Original Article

Pubertal Timing Is an Independent Predictor of Central Adiposity in Young Adult Males

The Gothenburg Osteoporosis and Obesity Determinants Study

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The role of puberty and normal variations in pubertal timing for the development of obesity in men is unclear. The aim of the current study was to investigate the impact of pubertal timing and prepubertal BMI (kg/m^2) for young adult BMI and fat mass distribution. Detailed growth charts from birth to age 18–20 years were retrieved for the men participating in the population-based Gothenburg Osteoporosis and Obesity Determinants study. Age at peak height velocity (PHV) and BMI at age 10 years were estimated for 579 subjects, and PHV was used as an assessment of pubertal timing. The fat mass characterization and distribution were analyzed using dual X-ray absorptiometry and peripheral as well as abdominal computed tomography at age 18.9 ± 0.5 years. We demonstrate that age at PHV is an independent negative predictor of young adult BMI and whole-body fat mass. Interestingly, age at PHV is an independent negative predictor of central, but not peripheral, fat mass. In contrast, BMI at 10 years of age predicts both central and peripheral subcutaneous fat mass. In conclusion, we demonstrate that early pubertal onset specifically predicts a central fat mass distribution, while a predominantly subcutaneous obese phenotype is strongly predicted by a high prepubertal BMI. *Diabetes* 55: 3047–3052, 2006

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Obesity has grown into an epidemic in the Western world, and not only adults but also children are now treated for metabolic syndrome disorders. Obesity is an important risk factor for the development of diabetes and cardiovascular disease, and in the National Health and Nutrition Examination Survey, 12.6% of boys aged 8–14 years were obese and 29.1% were overweight (1,2). Puberty has been identified as a critical period for the development of overweight (3), but the role of puberty and variations within the normal range in pubertal timing for the development of obesity in men are unknown. Age at pubertal onset is decreasing, and although girls are more extensively studied in this respect, there are reports of secular trends in pubertal onset for boys as well (4,5).

Kaplowitz et al. (6) reported a negative association between prepubertal BMI (weight in kilograms divided by the square of height in meters) and the timing of puberty in female subjects. Furthermore, an association between early age at menarche and increased adult BMI has been reported (7,8), but the impact of pubertal timing independent of prepubertal BMI was not investigated in these studies (7,8). The independent predictive role of pubertal timing, adjusted for prepubertal BMI, for adult BMI in female subjects has recently been investigated in two longitudinal studies: the Newton Study and the Bogalusa Heart Study. In the Newton Study (9), pubertal timing did not predict adult BMI independently of prepubertal BMI, and from the Bogalusa Heart Study (10) it was concluded that after adjustment for prepubertal BMI, menarcheal age no longer predicts adult BMI. Thus, pubertal timing is a negative predictor of adult BMI, but, after adjustment for prepubertal BMI, no clear independent impact of pubertal timing on adult BMI has been reported in female subjects.

The impact of male puberty for adult fat mass is not as thoroughly studied as the impact of female puberty. In the Amsterdam Growth and Health Study, van Lenthe et al. (11) studied the role of pubertal timing for adult fat phenotype in 177 boys and girls. In their study, pubertal timing was based on assessments of skeletal age and the results show higher BMI and sum of skinfold thickness for early maturers (11). Beunen et al. (12) report similar results from the Leuven Growth Study, including 173 longitudinally followed male subjects. Beunen et al. (12)

used age at peak height velocity (PHV) as an assessment of pubertal timing and demonstrated higher BMI in early maturers. Importantly, adjustment for prepubertal BMI was not performed in these two studies, investigating the role of pubertal timing for adult fat mass in men, and therefore the independent predictive role of pubertal timing could not be evaluated.

The aim of the current study was to investigate the independent predictive role of pubertal timing, adjusted for prepubertal BMI, for central and peripheral fat mass in a well-characterized, rather large cohort (n = 579) of young adult male subjects. We hypothesize that pubertal timing is an independent predictor of fat mass in young adult male subjects.

RESEARCH DESIGN AND METHODS

The Gothenburg Osteoporosis and Obesity Determinants (GOOD) study was initiated with the aim of determining both environmental and genetic factors involved in the regulation of fat mass. Study subjects were randomly identified using national population registers and were contacted by telephone and asked to participate in the present study. A total of 1,068 men, aged 18–20 years, representative for the greater Gothenburg area, were included between February and November 2003 in Gothenburg (13). To be included in the GOOD study, subjects had to be aged >18 and <20 years and willing to participate in the study. There were no other exclusion criteria; 48.6% of the contacted study subject candidates agreed to participate and were included in the study. The study population was homogenous with respect to ethnicity (98.6% white). A standardized questionnaire was used to collect information about the amount of present physical activity (hours per week, duration in years), nutritional intake (dairy products, vegetables, and vitamin intake), and smoking. Calcium intake was estimated from dairy product intake and semiquantified into quintiles before values were used in linear regression analyses. Of 1,068 subjects, complete growth and weight charts for determination of PHV and BMI at age 10 years were available for 579 subjects (i.e., with sufficient information for determination of both PHV and BMI) (10). Thus, the results presented here were obtained from a subsample of the original GOOD cohort.

The GOOD cohort was compared with age-matched, randomly selected conscripts living in the same area as the GOOD subjects. There was no difference in height, weight, or BMI between these two cohorts, indicating that the GOOD cohort is representative of the general young male population of Gothenburg (data not shown). Furthermore, height, age, and BMI did not differ between the present subsample and the complete GOOD cohort, indicating that the subsample is representative for the complete GOOD study (data not shown). Dual X-ray absorptiometry (DXA) and peripheral computed tomography (CT) analyses were performed on all subjects, while abdominal CT scans were performed on 190 of 579 study subjects. The GOOD study was approved by the local ethics committee at Gothenburg University. Written and oral informed consent were obtained from all study participants.

Anthropometrical measurements. Height was measured using a wall-mounted stadiometer, and weight was measured to the nearest 0.1 kg. All the measurements were carried out by the same trained staff. The coefficient of variation (CV) values were <1% for these measurements.

DXA. Fat mass, lean mass, and percent body fat of the whole body and fat mass of the trunk as well as upper and lower extremities were assessed using the Lunar Prodigy DXA (GE Lunar, Madison, WI).

Peripheral CT analyses of cross-sectional adipose tissue area in the distal arm and leg. A peripheral CT device (XCT-2000; Stratec Medizintechnik, Pforzheim, Germany) was used to scan the distal leg and the distal arm of the nondominant leg and arm, respectively. The peripheral CT was calibrated every week using a standard phantom and once every 30 days using a cone phantom provided by the manufacturer. A 2-mm-thick single tomographic slice was scanned with a voxel size of 0.50 mm. The subcutaneous cross-sectional adipose tissue area was measured using a scan through the diaphysis (at 25% of the bone length in the proximal direction of the distal end of the bone) of the radius and tibia. Image analyses were performed to determine the cross-sectional subcutaneous adipose tissue areas.

Abdominal CT analyses of cross-sectional adipose tissue areas. A CT technique was used to measure the cross-sectional adipose area of the abdomen. Adipose tissue areas were determined with the subject in a recumbent position with a General Electric High Speed Advantage CT system (HAS, version RP2; GE Medical Systems, Milwaukee, WI). Total abdominal, abdominal subcutaneous, abdominal deep subcutaneous, and two intra-abdominal (intraperitoneal and retroperitoneal) adipose tissue areas were determined as previously described (14). Adipose tissue areas were measured using one scan at the fourth lumbar vertebra level. The tube voltage was 120 kV. Slice thickness and tube current were set according to a dose reduction scheme (15). Precision errors were calculated from double determinations: subcutaneous adipose tissue (0.5%) and the sum of intraperitoneal and retroperitoneal adipose tissue areas (intra-abdominal adipose tissue area; 1.2%).

Estimation of PHV and BMI at 10 years of age. Growth charts from birth until age 18–20 years have been used for estimation of age at PHV according to the Infancy-Childhood-Puberty model (16), and PHV was used as an assessment of pubertal timing. The average number of measurements between birth and 19 years of age were 23. For each individual growth curve with sufficient information in all three growth phases, the Infancy-Childhood-Puberty model was fitted by minimizing the sum of squares using a modification of the Levenberg-Marquardt algorithm (16). To estimate PHV, two or more measurements during the critical "peri-PHV" period were needed, not >2 years from PHV and not >3 years from each other. Body weight at 10 years of age was 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 [available at www.r-project.org]). BMI at 10 years of age was then calculated from the estimated values of weight and height at age 10 years.

Age at PHV was defined as the age at maximum growth velocity during puberty and was estimated by the algorithm. PHV is generally believed to be reached within 2 years after pubertal onset (4,16). A total of 642 subjects had growth curves with enough information for the estimation of PHV, and of these, 579 subjects also had weight curves with enough information for the estimation of BMI at 10 years of age. Ten years of age as the prepubertal age was chosen close to pubertal onset to avoid confounding the independent role of age at PHV with genetic and environmental factors, which already have influenced BMI before onset of puberty. The use of a prepubertal age that lies close enough to pubertal onset is important in order to avoid the independent role of age at PHV, to some extent also reflecting the importance of prepubertal BMI for young adult BMI.

Statistical analysis. BMI at 10 years of age and age at PHV were tested as predictors of young adult BMI using linear regression analyses separately (nonadjusted or crude) or with BMI at 10 years of age and age at PHV included in the regression analyses (adjusted). Age at fat analysis was included as a covariate in both crude and adjusted analyses. BMI and variables from fat characterization (including variables from DXA, peripheral CT, and abdominal CT measurements) were not normally distributed as tested using the Kolmogorov-Smirnov test and have therefore been log transformed. Binary logistic regression analyses were performed for overweight (BMI >25 kg/m^2), according to BMI at 10 years of age (per SD) and age at PHV (1-year increments) and were given as odds ratios with 95% CIs. For all statistical analyses, SPSS software (version 13.0) was used. Values are given as means ± SD.

RESULTS

The current results were obtained from a subsample from the GOOD study. Descriptive characteristics of age at fat analyses, age at PHV, anthropometrics, and fat variables for the subsample as measured using DXA (n = 579), peripheral CT (n = 579), and abdominal CT (n = 190) are given in Table 1. The average age at fat analysis was 18.9 ± 0.5 years, and this is referred to as "young adult age." The average age at PHV was 13.6 ± 1.0 years (range 10.9–16.9).

BMI at 10 years of age predicts age at PHV. We first investigated whether BMI at 10 years of age (average 17.0 ± 2.1 kg/m^2; 10th and 90th percentiles 14.8 kg/m^2 and 19.6 kg/m^2, respectively) predicts age at PHV. Univariate analysis demonstrated that 5.0% of the variance in age at PHV is explained by BMI at 10 years of age (r = 0.22; Fig. 1*A*). Each extra unit (kg/m^2) of BMI at 10 years of age is associated with a reduction of age at PHV by 6 weeks, and the difference in age at PHV per SD in BMI at 10 years of age is 3 months (Fig. 1*A*).

Both BMI at 10 years of age and age at PHV predict young adult BMI. Univariate analyses demonstrated that young adult BMI is strongly positively associated with BMI at 10 years of age (r = 0.65; Fig. 1*B*) and moderately negatively associated with age at PHV (Fig. 1*C*). A clear impact on BMI at young adult age is demonstrated by the fact that each extra unit (kg/m^2) of

TABLE 1
Anthropometrics, age at PHV, and fat variables

Variables	Means ± SD	Median (range)
Anthropometrics (n = 579)		
Age at body fat analyses (years)	18.9 ± 0.5	18.8 (18.0–20.0)
Age at PHV (years)	13.6 ± 1.0	13.6 (10.9–16.9)
Height (cm)	181.6 ± 6.9	181.6 (161.0–202.8)
Weight (kg)	73.3 ± 11.5	71.7 (47.6–122.3)
Smoking (%)	8.7	—
Young adult BMI (kg/m^2)	22.2 ± 3.0	21.8 (16.1–36.5)
BMI at 10 years of age (kg/m^2)	17.0 ± 2.1	16.6 (13.0–26.9)
DXA measurements (n = 579)		
Whole-body fat (kg)	12.9 ± 7.3	11 (3.1–46.7)
Percentage body fat	16.8 ± 7.1	15.4 (5.2–42)
Whole-body lean mass (kg)	57.4 ± 6.3	57.1 (41.3–77.3)
Fat mass arm (kg)	0.5 ± 0.4	0.4 (0.1–2.8)
Fat mass leg (kg)	2.4 ± 1.3	2.1 (0.6–9.3)
Fat mass trunk (kg)	6.5 ± 4.0	5.6 (1.4–24.2)
Peripheral CT measurements (n = 579)		
Subcutaneous fat arm (cm^2)	7 ± 3	6 (1–22)
Subcutaneous fat leg (cm^2)	13 ± 5	13 (3–48)
Abdominal CT measurements (n = 190)		
Total fat area (cm^2)	136 ± 99	110 (20–580)
Subcutaneous fat (cm^2)	99 ± 83	73 (13–490)
Deep subcutaneous fat (cm^2)	45 ± 50	27 (2–350)
Intraabdominal fat (cm^2)	35 ± 19	32 (7–120)
Intraperitoneal fat (cm^2)	22 ± 12	20 (3–84)
Retroperitoneal fat (cm^2)	13 ± 8	11 (0–44)

BMI at 10 years of age is associated with 0.96 kg/m^2 extra BMI at young adult age, and, as expected, prepubertal BMI explains a large part (42%) of the variance in young adult BMI (Fig. 1*B*). Furthermore, univariate analysis demonstrated that age at PHV explains 5.2% of the variance in BMI at young adult age and that BMI at young adult age is decreased by 0.69 kg/m^2 per year increase of age at PHV (r = 0.23; Fig. 1*C*). Thus, BMI at

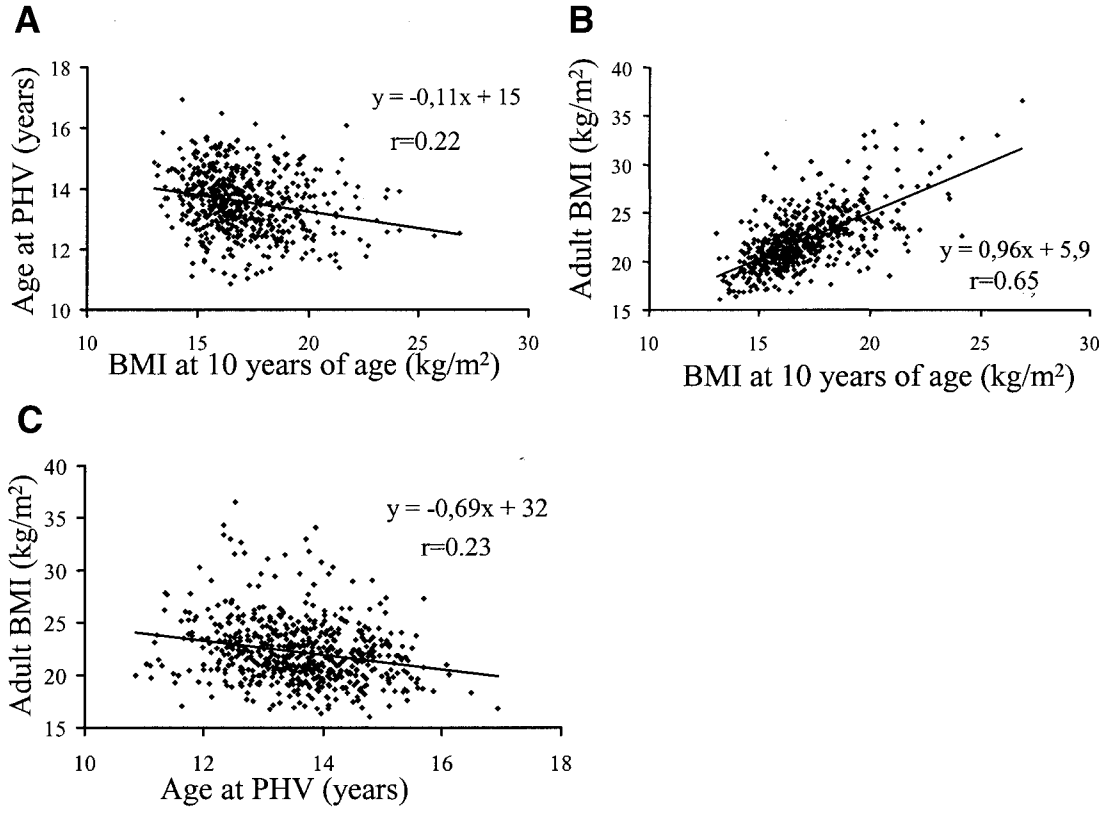

FIG. 1. Scatter plots of the associations between BMI at 10 years of age and age at PHV (*A*), BMI at 10 years of age and young adult BMI (*B*), and age at PHV and young adult BMI (*C*). *r* for the linear regressions are given in the figures. *P* < 0.001 for *A–C*.

TABLE 2
BMI at 10 years of age and age at PHV as predictors of young adult body composition

Variable	BMI at 10 years of age (crude)		PHV (crude)	
	β value	*P* value	β value	*P* value
Young adult BMI (n = 579)	0.66	<0.001	−0.23	<0.001
DXA				
Whole body (n = 579)				
Lean mass	0.38	<0.001	−0.04	0.286
Fat mass	0.53	<0.001	−0.20	<0.001
Total fat (%)	0.46	<0.001	−0.20	<0.001
Extremities (n = 579)				
Fat mass arm	0.54	<0.001	−0.19	<0.001
Fat mass leg	0.52	<0.001	−0.16	<0.001
Trunk (n = 579)				
Fat mass trunk	0.53	<0.001	−0.23	<0.001
CT				
Peripheral subcutaneous fat (n = 579)				
Distal arm	0.48	<0.001	−0.14	0.001
Distal leg	0.43	<0.001	−0.10	0.014
Abdominal fat (n = 190)				
Total fat area	0.53	<0.001	−0.27	<0.001
Subcutaneous fat	0.57	<0.001	−0.28	<0.001
Deep subcutaneous fat	0.51	<0.001	−0.29	<0.001
Intra-abdominal visceral fat	0.28	<0.001	−0.22	0.003
Intraperitoneal visceral fat	0.23	0.002	−0.18	0.014
Retroperitoneal visceral fat	0.28	<0.001	−0.23	0.002

Data presented are nonadjusted. The predictive role of BMI at 10 years of age and age at PHV for BMI and fat characterization at 18.9 ± 0.5 years of age were assessed through linear regression analyses (age at fat analysis included in linear regression analysis). Fat variables have been log transformed. Standardized β values are used.

young adult age is predicted both by BMI at 10 years of age and by age at PHV.

BMI at 10 years of age is a strong independent predictor of fat mass at young adult age. The impact of BMI at 10 years of age on BMI and fat mass at young adult age was examined using linear regression analyses. Nonadjusted linear regression analyses demonstrated that BMI at 10 years of age is a strong positive predictor of not only BMI (r = 0.65) but also fat mass at all locations examined in young adult men (fat mass r = 0.52; fat mass trunk r = 0.52; Table 2).

To differentiate the impact of BMI at 10 years of age from the impact of age at PHV for BMI and fat mass at young adult age, the impact of these two parameters was investigated with multiple linear regression analysis, including both age at PHV and BMI at 10 years of age. Fat mass and distribution were studied using DXA, peripheral CT, and abdominal CT. DXA measurements demonstrated that BMI at 10 years of age is an independent strong positive predictor of whole-body fat mass, percentage fat, and both central (trunk) and peripheral (lower- and upper-extremity) fat mass (Table 3). Fat mass and distribution were further studied using CT, demonstrating that BMI at 10 years of age is an independent strong predictor of the subcutaneous cross-sectional fat area in the arm, leg, and abdomen and a moderate predictor of visceral (intraperitoneal and retroperitoneal) fat areas in the abdomen (Table 3). When the ratios between subcutaneous and visceral (intraperitoneal and retroperitoneal) cross-sectional fat areas in the abdominal region were evaluated, we found that BMI at 10 years of age is an independent positive predictor of these ratios ($P < 0.01$; data not shown), suggesting that a high prepubertal BMI predominantly results in an increase of subcutaneous, and not so much visceral, fat deposits at young adult age. Thus, BMI at 10 years of age is associated with young adult BMI and fat mass at all sites investigated but with the strongest impact on subcutaneous fat deposits in young adult men.

Age at PHV is an independent predictor of central adiposity. We next studied the impact of age at PHV for young adult BMI and fat mass by using linear regression analysis, demonstrating that age at PHV is a negative predictor of young adult BMI (r = 0.23), whole-body fat, and all the different fat parameters of both central (DXA, trunk fat; abdominal CT, total, subcutaneous, deep subcutaneous, intraperitoneal, and retroperitoneal fat areas) and peripheral (DXA, upper- and lower-extremity fat mass; peripheral CT, subcutaneous fat area in arm and leg) fat mass (fat mass r = 0.20; trunk fat mass r = 0.23; nonadjusted; Table 2). Inclusion of BMI at 10 years of age as a covariate in the linear regression analysis demonstrated that age at PHV was an independent negative predictor of young adult BMI, whole-body fat mass, percentage body fat, and central (DXA, trunk) but not peripheral (DXA, upper and lower extremity; Table 3) fat mass. CT measurements for determination of cross-sectional fat areas confirmed that age at PHV is an independent negative predictor of measures of central fat mass (total fat, subcutaneous and deep subcutaneous fat, and visceral fat in the abdomen) but not of parameters reflecting peripheral fat mass (subcutaneous fat in the upper and lower extremity; Table 3). The largest independent impact of age at PHV for young adult fat mass is seen for the central fat deposits (Table 3). Linear regression analyses including either BMI at 10 years of age or age at PHV demonstrated that BMI at 10 years of age explains 7.9% and age at PHV explains 5.3% while linear regression analyses including both BMI and age at PHV explains 10.4% of the total variance in cross-sectional retroperitoneal fat area, supporting the notion that age at PHV has an independent

TABLE 3
BMI at 10 years of age and age at PHV as independent predictors of young adult body composition

Variable	BMI at 10 (adjusted)		PHV (adjusted)	
	β value	*P* value	β value	*P* value
Young adult BMI (n = 579)	0.64	<0.001	−0.09	0.007
DXA				
Whole body (n = 579)				
Lean mass	0.39	<0.001	−0.05	0.20
Fat mass	0.51	<0.001	−0.09	0.019
Total fat %	0.44	<0.001	−0.10	0.008
Extremities (n = 579)				
Fat mass arm	0.52	<0.001	−0.07	0.062
Fat mass leg	0.51	<0.001	−0.04	0.261
Trunk (n = 579)				
Fat mass trunk	0.50	<0.001	−0.12	0.001
CT				
Peripheral subcutaneous fat (n = 579)				
Distal arm	0.47	<0.001	−0.04	0.356
Distal leg	0.43	<0.001	0.00	0.939
Abdominal fat (n = 190)				
Total fat area	0.49	<0.001	−0.13	0.039
Subcutaneous fat	0.54	<0.001	−0.12	0.048
Deep subcutaneous fat	0.47	<0.001	−0.15	0.020
Intra-abdominal visceral fat	0.24	0.001	−0.15	0.039
Intraperitoneal visceral fat	0.19	0.011	−0.13	0.09
Retroperitoneal visceral fat	0.23	0.002	−0.16	0.027

The independent predictive role of BMI at 10 years of age and age at PHV for BMI and fat characterization at 18.9 ± 0.5 years of age were assessed through linear regression analyses. Fat variables have been log transformed. Both variables were included together with age at fat analysis as covariates in the linear regression analyses. Standardized β values are used.

impact on young adult central fat mass. Thus, age at PHV is inversely related to central, but not peripheral, adiposity in young adult men.

Both BMI at 10 years of age and age at PHV are independent predictors of young adult overweight. To quantify the impact of age at PHV for young adult overweight (BMI >25 kg/m^2, n = 89), odds ratios were computed by logistic regression analyses. Age at fat analyses was included as a covariate in all analyses. The analyses demonstrated that age at PHV (1-year increments) is inversely related to young adult overweight (not adjusted for BMI at 10 years of age [odds ratio 0.58 {95% CI 0.45–0.73}], $P < 0.001$; adjusted for BMI at 10 years of age [0.70 {0.53–0.91}], $P < 0.01$). BMI at 10 years of age (per SD) is an independent strong positive predictor of young adult overweight (not adjusted for age at PHV, 3.59 [2.72–4.76], $P < 0.001$, and adjusted for age at PHV, 3.43 [2.58–4.56], $P > 0.001$).

DISCUSSION

Studies on male puberty are scarce, and, consequently, our knowledge about this crucial event in development and its role for adult body composition is incomplete. In the present study, we have studied the independent impacts of pubertal timing and prepubertal BMI for young adult fat mass in a well-characterized cohort of young adult men. We demonstrate that early pubertal onset specifically predicts a central fat mass distribution, while a predominantly subcutaneous obese phenotype is strongly predicted by a high prepubertal BMI. Moreover, we have quantified the impact of prepubertal BMI for age at pubertal onset.

The decreased age at menarche in most European countries over the last century has paralleled the increasing incidence of obesity (5,17). In girls, menarcheal age is the most common way to assess pubertal timing. Several studies (6,9,10) describe a negative association between prepubertal BMI and age at menarche, and one may therefore speculate that the secular trend for menarcheal age is a part of the obesity epidemic in the Western world. For male subjects, no marked indicator for pubertal onset, such as age at menarche in female subjects, exists, and consequently, less is known about male puberty and pubertal timing (17). However, a secular trend for pubertal timing in male subjects has been described (4,5), but whether prepubertal BMI has an impact on male pubertal onset is not fully understood. In the present study, we demonstrate that prepubertal BMI is a predictor of pubertal timing and that it explains 5.0% of the variance in age at PHV. Thus, there is an association for male as well as for female subjects between prepubertal BMI and pubertal timing. The clinical consequence of the association between prepubertal body composition and pubertal timing might be that the assessment of pubertal development is biased and pubertal staging interpreted as more advanced in obese adolescents and delayed in thin adolescents. The data presented here enable adjustment of pubertal development for prepubertal BMI by using different references according to prepubertal BMI, which might be of some use in situations where other factors with a pathological influence on puberty need to be evaluated. Our data indicate that the pubertal development in boys should be adjusted by 6 weeks per unit of BMI.

The main finding in the current study is that pubertal timing independently of prepubertal BMI predicts BMI, fat mass, and distribution of fat mass at young adult age. Previous studies have not investigated the independent roles of prepubertal BMI and of pubertal timing for fat mass at young adult age in male subjects. Furthermore, we demonstrate that prepubertal BMI is a strong predictor of

young adult BMI, explaining 42% of the variance in BMI at young adult age.

In female subjects, the Newton study (9) concludes that the relation between early maturation and later obesity was largely explained by premenarcheal weight status with no extra information when menarcheal timing was included in the multivariate analysis. For male subjects, the Amsterdam Growth and Health Study and the Leuven Growth Study (11,12), both longitudinal, demonstrate an association between early sexual maturation and BMI at adult age. These two studies were initiated during puberty (ages 13–27 and 13–30 years of age, respectively), and thus no prepubertal measurements are included in these studies. Thus, an independent predictive role of age at PHV for young adult BMI and distribution of fat mass has not previously been shown. In the present study, we show for the first time that normal variations in the range of pubertal timing independently predict BMI and fat distribution in a well-characterized cohort of 579 young adult male subjects.

A central pattern of fat distribution has been associated with increased risk of cardiovascular morbidity and mortality and for the development of type 2 diabetes (18,19). In the Leuven Growth Study (12), investigating skinfold thickness as an indicator of subcutaneous fat, it was demonstrated that early maturers had increased skinfold thickness in the abdominal area. A similar pattern of increased central adiposity is seen in early maturing girls in the Fels Study (8), but neither of these studies have corrected the adult fat phenotype for prepubertal BMI. In the present study, adjusting for prepubertal BMI, we demonstrate that pubertal timing independently predicts central, but not peripheral, fat mass, implicating that the association between pubertal timing and young adult body composition is site dependent. In contrast, prepubertal BMI is an independent predictor of both central and peripheral fat mass. As a central distribution of fat mass has been associated with the metabolic syndrome and cardiovascular disease (18–20), one may speculate that early puberty is a metabolic risk factor for cardiovascular morbidity and diabetes in male subjects.

In conclusion, early pubertal onset specifically predicts a central fat mass distribution, while a predominantly subcutaneous obese phenotype is strongly predicted by a high prepubertal BMI. As a central pattern of fat distribution is known to be associated with increased risk of cardiovascular disease, one may speculate that early puberty is a previously unknown cardiovascular risk factor for men.

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