Childhood growth and chronic disease: evidence from countries undergoing the nutrition transition

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Abstract

Evidence is accumulating that the pattern of growth in childhood is associated with development of cardiovascular disease in adulthood, but such evidence comes mostly from developed countries. We conducted a review of studies from countries undergoing the nutrition transition. Five birth cohorts with measures of child growth and outcomes through adolescence were identified, from China, India, Guatemala, Brazil and the Philippines. Across studies there are major differences in data availability and in statistical approaches to modelling child growth and its effects. Nevertheless, generally consistent associations of growth failure in early childhood and development of overweight in later childhood with the risk of elevated blood pressure, glucose, and serum lipids in adulthood were observed. As these cohorts mature they will provide a wealth of critical information on the relation between early life factors and later disease risk, and efforts should be made to ensure ongoing follow-up using standardized approaches and more comprehensive assessments.

Keywords: birth cohorts, child growth, developing countries, life course, prospective studies.

Introduction

Prospective longitudinal studies provide strong data to test hypotheses related to the development of chronic disease across the life course. Birth cohorts

are especially valuable in testing hypotheses related to the concept of fetal programming of adult disease (Barker, 1998) and to investigation of interactions among fetal and postnatal exposures. To date, most such research has been conducted in industrialized countries, with major birth cohorts available from Hertfordshire England (Barker, 1998; Parsons *et al.*, 2001; Kuh *et al.*, 2002) and Helsinki, Finland (Eriksson *et al.*, 2002). Additional cohorts in which childhood exposures are recalled (rather than measured) are available from the United States (e.g.

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Curhan *et al.*, 1996) and elsewhere. Taken as a whole, and as reviewed elsewhere in this volume, these studies suggest that growth failure in the period from conception to age 2 years and accelerated growth in the period from childhood to adolescence are associated with increased risk of diabetes and cardiovascular disease and their metabolic underpinnings.

Identification and establishment of birth cohorts in developed countries has been facilitated by the widespread availability of birth records and, occasionally, the ability to link these to records of growth as monitored in clinic- and school-based surveillance programmes. Such programmes are rare in countries undergoing the nutrition transition where infrastructure and resources are limiting and a large proportion of births occur in homes. Countries in transition are characterized by widespread undernutrition and growth faltering. This is evident at birth, with mean birthweights often 0.5–1.5 standard deviations lower than those of western countries, and the degree of growth failure increases over the first years of postnatal life (Bustos *et al.*, 2001; Stein *et al.*, 2004). Poor growth is related to increased child morbidity (Victora *et al.*, 2001) and mortality (Caulfield *et al.*, 2004), and is therefore a concern for short-term outcomes. To the extent that size at birth and postnatal growth both relate to the development of chronic disease among survivors to adulthood, studies in transitional countries may be especially informative. The purpose of this review is to summarize the database currently available of prospective birth cohort studies where the cohort members have reached adulthood.

Methods

We searched the published literature for cohorts that met the following criteria: cohort inception at birth or during gestation, some follow-up during childhood and current age (in 2004) of 18 or greater. Five cohorts were identified, from Hong Kong, China, South Delhi, India, Pelotas, Brazil, Cebu, the Philippines and Oriente, Guatemala. Cohorts in Beijing, China (Mi *et al.*, 2000) and Mysore, India (Stein *et al.*, 1996), in which birth data and outcome data in adulthood are available but no intermediate measures were obtained, and a cohort in Kingston,

Jamaica (Gaskin & Walker, 2003), which was recruited in early childhood and followed through adolescence, were excluded.

We reviewed the publications derived from these cohorts to ascertain commonalities and differences in data collection protocols and findings related to the association between postnatal growth and its interaction with birth size on risk factors for cardiovascular disease.

Results

Data collection during childhood for the five cohorts is presented in Fig. 1. Three of the cohorts (Guatemala, Cebu, South Delhi) were identified prenatally and therefore some data are available on prenatal experience, while two cohorts (Hong Kong and Pelotas) were established as hospital-based birth series. Data collection was generally most intensive in the first years of life. A summary of key outcomes that have been related to postnatal growth is presented in Table 1.

Hong Kong, China

The Hong Kong birth cohort (Cheung *et al.*, 2000) included 523 ethnic Chinese infants born in one of

Fig. 1. Data collection through age 20 years in five birth cohort studies, by month of age. Each mark denotes a scheduled data collection point, although not all variables were collected at each age, and some data collection instruments were only administered to a sample of the original birth cohort. In the Oriente, Guatemala study, there was a single wave of data collection in 1988–89, at which time the birth cohort was 11–20 years old. See text for sources.

Table 1. Selected characteristics of identified birth cohorts

City, country	Birth years	Size of cohort at inception	Age at most recent published outcome assessment (years)	Sample size at most recent published outcome assessment N (% of birth cohort)	Published outcomes
Hong Kong, China	1967	523	30	133 (25%)	Blood pressure
South Delhi, India	1969–72	8181	27–32	1583 (19%)	Glucose
Oriente, Guatemala	1969–77	1301	21–29	473 (36%)	Body composition Glucose
Pelotas, Brazil	1982	5914	18	2250 males (79%) 1030 of a target 27% sample of 1749 (69%)	Blood pressure Body size and composition
Cebu, the Philippines	1983–84	3080	14–16	2089 (68%)	Blood pressure

two hospitals in Hong Kong between 24 February and 1 April 1967. Weight and length were measured monthly from birth through the first year, quarterly in years 2 and 3, and then at ages 4 and 5. No subsequent child measurements were obtained. The cohort subjects were followed up at age 30 and the 171 who could be located in Hong Kong were invited for study. In all, 133 (25.4% of the birth cohort) attended a clinical investigation session in 1997 at age 30 years. There were no differences in birth length or weight between responders and those lost to follow-up.

Based on 122 individuals with complete data, relations between child growth and adult systolic and diastolic blood pressure (DBP) have been published. The authors focused on measures of length, weight and ponderal index at birth, 6 months and 18 months, and on the change in these measures in the interval 0–6 months and 6–18 months, all expressed as standard deviation scores. Birth length standard deviation scores were inversely associated with DBP at age 30, but no other measures of anthropometric status or change were associated with DBP. Ponderal index at birth and postnatal changes in ponderal index from age 6 to 18 months were significantly and inversely associated with systolic blood pressure (SBP), with the change in ponderal index from 0 to 6 months being marginally significant.

This study is based on small numbers, and hence lacks power to detect modest effects. Furthermore the various measures entered into a single model raise statistical concerns of collinearity. Finally, out-

comes related to other risk factors for cardiovascular disease have not been published.

South Delhi, India

The South Delhi cohort (Bhargava *et al.*, 2004) was established by enrolling pregnant women between 1 December 1969 and 30 November 1972, and consisted of 8181 live births. The children were assessed every 3 months in the first year of life, and at 6-month intervals thereafter. Much of the cohort was lost to study following the demolition of unauthorized housing during the project period. Follow-up activities were conducted starting in 1998. A total of 2584 members of the cohort were located, of whom 1583 (19.3% of the birth cohort) agreed to participate in a clinical investigation. This examination focused on anthropometry, blood pressure and glucose metabolism (including a standard glucose tolerance test). After exclusions, analyses were reported on 1442 individuals aged 26–32 years.

At examination (mean age 30 years), 15.2% of the cohort already showed evidence of impaired glucose tolerance or diabetes. Both fasting and 120-min glucose concentrations showed the expected associations with adult age, body mass index (BMI) and waist-to-hip ratio. An inverse relationship was observed between 120-min plasma glucose concentrations and birthweight, ranging from 113 mg dL⁻¹ in those whose birthweight was 2.25 kg or less to 105 mg dL⁻¹ in those with birthweight greater than 3.5 kg. Similar

results were found for fasting insulin concentrations, 120-min insulin concentration and insulin resistance.

Development of impaired glucose tolerance (IGT) and diabetes was inversely related to both weight and BMI at 1 year of age. With respect to childhood growth, the highest prevalence of IGT and diabetes was among those who were in the lowest third of the group for BMI at age 2 years and the highest tertile at age 12 years. Both effects were significant in multiple regression models, suggesting the adverse effects of a pattern of early life growth failure followed by early adiposity rebound and development of overweight by adolescence.

This study is characterized by a relatively large size, very intensive data collection throughout childhood and a wide range of outcomes measured at mean age 29 years. Data on associations with blood pressure, serum lipids and other factors have not been published.

Oriente, Guatemala

Unique among the five cohorts reported here, the Guatemala study is a follow-up of participants in a supplementation trial begun in 1969 (Martorell *et al.*, 1995). Four villages were randomized to receive either Atole, a nutrient-rich gruel, or Fresco, a sweetened, protein-free soft drink. The supplement was available twice each day in a central location in the village, and intake was recorded for pregnant and lactating women and for children under 7 years of age. Extensive data were collected on the mothers ($n = 517$) and their children ($n = 1301$ born between 1969 and 1977). There was one cross-sectional wave of follow-up data collection in 1988, when the birth cohort was 11–18 years of age, and additional data collection has been conducted in the study villages since 1991. A survey of cardiovascular disease risk factors was conducted in 1997–98 among 473 members of the cohort born between 1969 and 1976, and hence who had available data on birthweight and growth through 12 months, who lived in one of the four villages or in Guatemala city (Torun *et al.*, 2002). Other out-migrants were not studied, and response rates are estimated at 60% of cohort members known to be alive. A fasting capillary sample was obtained

and analysed for lipids and glucose, anthropometry and blood pressure were measured, and additional data were obtained by interview.

The cohort (mean age 24.3 years at examination) is characterized by moderate levels of overweight and high prevalence of low HDL-cholesterol levels (Stein *et al.*, 2002). Size at birth was not related to the prevalence of clustering of four risk factors for the metabolic syndrome (SBP, HDL-cholesterol, triglycerides, glucose), while adult BMI was strongly related to this clustering. That study did not consider effects of postnatal growth. To date, two analyses that did consider growth to age 24 months have been published (Li *et al.*, 2003; Conlisk *et al.*, 2004), with others presented in abstract form (Webb *et al.*, 2004; Graff *et al.*, 2004). With respect to body composition in adulthood, Li *et al.* (2003) showed that increased length in infancy and increase change in length were independently associated with increased height and fat-free mass in adulthood, but not with increased fat mass. Conlisk *et al.* (2004) showed no main effect of size at birth or of postnatal growth on fasting glucose levels, but were able to demonstrate an interaction between these two measures, such that among children born small, above-average growth in length to 24 months was associated with decreased fasting glucose levels. Similar effects on LDL-Cholesterol and on blood pressure have been noted among recipients of Atole (Graff *et al.*, 2004; Webb *et al.*, 2004). To date, no data have been published on growth in later childhood and its association with cardiovascular disease risk.

Among the five studies reviewed here, this study is unique in several respects. First, it has an intervention design, with the intervention implemented as a community randomized trial. Second, it is the only study conducted in an exclusively rural birth cohort (the Cebu study included both rural and urban communities). Third, it is the only study in which out-migrants have been systematically studied, enhancing the ability to test for interactions of the child and adult environment as potential determinants of changes in body size and cardiovascular disease risk (Torun *et al.*, 2001). Finally, this is the only study in which two waves of data have been collected in adulthood. The current data collection wave, completed in July 2004, has involved even more extensive tracing efforts and

has resulted in slightly larger sample sizes. It will facilitate testing the consistency of interactions observed in the 1997–98 follow-up. Furthermore, as risk factor prevalence tends to increase with age, power to detect associations will increase overall. That said, however, the cohort is limited by a relatively small sample size, limiting power to test for differences in effect sizes between men and women, or between intervention treatment arms. Another limitation is that decreasing numbers of individuals provide data for measures of postnatal growth to increased ages, as the original study was terminated in September 1977. The 1988–89 data collection wave includes individuals at a wide range of ages, including both prepubertal and postpubertal participants, further complicating analyses that might include these data.

Pelotas, Brazil

This population-based birth cohort (Victora *et al.*, 2003) was identified and recruited through the hospitals where 99% of all deliveries in Pelotas occur. The inception cohort consists of 5914 live-born hospital births in 1982. Of the five cohorts identified, this study has the fewest measures of early childhood growth, consisting of two rounds of data collection at mean ages 19 month and 43 month, respectively. Male cohort members were studied at military induction in 2000 ($n = 2250$, mean age 18.2 y), and a systematic 27% sample of the cohort were further studied in 2001, at mean age 18.9 years. The study investigators estimate that 30% of the cohort has been lost to follow-up. At the most recent follow-up slightly more than 1000 individuals were interviewed, and assessed for anthropometry and blood pressure.

In this cohort, rapid growth between birth and 43 month was associated with overweight/obesity at 16 years, with the effects stronger in boys than in girls, and stronger when expressed as measures of weight for age or height than when expressed as growth in height alone (Monteiro *et al.*, 2003). Birth weight was inversely associated with blood pressure at age 20 years, while weight for age z-score at age 15 years was positively associated with blood pressure levels. Patterns were less evident for DBP (Horta *et al.*, 2003). Data on associations between

child growth and glucose or lipid metabolism have not been published.

This cohort is characterized by a large sample base and is representative of births in the city. However, the large sample size has been compromised by the sampling approach taken during follow-up waves, drastically reducing sample size. Furthermore, only limited data points are available in childhood with which to model child growth.

Cebu, Phillipines

The Cebu cohort (Adair & Cole, 2003) is population-based, representing births to pregnant women in 33 communities in metropolitan Cebu. The cohort was established in gestation by screening of women in these communities during 1983–84. Children were studied every 2 months during the first 2 years of life, and then at mean age 8.5 years, 11.5 years and 15.5 years. Of 3080 singleton live births recorded, 2089 (67.8%) were successfully traced in the 1998–99 follow-up, at age 15.5 years. Anthropometry and blood pressure were measured in all cohort members, and lipid levels were obtained on a subset (Kuzawa & Adair, 2003).

Among boys, there was no relationship between SBP or DBP and birth weight in unadjusted analyses. However, after controlling for birth length, current BMI, age and height, there was an inverse relationship between odds of high blood pressure and birthweight. In boys, increased weight gain from 0 to 2 year was associated with lower blood pressure, while there was a significant interaction between low birthweight and high weight gain, either between 8 and 11 years or between 11 and 15 year. In girls, there was no association between any measure of growth and blood pressure. However, girls had increased odds of high blood pressure if they had low birth BMI and high adolescent BMI compared with either those with low birth and low adolescent BMI or those girls with high birth and high adolescent BMI. Odds of high blood pressure in girls were primarily associated with larger growth increments from 8 to 16 year of age, independent of size at birth.

This study benefits from prenatal identification, from its large size and social and geographical gener-

alizability to the population of Cebu. It is the youngest of the cohorts reported here, and no data have yet been reported for the cohort as adults. Hence it is unclear if the relationships observed are in some way attributable in the short term by the processes of puberty. Future data collection rounds should aid in clarification of this point. No biochemical measures were obtained in the 1998 follow-up wave.

Discussion

We have identified five prospective birth cohort studies in which the cohort members are, in 2004, now adults, ranging in age from 37 years (Hong Kong) to 22 years (Cebu and Pelotas). Asia and the Americas are represented. Four of the studies recruited over 1000 individuals, the Hong Kong study being the exception. The population under follow-up also ranges considerably, from a mere 122 in Hong Kong to over 2000 who participated in the most recent data collection rounds in Cebu and Pelotas. Losses to follow-up have been considerable in Hong Kong and in South Delhi, with fewer than one quarter of the birth cohort being available for investigation as adults. The Guatemala cohort has shown very high trace rates, with fewer than 10% of the cohort being lost to follow-up, and response rates of 80% to the 2002–04 round of the cohort known to be alive and living in Guatemala. The Cebu and Pelotas cohort investigators have not published results of data collection among an adult population – it is likely that tracing and ensuring response rates will become progressively more challenging as the cohorts mature.

In general, the studies have shown that growth failure in early childhood and increased weight, whether expressed as weight, BMI, or weight for height, over later childhood, are associated with increased prevalence of risk factors for cardiovascular disease in late adolescence or early adult life. Taken as a whole, this is suggested for blood pressure (two studies), glucose metabolism (one study) and body composition (two studies). There is little evidence that increases in height alone are associated with any increased risk, indeed, there is evidence of null or even a protective effect of accelerated increases in height, especially among those born small, from Guatemala. Overall,

these data, which are broadly consistent with results from developed countries, suggests that intervention to increase height but not excessive weight might be health-promoting with respect to cardiovascular disease and its risk factors.

To date, there has been no attempt to conduct pooled analyses of the data from these studies. Such pooled analyses will enhance study power to assess the different effects of size at birth, growth in early childhood and growth in later childhood and adolescence. To do so would be challenging, given the variation in child measures available and adult outcomes studied, as well as differences in the ages at which these outcomes were measured, and the statistical approaches used to describe period of childhood and to model the associations between growth in these periods and adult outcomes. In some studies, exposure and outcome data were only collected on subsets of the population, further affecting available sample sizes, and in Guatemala, only capillary blood samples were obtained, precluding analyses of insulin status or of conducting glucose tolerance tests. At this time, data available across all studies appear to include anthropometry and blood pressure. Finally, the Guatemala study involved a community-level randomized trial of supplementation in childhood, while all the other studies were purely observational. Future rounds of follow-up in these cohorts should be developed to study the development of disease risk as these cohorts mature, and should be coordinated to ensure that outcome data are comparable across cohorts. A more comprehensive assessment of disease risk, including measures of glucose metabolism and endothelial function, is required. Addition of such measures will be relatively expensive, requiring the identification of research funds for this important purpose.

Several newer birth cohorts (e.g. Cameron *et al.*, 1998; Yajnik *et al.*, 2002) have been established in the past 15 years. These studies have expanded the geographic coverage of studies being conducted in transitional societies, with representation from Africa, India, and South and Central America. As these studies mature, investigators will face many of the same challenges of attrition via migration and non-response. We suggest that these study investigators

build in mechanisms to ensure feasibility of long-term follow-up. Here again, coordination across the studies at the planning stages will enhance generalizability, ease of later pooling of data for multi-study analyses and study power.

In conclusion, evidence is mounting for a coherent picture of associations between childhood exposures and risk of development of an adverse cardiovascular disease risk profile. These studies need to be sustained to facilitate study over the life course of the cohort members, and a network of such studies should be supported.

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