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Associations of Sleep Duration and Quality with Disinhibited Eating Behaviors in Adolescent Girls At-Risk for Type 2 Diabetes

Nichole R. Kelly, PhD^{1,2}, Lauren B. Shomaker, PhD^{1,3,*}, Rachel M. Radin, MS^{1,2}, Katherine A. Thompson, BA¹, Omni L. Cassidy, MS^{1,2}, Sheila Brady, MSN, CFNP¹, Rim Mehari, BA¹, Amber B. Courville, PhD, RD⁴, Kong Y. Chen, PhD⁵, Ovidiu A. Galescu, MD¹, Marian Tanofsky-Kraff, PhD^{1,2}, and Jack A. Yanovski, MD, PhD¹

¹Section on Growth and Obesity, National Institute of Child Health and Human Development (NICHD), National Institutes of Health (NIH), Department of Health and Human Services (DHHS), 10 Center Drive, MSC 1103, Bethesda, MD 20892

²Department of Medical and Clinical Psychology, Uniformed Services University of the Health Sciences, Department of Defense, 4301 Jones Bridge Road, Bethesda, Maryland 20814

³Department of Human Development and Family Studies, Colorado State University, 410 Pitkin Street, Campus Delivery 1570, Fort Collins, Colorado 80523

⁴Nutrition Department, NIH Clinical Center, DHHS, 10 Center Drive, MSC 1078, Bethesda, Maryland 20892

⁵Diabetes, Endocrinology, and Obesity Branch, National Institute of Diabetes and Digestive and Kidney Diseases, NIH, DHHS, 10 Center Drive, Bethesda, Maryland 20814

Abstract

Objectives—Short sleep duration and daytime sleepiness have been associated with an increased risk for the onset of type 2 diabetes in adults. There has been far less attention to the characterization of sleep in adolescents at-risk for diabetes or to the possible behavioral mechanisms, such as disinhibited eating, through which sleep may affect metabolic functioning.

Methods—We evaluated the associations of sleep duration and daytime sleepiness with a multimodal assessment of disinhibited eating in 119 adolescent girls at-risk for type 2 diabetes based upon being overweight/obese and having a family history of diabetes. Girls also endorsed mild-to-moderate depressive symptoms. Adolescents reported sleep duration and daytime sleepiness with the Sleep Habits Survey and Children's Sleep Habits Questionnaire. They were administered a series of successive test meals to measure total energy intake and eating in the absence of hunger (EAH). Adolescent binge eating was assessed with the Eating Disorder Examination interview.

Results—Accounting for age, race, puberty, body composition, depressive symptoms, and perceived stress, reported sleep duration was positively related to test meal total energy intake (p

*Correspondence to: Lauren B. Shomaker, Ph.D.; Present address: Colorado State University, Department of Human Development and Family Studies, 410 Pitkin Street, Campus Delivery 1570, Fort Collins, Colorado 80523. Lauren.Shomaker@colostate.edu.

= .04), but not to EAH. Adjusting for the same covariates, daytime sleepiness was associated with a greater odds of objective binge eating in the previous month ($p = .009$).

Conclusions—In adolescent girls at-risk for type 2 diabetes, reported sleep characteristics are associated with disinhibited eating behaviors that have been linked to excessive weight and adverse metabolic outcomes. Future studies are called for to evaluate these links using objective measures of sleep.

Keywords

Binge eating; Eating in the absence of hunger; Obesity; Sleep; Sleepiness

Consistent with extant data in adults (Cappuccio, D'Elia, Strazzullo, & Miller, 2010), accumulating evidence suggests that sleep may be a modifiable behavioral factor associated with obesity and type 2 diabetes risk among youth. Pediatric studies have identified significant associations between short sleep duration and higher adiposity among healthy youth of various weight strata (Bel et al., 2013; Hart, Cairns, & Jelalian, 2011; Navarro-Solera et al., 2015), as well as higher insulin resistance (Matthews, Dahl, Owens, Lee, & Hall, 2012), lower insulin secretion (Flint et al., 2007; Zhu et al., 2015), and hyperglycemia (Koren et al., 2011) among youth with obesity, all independent of adiposity. Subjective complaints of daytime sleepiness also have been associated with higher body and fat mass among youth (Calhoun et al., 2011; Gaina et al., 2007), even, in some cases, independent of sleep duration (Jarrin, McGrath, & Drake, 2013). Although links between daytime sleepiness and metabolic functioning have not been well-examined in pediatric samples, data from adults indicate that poor sleep quality, including daytime sleepiness, is associated with higher insulin resistance (Pyykkönen et al., 2012) and greater risk for type 2 diabetes (Kita et al., 2012), even after adjusting for body mass index (BMI). While emerging evidence indicates that shortened sleep duration and daytime sleepiness may be associated with a higher risk for excess weight gain and type 2 diabetes, the explanatory factors that account for the relationship of sleep with these adverse outcomes are poorly understood.

One strong possibility is that alterations in energy intake, which occur in response to inadequate sleep and daytime sleepiness, may be explanatory. In investigations of adults, experimentally-induced sleep loss has been associated with alterations in several physiological processes that may facilitate disinhibited eating, referring to behaviors characterized by a lack of self-regulation over intake, such as overeating, eating in the absence of hunger (EAH), and binge eating. For instance, alterations in appetitive hormones are common after sleep deprivation (Schmid, Hallschmid, Jauch-Chara, Born, & Schultes, 2008; Spiegel, Tasali, Penev, & Van Cauter, 2004) and may trigger disinhibited eating. Short sleep duration also prompts alterations in circadian schedules (Barion & Zee, 2007), which have been related to adults' disinhibited eating in the evening (Nedeltcheva et al., 2009). Additionally, excessive daytime sleepiness may be an indicator of inadequate slow wave sleep (Fernandez-Mendoza et al., 2015). In turn, inadequate slow wave sleep has demonstrated a positive association with carbohydrate and fat consumption among adults (Shechter et al., 2012).

A small body of available data in youth largely has been consistent with adult studies. In a randomized cross-over study, adolescents with and without overweight reported consuming significantly more total energy (Simon, Field, Miller, DiFrancesco, & Beebe, 2015) and servings of sweet/dessert foods (Beebe et al., 2013) following a 5-day sleep restriction condition compared to a 5-day lengthier sleep condition. Similarly, children (27% with overweight) asked to increase sleep by 1.5 hours for one week reported less total daily energy intake relative to a week when they decreased their sleep by the same amount (Hart et al., 2013). Further, self-reported short sleep duration (Bel et al., 2013; Kjeldsen et al., 2014; Weiss et al., 2010; Westerlund, Ray, & Roos, 2009) and daytime sleepiness (Westerlund et al., 2009) have been associated with lower self-reported diet quality, even after accounting for body composition, screen time, and physical activity. Measured continuously, sleep duration has been inversely associated with self-reported total energy intake among children (Firouzi, Poh, Ismail, & Sadeghilar, 2014; Golley, Maher, Matricciani, & Olds, 2013). By contrast, some studies have found the reverse effect. In adolescent boys with normal weight, three days of lengthier sleep increased observed energy intake and motivation to eat, compared to three days of sleep restriction (Klingenberg et al., 2012). Additional research evaluating the associations of sleep duration and daytime sleepiness with a multi-modal assessment of disinhibited eating, including the use of objective laboratory test meal studies, may help to clarify the nature of the associations between sleep dimensions and eating behavior.

In addition, there has been little attention to other, more specific forms of disinhibited eating behaviors associated with excess adiposity, such as EAH (Shomaker, Tanofsky-Kraff, Zocca, et al., 2010). EAH refers to the consumption of palatable foods in the absence of perceived physiological hunger (Kral & Faith, 2007). One study identified a significant inverse association between objectively-measured sleep duration and parent-reported EAH in response to external cues among young children, independent of children's BMI (Burt, Dube, Thibault, & Gruber, 2014). Binge eating, or the perception of losing control while consuming an objectively large amount of food (American Psychiatric Association, 2013), is another form of disinhibited eating that predicts excessive weight and fat gain in children and adolescents (Field et al., 2003; Sonnevile et al., 2013; Tanofsky-Kraff et al., 2006). Data from adult samples suggest that the associations of short sleep duration and poor sleep quality with adiposity may be explained by disinhibited eating behaviors such as binge eating (Chaput, Després, Bouchard, & Tremblay, 2011; Yeh & Brown, 2014). However, only one study examined the relationship between sleep characteristics and binge eating among youth. In this sample, children with obesity and binge eating had objectively worse sleep quality relative to children with obesity without binge eating (Tzischinsky & Latzer, 2006). Importantly, group differences may have been confounded by BMI, which was not statistically accounted for despite being significantly higher in the youth with binge eating. As such, carefully controlled evaluations of sleep characteristics and various disinhibited eating patterns are needed, particularly among adolescents, a developmental period during which obesogenic eating patterns may be particularly salient (Vannucci et al., 2014).

We therefore evaluated the associations of reported sleep duration and daytime sleepiness with disinhibited eating. It was hypothesized that lower reported sleep duration and higher daytime sleepiness would be associated with greater observed energy intake, including EAH,

and greater odds of binge eating. In all analyses, we adjusted for depressive symptoms and perceived stress, because prior studies indicate that associations between sleep and excess energy intake may be confounded by global mental health symptoms (Calamaro et al., 2010). All associations were evaluated in a sample of adolescent girls at elevated risk for type 2 diabetes.

Methods

Participants

The current study is a secondary analysis of baseline data from participants enrolled in a behavioral clinical trial for the prevention of type 2 diabetes (ClinicalTrials.gov: NCT01425905). All participants were adolescent girls (12–17 years) deemed at-risk for type 2 diabetes, as determined by being overweight or obese (≥ 85th BMI percentile for age and sex) and having a first- or second-degree relative with type 2 diabetes, gestational diabetes, or prediabetes. Additional eligibility criteria for study participation included having mild-to-moderate depressive symptoms, as indicated by a score of ≥ 16 on the Center for Epidemiologic Studies-Depression Scale (Roberts, Lewinsohn, & Seeley, 1991). Girls were excluded from participating if they had a major medical condition, including type 2 diabetes (fasting glucose level > 126 mg/dL or 2-hour glucose after an oral glucose administration > 200 mg/dL), psychiatric symptoms requiring immediate treatment referral, or major depressive disorder (American Psychiatric Association, 2013). Presence of psychiatric diagnosis was determined via the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Kaufman et al., 1997), a valid and reliable semi-structured interview for youth (Ambrosini, 2000). Other exclusion criteria included pregnancy, participation in a structured weight loss or psychotherapy program, or use of medication that could affect mood or eating behavior. Recruitment efforts included direct mailings, local area community postings, and flyers to physician offices. All procedures were approved by the Institutional Review Board (IRB) of the *Eunice Kennedy Shriver* NICHD.

Procedures and Measures

For the purposes of the current study, participants were studied at baseline, prior to the initiation of treatment, and after a parent/guardian and the participant signed IRB-approved consent and assent forms. All study visits were outpatient assessments completed at the NIH Mark O. Hatfield Clinical Center (Bethesda, Maryland) following an overnight fast.

Sleep Duration—Sleep duration was ascertained from a single item on the *Children's Sleep Habits Questionnaire* (Owens, Spirito, & McGuinn, 2000). This item asks youth to report, in hours and minutes, how much sleep they typically obtain on an average school/weekday night. Single items of habitual school/weekday sleep shows reasonable concurrent validity with actigraphy and diary data (Wolfson et al., 2003). Weekday (versus weekend) sleep was selected for the current study to facilitate comparisons across studies among adolescents. Consistent with prior research (Calamaro et al., 2010), participants who reported receiving less than two hours of sleep were re-coded as receiving two hours.

Daytime Sleepiness—Daytime sleepiness was evaluated with adolescents' report on the *Sleep Habits Survey* (Wolfson et al., 2003), a survey of general sleep patterns in the previous two weeks. Average scores from the 10-item Sleepiness subscale were calculated. This subscale measures perceived difficulties staying awake during daytime activities and has demonstrated good internal consistency in a large sample of adolescents (Wolfson & Carskadon, 1998).

Objective Energy Intake—Objective energy intake when hungry and eating in the absence of hunger (EAH) were observed during two successive test meals. At approximately 12:00pm, each participant was served a large food array (> 10,000 kcal), varied in macronutrients (55% carbohydrate, 12% protein, 33% fat) and comprised of foods that most adolescents like (Shomaker, Tanofsky-Kraff, Zocca, et al., 2010). The participant was instructed to “please eat until you are no longer hungry; take as much time as you need.” Average ratings of state hunger and fullness completed immediately before ($M \pm SD$ hunger = 66.6 ± 26.0 , M fullness = 8.5 ± 16.0 ; range 0–100) and after (M hunger = 3.1 ± 12.4 , M fullness = 76.8 ± 24.2 ; range 0–100) lunch indicated that this initial test meal was effective in inducing satiety among participants. Then, from approximately 1:00 to 1:15pm, adolescents were served an array of highly palatable snack foods (> 4,000 kcal) to measure EAH (Shomaker, Tanofsky-Kraff, Zocca, et al., 2010). Participants were instructed to taste each food item and rate how much they liked each item. After providing these ratings, participants were permitted to consume as much of the snack foods as they desired. All food items in each test meal paradigm were weighed to the nearest 0.1 g before and after consumption. Energy intakes (kcal) were calculated using the USDA National Nutrient Database for Standard Reference (Agricultural Research Service, Beltsville, Maryland, USA) and the manufacturers' nutrient information obtained from food labels.

Binge Eating—Objective binge eating was assessed with the overeating section of the Eating Disorder Examination (EDE) semi-structured interview, version 12.0D (Fairburn & Cooper, 1993). Binge eating was coded categorically as absent or present in the previous month. The EDE was administered to each participant by a trained interviewer. In prior adolescent samples, the EDE has demonstrated strong inter-rater reliability and discriminant validity for binge eating (Glasofer et al., 2007; Vannucci et al., 2013).

Body Measurements—All youth underwent a medical history and a physical examination, conducted by either an endocrinologist or nurse practitioner. Pubertal development was assigned by Tanner of the breast (Marshall & Tanner, 1969) based on both inspection and palpation, so as to discriminate lipomastia from true breast tissue (Bonat, Pathomvanich, Keil, Field, & Yanovski, 2002). Weight was measured using a calibrated scale and height was measured in triplicate by stadiometer. BMIz scores were derived from the Centers for Disease Control and Prevention 2000 growth charts (CDC, 2000). Body fat and total lean mass were derived from dual-energy x-ray absorptiometry (iDXA, GE Healthcare, Madison, WI).

Psychosocial Measurements—Depressive symptoms were assessed using the Center for Epidemiologic Studies-Depression Scale (Radloff, 1977). This measure has

demonstrated acceptable reliability and validity in adolescents (Radloff, 1991). Perceived stress was examined using the reliable and widely used Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983). The psychometric properties of this measure are acceptable for use with adolescents (Mahon, Yarcheski, Yarcheski, & Hanks, 2007).

Statistical Analyses

Statistical analyses were performed with SPSS 22.0 (IBM Corp, 2013). All variables approximated a normal distribution. Missing data were minimal (< 5%) and listwise deletion was employed. Bivariate correlations were used to evaluate the unadjusted associations among variables of interest, including reported sleep duration (min) and daytime sleepiness, test meal energy intake (kcal), test meal EAH (kcal), BMIz, fat mass (kg), depressive symptoms, and perceived stress. Independent samples *t*-tests were used to examine whether participants with or without binge eating in the previous month differed on self-reported sleep duration and daytime sleepiness without adjusting for relevant covariates. Hierarchical multiple linear regression models were employed to evaluate the association of sleep characteristics with disinhibited eating behaviors measured continuously after controlling for age, race (Cassidy et al., 2012), height, puberty (Shomaker, Tanofsky-Kraff, Savastano, et al., 2010), fat mass (arcsine transformed), lean mass (kg) (Goulding, Taylor, Gold, & Lewis-Barned, 1996), depressive symptoms, and perceived stress (Calamaro et al., 2010). Analyses examining observed EAH also controlled for total energy intake at the lunch test meal (Shomaker, Tanofsky-Kraff, Zocca, et al., 2010). Binary logistic regression models examined adjusted associations of self-reported sleep duration and daytime sleepiness with the odds of binge eating presence. Differences between groups were considered significant when *p* values were < .05. All tests were 2-tailed.

Results

Sample Characteristics

Participants were 119 girls with an average age of 14.5 ± 1.6 (SD) years and an average BMIz score of 2.0 ± 0.5 . The racial/ethnic composition of the sample was 62% non-Hispanic Black/African American, 16% non-Hispanic White, 11% Hispanic, 8% Multiple Races, and 3% Asian. The vast majority (90%) were in late puberty (Tanner stages 4 or 5). Table 1 displays sample characteristics of key study variables. Participants reported sleeping 7.0 ± 1.7 hours per night (range = 2.0–10.0) during the week; 13% reported obtaining the minimum hours of recommended sleep (9 hours or more) (Hirshkowitz et al., 2015) on an average school/weekday night.

Associations between Sleep and Disinhibited Eating

Objective Energy Intake—In unadjusted analyses, there was no significant association between self-reported sleep characteristics and energy intake during the lunch buffet meal ($ps > .37$). In analyses accounting for age, race, height, puberty, fat mass, lean mass, depressive symptoms and perceived stress, self-reported sleep duration was positively associated with objective energy intake ($p = .03$; Table 2), such that girls who reported more sleep consumed more energy during the lunch meal (Figure 1). Specifically, for every additional minute of sleep participants' reported, they consumed 1.39 kcal more during

lunch (or, for every hour, 83.4 more kcal). In adjusted analyses, the association between subjective daytime sleepiness and lunch meal energy intake was not significant ($p = .87$). Follow-up analyses were conducted to examine potential associations of self-reported sleep duration and daytime sleepiness with macronutrient, dessert, and snack food intake during the lunch array; none of these analyses were statistically significant ($ps > .16$).

Neither reported sleep duration nor daytime sleepiness related to test meal EAH in either unadjusted analyses ($ps > .54$) or analyses accounting for age, race, height, puberty, fat mass, lean mass, depressive symptoms and perceived stress ($ps > .79$; Table 3).

Binge Eating—In unadjusted analyses, the association between self-reported average school/weekday sleep duration and binge eating status was non-significant, $t(112) = 0.62$, $p = .54$ (M binge group = 407.19 ± 123.56 min; M non-binge group = 423.65 ± 94.96 min). In contrast, subjective daytime sleepiness was associated with binge eating status, $t(112) = -2.93$, $p = .005$; girls who endorsed binge eating in the previous month reported greater daytime sleepiness ($M = 2.10 \pm 0.58$) relative to girls who did not report binge eating ($M = 1.66 \pm 0.40$). In logistic regression analyses adjusting for age, race, height, puberty, fat mass, lean mass, depressive symptoms and perceived stress, the association between self-reported school/weekday sleep duration and binge eating remained non-significant, OR = 1.00, CI = 0.99, 1.01, $p = .99$. In adjusted analyses, subjective daytime sleepiness was associated with a greater odds of having objective binge eating, OR = 8.65, CI = 1.78, 42.16, $p = .009$.

Discussion

Among adolescent girls with overweight/obesity, a family history of diabetes and mild-to-moderate depressive symptoms, a small minority reported the recommended amount of daily sleep during the week (Hirshkowitz et al., 2015). Shorter than recommended levels of sleep are associated with excessive gains in adiposity and exacerbated metabolic functioning among children (Cappuccio et al., 2008; Fatima, Doi, & Mamun, 2015; Hart et al., 2011; Koren, O'Sullivan, & Mokhlesi, 2015; Matthews & Pantesco, 2015). Prior data suggest that this association may be explained, in part, by disinhibited eating patterns (Beebe et al., 2013; Burt et al., 2014; Firouzi et al., 2014; Golley et al., 2013; Hart et al., 2013; Simon et al., 2015). However, in the current study, reported sleep duration was not associated with objective EAH nor binge eating. Instead, sleep duration was positively associated with observed energy intake; girls who reported more sleep during the week consumed significantly more energy from a lunch buffet meal.

The significant and positive association between self-reported sleep duration and objective energy intake was unanticipated and contradictory to most, although not all (Klingenberg et al., 2012), prior research. Variability in sample characteristics and research methods may account for some of the discrepancies across studies. The current sample included adolescent girls with mild-to-moderate depressive symptoms. There is tremendous heterogeneity in the clinical presentation of depression, with some *atypical* phenotypes being characterized by elevated BMI, metabolic dysfunction, greater self-reported consumption of palatable foods, and hypersomnia (Lamers et al., 2010; Rahe et al., 2015;

Simmons et al., 2016). Recurrent patterns of elevated sleep duration and caloric consumption (in the current study, 90 more kcal per hour of additional sleep) could be anticipated to promote excessive weight gain and exacerbated metabolic functioning among girls with an atypical depressive presentation. It will be important for future research to examine this hypothesis using objective measures of sleep behavior as adolescents tend to overestimate their sleep duration (Short, Gradisar, Lack, Wright, & Carskadon, 2012). To that end, we cannot rule out the possibility that adolescents included time spent in bed attempting sleep, sleeping insufficiently, and/or engaging in leisure activities in their estimates of sleep duration.

In the current study, we also examined daytime sleepiness, a qualitative indicator of sleep that has been uniquely associated with type 2 diabetes risk (Kita et al., 2012). As hypothesized, self-reported daytime sleepiness was associated with greater odds of recent binge eating, even after accounting for body composition, depressive symptoms and perceived stress, but not objective intakes. Behavior regulation problems, emotional coping difficulties, and/or attentional problems, may underlie both perceptions of daytime sleepiness (Baum et al., 2014; Gruber et al., 2011) and self-reported disinhibited eating behaviors such as binge eating (Czaja, Rief, & Hilbert, 2009; Reinblatt et al., 2015). For instance, rumination may promote both binge eating and poor sleep quality (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Pillai, Steenburg, Ciesla, Roth, & Drake, 2014). Additional research is needed to explain the link, and the direction of the link, between sleepiness and binge eating. Binge eating, for instance, may lead to higher levels of daytime sleepiness following the overconsumption of foods high in saturated fat and low in dietary fiber (St-Onge, Roberts, Shechter, & Choudhury, 2015).

Results from the current study extend previous research in several important ways. Although the generalizability of the current study's findings are limited to adolescent girls at-risk for type 2 diabetes, little is known about the sleep and disinhibited eating behaviors of these individuals. Obesity (Park, Falconer, Viner, & Kinra, 2012), poor sleep (Flint et al., 2007; Koren et al., 2011; Koren et al., 2015; Matthews et al., 2012) and disinhibited eating (Tanofsky-Kraff et al., 2012) all increase risk for metabolic dysfunction and are thus important factors to consider concurrently when designing clinical efforts for the prevention of type 2 diabetes. Furthermore, the majority of prior studies examining sleep and eating behavior have relied on self-report methods of general energy intake and there has been little attention to specific disinhibited eating behaviors associated with excessive weight gain and exacerbated metabolic functioning in youth, such as binge eating (Field et al., 2003; Sonnevile et al., 2013; Tanofsky-Kraff et al., 2006). Sleep is also generally operationalized according to whether participants meet the recommended amount of sleep duration, despite research highlighting the importance of also examining qualitative indicators of sleep (Jarrin et al., 2013). The current study addressed many of these limitations through its inclusion of multi-modal assessments of disinhibited eating and a measure of daytime sleepiness, which allowed us to identify significant associations with obesity-promoting eating behaviors which would not have been uncovered otherwise.

Future research should evaluate the relationship of sleep and disinhibited eating using objective measures of sleep duration and quality. Prospective data are also needed to

determine whether binge eating accounts for links between sleep quality and obesity risk, as well as associated metabolic concerns, among youth. Of note, in the current study, sleep duration accounted for only a small amount of variance in the model examining observed energy intake. Moreover, multiple comparisons were conducted, increasing the odds of type 1 error. For these reasons, replication studies with larger samples of adolescents are needed. Future studies should include boys, as extant data from adults suggest that males and females may have differential metabolic and energy responses to short sleep duration (Cedernaes, Schiöth, & Benedict, 2015).

In summary, self-reported daytime sleepiness was associated with obesity-promoting binge eating behavior, after adjusting for body composition, depressive symptoms and general perceived stress. Reported sleep duration, in contrast, was not associated with binge eating or objective EAH, although it was positively associated with energy intake from a lunch buffet. This association may reflect an atypical depressive presentation, a surrogate for other sleep difficulties, and/or variability in sample characteristics across studies. Data from the current study highlight the importance of evaluating quantitative and qualitative indicators of sleep behavior in the context of obesity-promoting eating behaviors and type 2 diabetes prevention.

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Figure 1.

Accounting for age, race, height, puberty, fat mass, lean mass, depressive symptoms and perceived stress, reported average school/weekday sleep duration was positively associated with energy intake, such that girls who reported more sleep during the week consumed more total energy during the lunch buffet ($\beta = 1.39$, $SE = 0.62$, $p = .03$).

Bivariate correlations for reported sleep duration, daytime sleepiness, disinhibited eating behaviors, body/fat mass, depressive symptoms, and perceived stress

Table 1

	1	2	3	4	6	6	7	8
1 Sleep duration (min)	-							
2 Daytime sleepiness	-0.15	-						
3 Energy intake (kcal)	0.05	0.09	-					
4 EAH (kcal)	-0.06	-0.04	*0.19	-				
5 BMIz	-0.16	0.09	**0.33	*0.22	-			
6 Fat mass (kg)	** -0.26	0.01	**0.26	**0.27	**0.88	-		
7 Depressive symptoms	** -0.27	0.12	0.12	0.11	0.11	0.14	-	
8 Perceived stress	-0.12	0.09	0.05	0.15	-0.01	0.06	**0.26	-
Mean $\pm$ SD	421 $\pm$ 99	1.7 $\pm$ 0.5	1549 $\pm$ 623	385 $\pm$ 181	2.0 $\pm$ 0.5	37 $\pm$ 13	25 $\pm$ 7	30 $\pm$ 5

Note: EAH = eating in the absence of hunger; BMIz = body mass index standard score;

** $p < .01$,

* $p < .05$

Adjusted associations for reported sleep duration and daytime sleepiness with observed energy intake

Table 2

Sleep Variable	Variables Entered	β^a	SE	b^b	R^{2c}	R^2
Reported school/weekday sleep duration	Age	-15.91	44.05	-0.04	**0.21	**0.21
	Race	135.54	152.60	0.08		
	Puberty	-98.50	83.01	-0.13		
	Height	-17.48	10.97	-0.18		
	Fat mass	-21.68	643.80	-0.004		
	Lean mass	39.25	12.39	**0.51		
	Depressive symptoms	1.95	8.63	0.02		
Daytime sleepiness	Perceived stress	6.04	12.01	0.05		
	Sleep duration	1.39	0.62	*0.22	**0.25	*0.04
	Age	-14.97	44.00	-0.04	**0.20	**0.20
	Race	128.37	152.27	0.08		
	Puberty	-101.49	82.88	-0.13		
	Height	-18.55	10.90	-0.20		
	Fat mass	-27.98	643.25	-0.01		
	Lean mass	39.77	12.37	**0.51		
	Depressive symptoms	0.56	8.49	0.01		
	Perceived stress	6.41	11.99	0.05		
	Sleepiness	-21.41	128.50	-0.02	**0.20	<0.001

^a β = unstandardized regression coefficient at each step;^b b = standardized regression coefficient at each step;^c R^2 = proportion of variability in the dependent variable accounted for by model*** $p < .01$;* $p < .05$

Adjusted associations for self-reported sleep duration and daytime sleepiness with objective eating in the absence of hunger (EAH)

Table 3

Sleep Variable	Variables Entered	β^a	SE	b^b	R^{2c}	R^2
Self-reported school/weekday sleep duration	Age	-0.87	13.13	-0.01	* _{0.17}	* _{0.17}
	Race	-56.89	45.63	-0.12		
	Puberty	1.87	24.89	0.01		
	Height	4.36	3.31	0.16		
	Fat mass	544.49	191.75	** _{0.38}		
	Lean mass	-3.77	3.87	-0.17		
	Depressive symptoms	1.63	2.57	0.06		
Perceived daytime sleepiness	Perceived stress	4.06	3.58	0.11		
	Lunch energy intake	0.03	0.03	0.12		
	Sleep duration	0.004	0.19	0.002	* _{0.17}	<0.001
	Age	-0.28	13.24	-0.002	* _{0.17}	* _{0.17}
	Race	-61.42	45.96	-0.13		
	Puberty	0.68	25.11	0.003		
	Height	3.86	3.33	0.14		
Sleepiness	Fat mass	541.15	193.48	** _{0.38}		
	Lean mass	-3.66	3.90	-0.16		
	Depressive symptoms	0.86	2.55	0.03		
	Perceived stress	4.23	3.61	0.12		
	Lunch energy intake	0.04	0.03	0.13		
	Sleepiness	-9.83	38.65	-0.03	* _{0.17}	0.001

^a β = unstandardized regression coefficient at each step;^b b = standardized regression coefficient at each step;^c R^2 = proportion of variability in the dependent variable accounted for by model**
 $p < .01$;*
 $p < .05$