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Examining the longitudinal relationship between change in sleep and obesity risk in adolescents

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Abstract

Evidence is building regarding the association between inadequate amounts of sleep and the risk of obesity, especially in younger children. Less is known about the relationship between change in sleep and change in weight during adolescence. The objective of this study was to examine the longitudinal relationship between change in sleep duration and change in body mass index (BMI) and percent body fat (PBF) in a cohort of adolescents. The cohort included 723 adolescents (mean age 14.7 years at baseline) from Minnesota. Total sleep duration was assessed via self-report. BMI and PBF were objectively assessed. Covariates used in the multivariate analyses included energy intake as assessed through 24-hour recalls, activity levels as assessed by accelerometers, screen time/sedentary behavior, depression and socio-demographic characteristics. For both males and females, average BMI and PBF increased slightly over the 2 years and average sleep duration decreased by about 30 minutes. We saw no statistically significant longitudinal relationships between change in total sleep and change in BMI or PBF over time in either girls or boys. The only longitudinal relationship that approached statistical significance was a positive association between sleep and PBF in females (p=.068). This research contributes to the literature as the only study to date to examine how change in sleep duration during adolescence may be related to a concomitant change in BMI and body fat. Our findings do not support the hypothesis that a decline in sleep duration during adolescence increases obesity risk.

Introduction

Public health practitioners and clinicians are searching for an understanding of the obesity epidemic that is affecting both adult and youth populations(Flegal, Carroll, Kit, & Ogden, 2012; Ogden, Carroll, Kit, & Flegal, 2012). Poor sleep has emerged as a potential risk factor in the development of childhood and adult obesity and the research suggesting an association between lower amounts of sleep and increased risk of overweight and obesity is growing (Cappuccio et al., 2008; X. Chen, Beydoun, & Wang, 2008; Patel & Hu, 2008). The mechanisms involved in the relationship between sleep and obesity are not clear. Taheri (2006) suggests that lower amounts of sleep may affect energy balance: being tired may reduce levels of physical activity and energy expenditure and being awake more hours in a day may provide more time to eat and increase energy intake. In addition, there is some thought that low levels of sleep may impact levels of leptin and ghrelin, two hormones

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involved in appetite regulation. The mechanisms for how sleep may impact energy balance and obesity risk are poorly understood,

> Sleep duration is one aspect of sleep as a behavior. Sleeping problems (including difficulty falling asleep or staying asleep) and irregular sleep patterns (including differences in patterns of waking and going to bed) have been examined as well (National Sleep Foundation, 2007). However, the research to date has focused on examining the relationships between sleep duration and obesity risk

Several reviews of the literature on sleep and obesity in youth have been published. Chen et al (2008)(X. Chen et al., 2008) conducted a meta-analysis on this topic using 17 studies. The results showed that for each additional hour of sleep the risk of overweight or obesity was reduced by 9%. However, the relationship between sleep and obesity risk was only seen in younger children (<10 year olds) and was much stronger in boys as compared to girls. Another literature review of the relationship between sleep and obesity risk in youth was conducted and confirmed the relationship between sleep and body mass index (BMI), especially through cross-sectional studies, in younger children and in boys(Patel & Hu, 2008). Cappuccio et al (2008) reported that in 7 out of 11 studies included in their meta analysis a significant association was seen between short duration of sleep and obesity.

While these findings are intriguing, there is still much to be learned about the relationship between sleep and risk for overweight and obesity in youth. The assessment of sleep in population-based studies of youth is often limited to a single question assessing how often a child “gets adequate sleep” or sleeps at least 6–8 hours on weekdays(M. Chen, Wang, & Jeng, 2006; Knutson, 2005; Reilly et al., 2005). In addition, there is little consistency with which potentially important covariates are examined. Reviewing the list of confounders presented in the Chen et al (2008)(X. Chen et al., 2008) meta-analysis reveals that beyond demographic variables the other potential confounding variables (specifically energy intake, energy expenditure, and pubertal status) were poorly measured, if measured at all. Additionally, across the 17 articles included in Chen et al (2008)(X. Chen et al., 2008), 8 articles included children ages 10 and older. In four of these studies(Chaput, Brunet, & Tremblay, 2006; Eisenmann, Ekkekakis, & Holmes, 2006; Gibson, Lambert, & Neate, 2004; Knutson, 2005) the relationship between sleep and overweight or obesity was seen in boys but not girls and in three studies(M. Chen et al., 2006; Knutson & Lauderdale, 2007; Seicean et al., 2007) the relationship appeared to be strong only when sleep duration was six hours or less.

Our research team published data examining the cross-sectional relationships between sleep duration and BMI, percent body fat, and overweight status in a cohort of 723 adolescents (mean age=14.7)(Lytle, Pasch, & Farbakhsh, 2011). We included caloric intake, activity levels and depression as covariates and examined grade level and gender as potential moderators. We saw an inverse relationship between sleep duration and BMI ($p<0.001$) in middle school males and females and an inverse relationship between sleep duration and percent body fat and overweight status in the middle school males. However, we saw little evidence of a cross-sectional relationship between any of the weight related variables and sleep in the males and females in high school(Lytle et al., 2011).

The strongest evidence for a causal relationship between low levels of sleep and obesity in youth comes from a limited number of prospective studies that have been conducted in very young children. To date, there have been seven published studies that examine the longitudinal relationship between sleep patterns and overweight/obesity risk in youth (Agras, Hammer, McNicholas, & Kraemer, 2004; Bell & Zimmerman, 2010; Carter, Taylor, Williams, & Taylor, 2011; Dieu, Dibley, Sibbritt, & Hanh, 2007; Landhuis, Poulton, Welch, & RJ, 2008; Reilly et al., 2005; Snell, Adam, & Duncan, 2007). The evidence to date

suggests that lower levels of sleep at a young age (ages 3–7) is related to greater risk of obesity at adolescence.

Adolescents have been identified as a population at risk for sleep-related problems. Sleep duration decreases as children move into adolescence and only 15% of adolescents report sleeping 8.5 or more hours nightly. In addition, irregular sleep patterns between weekday and weekend nights are well documented in adolescents(National Sleep Foundation, 2007). Some of that irregularity may be due to early start times for school, challenging the adolescent to wake earlier than their circadian rhythms would prefer(Wolfson, Spaulding, Dandrow, & EM, 2007). Only three of the longitudinal studies to date included adolescents in the sample(Bell & Zimmerman, 2010; Landhuis et al., 2008; Snell et al., 2007) and in two of those studies(Bell & Zimmerman, 2010; Snell et al., 2007) the relationship between early sleep duration and obesity risk was not confirmed. None of the longitudinal studies on sleep and weight in youth adjusted for depression (a psychosocial factor that gains importance in adolescence and may impact both sleep and obesity) (Herva et al., 2006; Perlis, Giles, Buysse, Tu, & Kupfer, 1997) and none adjusted for puberty, which also impacts body weight(Carskadon, Vieira, & Acebo, 1993; Dahl & Lewin, 2002).

To date, the question being asked and answered in these longitudinal studies is: “Does sleep at a younger age predict body mass index or obesity risk at an older age?” Previous longitudinal analyses used baseline sleep patterns to predict body mass index or risk of overweight at a subsequent follow-up period. While documenting the relationship between early risk exposure and a later health outcome is an important epidemiological question, for applied public health purposes it is also important to examine the potential effect of change in the risk factor over time on change in the health outcome over time. In other words: Is change in sleep duration over time related to change in obesity risk over time?

The purpose of this study was to examine the longitudinal relationship between change in sleep duration and change in body mass index (BMI) and percent body fat (PBF) in a cohort of adolescents that were followed over 24 months controlling for sociodemographic characteristics, puberty, age, energy intake, physical activity, screen time/sedentary behavior and depression. Because the literature(X. Chen et al., 2008; Snell et al., 2007) and our own previous data(Lytle et al., 2011) suggest gender differences in the relationship between sleep and obesity, we conducted separate analyses by gender.

Methods

The sample is from the IDEA (NIH 1U54 CA 116849) and ECHO study (NIH 5 R01 HL085978); both studies are etiologic, longitudinal studies examining factors that may be related to unhealthy weight gain in youth(Lytle, 2009). Identical measurement protocols allowed us to combine these two samples increasing our potential power for understanding relationships. For the IDEA study, 349 youth ages 10–16 and one significant adult in their life (usually a parent) were recruited from within a seven-county metropolitan area from Minneapolis, St. Paul, Minnesota in 2006–2007. Youth were invited to participate regardless of weight status and were recruited from: 1) an existing cohort of youth participating in the Minnesota Adolescent Community Cohort (MACC) Tobacco Study(Widome, Forster, Hannan, & Perry, 2007), 2) a Minnesota Department of Motor Vehicle (DMV) list restricted to the seven-county metro area, and 3) a convenience sample drawn from local communities. Three measurement periods were included for IDEA (baseline, 12 months and 24 months).

For the ECHO study, 374 youth and a parent were recruited from the membership of Health Partners (HP) health plan within the seven-county metropolitan area of Minneapolis, St. Paul, Minnesota between June 2007 and March 2008. We used a recruitment procedure that

targeted a range of overweight and healthy weight youth and parents and that oversampled minorities. To be eligible, youth were required to be current HP members, in grades 6 through 11 in the fall of 2007, residing in one of the randomly selected middle or high-school districts included in the sample, have a parent willing to participate and be willing to allow their names and contact information to be sent from HP to the study team at University of Minnesota for further eligibility screening, consent and measurement. Two measurement periods were included for ECHO (baseline and 24 months).

In both the IDEA and ECHO studies, youth were excluded if they expected to move from the area in the next three years, had a medical condition that affected their growth, had any other physical or emotional condition that would affect their diet or activity levels or make it difficult to complete measurements or were non-English speaking or otherwise had difficulty comprehending English. Study resources did not allow for translation of the data collection instruments or for translators to assist participants in completing data collection instruments. The human subjects committees at the University of Minnesota and Ohio State University approved the study.

Measures

Obesity-related measures—Each youth attended a clinic visit at each measurement period. After confirming consent and assent, clinic staff measured the height of each youth using a Shorr height board (Irwin Shorr, Olney, MD), and weight and body composition using a Tanita scale, a bioelectrical impedance device that assesses body weight, lean and fat mass (Tanita TBF-300A Body Composition Analyzer, Arlington Heights, IL). While body fat assessment using bioelectrical impedance may vary by time of day and hydration levels, validation of bioelectrical impedance has occurred using hydrodensitometry as the criterion standard(Lukaski, Bolonchuk, Hall, & Siders, 1986). These assessments were used to calculate body mass index and percent body fat.

Student survey measures—At the clinic visit, each youth completed a survey. The following measures were taken from the survey.

Sleep duration: Sleep duration was assessed by asking the youth “what time they usually go to bed in the evening (i.e., turned out the lights in order to go to sleep) and “usually get out of bed in the morning” on a typical weekday and weekend. These items, adapted from the Night Eating Questionnaire, a validated instrument widely used in the field for assessing self-report sleep (Gluck, Geliebter, & Satov, 2001), were used to compute average total hours of daily sleep. Similar sleep questions have been used successfully with adolescents in previous studies(Laberge et al., 2001; Lee, McEnany, & Weekes, 1999; Wolfson, 1996).

Depression Measure: Depressive symptomology has been documented in adolescents and been found to be related to BMI(Herva et al., 2006; Kubik, Lytle, & Birnbaum, 2003). Depressive symptoms were measured using the 6-item Kandel-Davies scale (1982)(Kandel & Davies, 1982). These questions asked the students to respond “not at all,” “somewhat,” or “very much” to questions that included the extent to which, in the past twelve months, they felt bothered or troubled by feeling too tired to do things, feeling hopeless about the future, and felt sad, unhappy or depressed (range= 7–21). The Cronbach’s alpha for this scale was 0.78.

Screen time/sedentary behavior: Reilly(2005) and Crespo(2001) have documented a positive relationship between screen time and obesity risk in youth. Students were asked to estimate how many hours they spend each weekend and weekday on the following behaviors: watching television, watching DVDs or videos, reading/homework, playing

Nintendo/Play station/computer games; using the internet/computer; talking on the phone/ text messaging. Average daily screen time/sedentary behavior was computed.

Energy intake—Each cohort member was asked to participate in three telephone-administered 24-hour dietary recalls that were collected using the Nutrition Data Systems (NDS) software. Two recalls on weekdays and one recall on a weekend were obtained from each cohort member. The average number of kilocalories across the recalls (Average daily calories) was computed.

Physical activity—Each cohort member was asked to wear an accelerometer for 7 days, removing the device only when sleeping, showering, swimming, and at the request of a coach or teacher. The ActiGraph accelerometer, model 7164 (ActiGraph, LLC, Pensacola, FL) collected physical activity data using 30-second epochs (data collection intervals). The monitor is an objective measure of physical activity and has been previously validated for use with children in laboratory and field settings(Eston, Rowlands, & Ingledew, 1998; Louie et al., 1999; Trost et al., 1998). Accelerometry data were reduced using methods previously described(Catellier et al., 2005; Treuth et al., 2004). This method of imputation was used in the Trial of Activity in Adolescent Girls where physical activity was the primary outcome(Webber et al., 2008)(Stevens et al., 2005). Briefly, missing accelerometry data were replaced via imputation(Dempster, Laird, & Rubin, 1977). On average, approximately 25.5 hours of data (about 24.8%) for the IDEA sample and 23.3 hours of data (about 22.9%) for the ECHO sample per adolescent were imputed over all 7 days of data collection. Total physical activity was defined as the sum of light, moderate, and vigorous activity.

Demographic and pubertal measures—Data on socioeconomic status were obtained from the parent survey, and included the highest level of education for the parents who resided in the house and if the child qualified for free and reduced lunch at school. Race was reported by the child on the student survey. Because pubertal status is related to weight change, youth were asked to complete a 7- item self-report puberty scale(Peterson, Crockett, Richards, & Boxer, 1988).

Analysis Methods

The exposure variable was average total sleep duration combining average hours of sleep on both weekends and weekdays. For each participant, we calculated the mean across the two (ECHO) or three (IDEA) measurement periods; in addition, we calculated the deviation between the value observed at a given measurement period and that participant's mean. Both the mean and deviation scores were recorded on each measurement period record and included in a given regression analysis. The coefficient for the deviation score estimated the longitudinal relationship between sleep and weight; specifically, the change in the outcome within a youth who increased their sleep by one hour per night. We have used this decomposition scheme in previous papers related to obesity(Sherwood, Jeffery, French, Hannan, & Murray, 2000; Stevens et al., 2007) and this approach is discussed in a text on longitudinal data analysis(Singer & Willett, 2003). Gender was coded 1=male, 0=female. Race was coded 1=white, 0=other. Grade was coded 1=>=9th grade, 0=<9th grade. Parent's highest level of education, asked of the parent, was coded 1=college graduate, 0=other. School lunch, a measure of socioeconomic status, was coded 1= received free and reduced lunch program, 0=other. Puberty, depression, screen time/sedentary behavior, activity, and calories were modeled as continuous variables.

All analyses were conducted separately for males and females. To facilitate interpretation, all variables except the deviation scores were centered prior to analysis by subtracting the

gender-specific mean from each observed value. That was not necessary for the deviation scores, as they had a mean of zero by definition.

Random coefficient models were used to examine the relationship between sleep and BMI and PBF over time; these models fit a random slope and intercept for each participant (Murray, 1998; Raudenbush & Bryk, 2002; Singer & Willett, 2003). Consistent with the recommendations of Singer & Willett(2003), we used age as the index for time. We ran separate models for each dependent variable (BMI, PBF). In each model, we adjusted for race, grade, parent education, free/reduced school lunch, puberty, and study (IDEA vs. ECHO). We ran a second model for each dependent variable that also included depression, screen time/sedentary behavior, calories, and activity. In each model, we included age, the mean and deviation sleep variables, and their interactions. The interactions provided a test of whether the longitudinal relationship between sleep and the outcome depended on the age of the youth. We also fit parallel sets of models that excluded those interactions; those models provided a test of whether there were longitudinal associations and assumed that any such associations applied equally to older and younger participants. We used empirical sandwich standard errors to accommodate the complex pattern of correlation in the data due to the nesting of youth within schools and neighborhoods and to the nesting of repeat observations within youth.

Descriptive statistics were prepared using SAS PROC MEANS and SAS PROC FREQ. Regression models were run in SAS PROC MIXED. All analyses were run in SAS Version 9.1(*SAS/STAT 9.1 User's Guide*, 2004).

Results

Table 1 shows characteristics of the sample at each measurement period, stratified by gender. The total baseline sample of 723 was reduced only slightly at the two-year follow-up to a sample of 648, a 90% retention rate. For both males and females, BMI and PBF increased slightly over the two years of follow-up. For males, the mean increase in BMI and PBF was 1.62 (SD=1.8) and 0.58 (SD= 4.6), respectively. For females, the mean increase in BMI and PBF was 1.32 (SD= 1.8) and 1.77 (SD = 4.5), respectively. For males, total hours of sleep dropped from 8.7 hours to 8.4 hours across the two years of follow-up; females also saw a decline in average total sleep from 8.6 to 8.4 hours.

Table 2 presents the longitudinal associations between total sleep and BMI and PBF, with and without adjustment for screen time/sedentary behavior, depression, activity, and energy intake. We saw no statistically significant longitudinal relationships between total sleep and BMI or PBF in either males or females. The only longitudinal relationship that approached statistical significance was between sleep and PBF in females. Without adjustment for calories, activity, screen time/sedentary behavior, or depression, female participants whose sleep increased by one hour per night had an average increase in PBF of 0.268 units, or 0.268 percentage points (p= 0.068); however, after also adjusting for calories, activity, screen time/sedentary behavior, and depression, that relationship was attenuated slightly (0.259%) and no longer significant (p=0.253). We saw no evidence of an interaction between age and the sleep deviation scores in the longitudinal analysis, suggesting that the relationships were the same in both the younger and older youth in our sample.

Discussion

This research adds to the growing evidence on the relationship between sleep and obesity in youth. This research focused on adolescents, examined both BMI and PBF as outcomes, and explored the differences in relationships by gender. We report the effect of two levels of

adjustment, one that included only puberty and demographics and a second that also included other covariates that could serve as alternative explanations for an association between sleep and obesity. Importantly, the research examined if change in sleep duration over time was related to change in BMI or PBF over time. Although average sleep duration decreased over the 24 months in both males and females, that decrease in sleep was not associated with a statistically significant increase in BMI or PBF, controlling for socio-demographic and energy-related covariates.

The evidence from empirical research, especially longitudinal studies, is showing that the association between sleep and obesity risk is moderated by age, with lack of sleep in young children being an important risk factor for subsequent obesity. The evidence for the association in adolescents and adults is mixed. Snell (2007) followed a cohort that included adolescents for five years and found that sleep at baseline was only marginally related to BMI five years later. Bell and Zimmerman (2010) saw a similar pattern in the cohort that transitioned into adolescence during their 5 year longitudinal study. Landhuis et al (2008) examined a birth cohort from New Zealand and compared parental reports of sleep time at ages 5,7,9 and 11 with BMI measured when the cohort was 32; greater childhood sleep time predicted lower odds of adult obesity. None of these studies investigated if change in sleep duration predicted change in obesity risk during the adolescent period.

In adults, evidence of the longitudinal relationships between sleep and weight has also been mixed. Hasler (2004)(Hasler et al., 2004), Gangwisch (2005)(Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005) and Patel (2006)(Patel, Malhotra, White, Gottlieb, & Hu, 2006) reported an inverse relationship between sleep duration and future weight, but those studies have limitations including limited external validity, use of self-reported weight and assessment of sleep at only one time point(Chaput et al., 2006). Chaput (2006) examined the longitudinal relationship between sleep duration and subsequent body weight and weight gain in 276 adults over 6 years and found a U-shaped relationship: the risk of developing obesity was greater in adults who slept the most (9–10 hours) and the least (5–6 hours). The greatest benefit for healthy weight was seen in the group who, at baseline, slept 7–8 hours per night. Analysis of CARDIA data(Lauderdale et al., 2009) examined both cross-sectional and longitudinal associations between sleep and BMI in a sample of 612 adults with a mean age of 45.2. In the cross-sectional data an inverse association was seen between sleep duration and BMI; however, no longitudinal associations were found between the sleep measures and BMI.

To our knowledge, this is the first study to examine how change in sleep duration over time may be related to change in weight over time. Previous longitudinal studies in both youth and adults examined how baseline sleep predicted future risk of obesity. The distinction is important from a public health and clinical practice standpoint. If early sleep patterns predict future obesity, it suggests that inadequate sleep duration during early childhood may establish a risk for overweight and obesity that is fairly immutable remaining a part of the individual’s metabolic make-up. In this case, public health efforts should focus on parents and care providers of infants and young children with the message that developing good sleep patterns early in life is essential for a healthy weight as well as other physical and emotional outcomes.

A change in sleep duration that occurs over time that is related to a concomitant change in body weight or body fat over time might suggest that metabolic, hormonal, or behavioral factors are changing and impacting both sleep and weight. The corresponding public health or clinical message to adolescents would be to try to get more sleep in order to decrease obesity risk. Our findings did not support this hypothesis. Marshall and colleagues (2008) (Marshall, Glozier, & Grunstein, 2008) explore the importance of change in sleep duration

and obesity risk, asking if a consistent and adequate evidence base exists that would justify promoting increasing sleep duration as an intervention for obesity or prevention of weight gain. They note that no observational study has shown that change in habitual sleep duration is associated with weight change in adults. Although getting adequate sleep has many positive benefits for adolescents and adults (National Sleep Foundation, 2005), obesity prevention may not be one of them and should not be included in the rationale for getting adequate sleep until further research is done.

The limitations of this study include the use of self-report assessments of sleep duration. However, the preponderance of the current research examining the relationship between sleep and weight in youth is based on self-report. Another limitation is the relatively short amount of time that we followed the cohort. Additional measurement points following the youth into young adulthood may reveal relationships not seen in the current data. A third limitation is that our research was limited to a single metropolitan area, and while efforts were made to obtain as much heterogeneity as possible in the sociodemographic profile of the same, our results may still not apply to more diverse populations.

Limited variability in the change in BMI, PBF, or total sleep could stand as an alternative explanation for our finding of no longitudinal association between change in sleep and change in either BMI or PBF. To explore this possibility, we examined the variability in change over two years observed in our data. The mean, sd, and range for change in BMI were 1.64 BMI units (1.83, −4.62 to 8.07) for males and 1.32 BMI units (1.83, −7.61, 8.99) for females. The mean, sd, and range for change in PBF were 0.57% (4.65, −15.8 to 17.9) for males and 1.79% (4.54, −12.4 to 14.4) for females. The mean, sd, and range for change in total sleep were −0.20 hours/day (1.14, −3.85 to 4.07) for males and −0.19 hours/day (1.13, −3.71 to 3.25) for females. Given the range of change in each of the 3 measures, we think it unlikely that limited variability in change is responsible for our finding of no longitudinal association between change in total sleep and change in BMI or PBF.

This research contributes to the literature as the only study to date to examine how change in sleep may impact change in weight over time. Importantly, we examine this question in a sample of adolescents who are experiencing the typical reduction in sleep known to occur during the adolescent period. In addition, potentially important confounders of the relationship including estimates of energy intake and expenditure, screen time/sedentary behavior, depressive symptoms, socioeconomic status, race and pubertal status were all included in the models. Additional research in this area is warranted with larger and more diverse samples, particularly focusing on the mechanisms related to sleep and weight in youth and considering possible mediating variables on the suspected causal pathway between sleep and obesity risk.

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Table 1

Characteristics of the sample at each period of data collection.*

Males	**Baseline, N=353**		**1 Year Follow-up, N=161***		**2 Year Follow-up, N=320**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
age (yrs)	14.7	1.84	16.4	1.68	16.6	1.84
bmi (mean)	22.2	5.12	22.6	4.39	23.7	5.39
pbf (%)	16.3	9.66	14.4	7.13	16.6	9.28
total sleep (hrs)	8.66	1.04	8.33	1.09	8.44	1.05
energy intake (kcal)**	2194	672	NA	NA	2249	723
depression (mean)	14.9	3.65	14.9	3.30	15.11	3.86
puberty (mean)	2.59	0.67	3.13	0.60	3.28	0.63
activity (min/d)**	320.8	73.8	NA	NA	305.3	64.3
screen time (min/d)	346	232	362	236	350	226
	%		%		%	
white	85.8%		93.2%		88.8%	
grade 9–12	51.0%		83.2%		84.7%	
parent college graduate	76.9%		80.1%		79.6%	
free or reduced lunch	11.0%		5.59%		7.50%	
Females	Baseline, N=370		1 Year Follow-up, N=171*		2 Year Follow-up, N=328	
	Mean	SD	Mean	SD	Mean	SD
age (yrs)	14.7	1.83	16.3	1.72	16.6	1.88
bmi (mean)	22.0	5.01	22.3	4.61	22.9	4.82
pbf (%)	26.4	8.76	26.1	7.71	27.6	8.01
total sleep (hrs)	8.64	1.02	8.30	1.01	8.45	1.06
energy intake (kcal)**	1776	523	NA	NA	1777	506
depression (mean)	16.3	4.27	16.7	4.23	16.8	4.45
puberty (mean)	3.20	0.68	3.52	0.49	3.62	0.47
activity (min/d)**	299	63.1	NA	NA	280	52.0
screen time (min/d)	290	212	261	186	293	190

Males	Baseline, N=353		1 Year Follow-up, N=161*		2 Year Follow-up, N=320	
	Mean	SD	Mean	SD	Mean	SD
	%		%		%	
white	83.5%		93.6%		85.4%	
grade 9–12	55.9%		82.5%		82.0%	
parent college graduate	72.0%		74.0%		74.9%	
free or reduced lunch	12.7%		8.19%		8.81%	

* One year follow-up data were collected in IDEA only; baseline and two year follow-up data were collected in both studies.

** Energy intake and activity were not assessed at one year follow-up in IDEA

Table 2

Longitudinal associations between total sleep and body mass index (BMI) and percent body fat (PBF) by gender

		Coefficient	Longitudinal Association SE	p-value
Males (n=320): adjusting for demographic characteristics only*	BMI	−0.051	0.065	0.430
	PBF	0.113	0.189	0.552
Males (n=320): adjusting for demographic characteristics, depression and behaviors**	BMI	−0.091	0.110	0.410
	PBF	0.105	0.306	0.731
Females (n=328): adjusting for demographic characteristics only*	BMI	0.063	0.058	0.276
	PBF	0.268	0.147	0.068
Females (n=328): adjusting for demographic characteristics, depression and behaviors**	BMI	0.030	0.094	0.754
	PBF	0.259	0.226	0.253

*Adjusted for Race, grade, parent education, school lunch, puberty and study (IDEA vs. ECHO)

**Adjusted for demographics plus: screen time, depression, activity and energy intake