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Physical Activity, Not Sedentary Time, Predicts DXA-Measured Adiposity Age 5–19 Years

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Abstract

Purpose—To examine the associations among physical activity (PA), sedentary time (SED), and TV viewing (TV) with fat mass (FAT) and visceral adipose tissue mass (VAT) from childhood through adolescence (5–19 years).

Methods—Participants in the Iowa Bone Development Study (n = 230 males and 233 females) were examined at ages 5, 8, 11, 13, 15, 17 & 19 years. Accelerometers measured moderate- or vigorous-intensity PA (MVPA; min/day), light-intensity PA (LPA; min/day), and SED (h/day). Parent-proxy report (5 & 8 years) and child-report (11, 13, 15, 17, & 19 years) measured TV (h/ day). DXA scans measured FAT (kg) and VAT (g). Sex-specific growth models were used to create FAT and VAT growth curves for individual participants (level 1), and to test the effect of MVPA, LPA, SED, & TV (level 2) after adjusting for weight, height, linear age, non-linear age, and maturity.

Results—Growth models indicated that low levels of MVPA were associated with high levels of FAT and VAT for males and high levels of FAT for females. TV was positively associated with FAT and VAT for males and females. LPA was positively associated with FAT in males. SED was not associated with FAT or VAT for males or females (p > 0.05).

Conclusion—This study supports current PA guidelines focusing on MVPA rather than SED. The contribution of high TV, but not high SED, to high levels of adiposity suggests that TV's contribution to obesity is not just a function of low energy expenditure.

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The authors have no conflicts of interest to disclose in relation to this study. The results of the present study do not constitute endorsement by American College of Sports Medicine. The results of this study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

Body Composition; Fatness; Moderate- or Vigorous-Intensity PA; TV Viewing; Visceral Adipose Tissue

INTRODUCTION

Most infants are born with healthy body composition and, on average, every year children gain 1.9 kg of body fat (11). Excessive fat gain that is disproportional to total body mass leads to development of overweight or obesity (11). Excessive fat deposition, specifically excessive visceral adiposity, is associated with greater risk for hypertension and type II diabetes (3,32). The most commonly implicated behavioral factors associated with excessive fat gain are dietary behaviors contributing to increased energy intake and low levels of physical activity (PA) leading to low energy expenditure. The high prevalence (~17%) of childhood obesity supports the need to better understand which intensities of PA are most robustly associated with adiposity including visceral adiposity (5,26).

Based on the federal *2008 Physical Activity Guidelines for Americans* (27) children should engage in a minimum of 60 minutes or more of moderate- or vigorous-intensity PA (MVPA) everyday. This suggests that the most important PA intensity for health outcomes, including healthy body composition, is at least moderate-intensity, which is typically operationalized as 4 metabolic equivalents (METs). In agreement with this suggested intensity, Janz and colleagues demonstrated that objectively measured MVPA at age 5 years predicted dualenergy X-ray absorptiometry (DXA)-measured adiposity at ages 8 and 11 years (13). Similarly, Riddoch and colleagues in a two-year longitudinal study examining 4150 children (4th and 5th graders) showed that follow-up DXA-derived fat mass index was negatively associated with baseline objectively measured MVPA as well as total PA (28). Recently, in a multi-national, cross-sectional study of 6539 children (ages 9 to 11 years), Katzmarzyk and colleagues found that greater objectively measured MVPA was associated with lower risk of obesity (as classified by BMI z-scores) (16).

On the other hand, in their systematic review of 16 prospective observational studies and five trials of objectively-measured PA and obesity in youth (14 studies), and adults (7 studies), Wilks and colleagues concluded that PA is not strongly associated with adiposity and might not be the main predictor of excessive adiposity (34). Among the 14 studies of youth obesity included in Wilks and colleagues' systematic review, ten were observational and four were interventions. The ten observational studies were very different in quality and design and were not in agreement with regard to the association between PA and adiposity. Among the four intervention studies, time from baseline to follow-up measurements varied from four months to two years; only the study using two-year follow-up found a significant effect of objectively measured PA on adiposity (estimated by skin-folds). In contrast, Kelley and colleagues published a systematic review and meta-analysis of 20 randomized control trials examining the effects of PA intervention on BMI indicated obesity in children and adolescents. They concluded that exercise intervention was associated with significant reduction in BMI. However, the authors also reported significant heterogeneity and

inconsistency of the results (17). Variability in measurement precision of the physical activity exposure and adiposity outcomes probably contributed to inconsistent results cited in Kelly et al., and Wilks et al, reviews (17,34).

Aided by better PA measurement tools, researchers have expanded their examination of PA intensity to include light-intensity PA (LPA) (1.6 to 3.9 METs), sedentary time (SED) (1 to 1.5 METs), and specific sedentary behaviors, e.g., TV viewing (TV). In a large crosssectional study using data of 11,115 children and adolescents from the International Children's Accelerometry Database (ICAD), Mitchell and colleagues concluded that objectively measured MVPA was independently associated with lower BMI and waist circumference z-scores. The ICAD study also reported that TV was independently associated with higher BMI and waist circumference z-scores while total SED was not (23). Similarly, in their recent review article, Ekelund and colleagues reported that, out of 9 cohort studies that examined the relationship between SED and adiposity in youth, 8 studies indicated that SED and adiposity were not significantly related after controlling for MVPA (5). This review also suggested that TV might be related to adiposity independently of PA, but that the evidence is currently inconclusive and could be explained in part by residual confounding associated with snacking during TV. In their systematic review of 61 prospective cohort studies examining factors that predict excessive adiposity in youth, Pate and colleagues indicated that, although, some longitudinal studies demonstrate positive associations between SED and adiposity in children, most do not (26). The authors concluded that the mixed evidence likely could be explained by the differences in the precision of specific measures of adiposity (criterion vs. non-criterion), in the adjustment for maturation status, and in the characteristics of the populations assessed in different studies.

In this report, we analyze 14 years of prospective data to determine the contribution of PA, SED, and TV associated with DXA-derived total adiposity and visceral adiposity. Our study examines associations from the beginning of middle childhood (~5 years) through late adolescence (~ 19 years). Based on previous research, we hypothesized that MVPA is more strongly associated with adiposity than is LPA, or SED, and that, despite being a sub-set of sedentary time, TV would have a greater effect on adiposity than SED.

METHODS

Participants

The Iowa Bone Development Study is an ongoing longitudinal study investigating bone health and body composition from childhood to adulthood (13,14). Participants in this study are a subset of a larger cohort recruited immediately postnatally during 1998–2001 while participating in the Iowa Fluoride Study. Additional information about the Iowa Bone Development Study, the Iowa Fluoride Study and demographic information of participants is presented elsewhere (13,14). The current analysis used data collected in seven measurement waves at approximately ages 5 (wave 1), 8 (wave 2), 11 (wave 3), 13 (wave 4), 15 (wave 5), 17 (wave 6) and 19 (wave 7) years (n = 230 males and 233 females). Participants who completed at least two waves after wave two were included in the analysis. This strategy allowed us to model visceral adiposity which became available at wave 3. The University of Iowa Institutional Review Board approved this study. Parental written consent and assent

from children were obtained. At wave 7 (age 19), written consent was provided directly by the participants.

Anthropometry and Maturity Assessment

During each DXA visit, trained research nurses measured the participant's height (cm) using a Harpenden stadiometer (Holtain, UK), and body weight (kg) using a Healthometer physician's scale (Continental, Bridgeview, IL, USA). Both devices were calibrated routinely. Sitting height also was measured at waves 3 to 6. The offset of maturity, years from peak height velocity age (PHV), was estimated using prediction equations developed by Mirwald and colleagues (22). PHV was calculated using height, weight, age, sex, sitting height, leg length, and weight by height ratio as predictors of somatic maturity. Since we measured participants at age 11, 13, and 15 and therefore calculated multiple estimates of age at PHV (APHV), as advised by Malina and colleagues, the APHV estimate with the most precision (from measurement at assessment closest to estimated APHV) was used in our models (19). The maturity offset equation has been validated in white Canadian children and adolescents ($R^2 = 0.91, 0.92, SEE = 0.49, 0.50$, respectively). The maturity-offset variable was dichotomized as 0 (prior to APHV, or pre-mature) or 1 (APHV, or mature).

Physical Activity

Participant PA was measured using Actigraph uniaxial accelerometers model 7164 in waves 1 to 4, model GT1m in wave 5, and model GT3x+ in waves 6 and 7. Following the ICAD protocol, we did not adjust accelerometer data for different Actigraph models. Movement counts were measured in 1-minute epochs for waves 1, 2, and 3, and 5-s epochs for waves 4, 5, 6, and 7. The 5-s epochs later were converted to 1-min epochs to maintain consistency with previous measurements. The detailed procedures of physical activity assessment with accelerometry are described elsewhere (12). Briefly, during the autumn months participants were asked to wear the accelerometers during the waking hours for four consecutive days including one weekend day in waves 1 and 2, and 5 consecutive days including both weekend days in waves 3 to 7. Previous research has demonstrated that older children and adolescents have less stable activity patterns and lower day-to-day intraclass correlation coefficients when compared to younger children, indicating that an additional monitoring day was needed (15). Physical activity data were considered complete if participants wore the accelerometers for at least 10 hours per day and a minimum of 3 days within 15 months of the DXA scan.

Summary variables of daily minutes of MVPA, LPA, and SED were determined. Accelerometer variables were derived to intensity zones using Evenson cut points (6): Activity counts 2296 counts/min as MVPA, 100 to 2295 counts/min as LPA, and 0 to 99 counts/min as SED, respectively. Thus, the activity intensities were identified as MVPA 4 METs, LPA = 1.6 to 3.9 METs, and SED 1.5 METs. Total PA = sum > 99 counts/min also was determined and reported as counts/min per day.

TV Viewing

During clinical visits, questionnaires were administered that queried TV habits. The questionnaire for children in waves 1 and 2 was designed for parents (Proxy PAQ) and

consisted of an open-ended question about the average time per day (to the nearest quarter hour) the child spent watching TV (including videotapes, movies and programs). Using parental proxy to assess this type of sedentary behavior is common and has been shown to be moderately related to direct observation (r = 0.31 to 0.61) (29,30). In waves 3 to 7, participants self-reported their TV behaviors (Self PAQ). The following response categories were defined: (1) < 1 h/day or not at all; (2) 1 h/day, but < 2 h/day; (3) 2 h/day, but < 3 h/day; (4) 3 h/day, but < 4 h/day; and (5) 4 h/day. This method of assessing TV behavior has been used for children and adolescents in this age range (33). TV time of children in waves 1 and 2 was adjusted to match the response options of waves 3 to 7. Mean values for response categories were used to create continuous variable for TV viewing.

Adiposity

The adiposity of participants was determined using DXA during clinical visits at the University of Iowa General Clinical Research Center and Clinical Research Unit. All measurements were administered by one of three experienced research technicians in order to minimize operator-related variability. Total fat mass (FAT; kg) of children in waves 1 and 2 was determined by obtaining whole-body scans using Hologic QDR 2000 DXA with software version 7.20B and fan-beam mode. Starting with wave 3, the Hologic QDR 4500 DXA (Delphi upgrade) with software version 12.3 and fan-beam mode was used to obtain FAT. To adjust for machine differences, indicator variable was included in models for FAT.

Because DXA does not directly measure visceral adipose tissue mass (VAT; g) we used estimation equations recommended by the manufacturer to predict VAT from whole body 4500 DXA data at waves 3 to 7. Predicted VAT using this method has a very strong correlation with computed tomography (CT)-measured VAT in adults (r = 0.92) and in overweight children (r = 0.86) (2,21). This method estimates subcutaneous fat at the level of the 4th lumbar vertebra by identifying the amount of fat located between the abdominal muscle wall and the skin. The difference between total abdominal fat and subcutaneous fat at the area represented VAT (8). As part of the Iowa Bone Development Study, results of a pilot study of four children were used to identify the associations between VAT derived from DXA whole body scans and VAT derived from abdominal magnetic resonance imaging (MRI). The amount of VAT estimated from DXA and MRI had a strong correlation in these unpublished data (r < 0.99). Images from the Hologic QDR 2000 DXA cannot be processed to calculate VAT. Therefore, VAT information is not available for waves 1 and 2.

Statistical Analysis

Sex-specific descriptive analyses for participants' characteristics, time spent in MVPA, LPA, Total PA, SED, and TV, and amount of FAT, and VAT were conducted. T-tests were used for comparison between males and females. Spearman correlation coefficients were calculated among physical activity related variables by wave and sex.

For FAT and VAT, log transformations were used to make the positively skewed distributions more normal. After which, sex-specific mixed growth models for FAT and VAT were constructed. The model building process followed the recommendations of Cheng and colleagues for longitudinal data analysis (4). Marginal and conditional residual plots

confirmed that log transformation of FAT and VAT was required to satisfy mixed model assumptions for Gaussian residuals' distribution. Akaike's Information Criterion corrected for number of model parameters (AICC) and marginal and conditional residual plots were used to build the best models and select covariance parameters structure for random effects and residuals. Based on examined regression models that included centered age (time variable) polynomials, cubic polynomials described best the change over time for both males and females for FAT. For VAT, we used quadratic polynomials for males and cubic polynomials for females. FAT models included random intercept and slope with a variance component covariance structure. VAT models included random intercept only with heterogeneous autoregressive covariance structure for residuals per participant. After finalizing the structure of the models, the effects of MVPA, LPA, SED, and TV were examined in a step-wise fashion, after adjusting for overall growth trajectory (centered age polynomial, DXA machine type (FAT), height (cm), and maturity (pre-post peak height velocity age). Total PA was highly correlated with MVPA so it was not included in models. For stability of the models, we used centered age in all the models. The best reported models have the smallest AICC value.

Since growth models were developed for log FAT and log VAT, based on properties of natural logarithmic and exponential function, model parameters can be interpreted as following. In the log-linear part of the models (association with any physical activity related measure), the literal interpretation of the estimated coefficient β is that a one-unit increase in independent variable *X* will produce an expected increase in log *Y* of β units. In terms of *Y* itself, this means that the expected value of *Y* is multiplied by e^{β} . For small values of β , approximately $e^{\beta} \approx 1 + \beta$, so a 1-unit change in *X* corresponds to an expected change in *Y* of 100* β %. We presented β coefficients for 10 min increase in MVPA and 1-hour increase in LPA, SED, and TV.

SAS Software 9.4 (SAS Institute Inc., Cary, NC, USA) was used for all statistical computations and significance levels were set at p < 0.05.

RESULTS

Participants

Characteristics of participants are shown in Table 1 by measurement wave and sex. Males were taller than females at waves 2, 4, 5, 6, and 7 (p < 0.01) and heavier than females at waves 5, 6, and 7 (p < 0.01). Males reached PHV at a later age than females (p < 0.01). Males had lower FAT at waves 1, 5, 6, and 7, and higher VAT at waves 3 to 7 (p < 0.01) when compared to females. Males had greater MVPA and Total PA than females at every measurement wave (p < 0.01). There was no significant difference in minutes spent in LPA between males and females. Males spent fewer hours SED at wave 4 (p < 0.01) and 5 (p < 0.05) when compared to females. There were no significant differences between males and females at any measurement wave in the average number of minutes the accelerometers were worn per day. Males spent more time in TV at wave 6 (p < 0.01) than females. Peak mean TV time occurred at wave 3 in males and at wave 1 in females.

Associations among Physical Activity and Sedentary Variables

Spearman correlation coefficients were used to examine the bivariate associations between exposure variables by wave and sex. For males and female, MVPA and Total PA were consistently and highly associated across waves (r = 0.88 to 0.93). Associations between MVPA and SED changed over time. Higher levels of MVPA were moderately related to lower levels of SED in waves 1 to 4 (r = -0.50 to -0.36). In the later waves, the relationship between MVPA and SED was weak. The relationship between MVPA and TV was very weak. LPA was strongly related to SED in wave 1 for both males and females (r = -0.75) and in wave 2 for males (r = -0.76); they were moderately related in wave 2 for females (r = -0.66), waves 3, 4 and 7 for both sexes (r = -0.33 to -0.66), and waves 5 and 6 for males (r = -0.33). The association between SED and TV was weak and inconsistent.

Associations of Exposure Variables with FAT and VAT

Results from the mixed models are shown in Table 2. At all waves, low levels of MVPA were associated with high levels of FAT (p < 0.01) and VAT (p < 0.05) for males and high levels of FAT (p < 0.01) for females. LPA was positively related to FAT for males (p < 0.05) but not for females (see Model 2 & 3 FAT, Table 2). High levels of TV were positively related to FAT (p < 0.01) and VAT (p < 0.05) for males and females. Importantly, SED was not associated with FAT or VAT. For both males and females, Model 5 (see Table 2) was the best fitting model for FAT and VAT. This model included MVPA and TV for males (for FAT and VAT). For females, Model 5 included MVPA and TV for VAT. Model 5 suggested that for a 10 min increase in MVPA, one would expect approximately 1.9% decrease in (absolute) FAT for males and 1.4% decrease in females. An increase in 1 h for TV would increase FAT by 2.5% for males and 1.6% for females the association is only marginally significant), while an increase in 1 h for TV corresponded to an increase by 1.6% for males and a much larger increase for females, i.e., 4.3%.

DISCUSSION

This study used multi-level modeling of data from childhood through adolescence to quantify the contributions of physical activity, sedentary time, and TV viewing time associated with adiposity, while controlling for individual growth patterns including changes in maturity over time. Multi-level modeling is a powerful approach that reduces known attenuation of effects in cross-sectional studies of growing children. Physical activity was represented as two intensity bands based on metabolic demand: light and moderate or vigorous. We differentiated between sedentary time and TV viewing time because current research suggests that TV time has an independent role in childhood obesity (3,23). Adiposity was represented by two variables: total adiposity and visceral adiposity. When compared to total adiposity, visceral adiposity is more likely to promote the development of the metabolic syndrome (32). Our ability to quantify VAT using DXA scans is novel and a step forward in more fully understanding its role in children's health. Our results show that TV (but not SED) was associated with adiposity. In addition, lower levels of FAT for females. As an example, assuming all other variables are held constant in our multi-level

model, 13-year old girl in our study with average body fat (15 kg) and average weight (55.7 kg) who increased her daily MVPA from 29.9 minutes to the 60-minute guideline would expect to decrease her fat by 0.63 kg. On the other hand, an increase in her TV viewing from the average 2.2 hr/d to 3.2 hr/d would increase her fat by 0.24 kg.

Two results were unexpected. Specifically, lower levels of MVPA did not indicate higher levels of VAT in females, i.e., the association was not significant (p = 0.0708). The lower absolute level and limited range of MVPA and VAT in females, compared to males, may have contributed to this non-significant finding. And LPA had a positive association with FAT in males (see Table 2, FAT Models 3 & 4). This finding suggests that the current accelerometry cut-point for "healthy" physical activity intensity could be too low for males to maintain a healthy body composition.

In general, our findings support the results of other recent empirical studies by Katzmarzyk and colleagues (16), Mitchell and colleagues (23), and Riddoch and colleagues (28) as well as a systematic review by Kelley and colleagues (17). When coupled with these reports, our study suggests that the correct metabolic target for reducing the incidence of childhood obesity is at least moderate-intensity PA. We speculate that MVPA involves different physiological mechanisms than LPA (9,25). Future research in this area is warranted.

Our finding that TV, but not SED, was associated with adiposity suggests that interventions that do not discriminate among type of SED behavior could be ineffective. Similar to our findings, Mitchell et al also concluded that TV was positively associated with adiposity, independently of SED and MVPA (23). It is possible that self-reported TV viewing better represents true sedentary behavior than low accelerometer activity counts. For example, one limitation of the Actigraph accelerometer is its inability to distinguish standing and sitting (1). Furthermore, Fröberg has suggested that SED is an outcome of high levels of adiposity, rather than a cause (7). This assumption is in agreement with a longitudinal study of 785 children (ages 8 to 11 years) by Hjorth and colleagues where baseline DXA-derived fat mass index predicted follow-up SED, but not vice versa (10). Our finding that independent of MVPA, TV is associated with adiposity supports a review by Ekelund et al., that concluded that TV could be uniquely associated with adiposity (5). In another review, Sturm concluded that TV is associated with uncontrolled snacking and extended eating patterns, as well as unhealthy food preferences due to exposure to advertisements (31). TV is also associated with potential change in sleep patterns and sleep disturbances (24) which may influence adiposity. In fact, our multi-level model results suggest that TV viewing has an even more powerful influence on adiposity than MVPA. In short, TV viewing should be considered a sedentary behavior that captures more than just low energy expenditure and this insight should be considered when setting screen time guidelines and designing intervention strategies.

The strengths of our study are the longitudinal design that followed a relatively large sample of youth for 14 years, the use of objective measures of PA and SED, criterion measures of adiposity, and the use of multi-level growth models that controlled time dependent confounders of growth and maturation. However, this study is not without limitations. The study used a convenience sample of mostly white Midwestern individuals with relatively

high socioeconomic status. Throughout the study period, some of the participants dropped out of the study, leading to fewer participants in the later measurement waves. Moreover, not all potential causal variables for adiposity were included. It is possible that uncontrolled confounders, such as a high-energy diet contributed to the results. Lastly, there is a possibility of reverse causation, i.e., that the relationships between independent and dependent variables are bi-directional.

In conclusion, we found that MVPA and TV are independently associated with adiposity. Public health approaches for reducing childhood obesity, which advocate increasing *any* intensity of physical activity and decreasing *any* type of sedentary time should shift to focus on MVPA and pay special attention to reducing TV and the potential unhealthy eating habits associated with it.

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TABLE 1

Characteristics of participants by measurement wave and sex.

	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5	Wave 6	Wave 7
Males (N)	158	201	205	199	162	133	119
Age at DXA, years	5.2 (0.4)	8.7 (0.7)	11.2 (0.3)	13.3 (0.4)	15.4 (0.3)	17.5 (0.4)	19.8 (0.8)
Age at PHV^{a} , years	$13.6\left(0.8 ight)^{**}$	$13.6\left(0.8 ight)^{**}$	$13.6\ {(0.8)}^{**}$	$13.6\left(0.8 ight)^{**}$	$13.7 (0.8)^{**}$	13.7 (0.8) **	$13.7 (0.8)^{**}$
Height, cm	112.3 (5.6)	135.0 (7.3) **	149.2 (7.5)	$163.1 (9.5)^{**}$	175.3 (7.8) **	178.7 (7.5) **	179.8 (7.3) **
Weight, kg	20.5 (3.5)	33.4 (9.0)	45.4 (13.3)	57.9 (16.0)	70.8 (16.7)**	79.3 (17.8) **	$84.0(19.9)^{**}$
FAT ^{<i>a</i>} , kg	$3.8(1.9)^{**}$	8.2 (5.8)	13.8 (8.0)	16.1 (9.2)	$16.8 \left(9.5 ight)^{**}$	$18.6 (9.9)^{**}$	21.1 (12.0) **
Median (IQR)	3.4 (2.8, 4.1)	6.3 (4.5, 10.3)	11.3 (8.1, 17.2)	13.2 (9.2, 21.0)	13.5 (10.4, 20.0)	14.7 (11.5, 23.1)	17.1 (13.6, 23.8)
VAT^{a} , g			$225.3\left(100.1 ight)^{**}$	240.9 (117.8) ^{**}	$240.0 \left(105.3\right)^{**}$	273.9 (109.3) **	$307.0~(140.6)^{*}$
Median (IQR)			191.4 (161.7, 255.1)	204.3 (167.9, 277.9)	205.0 (181.6, 266.0)	238.3 (202.0, 310.5)	273.7 (233.1, 336.6)
Wear Time, h/day	12.0 (0.7)	12.5 (0.8)	12.5 (0.9)	12.7 (1.0)	12.9 (1.3)	12.9 (1.4)	12.7 (1.5)
MVPA ^a , min/day	50.0 (21.7) **	58.2 (25.8) **	59.2 (26.1) ^{**}	47.0 (22.3) **	33.6 (18.3) ^{**}	33.0 (18.2) ^{**}	36.9 (22.3) **
SED^{a} , h/day	4.6 (1.0)	5.4 (1.1)	5.8 (1.1)	6.7 (1.2) **	8.3 (1.2)*	8.4 (1.2)	8.3 (1.4)
TV, h/day	2.3 (1.1)	2.2 (1.0)	2.3 (1.1)	2.2 (1.2)	2.1 (1.2)	$2.0(1.1)^{**}$	2.0 (1.1)
LPA, min/day	393.5 (63.9)	363.9 (67.2)	346.1 (57.1)	308.2 (67.3)	241.9 (58.5)	238.8 (63.2)	229.6 (88.7)
Total PA b , counts/min	971.6 (199.9) **	1052.0 (243.3) **	1123.0 (254.2)**	1036.4 (236.8) **	972.1 (245.1) ^{**}	978.4 (228.3) **	1080.7 (368.7)**
Females (N)	181	214	212	203	156	161	149
Age at scan, years	5.3 (0.4)	8.7 (0.6)	11.2 (0.3)	13.2 (0.4)	15.3 (0.3)	17.5 (0.4)	19.7 (0.7)
Age at PHV, years	11.8 (0.6)	11.8 (0.6)	11.7 (0.6)	11.8 (0.6)	11.7 (0.6)	11.7 (0.6)	11.8 (0.5)
Height, cm	111.2 (5.5)	133.0 (6.8)	149.5 (7.5)	160.6 (6.6)	164.4 (6.3)	166.1 (6.9)	166.0 (7.0)
Weight, kg	20.1 (3.9)	32.0 (8.7)	44.8 (12.5)	55.7 (14.2)	61.9 (14.2)	67.3 (16.4)	69.7 (18.4)
FAT, kg	4.6 (2.4)	9.3 (5.9)	14.5 (7.4)	17.5 (8.4)	20.1 (9.3)	23.1 (10.5)	25.3 (12.0)
Median (IQR)	3.9 (3.3, 5.1)	7.5 (5.2, 12.4)	12.2 (9.0, 17.8)	15.0 (11.1, 21.3)	17.0 (13.8, 24.0)	20.2 (15.3, 27.2)	21.1 (16.6, 31.2)
VAT, g			158.4 (135.6)	174.3 (137.0)	188.2 (131.3)	231.7 (157.9)	256.1 (187.8)
Median (IQR)			108.8 (64.6, 211.7)	119.2 (86.4, 225.9)	142.3 (94.3, 237.5)	177.8 (118.3, 292.4)	184.6 (128.9, 302.3)
Wear Time, h/day	12.0 (0.7)	12.4 (0.9)	12.5 (0.8)	12.8 (1.1)	12.9 (1.3)	13.1 (1.3)	12.8 (1.3)
MVPA, min/day	39.2 (17.2)	40.6 (18.9)	36.0 (17.3)	29.9 (16.6)	22.8 (14.6)	23.2 (14.8)	28.8 (21.8)

	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5	Wave 6	Wave 7
Males (N)	158	201	205	199	162	133	119
SED, h/day	4.7 (0.9)	5.5 (1.1)	6.0 (1.0)	7.1 (1.2)	8.6 (1.1)	8.5 (1.2)	8.4 (1.4)
TV, h/day	2.3 (1.1)	2.0 (1.0)	2.1 (1.1)	2.2 (1.2)	1.9 (1.1)	1.6 (1.1)	1.8 (1.1)
LPA, min/day	400.3 (61.9)	369.8 (66.0)	353.0 (59.8)	309.4 (67.5)	234.9 (60.1)	249.3 (64.1)	238.1 (76.5)
Total PA, counts/min	889.4 (158.5)	936.0 (209.1)	916.0 (195.7)	881.3 (222.4)	838.1 (207.9)	854.4 (218.8)	927.6 (321.5)

VAT not available in Waves 1 and 2.

²PHV, peak height velocity; FAT, total adiposity; VAT, visceral adiposity; MVPA, moderate- or vigorous-intensity physical activity; SED, sedentary time; LPA, light-intensity physical activity. $b_{\text{Total PA counts/min} > 99.}$

 $_{p < 0.05.}^{*}$

 $^{**}_{p}<0.01$ from t-tests comparing males and females at each wave.

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TABLE 2

Associations of FAT and VAT with PA variables in mixed sex-specific growth models.

	Exposure Variables	Estimate	SEa	P-value	AICC ^b	Estimate	SE	P-value	AICC
log FAT			Ma	ıles			Fem	ales	
Model 1	MVPA	-0.020	0.003	<.0001	420.6	-0.013	0.004	0.0003	-42.5
Model 2	MVPA	-0.021	0.004	<.0001	425.2	-0.014	0.004	0.0002	-35.5
	LPA	0.014	0.007	0.0628		0.007	0.006	0.2170	
Model 3	MVPA	-0.021	0.004	<.0001	432.4	-0.014	0.004	0.0002	-27.2
	LPA	0.017	0.008	0.0465		0.006	0.006	0.3288	
	SED	0.006	0.008	0.4457		-0.001	0.006	0.8225	
Model 4	MVPA	-0.021	0.004	<.0001	427.2	-0.014	0.004	0.0002	-27.4
	LPA	0.019	0.008	0.0280		0.007	0.006	0.2690	
	SED	0.006	0.008	0.4490		-0.001	0.006	0.9301	
	TV	0.026	0.007	0.0003		0.016	0.006	0.0029	
Model 5	MVPA	-0.019	0.003	<.0001	416.4	-0.013	0.004	0.0003	-42.6
	TV	0.025	0.007	0.0004		0.016	0.005	0.0032	
log VAT									
Model 1	MVPA	-0.013	0.003	0.0001	-5.6	-0.014	0.008	0.0792	1116.5
Model 2	MVPA	-0.013	0.004	0.0003	1.8	-0.014	0.008	0.0736	1123.3
	LPA	-0.003	0.007	0.6878		0.005	0.012	0.6537	
Model 3	MVPA	-0.012	0.004	0.0006	8.6	-0.015	0.008	0.0680	1130.1
	LPA	0.001	0.008	0.8769		0.003	0.013	0.7992	
	SED	0.008	0.007	0.2373		-0.004	0.012	0.7194	
Model 4	MVPA	-0.013	0.004	0.0003	9.1	-0.015	0.008	0.0642	1123.3
	LPA	0.003	0.008	0.6966		0.006	0.013	0.6556	
	SED	0.008	0.007	0.2327		-0.001	0.012	0.9126	
	TV	0.019	0.007	0.0056		0.044	0.012	0.0002	
Model 5	MVPA	-0.014	0.003	<.0001	-5.6	-0.014	0.008	0.0708	1109.6
	TV	0.019	0.007	0.0053		0.043	0.012	0.0002	

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Mixed models include independent variables that describe growth and PA variables reported in the table. Growth models for FAT are mixed models that include DXA machine type (2000 vs. 4500), centered VAT are mixed models with random intercept and residual heterogeneous autoregressive covariance with independent variables that include centered age, centered age², maturity (pre vs post) for both males age, centered age², centered age³, height, maturity (pre vs post) for both males and females. Random intercept and slope for centered age are included to incorporate individual growth. Growth models for and females, and centered age³ and height for females only.

^aSE, standard error.

 $b_{\rm AICC}$, Akaike's Information Criteria corrected - takes into account the number of estimated parameters.

Unit for change for MVPA=10 min; for LPA, SED, and TV=1 h