Acute sleep restriction increases dietary intake in preschool-age children

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SUMMARY

Epidemiological findings suggest short sleep duration is associated with overweight and obesity across the lifespan. In adults, experimental sleep loss increases caloric intake more than total daily energy needs, thus leading to weight gain. To date, little is known about the relationship between sleep restriction and dietary intake in preschool children. Healthy children ($n = 10$; 41.2 ± 5.4 months; 5 females) followed a strict sleep schedule for 5 days before each experimental condition: 1 day of baseline sleep (nap and scheduled bedtime/wake time) and 1 day of sleep restriction (no-nap and ~2.3 h bedtime delay). Standardized parent-report dietary intake measures were obtained on baseline, sleep restriction and sleep recovery (*ad libitum* sleep opportunity in the 24-h following sleep restriction) days. As designed, children slept ~3 h less on the sleep restriction than the baseline day ($P < 0.001$), with no significant differences in sleep between baseline and recovery days (verified with actigraphy). Repeated-measures ANOVAs indicated differences across conditions in total kilocalories, sugar, carbohydrate and fat intake (all $P < 0.05$; no differences in protein). *Post hoc* tests revealed that compared with baseline, children consumed 21% more kilocalories, 25% more sugar and 26% more carbohydrates on the day of sleep restriction, as well as 14% more kilocalories and 23% more fat on the day of sleep recovery (all $P < 0.05$). Findings suggest that acute sleep loss increases dietary intake in preschoolers both on the day of and the day after sleep restriction. Increased kilocalorie intake may promote weight gain over time and be a mechanism through which short sleep contributes to childhood obesity risk.

INTRODUCTION

Obesity is a complex medical condition that in recent decades has grown to epidemic status worldwide. In 2011–2012, 32% of American youth were either overweight or obese, and 17% were considered obese (Ogden *et al.*, 2014). Childhood obesity is associated with increased risk for chronic illnesses (e.g. pulmonary, endocrine diseases), persistence of obesity into adulthood (Ebbeling *et al.*, 2002; Must *et al.*, 1999) and psychosocial consequences, including the development of a negative self-image and low self-esteem (Ebbeling *et al.*, 2002).

Meta-analysis of epidemiological data provides strong evidence of associations between obesity risk and insufficient sleep: children are twice as likely to be obese when experiencing short sleep (Cappucio *et al.*, 2008). In one prospective study, short sleep duration (<10.5 h) at 3 years of age was associated with an increased risk of obesity at age 7 years (Reilly *et al.*, 2005). Also, experimental sleep restriction increases kilocalorie intake and the overconsumption of highly palatable 'comfort foods' (i.e. high fat, sugar, carbohydrate), which over time promotes weight gain in adolescents (Beebe *et al.*, 2013) and adults (Dallman *et al.*, 2003; Markwald *et al.*, 2013). Experimental studies

examining sleep restriction and dietary intake in young children are scarce. Furthermore, we know of little data on the time course associated with the resumption of normal diet following sleep loss, but published findings suggest neurobehavioural deficits and hormonal imbalances are persistent for 1–2 nights after sleep restriction in adults (Drummond and Brown, 2001; Lamond *et al.*, 2007).

Early childhood is a sensitive period in the development of sleep and eating habits (Kelder *et al.*, 1994). Longitudinal data show that body mass index (BMI) has moderate-to-strong lifetime stability, as overweight youth are ~ 4 times more likely to maintain this status as adults than their normal-weight peers (Cunningham *et al.*, 2014). Sleep changes rapidly during early childhood (e.g. decrease in 24-h sleep duration, which is primarily attributed to a decline in daytime napping; Weissbluth, 1995). Also, ~ 30% of preschool children reportedly do not obtain adequate sleep (National Sleep Foundation, 2004). Insufficient sleep in early childhood is related to poor self-regulation (Miller *et al.*, 2015), which may promote the consumption of obesogenic foods (Pieper and Laugero, 2013) and rapid childhood weight gain (Francis and Susman, 2009). Lack of sleep has also been shown to disrupt appetite-regulating hormones (e.g. ghrelin, leptin) and thereby alter hunger when food intake is controlled (Spiegel *et al.*, 2004). We propose that dietary intake is a primary yet unexplored mechanism through which insufficient sleep may contribute to early childhood overweight/obesity risk.

In this study, we tested sleep-dependent changes in preschool children's dietary intake. Specifically, we examined the effects of a missed nap and a delayed bedtime on dietary intake at baseline compared to the day of and the day following acute sleep restriction. Based on studies of sleep restriction and dietary intake in adults, we hypothesized that in comparison to a baseline day of sleep, children would have greater overall energy intake and consume more sugar, fat and carbohydrates than during a day of acute sleep restriction. Because little is known about the changes in dietary intake following sleep loss, our analysis of differences in dietary outcomes during the 24-h recovery day was exploratory.

MATERIALS AND METHODS

Participants

Participants were 10 healthy, habitually napping children (ages 32–47 months; 41.2 ± 5.4 months; 5 females) of normal weight (BMI *z*-score −0.03 ± 1.4). Three additional children were enrolled but excluded for not falling asleep during their baseline nap opportunity. Participant recruitment and screening details have been previously published (Berger *et al.*, 2012). The University of Colorado Boulder Institutional Review Board approved all procedures, and the study was performed according to the Declaration of Helsinki. Families provided written consent and were compensated with cash. This study utilized children from a larger protocol designed to examine sleep, circadian rhythms and emotion processing in early childhood.

Protocol

Dietary recalls were completed on baseline, sleep restriction and sleep recovery days (baseline and sleep restriction condition days were counterbalanced; Fig. 1). Children followed a strict individualized daytime nap and night-time sleep schedule for ≥5 days before completing the sleep and dietary assessments. If the child had an accidental nap, a bedtime or wake time that deviated >15 min from the sleep schedule, illness, medications affecting sleep/alertness, or caffeine consumption, assessments were cancelled and rescheduled after an additional 5 consecutive days on the sleep schedule. For the baseline sleep condition, children maintained their individualized daytime nap and night-time sleep schedule. For the sleep restriction condition, children missed their nap and went to bed ~2.3 h (range: 1.1–3.1 h) past their scheduled bedtime and were awakened at their habitually scheduled time. For the 24-h sleep recovery condition, napping and night-time sleep was *ad libitum*. Sleep occurred in children's homes or at daycare/preschool on the days before the sleep and dietary intake assessments and at home on the day of the assessments. Sleep was tracked with actigraphy, sleep diaries and daily telephone calls/emails to researchers. Parents were instructed to refrain from providing foods and drinks containing caffeine, but were not given any additional instructions regarding their child's diet.

Measures

Sleep diary

Parents completed a daily sleep diary that inquired about times that the actigraph was off-wrist, night-time and nap time

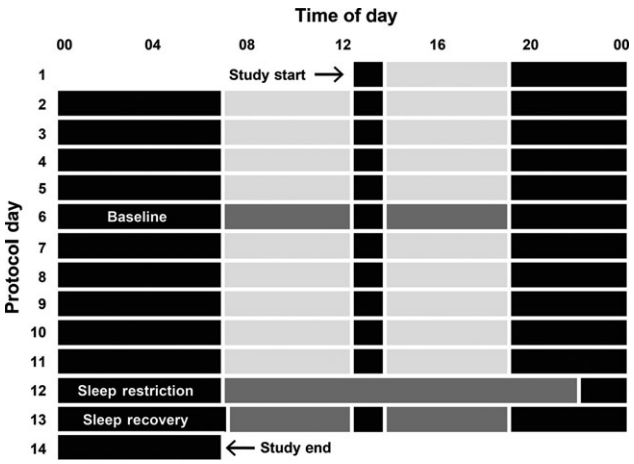

Figure 1. Individualized daytime nap and night-time sleep protocol for one child participant. Black bars indicate time in bed; grey bars indicate dietary recall assessments (baseline and sleep restriction conditions were counterbalanced).

sleep times, and sleep-onset latency (Akacem *et al.*, 2015). Sleep diaries were used to ensure compliance with the study protocol and to verify actigraphy data (Acebo *et al.*, 2005).

Actigraphy

Children wore an actigraph (AW Spectrum; Philips/Respironics, Pittsburg, PA, USA) on their non-dominant wrist. Continuous epoch-by-epoch (1 min) estimates of sleep or wakefulness were obtained using Respironics Actiware V5 software (Philips/Respironics, Pittsburg, PA, USA). We used our published laboratory procedures for establishing sleep intervals based upon diary reports completed by parents and actigraph event markers indicating 'lights-off' and 'lights-on' (Berger *et al.*, 2012). For each nap and night-time sleep interval, 3 actigraph variables were derived: (a) time in bed: min from lights-off to lights-on; (b) sleep period (duration; min from sleep start to sleep end); and (c) sleep efficiency (quality; % of sleep epochs between sleep start and sleep end time). When data were excluded or lost due to technical failure (6% of nights; 10% of naps), parent-reported data from the sleep diary were substituted. Although not a primary study outcome, we used actigraphy data to verify compliance to the sleep schedule and to measure children's sleep duration and quality during the 5 days before and the day of dietary assessments.

Dietary recall

Parents recorded all food and beverages consumed by their child using a pencil-and-paper food log on the baseline, sleep restriction and sleep recovery days. Children did not attend preschool or daycare on these days, thereby permitting continuous monitoring. Portion sizes, brand names and quantities for each food and beverage were recorded. Parents estimated portion sizes using household measures (e.g. grams, cups, teaspoons) and weights from food package labels. For homemade dishes, parents recorded ingredients, quantities and cooking methods. On the day after each dietary assessment, a researcher confirmed recorded data with parents and entered the child's food and beverage intake into the online system ASA24 (Subar *et al.*, 2012). This system is based upon the USDA Automated Multiple-Pass Method, which has been validated and shown to accurately estimate mean total kilocalorie and protein intakes compared with recovery biomarkers (i.e. doubly-labelled water/24-h urine collection protocol; Kipnis *et al.*, 2003; Moshfegh *et al.*, 2008; Subar *et al.*, 2012). It provides a comprehensive output of total daily energy, macronutrient, vitamin and mineral intake. We chose to analyse total kilocalorie, sugar, fat, carbohydrate and protein intake based upon prior data in adults showing sleep-dependent changes in these variables (Bosy-Westphal *et al.*, 2008; Nedeltcheva *et al.*, 2009; St-Onge *et al.*, 2011; Weiss *et al.*, 2010).

Analysis

Data analysis was performed with IBM SPSS Statistics Package 22.0 (IBM, Armonk, NY, USA). Paired *t*-tests compared sleep variables during the 5 days before baseline and sleep restriction assessments. Repeated-measures ANOVAs examined differences in sleep variables and dietary intake measures between the baseline, restriction and recovery days. Following a significant *F*-value, pairwise comparisons between the three conditions were made with a Bonferroni correction factor for multiple comparisons (one-tailed paired *t*-tests for baseline and sleep restriction planned comparisons; two-tailed paired *t*-tests for baseline and sleep recovery comparisons). Effect size was quantified as Cohen's *d* or eta^2. The alpha level was set at 0.05.

RESULTS

Sleep protocol verification

We found no differences between sleep measures during the 5 days before the baseline and sleep restriction assessments (Table 1). As designed, children missed their nap and went to bed later on the sleep restriction day and, thus, they had a shorter 24-h time in bed and 24-h sleep duration compared

Table 1 Descriptive statistics (*M, SD*) of actigraphic sleep variables during the 5 days before counterbalanced baseline and sleep restriction assessments

	Pre-baseline		*Pre-restriction*		*Statistics*		
	M	*SD*	*M*	*SD*	*t*	*d*	*P*
Night lights-out time	20:07	0:27	20:06	0.5	0.31	0.05	0.77
Morning wake time	6:45	0:34	6:45	0.55	−0.02	0.00	0.99
Nap lights-out time	13:05	0:24	13:01	0.57	0.66	0.13	0.53
Nap wake time	14:56	0:32	14:52	0.78	0.46	0.09	0.65
Time in bed (min)	735.0	44.4	728.5	34.6	0.40	0.16	0.70
Sleep period (min)	662.8	33.2	671.4	28.2	−1.03	−0.28	0.33
Sleep efficiency (%)	89.1	2.9	88.5	4.2	0.47	0.16	0.65

t, d and *P* are for the main effect of condition.

Table 2 Descriptive statistics (*M, SD*) for actigraphic sleep variables on the day of dietary assessments for baseline, sleep restriction and sleep recovery conditions

	Baseline		*Restriction*		*Recovery*		*Statistics*		
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*	*eta²*	*P*
Night lights-out time[a,c]	20:16	0:36	22:35	0:33	20:21	1:02	65.81	0.88	<0.001
Morning wake time[c]	6:42	0:42	6:47	0:39	7:09	0:39	5.13	0.36	0.02
Nap lights-out time	13:11	0:31	–	–	13:17	1:04	–	–	–
Nap wake time	15:02	0:31	–	–	15:29	1:29	–	–	–
Time in bed (min)[a,c]	735.7	58.0	509.1	23.7	776.2	63.0	122.7	0.94	<0.001
Sleep duration (min)[a,c]	674.4	43.8	489.0	29.3	707.4	53.9	78.2	0.92	<0.001
Sleep efficiency (%)	90.2	2.8	88.1	5.4	89.2	5.0	0.9	0.11	0.41

F, eta² and *P* are for the main effect of condition.
Significant *post hoc* pairwise comparisons are denoted as [a](baseline versus restriction), [b](baseline versus recovery) and [c](restriction versus recovery).

with baseline and recovery days (Table 2). We also observed that children awakened significantly later on the recovery compared with the sleep restriction day ($d = 0.53$; Table 2), with a non-significant trend for a later morning wake time on the recovery compared with the baseline day ($d = 0.66$, $P = 0.14$). Sleep efficiency was similar across all sleep conditions.

Dietary intake

Repeated-measures ANOVAs showed differences between conditions in total kilocalorie ($F = 5.43$; $P < 0.05$; $\text{eta}^2 = 0.34$), sugar ($F = 7.85$; $P < 0.05$; $\text{eta}^2 = 0.47$), carbohydrate ($F = 5.54$; $P < 0.05$; $\text{eta}^2 = 0.38$) and fat ($F = 5.72$; $P < 0.05$; $\text{eta}^2 = 0.39$) intake. Figure 2 shows that compared with baseline, children consumed 21% more kilocalories ($d = 0.95$), 25% more sugar ($d = 0.93$) and 26% more carbohydrates ($d = 0.93$) than on the day of sleep restriction, and 14% more kilocalories ($d = 0.56$) and 23% more fat ($d = 0.73$) than on the day of sleep recovery (all $P < 0.05$). We found no differences in protein intake across conditions.

DISCUSSION

In this study, we implemented a well-controlled experimental protocol to examine the immediate and recovery effects of acute sleep restriction on dietary intake in children. Compared with a baseline day of habitual sleep duration, acute sleep restriction (i.e. missed nap, delayed bedtime of ~2.3 h, habitual wake time) resulted in a significant increase in total kilocalorie, sugar and carbohydrate 24-h intake, with no observed differences in fat and protein intake. During the immediate recovery day of 24-h *ad libitum* sleep, overall kilocalorie intake remained elevated and fat intake was increased relative to baseline, whereas sugar and carbohydrate intake returned to baseline levels. No differences in protein intake were observed on the recovery compared to the baseline day.

These findings complement and extend the results of prior studies. Specifically, we observed a substantial increase in kilocalorie intake on the day of a missed nap and a late bedtime, which aligns with findings in adults and adolescents (Beebe *et al.*, 2013; Markwald *et al.*, 2013). Data from sleep restriction studies using whole-room calorimeters to assess 24-h energy expenditure provide support for the hypothesis that increased food intake during sleep loss represents a physiological adaptation to provide the body with the energy needed to sustain extended wakefulness; however, when food is readily available, overconsumption may ensue. When daily energy intake is greater than daily energy expenditure, a state of positive energy balance occurs, and over even a short period of time, sustained positive energy balance results in weight gain (Hill and Melanson, 1999). We did not measure 24-h energy expenditure, and therefore cannot conclude that participants were in a state of positive energy balance due to the observed increase in kilocalorie intake. Such studies in children are needed to determine how much sleep restriction is needed to increase 24-h energy expenditure and whether or not the well-known hyperactivity associated with sleep loss in childhood (Touchette *et al.*, 2009) elevates 24-h energy expenditure even more than that observed in adults.

Our experimental data provide novel insights into the macronutrient composition of dietary intake in response to acute sleep restriction in preschool children. In adults, data from studies utilizing acute and chronic sleep loss protocols to measure changes in macronutrient intake are inconsistent. Some studies illustrate that sleep restriction is associated with an increase in carbohydrate-rich foods (Bosy-Westphal *et al.*, 2008; Markwald *et al.*, 2013; Nedeltcheva *et al.*, 2009; Weiss *et al.*, 2010), whereas others show an increase in dietary fat (St-Onge *et al.*, 2011). We found that children's initial response to missing a nap and going to bed at a later clock time is an increase in carbohydrate and sugar intake. This selective overconsumption of foods high in sugar and carbohydrates may be particularly detrimental for overweight/

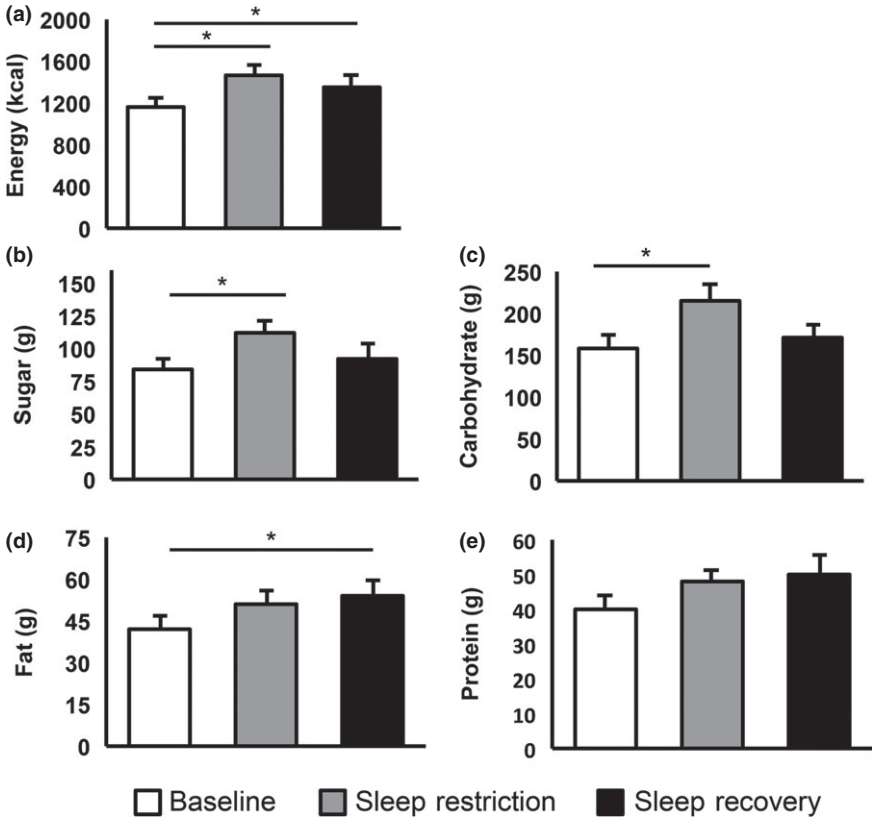

Figure 2. Average (a) energy, (b) sugar, (c) carbohydrate, (d) fat, and (e) protein intake between baseline, sleep restriction and sleep recovery conditions. Error bars represent ±SE. *Indicates significant *post hoc* pairwise comparison with Bonferroni correction.

obesity risk. Experimental sleep restriction results in impaired glucose tolerance and decreased insulin sensitivity (as reviewed in Knutson, 2007). Poor glucose regulation during sleep loss, in combination with our findings of selective increases in sugar and carbohydrate consumption, may further explain the relationship between reports of insufficient sleep and childhood obesity.

With regard to recovery after acute sleep loss, we found sustained effects on dietary intake when children were allowed to sleep *ad libitum* including their daytime nap. On the recovery day, kilocalorie intake remained higher (14%) than baseline. Specifically, fat intake increased from baseline, whereas sugar and carbohydrate intake returned to baseline levels. Little research has examined the changes in dietary composition during the first day of recovery from sleep loss. One relevant study (Brondel *et al*., 2010) found that study participants consumed 22% more kilocalories with an 8% increase in energy from fat in the 24-h period following 4 h acute sleep restriction compared with 8 h of sleep. These data align with the results of our experimental study and support the hypothesis that the first recovery day following sleep restriction is associated with increased dietary intake. Additionally, data from a recent study utilizing a simulated shiftwork protocol indicate that total daily fat utilization increases on the transition day from a stable night-time sleep schedule to the first night shift, as well as the second night shift. Fat utilization, however, was not increased on the third consecutive nightshift (McHill *et al*., 2014). These results may help to mechanistically explain the observed increased fat intake during immediate recovery sleep in our preschool sample. Following times of acute prolonged wakefulness and restricted sleep, physiological changes that upregulate the body's usage of fat ensue, which may lead to an increased drive to consume high-fat foods to meet this need. Notably, when chronically sleep-restricted individuals (5 days, 5 h nightly sleep opportunity) were allowed 5 days of adequate sleep, consumption of fats, carbohydrates and overall energy returned to baseline, leading to weight loss (Markwald *et al*., 2013). Further research addressing the pathways and specific time course associated with recovery in increased dietary intake in response to sleep restriction is necessary.

Although the mechanisms underlying relationships between sleep restriction and increased dietary intake are not fully understood, several physiological and behavioural changes associated with sleep loss may inform our observed effects. Levels of the appetitive hormones including leptin and ghrelin are disrupted during sleep restriction. Leptin is an appetite-inhibiting hormone that promotes feelings of satiety, while ghrelin is an appetite-stimulating peptide that promotes food intake (as reviewed in Knutson, 2007). In times of acute sleep loss, levels of both hormones are altered, facilitating

the drive to eat (as reviewed in Knutson, 2007). For example, Spiegel *et al.* (2004) showed that sleep restriction during controlled food intake in adults was associated with an 18% decrease in leptin, a 28% increase in ghrelin and a 33% increase in hunger for calorie-dense foods with higher carbohydrate content. Notably, Hart *et al.* (2013) showed that with increased sleep (1 week, 1.5 h increased time in bed), children (8–11 years) consumed fewer kilocalories and exhibited lower fasting morning leptin, with no difference in fasting ghrelin compared with the decreased sleep condition (1 week, 1.5 h less time in bed than baseline). Sleep restriction can also compromise or interfere with self-regulation (Miller *et al.*, 2015). Poor self-regulation (i.e. inability to delay gratification for food) in children has been associated with rapid weight gain and proposed as a pathway to obesity risk (Francis and Susman, 2009). Taken together, the hormonal disruptions and changes in neuronal activation occurring during sleep loss may in part explain our findings of increased dietary intake in response to sleep restriction in early childhood.

This experimental study represents a first step in understanding sleep-dependent effects on dietary intake in preschoolers, a sensitive window in the development of eating behaviour and a time during which early overweight/obesity risk trajectories emerge. Nonetheless, several limitations should be noted. First, our data are from a relatively small sample of healthy children without sleep disturbances, behavioural problems or overweight/obesity, which is a threat to external validity. Future research using experimental protocols (e.g. sleep restriction and/or sleep extension) from a larger community sample or cohorts of children at-risk for overweight/obesity is needed to extend the generalizability of our data. Second, we examined dietary intake in the child's natural home environment, which may be influenced by familial food preferences and attitudes; however, our repeated-measures design allowed each child to be his/her own control. Third, our protocol involved only a single night of sleep restriction; thus, it is unclear whether chronic sleep restriction would lead to long-term changes in dietary intake in young children. Furthermore, the inherent variability in dietary recalls and parental underreporting of foods via observational methods should be considered (Baranowski *et al.*, 1991). Although we utilized an established and detailed pencil-and-paper log of food and beverage intake that was reviewed the following day with a researcher, we believe future studies should use more controlled dietary intake protocols that regulate the types of food available, as well as quantify energy expenditure and dietician-measured food intake to assess energy balance. Protocols that assess the effects of sleep deprivation on accurate downregulation of intake in response to a caloric preload (Johnson, 2000), eating in the absence of hunger (Fisher and Birch, 2002), the ability to delay gratification for food (Francis and Susman, 2009), or the relative reinforcing value of food (Epstein *et al.*, 2007) may all be valuable. Finally, we found only a relatively small increase in sleep duration on the recovery versus the baseline day, which may reflect strong circadian entrainment as a result of children following a strict sleep-wakefulness schedule before each condition. Although not statistically significant with this sample size, a 33 min increase in sleep duration is consistent with a higher homeostatic sleep drive as a result of our experimental protocol, and future work should examine a larger number of children and use a more sensitive electroencephalography marker such as slow-wave activity.

In summary, our findings suggest that sleep restriction in early childhood may promote risk for weight gain and, thus over time, the development of childhood overweight and obesity. Insufficient sleep in preschool children is prevalent (National Sleep Foundation, 2004), which may arise from both daytime and night-time sleep loss (i.e. missed naps, bedtime delays). Our experimental simulation of this sleep restriction pattern resulted in increased dietary intake, which suggests this may be an important mechanism underlying associations between insufficient sleep and childhood overweight/obesity. The implications of our data are substantial when considering the cumulative effects of overeating. Nutritional status between conception and age 5 years is strongly associated with growth and development (Katz *et al.*, 2003) and future overweight (Herman *et al.*, 2009). Therefore, understanding how sleep loss may contribute to obesogenic eating behaviours early in development may inform the design of childhood obesity prevention programming.

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AUTHOR CONTRIBUTIONS

ENM, SC, ALM, JCL, KPW and MKL designed the research; ENM and SC performed the research; ENM and MKL analysed the data; ENM and MKL drafted the initial manuscript; ENM, ALM, JCL, KPW, SK and MKL reviewed and revised the manuscript drafts; all authors approved the final manuscript.

REFERENCES

Acebo, C., Sadeh, A., Seifer, R., Tzischinsky, O., Hafer, A. and Carskadon, M. A. Sleep/wake patterns derived from activity monitoring and maternal report for healthy 1- to 5-year-old children. *Sleep*, 2005, 28: 1568–1577.

Akacem, L. D., Simpkin, C. T., Carskadon, M. A. *et al.* The timing of the circadian clock and sleep differ between napping and non-napping toddlers. *PLoS ONE*, 2015, 10: e0125181.

Baranowski, T., Sprague, D., Baranowski, J. H. and Harrison, J. A. Accuracy of maternal dietary recall for preschool children. *J. Am. Diet. Assoc*., 1991, 91: 669–674.

Beebe, D. W., Simon, S., Summer, S., Hemmer, S., Strotman, D. and Dolan, L. M. Dietary intake following experimentally restricted sleep in adolescents. *Sleep*, 2013, 36: 827–834.

Berger, R. H., Miller, A. L., Seifer, R., Cares, S. R. and LeBourgeois, M. K. Acute sleep restriction effects on emotion responses in 30- to 36-month-old children. *J. Sleep Res*., 2012, 21: 235–246.

Bosy-Westphal, A., Hinrichs, S., Jauch-Chara, K. *et al.* Influence of partial sleep deprivation on energy balance and insulin sensitivity in healthy women. *Obes. Facts*, 2008, 1: 266–273.

Brondel, L., Romer, M. A., Nougues, P. M., Touyarou, P. and Davenne, D. Acute partial sleep deprivation increases food intake in healthy men. *Am. J. Clin. Nutr*., 2010, 91: 1550–1559.

Cappucio, F. P., Taggart, F. M., Kandala, N. B. *et al.* Meta-analysis of short sleep duration and obesity in children and adults. *Sleep*, 2008, 31: 619–626.

Cunningham, S. A., Kramer, M. R. and Narayan, K. M. Incidence of childhood obesity in the United States. *N. Engl. J. Med*., 2014, 370: 403–411.

Dallman, M. F., Pecoraro, N., Akana, S. F. *et al.* Chronic stress and obesity: a new view of "comfort food". *Proc. Natl Acad. Sci. USA*, 2003, 100: 11696–11701.

Drummond, S. P. and Brown, G. G. The effects of total sleep deprivation on cerebral responses to cognitive performance. *Neuropsychopharmacology*, 2001, 25: S68–S73.

Ebbeling, C. B., Pawlak, D. B. and Ludwig, D. S. Childhood obesity: public-health crisis, common sense cure. *Lancet*, 2002, 360: 473–482.

Epstein, L. H., Leddy, J. J., Temple, J. L. and Faith, M. S. Food reinforcement and eating: a multilevel analysis. *Psychol. Bull*., 2007, 133: 884–906.

Fisher, J. O. and Birch, L. L. Eating in the absence of hunger and overweight in girls from 5 to 7 y of age. *Am. J. Clin. Nutr*., 2002, 76: 226–231.

Francis, L. A. and Susman, E. J. Self-regulation and rapid weight gain in children from age 3 to 12 years. *Arch. Pediatr. Adolesc. Med*., 2009, 163: 297–302.

Hart, C. N., Carskadon, M. A., Considine, R. V. *et al.* Changes in children's sleep duration on food intake, weight, and leptin. *Pediatrics*, 2013, 132: e1473–e1480.

Herman, K. M., Craig, C. L., Gauvin, L. and Katzmarzyk, P. T. Tracking of obesity and physical activity from childhood to adulthood: the Physical Activity Longitudinal Study. *Int. J. Pediatr. Otorhinolaryngol*., 2009, 4: 281–288.

Hill, J. O. and Melanson, E. L. Overview of the determinants of overweight and obesity: current evidence and research issues. *Med. Sci. Sports Exerc*., 1999, 31: S515–S521.

Johnson, S. L. Improving preschoolers' self-regulation of energy intake. *Pediatrics*, 2000, 106: 1429–1435.

Katz, D., Yeh, M.-C., Webb, K. Nutrition, early childhood. In: T. Gullotta, M. Bloom, J. Kotch. C. Blakely (Eds) *Encyclopedia of Primary Prevention and Health Promotion*. New York, NY, USA: Kluwer Academic/Plenum Publishers, 2003: 713–721.

Kelder, S. H., Perry, C. L., Klepp, K. I. and Lytle, L. L. Longitudinal tracking of adolescent smoking, physical activity, and food choice behaviors. *Am. J. Public Health*, 1994, 84: 1121–1126.

Kipnis, V., Subar, A. F., Midthune, D. *et al.* Structure of dietary measurement error: results of the OPEN Biomarker Study. *Am. J. Epidemiol*., 2003, 158: 14–21.

Knutson, K. L. Impact of sleep and sleep loss on glucose homeostasis and appetite regulation. *Sleep Med. Clin*., 2007, 2: 187–197.

Lamond, N., Jay, S. M., Dorrian, J., Ferguson, S. A., Jones, C. and Dawson, D. The dynamics of neurobehavioural recovery following sleep loss. *J. Sleep Res*., 2007, 16: 33–41.

Markwald, R. R., Melanson, E. L., Smith, R. S. *et al.* Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. *Proc. Natl Acad. Sci. USA*, 2013, 110: 5696–5700.

McHill, A. W., Melanson, E. L., Higgins, J. *et al.* Impact of circadian misalignment on energy metabolism during simulated nightshift work. *Proc. Natl Acad. Sci. USA*, 2014, 111: 17302–17307.

Miller, A. L., Seifer, R., Crossin, R. and Lebourgeois, M. K. Toddler's self-regulation strategies in a challenge context are nap-dependent. *J. Sleep Res*., 2015, 24: 279–287.

Moshfegh, A. J., Rhodes, D. G., Baer, D. J. *et al.* The US Department of Agriculture automated multiple-pass method reduces bias in the collection of energy intakes. *Am. J. Clin. Nutr*., 2008, 88: 324–332.

Must, A., Spadano, J., Coakley, E. H., Field, A. E., Colditz, G. and Dietz, W. H. The disease burden associated with overweight and obesity. *J. Am. Med. Assoc*., 1999, 282: 1523–1529.

National Sleep Foundation. Sleep in America Poll: Children and Sleep. 2004. https://sleepfoundation.org/sleep-polls-data/sleep-in-america-poll/2004-children-and-sleep.

Nedeltcheva, A. V., Kilkus, J. M., Imperial, J., Kasza, K., Schoeller, D. A. and Penev, P. D. Sleep curtailment is accompanied by increased intake of calories from snacks. *Am. J. Clin. Nutr*., 2009, 89: 126–133.

Ogden, C. L., Carroll, M. D., Kit, B. K. and Flegal, K. M. Prevalence of childhood and adult obesity in the United States, 2011–2012. *J. Am. Med. Assoc*., 2014, 311: 806–814.

Pieper, J. R. and Laugero, K. D. Preschool children with lower executive function may be more vulnerable to emotional-based eating in the absence of hunger. *Appetite*, 2013, 62: 103–109.

Reilly, J. J., Armstrong, J., Dorosty, A. R. *et al.* Early life risk factors for obesity in childhood: cohort study. *BMJ*, 2005, 330: 1357.

Spiegel, K., Tasali, E., Penev, P. and Cauter, E. V. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Ann. Intern. Med*., 2004, 141: 846–850.

St-Onge, M.-P., Roberts, A. L., Chen, J. *et al.* Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals. *Am. J. Clin. Nutr*., 2011, 94: 410–416.

Subar, A. F., Kirkpatrick, S. I., Mittl, B. *et al.* The automated self-administered 24-hour dietary recall (ASA24): a resource for researchers, clinicians, and educators from the National Cancer Institute. *J. Acad. Nutr. Diet*., 2012, 112: 1134–1137.

Touchette, E., Cote, S. M., Petit, D. *et al.* Short nighttime sleep-duration and hyperactivity trajectories in early childhood. *Pediatrics*, 2009, 124: e985–e993.

Weiss, A., Xu, F., Storfer-Isser, A., Thomas, A., Ievers-Landis, C. E. and Redline, S. The association of sleep duration with adolescents' fat and carbohydrate consumption. *Sleep*, 2010, 33: 1201–1209.

Weissbluth, M. Naps in children: 6 months–7 years. *Sleep*, 1995, 18: 82–87.