Published in final edited form as:

Med Sci Sports Exerc. 2011 March ; 43(3): 443–448. doi:10.1249/MSS.0b013e3181ef3b0a.

Effects of Adiposity on Physical Activity in Childhood: Iowa Bone Development Study

Soyang Kwon¹, Kathleen F. Janz^{2,1}, Trudy L. Burns^{1,3}, and Steven M. Levy^{4,1}

¹Department of Epidemiology, University of Iowa, Iowa City, IA, USA

²Department of Health and Sport Studies, University of Iowa, Iowa City, IA, USA

³Department of Pediatrics, University of Iowa, Iowa City, IA, USA

⁴Department of Preventive and Community Dentistry, University of Iowa, Iowa City, IA, USA

Abstract

Purpose—The aim of this study was to examine whether adiposity level is associated with subsequent physical activity (PA) level in childhood.

Methods—Study participants were 326 children participating in the Iowa Bone Development Study. PA and fat mass were measured using accelerometers and dual energy X-ray absorptiometry (DXA) at approximately 5, 8, and 11 years of age. Data for relevant variables such as parents' education and PA levels and family income were also collected. Gender-specific generalized linear models were fit to examine the association between percent body fat (BF%) at age 8 and intensity-weighted moderate- to vigorous-intensity PA (IW-MVPA) at age 11.

Results—After adjusting for IW-MVPA at age 8, the interval between the age 5 and 8 examinations, residualized change scores of BF% and IW-MVPA from age 5 to 8, and mother's education level, BF% at age 8 was negatively associated with IW-MVPA at age 11 among boys ($P < 0.05$). After adjusting for IW-MVPA at age 8, physical maturity, and family income, BF% at age 8 was negatively associated with IW-MVPA at age 11 among girls ($P < 0.05$). Categorical data analysis also showed that the odds of being in the lowest quartile relative to the highest quartile of IW-MVPA at age 11 for boys and girls with high BF% at age 8 were approximately four times higher than the odds for those with low BF% at age 8 ($P < 0.05$).

Conclusion—This study suggests that adiposity level may be a determinant of PA behavior. Specific intervention strategies for overweight children may be needed to promote PA.

Keywords

Obesity; fatness; determinant; exercise; accelerometer; longitudinal

Corresponding author: Kathleen F. Janz, 130 FH, Department of Health and Sport Studies, University of Iowa, Iowa City IA 52242, Office phone: 319 335-9345, Office Fax: 319 335-6669, kathleen-janz@uiowa.edu.

DISCLAIMER

All manuscripts submitted to *Medicine & Science in Sports & Exercise*® for evaluation are protected by international copyright laws. Reviewers have permission to print this manuscript on paper in their individual effort to provide a thorough critique of the scientific quality of the manuscript to the Editorial Office. Any other use of the material contained in the manuscript or distribution of the manuscript to other individuals is strictly prohibited by federal, state, and international laws governing copyright. Such actions are also in direct conflict with the professional and ethical standards of the American College of Sports Medicine and its official journal, *Medicine & Science in Sports & Exercise*®.

The Editorial Office staff of *MSSE*® thanks you for participating in the review process. If you have any problems reviewing this manuscript, or you are unable to meet the deadline given, please write to the Editorial Office at msse@acsm.org.

INTRODUCTION

Physical inactivity is known to increase adiposity level in children. However, the relationship between physical activity (PA) and adiposity may be bi-directional. That is, not only may physical inactivity be a contributor to body fat gain, but adiposity status may influence PA behaviors (this has been referred to as the reverse causation (9, 18) hypothesis). Although cross-sectional studies have demonstrated a negative association between PA and adiposity levels in children (15), a review of prospective studies (28) concluded that low levels of baseline PA were only weakly or not at all associated with body fat gain. It is conceivable that the reported negative cross-sectional relationship may be due to a reduction of PA as a consequence of a high level of adiposity.

A high level of adiposity may negatively influence PA participation by children, presumably through psychological, societal, and physical functioning, such as low self-efficacy, poor body image, fear of being teased by peers, low athletic proficiency, and discomfort from heaviness. There is some evidence that obese youth are likely to be less active because of weight stigma (4). Storch *et al.* (25) showed that peer victimization among overweight youth is linked to lower levels of PA. Faith *et al.* (7) also showed that weight criticism during sports and PA is associated with negative attitudes about sports and lower participation in PA among overweight students. Must and Tybor (18) have suggested that, given the fact that PA in children often occurs as part of organized sport, overweight children may be less likely to want to participate in PA, either due to the fear of being teased or because they are “less athletic.”

Five prospective adult population-based cohort studies (3, 6, 17, 20, 29) consistently demonstrated that obesity is negatively associated with PA level later in life. To our knowledge, no study to date has explicitly addressed the reverse causation hypothesis in children. Testing this reverse causation hypothesis may provide evidence to promote PA at an early age before excess body fat is accumulated. If obesity leads to a reduction in PA later in life, specific intervention strategies for overweight children to promote PA would be warranted. The aim of this study was to examine whether adiposity level is associated with subsequent PA level in childhood.

METHODS

Participants

Study participants were a cohort of children participating in the Iowa Bone Development Study which is an ongoing longitudinal study of bone health during childhood and adolescence. The study participants are a sub-set of Midwestern children recruited during 1998 to 2001 from a cohort of 890 families then participating in the Iowa Fluoride Study. Detailed information about the study design and demographic characteristics of participants can be found elsewhere (10, 13, 14). Accelerometer and dual energy X-ray absorptiometry (DXA) measurements were conducted three times per child at approximately 5, 8, and 11 years of age (4.3 to 6.8 years of age at the first examination, 7.6 to 10.8 years at the second examination, and 10.5 to 12.4 years at the third examination). If the time interval between accelerometer measurement and DXA scanning was greater than 1.5 years for any age examination, the data for that examination were excluded. Four hundred thirty-six children completed both accelerometer and DXA examinations at the age 5 examination conducted between February 1998 and November 2000; 502 at the age 8 examination (September 2000 to December 2004); and 454 at the age 11 examination (October 2003 to September 2006). Five hundred seventy-seven children (51% girls) completed at least one examination and 326 (56% girls) completed all three examinations. Those 326 children (95% white) served as the study sample for this report. We examined the association between adiposity at age 8

and PA at age 11, so that data obtained at the age 5 examination could be used to account for the preceding three-year change in PA and adiposity (20). The study was approved by the University of Iowa Institutional Review Board (Human Subjects). Written informed consent was provided by the parents of the children and assent was obtained from the children.

Adiposity measurements

At the age 5 and 8 examinations, whole body scans were conducted using a Hologic QDR 2000 DXA (Hologic, Waltham, MA) with software version 7.20B in the fan-beam mode. At the age 11 examination, the Hologic QDR 4500A DXA (Delphi upgrade) with software version 12.3 and fan-beam mode was used for scan acquisition. Quality control scans were performed daily using the Hologic phantom. To adjust for the difference between the two DXA machines, translational equations from 4500A DXA measures to 2000 DXA measures for age 11 records were used. The translational equations (linear regression equations) were developed specifically for the two scanners in a methodological study where 60 of the children (32 boys, 28 girls) aged 9.9 to 12.4 years (mean = 11.4 years, SD = 0.4 years) were scanned on each machine in random order during one clinic visit (13). Fat mass (kg) was derived from the DXA scan images. Percent body fat (BF%) was calculated as fat mass (kg) divided by body weight (kg).

Physical activity measurements

Actigraph uniaxial accelerometers (model number 7164, Pensacola, FL) were used to measure PA level. The procedure for PA measurement has been described elsewhere (11, 12). Accelerometer movement counts were collected in a one-minute interval (one-minute epoch). At the time of the age 5 and 8 examinations, children were asked to wear the monitor during waking hours for four consecutive days, including one weekend day, during the fall season (September through November). At the time of the age 11 examination, they were asked to wear the monitor during waking hours for five consecutive days, including both weekend days during the fall season. In the accelerometer data reduction process, an interval of 20 or more consecutive minutes of zero accelerometer counts was considered as not wearing the monitor and invalid data (5). The two inclusion criteria for accelerometer data were having valid data for more than eight hours per day and wearing the monitor for three or more of the days. Intensity-weighted moderate- to vigorous-intensity PA (IW-MVPA) was defined as the daily sum of accelerometer counts derived during moderate- to vigorous-PA (MVPA) determined by 3,000 or greater accelerometer movement counts per minute (22, 26).

Covariate measurements

At each DXA visit, research nurses trained in anthropometry measured the child's height and weight. Sitting height was measured at age 11 to calculate maturity offset (year from peak height velocity) using predictive equations established by Mirwald and colleagues (16). The equations were developed in white Canadian children and adolescents and they have been cross-validated in another Canadian sample and a Flemish sample (16). To estimate physical maturity status, the maturity offset variable was dichotomized as pre-peak height velocity (pre-mature) or post-peak height velocity (mature).

Family income and parental education level data were obtained from a mailed family demographic questionnaire completed by each child's parents in 2007. Family income level was dichotomized into less than \$40,000/year and \$40,000/year or more for data analysis. Education levels of mothers and fathers were also dichotomized into some college or lower and college graduate or higher. The modified Baecke Physical Activity Questionnaire (2) was administered to the child's mother and father at the child's age 8 examination. The questionnaire was one of the most widely used tools for assessing PA in adults at the time of

data collection (19, 21). It was developed to evaluate a person's PA in three domains: work activity, sports activity, and leisure activity. Sports activity and leisure activity estimated by the questionnaire were used to determine PA levels of parents.

Statistical analysis

Gender-specific analyses were conducted using SAS version 9.2 (Cary, NC). Descriptive analyses, including frequency distributions and estimation of summary descriptive measures, were conducted. The age 11 IW-MVPA outcome variable was not normally distributed and a Box-Cox power transformation (labeled 'transformed IW-MVPA') was performed using the SAS TRANSREG procedure. Bivariate analyses were performed to identify a set of covariates included during the model development process; Pearson correlation coefficients between transformed IW-MVPA at age 11 and continuous covariates were estimated, and two-sample t-tests for IW-MVPA at age 11 and categorical covariates were performed. Potential covariates were considered and if the *P*-value was less than 0.10, the variable was considered for inclusion in the final model. The BF% and IW-MVPA data from the age 5 examination were used to account for the changes in BF% and IW-MVPA between the first two examinations (ages 5 and 8). Because the error terms of BF% at ages 5 and 8 were not independent (autocorrelated), the BF% residualized change score variable, which was defined as the residual from regressing the yearly change in BF% from age 5 to age 8 on BF% at age 8, was created using the SAS AUTOREG procedure (23). The IW-MVPA residualized change score variable was created in the same manner.

Gender-specific generalized linear models were fit using the SAS GENMOD procedure. The main exposure variable was BF% at age 8 and the main outcome variable was transformed IW-MVPA at age 11. Models included covariates which could possibly confound a relationship between IW-MVPA and BF% based on bivariate analysis results. Several nested models were fit to examine how a parameter estimate for the exposure variable behaved when each additional covariate was included one by one. The likelihood ratio test was performed to compare the fit of the nested models. After model fitting, model diagnostics were conducted. For categorical data analysis, BF% level at age 8 was dichotomized into low BF% (< 25% for boys and < 32% for girls) or high BF% ($\geq$ 25% for boys and $\geq$ 32% for girls) (24, 30). IW-MVPA at age 11 was divided into tertiles. Odds ratios and their 95% confidence intervals (CIs) were estimated to examine associations between BF% levels at age 8 and IW-MVPA levels at age 11, using logistic regression analysis models. These models included the same set of covariates as final generalized linear models. The significance level was set at 0.05.

RESULTS

Table 1 presents means and 95% CIs of study variables. Fat mass increased a mean of approximately 5.1 kg for boys and 5.3 kg for girls from age 8 to 11. Participants, on average, wore accelerometers for more than 12 hours per day. IW-MVPA was higher in boys than in girls. IW-MVPA tended to increase with age among boys, but not among girls. Table 2 shows results from descriptive analyses of potential categorical covariates and t-tests for the significance of the mean difference of IW-MVPA at age 11 between categories of those potential covariates. Approximately 13% of the study sample reported family income lower than \$40,000/year. Approximately two-thirds of parents reported college graduate or higher education levels. At age 11, all boys were classified as pre-peak height velocity (pre-mature), whereas 20% of girls were classified as post-peak height velocity (mature). In t-test results, IW-MVPA at age 11 was positively associated with mother's education level and father's education level for boys ($P < 0.10$). IW-MVPA at age 11 was positively associated with family income and negatively associated with maturity for girls ($P < 0.10$).

Based on the Box-Cox transformation, the IW-MVPA variable at age 11 was square root-transformed for boys ($\lambda = 0.5$) and log-transformed for girls ($\lambda = 0$). Pearson correlation coefficients for transformed IW-MVPA at age 11 and BF% at age 8, and transformed IW-MVPA at age 11 and potential covariates are presented in Table 3. A significant negative correlation between transformed IW-MVPA at age 11 and BF% at age 8 was observed in both boys and girls ($P < 0.05$). IW-MVPA at age 8 was significantly positively correlated with transformed IW-MVPA at age 11 in both boys and girls ($P < 0.05$). Transformed IW-MVPA at age 11 was positively correlated with the time interval between the age 8 and 11 examinations and negatively correlated with residualized change scores of BF% and IW-MVPA only among boys ($P < 0.10$). Age, mother's PA, and father's PA were not significantly correlated with transformed IW-MVPA at age 11 for either boys or girls. Ultimately, for boys, IW-MVPA at age 8, the interval between the age 8 and 11 examinations, residualized change scores of BF% and IW-MVPA, and mother's education were selected as covariates in the final model. Because of a modest association between mother's education level and father's education level ($\kappa = 0.37$, P for chi-square test < 0.0001), only mother's education level was included. For girls, IW-MVPA at age 8, family income, and physical maturity were selected as covariates in the final model.

Several nested models were fit to predict transformed IW-MVPA at age 11 based on BF% at age 8. Because full models provided a better fit to the data than reduced models in both boys and girls based on the likelihood ratio test, the full models are presented in Table 4. Two boys and two girls identified as outliers in model diagnostics were excluded from the final model. After adjusting for covariates, BF% at age 8 was significantly negatively associated with IW-MVPA at age 11 among boys ($P < 0.05$). After adjusting for covariates, BF% at age 8 was significantly negatively associated with IW-MVPA at age 11 among girls ($P < 0.05$).

In categorical data analysis, 23% of boys and 26% of girls were identified as having high BF% ($\geq 25\%$ BF for boys and $\geq 32\%$ BF for girls) at age 8. In a fully-adjusted logistic regression model (Table 5), boys and girls with high BF% at age 8 were more likely to be in the lowest tertile of IW-MVPA at age 11 than their counterparts with low BF% at age 8 (OR: 4.38, 95% CI: 1.05, 18.24 for boys; OR: 4.48, 95% CI: 1.35, 14.85 for girls, reference group: the highest tertile of IW-MVPA at age 11).

DISCUSSION

The aim of this study was to examine whether adiposity level is associated with subsequent PA level in childhood (the reverse causation hypothesis). This study found that, in continuous data analysis, BF% at age 8 was negatively associated with IW-MVPA at age 11 in both boys and girls. Categorical data analysis also showed that boys and girls with high BF% at age 8 were more likely to have low PA levels at age 11 than those with lower BF% at age 8. These findings are consistent with those of five adult cohort studies (3, 6, 17, 20, 29), where obesity was a significant predictor of PA level later in life. However, the current study results are inconsistent with Sallis *et al.* (23), who reported no association between skinfold thickness category at baseline and total activity accelerometer counts measured for one day at a 20-month follow-up among 732 children who were fourth graders at baseline.

In additional analysis, we examined the association between early adiposity and later PA using other PA indicators. BF% at age 8 was significantly negatively associated with total activity (the daily sum of ≥ 100 accelerometer counts per minutes) at age 11 in both boys and girls ($P < 0.05$). When time spent in MVPA (Time MVPA) was used as a PA indicator, the negative association between Time MVPA at age 11 and BF% at age 8 was significant among girls ($P < 0.05$) and suggestive among boys ($P < 0.10$). Considering that there was a

significant negative association between BF% at age 8 and IW-MVPA at age 11 among boys, these results may imply that boys with low BF% are more likely to engage in higher intensity PA than those with high BF%. From an energy expenditure perspective, IW-MVPA was expected to better represent PA level than Time MVPA in establishing a relationship between adiposity and PA. PA intensity may be critical particularly for boys. However, researchers should be cautious because the use of IW-MVPA may amplify measurement error, which is derived from the difference of relative intensities between individuals when absolute intensity is given.

The current study findings indicate that adiposity status may be a determinant of PA behavior in childhood. Godin *et al.* (8) suggested a theoretical model of the reverse causation hypothesis based on Ajzen's Theory of Planned Behavior (TPB) (1). According to this model, adiposity may impact PA behaviors by influencing cognition such as intention (motivation) and perceived behavioral control (ease or difficulty in engaging in the behavior, e.g., social barriers). Although we assumed that obesity-related psychological, societal, and physical functioning may negatively influence PA participation, we were not able to examine whether these are mediating factors in an association between early adiposity and subsequent PA because those potentially mediating variables were not assessed in the Iowa Bone Development Study.

The study results suggest at least two significant points in terms of public health implications. First, this study suggests that a new perspective is needed to best develop intervention strategies to promote PA and to prevent obesity in children. This study suggests weight status-specific intervention strategies for PA promotion, since overweight children may have different barriers to PA participation. Given the childhood obesity epidemic, it would be critical to identify specific barriers for overweight children and develop strategies to overcome these barriers. Second, the study results support PA promotion interventions from an early age, before excess fat is accumulated. Once excess fat is accumulated in early childhood possibly due to a low level of PA, it may lead to low PA participation. In turn, lack of PA may exacerbate fat accumulation. This assumption is supported by Valerio's study (27) showing that overweight and obese children 7 years of age had a higher BMI increase at three-year follow-up than normal-weight children. PA interventions from an early age are recommended to prevent excess fat accumulation throughout childhood and later in life.

Several limitations of this study should be acknowledged. We only included data from those who completed all three examinations. Loss to follow-up may have caused selection bias; but, IW-MVPA and BF% levels at age 8 were not significantly different between those who completed all three examinations and those who did not. The study sample was not randomly selected from Iowa Fluoride Study participants; which could also have led to selection bias. Therefore, an association between early adiposity and subsequent PA observed in this sample may not represent that in the general child population, and caution should be taken in generalizing the results. In the participant cohort, approximately 95% were white, which is a lower risk population for childhood obesity than the Hispanic or African American population. However, homogeneity of ethnicity and living-environment can be an advantage because unknown confounders are less likely to exist. Genetic predisposition was not considered. This observational study cannot eliminate error introduced by residual and unmeasured confounding factors.

Nonetheless, to our knowledge, this study is the first prospective cohort study in a fairly large childhood sample to explicitly examine the reverse causation hypothesis. The use of objective and accurate measures for both PA and adiposity helped reduce measurement error

and increase internal validity. Examinations at three time points allowed accounting for the preceding changes in adiposity and PA between the first two examinations.

In conclusion, this study showed that children with low adiposity were more likely to be active at three-year follow-up than their counterparts with high adiposity. Adiposity may be a determinant of PA behavior in childhood. Regarding future research, more evidence should be accumulated to support the reverse causation hypothesis in childhood. Research is required to understand the mechanism underlying the effect of adiposity status on PA behaviors. It would be valuable to test the hypothesis that obesity-related psychological, societal, and physical functioning are mediating factors in an association between early adiposity and subsequent PA, using existing datasets containing PA, adiposity, and related psycho-societal measurement data.

Acknowledgments

This study was supported by the National Institute of Dental and Craniofacial Research R01-DE12101 and R01-DE09551, and the General Clinical Research Centers Program from the National Center for Research Resources, M01-RR00059. We gratefully acknowledge and thank the children, parents, and staff of the Iowa Fluoride Study and the Iowa Bone Development Study, because without their contributions, this work would not have been possible. Results of the present study do not constitute endorsement by ACSM.

REFERENCES

1. Ajzen I. The theory of planned behavior. *Organ Behav Hum Decis Process*. 1991; 50:179–211.
2. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr*. 1982; 36(5):936–42. [PubMed: 7137077]
3. Bak H, Petersen L, Sorensen TI. Physical activity in relation to development and maintenance of obesity in men with and without juvenile onset obesity. *Int J Obes Relat Metab Disord*. 2004; 28(1): 99–104. [PubMed: 14610530]
4. Brownell KD, Schwartz MB, Puhl RM, Henderson KE, Harris JL. The need for bold action to prevent adolescent obesity. *J Adolesc Health*. 2009; 45(3 Suppl):S8–17. [PubMed: 19699441]
5. Cliff DP, Reilly JJ, Okely AD. Methodological considerations in using accelerometers to assess habitual physical activity in children aged 0-5 years. *J Sci Med Sport*. 2009; 12(5):557–67. [PubMed: 19147404]
6. Ekelund U, Brage S, Besson H, Sharp S, Wareham NJ. Time spent being sedentary and weight gain in healthy adults: reverse or bidirectional causality? *Am J Clin Nutr*. 2008; 88(3):612–7. [PubMed: 18779275]
7. Faith MS, Leone MA, Ayers TS, Heo M, Pietrobelli A. Weight criticism during physical activity, coping skills, and reported physical activity in children. *Pediatrics*. 2002; 110(2 Pt 1):e23. [PubMed: 12165622]
8. Godin G, Belanger-Gravel A, Nolin B. Mechanism by which BMI influences leisure-time physical activity behavior. *Obesity (Silver Spring)*. 2008; 16(6):1314–7. [PubMed: 18388892]
9. Hu, FB. Physical activity, sedentary behaviors, and obesity. In: Hu, FB., editor. *Obesity Epidemiology*. Oxford University Press; New York:NY: 2008. p. 301-319.
10. Janz KF, Burns TL, Levy SM. Iowa Bone Development Study. Tracking of activity and sedentary behaviors in childhood: the Iowa Bone Development Study. *Am J Prev Med*. 2005; 29(3):171–8. [PubMed: 16168865]
11. Janz KF, Burns TL, Torner JC, et al. Physical activity and bone measures in young children: the Iowa bone development study. *Pediatrics*. 2001; 107(6):1387–93. [PubMed: 11389262]
12. Janz KF, Gilmore JM, Burns TL, et al. Physical activity augments bone mineral accrual in young children: The Iowa Bone Development study. *J Pediatr*. 2006; 148(6):793–9. [PubMed: 16769389]
13. Janz KF, Kwon S, Letuchy EM, et al. Sustained effect of early physical activity on body fat mass in older children. *Am J Prev Med*. 2009; 37(1):35–40. [PubMed: 19423269]

14. Janz KF, Levy SM, Burns TL, Torner JC, Willing MC, Warren JJ. Fatness, physical activity, and television viewing in children during the adiposity rebound period: the Iowa Bone Development Study. *Prev Med.* 2002; 35(6):563–71. [PubMed: 12460524]
15. Jimenez-Pavon D, Kelly J, Reilly JJ. Associations between objectively measured habitual physical activity and adiposity in children and adolescents: Systematic review. *Int J Pediatr Obes.* 2009;1–16.
16. Mirwald RL, Baxter-Jones AD, Bailey DA, Beunen GP. An assessment of maturity from anthropometric measurements. *Med Sci Sports Exerc.* 2002; 34(4):689–94. [PubMed: 11932580]
17. Mortensen LH, Siegler IC, Barefoot JC, Gronbaek M, Sorensen TI. Prospective associations between sedentary lifestyle and BMI in midlife. *Obesity (Silver Spring).* 2006; 14(8):1462–71. [PubMed: 16988090]
18. Must A, Tybor DJ. Physical activity and sedentary behavior: a review of longitudinal studies of weight and adiposity in youth. *Int J Obes (Lond).* 2005; 29(Suppl 2):S84–96. [PubMed: 16385758]
19. Ono R, Hirata S, Yamada M, Nishiyama T, Kurosaka M, Tamura Y. Reliability and validity of the Baecke physical activity questionnaire in adult women with hip disorders. *BMC Musculoskelet Disord.* 2007; 8:61. [PubMed: 17610746]
20. Petersen L, Schnohr P, Sorensen TI. Longitudinal study of the long-term relation between physical activity and obesity in adults. *Int J Obes Relat Metab Disord.* 2004; 28(1):105–12. [PubMed: 14647181]
21. Pals MA, Peeters PH, Bueno-De-Mesquita HB, et al. Validity and repeatability of a modified Baecke questionnaire on physical activity. *Int J Epidemiol.* 1995; 24(2):381–8. [PubMed: 7635600]
22. Puyau MR, Adolph AL, Vohra FA, Butte NF. Validation and calibration of physical activity monitors in children. *Obes Res.* 2002; 10(3):150–7. [PubMed: 11886937]
23. Sallis JF, Alcaraz JE, McKenzie TL, Hovell MF. Predictors of change in children's physical activity over 20 months. Variations by gender and level of adiposity. *Am J Prev Med.* 1999; 16(3):222–9. [PubMed: 10198662]
24. Stevens J, Murray DM, Baggett CD, et al. Objectively assessed associations between physical activity and body composition in middle-school girls: the Trial of Activity for Adolescent Girls. *Am J Epidemiol.* 2007; 166(11):1298–305. [PubMed: 17855391]
25. Storch EA, Milsom VA, Debraganza N, Lewin AB, Geffken GR, Silverstein JH. Peer victimization, psychosocial adjustment, and physical activity in overweight and at-risk-for-overweight youth. *J Pediatr Psychol.* 2007; 32(1):80–9. [PubMed: 16601255]
26. Treuth MS, Schmitz K, Catellier DJ, et al. Defining accelerometer thresholds for activity intensities in adolescent girls. *Med Sci Sports Exerc.* 2004; 36(7):1259–66. [PubMed: 15235335]
27. Valerio G, D'Amico O, Adinolfi M, Munciguerra A, D'Amico R, Franzese A. Determinants of weight gain in children from 7 to 10 years. *Nutr Metab Cardiovasc Dis.* 2006; 16(4):272–8. [PubMed: 16679219]
28. Wareham NJ, van Sluijs EM, Ekelund U. Physical activity and obesity prevention: a review of the current evidence. *Proc Nutr Soc.* 2005; 64(2):229–47. [PubMed: 15960868]
29. Weiss DR, O'Loughlin JL, Platt RW, Paradis G. Five-year predictors of physical activity decline among adults in low-income communities: a prospective study. *Int J Behav Nutr Phys Act.* 2007; 4:2. [PubMed: 17233904]
30. Williams DP, Going SB, Lohman TG, et al. Body fatness and risk for elevated blood pressure, total cholesterol, and serum lipoprotein ratios in children and adolescents. *Am J Public Health.* 1992; 82(3):358–63. [PubMed: 1536350]

Table 1

Characteristics (means and 95% confidence intervals) of participants at the age 5, 8 and 11 examinations for 142 boys and 184 girls

	Age 5		Age 8		Age 11	
	Boys	Girls	Boys	Girls	Boys	Girls
	Mean (95% CI)		Mean (95% CI)		Mean (95% CI)	
Age (years)	5.2 (5.2, 5.3)	5.3 (5.2, 5.3)	8.7 (8.6, 8.8)	8.7 (8.6, 8.8)	11.2 (11.2, 11.3)	11.3 (11.2, 11.3)
Height (m)	1.12 (1.11, 1.13)	1.11 (1.10, 1.12)	1.34 (1.33, 1.35)	1.33 (1.32, 1.34)	1.49 (1.48, 1.50)	1.49 (1.48, 1.50)
Body weight (kg)	20.4 (19.8, 21.0)	20.0 (19.5, 20.6)	32.4 (31.0, 33.8)	31.8 (30.5, 33.0)	44.5 (42.4, 46.6)	44.6 (42.8, 46.4)
Time MVPA (min/d)	31 (28, 34)	24 (22, 26)	40 (36, 43)	25 (23, 27)	41 (38, 44)	23 (21, 25)
IW-MVPA ($\times 10^3$ ct/d)	143 (128, 159)	116 (103, 129)	188 (168, 208)	124 (112, 137)	204 (184, 224)	108 (97, 119)
Monitor-wearing time (hr/d)	12.1 (12.0, 12.2)	12.1 (12.0, 12.2)	12.5 (12.4, 12.6)	12.4 (12.3, 12.5)	12.4 (12.2, 12.5)	12.3 (12.2, 12.4)
Fat mass (kg)	3.8 (3.4, 4.1)	4.6 (4.2, 4.9)	7.6 (6.6, 8.5)	9.1 (8.3, 10.0)	12.7 (11.2, 14.3)	14.4 (13.1, 15.6)
Percent body fat (%)	17.9 (17.1, 18.8)	22.0 (21.2, 22.8)	21.5 (20.1, 22.9)	26.8 (25.5, 28.0)	26.0 (24.2, 27.9)	30.0 (28.6, 31.4)

CI, confidence interval; Time MVPA, daily minutes spent in moderate- to vigorous-intensity physical activity; IW-MVPA, the daily sum of accelerometer movement counts derived during moderate- to vigorous-intensity physical activity.

Table 2

Comparisons of the means of IW-MVPA at age 11 between potential covariate categories

Variable	Category	Boys (n=142)		Girls (n=184)	
		Percentage	Mean (95% CI) ($\times 10^3$ ct/d)	Percentage	Mean (95% CI) ($\times 10^3$ ct/d)
Physical maturity	Mature	0%	--	20%	88 (67, 109) ^a
Family income ^b	Pre-mature	100%	204 (184, 224)	80%	113 (100, 126)
	< \$40,000/yr	9%	197 (102, 292)	16%	88 (65, 111) ^a
	≥ \$40,000/yr	74%	206 (184, 229)	69%	116 (102, 130)
Mother's education	< college graduate	31%	178 (141, 215) ^a	33%	103 (86, 119)
	≥ college graduate	69%	216 (192, 241)	67%	111 (96, 126)
Father's education ^b	< college graduate	38%	182 (157, 206) ^a	44%	105 (85, 124)
	≥ college graduate	61%	220 (191, 250)	55%	112 (98, 125)

IW-MVPA, the daily sum of accelerometer movement counts during moderate- to vigorous-intensity physical activity.

^a $P < 0.10$.

^b Due to missing data, the sum of percentages is less than 100%.

Table 3

Associations between transformed IW-MVPA at age 11 and percent body fat and potential covariates

Variable	Boys (n=142)	Girls (n=184)
	<i>r</i> (95% CI)	<i>r</i> (95% CI)
Exposure variable		
BF% at age 8	−0.30 (−0.45, −0.15) ^a	−0.23 (−0.37, −0.09) ^a
Potential covariate		
Age	0.11 (−0.05, 0.27)	−0.08 (−0.22, 0.07)
Interval between age 8 and 11 examinations	0.18 (0.01, 0.33) ^a	0.05 (−0.10, 0.19)
IW-MVPA at age 8	0.42 (0.27, 0.55) ^a	0.27 (0.13, 0.40) ^a
IW-MVPA residualized change score from age 5 to 8	−0.16 (−0.31, 0.01) ^b	0.01 (−0.13, 0.16)
BF% residualized change score from age 5 to 8	−0.15 (−0.31, 0.01) ^b	0.04 (−0.10, 0.19)
Monitor-wearing time at age 11	0.11 (−0.06, 0.27)	0.04 (−0.10, 0.18)
Mother's sports activity	0.07 (−0.10, 0.24)	0.02 (−0.13, 0.16)
Mother's leisure activity	−0.02 (−0.19, 0.15)	0.08 (−0.07, 0.23)
Father's sports activity	0.07 (−0.11, 0.24)	0.06 (−0.11, 0.23)
Father's leisure activity	−0.12 (−0.30, 0.05)	0.02 (−0.14, 0.19)

IW-MVPA, the daily sum of accelerometer movement counts during moderate- to vigorous-intensity physical activity; BF%, percent body fat. In Box-Cox transformations of IW-MVPA at age 11, λ was 0.5 for boys and 0 for girls. Pearson correlation coefficients are presented.

^a $P < 0.05$.

^b $P < 0.10$.

Table 4

Generalized linear model analysis of transformed IW-MVPA at age 11 predicted by percent body fat at age 8

Boys (n=142) ^a		Girls (n=153) ^b	
Parameter	β (95% CI)	Parameter	β (95% CI)
BF% at age 8 (%)	-2.47 (-4.37, -0.57)	BF% at age 8 (%)	-1.36 (-2.53, -0.19)
IW-MVPA at age 8 ($\times 10^3$ ct/d)	0.37 (0.23, 0.51)	IW-MVPA at age 8 ($\times 10^3$ ct/d)	0.16 (0.05, 0.26)
Mother's education	31.5 (-1.9, 64.9)	Family income	23.6 (0.7, 46.7)
Interval between the age 8 and 11 examinations (year)	-23.5 (-49.1, 3.0)	Physical maturity	0.29 (-25.8, 26.3)
BF% residualized change score	-33.7 (-52.3, -15.1)	Intercept	242 (203, 281)
IW-MVPA residualized change score	-0.001 (-0.001, -0.0002)	--	--
Intercept	348 (249, 447)	--	--

IW-MVPA, the daily sum of accelerometer movement counts during moderate- to vigorous-intensity physical activity; β , parameter estimate; CI, confidence interval; BF% , percent body fat. In Box-Cox transformations of IW-MVPA at age 11 ($\times 10^3$ ct/d), λ was 0.5 for boys and 0 for girls.

^aBecause of two outliers, 140 boys were included in the final model.

^bBecause of missing data for physical maturity (missing n = 2) and family income (missing n = 28, missing data for both physical maturity and family income in one girl) and two outliers, 153 girls were included in the final model.

Table 5

Odds of being in the lowest tertile relative to the highest tertile of IW-MVPA at age 11 for boys and girls with high^a vs. low percent body fat at age 8

	Boys (n=142) ^b	Girls (n=155) ^c
IW-MVPA at age 11	Odds ratio (95% CI)	Odds ratio (95% CI)
The lowest tertile	4.38 (1.05, 18.24)	4.48 (1.35, 14.85)
The middle tertile	1.89 (0.40, 8.97)	2.40 (0.82, 6.07)
The highest tertile	Reference	Reference
<i>P</i> for trend	<0.05	<0.05

CI, confidence interval; IW-MVPA, the daily sum of accelerometer movement counts during moderate- to vigorous-intensity physical activity.

^aHigh percent body fat was defined as $\geq 25\%$ body fat for boys and $\geq 32\%$ body fat for girls, resulting in 23% of boys and 26% of girls being categorized into high percent body fat.

^bAdjusted for IW-MVPA at age 8, BF% residualized change score from age 5 to 8, IW-MVPA residualized change score from age 5 to 8, mother's education, and the interval between the age 8 and 11 examinations.

^cAdjusted for IW-MVPA at age 8, physical maturity at age 11, and family income. Because of missing data for physical maturity (missing $n = 2$) and family income (missing $n = 28$, missing data for both physical maturity and family income in one girl), 155 girls were included in the final model.