Postnatal weight and height growth velocities at different ages between birth and 5 y and body composition in adolescent boys and girls$^{1-3}$

Jérémie Botton, Barbara Heude, Jean Maccario, Pierre Ducimetière, and Marie-Aline Charles and the FLVS Study group

ABSTRACT

Background: Rapid weight gain in the first years of life is associated with adult obesity. Whether there are critical windows for this long-term effect is unclear.

Objective: The objective was to study anthropometric measures in adolescence by sex according to weight and height growth velocities at different ages between birth and 5 y.

Design: Anthropometric measures, including fat and fat-free mass by bipodal impedanceometry, were measured in 468 adolescents aged 8–17 y. We retrospectively collected early infancy data and individually estimated weight and height growth velocities in 69.4% of them using a mathematical model. Associations between birth variables, growth velocities, and anthropometric measures in adolescence were studied.

Results: Weight growth velocity at 3 mo was associated with overweight (odds ratio for a 1-SD increase: 1.52; 95% CI: 1.04, 2.22), fat mass, and waist circumference in adolescence in both sexes and with fat mass in boys ($r = 0.29, P < 0.001$) but not in girls ($r = -0.01, NS$). Weight growth velocities after 2 y were associated with all anthropometric measures in adolescence, in both sexes. Between 6 mo and 2 y, weight growth velocities were significantly associated only with adolescent height in boys; in girls, associations with fat mass in adolescence were weaker.

Conclusion: Our results support the hypothesis of 2 critical windows in early childhood associated with the later risk of obesity: up to 6 mo and from 2 y onward. The study of the determinants of growth during these 2 periods is of major importance for the prevention of obesity in adolescence.

INTRODUCTION

More and more evidence indicates that children who gain weight rapidly during early life are at higher risk of obesity in later life (1), although definitions of weight gain and later life are very different between the studies. The early life periods considered also vary between studies. Both the first months or years of life (2–4) and the period of the adiposity rebound (5–7) have been shown to be associated with later obesity, but it is difficult from available data to assess the relative contribution of each period. This is important to better understand the link between early weight and height growth and obesity later in life, so that the appropriate timing of potential preventive measures can be defined.

Most of the studies approximate height or weight growth velocity by the difference between 2 height or 2 weight measures (1, 8–11), and they use the average growth over the period. The contribution of different subperiods of age can therefore not be determined. Obesity is mostly assessed on the basis of body mass index (BMI), but this index does not distinguish between fat and fat-free mass. Some studies have shown that rapid growth rates in infancy or in childhood are specifically associated with more fat mass later in life (10, 12). Sex differences have rarely been assessed, even though both body composition and weight and height growth in infancy are quite different between boys and girls (13).

Our aim was to study body composition in adolescence, between 8 and 17 y of age, according to weight and height growth velocities at different ages between 3 mo and 5 y, separately in boys and girls. Velocities were estimated from a growth model that allowed us to smooth the data and to choose the periods of interest.

SUBJECTS AND METHODS

Subjects

The children were recruited in Fleurbaix and Lavantie, 2 neighboring towns in northern France, with respectively 2488 and 4426 inhabitants in 1992, when the Fleurbaix-Lavantie Ville Santé (FLVS) study started. The first part of the study (FLVS I), a 5-y follow-up of children involved in a nutritional education program at school (14), included 579 families.

The second part, FLVS II, was an epidemiologic study of the determinants of weight change in the population. This study was proposed to all families participating in the FLVS I Study that were still living in the 2 towns and who could be contacted (ie, 393 families). We recruited and examined 294 families between
April and September 1999 (ie, an acceptance rate of 75%). All family members aged \( \geq 8 \) y (\( n = 1113 \) subjects) were examined at home in 1999, and 255 boys and 251 girls aged 8–17 y were included. Fewer of the children studied were overweight than those not recruited (8.0% compared with 13.5%; \( P = 0.008 \) by the International Obesity Task Force (IOTF) definition; 15). Written consent was obtained from the children and from their parents. The FLVS II Study was approved by the ethics committee of Lille (France), and the computer files were declared to the Commission Nationale Informatique et Liberté.

At the end of the FLVS II Study in 2003, we collected data from the Carnet de Santé for 227 boys and 241 girls (92%). The Carnet de Santé is a health booklet given in France to all parents at the time of their children’s birth, which is filled in by health practitioners throughout childhood. These 468 children represent the study population for this report.

**Anthropometric data**

All weight and height measurements in the Carnet de Santé up until the age of 12 y were extracted for the present analysis, including measures at birth. Measurements at 9 mo and 2 y were available for most children because a medical examination is required by the French administration.

In addition, as part of the longitudinal follow-up of the children in the FLVS I and II studies, trained physicians collected anthropometric data on children on one or more occasions when they were between 5 and 17 y of age. Weight was measured to the nearest 0.1 kg and height to the nearest 1 cm while the children were wearing light clothes and no shoes. Waist circumference was measured to the nearest 0.5 cm, during expiration when breathing normally, at the smallest diameter between the iliac crest and the lower rib. Arm circumference was measured to the nearest 1 mm with a tape measure, on a relaxed arm, at the midpoint between the acromion and the olecranon. Four skinfold thicknesses were measured to the nearest 0.1 mm, in duplicate, with Harpenden calipers on the left side of the body: tricipital (posterior aspect of the arm, at the midpoint between the acromion and the olecranon), bicipital (anterior aspect of the arm, at the midpoint between the acromion and the olecranon), subscapular (1 cm below the inferior angle at the scapula), and supra-iliac (1 cm over the iliac crest, at the midaxillary line). The duplicate measures were averaged. The sum of all skinfold thicknesses, the sum of arm skinfold thicknesses (tricipital + bicipital), and BMI (weight/height\(^2\); in kg/m\(^2\)), were calculated. Overweight and obesity were defined according to the IOTF sex- and age-specific BMI cutoffs (15). Fat mass and fat-free mass were obtained with a bipedal impedanceometry device (TBF310; Tanita, Neuilly-sur-Seine, France) as part of the FLVS II Study. Pubertal stage was determined by the physicians according to Tanner’s classification.

**Statistical analysis**

The aim of the statistical analysis was to correlate weight and height growth velocities at different ages in early life with anthropometric measures in adolescence. The first step of the analysis was to derive the weight and height growth velocities for each child at different ages, from a mathematical model of a child’s growth between 0 and 12 y of age (described below). The second step was to correlate the estimated weight and height growth velocities at different ages between 3 mo and 5 y with the anthropometric measures at the last available examination of the children in 1999, the entry examination of the FLVS II Study: children aged 8–17 y are called “adolescents” for the sake of simplicity. Correlation coefficients were used instead of regression coefficients to ensure the comparability between each period of early life and each anthropometric measure. SAS software version 9.1 was used for all analyses. All significance tests were 2-sided, and a \( P \) value > 0.05 was considered nonsignificant.

**Weight and height growth models**

Measurements of weight and height from 0 to 12 y, obtained either from the Carnet de Santé or the clinical examination, allowed us to model each child’s growth individually, using an adaptation of the Jenss growth curve model (16, 17), by including an additional quadratic term in age \( (c_i \times t^2) \). This model was validated in our population by an analysis of the residuals to confirm the goodness of fit. Mean growth velocities estimated by piecewise models are more accurate but require many more measurements; they gave similar results to those estimated from our model (data not shown).

Our model allowed us to estimate a child’s growth from 0 to 12 y of age instead of 0 to 8 y of age, as in the initial Jenss growth model (16). Thus, we were able to include more children because many of them were measured during the FLVS I Study and have at least one measurement after 5 y. To be eligible for inclusion, the children had to meet some conditions: each child had to have \( \geq 6 \) measures between 0 and 12 y of age, with \( \geq 2 \) before 2 y of age and \( \geq 1 \) after 5 y of age; 161 boys and 164 girls met these conditions for the weight growth model (69.4%) and 160 boys and 152 girls for the height growth model (66.7%). Boys who met the weight or the height model conditions were 1 y younger than the others (\( P = 0.03 \)); however, after age adjustment, none of the anthropometric characteristic were significantly different. In girls, there were no significant differences. The number of weight measurements available for different age periods between 0 and 12 y are presented in Table 1 for the children studied; the number and distribution of available height measurements were similar.

The same model was used to fit both the weight and the height growth:

\[
y_i(t) = a_i + b_i \times t + c_i \times t^2 - \exp(d_i + e_i \times t) + e_i_i
\]

where \( t \) is age (in mo), \( y_i \) is weight (in kg) or height (in cm) of the \( i \)th child at age \( t \), and \( e_i \) is the residual error at age \( t \). The least-squares estimates \( a_i, b_i, c_i, d_i, \) and \( e_i \) were determined for each child with PROC NLIN by using Gauss-Newton iterative method in SAS.

This model is the sum of 2 components: 1) \( a_i + b_i \times t + c_i \times t^2 \), which principally models growth after 3 y of age, and 2) \( \exp(d_i + e_i \times t) \), which models growth during the first 3 y and is characterized by a sharp increase in the first months of life, followed by a slowly declining growth rate (18).

We constrained the parameters to obtain easier model convergence: for the height and the weight models, parameter \( b_i \) was constrained to be positive (positive mean growth), \( a_i > \exp(d_i) \) (positive birth weight), and \( e_i \) negative (declining growth rate during the first 3 y). For the weight model only, \( c_i \) was additionally constrained to be positive. Indeed, weight growth is accelerating after 8 y of age, whereas height growth is accelerating in some children, remaining stable or decelerating in others. The model and the observed data for 2 subjects with distinct growth velocities at 3 mo are shown in Figure 1.
The first derivative of the equation of the individual models is the growth velocity at a given age (Figure 2). We used ages 3 and 6 mo and 1, 2, 3, and 5 y as representative of different periods of early childhood for the analysis of the association with body composition in adolescence. The growth velocity at 10 y was computed for the descriptive analysis only.

**Analysis of the relations between early childhood growth rates and body composition in adolescence**

Results are given as means (± SD) or geometric means (and 95% CIs) for the log-transformed variables (total fat mass, sum of brachial skinfold thicknesses, and sum of 4 skinfold thicknesses) or as percentages (number). All between-sex comparisons were performed by using Student’s t tests or chi-square tests.

Anthropometric measures in adolescence (X1 variables) were correlated with growth rates of height and weight estimated at different ages during childhood (X2 variables). Partial correlation coefficients between an X1 variable and an X2 variable were the Pearson correlation coefficients between the residuals of the regressions of X1 and X2 on their respective adjustment factors. X1 variables were systematically adjusted for age and Tanner stage and for height when X1 was associated with height. When X1 was the arm circumference, we additionally adjusted for the sum of arm skinfold thicknesses to obtain a brachial fat-free mass index. X2 variables were systematically adjusted for gestational age when it was a birth outcome. Because of the presence of siblings in our study population, the analysis also took into account familial resemblance: in all regressions, we introduced a family variable as a random effect in a linear mixed model (19). Differences in relations between boys and girls were tested by including an interaction term with sex in linear regression models, with preadjusted X1 as the dependent variable and preadjusted X2 as the independent variable. Because our sample of adolescents covered a wide age range (8–17 y), we also tested interactions with an age class variable (defined by the median age of the population, 13.5 y) to evaluate whether relations were consistent across age categories.

The relations between weight and height growth velocities at different ages between 3 mo and 5 y and the risk of being overweight in adolescence were assessed from a logistic model adjusted for age, Tanner stage, and sex. Power was lacking to stratify this analysis by sex.

The quality of fit of the individual growth models could have been taken into account by an intrasubject variance parameter in the subsequent analyses; however, because this variance was ≥1

---

**TABLE 1**

<table>
<thead>
<tr>
<th>Age interval and measures (n)</th>
<th>Boys (n = 161)</th>
<th>Girls (n = 164)</th>
<th>Total (n = 325)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 2 y</td>
<td>6 (2–15)</td>
<td>7 (2–14)</td>
<td>7 (2–15)</td>
<td>%</td>
</tr>
<tr>
<td>2 ≤ n ≤ 5</td>
<td>80</td>
<td>66</td>
<td>146</td>
<td>45</td>
</tr>
<tr>
<td>n ≥ 6</td>
<td>81</td>
<td>98</td>
<td>179</td>
<td>55</td>
</tr>
<tr>
<td>&gt; 2 and ≤ 5 y</td>
<td>1 (0–11)</td>
<td>1 (0–8)</td>
<td>1 (0–11)</td>
<td>%</td>
</tr>
<tr>
<td>Median (min-max)</td>
<td>75</td>
<td>59</td>
<td>134</td>
<td>41</td>
</tr>
<tr>
<td>1 ≤ n ≤ 2</td>
<td>51</td>
<td>59</td>
<td>110</td>
<td>34</td>
</tr>
<tr>
<td>n ≥ 3</td>
<td>35</td>
<td>46</td>
<td>81</td>
<td>25</td>
</tr>
<tr>
<td>&gt; 5 and ≤ 12 y</td>
<td>4 (1–7)</td>
<td>4 (1–9)</td>
<td>4 (1–9)</td>
<td>%</td>
</tr>
<tr>
<td>Median (min-max)</td>
<td>20</td>
<td>23</td>
<td>43</td>
<td>13</td>
</tr>
<tr>
<td>1 ≤ n ≤ 2</td>
<td>141</td>
<td>141</td>
<td>282</td>
<td>87</td>
</tr>
<tr>
<td>n ≥ 3</td>
<td>22</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Median (min-max)</td>
<td>76</td>
<td>61</td>
<td>137</td>
<td>42</td>
</tr>
<tr>
<td>6 ≤ n ≤ 9</td>
<td>85</td>
<td>103</td>
<td>188</td>
<td>58</td>
</tr>
<tr>
<td>n ≥ 10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 Conditions of the model: ≥ 6 measures in the age range 0–12 y and ≥ 2 measures before 2 y and ≥ 1 measure after 5 y.

2 Percentage compared with the population meeting the conditions of the model (both sexes).

---

**FIGURE 1.** Fitted weight and height growth curve between 0 and 12 y in 2 selected subjects from the study with high and low growth velocities at 3 mo (descriptive data without statistical test).
times smaller than the intersubject variance at all ages (except 3 mo), we decided to ignore this parameter. At 3 mo, taking this variance component into account did not change our conclusions (data not shown).

RESULTS

Characteristics of the children

Birth, 9 mo, and 2 y

In our population, for a similar gestational age, birth weight was not significantly different between boys and girls, but boys were significantly longer than girls (Table 2). Therefore, ponderal index at birth was higher in girls. At 9 mo and 2 y, boys were heavier and taller than girls, but the mean BMIs were not significantly different.

Adolescence

At examination, the adolescents had a mean age of 13.5 y (Table 2). Girls were more mature, and boys were taller than girls. Girls had higher mean BMI, fat mass, and sum of skinfold thicknesses than did boys. Boys had more fat-free mass than girls. Arm circumference was not significantly different between boys and girls. The percentages of overweight or obesity were not significantly different between boys (10.2%) and girls (12.9%).

Growth velocity from 3 mo to 10 y

Weight and height growth at 3 mo were faster in boys than in girls: for weight, $0.70 \pm 0.13$ kg/mo in boys compared with $0.65 \pm 0.12$ kg/mo in girls ($P = 0.0003$; Table 3). Thereafter, the growth velocities were not significantly different until 10 y of age, when they were higher for girls than for boys: for weight, $0.27 \pm 0.10$ kg/mo in boys compared with $0.30 \pm 0.10$ kg/mo in girls ($P = 0.04$).

Relations between weight growth velocities in early childhood and overweight in adolescence

At 3 mo, a 1-SD increase in weight growth velocity (143 g/mo) increased the risk of being overweight or obese in adolescence (OR: 1.52; 95% CI: 1.04, 2.22; Figure 3). A 1-SD increase in weight growth velocity at 3 and 5 y (corresponding to 50 and 43 g/mo, respectively) increased the risk of being overweight (OR: 2.43; 95% CI: 1.75, 3.39) or obese (OR: 5.08; 95% CI: 3.19, 8.09) in adolescence, respectively. For weight growth velocity at 1 y, the increased risk was not significant (OR: 1.17; 95% CI: 0.84, 1.64). Overweight in both adolescent boys and girls was not significantly associated with any of the height growth velocities.

Relations between birth length and height growth velocities in early childhood with anthropometric measures in adolescence

Birth length and height growth velocities at all ages were positively correlated with the height in adolescence, the smallest correlations were for boys at 3 mo ($r = 0.17, P = 0.04$) and for girls at 12 mo ($r = 0.19, P = 0.02$). All other correlation coefficients were $>0.26 (P = 0.0008)$. In both sexes, birth length and height growth velocities at all ages in early childhood were not significantly associated with any of the other anthropometric measures in adolescence.

Relations between birth weight and weight growth velocities in early childhood with anthropometric measures in adolescence (Table 4)

Birth weight and height growth velocities between 3 mo and 5 y were significantly correlated with adolescent height in boys and girls (range: 0.19–0.46; $P < 0.02$), except birth weight in girls ($r = 0.13$). Birth weight was not significantly associated with adolescent BMI, either in boys or in girls. In contrast, correlations between weight growth velocity at 3 and 6 mo and adolescent BMI were significant ($r = 0.18–0.30$). In boys, correlations between 1 and 2 y decreased to low levels ($r = 0.09$ at 1 y; NS). Thereafter, they started increasing again. In girls, correlations between weight growth velocity and adolescent BMI increased more regularly with age.

Birth weight was not significantly associated with the amount of adolescent fat mass (adjusted for adolescent height, age, and Tanner stage). Weight growth velocities at 3 and 6 mo, but not at 1 y, were positively correlated with total fat mass and sum of skinfold thicknesses (same adjustments) in boys and in girls, with comparable coefficients for both variables and for both sexes. In girls only, there was a significant interaction with age for some correlations between weight growth and fat mass in adolescence (data not shown): correlations were only significant for the

![FIGURE 2](image-url) Weight and height growth velocity, predicted from the model, between 0 and 12 y in 2 selected subjects from the study with high and low growth velocities at 3 mo (descriptive data without statistical test).
younger girls (age ≤ 13.5 y) compared with the others (eg, 
[r]/L1155 0.38 and 
P/L1155 0.0003 compared with 
r/L1155/L1152 0.03 and 
P/L1155 0.80 
between weight growth velocity at 3 mo and fat mass in adoles-
ence).

There was a positive correlation between birth weight and 
adolescent fat-free body mass when sexes were pooled ( 
r/L1155 0.12, 
P/L1155 0.01). In boys, a faster weight growth velocity at 3 mo was 
associated with a higher adolescent fat-free mass ( 
r/L1155 0.29, 
P/L50141 0.001) and with a higher brachial fat-free mass index ( 
r/L1155 0.26, 
P/L50141 0.001). There was also a relation, albeit weaker, between 
weight growth velocity at 6 mo and fat-free mass ( 
r/L1155 0.16, 
P/L1155 0.05). However, in girls, rapid weight growth velocity at 3 mo 
was not associated with a higher amount of adolescent fat-free 
mass; the interaction term with sex was statistically significant ( 
P/L1155 0.002). Correlations between 6-mo, 1-y, and 2-y weight 
growth velocities and adolescent fat-free mass were low and not 
significantly different from zero for boys but increased progres-
sively for girls.

Weight growth velocities at 3 and 5 y were correlated with all 
anthropometric measures in adolescence. The correlation coef-
"TABLE 2" General characteristics of boys and girls and comparisons

<table>
<thead>
<tr>
<th></th>
<th>No. of subjects</th>
<th>Boys</th>
<th>Girls</th>
<th>(P^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (%)</td>
<td>468</td>
<td>48.5 (227)</td>
<td>51.5 (241)</td>
<td>0.52</td>
</tr>
<tr>
<td>Birth and infancy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gestational age (wk of pregnancy)</td>
<td>449</td>
<td>38.7 (2.0)</td>
<td>38.9 (1.6)</td>
<td>0.19</td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>463</td>
<td>3.33 (0.56)</td>
<td>3.30 (0.47)</td>
<td>0.58</td>
</tr>
<tr>
<td>Birth length (cm)</td>
<td>455</td>
<td>50.2 (2.1)</td>
<td>49.4 (2.0)</td>
<td>0.005</td>
</tr>
<tr>
<td>Birth BMI (kg/m²)</td>
<td>452</td>
<td>13.2 (1.4)</td>
<td>13.4 (1.3)</td>
<td>0.31</td>
</tr>
<tr>
<td>Birth ponderal index (kg/m³)</td>
<td>452</td>
<td>26.4 (2.7)</td>
<td>26.9 (2.6)</td>
<td>0.02</td>
</tr>
<tr>
<td>9-mo weight (kg)</td>
<td>437</td>
<td>8.91 (1.0)</td>
<td>8.49 (1.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>9-mo height (cm)</td>
<td>416</td>
<td>71.9 (2.8)</td>
<td>70.7 (3.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>9-mo BMI (kg/m²)</td>
<td>411</td>
<td>17.2 (1.6)</td>
<td>17.0 (1.7)</td>
<td>0.22</td>
</tr>
<tr>
<td>2-y weight (kg)</td>
<td>425</td>
<td>12.5 (1.6)</td>
<td>12.0 (1.5)</td>
<td>0.001</td>
</tr>
<tr>
<td>2-y height (cm)</td>
<td>411</td>
<td>87.3 (3.5)</td>
<td>86.0 (3.6)</td>
<td>0.0002</td>
</tr>
<tr>
<td>2-y BMI (kg/m²)</td>
<td>406</td>
<td>16.4 (1.7)</td>
<td>16.3 (1.7)</td>
<td>0.57</td>
</tr>
<tr>
<td>Adolescence</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>468</td>
<td>13.4 (2.4)</td>
<td>13.5 (2.7)</td>
<td>0.81</td>
</tr>
<tr>
<td>Tanner stage</td>
<td>468</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>22.0 (50)</td>
<td>19.5 (47)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>20.7 (47)</td>
<td>12.0 (29)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>19.4 (44)</td>
<td>19.9 (48)</td>
<td></td>
<td>0.02</td>
</tr>
<tr>
<td>4</td>
<td>26.0 (59)</td>
<td>27.4 (66)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>11.9 (27)</td>
<td>21.2 (51)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>467</td>
<td>1.59 (0.15)</td>
<td>1.56 (0.13)</td>
<td>0.004</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>468</td>
<td>47.9 (14.6)</td>
<td>47.1 (12.8)</td>
<td>0.51</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>467</td>
<td>18.4 (3.2)</td>
<td>19.1 (3.3)</td>
<td>0.03</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>462</td>
<td>5.5 (5.1, 6.0)</td>
<td>9.7 (8.9, 10.6)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>462</td>
<td>41.1 (11.7)</td>
<td>55.3 (7.1)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Sum of total skinfolds (mm)</td>
<td>463</td>
<td>32.8 (30.8, 35.0)</td>
<td>45.6 (43.0, 48.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Arm circumference (cm)</td>
<td>464</td>
<td>22.8 (3.5)</td>
<td>23.0 (3.2)</td>
<td>0.48</td>
</tr>
<tr>
<td>Sum of brachial skinfolds (mm)</td>
<td>465</td>
<td>16.1 (15.1, 17.0)</td>
<td>22.2 (21.0, 23.4)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Overweight (%)</td>
<td>467</td>
<td>8.9 (20)</td>
<td>10.0 (24)</td>
<td>0.45</td>
</tr>
<tr>
<td>Obese (%)</td>
<td>467</td>
<td>1.3 (3)</td>
<td>2.9 (7)</td>
<td></td>
</tr>
</tbody>
</table>

\(^1\) Student’s t test or chi-square test.

\(^2\) \(n\) in parentheses.

\(^3\) SD in parentheses.

\(^4\) 95% CI in parentheses.

DISCUSSION

Our results suggest that 2 periods in early childhood are asso-
ciated with body composition within the adolescent 8–17 y age 
range studied: the first 6 mo and the period from 2 y onward. In 
between, differences in weight growth contributed more to 
height than weight differences in adolescence. The relation be-
tween weight growth velocities and adolescent anthropometric 
measures other than height were not confounded by height 
growth because height growth velocities were not significantly 
associated with any of the adolescent measures.

The first critical period: before 6 mo

From our results and other studies, we can conclude that during 
this first period, weight growth appears to be associated, rather 
consistently, with body composition in later life (8, 10, 12, 20, 
21). This association might be stronger with fat-free mass than 
with fat mass, especially in developing countries (8, 10). As in 
our study, the association between weight growth and fat-free 
mass was significantly stronger in males than in females in the 
only other study that has examined sex differences (20).

These relations are not unexpected when one considers that it 
is the period of fastest growth in the entire life span. Any variation
in this process may therefore have long lasting consequences. Several determinants of growth and energy metabolism commence in this 6-mo period (22), among which is oral nutrition (23). In this period of intense development of adipose tissue (24), different foods have been shown to lead to different adipocyte development, in both size and in number (25), and this period is potentially determinant for the pool of fat cells, even though fat cells can be recruited over the entire life span (26). The offspring’s growth is also, for the first time, free of maternal intrauterine constraints and genetic factors, especially paternal, may be expressed (27). Some biological processes such as maturation of the gastrointestinal tract (24) or development of major hypothalamic regulatory functions (28)

TABLE 3

Mean weight and height growth velocity estimations between 3 mo and 10 y calculated from the model

<table>
<thead>
<tr>
<th>Weight velocities (kg/mo)</th>
<th>n</th>
<th>Boys</th>
<th>Girls</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 mo</td>
<td>161</td>
<td>0.70 ± 0.13</td>
<td>0.65 ± 0.12</td>
<td>0.0003</td>
</tr>
<tr>
<td>6 mo</td>
<td>164</td>
<td>0.48 ± 0.11</td>
<td>0.46 ± 0.09</td>
<td>0.10</td>
</tr>
<tr>
<td>1 y</td>
<td>161</td>
<td>0.29 ± 0.08</td>
<td>0.28 ± 0.07</td>
<td>0.55</td>
</tr>
<tr>
<td>2 y</td>
<td>162</td>
<td>0.18 ± 0.05</td>
<td>0.18 ± 0.05</td>
<td>0.40</td>
</tr>
<tr>
<td>3 y</td>
<td>162</td>
<td>0.16 ± 0.04</td>
<td>0.16 ± 0.05</td>
<td>0.49</td>
</tr>
<tr>
<td>5 y</td>
<td>162</td>
<td>0.19 ± 0.05</td>
<td>0.19 ± 0.05</td>
<td>0.54</td>
</tr>
<tr>
<td>10 y</td>
<td>162</td>
<td>0.27 ± 0.10</td>
<td>0.30 ± 0.10</td>
<td>0.04</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Height velocities (cm/mo)</th>
<th>n</th>
<th>Boys</th>
<th>Girls</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 mo</td>
<td>160</td>
<td>2.67 ± 0.38</td>
<td>2.56 ± 0.36</td>
<td>0.01</td>
</tr>
<tr>
<td>6 mo</td>
<td>152</td>
<td>1.91 ± 0.27</td>
<td>1.87 ± 0.27</td>
<td>0.18</td>
</tr>
<tr>
<td>1 y</td>
<td>152</td>
<td>1.21 ± 0.21</td>
<td>1.21 ± 0.21</td>
<td>0.81</td>
</tr>
<tr>
<td>2 y</td>
<td>152</td>
<td>0.78 ± 0.11</td>
<td>0.77 ± 0.11</td>
<td>0.63</td>
</tr>
<tr>
<td>3 y</td>
<td>152</td>
<td>0.65 ± 0.08</td>
<td>0.64 ± 0.09</td>
<td>0.43</td>
</tr>
<tr>
<td>5 y</td>
<td>152</td>
<td>0.55 ± 0.06</td>
<td>0.56 ± 0.08</td>
<td>0.39</td>
</tr>
<tr>
<td>10 y</td>
<td>152</td>
<td>0.42 ± 0.16</td>
<td>0.48 ± 0.20</td>
<td>0.005</td>
</tr>
</tbody>
</table>

* Student’s t test.

2 x ± SD (all such values).

in this process may therefore have long lasting consequences. Several determinants of growth and energy metabolism commence in this 6-mo period (22), among which is oral nutrition (23). In this period of intense development of adipose tissue (24), different foods have been shown to lead to different adipocyte development, in both size and in number (25), and this period is potentially determinant for the constitution of the pool of fat cells, even though fat cells can be recruited over the entire life span (26). The offspring’s growth is also, for the first time, free of maternal intrauterine constraints and genetic factors, especially paternal, may be expressed (27). Some biological processes such as maturation of the gastrointestinal tract (24) or development of major hypothalamic regulatory functions (28) that take place in early life. Finally, the early life period is characterized by transient hormonal secretions. In particular, we hypothesize that the physiologic peak of plasma testosterone at 3 mo in boys (29) could have anabolic effects stimulating the development of fat-free mass and explaining the stronger association between weight growth velocity at 3 mo and fat-free mass in males than in females.

The second critical period: from 2 y onward

Previous studies showed that either greater BMI gain or weight gain from ≈2 or 3 y of age was associated with adult adiposity, central obesity, and, to a lesser degree, a higher fat-free mass in later life (8, 10, 12, 21).

As suggested by Ekelund et al (12), the mechanisms leading to these relations may be different from those during the first critical period. Indeed, in our study, weight growth at 3 mo was not correlated with weight growth at 3 y in boys or in girls (data not shown). This second period is that of the adiposity rebound, which occurs earlier in fat children (29). Fat gain may therefore explain much of the variability in weight gain differences between 3 and 5 y. Again, nutrition could probably be a key factor in this period, and this is influenced by parental eating habits (30) and is the period during which taste is developing (31, 32). It is also a period during which physical activity habits start, even if tracking through childhood is not clear (33). Finally, specific genetic factors that would express preferentially from 2 y could be involved.

The gap: from 6 mo to 2 y

Correlations between weight gain velocities and adolescent anthropometric measures (except height) decreased in boys, as in girls, during this period. Several studies reported this pattern (8, 10, 21, 34), and it has been commented on in the 2 more recent ones (21, 34).

During this period, height growth seems to preferentially drive weight gain as previously reported (34). This period has also been noted as a period of critical height development, because nutritional
and girls

isome proliferator–activated receptor–
fat-free mass and in particular to bone growth. For example, perox-
allow a tradeoff and change of energy allocation from fat mass to
nutrient intake (36). Mechanisms have been described that may
may be stimulated preferentially during this period (21), during
improve growth rates (35). Height growth rather than fat storage
intervention (drinks rich in high-quality protein) has been shown to
improve growth rates (35). Height growth rather than fat storage

TABLE 4
Partial correlations between birth weight or weight growth velocities in infancy and anthropometric measures, adjusted for confounders, in adolescent boys and girls

<table>
<thead>
<tr>
<th>Measure</th>
<th>Boys</th>
<th>Girls</th>
<th>Weight at birth</th>
<th>3 mo</th>
<th>6 mo</th>
<th>1 y</th>
<th>2 y</th>
<th>3 y</th>
<th>5 y</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Weight growth velocities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td>0.39*</td>
<td>0.19*</td>
<td>0.27*</td>
<td>0.30*</td>
<td>0.32*</td>
<td>0.32*</td>
<td>0.39*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>0.13</td>
<td>0.30*</td>
<td>0.18*</td>
<td>0.09</td>
<td>0.14</td>
<td>0.35*</td>
<td>0.65*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat mass</td>
<td>0.10</td>
<td>0.26*</td>
<td>0.16*</td>
<td>0.07</td>
<td>0.12</td>
<td>0.31*</td>
<td>0.57*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sum of skinfolds</td>
<td>0.06</td>
<td>0.21*</td>
<td>0.12</td>
<td>0.04</td>
<td>0.09</td>
<td>0.27*</td>
<td>0.53*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waist circumference</td>
<td>0.09</td>
<td>0.23*</td>
<td>0.13</td>
<td>0.07</td>
<td>0.11</td>
<td>0.28*</td>
<td>0.56*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat-free mass</td>
<td>0.12</td>
<td>0.29*</td>
<td>0.14</td>
<td>0.04</td>
<td>0.03</td>
<td>0.18*</td>
<td>0.44*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brachial fat-free mass index</td>
<td>0.09</td>
<td>0.26*</td>
<td>0.09</td>
<td>-0.03</td>
<td>0.01</td>
<td>0.17*</td>
<td>0.41*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Girls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td>0.13</td>
<td>0.30*</td>
<td>0.34*</td>
<td>0.43*</td>
<td>0.46*</td>
<td>0.41*</td>
<td>0.37*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>0.03</td>
<td>0.19*</td>
<td>0.23*</td>
<td>0.19*</td>
<td>0.23*</td>
<td>0.36*</td>
<td>0.59*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat mass</td>
<td>-0.02</td>
<td>0.24*</td>
<td>0.21*</td>
<td>0.11</td>
<td>0.13</td>
<td>0.27*</td>
<td>0.51*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sum of skinfolds</td>
<td>-0.09</td>
<td>0.17*</td>
<td>0.17*</td>
<td>0.10</td>
<td>0.20*</td>
<td>0.33*</td>
<td>0.53*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waist circumference</td>
<td>-0.05</td>
<td>0.18*</td>
<td>0.20*</td>
<td>0.18*</td>
<td>0.19*</td>
<td>0.28*</td>
<td>0.45*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat-free mass</td>
<td>0.10</td>
<td>-0.01</td>
<td>0.10</td>
<td>0.16*</td>
<td>0.18*</td>
<td>0.25*</td>
<td>0.34*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brachial fat-free mass index</td>
<td>-0.01</td>
<td>-0.01</td>
<td>0.10</td>
<td>0.14</td>
<td>0.07</td>
<td>0.12</td>
<td>0.25*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Maximum of 5 missing values.
2 Residuals of variable adjusted for gestational age. n = 214 boys and 231 girls.
3 n = 161 boys and 164 girls.
4 Residuals of variable adjusted for age and Tanner stage.
5 P < 0.001.
6 P < 0.05.
7 P < 0.01.
8 Residuals of variable adjusted for age, Tanner stage, and height.
9 Residuals of arm circumference adjusted for age, Tanner stage, height and sum of arm skinfolds.

intervention (drinks rich in high-quality protein) has been shown to
improve growth rates (35). Height growth rather than fat storage
may be stimulated preferentially during this period (21), during
which there is an increase in the ratio of protein to lipids in macro-
nutrient intake (36). Mechanisms have been described that may
allow a tradeoff and change of energy allocation from fat mass to
fat-free mass and in particular to bone growth. For example, perox-
osome proliferator–activated receptor–γ is implicated as a transcrip-
tion factor in the differentiation from the common bone marrow
stromal cells in adipocytes or osteoblasts (37, 38). However, it might
still be possible that this period is characterized by the silent (as far
as weight is concerned) recruitment of preadipocytes in preparation
for the adiposity rebound (26).

Strengths and weaknesses

Our growth model was able to handle the heterogeneity of weight
and height measurements between 0 and 12 y in quality (no stan-
dardization for the measures of the Carnet de Santé) and in quantity,
because the model minimized the measurement errors (≥6 mea-
sures for 5 variables). It also allowed us to take into account the
nonlinearity of growth during infancy (Figure 1) and to investigate
different periods of infancy and early childhood, between 0 and 5 y.
Our model showed the known trends: faster growth of boys in the
first 6 mo after birth and faster growth of girls after 9 y, when they
begin their pubertal growth spurt (13). Another strength of our study
was the use of different indicators of fat mass and fat-free mass
obtained by both anthropometry and bioimpedancemetry.

However, we may discuss the generalizability of our results
because our population is not representative of French adolescents
and have a relatively low prevalence of obesity compared with
contemporary studies of French children (39, 40). This may have
reduced the variability and thus the power of the study, and the
main consequence would be an underestimation of the associa-
tions with fat mass in adolescence. The concordance of the results
with that of the other studies was the most convincing argument
that our results are not biased or chance findings. Moreover,
because of the constraints of the model in terms of number of
weight and height measurements required per child, we only used
66% of our initial population. Nevertheless, anthropometric vari-
ables for children whose weight growth velocities could or could
not be used in the models were not statistically different, except
for age in boys. Another weakness is the absence of accurate
measures to estimate fat and fat-free mass, measures such as
dual-energy X-ray absorptiometry or air-displacement plethys-
ography. However, the few results in common with a study
using air-displacement plethysmography (12) were similar to our
results. Last, our adolescent children were cross-sectionally ex-
amined at different ages between 8 and 17 y, a wide period of
ages. We adjusted all the adolescent measures for age, Tanner
stage, and, when it could be a supplementary confounding factor,
for height. We also tested the interaction between correlated
variables and age period, and such an interaction was only sig-
nificant with weight growth velocities in the relation with fat
mass variables in girls, with correlations lower in the older than
in the younger girls (ie, P for interaction < 0.05). We postulate
that the changes in fat mass induced by puberty, and by age, may
overcome the early childhood effect in the older girls.
Conclusion

In conclusion, our study supports the hypothesis that there are 2 critical periods in early childhood that are particularly associated with the later risk of overweight or obesity and not determined by the same factors because weight growth during these 2 periods is not significantly correlated, as was also observed in another study (11). The first months of life could be more critical for girls than for boys, because in girls this period of growth is significantly correlated with fat mass only, not with fat-free mass. The second period may be the most critical in the ages studied, as observed in the present study and in another study (21), for both sexes because higher weight growth velocities at 3 and 5 years appear more strongly related with gains in fat mass than in fat-free mass. In between, we suggest a period of preferential height growth. Before more knowledge is accumulated on that later period, our finding encourages to carefulness for any intervention during this “plateau” period: limiting energy intake could have an impact on height development rather than on overweight. For now, public health measures should probably focus on the 2 critical periods.

We thank the directors and teachers of the schools who made the study possible, the parents and their children who agreed to participate in the study, and Laboratoires Knoll, CEDUS, Groupe Fournier, Lesieur, Nestlé France, and Laboratoires de Recherche et Développement de Fournier Labo of Fournier Group for their financial support. The authors’ responsibilities were as follows—JB: conducted the data analyses and drafted the manuscript; BH and IM: provided statistical expertise (IM contributed more particularly to the growth model); M-AC: supervised the study; JB, BH, and M-AC: participated in the interpretation of the results and the writing of the manuscript; and BH, PD, and M-AC: provided critical input and advice concerning the analyses. All authors contributed to the revision of the manuscript. None of the authors had any conflicts of interest.

REFERENCES


