BMI Changes during Childhood and Adolescence as Predictors of Amount Adult Subcutaneous and Visceral Adipose Tissue in Men – the GOOD Study

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ABSTRACT

Objective. The amount of visceral adipose tissue is a risk factor for the metabolic syndrome. It is unclear how body mass index (BMI) changes during childhood and adolescence predict adult fat distribution. We hypothesized that there are critical periods during development for the prediction of adult subcutaneous and visceral fat mass by BMI changes during childhood and adolescence.

Research Design and Methods. Detailed growth charts were retrieved for the men participating in the population-based Gothenburg Osteoporosis and Obesity Determinants (GOOD) study (n=612). Body composition was analysed using Dual X-Ray Absorptiometry and adipose tissue areas using abdominal computed tomography at 18-20 years of age.

Results. The main finding in the present study was that subjects with increases in BMI Z-score of >1 SD during adolescence had, independent of prepubertal BMI, both larger subcutaneous (+138%; p<0.001) and visceral adipose tissue areas (+91%; p< 0.001) than subjects with unchanged BMI Z-score. In contrast, subjects with increases in BMI Z-score of >1 SD during late childhood had larger amount adult subcutaneous adipose tissue (+83%; p< 0.001) than subjects with unchanged BMI Z-score, but unaffected amount of visceral adipose tissue. BMI changes during adolescence predict both visceral and subcutaneous adipose tissue of the abdomen while BMI changes during late childhood predict only the subcutaneous adipose tissue.

Conclusions. The amount of visceral adipose tissue in young adult men was associated with BMI changes specifically during adolescence, while the amount of subcutaneous adipose tissue was associated with BMI changes during both late childhood and adolescence.
Childhood obesity has developed into an epidemic in the Western world and not only adults but also children are now treated for metabolic syndrome disorders (1). Obesity is an important risk factor for the development of diabetes and cardiovascular disease, and in the third US National Health and Nutrition Examination Survey (NHANES, 1999-2002) study, 16.8% of boys aged 6-19 were overweight and 31.8% were overweight or at risk for overweight (1; 2).

A large amount of visceral fat is a well-known risk-factor for the metabolic syndrome and for cardiovascular disease (3; 4). A recent study demonstrated that although the amount of subcutaneous adipose tissue is associated with the metabolic syndrome, the amount of visceral adipose tissue remains more strongly related and is therefore regarded as an independent risk-factor (5).

Anthropometric measures of obesity in childhood, such as BMI, have previously been shown to be associated with measures of obesity in adulthood (6). The concept of childhood and adolescent BMI changes as predictors for adult obesity, as well as adult fat mass and distribution, is attractive since BMI is easily obtained from standardized growth charts including height and weight. It has been reported that both BMI and obesity track from childhood to adulthood, the closer to adulthood, the stronger the tracking (7-9). Recent studies have also linked rapid alterations in BMI during childhood to an increased number of coronary events and impaired glucose tolerance (10-12).

In the New Delhi birth cohort study by Sachdev et al, children were followed with anthropometric measurements from birth until 21 years of age with the objective to identify indicators of adult body composition (13). The authors reported that BMI during early childhood predicted anthropometric indexes of adult lean mass stronger than anthropometric indexes of adult adiposity in Indian children. In contrast, BMI at 8 years of age was a clear predictor of indicators of adiposity in these children. Importantly, analyses of visceral fat depots were not performed in the New Delhi birth cohort study.

Dietz et al have previously identified both “adiposity rebound” (around 5 years of age after which BMI starts to incline) and adolescence as critical periods for the onset of obesity (14; 15). We and others have found support for the association between puberty and fat accumulation (16-20). We demonstrated an association between early puberty and high BMI at adult age in males and showed that pubertal timing, independently of prepubertal BMI, predicts a central pattern of fat distribution and visceral fat depots (16). Furthermore, Fox et al reported that the intra-abdominal/subcutaneous fat ratio increased during early puberty in boys (n=25 (21)). In contrast, in a small study by Brambilla et al, investigating eight obese children, it was found that the amount of intra-abdominal fat was stable during pubertal development (22). Thus, it is unclear if BMI changes during childhood and adolescence predict the amount of subcutaneous and visceral fat differentially. We used a well-characterized population-based cohort of Swedish young adult males and hypothesized that there are critical periods during development for the prediction of adult subcutaneous and visceral fat mass by BMI changes during childhood and adolescence. The main objective with the current study was, therefore, to investigate the role of BMI changes during childhood and adolescence for young adult subcutaneous and visceral fat depots.

**POPULATION AND METHODS**

The Gothenburg Osteoporosis and Obesity Determinants (GOOD) study was initiated with the aim to determine both
environmental and genetic factors involved in the regulation of fat mass, as previously described (23). To be included in the GOOD study, subjects had to be males, 18-20 years of age and willing to participate in the study. There were no other exclusion criteria. The study population was homogenous with respect to ethnicity. 1068 subjects, representative of the general young male population of Gothenburg (23) (data not shown), were enrolled in the study. Of the 1068 subjects, complete growth and weight charts for determination of BMI between 1 and 10 years of age were available for 612 subjects (=growth-chart subsample). Thus, the results presented here were obtained from a sub-sample of the original GOOD cohort. DXA analyses were performed on 610 subjects of the growth-chart subsample while abdominal CT scans were performed on 201 (= the CT-subsample) of the 612 study subjects in the growth chart-subsample. The growth-chart subsample is representative of the entire GOOD cohort in terms of age (GOOD 18.9±0.6 years; growth chart-subsample 18.9±0.5 years, non significant), weight (GOOD 73.9±11.9 kg; growth chart-subsample 73.3±11.5 kg, non significant), height (GOOD 181.4±6.8 cm; growth chart-subsample 182.0±6.8 cm, non significant) and BMI (GOOD 22.4±3.2 kg/m²; growth chart-subsample 22.1±3.1 kg/m², non significant). The CT-subsample is representative of both the growth chart-subsample and the entire GOOD cohort in terms of weight, height, and BMI (CT-subsample weight 72.7±11.2 kg, height 181.9±7.0 cm, BMI 21.9±3.0 kg/m², non significant versus both the entire GOOD cohort and the growth chart-subsample). However, the subjects of the CT-subsample were slightly younger (CT-subsample 18.7±0.5 years) than both the subjects of the entire GOOD cohort and the subjects of the growth chart-subsample (p<0.05).

The GOOD study was approved by the local ethics committee at Gothenburg University. Written and oral informed consent was obtained from all the study participants. 

**Anthropometrical measurements.** Height was measured using a wall-mounted stadiometer and weight was measured to the nearest 0.1 kg as previously described (16; 23).

**Dual X-Ray Absorptiometry (DXA).** Total body fat mass, percentage body fat, total body lean mass and fat mass of the trunk were assessed using the Lunar Prodigy DXA (GE Lunar Corp., Madison, WI USA).

**Abdominal CT analyses of cross-sectional adipose tissue areas.** A previously described CT technique was used to measure the cross-sectional adipose area of the abdomen (16; 24; 25). A single slice at the level of the fourth lumbar vertebra was acquired with a General Electric High Speed Advantage CT system (version RP2, GE Medical Systems, Milwaukee, Wisconsin, USA). First, subcutaneous and visceral adipose tissue areas were measured, then the visceral adipose tissue area was divided into an intraperitoneal and a retroperitoneal adipose tissue area. This enabled both subcutaneous and intra/retroperitoneal adipose tissue areas to be determined.

**Estimation of childhood BMI.** Growth and weight charts were collected for the subjects of the GOOD study and longitudinal growth was curve-fitted according to the Infancy-Childhood- Puberty (ICP) model (26) as previously described (16; 27). The ICP model represents a widely used model for fitting of human growth data to mathematical functions (26). The infancy part is represented by an exponential function, the childhood part by a quadratic function and the pubertal part by a logistic function. The model also includes the transformations between the different phases. By using this model we computed values of height at exact ages.

Body composition including BMI is greatly influenced by pubertal stage, and thereby pubertal timing (28). Complete growth charts with enough data to determine
pubertal timing, requiring several height and weight measurements around the pubertal growth spurt, were not available for the complete growth-chart subsample used in the present study. Therefore we have chosen to use measurements of BMI before and after, but not during, puberty. Collection and processing of childhood data took place between 2003 and 2008. Body weights between 1 and 10 years of age were estimated through fitting of the weight curve for each child using smooth splines (smooth.spline in the R package statistics, the R foundation for statistic computing, Vienna, Austria; www.r-project.org). BMI values between 1 and 10 years of age were then calculated from the estimated values of weight and height.

Statistical analysis. The Z-score, the standardization of a variable relative to the investigated population expressed in SDs, was calculated using the software SPSS 15.0. For each subject (n= 612) in this cohort and for each age, the height was calculated using the ICP model. These values were then used to calculate the Z-score for each subject at each age based on the variation (SD) in calculated height within the presently investigated population at that specific age. The variance of young adult body composition parameters (R²) explained by BMI or BMI Z-score changes and the corresponding BETA coefficients during childhood and adolescence was calculated using linear regression analyses with age-adjusted adult body composition variables as dependent variable. Age-adjustments were performed because the subjects differed somewhat in age at body composition analysis (18-20 years of age, mean±SD 18.9±0.5 years), and were performed between age at fat analysis and the body composition variables. All subsequent statistical calculations were performed using the age-adjusted body composition variables. For the calculations in Figure 2, adjustments for baseline BMI were performed. BMI and fat parameters, derived from body composition analyses have been log-transformed. For all the statistical analyses the software SPSS (version 15.0) was used. Values are given as means ± SD unless otherwise stated.

RESULTS

The population-based GOOD study has been thoroughly described earlier (23). The present study represents a subsample (n=612; =growth-chart subsample) of the original GOOD cohort (n=1068 ) in which subjects with available detailed growth charts from 1-10 years of age were included.

Anthropometrics and measurements of fat (DXA) / adipose tissue areas (abdominal CT) of the subjects participating in the present study are presented in Table 1.

BMI changes during both early and late childhood as well as during adolescence predicted young adult BMI. Average BMI in this cohort declined during early childhood years and adiposity rebound (=the lowest BMI) was reached slightly before six years of age, after which it began to increase (Figures 1a and b). BMI (Figure 1c and e) as well as BMI changes (Figure 2) during both early (age 1-4) and late (age 4-10) childhood were, as expected, clear positive predictors of adult BMI. During adolescence, pubertal stage influences body composition (29). Because inter-individual variations in pubertal onset will confound the calculations during adolescence, the predictive role of BMI for each individual year during adolescence for adult BMI and body composition is not shown. Instead the BETA coefficient for the correlation of the entire adolescence (age 10-19) is shown in Figure 2, demonstrating that, as expected, BMI changes during adolescence also predicted adult BMI.

Adult total body lean mass was predicted by changes in BMI during early childhood and adolescence. BMI alterations during development reflect changes in either fat mass, lean mass or both. In order to study the
role of childhood BMI and BMI changes for the prediction of different adult body compartments, the associations between BMI during development and age-adjusted adult body composition variables were analysed. The variance \((R^2)\) in adult total body lean mass explained by BMI as well as the corresponding BETA coefficients for the correlations increased during early childhood, levelled off during late childhood and increased again during adolescence (Figures 1c,e and 2). The variance in adult BMI and the variance in adult lean mass explained by childhood BMI and the corresponding BETA coefficients for the correlations were similar until 4 years of age when they separated distinctly (Figure 1c and e). The BETA coefficient for the correlation between change in BMI and adult lean mass was higher during adolescence than during late childhood (Fig 2). Thus, BMI changes during early childhood and adolescence but not during late childhood were major determinants of young adult lean mass.

**Adult total body fat mass and subcutaneous adipose tissue area were predicted by changes in BMI during late childhood and adolescence.** The prediction of young adult total body fat mass from BMI and BMI changes during different developmental periods followed a different pattern from that of lean mass. The variances in total body fat mass and percentage total body fat explained by childhood BMI and the corresponding BETA coefficients for the correlation were low before 4 years of age, but after 4 years of age the plotted cumulated variance \((R^2)\) and the corresponding BETA coefficients for the correlation for young adult total body fat mass and percentage body fat followed tightly the curve displaying the variance of adult BMI explained (Figure 1c and e). The predictive value of childhood BMI for young adult percentage total body fat increased substantially during late childhood years, as demonstrated by the fact that the variance in percentage total body fat explained by BMI at 4 years of age was only 1.7% while it was 20.8% at 10 years of age (Figure 1c).

In order to analyse fat distribution in detail we performed abdominal CT scans on a subset of the present GOOD sample = CT-subsample, \(n=201\) for measurements of abdominal subcutaneous and visceral adipose tissue areas. The abdominal CT measurements revealed that the contribution of BMI during development for the explanation of the variance in young adult subcutaneous adipose tissue area and the corresponding BETA coefficients for the correlations followed a pattern similar to that of total body fat mass and percentage total body fat, with a distinct increase after 4 years of age (Figure 1d and f). For both young adult percentage fat mass and subcutaneous adipose tissue area, the BETA coefficients for the correlations were of similar magnitude during late childhood and adolescence but clearly higher than seen during early childhood (Fig 2). Thus, BMI changes during late childhood and adolescence, but not during early childhood, clearly predicted young adult total fat mass and the amount of subcutaneous adipose tissue.

**Adult visceral adipose tissue areas were predicted by changes in BMI specifically during adolescence.** Interestingly, the contribution of BMI during the different developmental periods for the explanation of the variance in amount of young adult visceral adipose tissue, (intraperitoneal + retroperitoneal adipose tissue areas) and the corresponding BETA coefficients for the correlations, followed a different and clearly distinct pattern from that of total body fat mass and subcutaneous adipose tissue area. BMI and BMI changes during early and late childhood contributed only marginally to the explanation of the variance in intraperitoneal adipose tissue area, and the corresponding BETA coefficient for the correlation between
change in BMI and amount of young adult intraperitoneal adipose tissue was low (Figure 1d and f, Figure 2). Instead, adolescence was the developmental period when BMI changes started to contribute substantially to the explanation of the variance in amount adult intraperitoneal adipose tissue, and the corresponding BETA coefficients for the correlations between change in BMI and amount of young adult intraperitoneal adipose tissue were then higher than seen during early and late childhood (Figure 2). The results for retroperitoneal adipose tissue were similar as seen for intraperitoneal adipose tissue (data not shown). Moreover, analyses using weight for age during childhood and adolescence showed, in a similar manner as seen for BMI for age, clear age-dependent associations with adult fat parameters (data not shown). In contrast, analyses of height for age did not show any obvious age-dependent association with fat parameters (data not shown).

Thus, changes in BMI during adolescence, but not during early or late childhood, predicted the amount of adult visceral adipose tissue.

**Changes in BMI Z-score and adult fat mass.** To further describe the association between developmental changes in BMI and adult fat mass/adipose tissue areas, subjects were divided into groups based on change in BMI Z-score (>1 SD, <-1 SD and average BMI Z-score change) during the three different developmental time periods (Figure 3). Subjects with more than 1 SD increase in BMI Z-score during late childhood and during adolescence had clearly higher percentage total body fat mass (late childhood, +44%, p<0.001; adolescence, +60%, p<0.001, Fig 3a) and larger subcutaneous adipose tissue area (late childhood, +83%, p<0.001; adolescence, +138%, p<0.001, Fig 3b) than subjects with average change in BMI. In contrast, subjects with more than 1 SD increase in BMI Z-score during late childhood had unaffected intraperitoneal adipose tissue area compared with subjects with average change in BMI (Fig 3c). However for adolescence, subjects with more than 1 SD increase in BMI Z-score had larger intraperitoneal adipose tissue area (+91%, p<0.001) than subjects with unchanged BMI Z-score (Figure 3c). A specific association between high young adult subcutaneous, but not visceral (intraperitoneal + retroperitoneal), adipose tissue area, and increased BMI Z-score during late childhood is supported by the finding that young adult visceral adipose tissue area adjusted for total adipose tissue area was clearly reduced in subjects with more than 1 SD increase in BMI Z-score during late childhood (Fig 3d). In contrast, an increase in BMI Z-score of more than 1 SD during adolescence rather was associated with an increased visceral adipose tissue area adjusted for total adipose tissue area (Fig 3d). Subjects who decreased their BMI with more than one SD did not differ from those with no change in BMI Z-score for any of the investigated fat parameters (Figure 3a-d). Thus, the amount of visceral adipose tissue was predicted by large increases in standardized BMI specifically during adolescence, while a high amount of subcutaneous adipose tissue area was predicted by increases in BMI during both late childhood and adolescence.

**BMI increases during adolescence predict the amount of adult visceral adipose tissue independently of prepubertal BMI.** We next investigated if BMI increases in subjects with low prepubertal BMI had a similar impact on the amount of adult visceral adipose tissue as seen in subjects with a high prepubertal BMI. Therefore, subjects were divided into tertiles according to prepubertal (10 years of age) BMI (low 15.2±0.8 kg/m², average 16.7±0.4 kg/m² and high 19.3±1.5 kg/m²) and tertiles of BMI-Z-score change (low -1.0±0.6 SD, average -0.1±0.2 SD, and high 0.7±0.6 SD) during adolescence (Figure 4). Importantly, for all three prepubertal BMI groups, subjects
with greater increase in adolescence BMI had significantly larger visceral adipose tissue areas (low prepubertal BMI + 56%, average prepubertal BMI +53% and high prepubertal BMI +57% p<0.05 for all) than subjects with average adolescence BMI increase (Figure 4).

Thus, BMI increases during adolescence predicted the amount of adult visceral adipose tissue independently of prepubertal BMI.

DISCUSSION

Both BMI and obesity track strongly from childhood to adulthood (7-9; 30; 31) but the role for BMI changes during development as predictors of adult body composition and fat distribution is unclear. The main objective of the present study was to investigate the association between BMI changes during different developmental periods and young adult fat distribution in a well-characterized cohort of young adult men. Importantly, we demonstrate that the amount of adult subcutaneous adipose tissue and visceral adipose tissue were associated with BMI changes during distinct developmental periods. In addition, we identified the childhood age after which BMI increases had a clear impact on adult total body fat mass.

Our main finding was that the amount of adult visceral adipose tissue was associated with BMI changes specifically during adolescence, while the amount of subcutaneous adipose tissue was associated with BMI changes during both late childhood and adolescence. Importantly, subjects with more than 1 SD increase in BMI Z-score during adolescence had >90% greater visceral adipose tissue areas than subjects with average change in BMI Z-score. These data, hence, demonstrate that BMI increases during adolescence were associated with larger amount of visceral adipose tissue at young adult age. A large amount of visceral fat is a well-known risk-factor for cardiovascular disease. A recent study demonstrated that although the amount of subcutaneous adipose tissue is associated with the metabolic syndrome, the amount of visceral adipose tissue remains more strongly related and is therefore regarded as an independent risk-factor (5). Increased understanding of how and when subcutaneous and visceral fat compartments are predicted might, therefore, add important knowledge for the prevention of cardiovascular disease. In the present study, BMI increases during adolescence in subjects with a low prepubertal BMI had a similar impact on the amount of adult visceral adipose tissue as in subjects with a high prepubertal BMI. Thus, BMI increases during adolescence predicted the amount of adult visceral adipose tissue independently of prepubertal BMI.

An increase by > 1SD in standardized BMI both during late childhood and during adolescence was, in the present study, associated with approximately 50% more adult percentage total body fat. The New Delhi birth cohort study also demonstrated that BMI changes during late childhood were associated with anthropometric measures of adult fat mass (13). The New Delhi study represents a large longitudinally followed cohort, but indicators of fat mass and lean mass were only derived from anthropometric measurements and consequently the visceral fat could not be determined in that study. Moreover, the differences both in height and weight for age and in nutritional status between the Indian children in the New Delhi birth cohort and children in the Western world (13; 32-34) might also limit the interpretations of the data from the New Delhi birth cohort into the Western world. A major strength of the present study is that both the subcutaneous and the visceral adipose tissue areas were measured using abdominal CT scans, giving us the opportunity to determine the predictive value of developmental BMI changes for these two fat depots separately. In a Danish study, including men born between 1930 and 1956, it was shown that subjects
with both increases and decreases in BMI between 7 and 13 years of age had a higher risk of obesity in adulthood compared to those who maintained their BMI level (35). In contrast, in the present cohort of boys born between 1983 and 1985, increases but not decreases in BMI during growth were associated with increased adult fat mass and BMI. As the subjects in the Danish study were born between 1930 and 1956, it is possible that the decline in BMI during growth is related to environmental factors such as temporary under-nourishment among certain subjects during this period (including the Second World War). Importantly, although we in the present study have characterized the associations between childhood BMI and adult obesity, it is clear that a substantial part of adult obesity can not be explained by childhood obesity (6).

Furthermore, BMI changes during early childhood and adolescence were, in the present study, predicting adult lean mass, while BMI changes during late childhood and adolescence were predicting adult total body fat mass. These findings are in accordance with the findings in the New Delhi birth cohort study, demonstrating that BMI during early childhood predicts anthropometric measures of adult lean mass stronger than anthropometric measures of adult fat mass in Indian children (13). Both the present study and the New Delhi birth cohort study thus support the notion that BMI changes during early childhood are indicators of adult lean mass while BMI changes during late childhood and adolescence are indicators of adult total body fat mass. In addition, our data demonstrate that four years of age was the threshold-age for Swedish boys after which BMI increases were clearly associated with increased adult total body fat mass. A high BMI before this age was an indicator of high adult lean mass. However, it should be emphasized that the present findings are based on association studies and, therefore, should be interpreted with caution.

In conclusion, the present study demonstrates that the amount of young adult subcutaneous adipose tissue was associated with BMI changes both during late childhood and adolescence while young adult visceral adipose tissue areas were associated with BMI changes specifically during adolescence. These findings suggest that avoiding substantial BMI increases during adolescence might, independent of prepubertal BMI, result in lower adult visceral fat mass.

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Table 1. Anthropometrics and fat variables

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<th>Variables</th>
<th>Mean±SD</th>
<th>Median (Range)</th>
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<tr>
<td>Age (yr)</td>
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<td>Length (cm)</td>
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<td>BMI (kg/m$^2$)</td>
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<td>10 year of age</td>
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<td><strong>Adult DXA measurements (n=610)</strong></td>
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<td>Whole body fat (kg)</td>
<td>12,9 ± 7,5</td>
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<td>16,8 ± 7,1</td>
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<td>Whole body lean mass (kg)</td>
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<td>Subcutaneous fat (cm$^2$)</td>
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<td>Intraperitoneal fat (cm$^2$)</td>
<td>22 ± 13</td>
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Values are given as mean ± SD. DXA = Dual X-Ray Absorptiometry, CT = computed tomography, BMI= body mass index
Figure 1. Childhood BMI and variance in body composition and fat variables
(a and b) Body mass index (BMI, a) and BMI change (b) during early and late childhood. Values are expressed as means ± SD. (c-f) Association between childhood BMI between 1 and 10 years of age and young adult body composition parameters expressed as accumulated R² (%, c and d) or BETA coefficients (SD in adult body composition parameter per SD in BMI at that age, e and f). Variables of adult body composition and fat distribution have been age-adjusted. AT= adipose tissue, Sc= subcutaneous, Ip= intraperitoneal.
Figure 2. Correlation between BMI changes during development and adult body composition and fat distribution
Correlation between change in standardized body mass index (BMI) during different developmental time periods and adult body composition and fat distribution parameters (early childhood defined as 1-4 years; late childhood defined as 4-10 years and adolescence defined as 10 years- young adult). Bars indicate BETA coefficient (95% confidence intervals) expressed as SD in investigated adult body composition parameter per SD change in BMI during that growth period. Variables of adult body composition and fat distribution have been age-adjusted. AT= adipose tissue, Sc= subcutaneous, Ip= intraperitoneal. *** p<0.01, * p<0.05 significant association between the developmental time period and the investigated body composition parameter. NS= not significant. 95% confidence intervals are given for the BETA coefficients, making it possible to evaluate the differences in relative contribution of the three time periods for each dependent parameter.
Figure 3. The impact of developmental BMI changes for percentage body fat and fat distribution

The impact of developmental BMI changes for percentage total body fat (a), subcutaneous adipose tissue area (b), intraperitoneal adipose tissue area (c) and visceral (=intraperitoneal + retroperitoneal) adipose tissue adjusted for total abdominal (visceral + subcutaneous) adipose tissue area (d). Subjects with > +1 SD increase were compared with subjects with average change or < -1 SD decrease in standardized BMI during early childhood (1-4 years of age), late childhood (4-10 years of age) and adolescence (10-19 years of age).

Variables of adult body composition and fat distribution have been age-adjusted and are expressed as means ± SEM and were analysed using ANOVA followed by Tukey’s post-hoc analysis. *** p<0.001 versus average, **p<0.01 versus average, ### p<0.001 versus < -1 SD decrease, # p<0.05 versus < -1 SD. NS= not significant. Figure 1a: Early childhood < -1 SD n= 72, average n= 471, > 1 SD n = 67; Late childhood < -1 SD n= 75, average n= 462, > 1 SD n = 73; Adolescence < -1 SD n= 55, average n= 500, > 1 SD n = 55. Figures 1b, 1c and 1d: Early childhood < -1 SD n= 21, average n= 160, > 1 SD n = 20; Late childhood < -1 SD n= 22, average n= 154, > 1 SD n = 25; Adolescence < -1 SD n= 22, average n= 164, > 1 SD n = 15.
Figure 4. BMI increases during adolescence are associated with high adult visceral fat mass in subjects with low, average and high prepubertal BMI.

Intraperitoneal fat area (Y-axis) in subjects divided into tertiles according to (i) prepubertal BMI (X-axis; low, average and high) and (ii) BMI-Z-score increase during adolescence (Z-axis; low, average and high). The intraperitoneal adipose tissue area has been age-adjusted. Values are expressed as means and were analysed using ANOVA followed by Tukey’s post-hoc analysis. * p<0.05 ** p<0.01 versus average BMI Z-score change during adolescence (striped bars), ## p<0.01 versus low (white bar). BMI = body mass index.

N for lowest ΔBMI during adolescence (white bars) from low prepubertal BMI to high (left to right) 9, 19 and 39; for average ΔBMI during adolescence (striped bars) low prepubertal BMI to high (from left to right) 26, 25, 16; and for high ΔBMI during adolescence (black bars) from low prepubertal BMI to high (left to right) 32, 23 and 12. N for all = 201.