

# Weight gain in childhood and body composition at 18 years of age in Brazilian males

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## Abstract

**Aim:** To assess the association between weight gain at different time periods during childhood and measures of adiposity in late adolescence.

**Methods:** A population-based birth cohort carried out in Pelotas, a 320 000-inhabitant city in a relatively developed area in Southern Brazil. All newborns in the city's hospitals were enrolled in 1982. Weight gain from 0–1, 1–2, 2–4 and 4–15 years were expressed as changes in weight-for-age Z-scores relative to the NCHS/WHO reference. In 2000, 79% (2250) of all males were located when enrolling at the national Army. Weight and height were assessed. Body composition indicators (fat mass/height<sup>2</sup>, lean mass/height<sup>2</sup>, fat mass/lean mass<sup>2.3</sup>) were estimated through bioimpedance. Analyses were adjusted for maternal and social factors, as well as for gestational age.

**Results:** In the adjusted analyses, birthweight and weight gain in the first year of life were positively associated with attained height at age 18 years. Except for the fat mass/lean mass<sup>2.3</sup> ratio, all weight-related outcomes were positively associated with weight gain in different periods of life. Children who gained weight rapidly in more than one time period became fatter at age 18 years, independently of when fast growth took place.

**Conclusions:** Height was primarily determined by fetal and infant growth. Weight-related indices, including the fat/lean mass ratio, were more strongly influenced by later growth. No clear critical windows of growth during which absolute tissue masses are programmed could be identified.

## INTRODUCTION

Interest in the early determinants of chronic disease has led to concern that rapid weight gain in childhood may increase the risk of adult overweight and obesity. A recent systematic review confirmed that attained body size and growth during the first 2 years of life places children at an increased risk of subsequent obesity (1).

The data available from most studies on this topic did not allow the comparison of the effects of rapid growth in different periods during childhood. Also, the outcome used in most studies is the body mass index (BMI), which fails to discriminate between fat and lean body mass (used in this paper synonymously with fat-free mass).

In an earlier analysis of a cohort of Brazilian children born in 1993 (2), we showed that birthweight and weight gain in the first 6 months of life were associated with attained lean body mass, but not with fatness, measured at the age of 9 years. Weight gain between 1 and 4 years was associated both with later fat mass and lean mass, whereas weight gain 4–9 years was strongly associated with fat mass but not lean mass. We concluded that the association between early rapid weight gain and later overweight categorized by BMI was

not due to an effect on fatness, while later childhood weight gain was primarily associated with adiposity.

In this paper, we use data from another Brazilian birth cohort to assess associations between growth in different phases of childhood and body size and composition outcomes measured at 18 years of age.

## METHODS

The study was carried out in Pelotas (current population 320 000), a city in Southern Brazil. The population is mostly white, of Portuguese and Spanish background. The infant mortality rate at the time of the study was 38 per thousand live births.

Children born in 1982 in the city have been followed up on a number of occasions (3,4). The study started as a perinatal health survey including all 6011 infants born in three maternity hospitals (over 99% of all births in the city). The 5914 live-born infants were weighed using calibrated paediatric scales and their mothers interviewed on socioeconomic, demographic and health-related variables.

In early 1983, an attempt was made to locate at home all children born in January–April 1982 at the addresses

obtained during the hospital interview, and 79% of these were examined. In 1984 and 1986, the approximately 70 000 homes in the city were visited in search of children born in 1982; this approach led to tracing 87% and 84% of the original cohort, respectively. The average ages of the children were 11.3, 19.4 and 43.1 months in 1983, 1984 and 1986, respectively. In 1997, all homes in a 27% sample of the city's census tracts were visited, and 72% of cohort members expected to be living in these tracts were traced at the mean age of 14.7 years. In all visits, subjects were weighted with calibrated scales, and their height was measured using portable stadiometers.

In 2000, all males in the birth cohort who were still living in the city were legally obliged to take part in an examination at the local army base. Those who agreed signed a detailed informed consent form and underwent the physical examination; 79% of all males in the original cohort were traced. Anthropometric equipment were calibrated daily. The same anthropometrist carried out each measurement on all subjects. Extensive training sessions were carried out before the study started by an experienced anthropometrist. All subjects were also interviewed with a standardized questionnaire. A comparison of subjects who were located and those who were lost to follow-up at each visit is presented elsewhere (3).

Except for birthweight, weight measures were transformed into Z-scores of weight for age, using the NCHS/WHO international growth reference curves (5). For simplicity, weight gains are referred to in terms of approximate ages: 0–1, 1–2, 2–4 and 4–15 years, and represent the difference in Z-scores between consecutive weights. Birthweight Z-scores were calculated taking into account gestational age (obtained by recall of the last menstrual period) and using the Williams curves (6).

The following outcomes were measured during the examination carried out in 2000:

*Standing height:* Measured by a CMS (London, UK) stadiometer, to the nearest mm. Subjects were barefooted.

*Weight:* Subjects weighed in their underpants using an electronic Tanita Body Fat Analyser scale (model TBF-305; Tokyo, Japan), which also provided information on body composition through bioimpedance.

*Fat mass and lean mass:* Expressed in kg, estimated through bioimpedance. As these results may vary from one population to another, a validation substudy was carried out in 48 male subjects in the age range of the study using total body water (TBW) assessed through deuterium dilution as the gold standard (7). The results from the validation study were used to correct the bioimpedance equations for fat mass (TBW/0.732) and lean mass (weight – fat mass).

Anthropometric measures were used to calculate the body mass index (BMI; weight in kg/height in m<sup>2</sup>). Fat mass over height<sup>2</sup> and lean mass over height<sup>2</sup> were calculated. The power of two was selected because log–log regressions of fat and lean mass in kg over height showed regression co-

efficients of 2.12 and 2.05, respectively (8,9). Likewise, the ratio between fat and lean mass was obtained by elevating the denominator to the power of 2.3, based on a log–log regression of fat over lean mass (8,9).

The association between weight gain and each outcome was explored using regression analysis controlling for confounding factors. These factors included monthly family income, maternal education and maternal smoking during pregnancy, which were collected during the 1982 interview in the maternity hospitals, when maternal height was also measured. Birthweight for gestational age (6) was classified into three categories: small for dates, below the 10th centile; adequate weight for gestational age; and missing. The latter category included 21% of the sample for whom information on the date of the last menstrual period was unavailable. Birthweight and weight gain from birth to 1 year were not adjusted for weight for gestational age, because the latter is equivalent to the birthweight Z-score.

The confidentiality of all information was ensured and informed consent was obtained in all phases of the study (verbal consent in the 1980s and written consent in 2000). The Medical Ethics Committee of the University of Pelotas, affiliated with the Brazilian Medical Research Council, approved the study protocol.

## RESULTS

Table S1 (in Supplementary material online) shows the characteristics of the original cohort and of subjects included in the analyses, as well as the follow-up rates in the different visits, according to baseline characteristics. Middle class subjects were more often located than either poor or wealthier ones. On the other hand, follow-up rate was not associated with birthweight. The 110 subjects with complete data, in addition to a smaller prevalence of low birthweight, were also less poor than the whole cohort.

Table S2 (in Supplementary material online) shows the descriptive statistics for the outcome measures, for all subjects seen in 2000 as well as for the subset with complete data. The latter were slightly heavier, shorter and had higher BMI than the former. The fat mass/lean mass<sup>2.3</sup> ratio was similar in both groups.

Table S3 (in Supplementary material online) shows the crude regression coefficients for the outcomes, according to a difference of one Z-score in birthweight, or to a change of one Z-score in weight gain at different time periods. Attained height was positively associated with birthweight and negatively related to weight gain between 4 and 15 years of age; height was not associated with weight gain from birth to 4 years. On the other hand, regression coefficients for the relationship between weight gain and adult weight, BMI, and for fat/lean mass<sup>2.3</sup> almost double in size after the age of 4 years.

Except for fat/lean mass<sup>2.3</sup>, all weight-related outcomes measured in 2000 were positively associated with the weight gain variables in different periods of life. Fat/lean mass<sup>2.3</sup> was negatively associated with birthweight, whereas weight gain in the first 2 years of life were not associated with fat/lean mass<sup>2.3</sup>.

As the above associations may be confounded by maternal and social factors, as well as by gestational age, these analyses were repeated with adjustment for these factors. Table S4 (in Supplementary material online) shows the results after adjustment. Birthweight and weight gain in the first year of life were positively associated with attained height, while the other explanatory variables were not. The negative association between growth at 4–15 years and attained height was no longer significant.

All early-life variables were significantly associated with the weight-related outcomes, except for weight gain in the first 2 years, which were not associated with the fat/lean mass<sup>2,5</sup> index. Regression coefficients tended to increase with age for weight-related variables, but to decrease with age for the height outcome. These tendencies were maintained when the analyses were repeated using standardized regression coefficients or betas (data not shown).

The possibility of effect modification by maternal height, education and smoking, as well as by family income and gestational age, was examined through stratified analyses (results not presented), but there was not evidence of an interaction with any of these factors.

All of the above analyses were repeated by comparing children who gained weight above or below the sample mean in each time period, and the results were consistent with those reported above.

In Table S5 (in Supplementary material online), the 110 subjects measured in all visits were separated into several categories according to whether or not they grew faster than the sample mean in each time period. Weight gain in years 1 and 2 had to be combined; otherwise the sample sizes would be too small. There is no evidence for a critical period that would be more closely associated with the outcomes at age 18 years. Rather, it seems that children who grew faster in more than one time period became heavier, independently of when fast growth took place. These analyses were adjusted for confounding variables, but not for weight for gestational age because the latter is equivalent to birthweight, a component of the exposure variable.

## DISCUSSION

Following a large volume of research demonstrating associations between early growth patterns and diseases in adult life (10), increasing interest has been directed to the ontogenetic development of body composition. A number of studies have now described associations between birthweight and later body composition (2,11–15). However, few longitudinal studies exist where it is possible to examine the relative effects of weight gain or growth in different periods of the life course. This is particularly the case in low and middle-income populations, which may have enhanced susceptibility to early-origin diseases due to rapid shifts in their nutritional profile.

Concerning the limitations of the present study, we do not think that non-response or our inability to follow up all members of the original cohort will have biased the results, unless the relations between early weight gain and anthropometry in adolescence differ in non-responders. Losses to

follow-up, even if numbers vary by disease status or baseline characteristics, does not by itself bias estimates of risk. Bias arises, only if attrition in a disease category varies according to baseline characteristics. This variation might have occurred in our study if, for example, adolescents who were shorter at birth and had higher BMI had decided that they would not take part.

In the 1993 Pelotas Birth Cohort Study, birthweight and weight gain in the first 6 months of life were associated with attained height and lean body mass, but not with fatness, measured at the age of 9 years (2). Weight gain between 1–4 years was associated both with later fatness and lean mass, whereas weight gain 4–9 years was strongly associated with fatness but not lean mass. In the 1982 cohort described here, it was not possible to divide the first year of life into two time periods—birth to six, and 6–12 months. Therefore, our measure of growth in the first year may be combining two periods with different prognoses.

In the adjusted analyses, we confirmed our earlier finding that not only birthweight but also weight gain in the first year were associated with later height, whereas weight gain in subsequent periods did not show a significant association.

The pattern of associations between weight gain and adult weight was broadly similar to that for adult BMI. Weight gain in any period was associated with both these outcomes, however after 2 years of age the magnitude of the effects was substantially greater. Within this general pattern, we were able to observe age-specific variability in the extent to which weight gain was associated with lean versus fat in adulthood.

Consistent with several recent studies (2,11–14), we found an association between birthweight and later relative lean mass. Birthweight was also associated with relative adult fat mass, but the magnitude of the effect was much smaller than for lean mass. Our study is therefore broadly consistent with previous work in finding that fetal weight gain primarily programs lean mass.

Subsequent periods of weight gain exerted effects on both fat and lean, although at all ages the increment in adult lean was greater than in fat. Our previous study of boys aged 9 years discerned an age-related shift in the effect of weight gain from lean to fat (2). However, that study could not take into account the effect of puberty, a period of growth when lean deposition is greater than at earlier points in childhood. According to data on the reference child and the reference adolescent (16,17), in boys in particular, increases in BMI from around 6 years of age are primarily attributable to increases in relative lean mass rather than fat. It is clear therefore that more than one factor needs to be taken into account.

The ontogenetic pattern of body composition concerns two main influences. On the one hand, weight gain follows the intrinsic pattern of human growth. From mid-infancy onwards, this pattern is characterized primarily by gains in lean mass, but also by increases in fat mass in adolescent girls, leading to adult sexual dimorphism in body composition that exceeds the dimorphism in size (16,17). On the other hand, in contemporary environments weight gain also

reflects differential access to nutrition or exercise, and some individuals gain 'excess weight' which, not being part of intrinsic growth, primarily comprises fat mass. Both these effects are apparent in our data.

For any developmental period, increasing weight gain was associated with greater fat and lean masses in adulthood. Within this general pattern, the ratio of fat to lean was more strongly associated with weight gain from 4 years onwards. Thus, our data broadly show that whereas early weight gain is associated with 'larger' adults, weight gain from mid-childhood onwards is also associated with 'fatter' adults. Thus, despite differences between studies in the periods during which weight gain could be assessed, the present study is consistent with our earlier study in suggesting that weight gain *after* infancy is a stronger determinant of subsequent fatness than weight gain in fetal life and early infancy (2).

These findings are consistent with those from other recent studies. Sachdev and colleagues (14), using data on weight and height throughout infancy, childhood and adolescence in Indians from Delhi, found that birthweight and BMI gain in infancy predicted adult lean mass whereas BMI gain from late childhood onwards predicted adult adiposity. Sayer and colleagues (18) found birthweight to be associated with adult lean mass but not fat mass in older men from the UK, while weight at 1 year of age was associated with both lean and fat at follow-up.

These studies aid interpretation of the widespread finding that early weight gain is associated with childhood or adult obesity (1). The use of BMI to categorize obesity is problematic, given that BMI reflects variability in both lean mass and fat mass, whereas the health costs of obesity relate mainly to abdominal fat. There is a wide range in body fatness for a given BMI value (19), and a recent study further demonstrated variability in the BMI-fat relationship according to birthweight (15). It therefore remains unclear the extent to which the relationship between early weight gain and later 'obesity' categorized by BMI can be attributed to the programming of physique rather than fatness per se. Both size and physique must be taken into account before the possible programming of adiposity itself can be evaluated.

Several issues warrant further research. First, the association between birthweight and later body composition appears to be complex. Whereas several studies now link birthweight with later lean mass rather than fat mass, a number of studies have also associated birthweight with later fat distribution. Several studies have associated low birthweight with a more central distribution of fat in childhood or adulthood (15,20–22) and in one study of low birthweight Indian neonates the central fat distribution appeared to be present at birth itself (23). In our study, central fatness was not assessed at the age of 18 years. Second, there are currently too few studies to be confident about the duration of the sensitive period during which lean mass is programmed. In fact, the concept of critical windows for this outcome is problematic. Lean mass responds to nutritional constraint during more than one period of growth. It is clear that infancy is one such period, but puberty is known to be another. For ex-

ample, adolescents with eating disorders have markedly less lean mass than healthy controls (24). Furthermore, excess body weight is associated with increased lean mass as well as increased fat mass (25). Therefore, differential weight gain can impact on lean mass throughout childhood and adolescence.

What remains unclear is whether growth variability at different ages exerts common effects on lean mass, or whether the effects of differential weight gain in infancy are different to those in adolescence. This issue is important because public health policy needs simultaneously to promote optimal growth whilst constraining excess weight gain. This is particularly true for low and middle-income populations where slow growth in early life is associated with childhood morbidity and mortality (26,27). Several studies have now associated childhood obesity with *poor* growth in early life (28,29), and obesity and malnutrition are often present within the same family (28,30,31). Given growing evidence of the association between excess adiposity and other diseases such as cardiovascular disease, type 2 diabetes and hypertension, the programming and development of body composition requires further research.

In summary, our study has shown that height is primarily determined by fetal and infant growth. Weight-related indices tend to be more strongly influenced by later growth, and the same applies for the ratio between fat and lean mass. Nevertheless, it is difficult to discern clear critical windows of growth during which absolute tissue masses are programmed. More specific studies are now required to elucidate the effect of weight gain in different growth periods on the regional distribution of lean and fat mass. Finally, our study clearly relates only to males, and requires repeating in females whose growth pattern is distinct.

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#### Supplementary material

The following supplementary material is available for this article:

**Table S1** Characteristics of cohort subjects with data available at different time points. Males only.

**Table S2** Descriptive statistics of subjects examined at 18 years of age.

**Table S3** Unadjusted regression coefficients of body size and composition outcomes by weight gain for four time periods, for all subjects examined at 18 years of age. Each cell represents a regression model.

**Table S4** Adjusted regression coefficients of body size and composition outcomes by weight gain for four time periods, for all subjects examined at 18 years of age. Each cell represents a regression model.

**Table S5** Summary table of weight gain\* (above or below the mean) for three time periods, by six outcomes, for 110 subjects with complete data.

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