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Daily Associations between Objective Sleep and Consumption of Highly Palatable Food in Free-Living Conditions

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ORIGINAL ARTICLE

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Summary

Objectives

Prior studies have shown that individuals with shorter sleep duration and later sleep timing consume more highly palatable food (HPF). It is unclear if this relationship exists at the within-individual level, e.g. if sleeping less or later on one night is associated with greater HPF consumption in the following day in naturalistic environments. This study examined the daily associations between naturalistic sleep and HPF consumption.

Methods

Data were obtained from 78 healthy young adults (age = 20.38 [SD = 2.40] years). Participants carried a wrist actigraph and completed daily diaries tracking food consumption and covariates for seven consecutive days. Data were analysed using mixed models.

Results

Individuals with later bedtime were less likely to consume HPF at breakfast in the following day (odds ratio, OR [between] = 0.55 [0.44, 0.70], $p < 0.001$). This association was also significant at the within-individual level (OR (within) = 0.85 [0.74, 0.97], $p = 0.016$) – sleeping later on one night was associated with 15% decrease in the odds of consuming HPF at breakfast in the following day. Individual with later wake time had greater likelihood of consuming HPF at dinner (OR = 1.34 [1.03, 1.75], $p = 0.027$).

Conclusions

Sleep schedules characterized by later bedtimes and later wake times were associated with lower HPF consumption earlier in the following day but greater HPF consumption later in the day. This pattern of energy intake might mediate the association between sleep and the risk of obesity.

Keywords: Appetite regulation, energy intake, high-fat diet, sleep timing.

Poor sleep and short sleep duration are associated with weight gain and obesity (1–3). One of the most probable mechanisms by which sleep influences body mass is through its impact on energy balance (4). Experimental sleep restriction has been shown to be associated with increased caloric consumption (5–9). In particular, the increase in caloric consumption was found to be a result of increased consumption of highly palatable food (HPF), characterized by high fat or high carbohydrate content, especially during evening hours (5–7,10).

Studies on experimental sleep restriction and food consumption provided compelling evidence for the role of sleep in altering food consumption. However, food consumption in controlled settings, especially *ad libitum* food consumption in a constrained environment with restricted activities, might be substantially different from dietary habits in naturalistic environments. Although food is abundant in the modern world, work and school, social cues and norms, costs of food and food preparation, food advertising and so forth all influence food intake in free-living conditions (11–13). The generalizability of the effect of experimental sleep restriction on food consumption within the laboratory may be limited. Self-reported naturalistic sleep duration has been shown to be associated

with increased preference for calorie-dense food and increased caloric consumption (14). However, the association between self-reported sleep duration and food consumption might be explained by psychological distress underlying self-reported poor sleep quality (15). The use of sleep measures that are not biased by subjective distress is needed to clarify the association between naturalistic sleep and food consumption.

Several studies have found that actigraphy-assessed short sleep duration in naturalistic settings is associated with poorer dietary habits (16) and greater proportion of caloric consumption from fats (17). Individuals with actigraphy-assessed late sleep timing were also found to have greater consumption of calorie-dense food (18), especially in the evening hours (19), than individuals with early bedtime and wake time. These *between-individual* associations, however, could be explained by inter-individual differences in unmeasured variables. For instance, individuals who sleep less or have later sleep timing tend to engage in other unhealthy behaviour such as smoking, less physical activity, and greater alcohol use (20). Evening chronotype is also associated with higher levels of psychological stress (21), which is linked to emotional eating and increased caloric consumption (22). These factors could be contributing to both inadequate sleep or late sleep timing and greater consumption of HPF. Prior studies have not examined whether the association between sleep and consumption of HPF exists at the *within-individual* level, i.e. if sleeping less or later on one night is associated with greater consumption of HPF in the following day for the same individual in free-living conditions. An association at the within-individual level will be stronger evidence for the causal effect of sleep on dietary behaviour in naturalistic environments.

The present study examined the associations between actigraphy-assessed naturalistic sleep and consumption of HPF in free-living conditions in a sample of young adults. Based on the literature on food craving, HPF was defined as foods that have high fat or high carbohydrate content including high-fat foods, fast foods, sweets, starch and sugary drinks (23). It was hypothesized that shorter sleep duration, later bedtime, and later wake time would be associated with greater consumption of HPF at both the between-individual and within-individual levels.

Methods

Participants and procedure

This study recruited 84 relatively healthy young adults, who consumed no more than five cigarettes and three cups of coffee per day and no more than 15 alcoholic beverages per week, did not have a psychiatric diagnosis at the time, had not been diagnosed with any neurological disorders, were not on any psychostimulants or psychiatric medications and were not on any restrictive diets to lose weight at the time. They were recruited through flyers posted in the university library and psychology department. All participants were either undergraduate or graduate students. Informed consent was obtained from participants on paper forms by an experimenter. Participants were informed that the purpose of the study was to examine the associations between sleep and eating habits and that they would be compensated for their participation with $30 cash.

Participants were asked to wear a wrist actigraph for seven consecutive days and also complete daily tracking of food consumption for the same 7 d. Four of the recruited participants withdrew from the study and did not provide any data. Two participants did not complete the data collection procedures, and their actigraphy data indicated that they did not wear the actigraphs as instructed, i.e. they did not wear the actigraphs during their sleep. Hence, data from these two participants were excluded from the study. Data from a total of 78 participants were included in this study. The Institutional Review Board approved the research protocol before participant recruitment began.

Assessment

Sleep parameters

Sleep parameters were assessed using actigraphy (Ambulatory Monitoring, Inc., Ardsley, NY. USA). Participants wore an actigraph on their non-dominant wrist. They recorded their time in bed and time out of bed on a daily tracking form. The variables used in this study included *sleep duration*, the actual time asleep at night in minutes; *bedtime*, the time the first asleep episode was initiated; and *wake time*. The algorithm used to estimate these variables has been validated against polysomnography (24,25).

Food consumption in free-living conditions

Preference for HPF in free-living conditions was assessed by food diaries. Participants were instructed to record food and beverages consumed for breakfast, lunch, dinner and snacks each day on the daily tracking form. Based on prior research on food craving, five groups of food were identified as HPF – (i) high fats, such as chicken nuggets, sausages, bacon, beef, cheese and gravy; (ii) fast food, such as pizza, French fries, hamburgers and tacos; (iii) sweets, such as pastries, candies and ice cream; (iv) carbohydrates, such as pasta, bagels,

muffins, pancake and cereal; and (v) sugary drinks, such as flavoured milk, milkshake, fruit juice, soda and sport drinks (23). Because the amount of food consumed was not recorded, the dependent variable was the presence or absence of highly palatable food in a meal, coded as 1 or 0. The dependent variables derived from the daily food tracking include the consumption of highly palatable food in (i) breakfast, (ii) lunch, (iii) dinner and (iv) snacks.

Covariates

On the daily tracking form, participants were asked to report on each day their (i) daytime nap duration; (ii) the number of minutes of exercise and (iii) walking; (iv) the number of cups of alcohol and (v) caffeinated beverages they consumed; and (vi) their perceived stress each day. Perceived stress was measured by the 4-item Perceived Stress Scale (26). Items included "Feel unable to control important things in your life?" and "Feel confident about your ability to handle your personal problems?" Participants rated each of the items on a 1–5 response scale indicating how the statement applied to them with 1 indicating "never" and 5 "always." The scores for the 4 items were summed to form an overall perceived stress index. Body mass index was computed as weight in kilograms divided by height in metres squared. Body height was measured using a wall-mounted measuring tape, and weight was measured by an electronic digital scale, following the food tasting task.

Data analysis

Mixed modelling was conducted to examine the daily associations between sleep and consumption of HPF using the *lmer* package in R studio version 0.98.1103 (27). The *glmer* function was used to conduct logistic multilevel models predicting the odds of consuming HPF in each meal of the day. The rates of missing data ranged from 10% to 20%, typical of longitudinal psychological and behavioural research (28). Under the assumption that missing is at random, both full information maximum likelihood (FIML) and multiple imputation would be appropriate strategies for handling missing data. Given that FIML requires distributional assumptions and that the present dataset contained a mix of categorical and continuous variable, multiple imputation that allows more flexibility for data distributions was used to handle missing data (29). Multiple imputation was conducted using the *mice* package in *R* specifically designed for multilevel data (30).

In order to evaluate associations between sleep parameters and HPF consumption at both the within-individual and between-individual levels, both the fixed effect of the within-individual sleep parameter and the fixed effect of the between-individual variability of the sleep parameter were included in each model. For instance, the equation of the multilevel model for sleep duration is as follows:

$$\begin{aligned}(\text{consumption of HPF})ij = {} & \gamma_{00} + u_{00} \\ & + \gamma_{10}\,(\text{previous} - \text{night sleep duration})_{ij} \\ & + u_{10} \\ & + \gamma_{01}\,(\text{weekly average of sleep duration})_{j} \\ & + R_{ij}.\end{aligned}$$

The dependent variable is the consumption of HPF on a given day (*i*) by a participant (*j*), which is a function of the fixed intercept (γ_{00}), the random intercept (u_{00}), the fixed effect of previous-night sleep duration (γ_{10}, i.e. within-individual effect of sleep duration), the random slope (u_{10}), the fixed effect of the weekly average of sleep duration (γ_{01}, i.e. between-individual effect of sleep duration) and error (R_{ij}). Covariates that were found to be significantly associated with the outcome variables were added to the models as fixed effects. The same model was evaluated for each sleep parameter (sleep duration, bedtime and wake time). All within-individual variables were centred by group (person) means to capture only within-individual variability, while between-individual variables were centred by the grand mean (31). In order to evaluate the independent effects of each sleep parameter, a multivariate model consisting of all the within-individual and between-individual effects of sleep duration, bedtime and wake time were tested.

Results

The demographic information of the sample is presented in Table 1. The sample was primarily white, normal-weight young adults with roughly the same number of men and

Table 1 Participant characteristics

	Mean/percentage
Age	20.38 years (SD = 2.40; range = 18–30)
Race	
Asian or Pacific Islander	10%
Black	11%
Hispanic or Latino	3%
White	76%
Sex	56% female
BMI	23.0 kg m^{-2} (SD = 3.5; range: 17.0–35.3)

BMI, body mass index.

women. The means, standard deviations and ranges of values of all variables are presented in Table 2. Sleep duration is negatively correlated with bedtime ($r = -0.57$, $p < 0.001$) but not with wake time ($r = 0.04$, $p = 0.744$). Bedtime is positively correlated with wake time ($r = 0.56$, $p < 0.001$).

Table 3 presents the number of days over a week participants consumed HPF at each meal by sleep duration, sleep timing and sleep timing variability. Participants with average sleep duration shorter than 6 h, bedtime later than 2 a.m., bedtime variability greater than 90 min or wake time variability greater than 90 min consumed HPF at breakfast less frequently. Participants whose wake time variability was greater than 90 min consumed HPF at dinner more frequently. Among the covariates, women had greater likelihood of consuming HPF than men at breakfast (odds ratio, OR = 2.03 [1.33, 3.10], $p = 0.001$) and at lunch (OR = 1.52 [1.01, 2.59], $p = 0.044$). Caffeine consumption was negatively associated with likelihood of consuming HPF at lunch (OR = 0.50 [0.34, 0.74], $p < 0.001$). Exercise was associated with greater likelihood of consuming highly palatable snacks (OR = 1.01 [1.00, 1.01], $p = 0.029$). These covariates were then included in the subsequent analysis.

Table 4 presents the results of the mixed models. Both the between-individual and within-individual effects of bedtime on breakfast consumption of HPF were significant. The between-individual effect indicated that participants who had later bedtimes were less likely to consume HPF at breakfast than those who had earlier bedtimes (OR = 0.55, $p < 0.001$). There was a 45% reduction of the odds of consuming HPF for breakfast for each hour delay in bedtime. The within-individual effect indicated that participants were less likely (15% reduction of odds for each hour of delay) to consume HPF at breakfast on days when they went to bed later in the previous night than on other days when they went to bed earlier (OR = 0.85, $p = 0.016$). The between-individual effect of wake time on the likelihood of consuming HPF at dinner was significant, indicating that individuals who woke up later were more likely to consume HPF at dinner than those who woke up earlier (OR = 1.34, $p = 0.027$). There was an increase of 34% in odds of consuming HPF at dinner for each hour delay in wake time. Sleep duration was not significantly associated with the likelihood of HPF consumption at either between-individual or within-individual levels. However, there was a trend that individuals with longer sleep duration had greater odds of consuming HPF at breakfast (OR = 1.34, $p = 0.066$).

In the multivariate models with all sleep parameters and covariates included as predictors, the between-individual effect of bedtime was significant in predicting the odds of consuming HPF at breakfast and lunch (see Table 4). Individuals with later bedtimes had lower odds of consuming HPF (breakfast: OR = 0.51, $p = 0.001$; lunch: OR = 1.42, $p = 0.029$). On the other hand, individuals with later wake times had greater odds of consuming HPF at lunch.

Sensitivity analysis was conducted to evaluate if the results would be different using FIML instead of multiple imputations. All results were comparable and had the same patterns of association and levels of significance, except that the significance of the within-individual effect of bedtime on HPF consumption at breakfast reduced (OR = 0.83, $p = 0.16$). The reduction of significance level was likely due to reduced statistical power in FIML when about 30% of the rows (days) of data were omitted due to missing values.

Table 2 Means, standard deviations and ranges of values of main variables

	Mean	Standard deviation	Range
Sleep variables – weekly averages:			
Sleep duration (hr)	5.92	1.25	2.15–8.14
Bedtime (hh: mm)	02:28	70.12 min	23:32–05:03
Wake time (hh: mm)	08:58	61.20 min	6:38–11:21
Number of days participants consumed highly palatable food:			
Breakfast	2.98	2.19	0–7
Lunch	2.89	1.69	0–7
Dinner	3.49	1.79	0–7
Snacks	2.97	2.14	0–7
Covariates			
Perceived stress	7.64	2.28	4.00–11.84
Number of caffeinated beverages	0.29	0.53	0–2
Number of alcoholic beverages	0.15	0.64	0–4
Minutes of walking	35.9	42.9	0–340
Minutes of exercise	5.4	23.6	0–180

Discussion

The present study examined the daily associations between objective sleep and the likelihood of consuming HPF in free-living conditions in a sample of healthy young adults. Shorter sleep duration and later sleep timing were hypothesized to be associated with greater consumption of HPF. Partly consistent with the hypotheses, individuals who had later wake times had greater odds of consuming HPF at dinner compared with individuals who had earlier wake times. When sleep duration and bedtime were taken into account, individuals with later wake times had greater odds of consuming HPF at lunch. On the other hand, later bedtime was found to be associated with lower odds of consuming HPF at breakfast at both the between-individual and within-individual levels. The

Table 3 Consumption of highly palatable food (number of days in a week) by meal and sleep parameters

	Breakfast	Lunch	Dinner	Snack
Sleep duration >7 h	3.81	3.11	3.69	3.17
Sleep duration <7 h	2.63	3.00	3.37	3.34
	$t = 2.42$, $p = 0.018$	$t = 0.29$, $p = 0.770$	$t = 0.77$, $p = 0.447$	$t = -0.35$, $p = 0.726$
Bedtime earlier than 2 a.m.	4.03	3.40	3.53	3.23
Bedtime later than 2 a.m.	2.64	2.81	3.52	3.27
	$t = 2.86$, $p = 0.006$	$t = 1.52$, $p = 0.133$	$t = 0.02$, $p = 0.981$	$t = -0.08$, $p = 0.938$
Wake time earlier than 9 a.m.	3.60	3.17	3.54	3.26
Wake time later than 9 a.m.	2.85	2.95	3.51	3.26
	$t = 1.52$, $p = 0.132$	$t = 0.59$, $p = 0.555$	$t = 0.06$, $p = 0.944$	$t = 0.001$, $p = 0.999$
Sleep duration variability <90 min	371	2.88	3.59	3.32
Sleep duration variability >90 min	2.78	3.20	3.48	3.20
	$t = 1.88$, $p = 0.064$	$t = -0.84$, $p = 0.404$	$t = 0.26$, $p = 0.797$	$t = 0.24$, $p = 0.808$
Bedtime variability <90 min	3.78	2.96	3.44	3.31
Bedtime variability >90 min	2.31	3.21	3.66	3.17
	$t = 3.03$, $p = 0.004$	$t = 2.96$, $p = 0.512$	$t = -0.51$, $p = 0.613$	$t = 0.28$, $p = 0.778$
Wake time variability <90 min	3.86	3.03	2.94	3.40
Wake time variability >90 min	2.62	3.08	4.05	3.13
	$t = 2.54$, $p = 0.014$	$t = -0.13$ $p = 0.900$	$t = -2.67$, $p = 0.010$	$t = 0.54$, $p = 0.590$

within-individual association suggested that, for the same person, going to bed later for one night would decrease the likelihood of consuming HPF in the next morning. This association was unlikely confounded by inter-individual differences in the likelihood of engaging in health behaviour. Furthermore, individuals with later bedtimes had lower odds of consuming HPF at breakfast and lunch even after controlling for sleep duration and wake time.

Table 4 Within-individual and between-individual associations between sleep parameters and consumption of highly palatable food by meal

	Breakfast[a]		Lunch[b]		Dinner		Snacks[c]	
	Odds ratio	*p*	Odds ratio	*p*	Odds ratio	*p*	Odds ratio	*p*
Model 1								
Intercept	0.11 [0.31, 0.87]	0.013	0.75 [0.53, 1.05]	0.096	1.06 [0.81, 1.39]	0.655	0.63 [0.43, 0.94]	0.023
SD (within)	1.15 [0.97, 1.36]	0.104	0.98 [0.85, 1.14]	0.816	1.06 [0.91, 1.23]	0.453	0.98 [0.85, 1.13]	0.783
SD (between)	1.34 [0.98, 1.82]	0.066	0.98 [0.80, 1.19]	0.802	1.00 [0.81, 1.24]	0.984	1.00 [0.75, 1.33]	0.981
Model 2								
Intercept	0.60 [0.41, 0.87]	<0.007	0.79 [0.56, 1.12]	0.187	1.06 [0.81, 1.39]	0.649	0.63 [0.43, 0.94]	0.024
BT (within)	0.85 [0.74, 0.97]	0.016	0.93 [0.81, 1.07]	0.329	1.06 [0.92, 1.21]	0.419	0.95 [0.83, 1.10]	0.521
BT (between)	0.55 [0.44, 0.70]	<0.001	0.91 [0.74, 1.13]	0.407	1.17 [0.93, 1.47]	0.180	1.05 [0.77, 1.43]	0.745
Model 3								
Intercept	0.48 [0.29, 0.80]	0.005	0.75 [0.53, 1.04]	0.087	1.07 [0.82, 1.39]	0.636	0.63 [0.43, 0.94]	0.023
WT (within)	0.92 [0.80, 1.06]	0.257	0.94 [0.83, 1.07]	0.341	1.08 [0.94, 1.24]	0.260	0.99 [0.86, 1.47]	0.898
WT (between)	0.75 [0.52, 1.07]	0.111	1.09 [0.86, 1.37]	0.478	1.34 [1.03, 1.75]	0.027	1.23 [0.86, 1.76]	0.259
Model 4								
Intercept	0.60 [0.41, 0.89]	0.011	0.79 [0.56, 1.12]	0.189	1.07 [0.82, 1.39]	0.631	0.63 [0.43, 0.93]	0.022
SD (within)	1.21 [0.98, 1.48]	0.073	0.95 [0.77, 1.18]	0.664	1.10 [0.87, 1.38]	0.427	0.92 [0.75, 1.12]	0.409
SD (between)	0.98 [0.72, 1.33]	0.893	0.79 [0.61, 1.02]	0.072	1.01 [0.74, 1.37]	0.966	0.91 [0.60, 1.38]	0.652
BT (within)	0.97 [0.79, 1.20]	0.811	0.92 [0.75, 1.12]	0.394	1.10 [0.88, 1.37]	0.403	0.90 [0.74, 1.10]	0.307
BT (between)	0.51 [0.35, 0.76]	0.001	0.66 [0.47, 0.93]	0.018	1.02 [0.70, 1.51]	0.892	0.86 [0.50, 1.46]	0.565
WT (within)	0.86 [0.73, 1.02]	0.085	0.98 [0.82, 1.17]	0.812	1.02 [0.84, 1.23]	0.862	1.05 [0.87, 1.27]	0.610
WT (between)	1.12 [0.86, 2.77]	0.547	1.42 [1.04, 1.96]	0.029	1.33 [0.93, 1.90]	0.127	1.36 [0.83, 2.26]	0.221

[a]Adjusted for sex.
[b]Adjusted for sex and caffeine consumption.
[c]Adjusted for duration of exercise.
BT, bedtime; SD, sleep duration; WT, wake time.

Taken together, the present findings suggested that a sleep pattern characterized by later bedtime and later wake time was associated with a delayed shift of HPF consumption, which might predispose one to weight gain and obesity.

The present findings were largely consistent with prior studies in which greater caloric consumption was observed in the evening hours among individuals with experimentally delayed bedtime (5) or late sleep timing in free-living conditions (19). The significant association between later bedtime and lower odds of consuming HPF at breakfast in free-living conditions was novel. This effect was significant at the within-individual level indicating that delaying bedtime for one night was associated with lower likelihood of consuming HPF in the following day. The significant between-individual effect of bedtime remained significant after controlling for sleep duration and wake time, suggesting that individuals with later bedtimes had lower likelihood of consuming HPF at breakfast even after the effects of sleep duration and wake time. Although non-significant, this pattern of decreased caloric consumption earlier in the day was also observed in Baron *et al*. Lower caloric consumption was also observed from 08:00 to 14:59 during *ad libitum* food access following experimentally delayed bedtime (5). Nonetheless, without measuring the calories consumed, the present findings might reflect a preference for HPF but not an increase in the number of calories consumed.

Late sleep timing might increase consumption of HPF in the evening through two pathways. First, prior research has found that there is an endogenous circadian rhythm in appetite and appetite for HPF, characterized by low appetite in the morning and a peak in appetite in the evening, independent from the effect of calories consumed, sleep duration or wake time (32). Delayed bedtime extends wake hours in the evening and thus increases opportunities for eating when appetite and especially appetite for HPF is the highest. Second, delayed wake time reduces the opportunities for calorie intake earlier in the day and likely results in greater hunger in the later part of the day. Indeed, in a sample of free-living adults in Japan, greater energy intake and especially caloric intake from fats in the morning was found to be associated with decreased caloric consumption in the rest of the day (33). Recent research suggests that greater consumption of calorie-dense foods earlier in the day might be associated with lower risks of obesity in adults. For instance, in a longitudinal population-based cohort study in Spain, greater caloric consumption at lunch was associated with lower risk of gaining 3 kg of weight over three and a half years (34).

Participants with less than 7 h of sleep on average consumed HPF at breakfast for fewer days. There was a trend association between shorter sleep duration and lower likelihood of consuming HPF at breakfast. In the multivariate models in which bedtime and wake time were taken in account, the effects of sleep duration on HPF consumption were not significant. These findings suggest that the effect of sleep duration on HPF consumption might be explained by sleep timing. In a prior experimental study, increased caloric consumption was observed in healthy adults following a night of delayed bedtime but not following five nights of sleep restriction, suggesting that delayed bedtime rather than sleep duration was more predictive of caloric consumption in controlled settings (5). Alternatively, the non-significant effect of sleep duration might be due to inadequate statistical power for detecting small effects. Future studies with larger sample sizes in free-living conditions are needed to clarify the independent and combined effect of sleep duration and sleep timing on weight gain and the risk of obesity in naturalistic environments.

Participants with greater sleep duration variability and sleep timing variability also demonstrated a pattern of lower HPF consumption earlier in the day but greater consumption in the evening. Prior studies have found that higher sleep duration and sleep timing variability were associated with greater caloric intake and body mass index (35,36). The present findings suggest that the association between sleep variability and risk of obesity might also be mediated by a delayed shift of HPF consumption. Additionally, highly variable sleep schedules might lead to variable meal timing, which is associated with decreased thermic effect of food and subsequent decreased energy expenditure (37) and subsequently increases the risk of weight gain and obesity.

Most of the significant associations found in this study were small effects, and actual caloric consumption was not measured. While recent research has consistently found a relationship between sleep timing and dietary patterns, the association between sleep timing and obesity or body mass has not been consistently observed (38). The effects of sleep on weight changes might be rather small and might only be clinically significant over a long period of time. Alternatively, the relationship between sleep and weight might exist in some people but not others (36,38,39). Future studies are needed to examine the relationship between naturalistic sleep and risks of obesity for longer periods of time or in larger samples that allow for the examination of moderation effects. These studies are necessary to advance the understanding of the clinical implications of sleep in the prevention and treatment of obesity.

The sample was a convenient sample of young adults who were all students at the time whose sleep schedules might differ significantly from working young adults.

Hence, the present findings might not generalize to working young adults and older adults. Additionally, this sample consisted of relatively healthy adults. The relationship between sleep and food consumption might differ among individuals with obesity. The paper food diaries used to access HPF consumption might subject to self-report biases. Twenty-four-hour dietary recall interviews would provide more accurate estimations of food consumption. Moreover, the amount and nutritional contents of food consumed were not assessed. Without data on energy and nutrient intakes, the present findings of the effects of sleep timing on HPF preferences could not be interpreted as effects on actual caloric consumption. The timing of each meal was not recorded. Meal timing could vary across individuals and might confound the results. Additionally, the timing of menstruation was not assessed in the female participants. Research suggests that menstrual cycle might impact food consumption in female participants and confound the results of this study. Finally, although the rates of missing data of this study were typical of longitudinal behavioural research, new technology could be used to reduce the rates of missing data such as using wrist-worn actigraphs that can momentarily detect off-wrist periods and generate messages to remind participants to wear actigraphs and complete daily measurements.

Conclusions

This study found that individuals with later sleep timing had lower consumption of HPF earlier in the following day but greater consumption of HPF later in the following day in free-living conditions. Additionally, within-individual analysis indicated that going to bed later on one night was associated with lower consumption of HPF at breakfast in the following day. These findings suggest that later sleep timing was associated with a clockwise shift of the preference for HPF. Future research is needed to evaluate whether preference for HPF later in the day contributes to increased caloric consumption and/or increased risk of weight gain.

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Conflict of interest statement

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