

Increased Physical Activity and Reduced Adiposity in Overweight Hispanic Adolescents

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ABSTRACT

BYRD-WILLIAMS, C. E., B. R. BELCHER, D. SPRUIJT-METZ, J. N. DAVIS, E. E. VENTURA, L. KELLY, K. BERHANE, S. AZEN, and M. I. GORAN. Increased Physical Activity and Reduced Adiposity in Overweight Hispanic Adolescents. *Med. Sci. Sports Exerc.*, Vol. 42, No. 3, pp. 478–484, 2010. **Purpose:** The objectives of this study were to examine 1) whether changes in total physical activity (PA; counts per minute, cpm) and time spent in moderate to vigorous PA (MVPA) are associated with changes in adiposity and 2) whether energy intake influences the relationship between changes in PA and changes in adiposity in overweight Hispanic adolescents. **Methods:** Analysis included 38 overweight (body mass index, >85th percentile) Hispanic adolescents with complete pretest and posttest data on relevant variables after participating in a 16-wk intervention. The intervention treatment did not influence PA, so the sample was combined and the randomization group was adjusted for in the analysis. Body composition by dual-energy x-ray absorptiometry, 7-d PA by accelerometry, and dietary intake by 3-d diet records were assessed before and after intervention. **Results:** Within individuals, the mean increase of PA ($n = 19$) and mean decrease of PA ($n = 19$) was approximately 105 cpm. A 100-cpm increase in total PA was associated with a decrease of 1.3 kg of fat mass and 0.8% body fat after adjusting for pretest adiposity, PA, age, sex, and treatment ($P < 0.05$). Controlling for energy intake modestly strengthened the relationships between total PA and fat mass and percent body fat. Changes in MVPA were not related to changes in adiposity after controlling for total PA ($P > 0.05$). **Conclusions:** Increasing total PA by 28% (100 cpm) was associated with a decrease of 1.4 kg of fat mass and 1% body fat for 16 wk in overweight Hispanic adolescents independent of intervention group assignment. Increases in total PA, compared with MVPA, may be sufficient to improve body composition in overweight Hispanic adolescents. **Key Words:** OBESITY, CHILDREN, ACCELEROMETER, YOUTH, DEXA

National data from 2003 to 2006 indicate that 34% of adolescents in the United States are at risk for overweight and 17% are overweight (23). The prevalence among Mexican American adolescents is even higher than the national average, with 39% at risk for overweight and of those 21% are overweight (23). These high rates of overweight among Hispanic adolescents are an important public health concern because overweight adolescents are more likely to become overweight adults (31). In addition, being overweight contributes to the development of chronic diseases, such as type 2 diabetes, cardiovascular disease, and obesity-related cancers (19,24). Hispanics are the largest and fastest-growing ethnic minority in the United States (40); it is projected that by 2050 they will constitute almost one fourth of the US population. Given

the rate at which this population is growing and the incidence rates of obesity and related comorbidities among the youth of this population, it is imperative to identify the etiology of obesity in Hispanic adolescents.

Undoubtedly, there are many factors contributing to the alarming obesity rate, and one of the modifiable risk factors is physical activity (PA; 17,26). PA declines dramatically as children become adolescents (11,16,21). Only 8% of adolescents meet the current CDC PA recommendations (60 $\text{min}\cdot\text{d}^{-1}$ of at least moderate-intensity activity [38]). Whereas many studies in children and adolescents have found that increased PA is associated with decreased adiposity (20), there have been mixed reports (34). These discrepancies may be due to subjective methods used to assess PA and adiposity, such as questionnaires (28) and self-report body mass index (BMI). The use of objective and precise measures of PA and adiposity, such as accelerometry and DEXA (10,28,32,42), can reduce measurement error, thereby increasing the ability to detect effects and elucidate the relationship between PA and adiposity (29). Another way to increase the ability to detect an effect is to control for potential confounders, such as energy intake. Several studies have stated the importance of exploring the influence of energy intake when examining the relationship between physical activity and adiposity (6,22,35).

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Recent cross-sectional studies have shown that objectively measured PA is associated with adiposity in adolescents (1,18), but to date, no study has examined the short-term effects of changes in objectively measured PA on changes in adiposity, particularly in Hispanic youth. Therefore, the specific objectives of the current research were to 1) investigate whether changes in objectively measured total PA and percent of time spent in moderate to vigorous PA (MVPA) are associated with changes in adiposity measures, including total fat mass, percent body fat, and visceral fat and 2) examine the influence of energy intake on the relationship between changes in PA and changes in adiposity.

METHODS

Participants

Study participants consisted of a subgroup of 54 overweight Hispanic adolescents who participated in a randomized nutrition and strength training type 2 diabetes prevention intervention. The intervention had no effect on PA; as a result, the sample of the current study combined the treatment groups and controlled for treatment group in the analyses. Participants were recruited from local area high schools, health care centers, community centers, and by word of mouth, and newspaper ads.

Except for the accelerometry methodology that will be described in detail below, a complete description of the study methods has been reported elsewhere (5), so only a brief overview of the methods will be given here. Participants were 38 adolescents (19 girls, 19 boys) who had complete data for relevant measures at pretest and posttest (10 in the control group, 20 in the nutrition only group, 8 in the nutrition + strength training group). Participants included in the analyses were not significantly different from those excluded (sex, age, Tanner stage, BMI percentile, weight, total fat, or lean tissue mass, all $P > 0.10$). Informed written parental consent and child assent were obtained before testing. The institutional review board of the University of Southern California approved the study.

Procedures

Screening visit. Participants arrived at the General Clinical Research Center after an overnight fast. A licensed pediatric health care provider conducted a medical history examination and determined sexual maturation (37). To screen for diabetes, an oral glucose tolerance test was conducted. Participants who met the following criteria were invited back for further testing: 1) age- and sex-specific BMI ≥ 85 th percentile; 2) Hispanic ethnicity, assessed by parental report of maternal and paternal Hispanic grandparents; 3) grades 9th to 12th; 4) not currently taking medication or diagnosed with any syndrome or disease that influences fat distribution or insulin action; 5) not diagnosed with diabetes at screening or any major illness

(e.g., cancer) since birth; and 6) not participating in a structured exercise, nutrition, or weight loss program in the past 6 months.

Anthropometry and body composition. Weight and height were measured in triplicate using a beam medical scale and wall-mounted stadiometer, respectively, and then averaged. BMI percentiles for age and sex were determined using EpiInfo 2000, Version 1.1 (CDC, Atlanta, GA). Whole body fat, lean tissue, and percent body fat were measured by dual-energy x-ray absorptiometry (DEXA) using a Hologic QDR 4500W (Hologic, Bedford, MA).

Energy intake and PA. To assess energy intake participants completed 3-d diet records at home after being trained by study staff who were supervised by a registered dietician. When compared with 24-h recall and 5-d food frequency, the 3-d food record had the strongest agreement between observed and reported intakes (4). Staff clarified records when they were collected. Nutrition data were analyzed using the Nutrition Data System for Research (NDS-R version 5.0_35) developed by the University of Minnesota.

To assess PA, subjects were instructed to wear Actigraph accelerometers (GT1M or 7164; Actigraph, LLC, Pensacola, FL) for 7 d, except during water-based activities or when sleeping (27,41). The Actigraph accelerometer is a reliable instrument, with an intraclass correlation of 0.99 (7). Accelerometers were set to monitor activity in 15-s epochs, which were collapsed to 60-s epochs during analysis. Data were reduced using an adapted version of the SAS code used for the 2003–2004 National Health and Nutrition Examination Survey available at http://riskfactor.cancer.gov/tools/nhanes_pam. A correction factor was applied to allow for comparison between the two Actigraph monitor models (3).

The amount of time the participant wore the device was determined by subtracting nonwear time from 24 h. Nonwear time was defined by an interval ≥ 60 consecutive minutes of zero activity counts, with allowance for 1–2 min of counts between 0 and 100. Days with less than 6 h of wear data were not considered acceptable, and participants with ≥ 2 d of acceptable accelerometry data at pretest and posttest were included. There is no clear consensus on the length of acceptable monitoring periods (39), and the monitoring period of the current study was similar in duration to monitoring periods in other accelerometry studies (2,15,16). At pretest, participants with acceptable data wore the accelerometers for a mean \pm SD of 12.6 ± 1.3 h·d⁻¹ for 6.2 ± 2.3 d, which resulted in a mean monitoring period of 78.1 h. At posttest, the participants with acceptable data wore the accelerometers for 12.4 ± 1.4 h·d⁻¹ for 5.6 ± 2.7 d, which resulted in a mean monitoring period of 69.7 h. Statistical analyses were repeated in a subsample ($n = 36$) of participants with ≥ 3 d of accelerometry data, and similar results were obtained.

Data from all acceptable days were averaged and included the following variables: number of wear days, average number of minutes worn, total PA represented by

TABLE 1. Characteristics of overweight Hispanic participants at pretest and posttest ($n = 38$).

	Pretest	Posttest
Demographic factors		
Sex (% male)	50.00%	—
Age (yr)	15.3 (1.0)	15.8 (0.98)
Maturation/Tanner stage (1–5), median (SD)	5.0 (0.8)	—
Body composition variables		
Weight (kg) ^a	93.9 (23.3)	93.8 (21.9)
Total fat (kg)	35.9 (15.1)	34.7 (12.8)
Lean tissue (kg)	55.8 (10.9)	56.4 (11.6)
Body fat (%)	37.9 (8.0)	36.9 (7.4)
BMI z-score	2.1 (0.6)	2.0 (0.6)*
BMI percentile for age and sex	96.8 (3.9)	95.9 (5.6)**
PA variables		
Total PA (cpm)	359.9 (124.8)	380.1 (141.5)
Percent of time in MVPA	4.9 (2.9)	5.7 (3.4)
Energy intake (kcal)	1908.2 (681.1)	1762.3 (741.5)

Values are mean (SD) unless otherwise specified. Height and weight were an average of three measurements, energy intake was assessed with 3-d diet records, and PA was assessed with 7-d accelerometry.

^a BMI.

* $P < 0.01$, paired t -test.

** $P < 0.05$, paired t -test.

average counts per minute (cpm) on wear periods from all valid days, and percent of wear time spent in MVPA. The intensity cut points applied to categorize MVPA were those used for adults and older adolescents in NHANES (≥ 2020 cpm [38]) because the current sample of adolescents had an average weight of 94 kg and median Tanner stage of 5, suggesting that their biomechanics may be closer to those of adults than children. To ensure that results were not an artifact of the MVPA cut point used, the analyses were replicated using the age-dependent MVPA cut points on the basis of the pediatric equation of Freedson et al. (8,9).

Statistical analysis. Paired-sample t -tests were conducted to determine whether there were pretest and posttest mean differences in anthropometric, adiposity, PA, or dietary measures. Pearson correlations were conducted to describe bivariate relationships. Multiple regression analyses were conducted to assess whether changes in PA (e.g., total PA and MVPA) were associated with changes in adiposity (e.g., DEXA data) after controlling for covariates. The following standard covariates were included in all models: sex, age, pretest PA, pretest adiposity, and intervention group. Additional covariates included pretest and posttest DEXA lean mass when change in fat mass was the dependent variable.

Residual diagnostic analyses were completed to ensure that the assumptions of regression were not violated. Further diagnostic analyses were also evaluated to identify collinear predictors, specifically to ensure that tolerance was >0.1 and the variance inflation factor was <10 . For the regression models, unstandardized beta (β) coefficient estimates, SEs, and P values are reported. Throughout the regression analyses, total PA is discussed in increments of 100 cpm to ease the interpretation of the coefficient estimates. The value of the coefficient estimate is the amount of change in the PA-independent variable, e.g., MVPA (1%) or total PA (100 cpm).

To assess the influence of energy intake on the relationship between change in PA and change in adiposity, energy intake was included in the regression models. If including energy intake changed the coefficient estimate of the PA variable by $>10\%$, then it was considered a confounder (12,14). Analyses were conducted using SPSS for Windows (Version 16; SPSS, Inc., Chicago, IL) and SAS (v9.1; SAS Institute, Cary, NC). $P < 0.05$ denotes statistical significance.

RESULTS

Table 1 shows the participant characteristics at pretest and posttest. The participants consisted of 19 boys and 19 girls; 92% had Tanner stage of 4 or 5. The participants had an average age of 15 yr, average weight of 95 kg, and 97th BMI percentile for age and sex at pretest. There were statistically significant decreases between pretest and posttest in BMI z-score ($P = 0.01$) and BMI percentile ($P = 0.02$).

Although there were no group-level changes in PA or energy intake from pretest to posttest, there were noteworthy individual changes in total PA (maximum decrease -317.8 cpm vs maximum increase 339.3 cpm), percent of time spent in MVPA (maximum decrease -8.9% vs maximum increase 6.6%), and reported energy intake (maximum decrease was -1409 kcal vs maximum increase 2048 kcal). Figure 1 shows the individual values of change (post $-$ pre) in total PA for each participant; 22 participants increased and 16 decreased their total PA. Similarly, 23 participants increased (mean \pm SD, $2.8 \pm 2.1\%$) and 15 decreased ($-2.2 \pm 2.5\%$) their percent of time spent in MVPA (data not shown). Table 2 shows the Pearson correlations of change in adiposity variables, change in PA variables, change in energy intake, and demographic variables. Changes in total PA and percent time in MVPA were not correlated to the change in adiposity. Age was the only variable significantly correlated with change in adiposity; older participants had greater reductions in fat mass and percent body fat ($P < 0.05$).

Changes in total PA and adiposity. Regression analyses revealed that an increase in total PA (cpm) was

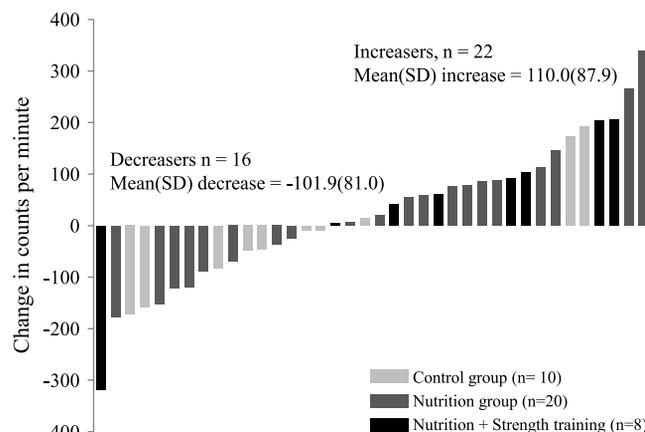


FIGURE 1—Change in total PA (cpm) by individual ($n = 38$).

TABLE 2. Pearson correlations of change in adiposity variables, change in PA variables, change in energy intake, and demographic variables ($n = 38$).

	Change in Adiposity	
	Change in Fat Mass (kg)	Change in Percent Body Fat
	Correlation Coefficient (P)	
Change in total PA (cpm)	-0.23 (0.17)	-0.29 (0.08)
Change in percent time in MVPA	-0.19 (0.25)	-0.22 (0.19)
Change in energy intake (kcal)	0.13 (0.44)	0.05 (0.76)
Age (yr)	-0.42 (0.01)	-0.37 (0.02)
Sex (% male)	0.30 (0.06)	0.15 (0.38)

significantly associated with a decrease in total fat mass after controlling for the standard covariates ($\beta = -1.3$, $P = 0.02$). Including energy intake in the model increased the coefficient estimate 8% (Table 3; $\beta = -1.4$, $P = 0.02$); thus, it cannot be concluded that energy intake confounded the relationship (because change in estimate <10%).

An increase in total PA (cpm) was significantly associated with a decrease in percent body fat ($\beta = -0.8$, $P = 0.03$), and energy intake confounded the relationship between total PA and percent body fat, increasing the coefficient estimate by 25% (Table 3; $\beta = -1.0$, $P = 0.01$). Figure 2 is a scatter plot of the change in total PA by the predicted values of change in total fat mass after controlling for the standard covariates, which shows a negative relationship between change in PA and predicted change in fat mass.

Changes in percent of time spent in MVPA and adiposity. After controlling for standard covariates, regression analyses revealed that an increase in percent time spent in MVPA was marginally associated with a decrease in percent body fat ($\beta = -0.26$, $P = 0.10$). When change in energy intake was included in the model, this relationship became significant (Table 4; $\beta = -0.33$, $P = 0.04$). The relationships between change in percent time in MVPA

TABLE 3. Regression analyses of changes in total PA (cpm) and changes in total fat mass and percent body fat ($n = 38$).

Predictor Variables	Dependent Variables	
	Change in Total Fat Mass (kg)	Change in Percent Body Fat
Intercept	30.12 (11.3)*	24.90 (8.2)**
Demographic factors		
Sex	3.87 (1.8)*	2.74 (0.9)**
Age (yr)	-2.07 (0.6)**	-1.62 (0.5)**
Body composition		
Adiposity measure at visit 1 (kg, %)	0.87 (0.10)***	0.85 (0.1)***
Lean tissue mass at visit 1 (kg)	-0.23 (0.2)	—
Lean tissue mass at visit 2 (kg)	0.21 (0.2)	—
Intervention assignment		
Nutrition + strength training group	-0.94 (1.4)	-0.12 (1.0)
Nutrition only group	-3.31 (1.7)	-2.58 (1.3)
PA variables		
Mean total PA at visit 1 (cpm)	0.005 (0.006)	0.003 (0.004)
Change in mean total PA (cpm)	-0.014 (0.005)*	-0.01 (0.004)*
Change in energy intake (kcal)	0.001 (0.001)	-0.001 (0.001)

Values are unstandardized coefficient estimates (SE). Multiple regression analyses were conducted to assess whether changes in mean total PA were associated with changes in total fat mass and percent body fat after controlling for covariates.
 * $P < 0.05$.
 ** $P < 0.01$.
 *** $P < 0.001$.

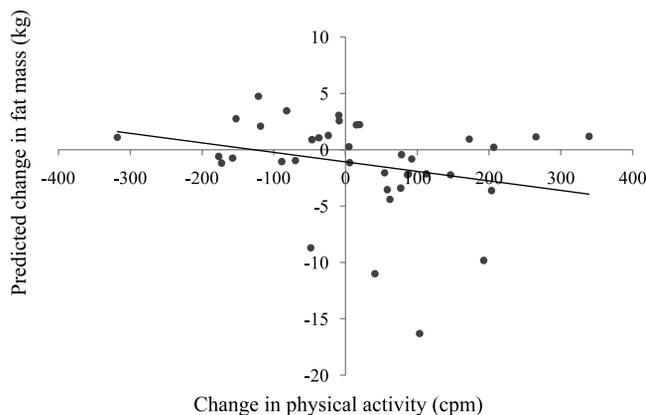


FIGURE 2—Change in total PA (cpm) by change in fat mass (kg). Predicted values are adjusted for sex, age, intervention group, lean tissue mass, and pretest PA ($n = 38$).

and percent body fat were no longer significant after accounting for change in total PA, regardless of whether change in energy intake was in the model ($\beta = -0.29$, $P = 0.43$) or not (Table 4; $\beta = 0.17$, $P = 0.64$).

Increases in percent of time spent in MVPA were associated with decreases in fat mass both before accounting for change in energy intake ($\beta = -0.49$, $P = 0.04$) and after ($\beta = -0.51$, $P = 0.03$), but not after controlling for change in total PA regardless of whether energy intake was excluded ($\beta = -0.02$, $P = 0.96$) or included in the model ($\beta = -0.15$, $P = 0.75$).

Parallel results were obtained when using MVPA cut points generated by the pediatric equation of Freedson et al.

TABLE 4. Regression analyses of changes in percentage of time spent in MVPA and changes in percent body fat with and without controlling for total PA (cpm; $n = 38$).

Predictor Variables	Dependent Variable = Change in Percent Body Fat	
	Model Without Controlling for Total PA (cpm)	Model Controlling for Total PA (cpm)
Intercept	23.56 (8.2)*	24.60 (7.9)*
Demographic factors		
Sex	2.7 (0.9)*	2.7 (0.9)*
Age (yr)	-1.5 (0.5)*	-1.6 (0.5)*
Body composition		
Percent body fat at visit 1	0.85 (0.1)**	0.87 (0.8)**
Intervention assignment		
Nutrition + strength training group	-0.21 (1.1)	-0.21 (1.1)
Nutrition only group	-2.9 (1.3)***	-2.6 (1.3)****
PA variables		
Percent time in MVPA	16.6 (20.6)	16.6 (20.1)
Change in percent time in MVPA	-0.33 (0.15)***	0.17 (0.40)
Change in mean total PA (cpm)	—	-0.01 (0.01)
Change in energy intake (kcal)	-0.001 (0.001)****	-0.001 (0.001)****

Values are unstandardized coefficient estimates (SE). Multiple regression analyses were conducted to assess whether changes in percent time in MVPA were associated with changes in percent body fat after controlling for covariates.
 * $P < 0.01$.
 ** $P < 0.001$.
 *** $P < 0.05$.
 **** $P < 0.10$.

(8,9). Increases in MVPA were significantly associated with decreases in percent body fat ($\beta = -0.37$, $P = 0.09$) and fat mass ($\beta = -0.81$, $P = 0.009$) after accounting for energy intake, but not after adjusting for total PA (all $P > 0.60$). Including the total hours of measurement at pretest and posttest as covariates in the models did not affect the results, and these were not included in the final models.

DISCUSSION

A major objective of this study was to examine how changes in PA during a 16-wk period are associated with changes in adiposity in overweight Hispanic adolescents. The primary findings are that a short-term increase in objectively measured total PA is significantly associated with a decrease in both total fat mass and percent body fat. Specifically, an increase of 28% of total PA, or 100 cpm, was associated with a decrease of 1.4 kg fat mass and 1% body fat after controlling for energy intake and standard covariates. To translate the accelerometry unit of counts per minute into more physiologically relevant terms, prediction equations based on previous observations in normal-weight adolescents (6) were used to estimate that an increase of 100 cpm is broadly similar to an increase of 250 kcal of energy expenditure.

A secondary objective was to examine energy intake as a confounder of the relationship between changes in PA and adiposity. Recent studies in adolescents state that adjusting for energy intake may strengthen the observed relationships between objectively measured PA and adiposity (6,35). The current study supports these assertions, finding that accounting for changes in energy intake strengthened the relationship between changes in PA and adiposity. Change in energy intake was identified as a confounder in the relationship between total PA and percent body fat because adjusting for change in energy intake increased the coefficient estimate by 25%, which was greater than the 10% change in the coefficient needed to identify it as a confounder. Change in energy intake was not identified as a confounder in the relationship between total PA and total fat mass because adjusting for change in energy intake only changed the coefficient estimate by 8%. The 10% change in coefficient to define a confounder is a rather arbitrary rule of thumb and is not based on a statistical test (14). Given that accounting for energy intake strengthened the relationship between changes in PA and adiposity, future studies attempting to quantify the magnitude of this relationship may consider whether to account for energy intake.

Accounting for the influence of energy intake had a modest effect on the relationship between PA and body fat. When the change in energy intake was accounted for, the decrease in fat mass that was associated with an increase of 100 cpm of total PA changed by 0.1 kg (1.3 to 1.4 kg), and the decrease in percent body fat changed by 0.2% (0.8%–1.0%). The modest change observed when accounting for change in reported energy intake may be partly explained

by the large variation in the change in energy intake from pretest to posttest (from a decrease of -1409 kcal to an increase of 2048 kcal). This variation may be due to the self-report nature of the dietary measure. The variation could also be a reflection of a fasting/gorging cycle of eating that may be practiced by the adolescents in this sample. No studies describing the eating behaviors of overweight Hispanic adolescents were identified. Future studies that assess diet over a longer measurement period (7–14 d) may help to identify the dietary patterns of overweight Hispanic adolescents.

An increase in the percent of time spent in MVPA was not associated with a decrease in either percent body fat or total fat mass after accounting for total PA. These findings suggest that increases in total PA may be sufficient to result in improvements in body composition in overweight Hispanic adolescents. It should be noted that these findings do not suggest that increases in MVPA will not lead to reductions in fat mass, but MVPA may be difficult for and not well tolerated by this population. As such, it is important for future randomized trials to determine whether increases in total physical, independent of MVPA, can reduce adiposity in overweight Hispanic adolescents.

There is no clear consensus on whether the intensity level of PA influences changes in adiposity, especially in overweight adolescents. Some longitudinal studies that report a relationship between objectively measured MVPA and body fat have accounted for total PA (6), whereas others have not (35). Therefore, it remains unclear whether the reductions in body fat observed in these studies were associated with MVPA independent of total PA. In a large cohort of 12-yr-olds, time spent in MVPA was negatively associated with fat mass after adjusting for total PA (22), suggesting that the physiological effects of MVPA may be importantly related to adiposity. Future longitudinal studies examining the relationship between MVPA and adiposity should control for total PA to determine whether decreases in adiposity are related specifically to an increase in MVPA or whether the effects are due simply to an increase in total PA. This has important public health implications given that the barriers to increasing MVPA may be different than the barriers to increasing total daily PA, especially in overweight youth (43).

It is also possible that a significant relationship between changes in percent of time spent in MVPA and adiposity was not detected because of the cut points used to designate time spent in MVPA. There is no agreement on which cut points should be used in different populations, and using cut points derived from different prediction equations can yield markedly different results (13). The prediction equations also introduce residual error into the measurement, and thus, counts per minute may be a more valid measure of activity than intensity level (8). It is unlikely, however, that the MVPA cut point used in the current study contributed to the failure to detect a relationship between changes in MVPA and adiposity independent of total activity because

when cut points from a different prediction equation were used to designate MVPA (8,9), the results were replicated.

In a prospective 4-yr study with older adolescents, Ekelund et al. (6) also reported that change in total activity, not MVPA, was related to change in adiposity, although they only reported these results in normal-weight participants. The authors postulate that a failure to detect an association in overweight adolescents may be due to the lack of change in activity in this group. In the current study, we also reported a lack of overall change, but there was a nicely distributed variability in change in PA at the individual level. One reason for the apparently discrepant findings may be that the distribution of change in activity was differently dispersed in Ekelund et al., making it difficult to identify a relationship. It is difficult to compare results from these studies, however, because of the differences in the populations studied, the length of the follow-up period, and the different covariates, such as dietary intake, included in the analyses.

Some longitudinal studies using BMI to measure adiposity have failed to find a relationship with PA (35,36). One strength of the current study was the ability to examine the relationship with objective measures of PA and adiposity, e.g., activity by accelerometry (29) and total fat mass and percent body fat measured by DEXA (10). Another strength of the current study is the homogeneity of the demographic characteristics (e.g., ethnicity, age, Tanner stage, overweight status) of the sample of the participants. This homogeneity helps to reduce the effects of maturation bias, although this also restricts the generalizability of the findings to those who are similar to the study participants.

Our findings are supported by cross-sectional studies that have found a significant association between PA and adiposity (30,33). Coupled with cross-sectional and longitudinal studies, the current analysis lends support to a causal inverse association between changes in PA and adiposity, but caution should be used when interpreting these findings as definitive evidence of causation.

A potential limitation to this study is the fact that accelerometry is not a perfect measure of PA because it is

worn on the hip and does not accurately detect bicycling or upper body movement, such as weight lifting. Similarly, the accelerometer cannot be worn during water-based activities, such as swimming. A recent large cohort study reported that swimming is not a common activity among Hispanic adolescents (1), so this limitation may not be of concern in the current sample. The definition of valid accelerometry data used in the current study, 2 d and 6 h, is another potential limitation, although the average measurement period was approximately 70 h. The self-report nature of the dietary data may also be a limitation, especially given the overweight status of the sample and the likelihood of overweight participants to underreport their dietary intake (25). Another possible limitation of the study is the fact that dietary intake was not examined as either an effect modifier or a mediator of the relationship between PA and adiposity. The authors acknowledge the probability that energy intake may act as a mediator and/or effect modifier, but that investigation is outside the scope of the current study.

To our knowledge, this is the first study to examine the relationship between short-term changes in objectively measured activity by accelerometry and adiposity and to examine the influence of energy intake on this relationship in overweight Hispanic adolescents. In summary, after accounting for changes in energy intake, an increase of 28% of total physical, or roughly 250 kcal, was associated with a modest, yet significant, decrease of 1.4 kg of fat mass and 1% body fat in overweight Hispanic adolescents for 16 wk, although there was no association between change in time spent in MVPA and adiposity independent of total PA.

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The results of the present study do not constitute endorsement by the American College of Sports Medicine.

No conflicts of interest existed for any of the authors.

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