The Terneuzen Birth Cohort: BMI Changes between 2 and 6 Years Correlate Strongest with Adult Overweight

Marlou L. A. De Kroon1*, Carry M. Renders1, Jacobus P. Van Wouwe2, Stef Van Buuren2,3, Remy A. Hirasing1

1 Department of Public and Occupational Health, The EMGO Institute for Health and Care Research, VU University Medical Centre, Amsterdam, The Netherlands, 2 Netherlands Organisation for Applied Scientific Research, TNO Quality of Life, Prevention and Health Care, Leiden, The Netherlands, 3 Department of Methodology and Statistics, Faculty of Social Sciences, University of Utrecht, Utrecht, The Netherlands

Abstract

Background: Complications of overweight amplify with age, and irreversible damage already exists in young persons. Identifying the most sensitive age interval(s) for adult overweight is relevant for primary prevention. The aim of the study was to assess the relative contribution of body mass index (BMI) changes between 0 and 18 years to adult overweight, and to identify the earliest critical growth period.

Methods and Findings: Data from 762 subjects in the Terneuzen Birth Cohort with an average of 21 growth measurements per subject from birth until 18 years were used. The main outcome measure was the BMI standard deviation score (SDS) at young adulthood. For each subject BMI SDS was fitted by a piecewise linear model at eight different ages and correlated to adult BMI SDS. The age intervals in between are considered critical according to three criteria, tested by respectively Students’ t-tests, multiple linear regression analyses and Pearson’s correlation tests. In the age intervals 4 months(m) -1 year(y), 2–6 y, 6–10 y and 10–18 y the BMI SDS change differs between adults with and without overweight (P<0.001). The age intervals 2–6 y and 10–18 y also meet the second criterion, implying that the BMI change during this period has a predictive value for adult BMI SDS in addition to BMI SDS at the end of the period. The largest rise in correlation between estimated BMI SDS and measured adult BMI SDS occurs during the period 2–6 y (from 0.36 to 0.63), which results in a high sensitivity (0.6) and specificity (0.8) by the age of 6 y.

Conclusions/Significance: The age interval from 2 y to 6 y is the earliest and most critical growth period for adult overweight. Therefore, primary prevention of adult overweight seems most likely to be successful if targeted at this specific age interval. By identifying those with an upwards centile crossing between 2 and 6 years, the development towards adult overweight might be reversed.

Introduction

The effect of overweight on later cardiovascular health problems amplifies with age [1], and irreversible precursors of diabetes and cardiovascular disease already exist in young persons [2]. Not only is weight in itself a risk factor, but so is also a fast BMI increase during childhood [1,3–7]. For the prevention of adult overweight, research has focused on the identification of sensitive or so-called ‘critical’ growth periods. A growth period is critical for adult overweight if changes within this period increase the risk of adult overweight [8]. Several investigators have distinguished growth periods with increased risk [4,9–17].

Figure 1 illustrates how the BMI standard deviation score (BMI SDS) in five hypothetical growth patterns evolves into adult overweight. The first pattern is a simple trajectory with a constant increase in BMI SDS over a prolonged time interval, e.g. 0–20y. Every period seems to be critical here (a ‘long critical’ period). Another simple trajectory occurs if children are already overweight at birth and remain overweight until adulthood, so in essence no critical period exists (‘no critical’ period). By contrast, the ‘short early’ and ‘short late’ trajectories have large increases in BMI SDS during short time periods. The rise in BMI SDS could also be broken into a smaller number of critical periods, e.g. ‘two critical’ periods. The last three patterns (‘short early’, ‘short late’ and ‘two critical’ periods) suggest that prevention opportunities are to be found before or within the periods of BMI SDS increase, rather than after. In all situations statistical evidence is required to confirm that changes in BMI SDS effectively influence the risk of adult overweight.

Few studies have followed the BMI changes in children from birth to adulthood; most studies limit themselves to a time interval during childhood [9,10,12–14,18–21] or have a follow-up that does not exceed puberty [11,12,17,20,21]. Also, their results are sometimes contradictory [6,16,20,21]. Two recent studies without these shortcomings included respectively males only [16] and no Caucasians [15].
We aim to assess the relative contribution of BMI SDS changes between 0–18 y of age to adult overweight, and to identify the earliest relevant, critical growth period for adult overweight.

Methods

Population and Study Design

The original cohort consists of all 2,604 children born between 1977 and 1986 in the city of Terneuzen. Of the 1,701 subjects data for weight and length as routinely registered by the Municipal Health Services were available from birth. Of these subjects, 762 persons (45%) were willing to participate in a follow-up study at young adulthood that included measurements of weight, height and waist circumference and a questionnaire to collect sociodemographic characteristics. This is described in more detail elsewhere [22]. The participants in the follow-up study did not differ from the original cohort regarding baseline characteristics collected at birth, e.g. date of birth, birth weight, BMI SDS at birth, age of the mother, and parity, except for gender (41% were males vs 51% in the original cohort, $P<0.05$). We used BMI values (kg/m²) as the measure for (over-)weight, converted to age-specific standard deviation scores (BMI SDS) based on Dutch reference data [23], because these are most comparable to our study population. The criterion for being overweight in young adulthood is defined as BMI SDS $\geq 1.3$ (roughly a BMI $\geq 25$).

In contrast to most studies that are limited to a specific period (infancy, childhood or adolescence) and lack of follow-up to adulthood, our cohort covers the complete growth from birth to adulthood. For comparison purposes with other studies, we divided the growth period of our cohort into the following age intervals: 0–8 days (0–8 d) [9], 8 days-4 months (8 d-4 m) [9], 4 months-1 year (4 m-1 y) [24], 1-2 years (1–2 y) [4], 2–6 years (2–6 y) [12], 6–10 years (6–10 y), and 10–18 years (10–18 y) [14,25]. The upper limit in the age interval 6–10 y was set since Dutch children go into puberty after 10 years of age; the upper limit of 18 years marks the start of adulthood. The limits of all periods (0 d, 8 d, 4 m, 1 y, 2 y, 6 y, 10 y and 18 y) are called break ages.

Statistical Analysis

The major analytic problem was that the number and the timing of the measurements vary between individuals. We solved this by fitting each individual BMI SDS trajectory by a piecewise linear model, otherwise known as a broken stick-model [26], with the knots set equal to the break ages. We also dealt with missing data in this way. This model approximates each person’s observed BMI SDS trajectory by a series of straight lines that connect to each other at the break ages. In order to stabilize the parameter estimates, we fitted these parameters as randomly varying slopes in a linear multilevel model [27]. We used the S Plus 8.0 function bs() to code the data into the appropriate form, and used the function lm() to estimate the parameters as random effects. The procedure resulted in eight parameters per person that together describe the persons’ BMI SDS trajectory. Each parameter corresponds to the predicted value for each individual, using both random and fixed estimates. We call these status scores. They are represented as $Z_{0d}, Z_{8d}$, and so on. The change in BMI SDS per period is equivalent to the difference between two successive status scores, i.e. $Z_{8d}-Z_{0d}, Z_{4m}-Z_{8d}$, and so on. We call these change scores.

We define a growth period, bounded by ages T1 and T2, as critical if:
a. the mean change score \( z_{T2}-z_{T1} \) is significantly different between those with and without adult overweight,
b. the change score \( z_{T2}-z_{T1} \) and \( z_{T2} \) are both significantly related to adult BMI SDS in a multiple regression analysis, which is, as has been suggested by Lucas [28], equivalent to the significance of \( z_{T1} \) as predictor in addition to the significance of \( z_{T2} \) as predictor (see Addendum S1 for further explanation), and
c. the score \( z_{T2} \) is relatively close to BMI SDS at adult age.

Criterion a will filter out periods during which the two mean curves of the BMI SDS trajectory diverge, so significant differences in growth of those who do and those who do not become overweight emerge. Criterion b indicates if the preceding change score has additional value to the status score at the end of the period, in predicting the BMI SDS at adulthood. Criterion c will select periods for which it is easier (i.e. with higher sensitivity and specificity) to identify children at risk for adult overweight.

We tested for these criteria in SPSS 14.0 by applying Student’s t-tests (2-sided), Pearson’s correlation coefficients and multiple regression analysis (with alpha = 0.05 for statistical significance). In the multiple regression analyses multiplicative interaction effects were entered to explore whether early weight is modifying the effect of later weight size on adult overweight with a type I error rate of 0.10 [29]. Age, gender, parity, exclusive breastfeeding (duration did not influence the results) were entered to explore whether early weight is modifying the effect of later weight size on adult overweight. Significant differences were found for four age intervals: 4 m-1 y, 2-6 y, 6-10 y and 10-18 y. No differences in the change scores were found for the age interval 1-2 y (Table 2). Similar results were found for males and females, although for the age interval 4 m-1 y the difference became non-significant in males (P = 0.078). In addition t-tests were applied to test differences in BMI SDS changes for those with or without increased waist circumference at young adulthood as defined by IDF criteria. These analyses showed significant results for exactly the same intervals (P<0.001).

The results of the multiple regression analyses to test criterion b are shown in Table 3, in which adult BMI SDS is the outcome, and the status score(s) the predictor(s). Because no effect-modification was found for gender (P>0.3), in applying multiple regression analyses and correlation coefficients males and females could be analyzed as one group, increasing statistical power. As parity and breastfeeding duration did not influence the results (P>0.05), these variables were not included in the final models. Not surprisingly, in the simple linear regression analyses BMI SDS is significantly related to adult weight at all ages. After including the previous status score as a second (linear) predictor, only two age intervals, 2–6 y and 10–18 y, met both criterion a and criterion b. Moreover, these periods are both characterized by significant predictors with opposite regression signs, which means that especially the BMI SDS changes in these age intervals are relevant [28].

---

**Table 1.** General characteristics at birth and at adulthood, number of subjects (N), and their mean (SD) number of height and weight measurements per age interval.

| Characteristics | Males | | | Females | | |
|----------------|-------|------|-------|----------|------|-------|
|                | N     | Mean | SD    | N        | Mean | SD    |
| birth weight (g) | 830   | 3481.4 | 549.3 | 870      | 3348.2 | 541.0 |
| birth length (cm) | 804   | 50.9 | 2.5  | 839      | 50.2 | 2.2   |
| gestational age (wk) | 765   | 39.9 | 1.7  | 819      | 39.8 | 2.4   |
| adult height (cm) | 307   | 182.6 | 6.8  | 455      | 169.6 | 6.2   |
| adult weight (kg) | 307   | 77.0 | 12.3 | 455      | 67.8 | 11.9  |
| adult BMI (kg/m²) | 307   | 23.1 | 3.4  | 455      | 23.4 | 3.9   |

| Age interval | height | weight | height | weight |
|--------------|--------|--------|--------|--------|
|              | N | mean (SD) | N | mean (SD) | N | mean (SD) | N | mean (SD) |
| 0-8 d       | 810 | 1.0 (0.1) | 1311 | 2.6 (1.8) | 852 | 1.0 (0.1) | 1284 | 2.5 (1.9) |
| 8 d-4 m     | 735 | 2.8 (1.1) | 754 | 4.3 (1.7) | 799 | 2.7 (1.1) | 818 | 4.5 (1.9) |
| 4 m-1 y     | 751 | 4.8 (1.6) | 753 | 5.4 (1.8) | 815 | 4.8 (1.5) | 818 | 5.6 (2.0) |
| 1-2 y       | 709 | 1.8 (0.7) | 710 | 1.8 (0.8) | 765 | 1.8 (0.7) | 767 | 1.8 (0.7) |
| 2-6 y       | 802 | 2.7 (0.9) | 804 | 2.7 (1.0) | 848 | 2.7 (0.9) | 850 | 2.7 (0.9) |
| 6-10 y      | 734 | 1.6 (0.6) | 735 | 1.6 (0.6) | 787 | 1.6 (0.7) | 788 | 1.6 (0.7) |
| 10-18 y     | 723 | 1.7 (0.8) | 724 | 1.7 (0.8) | 766 | 1.6 (0.7) | 767 | 1.6 (0.7) |

doi:10.1371/journal.pone.0009155.t001
Figure 2. Broken sticks trajectories of BMI SDS changes. A. Broken sticks trajectories for subjects with normal weight (green lines) versus subjects with overweight (red lines) at young adulthood, B. Broken-stick model of mean increments for subjects with normal weight and with overweight at young adulthood. The green, dotted line represents the mean increments of subjects with normal weight, the red line the mean increments of subjects with overweight at young adulthood.

doi:10.1371/journal.pone.0009155.g002
the smaller time intervals between birth and the age of 2 years to one age interval, in order to assess if the length of the age intervals influenced the results of the analyses. However, no significant effect has been shown by adding the status score at birth to the status score at 2 y; the increase in explained variance is zero; (β in the multiple regression model at 2 y and at 0 d are respectively 0.548 (SE 0.055, P=0.145) and 0.703 (SE 0.065, P=0.039). The increase of explained variance caused by including BMI-SDS at T0 into the model containing BMI-SDS at T1 was largest for the period 2–6 y. This implicates that the influence of the change score on adult overweight is largest for the age interval 2–6 y. Because the relative changes in regression signs after extending the models is highest in the age interval 2–6 y, especially in this age interval upwards centile crossing is an additional risk to the status score at the end of these age intervals (see Addendum S1). For comparisons reasons with a recent study [16], we added an additional breakpoint at 4 y, and found that the proportion of increased variance as a function of the status score at the end of the period for the age intervals 2–4 y and 4–6 y are respectively 0.03 and 0.04 by adding the status score at the start of the period to the model. In modeling the Z-score of the waist circumference at young adulthood as the outcome measure (number of missing outcomes is 5), we obtained similar results for the age interval 2–6 y. In the multiple regression the coefficients of the status scores at 6 y and at 2 y coefficients are respectively 0.31 (SE 0.09, P<0.001) and −0.14 (SE 0.00, P=0.040), with an increased explained variance of 3% by augmenting the model with the preceding BMI SDS. Finally, because extreme high BMI at adulthood is more closely related to fat mass than lower values of BMI, we performed additional analyses by using adult obesity (BMI ≥20) as the outcome. These analyses identified only the period 2–6 y as critical (OR of BMI SDS at respectively 6 y and 2 y were 41.27, 95% CI 15.8–107.7 and 0.24, 95% CI 0.12–0.50), whereas none of the other periods were found to comply with the conditions of a critical period.

Criterion r was assessed by Pearson’s correlation between status scores and adult BMI SDS (Figure 3). From the age of 6 years onwards the correlation between the status score and adult BMI SDS is greater than 0.6, which implies that prevention of a (relatively) high BMI SDS at the age of 6 y is relevant in terms of health outcome at adulthood.

**Table 3.** Linear relation between BMI SDS at young adulthood and BMI SDS at earlier age: Model A includes one status score as independent variable (a), model B is model A extended with the preceding BMI SDS as (b) independent variable.

| Independent variables | Models A | Models B |
|-----------------------|----------|----------|
|                       | β        | SE       | P        | Adj R²   | β₁        | SE       | P        | Adj R²   |
| (a) BMI sds at birth  | 0.158    | 0.047    | 0.001    | 0.035    | –         | –        | –        | –        |
| (b) BMI sds at 8 d    | 0.320    | 0.057    | <0.001   | 0.060    | 0.390     | 0.084    | <0.001   | 0.061    |
| (c) BMI sds at 4 m    | 0.307    | 0.060    | <0.001   | 0.054    | 0.210     | 0.066    | 0.002    | 0.071**  |
| (d) BMI sds at 8 d    | 0.239    | 0.063    | <0.001   | –        | 0.591     | 0.068    | <0.001   | 0.138    |
| (e) BMI sds at 1 y    | 0.562    | 0.057    | <0.001   | 0.138    | 0.591     | 0.068    | <0.001   | 0.138    |
| (f) BMI sds at 4 m    | 0.359    | 0.053    | <0.001   | 0.146    | 0.346     | 0.081    | <0.001   | 0.158**  |
| (g) BMI sds at 1 y    | 0.291    | 0.083    | 0.001    | –        | 0.291     | 0.083    | 0.001    | –        |
| (h) BMI sds at 6 y    | 1.095    | 0.049    | <0.001   | 0.407    | 1.583     | 0.077    | <0.001   | 0.045**  |
| (i) BMI sds at 2 y    | 1.014    | 0.035    | <0.001   | 0.530    | 1.126     | 0.080    | <0.001   | 0.530    |
| (j) BMI sds at 10 y   | 0.155    | 0.099    | 0.017    | –        | 0.155     | 0.099    | 0.017    | –        |
| (k) BMI sds at 6 y    | 1.065    | 0.020    | <0.001   | 0.790    | 1.292     | 0.041    | <0.001   | 0.790**  |
| (l) BMI sds at 10 y   | –0.305   | 0.047    | <0.001   | –        | –0.305    | 0.047    | <0.001   | –        |

*Intercepts not reported  
Interaction effects between BMI SDS at the end and at the start of the periods were not included in models B, because they were all non-significant.

F-test for comparing the multiple regression model with the simple regression model is significant (P<0.001).

Values of β, β₁, and β₂ are adjusted for gender and the age at the measurement of BMI SDS at young adulthood. – Not applicable, Adj R² adjusted variance.
that the age interval 2–6 years is especially important to develop strategies for primary prevention of overweight. Our study shows that two children with identical BMI SDS at age 6 y have different risks for becoming overweight depending on their BMI SDS at 2 y. Also the correlation with adult overweight rises most during this age period (from 0.36 to 0.63), indicating that the rise in sensitivity and specificity for predicting adult overweight based on childhood BMI SDS in this period is high. Ideally, primary prevention should be realized before the point of high sensitivity and specificity has been reached. For the age interval 6–10 y and up to the age of 2 years, change scores are not very predictive for adult overweight.

At first sight, our results deviate from the GOOD study in young male adults [16]. In this study both early and late childhood (defined as 1–4 y and 4–10 y) were found to be predictors of adult BMI. Their breakpoint was chosen at 4 y which is exactly in the middle of the age interval 2–6 y. By additional analyses, we found that the age intervals 2–4 y and 4–6 y are quite similar in terms of their predictive ability. Therefore it might be possible that the predictive value of the early child period in the GOOD study might be mainly explained by the predictive value of the period 2–4 years and the predictive value of the late childhood period mainly by the period 4–6 years.

We found that changes in BMI SDS up to the age of 2 years have hardly any predictive value for adult overweight. The change score in the period 4m-1y differs significantly between adults with and without overweight, but this effect disappears once BMI SDS at 1 y is included in the statistical model. Thus, a change score in the age interval from 4 months to 1 year of age seems not to correlate with a higher adult overweight risk at the age of 1 year.

Our study confirms the results from other studies that growth during certain age intervals in childhood are more sensitive in predicting overweight. However the explanation for these ‘critical’ growth periods is still unclear [4,9,12,14,24,25]. It is possible that changing relations between BMI SDS and fat, lean and bone mass at different ages [30] and other biological explanations concerning the changing growth velocity of fat tissue play a role [15,16,17,31,32]. The results of this study did not show that rapid growth during the first years of life is a predictor for adult overweight, which is in contrast to the results from similar studies.

### Table 4. Summary of the results of the analyses and the interpretation per age interval based on criteria a, b and c.

| Age interval | Criterion a. Relation with adult overweight according to Students t tests | Criterion b. Significance of $Z_{11}$ as predictor in addition to the significance of $Z_{12}$ as predictor in a multiple linear regression analyses | Criterion c. High correlation of BMI SDS at a certain age with BMI SDS at adulthood at the end of the period | Critical age interval based on results of analyses concerning criteria a, b and c. | Confirmation of other study results [reference(s)] |
|--------------|--------------------------------------------------------------------------|---------------------------------------------------------------------------------|---------------------------------------------------------------------------------|---------------------------------------------------------------------------------|---------------------------------------------------------------------------------|
| 0–8 d        | NS                                                                       | NS                                                                              | no                                                                              | no                                                                              | no [9]                                                                           |
| 8 d–4 m      | NS                                                                       | yes**                                                                           | no                                                                              | no                                                                              | yes [9,10,17]                                                                   |
| 4 m–1 y      | yes*                                                                    | NS                                                                              | no                                                                              | no                                                                              | partly, concerning results of t-tests [10]                                      |
| 1–2 y        | NS                                                                       | yes*                                                                           | no                                                                              | no                                                                              | yes [17]                                                                       |
| 2–6 y        | yes**                                                                   | yes**                                                                           | yes                                                                              | yes                                                                              | yes [5,15,21]                                                                   |
| 6–10 y       | yes**                                                                   | NS                                                                              | yes                                                                              | yo                                                                              | possibly, not validated yet [16]                                               |
| 10–18 y      | yes**                                                                   | yes**                                                                           | yes                                                                              | yes                                                                              | yes [25]                                                                       |

NS not statistically significant (p>0.05),

*P<0.001, **P<0.001.

doi:10.1371/journal.pone.0009155.t004
Possible explanations are a shorter follow-up [17], the selection of the study population [9,15], or higher statistical power due to a larger study population [13]. Our conclusion that the age period between 2 and 6 years emerges to be critical for adult (over-)weight confirms other study results, that show a rapid elevation in the deposition of body fat rather than lean tissue mass just before the age of 6 years in children with a related early adiposity rebound (AR) [33,34]. Other studies have also pointed to this crucial age period, with an early AR as a risk for adult overweight [4,7,9,13,20,21]. The importance of adolescence for developing adult overweight was also reported in another study [16], which showed that changes in BMI SDS during adolescence reflect changes in visceral fat mass, more than in other periods.

The strengths of our study are that it was carried out in a general population, and weight and height were frequently measured between 0 and 14 years according to the protocol used within Youth Health Care. The addition of protocolized measurements of weight and height at adulthood offered the opportunity to study the importance of all subsequent growth periods from birth to adulthood in the prediction of overweight at young adulthood. We also had to deal with limitations. As in most birth cohort studies, there was a substantial loss in the follow-up. Therefore sampling bias might be possible. However, there is no reason to assume that loss to follow up is related to the strength of the relation between BMI changes in childhood and adult BMI. Moreover, no significant differences were found for the baseline characteristics and the study population of the original cohort except for gender.

Another limitation of our study is that we had to deal with missing data. This problem was solved by applying the broken stick method. The broken stick method results in estimates that are closer to the mean. This implies that any tests of differences will be conservative, and possibly underestimates the effects of BMI changes in periods in which fewer measurements are recorded. Also using BMI SDS (changes) as a predictor and as an outcome has limitations, although the correlation between BMI SDS and body fat% is reasonable and increases from 0.62 to 0.78 (between the ages of 3.5 and 7 years) [21]. Post hoc analyses with waist circumference, a proxy of central fat tissue considered most harmful to health [31,35], and with adult obesity as the outcome measure, showed similar results for the period 2–6 y. This strengthens our impression that BMI SDS change, especially in the period 2–6 y, has a strong relationship with bodyfat% over the years. More fundamental research is needed to study the age dependency of the relation between BMI and several body components.

Our study indicates that the BMI change between 2 and 6 years of age (and, to a lesser degree, the age interval 10–18 y) has relatively the largest contribution to adult overweight. It would be interesting to study if in younger cohorts, living in an increasingly obese society, the age interval between 2 and 6 years is also more predictive for adult overweight than other age intervals. If replicated in other studies, primary prevention of overweight should be more directed towards upwards centile crossing in the age interval 2–6 years. Especially in children with a normal weight, this may have a large payoff in terms of overweight reduction at adulthood, and the development towards adult overweight might be reversed.

Supporting Information

Addendum S1 Explanation of criterion b of the definition of a critical growth period. Found at: doi:10.1371/journal.pone.0009155.s001 (0.02 MB DOC)

Acknowledgments

We gratefully thank all participants for their time, the assistants for their contribution in the field work, the laboratory of the Community Hospital in the city of Terneuzen, especially Ruud Munzze, PhD, the Municipal Health Services of Terneuzen (GGD Zeeland) for support and cooperation, and Guus A. de Jonge, PhD, professor emeritus, for laying the foundations of this study in 1977–1986.

Author Contributions

Analyzed the data: MLADK SVB. Wrote the paper: MLADK CMR JPVW SVB RAH. Performed the study design: MLADK.

References

1. Singhal A, Lucas A (2004) Early origins of cardiovascular disease: is there a unifying hypothesis? Lancet 363: 1642–45.
2. Tounian P, Aggoun Y, Dubern B, Varille V, Guy-Graaud B, et al. (2001) Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study. Lancet 358: 1490–4.
3. Barker DJP, Osmond C, Forsén T, Kajantie E, Eriksson JG (2005) Trajectories of growth among children who have coronary events as adults. N Engl J Med 353: 1802–09.
4. Monteiro POA, Victora CG, Barros FC, Monteiro LMA (2003) Birth size, early childhood growth, and adolescent obesity in a Brazilian birth cohort. Int J of Obesity 27: 1274–1282.
5. McCarthy A, Hughes R, Tilling K, Davies D, Smith GD, et al. Birth weight; postnatal, infant, and childhood growth; and obesity in young adulthood: evidence from the Barry Caerphilly Growth Study. Am J Clin Nutr 86: 907–15.
6. Eriksson J, Forsén T, Osmond C, Barker D (2003) Obesity from cradle to grave. Int J of Obesity: 727–727.
7. Baird J, Fisher D, Lucas P, Kleijnen J, Roberts H, et al. (2005) Being big or growing fast: systematic review of size and growth in infancy and later obesity. BMJ 331: 929–931.
8. Dietz WH (1994) Critical periods in childhood for the development of obesity. Am J Clin Nutr 59: 955–959.
9. Stettler N, Zemel BS, Kumanayika S,stellings VA, Troxel AB, Zhao J, Schinnar R, et al. (2005) Comparison of weight gain in the first week of life and overweight in adulthood. Circulation 111: 1897–1903.
10. Ong KKL, Ahmed ML, Emmett PM, Preece MA, Dunger DB (2000) Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. BMJ 320: 967–971.
11. Whitaker RC, Pepe MS, Wright JA, Seidell JK, Dietz WH (1998) Early adiposity rebound and the risk of adult obesity. Pediatrics 101(3): 1–6.
12. Ong KKL, Ahmed ML, Emmett PM, Preece MA, Dunger DB (2000) Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. BMJ 320: 967–971.
13. Botton J, Heude B, Maccario J, Ducimetie`re P, Charles M-A, et al. (2008) Growth patterns and obesity development in overweight or normal-weight 13-year-old adolescents: The STRIP study. Pediatrics 122: e656–e663.
21. Blair NJ, Thompson JM, Black PN, Becroft DM, Clark PM, et al. (2007) Risk factors for obesity in 7-year-old children: the Auckland Birthweight Collaborative Study. Arch Dis Child 92: 866–871.
22. De Kroon ML, Renders CM, Kuipers EC, van Wouwe JP, van Buuren S, et al. (2008) Identifying metabolic syndrome without blood tests in young adults - the Terneuzen Birth Cohort. Eur J Pub Health 6: 656–60.
23. Fredriks AM, Van Buuren S, Wit JM, Verloove-Vanhorick SP (2000) Body index measurements in 1996-7 compared with 1980. Archives of Diseases in Childhood 82: 107–12.
24. Fredriks AM, Van Buuren S, Wit JM, Verloove-Vanhorick SP. (2000) Body index measurements in 1996-7 compared with 1980. Archives of Diseases in Childhood 82: 107–12.
25. Scholtens S, Gelring U, Brunekreef B, Smit HA, de Jongste JC, et al. (2007) Breastfeeding, weight gain in infancy, and overweight at seven years of age. Am Journal of Epidemiology 163(8): 919–926.
26. Oren A, Vos LE, Uiterwaal CS, Gorissen WH, Grobbee DE, et al. (2003) Change in body mass index from adolescence to young adulthood and increased carotid intima-media thickness at 28 years of age: the Artherosclerosis Risk in Young Adults Study. Int J Obes Relat Metab Disord 27: 1383–90.
27. Snijders TAB, Bosker RJ (1999) Multilevel analysis. London: Sage.
28. Lucas A,Fewtrell MS, Cole TJ (1999) Fetal origins of adult disease - the hypothesis revisited. BMJ 319: 245–249.
29. Jones HE, Spiegelhalter DJ (2009) Accounting for regression-to-the-mean in tests for recent changes in institutional performance: analysis and power. Stat Med 30(12): 1645–67.
30. Freedman DS, Wang J, Thornton JC, Mei Z, Sopher AB, et al. (2009) Classification of body fatness by Body mass-index-for-age categories among children. Arch Pediatr Adolesc Med; 163(9): 805–811.
31. McCarthy A, Hughes R, Tilling K, Davies D, Smith GD, et al. (2007) Birth weight, postnatal, infant and childhood growth and obesity in young adulthood: evidence from the Barry Caerphilly Growth Study. Am J Clin Nutr 86: 907–13.
32. Martorell R (1995) Results and implications of the INCAP follow-up study. J Nutr 125(suppl): 1127S–38S.
33. Cole TJ (2004) Children grow and horses race: is the adiposity rebound a critical period for later obesity? BMC Pediatrics 4: 6.
34. Taylor RW, Grant AM, Goulding A, Williams SM (2003) Early adiposity rebound: review of papers linking to this to subsequent obesity in children and adults. Curr Opin Clin Nutr Metab Care 8: 607–612.
35. Wells JCK, Chomtho S, Fewtrell MS (2007) Programming of body composition by early growth and nutrition. Proceedings of the Nutrition Society 66: 425–434.