponderal index	273	2.6 ± 0.27	2.6 ± 0.27	2.6 ± 0.28
% low birth weight (<2500 g)	279	15.4	16.8	14
% birth weight <10th (SGA)	279	24.7	26.7	22.8
Child data				
Age at follow-up (years)	279	11.4 ± 1.2	11.6 ± 1.2	11.2 ± 1.2
% commenced puberty	279	50	36	63
Weight (kg)	279	35.9 ± 12.3	35.6 ± 12.3	36.2 ± 12.4
Height (cm)	279	143.5 ± 10.9	143.9 ± 10.7	143.1 ± 11
BMI (kg/m^2)	279	17.0 ± 3.6	16.8 ± 3.6	17.2 ± 3.7
Weight for age *z* score (WAZ)	279	−0.8	−0.9	−0.7
Height for age *z* score (HAZ)	279	−0.5	−0.5	−0.4
Fasting insulin (μU/l)	279	9.0 ± 11.4	7.4 ± 7.0	10.8 ± 14
Fasting glucose (mmol/l)	279	4.5 ± 0.6	4.5 ± 0.6	4.5 ± 0.7
HOMA-IR	279	1.9 ± 3	1.5 ± 1.6	2.3 ± 4.1

Table 3
Growth outcomes of fasting appropriate for gestational age and small for gestational age Aboriginal children[a]

Characteristics	AGA ($n = 210$)	SGA ($n = 69$)	*P*
Age, years (mean ± S.D.)	11.3 ± 1.2	11.7 ± 1.1	0.02
% male	50	55	0.46
% commenced puberty	49.5	50	0.94
Body mass index (kg/m^2) (mean ± S.D.)	17.5 ± 3.9	15.7 ± 2.3	<0.001
Weight for age *z* score (mean ± S.D.)	−0.6 ± 1.4	−1.4 ± 1.2	<0.001
Height for age *z* score (mean ± S.D.)	−0.4 ± 1.1	−0.8 ± 1.1	0.006

[a] AGA: birth weight equal or above the 10th percentile for gestational age; SGA: birth weight below the 10th percentile for gestational age [21].

Table 4
Size at birth and size in childhood: relations with fasting insulin concentrations (μU/l) in childhood

Model	Variables	Birth size variables			Child size variables		
		Ratio	95% CI	*P*	Ratio	95% CI	*P*
Early (birth size)	BW (per 500 g)	1.04	1.0–1.1	0.07			
Combined	With adjustment for child height	1.0	0.96–1.05	0.9	1.01	1.01–1.02	<0.001
Combined	With adjustment for child weight	0.97	0.93–1.01	0.2	1.02	1.01–1.02	<0.001
Early (birth size)	Ponderal index[a] (per ratio unit)	1.08	0.88–1.25	0.6			
Combined	With adjustment for child height	1.04	0.9–1.28	0.4	1.01	1.01–1.02	<0.001
Combined	With adjustment for child weight	0.97	0.83–1.1	0.7	1.02	1.01–1.02	<0.001
Early (birth size)	SGA vs. AGA[b]	0.94	0.84–1.04	0.2			
Combined	With adjustment for child height	0.97	0.89–1.08	0.7	1.01	1.01–1.02	<0.001
Combined	With adjustment for child weight	1.03	0.94–1.14	0.5	1.02	1.01–1.02	<0.001
Late (child size)	Current height per (cm)				1.02	1.01–1.02	<0.001
Late (child size)	Current weight (per kg)				1.02	1.01–1.02	<0.001

All models adjusted for gestational age, sex and chronological age.

[a] Ponderal index = (g/cm^3) × 100.

[b] SGA: birth weight below the 10th percentile for gestational age; AGA: birth weight equal or above the 10th percentile for gestational age [21].

Table 5
Size at birth and size in childhood: relations with fasting glucose concentrations (mmol/l) in childhood

Model	Variables	Birth size variables			Child size variables		
		Ratio	95% CI	*P*	Ratio	95% CI	*P*
Early (birth size)	BW (BW per 500 g)	1.002	0.99–1.01	0.7			
Combined	With adjustment for child height	1.0	1.0–1.01	0.5	1.0	1.001–1.003	0.002
Combined	With adjustment for child weight	0.99	0.99–1.004	0.3	1.001	1.001–1.002	<0.001
Early (birth size)	Ponderal index[a] (per ratio unit)	1.0	0.97–1.03	0.9			
Combined	With adjustment for child height	1.0	0.97–1.03	0.9	1.0	1.001–1.002	0.003
Combined	With adjustment for child weight	1.0	0.96–1.02	0.5	1.001	1.001–1.002	0.001
Early (birth size)	SGA vs. AGA[b]	0.99	0.97–1.01	0.3			
Combined	With adjustment for child height	1.0	0.98–1.02	0.9	1.001	1.001–1.002	0.002
Combined	With adjustment for child weight	1.0	0.98–1.02	0.8	1.001	1.0–1.002	0.002
Late (child size)	Current height (per cm)				1.0	1.001–1.002	0.003
Late (child size)	Current weight (per kg)				1.0	1.001–1.002	0.001

All models adjusted for gestational age, sex and chronological age.
[a] Ponderal index = $(g/cm^3) \times 100$.
[b] SGA: birth weight below the 10th percentile for gestational age; AGA: birth weight equal or above the 10th percentile for gestational age [21].

or centimeter increment in height, fasting insulin rose by a factor of 1.02 or 2% (late models, Table 4) and fasting glucose rose by a factor of 1.001 or 0.1% (late models, Table 5) HOMA-IR also had a significant positive relationship with child size, with an increase of 2% (95% CI: 1.01–1.02, $P =< 0.001$) for every kilogram increment of current weight or every centimeter change in current child height.

There was no indication that the linear assumption in the models was not appropriate and that a U-shape existed. For example, in the univariate analysis containing Io and quartiles of birth weight, the levels of insulin were 6, 0.4 and 5% higher (overall $P = 0.7$) in the second, third and highest quartile compared to the lowest quartile.

4. Discussion

This prospective Aboriginal birth cohort is an opportunity to test hypotheses about the effect of intrauterine environment on measures of glucose and insulin metabolism in childhood. In this population in childhood, neither birth size nor shape were associated with fasting insulin or glucose concentrations and insulin resistance but there were direct relationships of current child size with fasting insulin, glucose and insulin resistance.

These findings are similar to two studies of contemporary British children from populations where low birth weight (LBW) is uncommon [8,9]. One study based on maternal recall of birth weight found current size was the main determinant of insulin and glucose concentrations in childhood although there was a small inverse relationship between fasting insulin and birth weight [8]. The other, a non-intervention prospective study of Plymouth children also showed insulin resistance was a function of current weight rather than low birth weight [9]. However, our findings and the British ones are in contrast to two studies from populations where LBW is common. Among 8-year-old Indian children (mean birth weight 2.75 kg), higher insulin resistance was seen in those who were small at birth and had grown "big" compared to others in their cohort [10]. For 7-year-old South African children (mean birth weight 2.75 kg), those with more rapid growth velocities had the highest measures of insulin resistance [11]. These studies are consistent with the speculation that the fetal adaptations may only become apparent in later life after modification by particular childhood growth patterns responding to the external environment [12]. The rapid weight gain of the small babies in the Indian study and the increased post-natal growth velocity in the South African subjects may be due to improved childhood environments relative to that of their fetal life. However, the

Plymouth study with a 2.5% LBW suggests the starting point of the growth trajectory may be important as there was no difference in the insulin resistance between the low birth weight/high current weight children and the high birth weight/high current weight children.

In this Aboriginal birth cohort with 13.5% LBW, growth outcomes are poor with negative mean *z* scores for both height and weight (Table 2). At a mean age of 11.4 years, there are significant weight, height and BMI differentials between the SGA and AGA children (Table 3), which means it is unlikely that babies who were smaller at birth have grown relatively more than others in the cohort. For these children it is likely that the undernourishment of fetal life due in part to maternal smoking and under nutrition in this cohort [23], has not markedly improved post-natally resulting in the prenatal growth trajectory remaining unchanged. These children are still relatively undersized and the failure of catch-up growth may explain why only current size relates to measures of glucose and insulin control in our group. On the other hand current weight may well be the only determinant of glucose and insulin metabolism, with catch-up growth representing overfeeding leading to later obesity [11].

Given the high prevalence of overweight and obesity currently seen in Aboriginal adults [24], subsequent follow-up with sequential, multiple measures of height and weight will give us ample opportunity to determine if and when the effects of poor intrauterine growth are modified by post-natal environmental factors. The relationships we have described in childhood may change with the development of adult obesity and the effects of fetal programming may only become important once obesity is expressed.

In the meantime our findings confirm that measures of glucose and insulin metabolism cannot be determined solely by intrauterine events. This shifts the focus from fetal growth to post-natal growth and implies that the current high rates of type 2 diabetes seen in the adults of this indigenous population may have the potential to be decreased by behavioral interventions in such settings as preschool and school particularly if the wider issues of community control and ownership are properly addressed [25].

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References

[1] S. Sayers, A. Reid, D. Mackerras, Current childhood size not birth size is related to childhood fasting plasma insulin and glucose concentrations in Australian Aboriginal children aged 8–14 years, Diabetes Res. Clin. Pract. Suppl. 57 (2002) S30.
[2] M. de Courten, A. Hodge, G. Dowse, I. King, J. Vickery, P. Zimmet, Review of the Epidemiology, Aetiology, Pathogenesis and Preventability of Diabetes in Aboriginal and Torres Strait Islander populations, OATSIH & DHFS, Canberra, 1998.
[3] B. Braun, M.B. Zimmermann, N. Kretchmer, R.M. Spargo, R.M. Smith, M. Gracey, Risk factors for diabetes and cardiovascular disease in young Australian Aborigines, Diabetes Care 19 (1996) 472–479.
[4] J. Cunningham, J.R. Condon, Premature mortality in Aboriginal adults in the Northern Territory, 1979–1991, Med. J. Aust. 165 (1996) 309–312.
[5] C.A. Newsome, A.W. Shiell, C.H.D. Fall, D.I.W. Phillips, R. Shier, C.M. Law, Is birth weight related to later glucose and insulin metabolism?: a systematic review, Diabetes Med. 20 (2003) 339–348.
[6] D. Dabelea, D.J. Pettit, R.L. Hanson, G. Imperatore, P.H. Bennett, W.C. Know, Birth weight, type 2 diabetes, and insulin resistance in Pima Indian children and young adults, Diabetes Care 6 (1999) 944–950.
[7] J.N. Wei, F.C. Sung, C.Y. Li, C.H. Chang, R.S. Lin, C.C. Lin, et al., Low birth weight and high birth weight infants are both at an increased risk to have type 2 diabetes among schoolchildren in Taiwan, Diabetes Care 2 (2003) 343–348.
[8] P.H. Whincup, D.G. Cook, F. Adshead, S.J.C. Taylor, M. Walker, O. Papacosta, et al., Childhood size is more strongly related than size at birth to glucose and insulin levels in 10–11-year-old children, Diabetologia 40 (1997) 319–326.
[9] T.J. Wilkin, B.S. Metcalf, M.M. Murphy, J. Kirkby, A.N. Jeffery, L.D. Voss, The relative contributions of birth weight, weight change, and current weight to insulin resistance in contemporary 5 year olds. The Early Bird Study, Diabetes 51 (2002) 3468–3471.

[10] C. Yajnik, Interactions of perturbations in intrauterine growth and growth during childhood on the risk of adult onset disease, Proc. Nutr. Soc. 59 (2000) 257–265.
[11] N.J. Crowther, N. Cameron, J. Trusler, I.P. Gray, Association between poor glucose tolerance and rapid postnatal weight gain in seven year old children, Diabetologia 41 (1998) 1163–1167.
[12] T. Forsen, J. Eriksson, J. Tuomilehto, A. Reunanen, C. Osmond, D. Barker, The fetal and childhood growth of persons who develop Type 2 diabetes, Ann. Intern. Med. 133 (2000) 176–182.
[13] N. Nasser, E.A. Sullivan, Australia's mothers and babies 1999, AIHW Cat. No. PER 19, Perinatal Statistics Series no. 11, Australian National Perinatal Statistics Unit, Sydney, 2001.
[14] S.M. Sayers, D. Mackerras, G. Singh, I. Bucens, K.A. Flynn, A. Reid, An Australian Aboriginal birth cohort: a unique resource for a life course study of an indigenous population. A study protocol, BMC Int. Health Hum. Rights 3 (2003) 1, http//www.biomedcentral.com/1472-698X/3/1.
[15] D.B. Jellife, The assessment of the nutritional status of the community, Monograph No. 53, WHO, Geneva, 1966, 1997 pp.
[16] L.M.S. Dubowitz, V. Dubowitz, A clinical manual: gestational age of the newborn, Addison-Wesley, Philippines.
[17] A.G. Dean, T.G. Arner, S. Sangam, G.G. Sunki, R. Friedman, M. Lanting, et al., Epi Info 2000, A database and statistical program for public health professionals for use on Windows 95, 98, NT and 2000 computers, Centers for Disease Control and Prevention Atlanta, GA, USA, 2000.
[18] D.R. Matthews, J.P. Hosker, A.S. Rudenski, B.A. Naylor, D.F. Treacher, et al., Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man, Diabetologia 28 (1985) 412–419.
[19] H. Miller, K. Hassanein, Diagnosis of impaired fetal growth in newborn infants, Pediatrics 48 (1971) 511–522.
[20] R. Guaran, P. Wein, M. Sheedy, J. Walstab, N.A. Bleischer, Update of growth percentiles for infants born in an Australian population, Aust. NZ J. Obstet. Gynaecol. 4 (1994) 39–49.
[21] A. Lucas, M.S. Fewtrell, T.J. Cole, Fetal origins of adult disease revisited, BMJ 319 (1999) 245–249.
[22] StataCorp Stata Statistical Software: Release 7.0, Stata Corporation, College Station, TX, 2001.
[23] S. Sayers, J. Powers, Risk factors for Aboriginal low birthweight, intrauterine growth retardation and preterm births in the Darwin Health Region, Aust. NZ J. Public Health 121 (1997) 524–530.
[24] J. Cunningham, D. Mackerras, Overweight and obesity, indigenous Australians 1994, ABS Catalogue No. 4702.0, Australian Bureau of Statistics, Canberra, 1998.
[25] K.G. Rowley, M. Daniel, K. Skinner, M. Skinner, G.A. White, K. O'Dea, Effectiveness of a community-directed "healthy lifestyle" program in a remote Australian Aboriginal community, Aust. NZ J. Public Health 24 (2000) 136–144.