# Association between Sleep, Sedentary Time, Physical Activity, and Adiposity in Adolescents: A Prospective Observational Study

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## ABSTRACT

KRACHT, C. L., P. T. KATZMARZYK, C. M. CHAMPAGNE, S. T. BROYLES, D. S. HSIA, R. L. NEWTON JR, and A. E. STAIANO. Association between Sleep, Sedentary Time, Physical Activity, and Adiposity in Adolescents: A Prospective Observational Study. *Med. Sci. Sports Exerc.*, Vol. 55, No. 1, pp. 110–118, 2023. **Purpose:** This study aimed to examine the effects of substituting sedentary time with sleep or physical activity on adiposity in a longitudinal sample of adolescents. **Methods:** Adolescents (10–16 yr) were recruited for a prospective observational cohort. Parents and adolescents reported demographic characteristics and pubertal development. Accelerometry was used to measure sleep, physical activity, and sedentary time. Adiposity was quantified with imaging techniques. Isotemporal substitution modeling was conducted to examine the effect of substituting 10 min of sedentary time with sleep or differing intensities of physical activity. Results were stratified by sex and race and adjusted for covariates. **Results:** A total of 217 adolescents provided complete measures at both baseline and 2 yr later (58.1% White, 51.8% girls; 12.9 ± 1.9 yr at baseline). Sleep was negatively related to adiposity 2 yr later when considering other movement behaviors, but substituting baseline sedentary time with sleep was not related to future adiposity ($P > 0.05$). In boys and non-White adolescents, substituting sedentary time with vigorous-intensity physical activity was related to lower adiposity 2 yr later ($P < 0.05$). Substituting sedentary time for moderate- to vigorous-intensity physical activity was not associated with future adiposity. **Conclusions:** Substituting sedentary time with vigorous-intensity physical activity was related to lower adiposity in later adolescence in certain groups. Opportunities to promote an adequate balance of sleep, sedentary time, and physical activity in all adolescents are encouraged for optimal development. **Key Words:** OBESITY, EXERCISE, CHILDREN, COMPOSITIONAL DATA ANALYSIS

Physical activity is paramount for child health and development, including as a deterrent for excess weight gain in adolescents (1). Time spent sedentary, especially for prolonged periods, is associated with a higher body mass index (BMI) and cardiometabolic risk factors in adolescents (2). Despite the benefits of additional physical activity and less time spent sedentary, many children increase their time spent sedentary at around 7 yr of age while reducing physical activity (3). This pattern continues into later adolescence (3). Another important health behavior, sleep, is also associated with childhood obesity and cardiometabolic risk as children with short sleep duration are more likely to have overweight or obesity

(4). Recognizing the collective influence of these three health behaviors that occur within the 24-h day, Canada released the 24-Hour Movement Guidelines for physical activity, sedentary screen time, and sleep for children and adolescents in 2016 (5). The World Health Organization (WHO) recently built on these efforts by releasing the 2020 WHO guidelines on physical activity and sedentary time for children and adolescents (ages 5–17 yr) (6). The WHO guidelines recommend $\geq 60$ min·d$^{-1}$ in moderate- to vigorous-intensity physical activity (MVPA) and reduction of time spent sedentary. Further, the WHO guidelines suggest the substitution of sedentary time with physical activity for maximal benefit (7).

Although the ideal balance of all movement behaviors is unknown, recent studies have begun using statistical analysis techniques to model the interplay of these behaviors within the 24-h day. Substitution techniques, including compositional data analysis, are used to understand the relationship between time spent in one behavior compared with time spent in another behavior with health outcomes (8). Compositional data analysis includes multiple approaches, including the isometric log ratio approach, whereby movement intensities are represented as ratios of the 24-h day. This approach may be limited by any missing or nonwear data and undetected sleep, and the results can be difficult to translate into recommendations (8). Another technique is the traditional approach, which includes

all movement behaviors as raw values in the same model. In these models, movement behaviors are sequentially added and removed from the models to represent the substitution of one behavior for another. This technique may also be used for comparing reallocation of time (min) for sleep and may be used for interpretating changes among movement behaviors (9). As an example, substituting 30 min of sedentary time with sleep was associated with a lower BMI in a cross-sectional study of 1718 older adults (ages 45–84 yr) (10). Similarly, a recent systematic review of eight studies (seven cross-sectional and one longitudinal) reported that reallocating time spent sedentary with time spent in light physical activity (LPA) and MVPA was associated with lower adiposity in adults (11).

These analytic techniques and explorations are emerging in adolescents because adolescence is a critical period when sedentary time increases and physical activity decreases (12). Within this age-group, there are also potential differences by sex (3) and race (13), with girls and non-White adolescents engaging in more sedentary behavior and less physical activity relative to boys and White adolescents, respectively (3,13). Even so, cross-sectional analyses have found that time spent in vigorous-intensity physical activity (VPA) relative to all other behaviors (sedentary time, LPA, and MVPA) was associated with lower BMI $z$-scores in children and adolescents (6–17 yr) (14), and reallocating sedentary time to LPA and VPA was associated with lower BMI $z$-scores in 782 children (ages 7–13 yr) (15). Furthermore, substituting sedentary time with MVPA was associated with lower visceral adipose tissue (VAT) in 412 children (ages 7–12 yr) (16). A longitudinal study of 88 children (ages $9.2 \pm 0.9$ yr) found that allocating sedentary time to VPA was associated with lower VAT 5 yr later (17). These results provide evidence that reallocating sedentary time with higher-intensity activity is important for adolescent development.

Another consideration is sleep, as others have found allocating time from MVPA to sleep to be related to higher adiposity, but it is unclear if allocating sedentary time to sleep provides any benefit (18). Examining the substitution sleep for sedentary time (14,16) and assessing more than a single time point (14–16) may improve on existing work. Evaluating the effects of substituting sedentary time with all other movement behaviors on various measures of adiposity using longitudinal data may add to understanding if reallocation of movement behaviors is related to the development of excess weight and to finding actionable targets for future behavioral interventions. Therefore, the purpose of this study was to examine the effects of substituting sedentary time with sleep or physical activity on adiposity in a longitudinal sample of adolescents. Based on existing literature, we hypothesized that substituting sedentary time with VPA will be related to lower VAT accumulation.

## METHODS

**Participants.** Adolescents (ages 10–16 yr) were recruited from a metropolitan area in Louisiana between 2016 and 2018 to participate in a prospective observational cohort study, the Translational Investigation of Growth and Everyday Routines in Kids (TIGER Kids) study (NCT02784509). Adolescents were eligible at baseline if they weighed less than 500 lb or 226 kg to not exceed weight capacity of the imaging instruments, were not pregnant, were not on a restrictive diet due to illness, had no significant physical and/or mental disability that interfered with the ability to walk or wear an accelerometer, and were able to comprehend and complete all study procedures. The primary aim of the TIGER Kids study was to examine the relationship between changes in physical activity and sedentary time with fat accumulation during childhood and adolescence and explore differences by race (White vs non-White adolescents). Therefore, the target sample size was at least 334 participants to detect a 24-min difference in sedentary time between adolescents with and without obesity ($\alpha = 0.05$, 80% power) and allow for a dropout rate of 25%, based on a previous study (19). The study team aimed to recruit 340 adolescents at baseline to assess this primary research question.

Recruitment occurred through flyers, e-mail listservs, social media (e.g., Facebook) advertisements, local news outlets, health events, outreach at local schools, and other community events. Follow-up measures occurred during August 2018 and August 2020, approximately 2 yr after baseline measures (range, 18–30 months). Retention in the study was facilitated by communicating with the parent or guardian semiannually over e-mail or phone, which included reviewing health and weight status changes in the participating adolescent and scheduling the follow-up visit.

**Procedure.** Informed assent of the adolescent and written consent of the parent were obtained before undertaking any measurements. At an orientation event, adolescents were instructed to wear an accelerometer and return for a clinic visit after at least 7 d. Adolescents were asked to arrive at the clinic visit in a fasted state. At this same visit, the parent completed a demographics survey, which included adolescent age, sex, race, marital status, and household income. Adolescents also reported their stage of sexual maturation based on standardized validated drawings that showed advanced pubertal stages from incomplete development to complete development for male or female bodies, depending on the adolescent's sex (20). Adolescents completed one dietary recall using the Automated Self-Administered 24-Hour Dietary Assessment Tool (ASA-24 2016) (21) at the clinic visit and up to two additional dietary recalls at home as described elsewhere (22). All questionnaires were completed using a secure data capture software, Research Electronic Data Capture (REDCap), hosted at Pennington Biomedical Research Center (23).

A trained study staff member measured height and weight twice according to clinic protocol and averaged these measurements. If the measurements differed by more than 0.5 units, then a third measurement was taken. BMI was calculated by dividing weight (in kilograms) by squared height (in meters). BMI was compared with age- and sex-specific percentiles using the CDC SAS Macro program (24). Obesity was defined as having a BMI ≥95th percentile, overweight was defined as a BMI ≥85th to 95th percentile, and underweight was defined as

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a BMI ≤5th percentile for age and sex (25). All clinical procedures from the initial clinic visit were repeated during the follow-up clinic visit. The study protocol was approved by the Pennington Biomedical Research Center's Institutional Review Board.

**Sleep, sedentary time, and physical activity.** Sleep, sedentary time, and physical activity were measured using accelerometry. An ActiGraph GT3X+ accelerometer (Pensacola, FL) was placed on the right hip of the adolescent with an elastic waistband, and data were recorded in 15-s epochs. The adolescent was instructed to wear the device continuously (including overnight) for at least 7 d and only remove the device for water-based activities (e.g., bathing). A previously published algorithm, developed using a pediatric cohort, was used to differentiate between sleep, nonwear, and wear time (26). Age-appropriate cut points for 15-s epochs were applied, including ≤25 counts for sedentary time, 26–573 counts for LPA, 574–1003 counts for moderate-intensity physical activity (MPA), and ≥1003 counts for VPA (27). MPA and VPA were considered separately, and together as a sum in analysis (as MVPA), because of the unique relationship between VPA and adiposity (28). Adolescents who had at least 4 d (including one weekend day) with 10 h of wear time (excluding nonwear or sleep) at baseline were included in the analysis (29). Sleep time was the amount between algorithm-determined bedtime and wake time. Adolescents with at least 3 d (with one weekend day) with ≥160 min of overnight sleep were included in the analysis based on a previously published algorithm (26). The algorithm using 2 d was validated in a free-living environment against self-report logs in children (30). Sleep, sedentary time, physical activity, and wear time (min) were averaged over valid days available (min·d$^{-1}$).

**Adiposity.** A trained technician performed a whole-body scan using a General Electric (GE) Lunar iDXA scanner (GE Medical Systems, Milwaukee, WI) and a standard positioning protocol. The amount of body fat mass and lean mass was estimated (kg). Adolescents who exceeded the scan region were positioned with their left arm excluded, and their right arm measurements were duplicated for analysis. A water-fat shifting MRI scan was used to assess VAT and subcutaneous adipose tissue (SAT) with a GE Discovery 750w 3.0 Tesla (GE Medical Systems). Images were captured from the highest point of the liver to the bottom of the right kidney during a 20-s breath hold and processed using IDEAL-IQ imaging software. A trained technician manually drew VAT and SAT areas, and additional calculations were used to estimate volume (kg) (31). Total abdominal adipose tissue (TAT) was the sum of VAT (kg) and SAT (kg).

**Statistical analysis.** Adolescents with complete data for demographics and accelerometry at baseline and adiposity at both time points were included in the analysis. Fat mass, VAT, SAT, and TAT values were reported for the entire sample but were log-transformed for analysis because of nonnormal distribution. Pearson correlation coefficients were used to examine associations among baseline movement behaviors, and between baseline movement behaviors and adiposity at both time points. A series of models were conducted to examine the relationship between movement behavior (baseline) and adiposity (follow-up), including singular, partition, and isotemporal substitution models. For singular models, serial independent linear regression models were used to examine the association between baseline movement behaviors (one independent variable per model) and follow-up adiposity (dependent variables). Four models, one for each dependent variable, were run as for the partition component and then for isotemporal substitution component. For partition models, linear regression models were used to examine the association between baseline movement behaviors (all five independent variables together in the model [sedentary time, LPA, MPA, VPA, and sleep]) and follow-up adiposity (dependent variables). For isotemporal substitution models, movement behaviors were summed to create a total time variable (sum of sedentary time, LPA, MPA, VPA, and sleep), and individual movement behaviors were also divided by 10 to create 10-min increments like other studies with low amounts of VPA relative to MPA and LPA (15,17,32). To evaluate the association of substituting 10 min of sedentary time, all other movement behaviors (LPA, MPA, VPA, and sleep represented as 10-min increments) were included as independent variables, total time was included as a covariate, and follow-up adiposity was the dependent variable.

Models were adjusted for baseline age, sex, race, puberty status, baseline adiposity, time between visits (months), and baseline lean mass in the models with fat mass as dependent variable, along with time between visits (months). Models were repeated with adjustment for wear time, and with MVPA as the independent variable instead of MPA and VPA. A sensitivity analysis was conducted with singular, partition, and isotemporal substitution models with additional adjustment for baseline dietary quality via the average daily Healthy Eating Index 2015 score calculated from available dietary recalls (1–3 d possible) (33). In addition, interactions between sex and movement behaviors, along with race and movement behaviors, were explored in singular models. Levene's test was conducted to examine if sex and race groups should be modeled together or separately. Significantly different variances were found in VAT among sexes ($P < 0.05$), and fat mass had differences that were trending. Similar results were found when repeated for race. Thus, model that considered race and genders were separated by group levels. Singular, partition, and isotemporal substitution models were then repeated separately by sex. The stratified samples by sex and race were moderately powered (power-level 70% for 91 adolescents and 80%–89% for 104–126 adolescents) to detect an effect at 0.05 significance level assuming a moderate effect size (i.e., Cohen's $f^2 = 0.15$; calculated using G*Power Version 3.1). Variance inflation was examined in each model because of possible collinearity with movement behaviors, although no variance inflation was found (<10 variance inflation factor per variable). Up to five outliers were removed from each model because of departure from normality in the residuals. All analyses were conducted in SAS 9.4 (Cary, NC), and

significance was set at $P < 0.05$ for main effects and $P < 0.10$ for interaction effects.

## RESULTS

Three hundred and forty-two adolescents completed a TIGER Kids baseline visit, and 286 completed all baseline measures for the present analysis (45 had incomplete accelerometry data, 10 were missing MRI data, and 1 was missing puberty data). Of these, 222 adolescents participated in the follow-up TIGER Kids visit, and 217 provided complete measures. Those who were included had higher MPA ($25.2 \pm 12.4$ min) and VPA ($10.1 \pm 9.7$ min) and wore the accelerometer longer ($868.0 \pm 56.4$ min) at baseline compared with those not included (MPA, $21.9 \pm 10.1$ min, $P = 0.01$; VPA, $7.2 \pm 7.0$ min, $P = 0.003$; wear time, $847.2 \pm 70.2$ min, $P = 0.01$). Those who were included had lower fat mass and lean mass at baseline and lower values in all adiposity measures at follow-up ($P < 0.05$ for all). No other differences in demographics, independent variables, or dependent variables were found between those included and not included in analysis.

As shown in Table 1, adolescents were $12.9 \pm 1.9$ yr of age, over half were White (58.1%), about one-third were African American (36.4%), and few identified as another race (5.5%). The sample's racial composition is similar to the U.S. census data for the Baton Rouge metropolitan area (56% White, 36% African American) (34). Almost half the sample had overweight (12.2%) or obesity (30.9%). There was a significant increase in BMI percentile and all adiposity measures between baseline and follow-up ($P < 0.01$ for all). There was a difference in sedentary time, MPA, VPA, and

multiple adiposity measures at baseline and follow-up between boys and girls (Supplemental Digital Content, Table S1, Descriptive characteristics of sample stratified by sex and race, http://links.lww.com/MSS/C699). White and non-White adolescents differed in baseline LPA, sleep, and multiple adiposity measures at baseline and follow-up (Supplemental Digital Content, Table S1, Descriptive characteristics of sample stratified by sex and race, http://links.lww.com/MSS/C699).

In unadjusted models (Supplemental Digital Content, Table S2, Unadjusted correlations between baseline movement behaviors and adiposity, http://links.lww.com/MSS/C699), baseline sedentary time was negatively related to baseline LPA, MPA, VPA, and sleep ($P < 0.001$). LPA was positively correlated with MPA, VPA, and negatively correlated with sleep ($P < 0.05$), and MPA was positively correlated with VPA ($r = 0.77$, $P = 0.001$). Baseline MPA, VPA, and sleep were negatively related to all measures of adipose and lean mass at both time points ($P < 0.05$), except for follow-up MPA and lean mass ($P = 0.62$). Baseline sedentary time was also positively related to all measures of adipose and lean mass at both time points ($P < 0.05$) and related to less change in lean mass ($r = -0.33$, $P = 0.001$). LPA was negatively related to VAT at both time points, with change in VAT, and with lean mass at baseline ($P < 0.05$). LPA, MPA, and VPA were each related to a larger change in lean mass in separate models ($P < 0.05$).

**Full sample.** In singular models, baseline sedentary time, LPA, MPA, VPA, or sleep were not related to follow-up fat mass, VAT, SAT, or TAT ($P > 0.05$, Supplemental Digital Content, Table S3, Singular models for associations among baseline movement behaviors and adiposity at follow-up, http://links.lww.com/MSS/C699). Partition and isotemporal

TABLE 1. Descriptive characteristics of sample ($n = 217$).

| | Baseline | | Follow-Up | | Difference | |
|---|---|---|---|---|---|---|
| | Mean ± SD | Pct. | Mean ± SD | Pct. | Mean ± SD | P |
| Age | 12.9 ± 1.9 | | 14.8 ± 2.0 | | | |
| Male | | 48.2 | | | | |
| Race | | | | | | |
| White | | 58.1 | | | | |
| African American | | 36.4 | | | | |
| Other | | 5.5 | | | | |
| Puberty | | | | | | |
| Prepuberty | | 12.4 | | | | |
| Peripuberty | | 52.1 | | | | |
| Postpuberty | | 35.5 | | | | |
| Movement behaviors | | | | | | |
| Sedentary time (min·d⁻¹) | 591.2 ± 72.2 | | | | | |
| LPA (min·d⁻¹) | 241.2 ± 57.6 | | | | | |
| MPA (min·d⁻¹) | 25.2 ± 12.5 | | | | | |
| VPA (min·d⁻¹) | 10.1 ± 9.7 | | | | | |
| Sleep (h·d⁻¹) | 8.7 ± 1.0 | | | | | |
| Weight status and adiposity | | | | | | |
| BMI percentile | 68.5 ± 31.5 | | 71.2 ± 29.6 | | 3.2 ± 15.0 | 0.003* |
| BMI category | | | | | | 0.001* |
| Underweight | | 3.6 | | 2.5 | | |
| Normal | | 53.4 | | 51.5 | | |
| Overweight | | 12.2 | | 14.5 | | |
| Obesity | | 30.9 | | 31.5 | | |
| VAT (kg) | 0.5 ± 0.4 | | 0.6 ± 0.5 | | 0.1 ± 0.2 | 0.001* |
| SAT (kg) | 5.0 ± 4.6 | | 5.8 ± 5.3 | | 0.8 ± 1.7 | 0.001* |
| TAT (kg) | 5.5 ± 5.0 | | 6.4 ± 5.8 | | 0.9 ± 1.8 | 0.001* |
| Lean mass (kg) | 36.7 ± 10.3 | | 43.3 ± 10.3 | | 6.6 ± 5.2 | 0.001* |
| Fat mass (kg) | 20.8 ± 13.9 | | 24.0 ± 16.2 | | 3.2 ± 5.9 | 0.001* |

VAT, SAT, TAT, lean mass, and fat mass are presented without transformation. Assessed using paired $t$-tests and chi-square analysis; *$P < 0.05$.

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TABLE 2. Partition and isotemporal substitution models for associations among baseline movement behaviors and adiposity at follow-up (n = 217).

| | Fat Mass (kg) | | VAT (kg) | | SAT (kg) | | TAT (kg) | |
|---|---|---|---|---|---|---|---|---|
| | β (95% CI) | P | β (95% CI) | P | β (95% CI) | P | β (95% CI) | P |
| Partition models[a] | | | | | | | | |
| Sedentary time | −0.00007 (−0.0007 to 0.00006) | 0.83 | −0.001 (−0.002 to 0.0003) | 0.13 | −0.0002 (−0.001 to 0.0006) | 0.57 | −0.0003 (−0.001 to 0.0005) | 0.42 |
| LPA | −0.0006 (−0.001 to 0.0002) | 0.16 | −0.0007 (−0.002 to 0.0007) | 0.28 | −0.0006 (−0.001 to 0.0004) | 0.24 | −0.0007 (−0.001 to 0.0004) | 0.21 |
| MPA | 0.002 (−0.002 to 0.007) | 0.32 | 0.0004 (−0.008 to 0.008) | 0.98 | −0.0001 (−0.007 to 0.004) | 0.69 | −0.001 (−0.007 to 0.004) | 0.63 |
| VPA | −0.002 (−0.007 to 0.002) | 0.24 | −0.006 (−0.01 to 0.002) | 0.15 | −0.001 (−0.005 to 0.008) | 0.66 | −0.001 (−0.005 to 0.009) | 0.68 |
| Sleep | −0.0005 (−0.001 to 0.0007) | 0.08 | −0.0006 (−0.001 to 0.0004) | 0.23 | −0.001 (−0.001 to −0.0004) | 0.03* | −0.0008 (−0.001 to −0.0002) | 0.04* |
| Isotemporal substitution models[b] | | | | | | | | |
| Sedentary time | Dropped | | Dropped | | Dropped | | Dropped | |
| LPA | −0.004 (−0.01 to 0.003) | 0.24 | 0.001 (−0.01 to 0.01) | 0.85 | −0.003 (−0.01 to 0.005) | 0.40 | −0.003 (−0.01 to 0.005) | 0.45 |
| MPA | −0.02 (−0.02 to 0.06) | 0.35 | 0.01 (−0.07 to 0.09) | 0.79 | −0.007 (−0.06 to 0.04) | 0.77 | −0.009 (−0.06 to 0.04) | 0.73 |
| VPA | −0.03 (−0.08 to 0.02) | 0.25 | −0.05 (−0.15 to 0.03) | 0.23 | 0.005 (−0.05 to 0.106) | 0.86 | 0.005 (−0.06 to 0.17) | 0.57 |
| Sleep | −0.004 (−0.01 to 0.001) | 0.16 | 0.003 (−0.008 to 0.01) | 0.57 | −0.004 (−0.01 to 0.002) | 0.18 | −0.003 (−0.001 to 0.003) | 0.30 |

Fat mass, VAT, SAT, and TAT values were log-transformed due to nonnormal distribution.
*P < 0.05.
[a]Assessed using linear regression with all movement behaviors included, and adjustment for baseline age, sex, race, puberty status, adiposity and lean mass for fat model, and time between visits (months).
[b]Assessed using the linear regression with sedentary time removed, and adjustment for the same covariates as the partition models along with total time in behaviors (sum of sedentary time, LPA, moderate physical activity, vigorous physical activity, and sleep) at baseline.

substitution models with the full sample are presented in Table 2. In partition models, baseline sleep was negatively associated with follow-up SAT and TAT after accounting for other movement behaviors (P < 0.05). Baseline movement behaviors explained 23% of the variance of follow-up SAT and TAT. In isotemporal substitution models, there were no significant associations between the substitution of 10 min of movement behaviors with sedentary time and adiposity at follow-up (P > 0.05). Singular, partition, and isotemporal substitution results were unchanged when MVPA replaced MPA and VPA in models or adjusting for accelerometer wear time, or when adjusting for Healthy Eating Index total score at baseline in sensitivity analysis (n = 202).

**Stratification by sex.** In singular models, there was a significant interaction between LPA and sex for fat mass (P = 0.05) and between VPA and sex for fat mass (P = 0.06) and VAT (P = 0.04). Singular and partition model results for girls and boys are shown in Tables S4 and S5 (Supplemental Digital Content, Singular, partition, and isotemporal substitution models for associations among baseline movement behaviors and adiposity at follow-up in girls [Table S4] and in boys [Table S5], http://links.lww.com/MSS/C699), respectively.

Isotemporal substitution model results stratified by sex are presented in Table 3.

In models including only girls (n = 113), baseline VPA was negatively associated with follow-up VAT (P = 0.04). Baseline VPA explained 13% of the variance in follow-up VAT. There were no other significant relationships between movement behaviors and adiposity in singular (P > 0.05), partition (P > 0.05), or isotemporal substitution models (P > 0.05). In girls, age was negatively related to follow-up adiposity in singular models and baseline adiposity was positively related to follow-up adiposity in all models (P < 0.05). In singular models including only boys (n = 104), baseline VPA was negatively associated with follow-up VAT (P = 0.01). Baseline sleep was negatively associated with follow-up fat mass (P = 0.02), SAT (P = 0.01), and TAT (P = 0.01). Baseline VPA explained 15% of the variance of follow-up VAT, and baseline sleep account for 6% to 7% of the variance of follow-up fat mass, SAT, and TAT. In boys' partition models, baseline LPA (P = 0.03) and sleep (P = 0.01) were negatively associated with follow-up fat mass, although together only explained 7% of the variance. Baseline VPA was negatively associated with follow-up VAT (P = 0.01), and baseline sleep

TABLE 3. Isotemporal substitution models for adjusted associations among baseline movement behaviors and adiposity at follow-up stratified by sex (n = 217).

| | Fat Mass (kg) | | VAT (kg) | | SAT (kg) | | TAT (kg) | |
|---|---|---|---|---|---|---|---|---|
| | β (95% CI) | P | β (95% CI) | P | β (95% CI) | P | β (95% CI) | P |
| Girls (n = 113) | | | | | | | | |
| Sedentary time | Dropped | | Dropped | | Dropped | | Dropped | |
| LPA | −0.004 (−0.01 to 0.003) | 0.25 | 0.004 (−0.01 to 0.02) | 0.66 | −0.005 (−0.01 to 0.003) | 0.21 | −0.003 (−0.01 to 0.005) | 0.42 |
| MPA | 0.01 (−0.02 to 0.05) | 0.50 | −0.03 (−0.13 to 0.08) | 0.66 | −0.01 (−0.06 to 0.04) | 0.69 | −0.01 (−0.07 to 0.04) | 0.57 |
| VPA | −0.02 (−0.08 to 0.02) | 0.29 | 0.04 (−0.10 to 0.18) | 0.58 | 0.007 (−0.05 to 0.07) | 0.80 | 0.006 (−0.05 to 0.07) | 0.85 |
| Sleep | −0.003 (−0.009 to 0.002) | 0.26 | 0.007 (−0.006 to 0.02) | 0.27 | −0.004 (−0.02 to 0.002) | 0.18 | −0.003 (−0.01 to 0.003) | 0.35 |
| Boys (n = 104) | | | | | | | | |
| Sedentary time | Dropped | | Dropped | | Dropped | | Dropped | |
| LPA | −0.01 (−0.02 to 0.002) | 0.12 | −0.006 (−0.03 to 0.01) | 0.54 | 0.01 (0.001 to 0.02) | 0.02* | −0.005 (−0.02 to 0.01) | 0.54 |
| MPA | 0.06 (−0.02 to 0.14) | 0.15 | 0.09 (−0.04 to 0.22) | 0.15 | −0.10 (−0.17 to −0.03) | 0.03* | 0.03 (−0.07 to 0.13) | 0.56 |
| VPA | −0.07 (−0.17 to 0.01) | 0.09 | −0.17 (−0.31 to −0.03) | 0.01* | 0.01 (−0.05 to 0.09) | 0.63 | −0.07 (−0.19 to 0.04) | 0.19 |
| Sleep | −0.007 (−0.01 to 0.004) | 0.21 | −0.002 (−0.02 to 0.01) | 0.79 | −0.01 (−0.02 to 0.004) | 0.01* | −0.01 (−0.02 to 0.002) | 0.09 |

Fat mass, VAT, SAT, and TAT values were log-transformed due to nonnormal distribution.
*P < 0.05.
[a]Assessed using linear regression with all movement behaviors included except sedentary time, adjustment for baseline age, sex, race, puberty status, adiposity, lean mass in fat mass models, time between visits (months), and total time in behaviors (sum of sedentary time, LPA, moderate physical activity, vigorous physical activity, and sleep).

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was also negatively associated with follow-up SAT ($P = 0.04$) and TAT ($P = 0.01$) in partition models. In boys' isotemporal substitution models, the substitution of 10 min of sedentary time for 10 min of VPA at baseline was negatively associated with follow-up VAT ($P = 0.01$). Substituting 10 min of sedentary time for MPA or sleep at baseline was associated with lower follow-up SAT, whereas substituting 10 min of sedentary time for LPA was associated with higher follow-up SAT ($P < 0.05$, Table 3).

When MVPA was used instead of MPA and VPA separately, sleep was negatively related to SAT in partition models for girls ($P = 0.01$). For boys, baseline LPA and sleep were no longer associated with follow-up SAT in isotemporal substitution models, but MVPA was not associated with follow-up SAT ($P > 0.05$). There were no other significant relationships when using MVPA rather than MPA and VPA.

**Stratification by race.** There were no significant interactions between movement behaviors and race with adiposity measures ($P > 0.10$), even when including MVPA rather than MPA or VPA. This investigation was established *a priori* and pursued. Singular and partition model results for White and non-White adolescents are shown in Tables S6 and S7 (Supplemental Digital Content, Singular, partition, and isotemporal substitution models for associations among baseline movement behaviors and adiposity at follow-up in White adolescents [Table S6] and in non-White adolescents [Table S7], http://links.lww.com/MSS/C699), respectively. Results of isotemporal substitution models stratified by race are presented in Table 4.

In models including only White adolescents ($n = 126$), there were no significant relationships between movement behaviors and adiposity in singular ($P > 0.05$), partition ($P > 0.05$), or isotemporal substitution models ($P > 0.05$, Table 4). Results did not change when including MVPA rather than MPA and VPA. There were no significant associations in singular models including non-White adolescents ($n = 104$). In partition models with non-White adolescents, baseline VPA and sleep were negatively associated with follow-up fat mass, and MPA was positively associated with follow-up fat mass ($P < 0.05$). Baseline VPA was also negatively related to follow-up VAT ($P = 0.02$). Baseline sleep was negatively associated with follow-up SAT

and with TAT ($P = 0.01$ for both). As for isotemporal substitution models, substituting 10 min of sedentary time with 10 min of VPA was associated with lower fat mass ($P = 0.04$) and VAT at follow-up ($P = 0.03$, Table 4). Substituting 10 min of sedentary time with MPA was associated with higher fat mass at follow-up ($P = 0.01$). When including MVPA rather than MPA and VPA, there were no significant associations in partition models or isotemporal substitution models with non-White adolescents.

## DISCUSSION

The purpose of this study was to examine the longitudinal association between movement behaviors and adiposity in adolescents. Sleep was negatively related to adiposity when considering other movement behaviors. After evaluating by race and sex by using four different models, VPA was related to less VAT and sleep was related to less SAT in boys and non-White adolescents independently when evaluated as a substitution for sedentary behavior. There were no significant relationships between MVPA and adiposity. Taken together, efforts to encourage VPA and sleep in adolescents may promote healthy development and deter excess adipose development.

In this sample, sleep was associated with various markers of abdominal fat 2 yr later, but there were no associations between substituting sedentary time with sleep. Most posit the relationship between sleep and adiposity to be linear, whereby increasing sleep is related to lower adiposity, but this relationship is predominately based on examination of short sleep in adolescents as longitudinal and experimental studies of long-sleep and adiposity in this age range are limited (35,36). As for other isotemporal substitution studies, a cross-sectional study in adults found that substituting 30 min of sedentary time for sleep was associated with lower BMI (10), which was not replicated in the current study when sleep was evaluated as substituting of sedentary time. The adolescent isotemporal substitution literature is mixed, with Dumuid et al. (2018) finding an association between longer sleep days and lower adiposity composite score (including body fat percentage, BMI *z*-score, and waist-to-height ratio) (37) and Verswijveren

TABLE 4. Isotemporal substitution models for adjusted associations among baseline movement behaviors and adiposity at follow-up stratified by race ($n = 217$).

| | Fat Mass (kg) | | VAT (kg) | | SAT (kg) | | TAT (kg) | |
|---|---|---|---|---|---|---|---|---|
| | β (95% CI) | P | β (95% CI) | P | β (95% CI) | P | β (95% CI) | P |
| White ($n = 126$) | | | | | | | | |
| Sedentary time | Dropped | | Dropped | | Dropped | | | |
| LPA | −0.001 (−0.01 to 0.08) | 0.81 | −0.004 (−0.02 to 0.01) | 0.64 | 0.003 (−0.009 to 0.01) | 0.58 | 0.002 (−0.01 to 0.01) | 0.69 |
| MPA | 0.003 (−0.05 to 0.06) | 0.90 | −0.03 (−0.15 to 0.08) | 0.56 | −0.03 (−0.11 to 0.04) | 0.38 | −0.03 (−0.11 to 0.04) | 0.33 |
| VPA | −0.01 (−0.08 to 0.05) | 0.65 | −0.01 (−0.15 to 0.12) | 0.84 | −0.01 (−0.09 to 0.07) | 0.80 | −0.004 (−0.09 to 0.09) | 0.91 |
| Sleep | −0.002 (−0.01 to 0.005) | 0.54 | −0.001 (−0.01 to 0.01) | 0.89 | −0.005 (−0.01 to 0.004) | 0.24 | −0.004 (−0.01 to 0.005) | 0.34 |
| Non-White ($n = 91$) | | | | | | | | |
| Sedentary time | Dropped | | Dropped | | Dropped | | Dropped | |
| LPA | −0.007 (−0.02 to 0.006) | 0.28 | 0.002 (−0.01 to 0.02) | 0.83 | −0.01 (−0.02 to 0.004) | 0.17 | −0.009 (−0.02 to 0.005) | 0.21 |
| MPA | 0.09 (0.009 to 0.18) | 0.03* | 0.10 (−0.02 to 0.23) | 0.11 | 0.04 (−0.04 to 0.14) | 0.32 | 0.04 (−0.04 to 0.14) | 0.29 |
| VPA | −0.12 (−0.23 to −0.02) | 0.01* | −0.15 (−0.30 to −0.01) | 0.03* | −0.05 (−0.16 to 0.06) | 0.38 | −0.05 (−0.17 to 0.05) | 0.31 |
| Sleep | −0.005 (−0.01 to 0.006) | 0.34 | 0.009 (−0.006 to 0.02) | 0.23 | −0.003 (−0.01 to 0.008) | 0.52 | −0.002 (−0.01 to 0.009) | 0.65 |

Fat mass, VAT, SAT, and TAT values were log-transformed due to nonnormal distribution.
* $P < 0.05$.
[a]Assessed using linear regression with all movement behaviors included except sedentary time, adjustment for baseline age, sex, puberty status, adiposity, lean mass in fat mass models, time between visits (months), and total time in behaviors (sum of sedentary time, LPA, moderate physical activity, vigorous physical activity, and sleep).

et al. (2021) finding no association between sleep and BMI or waist circumference (15). Yet, a recent longitudinal study of 137 children and adolescents (8–17 yr) reported that characteristics of sleep (e.g., wake time and sleep midpoint) were associated with percent fat mass 1 yr later but not associated with sleep duration (38). The differences in results may reflect the current study's use of abdominal adipose tissue compared with anthropometry and longer follow-up time (2 yr) compared with others (1 yr) (38). These differences in design allow for more specific assessment of adiposity and additional time for adipose tissue to accumulate, assuming these movement behavior patterns continued over 2 yr. Still, the exact mechanism of sleep and SAT accumulation is unclear. It may be that substituting sleep for sedentary time would result in a reduction in nighttime food consumption and screen time, although detailed assessment and study designs in children are still limited for this precise mechanism (39). A separate longitudinal analysis of the TIGER Kids cohort found those who watched multiple screens at night had less sleep and higher adiposity compared with those who watched no screens at night, but this relationship was attenuated when accounting for demographic factors such as age (40). A cross-sectional study of 357 White and Black adolescents found that those who met more 24-Hour Movement Guidelines had lower SAT (41). This finding suggests that those who have additional sleep may also have an adequate balance of physical activity and sedentary time that may promote less SAT development, which would align with partition models that account for other movement behaviors but not necessarily substituting for sedentary time.

In the isotemporal substitution models, substituting sedentary time with VPA was associated with lower fat mass and VAT as hypothesized, but only in boys and non-White adolescents independently but not in the full sample. The association between VPA and adiposity aligns with a review that found among 13 articles that VPA was consistently associated with lower body fat (28). Comparable results were found in a pooled meta-analysis of device-based measures whereby VPA was negatively associated with overall adiposity (42). Authors did note that about half the amount of VPA was needed compared with MPA for body composition benefit (28). However, there were no associations between MPA and adiposity in this sample, whether evaluated by itself, with other movement behaviors, or as a substitute for sedentary time. The current study's results suggest a benefit of higher-intensity activity with less adipose growth, especially among boys and non-White adolescents. The current report focused on substitutions of 10 min of sedentary time with VPA, although some adolescents still failed to this threshold and obtained little MPA throughout the day. Programs focused on replacing sedentary time with VPA in these groups, and adolescents in general, may benefit for attenuation of excess fat gain.

Another consideration is that those who can obtain VPA-level intensity during their activities may have lower adiposity already. The current analysis adjusted for baseline levels of adiposity, but there may still be larger barriers to obtaining VPA (e.g., sports opportunities), especially among girls and as they age into adolescence (43). Indeed, VPA was associated with lower fat in singular models, but there were no other associations between movement behaviors and adiposity in girls within this sample. This lack of relationship may be due to the low levels of MPA ($22.3 \pm 10.6$ min·d$^{-1}$) and VPA ($7.9 \pm 7.9$ min·d$^{-1}$) within this group overall, which may be expected as MVPA declines during adolescence, especially in girls (3). There were also no associations between movement behaviors and adiposity in White adolescents, in any model representation, although others have found White adolescents have higher VAT relative to non-White adolescents (44). Potential explanations all center around shifts between baseline and follow-up, including few changes in VAT, lean mass and fat mass changing proportionally, and changes in movement behavior patterns.

Another interesting finding was that there were no associations among MVPA and adiposity. This finding may be due to the low amounts of MPA ($25.2 \pm 12.5$ min·d$^{-1}$) and VPA ($10.1 \pm 9.7$ min·d$^{-1}$) within this sample, which is around half of the recommended amount per day (60 min MVPA·d$^{-1}$) (6). MPA and VPA were captured using device-based measures and represent differing ranges of metabolic equivalent of tasks (3.0–6.0 METs or METy for the youth equivalent for MPA, and >6.0 METs or METy for VPA) (45). It is unclear what MET level was achieved in this study, as accelerometer counts are divided into large categories of activity. Obtaining MPA or VPA may differ within this age range (10–16 yr) as technical expertise in structured movements and weight both increase into adolescence (45). For example, playing baseball with the whole body using active video games is around 4.7 METy for ages 10–12 yr but is 5.7 METy for ages 13–15 yr and could become VPA (45). Overall, the authors caution against an interpretation that examining total MVPA is not valuable, as there are many who report otherwise (6,46). The current study's results suggest that the amount of MVPA achieved in this sample was not related to future adiposity, although MVPA was low and may be due to individual factors. Supplementary measures, such as a self-report questionnaire of activities, may better characterize activities of MVPA within this age range and provide insight into the physical activity level achieved.

The current study had multiple strengths, including a prospective study design during a critical time in growth (i.e., adolescence); use of device-based measures for movement behaviors, along with imaging techniques for assessment of body fat and abdominal adipose tissue; and an advanced statistical technique for analysis (isotemporal substitution analysis). Further, this sample comprised 41.9% non-White adolescents, and 30.9% had obesity, allowing for assessment among a diverse cohort and representing differing levels of adiposity. This study still had limitations. This study did return around 75% of the baseline sample, but there were still many missing critical components for analysis (e.g., 45 adolescents did not meet accelerometry wear time thresholds at baseline). This study did use a device-based measure for activity levels but cannot determine the exact activity performed while sedentary

(e.g., reading, sitting, and watching TV, etc.), limiting the assessment of specific activities. This limitation precludes the current study from making explicit recommendations for adherence to the 24-Hour Movement Guidelines, which specify sedentary screen time rather than overall sedentary time. Results from baseline measures of the TIGER Kids study found that meeting the physical activity guideline was associated with lower BMI percentile, adiposity, and individual and total cardiometabolic risk scores (22). Additional investigation into adherence to the 24-Hour Movement Guidelines (including sedentary screen time) in a longitudinal manner may better address the effect of meeting these public health metrics. This study also had smaller sample sizes for subgroup analysis (range, 91–126 adolescents), which were moderately powered. Additional participants may increase the power of these analysis. Because of the log-transformation of adipose measures, we cannot deduce the exact trade-off from substituting behaviors or if these changes are clinically meaningful. The isotemporal substitution techniques also limit the ability to create effect sizes, and the log-transformation of values does limit the interpretation of results. The low amount of MPA and VPA obtained in this sample may limit the interpretation of substitution for another movement behavior and the result on subsequent adiposity. Further, this study conducted multiple analysis and may detect some false-positives from these additional analyses. A traditional approach, as in the sequential addition and subtraction of behaviors, was chosen to address the use of sleep, to allow for the incorporation of longitudinal data, and to improve translation of results, although other compositional techniques are possible (32). Finally, additional considerations of movement behaviors and dietary habits changing from baseline to follow-up were not included in analysis but are important contributors to adipose development.

Still, the current study recommends three specific future directions for upcoming research. First, it is critical to design opportunities at home and at school for all adolescents to obtain physical activity, including VPA. The current study assessed an overall change of 10 min·d$^{-1}$, not necessarily 10-min bouts. The recent *Physical Activity Guidelines for Americans, 2nd Edition*, recommend any physical activity interspersed throughout the day, even 1 min, as brief active transport or breaks during the day is beneficial compared with the previous recommendation of bouts of 10 min (47). Within this future direction,

special attention should be paid to girls within this age range as disparities emerge even before adolescence (3). Support throughout childhood and adolescence for girls to be physically active is needed to reduce this gap. Finally, additional attention should be paid to sleep metrics beyond duration. As described with physical activity intensity, other contextual metrics should be considered, which for sleep may include social jetlag (i.e., shift from school night to weekend bedtime), sleep efficiency, and sleep hygiene as explored in other studies in this age range (38,48). The analytic sample included those adolescents who had accelerometry data for at least one weekend day; however, the analysis may be influenced by the differing composition of weekend and weekdays included (e.g., sleep data from some adolescents may be from all weekend days whereas others may have two weekdays and one weekend day). The variability and quality of sleep should be considered in adipose formation and considered as research continues to untangle the sleep and obesity connection.

## CONCLUSIONS

Overall, this study improved on existing isotemporal substitution literature in adolescence using longitudinal data, inclusion of sleep, and various indices of adiposity. Sleep was associated with lower adiposity 2 yr later and when used in place of sedentary time for some groups. Substituting sedentary time for VPA was related to lower adiposity 2 yr later, but only in boys and non-White adolescents. Opportunities to promote an adequate balance of sleep, sedentary time, and all levels of physical activity intensity are encouraged within this age range for optimal development and reducing risk of future chronic disease.

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