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Leptin predicts a decline in moderate to vigorous physical activity in minority female children at risk for obesity

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Abstract

Background—Leptin may influence moderate to vigorous physical activity (MVPA) at the start of puberty. The direction and magnitude of this association is unclear.

Objectives—To determine the effect of baseline leptin on MVPA over one year in minority girls at high-risk for obesity.

Methods—Data came from TRANSITIONS, a longitudinal observational study on the age-related MVPA decline. Fifty peri-pubertal girls aged 8–11 years at baseline participated. Baseline leptin (ng/mL) was collected via a duplicated assay using a double antibody Radio Immune Assay. MVPA (min/day) was measured using accelerometers for at least four 10-hr days on a quarterly basis for up to one year.

Results—Continuous leptin was negatively related to MVPA ($p=0.001$) independent of central adiposity at baseline and predicted the MVPA decline over one year ($p=0.002$). For descriptive purposes, baseline leptin was dichotomized at the sample median into ‘high leptin’ and ‘low leptin’ categories to determine whether MVPA trajectories differed between these groups. Girls with ‘low leptin’ at baseline had significantly higher levels of MPVA at baseline, visit 1, and visit 2 compared to girls with ‘high leptin’.

Conclusions—High leptin levels predicted nearly a 12.6% decline in MVPA over one year. These findings provide support for the biological basis of declining MVPA as girls enter puberty.

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Disclosure Statement

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Keywords

leptin; accelerometer; biological basis; physical activity; adolescent

Introduction

Obesity prevention in children is a primary public health concern. Among youth 6–11 year olds, 35.5% are overweight (defined as having an age- and sex- specific body mass index (BMI) percentile ≥ 85)¹. The epidemic is more severe in minority youth. Recent national estimates indicate that 42.6% of Hispanic youth (versus 34.5% of non-Hispanic whites) are overweight¹. Pediatric obesity has deleterious health consequences such as metabolic syndrome² and type 2 diabetes³. Furthermore, overweight and obese youth have an increased risk for all-cause mortality as adults⁴.

Many factors are associated with increased risk for obesity, including low levels of physical activity⁵. There is a well-documented age-related decline in physical activity^{6,7}, especially as youth enter puberty⁸. Based on a nationally-representative sample of youth, only 41.4% meet the current youth physical activity recommendations of at least 60 minutes or more of moderate to vigorous physical activity (MVPA) per day⁹. Pate and colleagues⁷ found a 4% decline in accelerometer-measured physical activity in girls over one year, however other studies using self-reported physical activity report higher rates of decline^{10–12}. To date, public health efforts have had little impact on declining physical activity levels as youth traverse puberty. It seems necessary to look beyond solely behavioral determinants of activity and pursue other potential causes underlying the adolescent physical activity decline.

Biological factors have been hypothesized to contribute to the declining levels of physical activity as children age¹³; however their magnitude of influence and mechanisms are unclear. Leptin, an adipose-derived hormone secreted in direct proportion to adipose tissue mass¹⁴, may influence activity levels. Leptin signals to the brain that fat stores are sufficient and energy intake can decrease while energy expenditure can increase¹⁵. In lean individuals, normal leptin levels are related to increases in energy expenditure¹⁶. However, in overweight and obese individuals, there is evidence of ‘leptin resistance’ where plasma leptin levels are chronically high, yet do not produce the expected decrease in energy intake and increase in energy expenditure^{17,18}. The excess adipose tissue found in obese individuals results in high circulating leptin levels that saturate the leptin receptors and diminish their ability to regulate energy balance^{16,19}. In a sample of White children (mean age 10.6 years), overweight youth had higher fasting leptin levels than normal weight youth at baseline and at a one-year follow-up. The authors noted that the leptin levels remained consistently higher in the overweight children, which may indicate some degree of leptin resistance²⁰.

Due to its central role in energy regulation, leptin is hypothesized to be related to observed physical activity declines as youth begin puberty¹⁶. However, the direction of the relationship between leptin and physical activity has not been conclusively established in youth, and it may be influenced by other factors such as age, pubertal status, and sex. This study aims to determine if leptin influences MVPA levels. We hypothesize that baseline leptin levels will predict a decline in MVPA over one year in peri-pubertal girls.

Methods

Participants

Participants were recruited from clinics, churches, schools, and community centers in the Los Angeles area from June 2006 to August 2009. All participants were at-risk for adult obesity, defined as (1) being overweight or obese; or (2) being of normal weight with at least one overweight/obese parent. All subjects were of self-reported Latina (n= 39) or African American ancestry (n=11) (all four grandparents of Latino or African American origin as determined by parental self-report), non-menstruating girls, between the ages of eight and 11, who were at pubertal Tanner breast stage 1 or 2²¹. Participants were ineligible if they were taking medications known to affect body composition, had syndromes or diseases known to affect body composition or fat distribution, or had any major illness since birth. Participants were excluded from the study if they had diabetes (defined as fasting plasma glucose ≥ 126 mg/dl). Informed written parental consent and participant assent (in the primary language of each) was obtained prior to participation. The study was approved by the Institutional Review Board of the University of Southern California.

Study design

Participants completed an initial visit to the Clinical Trials Unit (CTU) in order to screen for diabetes and determine Tanner breast stage. Eligible participants then completed an annual inpatient visit at the CTU. In addition to the annual visit, accelerometer measured physical activity was assessed at quarterly visits every three months.

Procedures

At the yearly inpatient visits, participants arrived at the CTU in the afternoon on Day 1. The participants completed anthropometric and body composition measures before receiving a complete medical examination and health history conducted by a licensed pediatric health care provider. After the examination, participants were given a standard meal and snack before 20.00h, after which they were only permitted to drink water. After spending the night, on Day 2 at 08.00h, fasting blood samples were taken to measure leptin. The Frequently Sampled Intravenous Glucose Tolerance Test (FSIVGTT) was performed over the course of three hours to assess insulin sensitivity (SI). Participants were then instructed on how to wear an accelerometer for the seven days following the inpatient visit. After instruction and lunch, participants were discharged from the CTU. A study team member subsequently contacted the participant to ensure that the accelerometer was worn and to retrieve the device.

In between the yearly visits, quarterly home visits were conducted at three month intervals. At these visits, the participants were provided with an accelerometer and requested to wear it during waking hours for seven days. Accelerometer data from the Year 1 (baseline) inpatient visit, three subsequent quarterly assessments, and the Year 2 inpatient visit are included in this analysis, for a total of five time points.

Measures

Demographics & Physical Exam—Baseline age (in years) was recorded at the Year 1 baseline visit. Child race/ethnicity was self-reported by the parent. Pubertal Tanner breast stage²¹ was assessed via palpation by the licensed pediatric medical care provider at the screening visit.

Body Composition—Subcutaneous abdominal adipose tissue (SAAT; in L) and visceral adipose tissue (VAT; in L) were measured by magnetic resonance imaging (MRI) at the

inpatient visits. Percent body fat was measured using air displacement plethysmography (Bod Pod®). Height (cm) and weight (kg) were recorded in triplicate by the nursing staff and the average of the three readings was calculated. Body mass index (BMI) was calculated as kg/m^2 from the average height and weight measurements. The 2000 Centers for Disease Control and Prevention (CDC) age- and sex-specific growth charts were used to calculate BMI percentiles ²².

Clinical Measures—Leptin was assessed at the inpatient visits. Fasting plasma leptin (ng/mL) levels were determined in duplicate using a double antibody Radio Immune Assay (RIA) kit (Millipore, St. Charles, MO) with a 0.5 ng/mL limit of sensitivity. Leptin intra- and inter- assay CV were up to 6.2% and up to 5.3%, respectively. All samples were measured in duplicate.

Physical Activity—Accelerometers assessed physical activity. The participants wore a uniaxial Actigraph GT1M accelerometer (Actigraph, LLC, Fort Walton Beach, FL) on the right hip. To be included, the participants needed to have at least four 10-hour days of wear ^{5, 6}. Data were downloaded after each visit and reviewed to ensure the monitor was functioning correctly. The data was processed using an adaptation of SAS code developed by the National Cancer Institute for use with NHANES data (available at: http://riskfactor.cancer.gov/tools/nhanes_pam). Using this program, non-wear time was defined as 60 minutes or more of zero intensity counts, with allowance for up to two consecutive minutes of counts between one and 100. The non-wear period ended when at least one of the following conditions were met: one minute of an intensity above 100; one minute with a missing intensity count; three or more minutes with intensity counts between one and 100 counts; or the last minute of the day. Wear time was calculated by subtracting non-wear time from the total observation time for each day.

Using accelerometers, movement intensity is captured by ‘counts’. Counts from the valid days were categorized into activity levels using intensity thresholds developed by Evenson and colleagues ²³. These intensity thresholds have been shown to have acceptable classification accuracy for children ²⁴. Mean minutes per day (min/day) spent in MVPA is calculated by summing each minute spent above the user-defined thresholds for MVPA, and averaging across all valid days of wear.

Data Analysis

All analyses were conducted using SAS v9.2 (SAS Institute, Inc., Cary, NC) with statistical significance set at $p = 0.05$. Mean estimates and standard deviations and frequencies describe the sample. Normality was assessed for the primary independent (baseline leptin) and dependent (MVPA) variables. Because leptin and MVPA were not normally distributed, a log transformation was performed. Differences in estimates of mean physical activity variables across the visits were assessed using paired t-tests. For descriptive purposes, baseline leptin was dichotomized at the sample median into ‘high leptin’ and ‘low leptin’ categories. Mean min/day spent in MVPA at each study visit were calculated for each of the groups. Differences in estimates of mean MVPA between the leptin groups were assessed using t-tests.

Generalized linear regression assessed the baseline cross-sectional association between continuous leptin concentration and MVPA. Covariates were age (years), race/ethnicity (Latina vs. African American), SAAT (L), and VAT (L). SAAT and VAT were included in the models as central adiposity measures because leptin is predominantly secreted by adipose tissue in these regions ²⁵.

A mixed model assessed the longitudinal effects of leptin levels on MVPA over one year. The models used all available data for each participant and a compound symmetric covariance matrix. Variance components were estimated using the restricted maximum likelihood estimate method to control for within-individual correlations over the five visits. Baseline continuous leptin was the independent variable and mean min/day spent in MVPA was the dependent variable. Baseline covariates age, race/ethnicity, SAAT, and VAT were treated as time-invariant variables. Visit number (zero to four) was treated as a repeated measure, and model-based estimates of mean differences between baseline and subsequent visits were calculated. Pubertal Tanner stage was not significant in any models, therefore it was not included as a covariate.

Results

Participants with missing data were excluded from the analyses. The original sample size was 79 participants²⁶. Of these, 14 had missing accelerometer data (due to monitor malfunction or non-compliance), nine had an insufficient number of days of physical activity data, one had missing leptin data, and five had missing MRI data. The final sample size for these analyses was 50 participants. Excluded participants were not significantly different on baseline age, race/ethnicity, and BMI percentile than those with complete data (data not shown).

Table 1 presents baseline sample characteristics for the analyzed sample. The majority of the analyzed sample was Latina (78%) with a mean age of 9.4 (± 0.9) years. A large proportion of the sample was overweight (24.0%) or obese (38%). Table 2 presents the estimates of physical activity variables across the visits. Mean monitor wear time was stable from baseline to visit three, and was 28 min/day higher in visit four ($p < 0.050$) compared to baseline. Mean counts per minute (cpm) were significantly different at visits two and four compared to baseline ($p < 0.050$). MVPA declined by four minutes (12.6%) between baseline and visit four. MVPA (min/day) levels at visits three and four were significantly lower than baseline levels ($p < 0.050$). Paired t-tests between baseline and year two body composition measures (percent body fat, BMI percentile, BMI z-score) examined whether the means were significantly different between the last and first visits. The results indicated that there were no significant differences in these variable means (data not shown).

There is currently a debate in the field as to how many days of accelerometer data are necessary to produce reliable estimates of MVPA. Thus, we conducted an analysis that included the nine participants with one or more days of data. The results using 59 participants did not appreciably change from the results with 50 participants with four or more days of data. Previous research indicates that four or more days of data achieve a higher reliability in younger children²⁷. Therefore, we present regression results for the 50 participants with four or more days of data. Table 3 presents the cross-sectional baseline linear regression results. The model explained 39% of the variance in mean MVPA (min/d). Leptin ($p = 0.001$) was negatively associated with MVPA independent of central adiposity. Table 4 presents the longitudinal mixed model results. In an attempt to account for potential differences in MVPA estimates due to variation in monitor wear time across the visits, mean wear time was included in the mixed models. This variable did not substantially change the results (data not shown); therefore this variable was not included in the final model. Continuous baseline leptin concentration significantly predicted a decline in MVPA over one year in this sample ($\beta = -0.36 (\pm 0.11)$; $p = 0.002$), independent of age, SAAT, and VAT. Figure 1 presents the mean MVPA levels over all of the visits for the entire analyzed sample, and for the high and low leptin groups. Compared to those with high baseline leptin values, the low leptin group had significantly higher min/day in MVPA at baseline, visit one and visit two ($p < 0.050$).

Discussion

To our knowledge, this is the first study to demonstrate the effects of leptin on the decline in physical activity in a sample of minority peri-pubertal girls. Baseline leptin levels were related to both cross-sectional and longitudinal physical activity levels. These results suggest that leptin levels at the beginning of puberty may be a salient factor in the steady decline of physical activity levels in girls.

Findings from previous studies on the relationship between leptin and physical activity have been inconsistent. Romon and colleagues²⁸ reported leptin was negatively correlated to pedometer-calculated steps per day in girls in a cross-sectional sample of 510 White youth aged eight to 18 years. Conversely, in a sample of 125 Pima Indian children (mean weight=23.2 kg) Salbe and colleagues²⁹ found that plasma leptin concentrations were positively associated with physical activity levels (measured as the ratio of TEE:RMR). However, this relationship was not found in a longitudinal study of 213 healthy children, where accelerometer-measured physical activity did not significantly correlate with leptin levels³⁰. The results from the current study support the findings of Romon and colleagues, suggesting that higher circulating leptin concentrations are associated with lower levels of physical activity in girls. The correspondence between the findings from this study and those from Romon may be due to the fact that both studies employed device-based measures of physical activity, which provides more robust estimates of activity in youth than other measures. Also, although sex differences in leptin levels during puberty have been shown³¹, Salbe and Metcalf did not stratify by sex, while our study and that of Romon were conducted in girls of similar ages.

The decrease in physical activity with higher leptin levels in this sample is not congruent with the expected relationship between leptin and physical activity. Based on known biological mechanisms, high leptin levels signal the brain that there is excessive energy intake, which results in decreased intake and increased energy expenditure³². However, the results indicate higher baseline leptin levels predicted a decline in physical activity. One possible reason for this unexpected result may be because of higher than normal leptin levels in our sample. Normal leptin levels for peri-pubertal normal weight girls averages 5.0 ng/mL³³, while this sample's average was 15.2 ng/mL. The higher than normal baseline leptin levels suggest that this sample may more prone to energy imbalance³⁴. Furthermore, girls are more prone to increasing fat mass at the initiation of puberty, which may act to raise leptin levels³¹ and determine a decrease in physical activity³⁵. The findings from this study support a biological basis for the age-related decline in physical activity.

This study has a number of limitations. This analysis included only baseline leptin concentrations and therefore prevented the assessment of the simultaneous change in leptin and physical activity over one year. Collecting leptin at more frequent intervals may help improve the ability to detect simultaneous changes in leptin and physical activity during puberty when hormone levels fluctuate. A majority of the sample (62.0%) was overweight or obese, and therefore these findings may not be generalizable to normal weight youth. Also, the small sample size precluded stratified analysis based on race/ethnic group, although African Americans recorded more physical activity than Latinas. Previous studies have reported that there are race/ethnic differences in physical activity levels⁹ and future studies should attempt to discern whether these differences are influenced by variations in leptin levels between groups. Also, while these accelerometers are an excellent device-based measure of physical activity in youth³⁶, they do not capture all types of activities (e.g.: swimming, load carrying) and may bias our physical activity estimates.

In conclusion, leptin levels promote a decline in physical activity over one year. In minority youth, girls are consistently less active than boys of all weight status categories⁹. Entering puberty with high leptin levels puts them at a greater disadvantage during the observed decline in physical activity that may have negative health consequences³⁷. To our knowledge this is the first study to find this relationship in a longitudinal model in minority girls. These findings add to the growing support for the biological basis of declining activity levels in pubertal girls and may explain in part why interventions to increase physical activity based solely on behavioral and psychosocial theories have shown little success to date. Future studies are needed to further investigate how biological mechanisms, such as leptin concentrations, impact physical activity throughout puberty in order to better understand how these mechanisms contribute to the age-related physical activity decline.

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What is already known about this subject

- Physical activity declines as children enter puberty
- Leptin is cross-sectionally associated with physical activity, but there are conflicting findings on the magnitude and direction of this association
- Leptin concentrations fluctuate during puberty, and may impact energy balance

What this study adds

- Leptin predicts the decline in physical activity during the start of puberty independent of central adiposity
- Based on a median split of leptin, girls with low leptin levels have higher levels of physical activity than girls with high leptin levels at the start of puberty
- Leptin levels at the start of puberty may provide a biological basis for the age-related physical activity decline in girls

Figure 1.

Mean MVPA (min/day) at each visit for the whole sample and by leptin category

MVPA= moderate to vigorous physical activity

* High versus low leptin groups significantly differ at visits 0, 1, 2 (p < .050)

Table 1

Baseline descriptive characteristics of the sample (n= 50)

Variable	Mean (SD) ^a
Latina (N (%))	39 (78.0)
Age (years)	9.4 (0.9)
Tanner Stage 1 (N (%))	26 (52.0)
BMI z-score	1.2 (1.0)
BMI	22.1 (4.6)
BMI percentile	81.4 (23.1)
Overweight (N (%))	12 (24.0)
Obese (N (%))	19 (38.0)
Body fat (%)	26.6 (10.9)
Insulin Sensitivity	3.23 (1.95)
SAAT (L)	4.46 (3.21)
VAT (L)	0.69 (0.60)
Mean MVPA (min/day)	31.0 (22.0)
25 th percentile	16.8
50 th percentile	25.8
75 th percentile	37.6

SD= standard deviation; BMI= body mass index; SAAT= subcutaneous abdominal adipose tissue; VAT= visceral adipose tissue; MVPA= moderate to vigorous physical activity

^aMean (SD) unless otherwise indicated

Table 2

Mean (SD) physical activity estimates for all visits

Variable	Visit			
	0	1	2	3
Mean wear time (hr/day)	13.7 (0.9)	13.9 (1.0)	13.9 (1.1)	14.3 (0.8)*
Mean cpm	442.3 (143.8)	429.1 (159.3)	448.5 (198.7)*	374.8 (125.3)
Mean MVPA (min/day)	31.0 (22.0)	32.6 (23.8)	31.8 (25.6)	25.7 (16.4)*
				27.1 (24.3)*

SD= standard deviation; cpm= counts per minute; MVPA= moderate to vigorous physical activity

* significantly different from visit 0 (p< .050)

Table 3

Baseline generalized linear model of the association between continuous leptin concentrations and MVPA (N=50)

Variable	MVPA ^a Model R ² = 0.39	
	β (SE)	p value
Intercept	4.80 (0.86)	< .001
Ethnicity (Latina vs. AA)	−0.53 (0.19)	.009
Age (years)	−0.13 (0.09)	.887
SAAT (L)	−0.03 (0.05)	.591
VAT (L)	0.20 (0.24)	.425
Leptin (ng/mL) ^a	−0.43 (0.12)	.001

MVPA= moderate to vigorous physical activity; SE= Standard Error; AA= African American; SAAT= subcutaneous abdominal adipose tissue; VAT= visceral adipose tissue.

^aMVPA and leptin values are log-transformed

Table 4

Longitudinal mixed model^a with baseline leptin predicting change in MVPA over all visits (n=50)

Baseline Variable	MVPA ^b			
	β	SE	t	p
Intercept	3.85	0.76	5.09	<.001
Visit ^c	0.72	-	-	.579
Ethnicity (vs. AA)	-0.43	0.16	-2.64	.012
Age (years)	0.06	0.08	0.83	.411
SAAT (L)	-0.01	0.04	-0.34	.734
VAT (L)	-0.08	0.21	-0.40	.692
Leptin (ng/mL) ^b	-0.36	0.11	-3.35	.002

MVPA= moderate to vigorous physical activity; SE= Standard Error; AA= African American; SAAT= subcutaneous abdominal adipose tissue; VAT= visceral adipose tissue.

^a model has a random intercept

^b MVPA and leptin values are log-transformed

^c F statistic with df= 4,91; visit has been coded as 0–4

Table of sensitivity analysis

Baseline Variable	MVPA ^a 4+ valid days of data N=50				MVPA ^a 1+ valid days of data N=59			
	β	SE	t	p	β	SE	t	p
Intercept	3.85	0.76	5.09	<.001	3.52	0.70	5.02	<.001
Visit ^b	0.72	-	-	.579	1.56	-	-	.189
Ethnicity (vs. AA)	-0.43	0.16	-2.64	.012	-0.30	0.16	-1.90	.063
Age (yrs)	0.06	0.08	0.83	.411	0.07	0.07	0.95	.347
SAAT (L)	-0.01	0.04	-0.34	.734	-0.02	0.04	-0.60	.552
VAT (L)	-0.08	0.21	-0.40	.692	-0.04	0.19	-0.20	.845
Leptin (ng/ml) ^a	-0.36	0.11	-3.35	.002	-0.30	0.10	-2.91	.005

SE is the standard error & model has a random intercept

MVPA= moderate to vigorous physical activity; SE= Standard Error; AA= African American; SAAT= subcutaneous abdominal adipose tissue; VAT= visceral adipose tissue.

^aMVPA and leptin values are log-transformed

^bF statistic with df= 4,91; visit has been coded as 0-4