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Dimensions of sleep quality are related to objectively-measured eating behaviors among children at high familial risk for obesity

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Abstract

Objective: To evaluate whether dimensions of sleep quality were associated with homeostatic and hedonic eating behaviors among children with healthy weight (BMI-for-age<85%) but varying maternal weight status.

Methods: Seventy-seven children (age:7.4±0.6 y; BMI z-score:−0.10±0.7) with healthy weight and high (n=32) or low (n=45) familial obesity risk based on maternal weight status were served an *ad libitum* meal (homeostatic eating) followed by palatable snacks to assess eating in the absence of hunger (EAH; hedonic eating). Habitual sleep quality was quantified from 7 nights of wrist actigraphy. Partial correlations, adjusted for child energy needs, pre-meal hunger, food liking, and socioeconomic status evaluated associations of sleep with meal intake and EAH. Additionally, sleep-by-obesity risk interactions were assessed.

Results: Greater sleep fragmentation was associated with higher homeostatic meal energy intake, but only among children at high familial obesity risk (p-interaction=0.001; β(high-risk=48.6, p<0.001). Sleep fragmentation was not associated with total EAH but was related to higher and lower intake of carbohydrates (r=0.33, *p*=0.003) and fat (r=−0.33, *p*=0.003), respectively.

Conclusions: Adverse associations of poor sleep with energy intake may be amplified among children already predisposed to obesity. Furthermore, that fragmented sleep relates to preferential intake of carbohydrates over fat during EAH may suggest alterations in taste preferences with poor sleep.

Keywords

Sleep; Sleep quality; Eating behaviors; Food intake; Eating in the absence of hunger; Children

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INTRODUCTION

Suboptimal sleep is an emerging risk factor for obesity among children (1). This has broad public health implications since an estimated 25% of US children experience sleep problems (2). Elucidating mechanisms underlying the sleep-obesity association is a critical step in the development of new strategies to treat and/or prevent obesity. Studies in adults and children demonstrate that sleep loss leads to increased energy intake (3, 4). However, little is known about the role of sleep quality in children's eating behaviors.

Suboptimal sleep could promote weight gain by increasing the homeostatic drive to eat, which can be quantified through assessment of *ad libitum* energy intake at a meal (5). Despite conflicting results of observational studies evaluating associations of sleep duration with energy intake in children (6, 7), experimental studies varying sleep duration observe increased 24-h energy intake (8, 9), particularly in the evening (10), in response to curtailed sleep. Whether quality, beyond quantity, of sleep relates to differences in homeostatic eating among children, however, is unclear. Only 2 studies have evaluated dimensions of sleep quality in relation to intake in children; results demonstrated that poorer sleep efficiency was associated with more frequent intake of energy-dense, nutrient-poor foods (11) and higher overall energy intake (12). While these findings point towards a link between poor sleep and greater homeostatic eating, results are equivocal and rely on self-reported intake. Studies are needed to evaluate the association between sleep quality and objectively-measured food intake to better understand the role of sleep in energy balance.

It has also been postulated that heightened responsiveness to hedonic food properties could underlie the relation of suboptimal sleep with obesity (13). Among children, eating in the absence of hunger (EAH) is a widely-used method to assess hedonic eating (14). Studies evaluating sleep exposures in relation to EAH have produced mixed results (15–17). For example, sleep duration, but not quality, was inversely associated with children's EAH (17); however, this study relied on self-reported measures of sleep, which may not reflect actual sleep quality or capture its multidimensionality. In fact, no study to date has evaluated free-living sleep in relation to EAH in children using objective measures of both sleep and food intake.

Middle childhood represents a particularly important life stage to study associations between sleep and eating, given growing autonomy in making lifestyle choices. Therefore, we used objective sleep and food intake data from a longitudinal study of obesity risk in 7–8 y-old children of varying familial risk status to evaluate cross-sectional associations of habitual sleep quality with homeostatic and hedonic eating. Homeostatic eating was quantified by measuring *ad libitum* intake at a meal eaten when children were hungry, and hedonic eating was assessed using an EAH paradigm when children were sated. We hypothesized that dimensions of poor sleep, including lower sleep efficiency, longer wake after sleep onset, and more disrupted sleep, would be associated with higher energy intakes at both eating paradigms. Furthermore, because the heritability of obesity is strengthened in obesogenic home environments (18), and familial obesity risk moderates the association between sleep and child weight status (19), we also examined whether familial obesity risk, based on maternal weight status, would moderate associations of sleep quality with intake. We

hypothesized that associations between poorer sleep and higher energy intake would be stronger among children with high relative to low familial obesity risk.

METHODS

Study Design

This was a cross-sectional analysis among children with healthy weight who were at low- or high-risk for obesity based on maternal weight status (see NCT02759523). Complete baseline data on habitual sleep patterns, food intake, and EAH were available from 77 children enrolled in a larger prospective study to characterize neurobiological and appetitive characteristics that predict gains in adiposity (NCT03341247). Dimensions of sleep were assessed over 7 nights. Homeostatic eating was assessed at an *ad libitum* meal, and EAH was evaluated through provision of palatable foods following the meal.

All procedures were approved by the Institutional Review Board of The Pennsylvania State University. Prior to participating, parents provided written informed consent, and children provided written and verbal assent. Families received compensation for each study visit.

Participants

This analysis was a component of a larger prospective cohort study enrolling healthy 7- and 8-y-old children and the parent identified as having primary responsibility for feeding (87.4% mothers). The narrow age range reflects the larger aim of this project, which was intended to identify neurocognitive determinants of pre-pubertal adiposity gain. Families were recruited through flyers placed around the community, advertisements on social media and radio, monthly mailings, and word-of-mouth. Families interested in participating were invited to the laboratory for assessment of eligibility. At this screening visit, height and weight were measured in duplicate for both parent and child using a stadiometer and calibrated scale (ScaleTronix 5002, Welch Allyn, Chicago, IL, USA), respectively. Weight status was quantified using BMI for parents and BMI z-score and BMI percentile for children. These values were calculated using the Centers for Disease Control and Prevention conversion program (20). Families with children meeting the following criteria were eligible for the study: 7–8 y old, BMI between the 5th and 90th percentiles, ability to safely undergo magnetic resonance imaging scanning, and no conditions affecting appetite or cognition. Medical and developmental conditions were reported by the parents prior to the visit. Furthermore, for families to be eligible, the mother of the child was required to have BMI of 18–26 kg/m^2 or >29 kg/m^2, so that children could be categorized as either low- or high-risk for obesity, respectively. In the full study, 117 families consented and were enrolled. Ninety-eight children from these families completed the baseline visits, and 77 (78.6%) provided complete exposure and outcome data for the current analysis. There were no differences in family income, weight status, or demographic characteristics between the 77 children who provided complete data and those who did not (p>0.05 for all).

Procedures

Baseline data collection for the full study occurred between July 2017 to July 2022 but was paused for ~8 months due to the COVID-19 pandemic. Therefore, only baseline data were

available for the current analysis. The data included in the current analysis were collected in conjunction with the first baseline visit.

The baseline visit was conducted at the General Clinical Research Center, and parents were instructed to have children refrain from eating or drinking (except water) for ≥3 h prior to the visit. Children were escorted to a separate room for assessment of homeostatic eating (i.e., following a 3-h fast) via provision of a multi-item meal (see section labeled ad libitum food intake), followed by measurement of hedonic (i.e., when they were full) eating via EAH (see section labeled EAH). Before and after each eating paradigm, children rated fullness using a validated, age-appropriate, visual analog scale (21). After completing pre-meal fullness and immediately before beginning the meal, children tasted small (<3 g) samples of each food and rated liking on a 5-point hedonic scale (1=hate it, 5=love it).

At the conclusion of the baseline visit, children were provided a triaxial accelerometer for assessment of habitual sleep. Parents were instructed to have their child wear the device on their non-dominant wrist at all times over the 7 days following the visit to capture their typical patterns of sleep (22).

Measures

Ad libitum food intake (homeostatic eating)—To examine typical meal intake when hungry, children were presented with a multi-item meal of age-appropriate, commonly-consumed foods, including sandwiches, cookies, chips, carrots, and applesauce (Table 1 and Figure 1A). The same foods and amounts were provided to each child. As one of the goals of this meal was for children to eat until sated, we provided a range of sandwich types (e.g., cold cuts, cheese, peanut butter) from which children could select. Foods were pre-weighed on a research-grade scale (Nimbus NBL-3602e, Adam Equipment, Oxford, CT) and presented on trays with no packaging. Children were told that they had up to 30 min to eat as much or as little as they wanted. Before the COVID-19 pandemic, a researcher sat with the child (n=49) during the meal and read a neutral book to reduce the likelihood of the child engaging in conversations about food. To maintain safety, when research activities resumed, the parent read to the child (n=14) or audio books were played (n=14) (Audible by Amazon, Newark, NJ).

After 30 min of eating, or once children indicated that they had eaten to satisfaction, uneaten foods and beverages were removed and individually weighed to the nearest 0.01 g on a scale. Post-meal values were subtracted from the respective pre-meal weights (amount served) to determine amounts (g) consumed. Food and beverage intakes by weight were then multiplied by the energy density of the corresponding item to determine energy intake (kcal). Food and beverage nutrient information were obtained from nutritional facts panels and/or from standard nutrient databases (https://fdc.nal.usda.gov/).

EAH (hedonic eating)—To measure hedonic eating, children were provided a selection of energy-dense sweet and savory snacks 15 min after the meal (Table 2 and Figure 1B), along with a selection of activities and toys (e.g., coloring, toy cars). Children were told that they could play with any of the toys and/or eat from the selection of foods while a researcher worked in an adjacent room. After 10 min, the researcher returned and collected the uneaten

food. Similar to the main meal, pre-specified amounts of foods were served, and post-meal weights were subtracted from pre-meal weights to determine food intake. Energy intake was computed using the methods previously described.

Dimensions of sleep—Free-living sleep patterns were assessed using the validated ActiGraph GT3X+ (ActiGraph Corp, Pensacola, FL) worn on the non-dominant wrist. Wrist actigraphy for assessment of sleep in children has been compared with polysomnography and performs well for accuracy and sensitivity (23, 24), and it is endorsed for sleep assessment in children by the American Academy of Sleep Medicine (25). Furthermore, multi-day wrist actigraphy represents a strong index of habitual sleep (22). Therefore, to capture typical sleep patterns, children were asked to wear the device continuously (aside from swimming and bathing) for 1 week. Following completion of the week, actigraphy devices were returned for sleep scoring. Data were validated and scored using the ActiLife 6 software (ActiGraph Corp Pensacola, FL). Sleep onset and offset were assessed via Tudor-Locke algorithm (26). Data were visually inspected independently by 2 researchers (JB, MS) for verification of bed and waketimes provided by ActiLife. All sleep dimensions were quantified using the Sadeh algorithm (27). Sleep quality exposures of interest included: *sleep efficiency:* the percentage of time in bed that is spent asleep; *wake after sleep onset*: time spent awake after sleep onset; and *fragmentation index*: the percentage of 1-min periods of sleep vs. all sleep periods (representing shifts from deep to light sleep). In addition to sleep quality, we also examined associations between intake and *total sleep time*: number of minutes scored as sleep between sleep onset and offset (28). Children who did not wear the watch for ≥3 consecutive nights (n=14) were removed from analysis, and 1 additional participant was removed due to outlier sleep data (values > 3 SD outside of the mean).

Data analysis

Descriptive statistics are presented as mean±SD for continuous variables and frequency (%) for categorical variables. Prior to conducting analyses, all variables were checked for normality using the Shapiro-Wilk test. Associations of sleep with eating behaviors were evaluated using Pearson's correlation coefficients. Partial correlations, with addition of covariates that could influence energy intake, were then conducted to determine if results persisted. Covariates included estimated basal metabolic rate (BMR) determined by the Schofield equation (29), average liking of foods, pre-meal fullness, and family income. Sex and age were not added as covariates in models since they are included in the BMR calculation. Total sleep time was also included in adjusted models to determine whether associations of sleep quality with intake were independent of sleep duration. To rule out the potential for systematic differences in primary findings between children tested before (n=49) and after (n=28) the COVID-19 lockdowns, additional models included a dichotomous covariate for testing period: pre- vs. post-lockdown. As inclusion of testing time did not affect significance, the term was dropped from presented analyses; however, results of fully-adjusted models are in the Supplementary Text. The PROCESS (v4.1) macro in the Statistical Package for Social Sciences (SPSS, v28) was used to test whether familial risk status moderated associations between sleep and intake. To control for false discovery rate (FDR), Benjamini-Hochberg correction was applied with a FDR of 5% based on the

number of individual tests run (n=48). (30) All hypotheses were 2-tailed, and an adjusted p-value of 0.05 was used to denote statistical significance.

RESULTS

Descriptive characteristics

Descriptive statistics for the analytic sample are provided in Table 3. There were no differences in child age, sex, or estimated BMR between familial risk groups. Although children were at a healthy weight status on average (BMI-for-age %: 46.9±24.5), BMI z-score was greater for high-risk relative to low-risk children ($p<0.01$). Children in this sample were predominantly white, and nearly 40% of parents reported yearly family income >$100,000 across the full sample. However, families of children with low-familial obesity risk were more likely to report earning >$100,000 than high-risk families. On average, 6.3 nights of sleep data were obtained from children (ranging from 3–7). Total sleep time across the full sample was 9.7±0.6 h/night; neither sleep duration nor parameters of sleep quality differed by familial risk status ($p>0.05$ for all). Measured energy intake at the meal and EAH paradigm did not differ between children at high- vs. low-risk for obesity ($p>0.05$ for both).

Associations between sleep and intake at the buffet meal (homeostatic eating)

Pearson's correlation coefficients between sleep and both energy and nutrient intakes at the main meal are provided in Table 4. Prior to adjustment for covariates, there were modest associations between fragmented sleep and greater meal energy intake (r=0.25, $p=0.052$, FDR adjusted). Following adjustment for BMR, food liking, pre-meal fullness, income, and total sleep time, none of the associations between sleep quality and intake at the homeostatic meal were significant (p>0.05 for all). There were no associations between total sleep time and any of the homeostatic intake measures ($p>0.05$ for all). Exploratory analyses were also conducted to identify patterns of food intake at the meal that were related to sleep quality (see Supplementary Text).

Associations between sleep and EAH (hedonic eating)

In fully-adjusted models, associations of sleep fragmentation with EAH were not statistically significant (Table 4). However, in terms of nutrient intakes in the EAH paradigm, partial correlations from adjusted models demonstrated that more fragmented sleep was associated with higher percent of calories consumed from carbohydrates (r=0.32, $p=0.003$, FDR adjusted) but lower percent of calories from fat (r=−0.32, $p=0.003$, FDR adjusted). Although not reported in Table 4, there were no associations between sleep duration and any of the hedonic intake measures ($p>0.05$ for all). Exploratory analyses were also conducted to identify patterns of intake at the hedonic eating paradigm that were related to sleep quality (see Supplementary Text).

Moderating role of familial risk in the association between sleep quality and intake.

After accounting for the effects of covariates, a significant proportion of variance was explained by fragmentation index and the fragmentation index-by-risk interaction ($F(7,66)=7.5$, $R^2=0.44$, $p<0.001$, FDR adjusted). In this model, there was a main effect of fragmentation index ($\beta=17.2$, $t(66)=2.5$, $p=0.005$, FDR adjusted). However, due

to a fragmentation index-by-risk status interaction (β=52.8, t(66)=3.8, p=0.001, FDR adjusted), the main effects will not be interpreted. Evaluation of the slopes by obesity risk demonstrated a positive relationship between sleep fragmentation and meal intake in high-risk, such that, among high-risk children, energy intake at the meal increased by 48.6 kcal for every unit increase in fragmentation index (β=48.6; t(66)=4.39; p=0.001, FDR adjusted), while no association was observed between fragmentation index and meal intake in low-risk children (β=−4.1; p=0.63) (Figure 2). None of the other relationships between sleep quality and intake at either the meal or EAH were moderated by familial risk.

DISCUSSION

This study contributes novel findings on sleep quality and eating behaviors in children by demonstrating that, in children with healthy weight but high familial risk for obesity, fragmented sleep is associated with greater meal energy intake. That maternal obesity status moderated the association between sleep and intake supports prior literature demonstrating that adverse associations of suboptimal sleep with obesity risk are not equal across children (19, 31). Notably, associations of poor sleep with homeostatic eating in this study were observed among children who met recommendations for sleep duration on average, with over 95% sleeping more than 8.5 h/night, indicating that fragmented sleep could be an independent risk factor for overconsumption.

Several mechanisms have been proposed to explain the association between sleep and food intake, including biological (i.e., the release of hormones influencing hunger and satiety) and behavioral (i.e., greater opportunity to eat with short sleep) (13). Particularly strong evidence comes from neuroimaging studies showing sleep-based alterations in neural responses to food cues in regions implicated in reward-based feeding (32, 33); however, data exists to support roles for homeostatic underpinnings of the sleep-intake relation as well (34). The present study addresses both mechanisms by measuring food intake under fasted and fed conditions, with current results suggesting that the association between sleep quality and intake is more prominent among children at high-familial risk for obesity during a homeostatic eating occasion. Despite our data supporting homeostatic mechanisms underlying the sleep-food intake association, we cannot preclude a potential contribution of hedonic underpinnings, since both homeostatic and hedonic processes facilitate ingestive behaviors across a variety of contexts. For example, it is possible that the buffet meal in the homeostatic eating paradigm, which consisted of a variety of sandwiches, as well as chips and cookies, might have also generated overconsumption among children for hedonic reasons; both variety (35) and palatability (36) can stimulate intake. Systematic assessment of selection and intake of palatable, energy-dense foods under varying levels of sleep quality will be needed to disentangle the mechanistic underpinnings of the sleep-intake association. Regardless of the mechanism involved, our findings expand the literature by showing that children who are at high-familial risk for obesity may be susceptible to greater homeostatic energy intake under conditions of poor sleep quality.

In the present study, we found no significant association between sleep quality and energy intake during EAH. This null result contributes to existing inconsistency in the literature relating sleep to EAH; an observational study found an inverse association between sleep

duration and EAH observed in low-income toddlers' homes (17), while an experimental study varying sleep duration in 8–12 year-olds found no effect on EAH observed in the laboratory (16). Given the heterogeneity across studies in terms of design and findings, it is not possible to preclude a role for sleep quality in EAH. Despite the null findings with total EAH, fragmented sleep was associated with preferential intake of carbohydrates over fat. Given the selection of foods provided to children, this implies that children with poorer sleep may have preferred the sweeter and/or higher carbohydrate food options (e.g., fruit candies) as opposed to higher-fat selections, such as cookies and ice cream. Thus, in addition to its association with meal intake, fragmented sleep might also act specifically on taste preferences in a manner that promotes intake of sweet foods. In adults, curtailing sleep shifted sweet preference to higher concentrations (37), and in youth, sleep restriction promotes intake of desserts (38) and dietary carbohydrates and sugar (39, 40), potentially by enhancing their appeal (41). Given the paucity of studies in children with objective measures of both sleep and food intake and the inconsistent findings across these studies, systematic investigation into whether impaired sleep contributes to a positive energy balance through increased hedonic feeding is warranted.

Our investigation of individual differences in the associations of sleep quality with food intake revealed that familial obesity risk moderated this relation. More specifically, adverse associations of greater sleep fragmentation with food intake were apparent only for children who had mothers with obesity. While parental obesity is one of the strongest predictors of childhood obesity, inter-generational effects are particularly strong between mother and child (42). Beginning with the intrauterine environment, maternal obesity can "program" fetal risk for future metabolic disease, potentially priming the central nervous system to exhibit heightened reward responses to foods present in the environment (43). As evidence to support the durability of this relationship, mothers and children show similar patterns of food intake (44) and eating behaviors (45). Furthermore, a healthy home food environment can reduce the impact of genes on the development of obesity (18), while an obesogenic home food environment can exacerbate genetic risk for obesity. Thus, it could be that in children at high risk for obesity, biological susceptibility interacts with the home food environment to increase appeal of energy-dense foods; in this study, this could have increased vulnerability of high-risk to eating such foods in the laboratory.

It is notable that associations between sleep fragmentation and food intake were observed among young children with healthy body weights. Sleep problems are widely prevalent among children and adolescents, and greater sleep disturbances are associated with higher odds for obesity (46). Given that energy intake is a key determinant of weight change, it is possible that overeating in response to fragmented sleep could promote the development of obesity. Our data linking more fragmented sleep with higher meal energy intakes would support this postulation, although longitudinal data will be needed for confirmation, as a temporal relationship cannot be determined from this study. Notably, in animal models, chronic sleep fragmentation promotes hyperphagia and obesity (47). In humans, experimental sleep fragmentation suppressed feelings of fullness and satiety hormones (48); however, neither *ad libitum* food intake nor long-term weight change were assessed in that study. That associations of sleep fragmentation with eating behaviors in this study were observed in children that have not yet developed obesity but are at high risk suggests that

this measure may warrant additional consideration as a potential early indicator of the risk for more serious metabolic disease.

Several strengths of this study should be noted. Foremost, this is the first study in middle childhood to apply objective measures of both sleep and food intake across multiple contexts (i.e., meal and snack). These paradigms allowed us to precisely measure both the types and amounts of foods consumed under controlled conditions. It has been proposed that overeating in response to insufficient sleep results from more time awake for snacking (13), but current results from a controlled environment suggest mechanisms extend beyond greater opportunity to eat. Sleep was assessed over 7 days with actigraphy, which is recommended for characterizing habitual, free-living sleep patterns (49). In addition, because these measures were collected as part of an ongoing clinical trial, we were able to account for a number of family-related risk factors for obesity. As a result, we identified potential maternal obesity as a moderator that magnified the impact of fragmented sleep, a critical step needed for the development of tailored interventions. Furthermore, we collected these data in a primarily healthy weight cohort of children, thereby highlighting dimensions of sleep that may be of concern prior to the development of obesity.

Despite these strengths, several limitations should be addressed. First, although the sample was representative of the geographic region, it lacked ethnic and racial diversity. Replication in a more diverse sample of children is needed, given growing evidence of sleep health disparities (50). In addition, this was a secondary analysis with a modest sample size, necessitating the need to be conservative in our selection of covariates for adjusted models. Because overfitting of models can occur with limited sample sizes, we also presented results of unadjusted analyses. To enhance generalizability of this laboratory-based study, various commonly-consumed foods were presented at both the hedonic and homeostatic eating paradigms. However, because the *ad libitum* meal included a variety of highly palatable foods (i.e., chips, cookies), which can evoke hedonic eating, it is challenging to clearly separate homeostatic from hedonic mechanisms in this study. In addition, the data from this cohort were cross-sectional, so we cannot confirm directionality of the sleep-intake relation. Future studies should prioritize evaluating prospective relations between objectively assessed sleep quality and food intake.

In conclusion, actigraphy-derived sleep fragmentation was positively related to measured meal intake in healthy weight children at high familial risk for obesity. Interestingly, we do not find associations of sleep duration with either homeostatic or hedonic eating, despite other studies reporting greater energy intakes among short sleepers (40, 51). The results of the current study may be evidence of an independent role of disrupted sleep in promoting overconsumption; however, this must be interpreted with caution, given the limited variability in total sleep time across the children, most of whom routinely achieved adequate sleep duration. Although sleep was not associated with children's total intake in the absence of hunger, fragmented sleep was positively associated with preferential selection of carbohydrates over fat at the hedonic eating paradigm. These findings imply that dimensions of sleep quality warrant exploration as potential behavioral targets associated with the future development of obesity.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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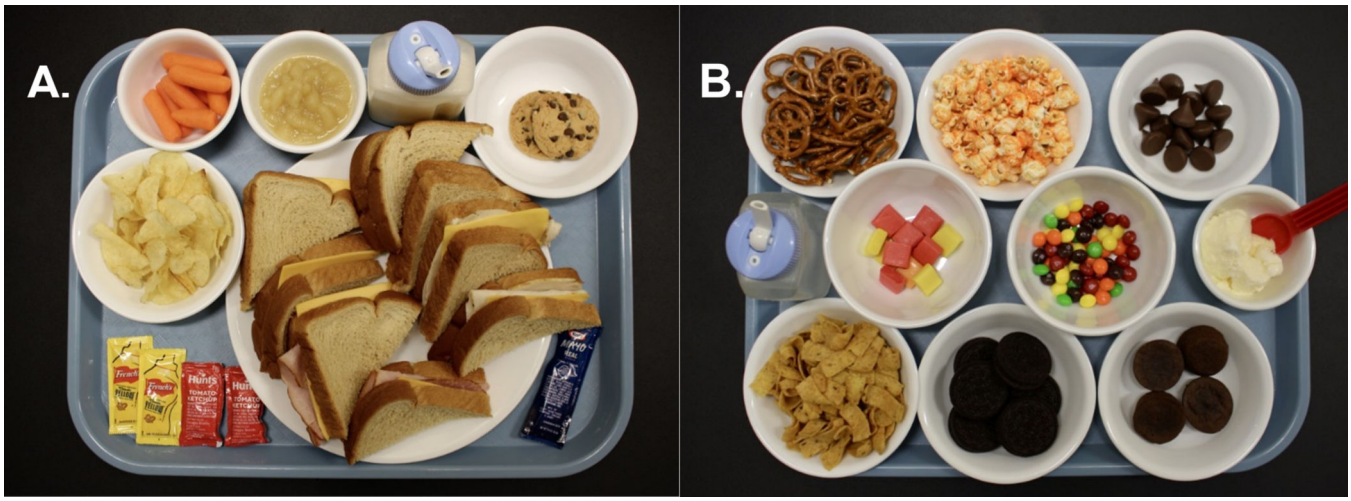

Figure 1.
Images of the foods served at the ad libitum test meal (A) and the subsequent eating in the absence of hunger paradigm (B).

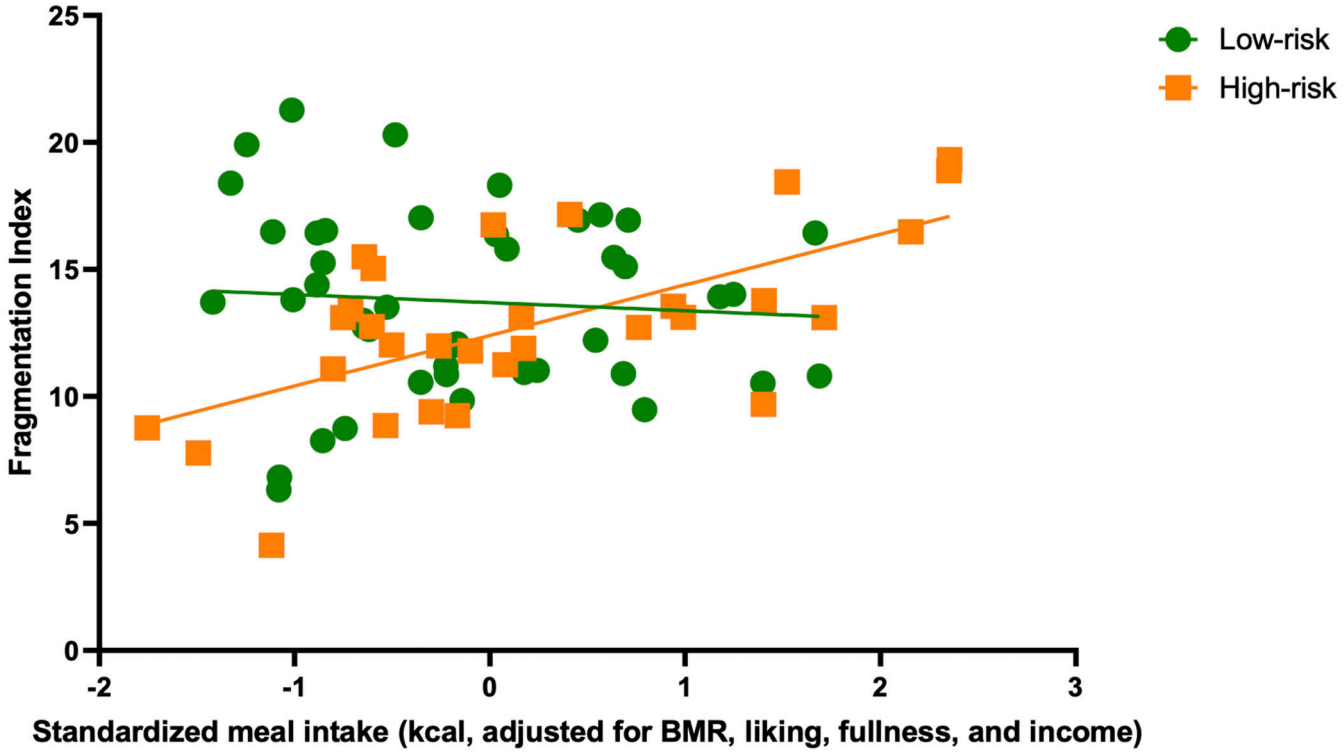

Figure 2.

Fragmentation index plotted against the standardized residual (z-score) of meal intake (adjusted for BMR, liking, fullness, and income). Fragmentation index was positively associated with intake at the buffet meal in children who were high-familial risk for obesity, classified by maternal obesity status (β=48.6; t(66)=4.39; p<0.001). In high-risk children, for every 1 unit increase in fragmentation index, intake at the buffet meal increased by 48.6 kcal

Table 1.

Foods served at the *ad libitum* buffet meal

Food item	Weight (g) served	Energy (kcal) served	ED (kcal/g) served	CHO (% kcal) served	FAT (% kcal) served	PRO (% kcal) served
Cheese sandwich [a]	98	270	2.8	32	51	17
Ham & cheese sandwich [a]	154	340	2.2	39	37	24
Turkey & cheese sandwich [a]	154	320	2.1	40	36	24
Peanut butter & jelly [b]	113	390	3.5	49	39	12
Applesauce [c]	110	49	0.5	100	0	0
Baby carrots [a]	60	25	0.4	89	0	11
Cookies [d]	22	107	4.9	55	42	3
Potato chips [e]	30	171	5.7	40	53	7
Milk – 2% fat [a]	290	157	0.5	40	35	25
Ketchup [f]	18	20	1.1	100	0	0
Mayo [g]	24.8	180	7.3	0	0	100
Mustard [h]	14	0	0	0	0	0
Total	1,087.8	2,029	NA	NA	NA	NA

[a] Wegman's, Rochester, NY, USA;

[b] Smucker's, Orrville, OH, USA, Jif Creamy, Lexington, KY, USA;

[c] Motts, Plano, TX, USA;

[d] Chips Ahoy, East Hanover, NJ, USA;

[e] Lays, Plano, TX, USA;

[f] Heinz, Pittsburgh, PA, USA;

[g] Kraft, Chicago, IL, USA;

[h] French's, Rochester, NY, USA

Table 2.

Foods served at the EAH paradigm

Food item	Weight (g) served	Energy (kcal) served	ED (kcal/g) served	CHO (% kcal) served	FAT (% kcal) served	PRO (% kcal) served
Chocolate kisses [a]	66	330	5.0	45	50	5
Fruit chews [b]	66	264	4.0	85	15	0
Fruit candy [c]	66	264	4.0	92	8	0
Brownies [d]	55	240	4.4	50	45	5
Vanilla ice cream [e]	48	90	1.9	50	45	5
Cheese popcorn [e]	30	170	5.7	28	64	8
Corn chips [f]	58	331	5.7	40	56	4
Pretzels [g]	39	153	3.9	84	8	8
Total	494	2,153	NA	NA	NA	NA

[a] Hershey's, Hershey, PA, USA;

[b] Starburst, Mars, McLean, VA, USA;

[c] Skittles, Mars, McLean, VA, USA;

[d] Oreos, Nabisco, East Hanover, NJ, USA;

[e] Wegman's, Rochester, NY, USA;

[f] Fritos, Pepsico, Purchase, NY, USA;

[g] Rold Gold, Pepsico, Purchase, NY, USA

Table 3.

Characteristics of the analytic sample at baseline

Participant characteristics	Total (*n*=77)	Low-Risk (*n*=45)	High-Risk (*n*=32)
Physical: Continuous (mean (SD))			
Age (y)	7.4 (0.6)	7.4 (0.6)	7.4 (0.6)
BMI z-score	−0.10 (0.7)	−0.29 (0.7) **	0.16 (0.7) **
BMR (kcal) [a]	1046.5 (76.6)	1041.4 (78.1)	1053.6 (75.2)
Demographic: Categorical (%(n))			
Sex			
Male	51 (39)	55.6 (25)	43.8 (14)
Female	49 (38)	44.4 (20)	56.2 (18)
Race			
White	97 (75)	95.6 (43)	100.0 (32)
Non-White	3 (2)	4.4 (2)	0 (0)
Household Income ($) [b]			
< 36,000	5.4 (4)	2.3 (1)	10.0 (3)
36,000–50,999	9.5 (7)	6.8 (3)	13.3 (4)
51,000–75,999	22.9 (17)	13.6 (6)	36.7 (11)
76,000–100,000	22.9 (17)	25.0 (11)	20.0 (6)
> 100,000	39.3 (29)	52.3 (23)	20.0 (6)
Parent Education			
High School	11.7 (9)	6.7 (3)	18.8 (6)
2-year, Vocational, etc.	10.4 (8)	4.4 (2)	18.8 (6)
Bachelor's Degree	42.9 (33)	42.2 (19)	43.7 (14)
Master's Degree	22.1 (17)	28.9 (13)	12.5 (4)
Doctoral Degree	12.9 (10)	17.8 (8)	6.2 (2)
Sleep: Continuous (mean (SD))			
Sleep efficiency (%)	83.5 (4.1)	83.2 (4.1)	83.9 (4.2)
Sleep fragmentation index	27.3 (5.1)	28.2 (5.4)	26.2 (4.6)
Fragmentation index	13.4 (3.5)	13.7 (3.5)	12.9 (3.5)
Wake after sleep onset (min)	91.1 (25.2)	94.0 (26.1)	87.0 (23.7)
Total sleep time (min)	578.7 (33.7)	584.6 (33.0)	570.5 (33.5)
Eating Behaviors (mean (SD)			
Meal intake (kcal)	584.1 (255.9)	562.9 (238.2)	614.0 (280.0)
EAH intake (kcal)	271.1 (130.3)	254.2 (129.3)	294.9 (130.1)

** Significantly different via t-test, p<0.01;

[a] Determined from Schofield equation;

[b]Missing data for 3 families who did not report income. Abbreviations: EAH – Eating in the Absence of Hunger

Table 4.

Associations of sleep quality measures with laboratory intakes (n=77)

		Intake at *ad libitum* meal				Intake at EAH paradigm			
		Model 1 [a]		Model 2 [b]		Model 1 [a]		Model 2 [b]	
Sleep exposure	**Diet outcome**	**Correlation (*r*)**	***p*** [d]	**Partial corr. (*r*)**	***p*** [d]	**Correlation (*r*)**	***p*** [d]	**Partial corr. (*r*)**	***p*** [d]
Sleep efficiency (%)	Total kcal	−0.17	0.13	−0.02	0.10	0.13	0.25	0.12	0.34
	% CHO	0.15	0.20	0.14	0.25	−0.06	0.60	−0.06	0.64
	% FAT	0.03	0.79	0.00	0.99	0.06	0.60	0.07	0.58
	% PRO	−0.19	0.10	−0.17	0.16	0.04	0.71	−0.02	0.87
Fragment. index	Total kcal	0.25	0.05	0.23	0.06	−0.18	0.13	−0.20	0.10
	% CHO	−0.12	0.30	−.0.14	0.24	0.33	0.004	0.32	0.003
	% FAT	0.14	0.22	0.21	0.09	−0.33	0.004	−0.32	0.003
	% PRO	−0.02	0.89	−0.06	0.65	−0.27	0.02	−0.26	0.03
Wake after sleep onset (min)	Total kcal	0.18	0.12	0.20	0.10	−0.12	0.31	−0.10	0.42
	% CHO	−0.10	0.38	−0.15	0.24	0.06	0.61	0.05	0.68
	% FAT	−0.01	0.93	−0.02	0.88	−0.05	0.64	−0.06	0.62
	% PRO	0.12	0.29	0.15	0.21	−0.08	0.48	0.02	0.85
Total sleep time (min) [c]	Total kcal	0.08	0.49	0.12	0.32	−0.07	0.56	−0.01	0.92
	% CHO	0.17	0.14	0.10	0.43	0.03	0.83	−0.01	0.92
	% FAT	−0.01	0.93	−0.07	0.57	0.00	0.99	0.04	0.77
	% PRO	−0.18	0.13	−0.04	0.75	−0.17	0.13	−0.14	0.25

[a] Results of unadjusted Pearson's correlations;

[b] Results of partial correlations adjusted for estimated BMR, average liking of foods, pre-meal fullness, family income, and total sleep time.

[c] Results of partial correlations for *total sleep time* adjusted for estimated BMR, average liking of foods, pre-meal fullness, and family income.

[d] Adjusted for false discovery rate.