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Role of Sleep Timing in Caloric Intake and BMI

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Sleep duration has been linked to obesity and there is also an emerging literature in animals demonstrating a relationship between the timing of feeding and weight regulation. However, there is a paucity of research evaluating timing of sleep and feeding on weight regulation in humans. The goal of this study was to evaluate the role of sleep timing in dietary patterns and BMI. Participants included 52 (25 females) volunteers who completed 7 days of wrist actigraphy and food logs. Fifty-six percent were "normal sleepers" (midpoint of <5:30 AM) and 44% were "late sleepers" (midpoint of sleep ≥5:30 AM). Late sleepers had shorter sleep duration, later sleep onset and sleep offset and meal times. Late sleepers consumed more calories at dinner and after 8:00 PM, had higher fast food, full-calorie soda and lower fruit and vegetable consumption. Higher BMI was associated with shorter sleep duration, later sleep timing, caloric consumption after 8:00 PM, and fast food meals. In multivariate models, sleep timing was independently associated with calories consumed after 8:00 PM and fruit and vegetable consumption but did not predict BMI after controlling for sleep duration. Calories consumed after 8:00 PM predicted BMI after controlling for sleep timing and duration. These findings indicate that caloric intake after 8:00 PM may increase the risk of obesity, independent of sleep timing and duration. Future studies should investigate the biological and social mechanisms linking timing of sleep and feeding in order to develop novel time-based interventions for weight management.

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Recent research has begun to examine the impact of sleep timing on cardiometabolic health (1). Sleep timing is a behavior that exhibits significant variation within the population and is determined by both biological and social factors. Biologically, the timing and duration of sleep is controlled by an interaction between both the circadian clock and homeostatic sleep pressure (2). Results from over 55,000 online surveys demonstrate that the most commonly reported preferred sleep timing when free from occupational and social obligations is approximately midnight to 8:00 AM. In general, sleep timing appears to have a normal distribution within the population, although a significant percentage (8.3%) report a preferred sleep onset time of 3:00 AM. or later. Sleep timing is also highly age dependent; sleep times are earlier in childhood, delay across adolescence, then advance again in older adulthood (3).

The majority of research on sleep and obesity has focused on sleep duration and quality. Cross sectional (4,5) and prospective studies (6–8) have found associations between short sleep duration and increased risk for obesity, greater percentage body fat, and weight gain over time. Several recent studies using dietary assessments have reported associations between short sleep duration and increased energy intake, particularly from fats (9–11). However, dietary intake alone does not fully explain the effects of sleep duration on weight gain. In addition to duration, poor sleep quality, such as disruption of slow wave sleep or sleep fragmentation due to obstructive sleep apnea, has also been associated with metabolic dysregulation (12,13).

In addition to the duration of sleep, the timing of the sleep–wake cycle, particularly when it is at odds with work and social demands, may also play a role in health, including weight regulation and metabolism. Alterations in the timing of sleep may lead to shorter sleep duration and/or circadian misalignment between sleep and dietary intake. Several studies have reported shorter sleep duration in individuals with late sleep timing, particularly on weekdays, and then greater "catch up" sleep on weekends (14,15). In addition, altered sleep timing may lead to circadian disruption, which can have independent effects on biology and behavior (1). For example, misaligned sleep timing with the timing of the endogenous circadian rhythm in the laboratory has been shown to lead to alterations in leptin and glucose (16).

Furthermore, being awake late at night may be related to social and environmental factors that place individuals at greater risk for obesity via an increase in the opportunity to consume calories and poor availability of healthful foods at night (17,18). Few studies have evaluated the role of sleep timing in dietary behavior or indicators of health risk such as BMI. Later sleep timing, and a self-reported "preference" for later sleep and activity have been associated with poorer health behaviors in other domains, such as increased smoking, caffeine, and alcohol use

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(14,19,20). A recent study conducted in adolescents found that later sleep timing on days off was associated with greater fast food consumption and fewer servings of dairy (21). No studies have specifically assessed associations between sleep timing and BMI, however, a case control study reported higher BMI in those with delayed sleep phase disorder (who generally have later sleep times) compared with controls (22). In addition, individuals with Night Eating Syndrome, which is characterized by evening hyperphasia and/or nocturnal awakenings to eat, report a preference for later sleep timing, demonstrate phase delays in caloric consumption and are more likely to be obese (23–26).

The primary goal of this study was to test the hypothesis that the timing of sleep is associated with poor dietary patterns and elevated BMI above and beyond sleep duration. The use of wrist actigraphy and a 7-day sleep and food log where participants recorded the timing and content of all meals and snacks allowed us to compare the timing of sleep, meals, and caloric intake across the day and night. We predicted that those with later sleep timing would have later meal times, poorer dietary habits (i.e., greater caloric intake, more frequent fast food consumption, lower fruit and vegetable consumption, greater soda and caffeine consumption), and higher self-reported BMI.

METHODS AND PROCEDURES

Participants

Participants were recruited from the community using flyers and online advertisements as part of a larger study of circadian rhythms and sleep–wake timing. This study was approved by the institutional review board of Northwestern University and all participants gave written informed consent prior to enrollment. This analysis included only individuals who reported an evening or intermediate (neither evening nor morning) diurnal preference. Although being a shift worker was not an exclusion criterion from this study, none of the participants reported shift work schedules. Those with a morning type diurnal preference were excluded from analyses due to low numbers. Additional exclusionary criteria included elevated depressive symptoms, as indicated by a score >16 on the Center for Epidemiologic Studies-Depression Scale (27).

Procedure

Participants underwent preliminary telephone or internet screening to determine eligibility and willingness to participate in the study. After informed consent was obtained, eligible participants were provided with 7 days of food logs, sleep logs, and a wrist actigraph which was worn for at least 7 days. Participants were asked to list every food or drink they consumed across each 24-h period, and give a time, type, location, description, and quantity corresponding to each meal or snack. Participants also completed a daily sleep log and wore an actigraph (AW-L Actiwatch; Mini Mitter Co., Bend, OR) to determine sleep and wake timing, sleep duration, and sleep quality. Sleep onset and offset times were subsequently retrieved from actigraphy recordings using Actiware-Sleep 3.4 software (Mini Mitter Co.).

Screening questionnaires

Diurnal preference was measured for screening purposes by the Horne Ostberg Morningness Eveningness Questionnaire. Scores on this 19-item scale range from 16 to 86, with higher scores indicating greater preference for morning. Questions assess individual preferences for sleep/wake times including "Considering your own "feeling best" rhythm, at what time would you get up if you were entirely free to plan your day?". Adequate internal consistency has been reported for this measure ($\alpha = 0.86$) (28). Horne Ostberg scores have been demonstrated to be correlated with measures of circadian timing, such as body temperature (29,30).

Depressive symptoms were measured using the Center for Epidemiologic Studies-Depression Scale (27). Adequate internal consistency has been reported for this widely used 10-item questionnaire (27). Participants with Center for Epidemiologic Studies-Depression Scale scores >16 were excluded from this sample in order to reduce confounders. Scores >16 have been associated with clinically significant levels of depression (31).

Dietary assessment

Dietary intake was assessed using a food log in which participants recorded all food and drinks consumed for a 7-day period. We asked participants to record the time the food or drink was consumed, meal (breakfast, lunch, dinner, or snack), type of food with brand name if possible, setting it was eaten in (i.e., home or restaurant), portion size, and whether it was a typical or atypical diet that day.

Meal times were recorded as the start of each period of consumption. However, two or more meal times listed within 30 min of one another were grouped as one meal, with the time that occurred first chronologically used in calculations. Meals were classified as breakfast, lunch, or dinner based upon the meal type each participant indicated on the diet log. When a participant assigned multiple meals to one of these three meal types, the meal that was consumed first was used in calculations. A meal listed as "brunch" was considered neither breakfast nor lunch in order to avoid ambiguity and redundancy. Logs were considered valid if there were at least 2 weekdays and 1 weekend days completed. Dietary logs were considered incomplete if total calories per day were <500. If participants had fewer than 7 days recorded, all of the available data was used; alternatively, if an excess of 7 days were completed, the investigators used the first 7 consecutive days that best coincided with actigraphy recordings. In the case of conflicting data, such as a breakfast time listed prior to a wake time, calorie information was utilized but meal time was omitted.

Caloric content of the meals was analyzed using publicly available nutrition information (http://www.sparkpeople.com) as well as restaurant and manufacturer websites. Caloric analysis yielded total calories, grams of fat, protein, and carbohydrates. Cumulative calories across the day and calories consumed in the evening were also calculated. Calories in the evening were defined as calories consumed after 8:00 PM since this has previously been reported as the average time of maximum caloric intake in normal eaters (32). In addition, we calculated frequencies of several types of food: servings of fruits and vegetables, full-calorie sodas, caffeinated drinks and fast food meals. Fast food was defined as anything that can be purchased from a drive-through. Frozen and convenience foods, as well as snacks such as potato chips, were not included as fast food. Full-calorie soda intake was defined as number of 12 oz. servings of full-calorie soda per week. Intake of caffeinated drinks was defined as number of servings of caffeinated drinks (e.g., cups of coffee or 12 oz. servings of diet or regular soda) per week. Although night eating syndrome was not formally assessed using research diagnostic criteria (23), features of this disorder were defined as ≥25% of caloric consumption after the evening meal and/or two or more episodes of eating after actigraphically documented sleep onset.

Sleep timing and duration

Sleep timing and duration were assessed using sleep logs and wrist actigraphy. The following variables were determined: sleep start (sleep onset), sleep end (actual wake time), and actual sleep time (total sleep time). Sleep start was defined as the first 10-min period in which no more than one epoch was scored as mobile. Sleep end was defined as the last 10-min period in which no more than one epoch was scored as immobile. Actual sleep time was defined as the amount of time between sleep start and sleep end that was scored as sleep. We calculated midpoint of sleep based on the average of the sleep onset and sleep offset for the 7-day period. In categorical analyses, participants were classified as having normal sleep times if midpoint of sleep was between 1:00 AM to 5:29 AM, and participants were classified as having late sleep times if

midpoint of sleep was 5:30 AM or later, which is past the 50th percentile of sleep times in the population (4:00 AM) (3) and comprises nearly half of our sample, allowing for power to conduct categorical analyses. Sleep logs and actigraphy were conducted during the same 7 days as the food logs in all but four participants. For these four participants, we used actigraphy data from within the same month as the food logs to calculate sleep times but we did not calculate relationships between sleep times and meal times because actigraphy was conducted at a different time. In addition, four participants were missing actigraphy but not food log data: two due to equipment failure, one participant was noncompliant with the actigraph recording and one participant did not have valid actigraphy data for a month before or after the food log. Despite not having actigraphy data, we were able to estimate sleep times for the categorical analyses using the sleep log data in three of these four participants because they clearly fell into a category based on self-reported sleep times. One participant could not be classified due to lack of both sleep logs and actigraphy data, and was therefore excluded in the sleep timing analyses.

Activity

In order to approximate physical activity and movement, wrist actigraphy was also used to approximate activity. The average daily 24-h activity counts for each participant were recorded based on 7 days of recording. This was available for all except four participants.

Statistical analysis

Data were analyzed using IBM SPSS v. 19 (Somers, NY). Analyses were conducted using *t*-tests for independent means, repeated measures ANOVA, correlations, partial correlations, and linear regression analyses. We conducted *post hoc* analyses for significant time by sleep timing interactions in repeated measures ANOVA were followed up by *t*-tests with a Bonferroni correction. For all other analyses, significance was determined as 0.05 on two-tailed tests.

RESULTS

Participants

Participant characteristics are listed in Table 1. The sample included 27 males and 25 females. The majority of the sample (71%) was white. Comparisons between sleep timing groups on demographic, mood, and sleep characteristics are listed in Table 2. Based on actigraphy and sleep logs, 28 participants were classified as having normal sleep times (midpoint of sleep earlier than 5:30 AM) and 23 participants were classified as having late sleep timing (midpoint of sleep later than 5:30 AM). Late sleepers had later sleep onset ($P < 0.001$) and sleep offset times ($P < 0.001$), later midpoint of sleep ($P < 0.001$) and shorter sleep duration ($P < 0.001$). Late sleepers had lower scores on the Horne Ostberg Questionnaire, indicating a preference for later sleep and activity timing (M = 45.3, s.d. = 7.7) in normal sleepers vs. late sleepers (M = 29.1, s.d. = 5.9), $P < 0.001$. Although none of the participants had significant levels of depression, depressive symptoms were higher in late sleepers (M = 6.1 s.d. = 4.1 in normal sleepers vs. M = 8.9, s.d. = 5.6 in late sleepers, $P < 0.05$).

Meal frequency and timing

There were no differences in number of meals consumed each day between normal and late sleepers (normal sleep times M = 4.6 s.d. = 1.4, late sleep times M = 4.2, s.d. = 1.5). The frequency of participants who skipped breakfast (defined as eating breakfast <3 days of 7) was similar in both groups (4% normal sleepers and 16% of late sleepers $P = 0.31$). Timing of meals and sleep is presented in Figure 1. Those with later sleep times had later breakfast ($P < 0.001$), lunch ($P < 0.001$), dinner ($P < 0.05$) and last meal times ($P < 0.001$) compared to those with normal sleep times. The duration between breakfast and lunch was shorter in late sleepers compared with normal sleepers ($P < 0.01$). The duration between the last meal or snack to sleep onset was longer in late sleepers ($P < 0.05$).

Night eating features

Average percentage of calories consumed after the participants' evening meal was similar in normal and late sleepers (7.6% vs. 9.9%, $P = 0.25$). Two participants exhibited characteristics of Night Eating Syndrome. One participant (a late sleeper) consumed 28% of daily calories after the evening meal and one participant (a normal sleeper) reported two episodes of caloric consumption after sleep onset.

Caloric intake, macronutrient, and food type intake

Table 3 displays caloric, macronutrient, and food type intake. Late sleepers consumed on average 248 more calories per day than normal sleepers but this was not statistically significant ($P = 0.10$). Late sleepers reported higher caloric intake at dinner

Table 1 Participant characteristics (*N* = 52)

	Mean (s.d.) or *N* (%)
Age	31 (12), range 18–71
Sex	
Males	27 (52%)
Females	25 (48%)
Race	
White	37 (71%)
Black	4 (8%)
Asian	6 (12%)
Hispanic	3 (6%)
Other	2 (4%)

Table 2 Participant characteristics according to sleep timing

	Normal sleep timing	Late sleep timing
	N = 28	*N* = 23
	M (s.d.)	M (s.d.)
Age	33.11 (14.80)	30.04 (9.49)
Sex	13 males, 15 females	13 males, 10 females
Sleep onset time*	12:36 AM (0:52)	3:45 AM (1:13)
Sleep offset time*	8:07 AM (1:12)	10:42 AM (1:29)
Midpoint of sleep*	4:08 AM (1:01)	7:15 AM (1:17)
Actual sleep*	6:38 h (0:48)	5:33 h (1:04)

Missing actigraphy data: $n = 2$ equipment malfunction, $n = 1$ participant noncompliance, $n = 1$ actigraphy was not conducted in the same time period as food diary, therefore it was not included.
*$P < 0.001$

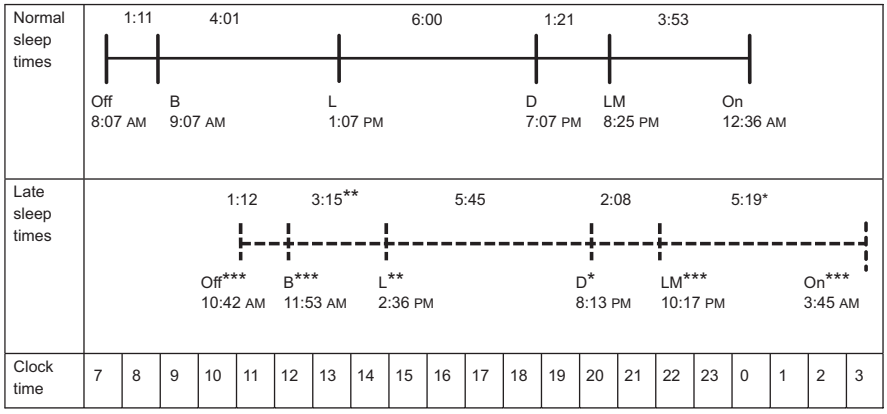

Figure 1 Sleep and meal timing according to sleep timing. B, breakfast; D, dinner; L, lunch; LM, last meal or snack of the day; Off, sleep offset via actigraphy; On, sleep onset via actigraphy. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

Table 3 Caloric intake and food type frequency according to sleep timing

	Normal sleep timing	Late sleep timing
	M (s.d.)	M (s.d.)
Total calories	1,905 (526)	2,153 (524)
% Protein	14 (2.7)	15 (2.0)
% Carbohydrate	49 (7.9)	49 (7.8)
% Fat	38 (7.2)	35 (7.7)
Breakfast	355 (133)	285 (143)
Lunch	528 (188)	503 (378)
Dinner*	630 (198)	825 (352)
Snacks	405 (284)	536 (323)
Calories after dinner	150 (151)	208 (166)
Calories after 8:00 PM***	376 (237)	754 (373)
Fast food meals per week*	3.0 (1.8)	5.2 (3.8)
Fruit and vegetable servings per day**	3.4 (1.8)	1.9 (1.1)
Sugar soda servings per week*	1.3 (2.5)	4.5 (6.5)
Caffeinated drinks per week†	7.3 (6.5)	13.0 (12.6)

One participant in the late sleep timing group did not label meals, and therefore was not included in the analyses for each meal but was included in total caloric and macronutrient analyses.
*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$, †$P < 0.1$.

($P < 0.05$), higher caloric intake after 8:00 PM ($P < 0.001$), more fast food ($P < 0.05$), more full-calorie sodas ($P < 0.05$) and fewer fruits and vegetables ($P < 0.01$) compared with normal sleepers. There was a trend for more caffeinated drinks in late sleepers ($P < 0.10$). There were no significant differences in caloric intake at breakfast, lunch, or snacks or percentage of macronutrients.

Figure 2 depicts cumulative calorie intake across the day in 1-h increments. There was a significant effect for time ($P < 0.001$) and time × sleep timing ($P < 0.01$). Late sleepers consumed fewer calories early in the day including 9:00 AM ($P < 0.001$), 10:00 AM, 11:00 AM, and 12:00 PM ($P = 0.001$). Although late sleepers consumed more cumulative calories beginning at 10:00 PM, these differences were not statistically significant.

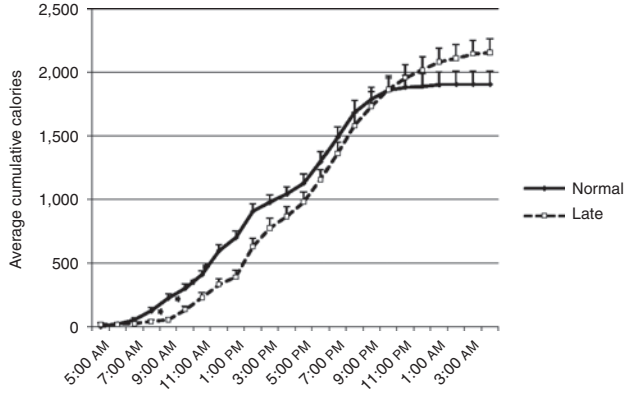

Figure 2 Cumulative caloric intake according to sleep timing. *$P < 0.002$.

Relationship between sleep timing and duration, caloric intake and BMI

Average BMI was 23.7 (s.d. = 3.2) in normal sleepers and 26.0 (s.d. = 6.9) in late sleepers ($P = 0.15$). A greater percentage of late sleepers reported BMI ≥30, but this was not statistically significant (2 of 27 normal sleepers and 6 of 22 late sleepers, $P = 0.12$). In correlation analyses (Table 4), BMI was positively correlated with later sleep timing ($P < 0.01$), calories consumed after 8:00 PM ($P < 0.01$), and fast food consumption ($P < 0.05$). There was a trend for a higher BMI in participants with shorter sleep duration ($P = 0.05$). Later sleep timing was also associated with fewer servings of fruits and vegetables ($P < 0.001$). Higher consumption of calories after 8:00 PM was associated with later sleep timing ($P < 0.001$), higher daily caloric intake ($P < 0.001$) and shorter sleep duration ($P < 0.001$). Later timing of last meal or snack of the day was associated with later sleep timing ($P < 0.001$) shorter sleep duration ($P < 0.001$) and also higher caloric intake ($P < 0.001$). Higher intake of full-calorie soda was associated with higher caloric intake ($P < 0.01$).

In multivariate regression analyses controlling for age and sleep duration, later sleep timing was associated with greater calories consumed after 8:00 PM ($\beta = 0.45$, $r^2_{\Delta} = 0.18$, $P = 0.001$) and fewer servings of fruits and vegetables ($\beta = -0.51$, $r^2_{\Delta} = 0.22$, $P = 0.002$). After controlling for age and sleep duration, sleep timing was not associated with BMI. Caloric

Table 4 Correlations between sleep duration and timing with BMI, caloric intake, and food types

	Sleep timing	Sleep duration	BMI	Total calories	Calories after 8:00 PM	Last meal or snack	Fast food	Fruits and veg	Soda
Sleep timing	—								
Sleep duration	–0.33*	—							
BMI	0.41**	–0.29†	—						
Total calories	–0.19	–0.19	0.26†	—					
Calories after 8:00 PM	0.56***	–0.57***	0.37**	0.59***	—				
Last meal or snack	0.52***	–0.50***	0.26†	0.52***	0.74***	—			
Fast food	0.25	–0.31*	0.31*	0.15	0.15	0.27†	—		
Fruits and veg	–0.49**	0.18	–0.17	0.20	–0.14	0.18	–0.45**	—	
Soda	0.27†	–0.07	0.16	0.39**	0.27†	0.25†	0.16	–0.12	—

Fruits and veg, servings of fruits and vegetables per day; sleep duration, actual sleep determined by actigraphy; sleep timing, midpoint of sleep determined by 7 days of actigraphy; soda, servings of full-calorie soda per week; total calories, average caloric intake per day.
*$P < 0.05$, **$P < 0.01$,***$P < 0.001$, †$P < 0.10$.

consumption after 8:00 PM was an independent predictor of BMI ($\beta = 0.44$, $r^2_{\Delta} = 0.09$, $P = 0.03$), after controlling for age, sleep duration, and sleep timing. Furthermore, caloric intake after 8:00 PM predicted total caloric intake after controlling for age, sleep duration, and sleep timing ($\beta = 0.72$, $r^2_{\Delta} = 0.35$, $P < 0.001$).

Activity

Average 24-h activity counts based on 7 days of actigraphy were similar for normal and late sleepers (M = 277,794 s.d. = 101,680 in normal sleepers and M = 307,752 s.d. = 81,372 in late sleepers, $P = 0.27$) and activity was not correlated with sleep timing, sleep duration, total caloric intake, or calories consumed after 8:00 PM. There was a trend for a negative association between activity and BMI ($r = -0.27$, $P = 0.07$).

DISCUSSION

The aim of this study was to determine whether the timing of the habitual sleep–wake schedule was associated with dietary patterns and BMI. As expected, individuals with later sleep–wake timing consumed meals at a later clock time than those with normal sleep–wake timing. Later sleep timing was also associated with greater calories consumed after 8:00 PM and fewer servings of fruits and vegetables even after controlling for age and sleep duration. In categorical analyses, late sleepers consumed on average 248 more calories per day than normal sleepers, with the majority of the excess calories occurring at dinner and after 8:00 PM. Although the difference in total daily calories was not statistically significant, this difference would roughly equate to 2 lb per month if this difference persisted over time and was not balanced by greater energy expenditure. We also found some support for our hypothesis that sleep timing would be associated with BMI, however, it was not independent of sleep duration. This finding should be explored further under more controlled conditions because in our sample, sleep timing was associated with sleep duration, making it difficult to decipher the independent effects. Perhaps the most robust finding was that caloric consumption after 8:00 PM was associated with BMI, independent of age, sleep timing, and sleep duration. This suggests that greater caloric consumption in the evening may be a behavior that links later sleep timing to obesity risk.

In addition to all meal and snack times occurring at a later clock time in late sleepers, there were also differences in the duration between meals and sleep. In the late sleepers, there was a shorter duration between breakfast and lunch and a longer duration between their last reported meal or snack, and sleep onset. It is unclear whether these differences are related to misalignment of their appetite or other physiological rhythms with social meal times, but it does indicate that there are alterations in the meal patterns of late sleepers compared with normal sleepers.

This alteration in meal pattern was also reflected in the pattern of caloric intake across the day. In this study, caloric intake was examined in several ways including calories consumed at each meal, calories after dinner, calories after 8:00 PM and cumulative calories across the 24-h day. Late sleepers consumed significantly more calories at dinner and after 8:00 PM. We also found that those who ate later in the evening consumed more calories over the 24-h period. Previous studies have also shown an association between calories consumed in the morning or at breakfast and obesity risk. For example, greater energy intake at breakfast has predicted concurrent BMI and weight gain over time (33,34). In our sample, when we examined frequency of breakfast consumption, there were no differences between the sleep timing groups in caloric intake at breakfast or the number of individuals who ate or "skipped" breakfast. However, evaluation of cumulative caloric intake across the day demonstrated that late sleepers consumed significantly fewer calories in the morning hours, and then steeply increased their intake in the afternoon at which point their caloric intake matched and began to exceed normal sleepers around average dinner time. Caloric intake of late sleepers continued to rise after 11:00 PM but normal sleepers reached a plateau as early as 9:00 PM. This may simply be the result of later sleepers not being awake until a later clock time and thus eating less in the morning and then overconsuming calories later in the day.

In addition to the difference in meal timing and caloric intake, late sleepers reported poorer food choices including nearly half the servings of fruits and vegetables, twice the fast foods and twice the full-calorie sodas as normal sleepers. These findings are important given the role of diet quality in weight regulation. For example, multiple observational and intervention studies support that increasing high fiber and low calorie foods is a useful strategy in weight management (35,36). It is interesting that despite these differences in food choices, there were no differences in the macronutrient content of the foods consumed between the groups and not a statistically significant difference in overall caloric intake. This may reflect biases in reporting on diet logs, which is more pronounced in overweight individuals (37). In addition, analyses of food diaries using public databases are likely to be less precise than using a nutrition research database such as the Nutrition Data System for Research (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN).

Our results are consistent with a few recent studies that have found poorer dietary patterns associated with later sleep timing and diurnal preference (21,38). In a study conducted in German adolescents, later self-reported sleep timing on days off from school was related to poorer dietary habits, including caffeine, fast food consumption, and dairy. In addition, Schubert and colleagues (38) reported lower perceived hunger and disinhibition (i.e., tendency to overeat) in those who saw themselves as more "morning types". However, this study neither evaluated evening preference nor the effect of sleep timing. Our study builds on the results of these two studies by objectively measuring sleep timing, controlling for sleep duration, and extending the results to adults.

In our sample, there were only two individuals (one normal sleeper, one late sleeper) who exhibited at least one of the core criteria for Night Eating Syndrome. Despite the infrequency of this syndrome in our study, the pattern of caloric intake in our sample over the 24-h period was similar to that of described in several previous studies of Night Eating Syndrome (24,25,39). Combined with our results, these studies suggest that Night Eating Syndrome is associated with a similar 24-h pattern caloric consumption, but having a late sleep pattern alone is not necessarily consistent with this eating disorder diagnosis. Furthermore, the delayed consumption of calories may be a shared risk factor for obesity between these two overlapping populations.

Our results are limited by use of a convenience sample, reliance on self-reported measures of dietary behavior and weight, and lack of objective metabolic and hormonal markers. Thus, this study cannot directly address the biological mechanisms linking sleep or circadian timing with appetite and weight regulation. Another limitation is that it is very difficult to completely dissociate the role of timing of sleep and sleep duration. Alteration in sleep or circadian timing is invariably accompanied by changes in sleep duration. In our study, late sleepers slept on average ~1 h less than the normal sleepers. This finding may be due to the decreased opportunity for sleep or inability to sleep at adverse circadian times. There is evidence that sleeping at the "wrong" circadian time affects sleep quality (40). But also there is evidence to support the notion that misalignment between sleep time and endogenous circadian rhythms (i.e., sleeping and eating 12 h out of phase), can produce alterations in cardiometabolic function after controlling for sleep efficiency, which suggests that circadian misalignment plays an independent role (16).

Future studies with circadian markers would be useful to determine whether late sleeping is due to delayed circadian timing, and whether there is misalignment between circadian rhythms with feeding and sleeping time. For example, feeding during the nonoptimal time may affect meal pattern and food choice. In addition, there may also be a genetic vulnerability in late sleepers that may predispose them to poor eating patterns, metabolic dysregulation, and obesity (41). Kohsaka and colleagues reported that mice that were fed a high-fat diet had alterations in the timing of activity, feeding behavior, and expression of circadian clock genes and related transcription factors (42). Moreover, Turek *et al.* found that mice with a mutation in the circadian *Clock* gene that results in an unusually long circadian period and shorter sleep duration are particularly susceptible to weight gain and metabolic disturbance when placed on a high-fat diet relative to wild-type mice (43,44). In addition, one study indicates that humans with variants in a *Clock* transcription factor are at increased risk for obesity (45), while another implicates *Clock* gene polymorphisms with a role in metabolic disorders (46). Such findings suggest that the interactions between circadian timing, circadian genes, and dietary behaviors regulate body weight. How these factors interact require further investigation.

Results of this study are of further interest in view of recent studies in mice indicating that feeding time relative to sleep–wake and light–dark cycle can influence body weight gain. A recent study by Arble and colleagues demonstrated increased weight gain in mice fed only during the light phase (a time when they are typically less active) compared to mice fed during the dark phase (a time when they are most active and typically feed) (47). Another study found that animals exposed to constant dim light or constant bright light had increased body mass and impaired glucose metabolism, compared to mice on a typical 12-h light–dark cycle (48). They also showed that restricting feeding to only the active phase prevented the body mass increases regardless of light levels. The relationship between the time of feeding and light exposure have not been explored in humans. We can postulate that having a later sleep time is likely to influence exposure to bright light, which may be an additional mechanism that may influence obesity. Late sleepers may have reduced light exposure during the day and/or higher light exposure at night, which may lead to consuming calories at that time. There is data to support that individuals with a preference for the evening have reduced light exposure over the day but higher light levels in the late evening (49). Future studies examining light exposure in relation to diet, or manipulating the timing of feeding in humans in relation to the light–dark cycle may be useful in determining whether controlling light exposure and/or timing of food intake can influence weight regulation in humans.

In summary, our data demonstrate that the timing of sleep affects dietary behavior and timing of caloric intake. Interestingly, even after controlling for sleep duration and timing, consuming calories in the evening was associated with a higher BMI. When taken together with previous human and animal data, these results underscore the importance of sleep timing and the timing of calorie intake when assessing the relationship between food intake and obesity. Large population studies with detailed timing of both feeding and sleep–wake are needed to determine the generalizability of our findings. In addition, well-controlled physiological studies using biomarkers of sleep, circadian rhythms, and metabolism will be necessary to understand the processes linking circadian rhythms and sleep with behavioral and biological mechanisms involved in weight regulation.

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DISCLOSURE

The authors declared no conflict of interest.

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