ORIGINAL ARTICLE

Short Sleep Duration Is Associated With Eating More Carbohydrates and Less Dietary Fat in Mexican American Children

Suzanna M. Martinez, PhD[1]; Jeanne M. Tschann, PhD[2]; Nancy F. Butte, PhD[3]; Steve E. Gregorich, PhD[4]; Carlos Penilla, MS[5]; Elena Flores, PhD[6]; Louise C. Greenspan, MD[7]; Lauri A. Pasch, PhD[8]; Julianna Deardorff, PhD[9]

[1]Nutrition Policy Institute, Division of Agriculture and Natural Resources, University of California, Berkeley, CA; [2]Department of Psychiatry, University of California at San Francisco, San Francisco, CA; [3]Department of Pediatrics, USDA/ARS Children's Nutrition Research Center, Baylor College of Medicine, Houston, TX; [4]Department of Medicine, University of California at San Francisco, San Francisco, CA; [5]School of Public Health, University of California at Berkeley, Berkeley, CA; [6]Counseling Psychology Department, School of Education, University of San Francisco, San Francisco, CA; [7]Kaiser Permanente, San Francisco, CA; [8]Department of Psychiatry, University of California at San Francisco, San Francisco, CA; [9]Division of Community Health and Human Development, School of Public Health, University of California at Berkeley, Berkeley, CA

Study Objective: Short sleep duration is a risk factor for childhood obesity. Mechanisms are unclear, but may involve selection of high carbohydrate foods. This study examined the association between estimated sleep duration and macronutrient intake as percentages of total energy among Mexican American (MA) 9–11 year olds.

Methods: This cross-sectional study measured diet using two 24-hour recalls and estimated sleep duration using hip-worn accelerometry in MA children (n = 247) who were part of a cohort study. Child and maternal anthropometry were obtained; mothers reported on demographic information. Using linear regression, we examined the relationship of sleep duration with energy intake, sugar intake, and the percentage of energy intake from carbohydrates, fat, and protein.

Results: Children were 47% male; mean age was 10 (SD = 0.9) years. Mean sleep duration was 9.6 (SD = 0.8) hours; 53% were overweight/obese, with a mean energy intake of 1759 (SD = 514) calories. Longer sleep duration was independently associated with a lower percentage of energy intake from carbohydrates (β = −0.22, p < .01) and a higher percentage of energy from fat (β = 0.19, p < .01), driven by the percentage of energy from polyunsaturated fatty acids (PUFA; β = 0.17, p < .05). No association was found with the intake of energy or total sugars, or the percent of calories from protein.

Conclusions: MA children who slept longer consumed diets with a lower percentage of calories from carbohydrates and a higher percentage from fat, especially from PUFA. Short sleep duration may be a risk factor for food cravings that are high in carbohydrate content and may displace heart-healthy dietary fat, and thereby increase obesity risk among children.

Keywords: Sleep duration, diet, carbohydrates, fat, Mexican American children, accelerometer.

Statement of Significance

In free-living Mexican American children, longer sleep duration was associated with less energy intake from carbohydrates and more energy intake from fat. Longer sleep duration may play a protective role in obesity prevention. Longitudinal research is needed to investigate sleep duration impacts diet and appetite during childhood and the long-term impact on obesity.

INTRODUCTION

Childhood obesity remains a public health concern. US Latino children, the fastest growing segment of the US population, remain a vulnerable population with high obesity rates; nearly 40% of Latino children are overweight/obese.[1,2] The severity of pediatric obesity in this population has important implications such as lifelong obesity risk, metabolic syndrome as adolescents, and diabetes.[3] In a recent study of Mexican American (MA) children, short sleep duration was a risk factor for obesity.[4] Understanding the relationship between sleep duration and dietary intake and behaviors is necessary to help develop new interventions that could be beneficial in preventing childhood obesity. For Latino children, a clearer understanding of this information could help to improve how obesity is addressed.

The National Sleep Foundation and the American Academy of Pediatrics provide sleep recommendations for children of all ages: 11–14 hours for preschoolers, 9–11 hours for school-aged children, and 8–10 hours for teenagers.[5] Empirical evidence, however, suggests that children do not sleep adequately,[6,7] thereby increasing risk for obesity.[7,8] Mechanisms are unclear, but research suggests a connection between short sleep duration and increased hunger through dysregulated hormonal mechanisms that involve decreased leptin and increased ghrelin levels.[9,10] Studies have attempted to understand the mechanisms between sleep and obesity, but have been limited to clinical studies among adults in controlled environments. This research suggests that sleep disruption increases a homeostatic drive to eat[11] and that sleep restriction results in subsequent increased caloric intake from energy-dense foods and larger portion sizes.[11–14] Additionally, studies of adults have also shown that insufficient sleep (less than 7 h) and oversleeping (more than 9–10 h) may increase risk for obesity, type 2 diabetes, heart disease, stroke, and depression.[15–17]

Research on children has found a relationship between sleep patterns and dietary patterns as early as age 2.[18] Sleep difficulties in infancy may influence irregular eating patterns at ages 2–4 years.[18] A large study of mostly non-Hispanic white and Hispanic school-age children (n = 1870), using self-reported measures of diet and sleep, found that children who slept less than 10 hours drank more soda and ate vegetables less frequently than children who slept at least 10 hours.[19] Self-reported dietary patterns and sleep duration were examined in a sample of Canadian boys and girls ($n \approx 1000$); authors reported that short sleep duration was associated with decreased fruit and vegetable intake in both boys and girls, increased meat consumption among boys, and decreased dairy intake and increased soft drink intake among girls.[20] For boys, they also found a relationship between short sleep duration and problematic eating behavior (ie, eating at irregular hours and eating too much/too fast) at age 6, which in

turn was associated with overweight/obesity at 6–7 years old. In a study of 9 to 11-year-old Finnish children, shorter sleep duration was associated with the consumption of energy-rich foods such as hot dogs, pizza, sweets, and sweetened breakfast cereal.[21]

To better understand how sleep impacts diet, studies have experimentally manipulated sleep duration among children and adolescents. Using a crossover design, Hart et al. increased sleep duration in one group of 8 to 11-year-old children and restricted sleep duration in another for 1 week and then children crossed over to the opposite sleep condition. They found that increased sleep compared to restricted sleep, a targeted difference of 3 hours, resulted in a reduced energy intake of 134 calories/day.[22] Beebe et al. used a similar crossover design in adolescents and found that those who were sleep restricted consumed more foods with a high glycemic index (ie, sweets and desserts).[23]

Although these studies have increased knowledge about the relationship between sleep and dietary intake, they used self-reported measures of sleep and/or did not use 24-hour dietary recall,[18,20,23] or were not conducted in free-living conditions.[22,23] An understanding of how sleep naturally impacts diet using more rigorous methods is still lacking, particularly among children most at risk for obesity. Studies should also account for socioeconomic context, given that shorter sleep duration has been observed among low-income adults and children,[24,25] including Latino children.[26] Latino children are at dual risk for both short sleep duration and unhealthful dietary intake,[27] and studies of sleep and diet are needed to help inform strategies to reduce obesity in this population.

This study aimed to examine the relationship between sleep duration and dietary intake, accounting for maternal education, among free-living MA 9–11 year olds in the San Francisco Bay Area, California, United States. We expected that longer sleep duration would be related to lower total energy and sugar intake, and lower percentage of energy intake from carbohydrates (%CARB). We explored the relationship between sleep duration and percentage of energy intake from fat (%FAT) and protein (%PROT). Finally, we tested for the possibility of a sleep duration by gender interaction for energy and sugar intake, and %CARB, %FAT, and %PROT.

METHODS

Participants and Data Collection

We recruited 322 families who were members of Kaiser Permanente Northern California, an integrated health care delivery organization, to participate in a 2-year study. Parents were sent letters and parent–child pairs were invited to participate if (1) the mother was of Mexican origin (born in the United States/Mexico), (2) the child was 9–11 years of age, and (3) the child had no major illnesses. Bilingual interviewers obtained parental informed consent and child assent to participate in the research. They interviewed parent–child pairs in their homes in the participants' preferred language. For this study, we used data at 1-year follow-up when children were all in the same age group for dietary recommended allowances.[28] At 1-year follow-up, a total of 251 families participated in sleep and dietary assessments. Mother–child pairs who had missing data on children's dietary variables were excluded from the study (n = 4), resulting in a final sample of 247 mother–child pairs, thus less than 1% of the sample was excluded due to incomplete data. The study was approved by the University of California San Francisco and Kaiser Permanente Northern California Research Foundation Institutional Review Boards.

Measures

Sleep Duration and Sedentary Time

Accelerometer-estimated sleep duration and sedentary behaviors were assessed consecutively for three consecutive 24-hour periods, including two weekdays and one weekend day (Wednesday through Saturday), using the hip-worn Actical accelerometer (Philips Respironics, Bend, OR). When positioned on the hip, the device is most sensitive to vertical movements of the torso. Actical is sensitive to movements in the 0.5–3 Hz range, allowing for detection of sedentary movements and high-energy movements. Actical's frequency range minimizes the effect of undesirable noise impulses, which tend to skew results. Sleep duration estimated by hip-worn accelerometry has been found to be highly correlated (r = 0.93) with sleep duration measured with a wrist-worn accelerometer in children aged 10–11 years.[29] Three consecutive 24-hour periods were chosen to reduce participant burden and maximize study participation.

The monitor was attached to an elastic belt with an adjustable buckle and positioned on the child above the iliac crest of the right hip. This method has been used to extract sleep duration from hip-worn accelerometry and compared to wrist-worn actigraphy,[29,30] but not other characteristics that are determined by polysomnography. Research assistants provided verbal and written instructions for care and placement of the monitor and belt at the time of the home visit. The child was instructed to wear the monitor at all times for three consecutive days, except during bathing. Accelerometers were collected after the third day.

Sleep Duration. In this study, accelerometers were programmed to collect data at 1-minute intervals at a specified start time.[31] Data were downloaded into a computer, and data output included the time stamp and total accelerometer counts. In the initial examination, data completeness was verified against the participant's log. Times and reasons for monitor removal were coded in the file. To minimize inter-interpreter variation, a single trained research assistant conducted visual inspections and cross-checked bedtimes and wake times with child-reported sleep diaries, using a plot of activity counts per minute for each 24-hour period. A plot of activity counts per minute for each 24-hour period was used to identify the time of sleep onset and termination. During the nighttime sleep period, the activity counts are usually zero. Sleep periods were cross-checked with the participants' wear log for "off" times. Any minutes scored as awake were removed from the sleep duration. Sleep duration was determined as the mean of the three 24-hour accelerometer measurements.

Sedentary Time. Child participants (assisted by parent) were asked to note any daytime naps in an activity log/diary. During the day, if accelerometer data showed 20 minutes of zeros that

were not accounted for by the activity log/diary, the monitor was considered "removed." Valid data included 1000 minutes or more out of 1440 min/day in a 24-hour period. Activity counts were summed for each 24-hour period and awake time was categorized into sedentary time, and light, moderate, and vigorous levels of physical activity according to the thresholds. The amount of sedentary time was computed using a cut-off equivalent to activity energy expenditure (AEE) < 0.01 kcal·kg^{-1}·min^{-1} or physical activity ratio (PAR) < 1.5 and encompassing physical activities of minimal body movements in the sitting or reclined position.[32] In this analysis, sedentary time was expressed as a percent of awake time (%SED).

Dietary Intake

At two home assessments within the same week (one weekend day and one weekday), interviewers conducted an individual 24-hour dietary recall with the child in his/her preferred language (Spanish/English). Trained interviewers collected dietary data using laptop computers equipped with the Nutrition Database System for Research (NDS-R), a dietary analysis program designed for the collection and analyses of 24-hour dietary recalls, food records, menus, and recipes.[33] Interviewers conducted the dietary recalls according to the prompts offered by the NDS-R program.[34]

NDS-R calculations were obtained for energy and total sugar intakes, carbohydrates, fat, and protein for each recall. Averages were computed for the two 24-hour dietary recall assessments. We used total energy intake (kcal) and total sugar intake (g) and calculated the %CARB, %FAT, and %PROT. We also calculated the percentage of calories from polyunsaturated fatty acids (%PUFA), monounsaturated fatty acids (%MUFA), and saturated fatty acids (%SFA).

Covariates

Covariates were based on previous research and factors known to influence diet. We controlled for child age, gender, height and weight, body mass index (BMI) *z*-score, and percent of awake time spent being sedentary (%SED). For child BMI *z*-score, height and weight were obtained using standard procedures in duplicate while the participants were wearing light indoor clothing and no shoes.[35,36] Children's BMI was calculated (weight[kg]/height[m]^2) and then converted to *z*-scores using National Child Health Statistics growth reference.[37] Weight status was computed using Center for Disease Control and Prevention (CDC) percentiles and cutoffs at the 85th percentile for overweight status and 95th percentiles for obese status.[38]

We controlled for maternal factors including self-reported maternal education in years and BMI. Mothers' BMI was computed using measured height and weight obtained by a trained research assistant.

Finally, we also tested for household income, mother's country of birth, number of children in the household, and child height and weight, but these factors were not significant in preliminary models and were removed for parsimony ($p > .20$).

Statistical Analyses

All statistics were computed using IBM SPSS 22 Statistics for Windows (Armonk, NY: IBM Corp). Descriptive statistics were computed using means and frequencies. We conducted multivariable linear regressions. We examined sleep duration with total caloric intake (kcal), total sugar intake (g), and percentages of energy intake from carbohydrates, fat, and protein. In post hoc analyses, we examined sleep duration with %PUFA, %MUFA, and %SFA. All models were adjusted for child age, gender and BMI *z*-score, %SED, maternal education, and maternal BMI.

RESULTS

Participant Sample

Participant characteristics are presented in Table 1. Children were 47% male; mean age of 10.3 (*SD* = 0.9) years; average sleep duration 9.6 (*SD* = 0.8) hours. Children had a BMI *z*-score of 0.92 (*SD* = 1.1), with 53% being overweight or obese. On average, children consumed 1759 calories, with caloric intake comprised of 53% carbohydrates, 31% fat, and 15% protein. Total sugar intake was 107 g (equivalent to 414 calories).

Multivariable Linear Regressions Models

Regression results are included in Table 2. We examined sleep duration with total energy intake, total sugar intake, and the %CARB, %FAT, %PROT. Longer sleep duration was marginally associated with lower sugar consumption ($\beta = -0.13$, $p = .05$), significantly associated with a lower %CARB ($\beta = -0.22$, $p < .01$), and significantly associated with a higher %FAT ($\beta = 0.19$, $p < .01$), after controlling for covariates. Compared

Table 1—Sample Characteristics of 9 to 11-Year-Old Mexican American Children in the San Francisco Bay Area (*n* = 247).

Child characteristics	Mean (*SD*) or %
Sleep duration (h)	9.6 (0.8)
Total energy intake (kcal)	1759 (514)
% Energy from carbohydrate	53 (6)
% Energy from fat	31 (5)
Polyunsaturated fatty acids	7 (2)
Monounsaturated fatty acids	11 (2)
Saturated fatty acids	11 (3)
% Energy from protein	15 (3)
Total sugar intake (g)	107 (39)
Age (y)	10.3 (0.9)
% Female	52.6
BMI *z*-score	0.92 (1.1)
% Overweight or obese	52.8
% Awake time spent being sedentary	60 (0.1)
Maternal characteristics	
Education (y)	10.8 (3.7)
BMI	30.7 (7.1)
Obese	49.6

BMI = body mass index.

Table 2—Multivariable Linear Regressions With Sleep Duration and Consumption of Macronutrients in 9 to 11-Year-Old Mexican American Children, Showing Standardized β Coefficients (n = 247).

			% Calories from		
	Total calories	Total sugar	Carbohydrate	Fat	Protein
	β (*b*)	β (*b*)	β (*b*)	β (*b*)	β (*b*)
Independent variable					
Sleep duration (h)	0.01 (3.37)	−0.13 (−6.38)†	−0.22 (−1.77)**	0.19 (1.32)*	0.10 (0.43)
Covariates					
Age (y)	−0.03 (−14.62)	−0.09 (−3.97)	−0.12 (−0.85)	0.02 (0.09)	0.20 (0.78)**
Female	0.27 (−269.77)***	−0.20 (−15.31)**	0.04 (0.52)	−0.08 (−0.81)	0.03 (0.23)
Child BMI z-score	0.07 (33.64)	0.05 (1.57)	−0.06 (−0.03)	−0.03 (0.16)	0.04 (0.12)
% Awake time spent being sedentary	−0.01 (−78.90)	0.05 (19.32)	0.05 (3.57)	−0.05 (−2.73)	−0.02 (−0.73)
Maternal education (y)	0.16 (21.85)*	0.13 (1.32)*	−0.06 (−0.10)	0.15 (0.22)*	−0.13 (−0.13)*
Maternal BMI	−0.06 (−3.99)	−0.08 (−0.45)	−0.11 (−0.10)	0.08 (0.06)	0.07 (0.03)

†p= .05; *p < .05; **p <.01; ***p< .001.
β = standardized linear regression coefficient; b = unstandardized linear regression coefficient; BMI = body mass index.

to boys, girls consumed less energy (β = −0.27, p < .001) and less sugar (β = −0.20, p < .01). Older children obtained a higher %PROT (β = 0.20, p < .01). More maternal education was associated with increased energy intake (β = 0.16, p < .05), increased %FAT (β = 0.15, p < .05), and deceased %PROT (β = −0.13, p < .05).

In post hoc analyses, we examined the association between sleep duration and the %PUFA, %MUFA, and %SFA. Longer sleep duration was related to a higher %PUFA (β = 0.17, p < .05), controlling for covariates. More maternal education was associated with increased %SFA (β = 0.23, p < .05) (Tables 3 and 4).

We examined whether child gender moderated the relationship between sleep duration and macronutrient intake, but no interactions were significant.

DISCUSSION

Studies of adults suggest that insufficient sleep affects diet quality, but pediatric studies in this area are scant. The aim of the current study was to examine the relationship between sleep duration and macronutrient intake in a cohort of MA children, who are at high risk for obesity and future chronic disease. Using 24-hour food recalls and accelerometer-estimated sleep duration, we found that longer sleep duration was related to a lower percentage of energy intake from carbohydrates and a higher percentage of energy from fat, driven by polyunsaturated fat.

In this study of free-living 9 to 11-year-old MA children, we found that carbohydrate intake decreased with longer sleep duration. Similarly, a study of Finnish children found that shorter sleep duration was associated with the intake of energy-rich foods, and among girls, longer sleep was related to increased nutrient-dense foods (diet based on food frequency patterns).[21] This is also consistent with previous studies using protocols to manipulate sleep among children and adolescents. Hart et al.[22] found that when children were in an increased sleep condition, they consumed 134 fewer calories/day than when they were in a decreased sleep condition. Beebe et al.[23] found that sleep-deprived adolescents increased their consumption of foods with a high glycemic index, consistent with our study. These findings suggest that insufficient sleep may increase cravings for foods that are high in carbohydrate content, thereby increasing obesity risk. More research is needed to investigate what causes cravings for energy-dense foods and the drive to eat among children with decreased sleep and whether there is a tipping point at which too little sleep affects dietary intake among children.

We found that children who slept longer ate a higher percentage of energy from fat, which is inconsistent with two other studies of young children and older adolescents showing that short sleep duration was related to increased fat intake.[39,40] Inconsistent findings may have been a result of one study examining adolescents, or the other study using self-reported measures to assess sleep in young children. In a further examination of the relationship between sleep duration and fat intake, we found that longer sleep duration was associated with increased %PUFA. This finding is similar to a recent meta-analysis of nine cohort studies that found that longer sleep duration was related to higher total fat intake, primarily driven by higher PUFA intake, in older women of European descent.[41] To our knowledge, this is the first study to observe this association in children of Mexican descent. It is possible that increased carbohydrate intake may replace the intake or cravings for foods high in PUFA. It is also possible that longer sleep duration promotes fat intake to replenish fat that is oxidized during nonfragmented sleep.[42] Future studies could help to illuminate these relationships, such as examining the temporal relationships between dietary intake and sleep duration.

This study has several limitations. We studied children from middle-late childhood and therefore our findings should not

Table 3—Multivariable Linear Regressions With Sleep Duration and % Calories From Fat Intake in a 9 to 11-Year-Old Mexican American Children (*n* = 247).

	% Calories from		
	PUFA	MUFA	SFA
	β (*b*)	β (*b*)	β (*b*)
Independent variable			
Sleep duration (h)	0.17 (0.47)*	0.12 (0.39)	0.12 (0.39)
Covariates			
Age (y)	−0.05 (−0.12)	0.03 (0.10)	0.05 (0.15)
Female	0.00 (−0.01)	−0.12 (−0.60)	−0.02 (−0.12)
Child BMI z-score	0.06 (0.13)	0.08 (0.17)	−0.08 (−0.20)
% Awake time spent being sedentary	−0.03 (−0.83)	−0.05 (−1.27)	−0.05 (−1.54)
Maternal education (y)	0.01 (0.01)	0.08 (0.05)	0.23 (0.16)*
Maternal BMI	0.14 (0.05)*	0.10 (0.03)	−0.04 (−0.02)

*$p < .05$.
PUFA = polyunsaturated fatty acid; MUFA = monounsaturated fatty acid; SFA = saturated fatty acid; BMI = body mass index; β = standardized linear regression coefficient; *b* = unstandardized linear regression coefficient.

Table 4—Multivariable Linear Regressions With Sleep Quartiles and % Calories From Carbohydrate, Fat, and PUFA Intake in a 9 to 11-Year-Old Mexican American Children, Showing Standardized β Coefficients (*n* = 247).

Independent variable	% Calories from		
	CARB	FAT	PUFA
Sleep quartiles			
1st (<9 h)	0.17*	−0.12	−0.10
2nd (9.1–9.5 h)	0.23**	−0.20*	−0.23**
3rd (9.6–10 h)	0.07	−0.003	−0.06
4th (>10 h)	Reference	Reference	Reference
Covariates			
Age (y)	−0.12	0.02	−0.07
Female	0.05	−0.10	−0.03
Child BMI z-score	0.06	0.02	0.05
% Awake time spent being sedentary	0.08	−0.04	−0.01
Maternal education	−0.04	0.14	−0.02
Maternal BMI	−0.13	0.09	0.15*

*$p < .05$; **$p < .01$.
BMI = body mass index; CARB = carbohydrate; PUFA = polyunsaturated fatty acid.

be generalized beyond this age range. The cross-sectional design of this study did not allow us to determine causal relationships; sequential assessment of sleep and dietary intake, such as subsequent dietary intake after prior night's sleep, would be necessary to draw causal inferences. Also, children in this study were from families with health insurance covered through Kaiser Permanente which may not be representative of all MA children. The study, however, comprised families across a broad range of occupations: 44% of fathers were unskilled workers, 20% were semi-skilled and 16% were skilled manual workers, and 20% clerical to professional workers. We estimated sleep using accelerometers used on the hip as opposed to on the wrist, which is typically used for sleep measurement; however, several studies have used other hip-worn accelerometers to assess sleep in children,[29,30] of which one reported that results were highly correlated with those obtained using wrist-worn accelerometers. To measure sleep, polysomnography (a clinical test that records brain waves, oxygen level in the blood, heart and breathing rates, and movements) is the gold standard for sleep studies, but it is expensive and subject burden is high; therefore, it is less feasible in research with children. Hip-worn accelerometry was used for the purpose of estimating sleep duration and not other sleep characteristics that should be determined by polysomnography. Finally, we considered nighttime sleep duration, but not daytime napping. During the day, data consisting of 20 minutes of consecutive zeros was interpreted as removal of the accelerometer. Children were asked only to remove the accelerometer when they bathed. Yet, because the activity logs/diaries may have been incompletely recorded, there is the possibility that naps went undetected and unreported. Daytime napping, however, is uncommon among 9 to 11-year-old children.[43] It is also possible that dietary intake, such as increased carbohydrate intake before bedtime, impacts sleep duration; however, the parent study was not designed to answer this research question.

The strengths of our study include the relatively large free-living sample size of MA children. We estimated sleep duration for three consecutive nights (two weeknights and one weekend night) using hip-worn accelerometry. We used two 24-hour food recalls to assess macronutrient intake. Another strength was the focus on MA children, a group with some of the highest obesity rates, but about whom little is known regarding sleep as a risk factor for obesity. Although our findings may not be generalizable to other Latino subgroups or other ethnic groups, our results provide support for the relationship between insufficient sleep duration and macronutrient intake among MA children. Future studies could examine these relationships in younger children and other ethnic groups. Longitudinal research is needed to examine the temporal effects of measured sleep duration and dietary intake.

CONCLUSION

Longitudinal research is needed to investigate how inadequate sleep duration impacts diet and appetite during childhood and the long-term impact on obesity risk. This study provides cross-sectional evidence of the link between sleep duration and dietary intake among MA children and underscores the protective role that sleep may play in obesity prevention. These findings have important implications for children's health as inadequate sleep may contribute to overconsumption and cravings for high carbohydrate foods, which may impact the

intake of healthy fat. Highlighting the sleep recommendations supported by the National Sleep Foundation and the American Academy of Pediatrics is an important step toward increasing awareness and national policies to support parent education about sleep health.

REFERENCES

1. US Bureau of the Census. Statistical Abstract of the United States: 2012. Population 2012. https://www.census.gov/prod/2011pubs/12statab/pop.pdf. Accessed April 17, 2014.
2. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014; 311(8): 806–814.
3. Rendall MS, Weden MM, Fernandes M, Vaynman I. Hispanic and black US children's paths to high adolescent obesity prevalence. Pediatr Obes. 2012; 7(6): 423–435.
4. Martinez S, Tschann J, Greenspan L, et al. Is it time for bed? Short sleep duration increases risk for obesity in Mexican American children. Sleep Med. 2014; 15(12): 1484–1489.
5. National Sleep Foundation. National Sleep Foundation Recommends New Sleep Durations. 2015. https://sleepfoundation.org/media-center/press-release/national-sleep-foundation-recommends-new-sleep-times. Accessed September 28, 2012.
6. Martinez S, Greenspan L, Tschann J, et al. Mother-reported sleep, accelerometer-estimated sleep and weight status in Mexican American children: sleep duration is associated with increased adiposity and risk for overweight/obese status. J Sleep Res. 2014; 23(3): 326–334
7. Cappuccio FP, Taggart FM, Kandala NB, et al. Meta-analysis of short sleep duration and obesity in children and adults. Sleep. 2008; 31(5): 619–626.
8. Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity: a systematic review and meta-analysis. Obesity. 2008; 16(2): 265–274.
9. Spiegel K, Tasali E, Penev P, Cauter EV. 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(11): 846–850.
10. Taheri S. The link between short sleep duration and obesity: we should recommend more sleep to prevent obesity. Arch Dis Childhood. 2006; 91(11): 881–884.
11. Hogenkamp PS, Nilsson E, Nilsson VC, et al. Acute sleep deprivation increases portion size and affects food choice in young men. Psychoneuroendocrinology. 2013; 38(9): 1668–1674.
12. Nedeltcheva AV, Kilkus JM, Imperial J, Kasza K, Schoeller DA, Penev PD. Sleep curtailment is accompanied by increased intake of calories from snacks. Am J Clin Nutr. 2009; 89(1): 126–133.
13. Brondel L, Romer MA, Nougues PM, Touyarou P, Davenne D. Acute partial sleep deprivation increases food intake in healthy men. Am J Clin Nutr. 2010; 91(6): 1550–1559.
14. 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(5): 266–273.
15. Liu Y, Wheaton AG, Chapman DP, Croft JB. Sleep duration and chronic diseases among U.S. adults age 45 years and older: evidence from the 2010 behavioral risk factor surveillance system. Sleep. 2013; 36(10): 1421–1427.
16. Watson N, Harden K, Buchwald D, et al. Sleep duration and depressive symptoms: a gene–environment interaction. Sleep. 2014; 37(2): 351–358.
17. Shan Z, Ma H, Xie M, et al. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care. 2015; 38(3): 529–537.
18. McDermott BM, Mamun AA, Najman JM, Williams GM, O'Callaghan MJ, Bor W. Preschool children perceived by mothers as irregular eaters: physical and psychosocial predictors from a birth cohort study. JDBP. 2008; 29(3): 197–205.
19. Franckle RL, Falbe J, Gortmaker S, et al. Insufficient sleep among elementary and middle school students is linked with elevated soda consumption and other unhealthy dietary behaviors. Prev Med. 2015; 74: 36–41.
20. Tatone-Tokuda F, Dubois L, Ramsay T, et al. Sex differences in the association between sleep duration, diet and body mass index: a birth cohort study. J Sleep Res. 2012; 21(4): 448–460.
21. Westerlund L, Ray C, Roos E. Associations between sleeping habits and food consumption patterns among 10–11-year-old children in Finland. Br J Nutr. 2009; 102(10): 1531–1537.
22. Hart CN, Carskadon MA, Considine RV, et al. Changes in children's sleep duration on food intake, weight, and leptin. Pediatrics. 2013; 132(6):E1473–E1480.
23. Beebe DW, Simon S, Summer S, Hemmer S, Strotman D, Dolan LM. Dietary intake following experimentally restricted sleep in adolescents. Sleep. 2013; 36(6): 827–834.
24. Mezick EJ, Matthews KA, Hall M, et al. Influence of race and socioeconomic status on sleep: Pittsburgh SleepSCORE project. Psychosom Med. 2008; 70(4): 410–416.
25. Crosby B, LeBourgeois MK, Harsh J. Racial differences in reported napping and nocturnal sleep in 2- to 8-year-old children. Pediatrics. 2005; 115(1 Suppl): 225–232.
26. Martinez S, Jostad-Laswell A. Latino parents' views on optimal sleep, bedtime routines and sleep hygiene among urban preschool-age children: what do parents have to say? Acad Pediatr. 2015; 15(6): 636–643.
27. Wilson T, Adolph A, Butte N. Nutrient adequacy and diet quality in non-overweight and overweight Hispanic children of low socioeconomic status: the Viva la Familia Study. J Am Diet Assoc. 2009; 109(6): 1012–1021.
28. Institute of Medicine. Dietary Reference Intakes Tables and Application. http://iom.nationalacademies.org/Activities/Nutrition/SummaryDRIs/DRI-Tables.aspx. Accessed August 12, 2015.
29. Kinder JR, Lee KA, Thompson H, Hicks K, Topp K, Madsen KA. Validation of a hip-worn accelerometer in measuring sleep time in children. J Pediatr Nurs. 2012; 27(2): 127–133.
30. Nixon GM, Thompson JMD, Han DY, et al. Short sleep duration in middle childhood: risk factors and consequences. Sleep. 2008; 31(1): 71–78.
31. Butte NF, Gregorich SE, Tschann JM, et al. Longitudinal effects of parental, child and neighborhood factors on moderate-vigorous physical activity and sedentary time in Latino children. Int J Behav Nutr Phys Act. 2014; 11: 108.
32. Puyau MR, Adolph AL, Vohra FA, Zakeri I, Butte NF. Prediction of activity energy expenditure using accelerometers in children. Med Sci Sports Exerc. 2004; 36(9): 1625–1631.
33. Feskanich D, Sielaff B, Chong K, G B. Computerized collection and analysis of dietary intake information. Comput Methods Programs Biomed. 1989; 30(1): 47–57.
34. Lytle LA, Nichaman MZ, Obarzanek E, et al. Validation of 24-hour recalls assisted by food records in third-grade children. The CATCH collaborative group. J Am Diet Assoc. 1993; 93(12): 1431–1436.
35. Stallings VA, Fung EB. Clinical nutritional assessment of infants and children. In: Shils ME, Olson JA, Shike M, Ross AC, eds. Modern Nutrition in Health and Disease. 9th ed. Philadelphia: Lippincott, Williams & Wilkins; 1999: 885–893.
36. Lohman TG, Roche AF, Martorell R. Anthropometric Standardization Reference Manual. Champaign, IL: Human Kinetics Books; 1989.
37. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. Adv Data. 2000; 314: 1–27.
38. Centers for Disease Control and Prevention (CDC). Clinical Growth Charts. http://www.cdc.gov/growthcharts/cdc_charts.htm. Accessed January 15, 2013.
39. Fisher A, McDonald L, van Jaarsveld CHM, et al. Sleep and energy intake in early childhood. Int J Obes. 2014; 38(7): 926–929.
40. Weiss A, Xu F, Storfer-Isser A, Thomas A, Ievers-Landis CE, Redline S. The association of sleep duration with adolescents' fat and carbohydrate consumption. Sleep. 2010; 33(9): 1201–1209.
41. Dashti HS, Follis JL, Smith CE, et al. Habitual sleep duration is associated with BMI and macronutrient intake and may be modified by CLOCK genetic variants. Am J Clin Nutr. 2015; 101(1): 135–143.
42. Hursel R, Rutters F, Gonnissen HK, Martens EA, Westerterp-Plantenga MS. Effects of sleep fragmentation in healthy men on energy

expenditure, substrate oxidation, physical activity, and exhaustion measured over 48 h in a respiratory chamber. Am J Clin Nutr. 2011; 94(3): 804–808.
43. Thorleifsdottir B, Björnsson JK, Benediktsdottir B, Gislason T, Kristbjarnarson H. Sleep and sleep habits from childhood to young adulthood over a 10-year period. J Psychosom Res. 2002; 53(1): 529–537.

FUNDING

This study was funded by National Heart, Lung, and Blood Institute, 1 R01 HL084404 (PI: Tschann).

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication May, 2016
Submitted in final revised form November, 2016
Accepted for publication November, 2016
Address correspondence to: Suzanna M. Martinez, PhD, Nutrition Policy Institute, University of California, 1111 Franklin St, 10th Floor, Oakland, CA 94607, USA. Telephone: +510-587-6264; Fax: +510-643-8197; E-mail: suzanna.martinez@ucop.edu

DISCLOSURE STATEMENT

None declared.