ORIGINAL RESEARCH

CURRENT DEVELOPMENTS IN NUTRITION

Eating behavior and qualitative assessments

Breakfast Consumption Augments Appetite, Eating Behavior, and Exploratory Markers of Sleep Quality Compared with Skipping Breakfast in Healthy Young Adults

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Abstract

Background: Observational studies show associations between breakfast skipping, reduced satiety, and poor sleep quality; however, intervention studies are lacking.

Objective: The purpose of this study was to examine the effects of consuming breakfast compared with breakfast skipping on appetitive, hormonal, and neural markers of appetite and satiety; ad libitum food intake; and exploratory measures of sleep health in young adults.

Methods: Thirteen adults [aged 23.5 ± 0.9 y (mean ± SEMs); body mass index (kg/m^2): 23.6 ± 0.6] completed the following randomized crossover-design study. Participants consumed a high-protein breakfast ("Breakfast"; 340 kcal, 30 g protein, 36 g carbohydrate, 9 g fat) or skipped breakfast ("Skip") for 7 d/treatment. On day 7, an 8-h clinical testing day was completed including assessments of hunger, fullness, desire to eat, prospective food consumption (PFC), related hormones, food cue–stimulated functional magnetic resonance imaging brain scans, and ad libitum evening food intake. Sleep quantity and quality were assessed with 7-d actigraphy, 7-d sleep diaries, and sleep-related hormones.

Results: Morning and daily hunger, desire to eat, PFC, and ghrelin decreased, whereas fullness increased after the Breakfast pattern compared with after the Skip pattern (all, $P < 0.05$). No difference in peptide YY (PYY) concentrations were detected. Hippocampal, parahippocampal, and middle frontal gyrus activations were reduced after the Breakfast pattern compared with the Skip pattern (all, $P < 0.01$). Although no differences in daily food intake were observed, the Breakfast pattern reduced evening intake of high-carbohydrate and high-fat foods ($P < 0.05$), whereas evening sugar intake tended to be reduced compared with the Skip pattern ($P = 0.085$). Although Breakfast led to shorter total sleep time (TST) compared with Skip ($P < 0.05$), no difference in sleep efficiency (TST/sleep period) was detected. Perceived sleep quality and sleep onset tended to improve after Breakfast compared with after Skip ($P = 0.060$ and $P = 0.07$, respectively).

Conclusion: Breakfast consumption improved appetite, satiety, and diet quality and may support some aspects of sleep health in healthy young adults. This trial was registered at clinicaltrials.gov as NCT03031132. *Curr Dev Nutr* 2018;0:nzy074.

Introduction

The prevalence of poor sleep quality in the United States has increased over the past 30 y at an alarming rate (1,2). According to the Behavioral Risk Factor Surveillance System (3, 4), >35% of young adults aged 18–34 y currently experience reduced sleep duration, poor sleep quality, or sleepiness during the day. Each of these components of sleep health contribute to increased public safety concerns, jeopardized work productivity, and increased health care needs and are associated with overall morbidity (4–6). Although sleep is multidimensional, as stated by the National Sleep Foundation (7), longer sleep latencies (i.e., time to sleep onset), greater number of awakenings,

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Supplemental Figures 1–3 and Supplemental Table 1 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/cdn/.

Abbreviations used: ES, effect size; PYY, peptide YY; TST, total sleep time.

longer wake time after sleep onset, and decreased sleep efficiency are all components of poor sleep quality.

There is increasing interest in the interactions between dietary patterns, ingestive behavior (i.e., markers of appetite and satiety), and sleep quality and their impact on the progression of obesity. Suboptimal sleep (i.e., reduced sleep time, poor sleep quality, and continuity) alters the physiologic and hedonic signals controlling eating behavior and contributes to overeating, weight gain, and obesity (1, 2, 8). Daily hunger (9–13); circulating ghrelin, which is associated with hunger and meal initiation (9, 13, 14); food cravings (14, 15); and the desire to overeat (9,13) are increased in adults with poor sleep compared with those with healthy sleep patterns and good sleep quality. In contrast, fullness and circulating peptide YY (PYY), a potent satiety signal, are blunted with poor sleep (16). These responses are accompanied by increases in daily food intake, primarily as evening snacking (9, 13), and a shift toward unhealthy eating practices that include skipping breakfast (17–19). In addition, orexin-A is a regulatory neuropeptide that controls arousal, wakefulness, sleep, and eating behaviors and is thought to increase feeding when sleep is shortened and a state of arousal or wakefulness is maintained (20, 21).

One potential target for promoting better sleep patterns includes the timing of food intake. Irregular eating practices, such as breakfast skipping, disrupt behavior patterns related to sleep-wake cycles and peripheral circadian "clocks," leading to impaired food intake regulation among other things (22).

The dietary habit of skipping breakfast is quite common in the United States, with as many as 60% of young adults frequently skipping the morning meal (23–25). Of concern is the relation between breakfast skipping, unhealthy eating behavior, and obesity (26). Our initial work centered around the addition of breakfast, particularly high-protein versions containing ~30 g protein, to support weight management by promoting appetite and satiety. The consumption of high-protein breakfasts augments satiety, through increased fullness and PYY concentrations; reduces neural food cravings; and reduces unhealthy evening snacking compared with skipping breakfast or consuming a normal-protein breakfast (27–30). Furthermore, high-protein breakfasts reduce daily food intake and prevent body fat gains over the longer term (31). These data support the daily consumption of a high-protein breakfast as a successful dietary strategy to assist in promoting healthy weight status. However, few studies have examined whether nutrition interventions, like breakfast consumption, influence healthy sleep patterns. Thus, the purpose of this study was to examine the effects of consuming a high-protein breakfast compared with breakfast skipping on markers of appetite, satiety, food reward, and food intake in healthy young adults. In addition, as an exploratory aim, we sought to examine whether breakfast behaviors affect sleep quality in an age group susceptible to poor sleep.

Methods

Experimental design

Thirteen healthy, normal-weight individuals completed a randomized crossover-design study (**Supplemental Figure 1**). Breakfast was either consumed ("Breakfast") or skipped ("Skip") for 7 d/pattern. Sleep quality was assessed throughout each 7-d pattern via daily actigraphy and daily sleep diaries. Salivary sampling was conducted during days 5–7 of each pattern to examine cortisol responses, which are related to both circadian timing and sleep (32). On day 7 of each pattern, participants arrived at the testing facility 1 h before their habitual breakfast time to complete a tightly controlled clinical testing day. The participants began the testing day by following their respective breakfast treatment. Questionnaires assessing hunger, fullness, desire to eat, prospective food consumption, sleepiness, and perceived energy in combination with blood sampling to measure plasma ghrelin, PYY, and orexin-A concentrations were completed throughout the testing day. Before lunch, participants were transported to the brain imaging center and a brain scan was completed using fMRI to identify neural activation in response to food stimuli. After the fMRI the participants returned to the clinical testing facility for the remainder of the testing day. Food intake and food choice were also assessed using a collection of foods provided when the clinical testing portion of the day was completed. This collection of foods is referred to hereafter as an ad libitum food packout. There was a 3- to 7-d washout period between treatments. This trial was registered at clinicaltrials.gov (NCT03031132).

Study participants

From June 2016 to August 2016, young adults were recruited through advertisements, fliers, e-mail listservs, and word-of-mouth to participate in the study. Eligibility was determined through the following inclusion criteria: *1*) age 20–32 y; *2*) BMI (kg/m^2) of 22–30; *3*) nonsmoker and nonuser of tobacco products; *4*) generally healthy (as assessed by medical history questionnaire); *5*) not pregnant or lactating in the past 6 mo; *6*) not clinically diagnosed with an eating disorder; *7*) no metabolic, hormonal, and/or neural conditions, diseases, or medications that influence metabolism or food intake; *8*) no known bleeding disorders; *9*) not currently or previously (in the past 6 mo) following a weight-loss or other special diet; *10*) no weight loss or gain (>4.5 kg) in the past 6 mo; *11*) normal cognitive restraint, as assessed by a score of <4 on the Three-Factor Eating Questionnaire; *12*) rating of ≥5 showing a minimum of "neither like nor dislike" on a 9-point hedonic scale rating for the study breakfasts; and *13*) not clinically diagnosed with obstructive sleep apnea or insomnia and does not participate in shiftwork.

Nineteen individuals met the initial screening criteria, and 13 ($n = 6$ men, $n = 7$ women) signed the study consent form and completed all study procedures. The participants were 23.5 ± 0.9 y of age (mean ± SEMs) and had a BMI of 23.6 ± 0.6. Furthermore, 12 of the 13 participants were habitual breakfast consumers and all participants had healthy sleep patterns, sleeping an average of 7.8 ± 0.3 h/night as indicated by the Pittsburgh Sleep Quality Index (33). Habitual wake time for all participants was 0709, ±65 min.

All of the participants were informed of the study purpose, procedures, and risks and signed the study consent forms. The study was approved by the institutional review board, and all procedures were followed in accordance with the ethical standards of the University of Missouri Institutional Review Board. The participants were compensated for completing all study procedures.

Breakfast treatments

During the 7-d Breakfast treatment, the participants were provided with their respective breakfasts and asked to consume these between 0700 and 0830 each day according to his or her habitual breakfast time. During the 7-d Skip treatment, the participants had nothing to eat or drink (besides water) until 1200.

For the Breakfast treatment, all foods were fully prepared in the metabolic kitchen using similar procedures as previously described (34). Participants picked up the meals on the day before the 7-d pattern, and reheating instructions were provided. As a measure of compliance, the participants were required to complete a breakfast food inventory log. The logs also provided instruction for when breakfast was to be consumed, which was set according to each participant's habitual weekday breakfast meal time. Participants were instructed to consume only foods provided to them or to skip breakfast, document all deviations (i.e., foods not consumed or extra foods consumed), and return all wrappers and uneaten foods to be reweighed. Compliance to the breakfast interventions was >90%.

The meals provided throughout the 7-d Breakfast pattern were isocaloric (340 kcal) and matched for macronutrient composition (30 g protein, 36 g carbohydrate, 9 g fat). The breakfasts included commonly consumed breakfast foods and were provided on the following days—days 1 and 4: waffles with applesauce dipping cup and scrambled eggs with Canadian bacon; days 2 and 6: scramble with peach cup; day 3: quesadilla with pineapple cup; and days 5 and 7 (testing day): French toast with syrup.

Appetite and satiety

Computerized questionnaires assessing perceived hunger, fullness, desire to eat, and prospective food consumption were completed during day 7 of each treatment as previously described (31). Blood samples to assess appetite- and satiety-related hormonal responses were also completed during day 7 of each treatment (29). Plasma active ghrelin and total PYY concentrations were assessed at each time point using ELISA assays (EMD Millipore). Plasma orexin-A concentrations were assessed at baseline and at +420 min using ELISA assays (Phoenix Pharmaceuticals).

fMRI neural responses

Brain activation responses were assessed before lunch during each testing day using food cue–stimulated fMRI. After the structural scan, the participants were instructed to focus on a set of photographs projected onto a screen, which incorporated stimuli from food, nonfood (e.g., animals), and blurred baseline images. The animal pictures were used to control for visual richness and general interest (i.e., appealing but not appetizing). The pictures from each category were presented in blocks of images. The functional scan lasted 7 min and was performed in triplicate. Scanning was performed on a 3-Tesla Siemens Trio scanner (Siemens Medical Solutions). Additional information is described in our previous studies (28, 29, 35).

Additional food provided

A standardized lunch (i.e., 500 kcal; 19 g protein, 69 g carbohydrate, 17 g fat) was provided on the clinical testing day (+300 min postbreakfast). The meal consisted of a deli sandwich (roll, cheese slice, turkey, lettuce, mayonnaise or butter), baked potato chips, and water (237 mL).

Before leaving the clinical testing facility (+450 min postbreakfast), the participants were provided with a cooler containing a variety of foods commonly consumed in the afternoon and evening as meals and snacks (**Supplemental Table 1**). All food items were preweighed and recorded. The participants were instructed to consume as much or as little of the foods as they wished (ad libitum) from this cooler until going to bed. Time of consumption was also recorded by participants and confirmed by study staff. All empty, partially consumed, and uneaten foods were returned and re-weighed (29). Energy and macronutrient content, time of food intake, and food type were assessed. The foods were then retrospectively grouped according to high-fat foods (i.e., >5 g fat/serving) and high-carbohydrate foods (i.e., >30 g carbohydrate/serving). In total, the packout included ~6400 kcal [430 g protein, 920 g carbohydrate (470 g sugar), 260 g fat].

Sleep measures

An actigraph (BodyMedia SenseWear; Jawbone) was worn on the upper arm starting on day 1 through the night of day 7. The armband uses an accelerometer, heat flux, galvanic skin response, skin temperature, and near-body ambient temperature sensors and is validated for the assessment of sleep-onset latency, total sleep time (TST), time in bed, awakenings (and duration), sleep efficiency (TST/total time in bed), and naps during daytime (36, 37). To support the actigraphy data, the validated Pittsburgh Sleep Diary (38) was completed every morning and night during days 1–7. Sleep and wake times, the day's events (that could influence sleep patterns), awakenings (and duration), and perceived sleep quality were collected. On day 7, computerized questionnaires (39) assessing perceived daytime sleepiness and daytime energy were completed throughout the testing period. The questionnaires contained visual analog scales incorporating a 100-mm horizontal line rating scale for each response. The questions were worded as "How sleepy do you feel" with anchors of "not at all" to "extremely."

Salivary sampling

Salivary samples were collected during day 7 of each treatment. Passive drool was collected using established procedures (40) through a Saliva Collection Aid (Salimetrics). The samples were frozen immediately at −20°C and then stored at −80°C until analysis. Salivary cortisol was measured using ELISA assays (Salimetrics).

Data and statistical analysis

Power analyses were performed before the start of the study to determine the sample size required to detect significant differences between the Breakfast and the Skip treatments. The effect size (ES) from our previous studies was determined for the following outcomes: postprandial fullness (ES = 1.03) (29), postprandial PYY (ES = 0.77) (29), and daily intake (ES = 1.77) (41). Given these ESs, a final sample size of $n = 12$ was determined as adequate to provide 80% power to detect differences in the respective outcomes.

Summary statistics (means ± SEMs) were computed for energy intake, macronutrient intake, sugar intake, TST, sleep efficiency, sleep quality, sleep onset, and cortisol and orexin-A concentrations. Net incremental AUC was computed for hunger, fullness, desire to eat, prospective food consumption, ghrelin, PYY, morning sleepiness,

daytime sleepiness, morning energy, and daytime energy. A repeated-measures ANOVA was then applied to detect main effects of treatment, time, and treatment-by-time interactions for the perceived sensations, hormonal responses, and sleep measures. When main effects were detected, pairwise comparisons using the least-significant-difference test were applied to compare differences between treatments and interactions. A repeated-measures ANOVA was also applied to detect main effects of treatment for the energy content, macronutrient composition, and/or food categories from the ad libitum evening packout and daily intake measures. When main effects were detected, pairwise comparisons using the least-significant-difference test were applied.

With regard to the fMRI neural responses, the prelunch brain activation responses to the food stimuli were preprocessed using procedures described previously (35). Repeated-measures ANOVAs were performed on the brain activation maps within the Brain Voyager software (Brain Innovation BV) with the use of stimulus [food (i.e., appetizing and appealing) compared with nonfood (i.e., animal, nonappetizing but appealing)] and breakfast (Breakfast and Skip) comparisons. To identify significant activations in a priori regions, a cluster-level statistical threshold was applied to correct for multiple comparisons. Significance was set at $P = 0.01$ with a cluster-level false-positive rate of $\alpha = 0.05$.

Pearson correlations were conducted to determine associations between select markers of sleep, appetite and satiety, cortisol, and food intake. Multiple linear regressions were performed to determine associations between sleep onset and caloric intake and percentage of calories from high-fat and high-carbohydrate foods.

In addition, menstrual cycle phase, which has been shown to affect appetite and food intake measures (42), was not controlled for within this study. Pearson correlation analyses were conducted on menstrual phase day and study outcomes to identify potential associations to identify potential study confounders. Because no associations were detected for any outcomes, no subsequent covariate analyses were performed.

Analyses were conducted using SPSS (version 24; SPSS, Inc.). $P < 0.05$ were considered significant. Trends were identified when $P < 0.09$ but >0.05.

Results

Appetite and satiety

The pre- and postprandial appetitive and hormonal appetite and satiety responses across the clinical testing day 7 are depicted in **Supplemental Figures 2** and **3**, and the AUC analyses are shown in **Table 1**. Main effects of time ($P < 0.0001$) and treatment ($P < 0.05$) were observed for all postbreakfast measures. Specifically, hunger, desire to eat, prospective food consumption, and ghrelin decreased, whereas fullness and PYY AUC increased throughout the day after the Breakfast pattern compared with the Skip pattern (all, $P < 0.05$).

fMRI brain activation. Prelunch cortico-limbic neural activation in response to visual food stimuli is shown in **Figure 1**. The consumption of breakfast led to reductions in neural activation in the hippocampus, parahippocampus, and middle frontal gyrus compared with skipping breakfast (all, $P < 0.01$).

TABLE 1 Daily appetite and satiety AUCs for the day 7 testing day in healthy young adults[1]

	Daily AUC, mm × 420 min	
	Breakfast	Skip
Hunger	−12,200 ± 2390*	754 ± 1580
Fullness	14,400 ± 2250*	4056 ± 1458
Desire to eat	−11,500 ± 2490*	−2560 ± 1450
PFC	−7570 ± 2040*	−797 ± 1580
Ghrelin	−30,600 ± 9700*	−9190 ± 5430
PYY	12,500 ± 3990*	−2210 ± 5670

[1]Values are means ± SEMs, $n = 13$. *Different from Skip, $P < 0.05$. Breakfast, consumption of a high-protein breakfast; PFC, prospective food consumption; PYY, peptide YY; Skip, skipping breakfast.

Intake assessments. Amount, energy, and macronutrient contents of the required and ad libitum meals and snacks consumed throughout the day after the breakfast treatments are shown in **Table 2**. Although the energy consumed within the afternoon or evening ad libitum packout was not different between the Breakfast and the Skip treatments, the amount of food consumed was ∼20% less after the Breakfast treatment than after the Skip treatment ($P < 0.05$).

Specifically, the participants consumed ∼30% fewer evening snacks that were high in carbohydrates and fats after the Breakfast treatment compared with the Skip treatment ($P < 0.05$), leading to ∼300 ± 100 fewer calories ($P < 0.05$). In addition, sugar intake throughout the evening period tended to be lower after the Breakfast treatment than after the Skip treatment ($P = 0.085$). The time of last eating occasion within the packout did not differ after the Breakfast (2237, ±98 min) compared with the Skip (2333, ±116 min) treatments nor was the energy load and time of largest eating occasion different between the Breakfast (570 ± 49 kcal and 1932, ±47 min, respectively) and Skip (589 ± 73 kcal and 1958, ±38 min, respectively) treatments.

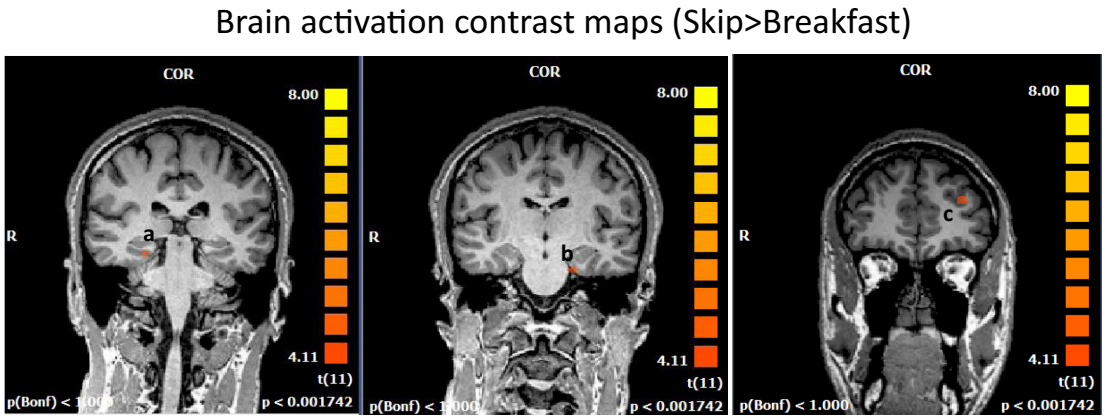
Regardless of the modest differences in evening snacking, no differences in total daily energy, carbohydrate, or fat intakes were detected between treatments (Table 2). However, daily protein intake was greater after the Breakfast treatment than after the Skip treatment ($P < 0.05$).

Sleep-related outcomes

All sleep-related outcomes are shown in **Table 3**. Main effects of treatment were detected for TST (actigraphy). The Breakfast treatment led to shorter TST of 36 ± 11 min compared with the Skip treatment ($P < 0.05$). However, regardless of the differences in TST, sleep efficiency was quite high in the Breakfast and Skip treatments (i.e., 99% ± 0% and 98% ± 0%, respectively), with no differences between breakfast treatments. Perceived sleep quality and sleep-onset time tended to improve after the consumption of breakfast compared with skipping breakfast ($P = 0.060$ and $P = 0.077$, respectively). No main effects of treatment were detected for perceived daytime sleepiness or energy.

Sleep-related hormonal responses

Main effects of treatment were detected for the salivary cortisol response, such that the Breakfast treatment led to higher waking and prebedtime cortisol concentrations than the Skip treatment ($P < 0.05$; **Figure 2**). In addition, although the Breakfast treatment elicited a typical diurnal pattern, the Skip treatment blunted this response. Main



Brain Regions	Talairach Coordinates			t	Cluster extent (voxels)
	X	Y	Z		
Skip > Breakfast					
a priori regions:					
(a) Hippocampus	24	−28	−1	4.65	51
(b) Parahippocampus	−18	−13	−23	5.32	112
(c) Middle Frontal Gyrus	−33	41	25	4.63	191

FIGURE 1 Prelunch fMRI brain activation contrast maps in the coronal view in response to food stimuli during the Breakfast and Skip treatments in healthy young adults. Bonf, Bonferroni; Breakfast, consumption of a high-protein breakfast; COR, coronal; Skip, skipping breakfast.

effects of treatment were not detected for fasting and afternoon plasma orexin-A concentrations (Table 3).

Ingestive behavior and sleep interactions

Pearson's correlational analyses showed a positive association between measured TST and postprandial ghrelin concentrations ($r = 0.4220$, $P < 0.05$), whereas TST was negatively associated with evening food intake (kcal; $r = -0.623$, $P < 0.001$), evening carbohydrate and fat-rich food intake (kcal; $r = -0.460$, $P < 0.05$), and evening sugar intake (kcal; $r = -0.553$, $P = 0.003$). Perceived sleep quality tended to be positively associated with daily PYY responses ($r = 0.375$, $P = 0.078$) and negatively associated with evening food intake (kcal; $r = -0.505$, $P < 0.01$), evening carbohydrate and fat-rich food intake (kcal; $r = -0.488$, $P = 0.01$), and evening sugar intake (kcal; $r = -0.399$, $P < 0.05$). Finally, sleep efficiency was positively associated with daily PYY concentrations ($r = 0.644$, $P = 0.001$) and negatively associated with evening intake (kcal; $r = -0.404$, $P < 0.05$) and evening carbohydrate and fat-rich food intake (kcal; $r = -0.392$, $P < 0.05$).

Discussion

In the current study, consuming a high-protein breakfast consistently improved markers of ingestive behavior and diet quality but led to mixed effects with regard to sleep health in healthy young adults. Specifically, breakfast led to reduced appetite and food reward, increased satiety, and reduced unhealthy snacking behavior compared with skipping breakfast. With regard to sleep health, breakfast consumption led to modest reductions in measured sleep quantity but improved perceived sleep quality and sleep onset. These data suggest that breakfast consumption may be one dietary strategy to promote improvements in appetite, eating behavior, and indexes of perceived sleep quality in healthy young adults.

The question of whether breakfast consumption affects weight management remains controversial due to insufficient causal evidence (26). However, a number of studies have been published exploring the potential mechanisms of action, including, but not limited to, appetitive and hormonal markers of appetite and satiety, neural food cravings, and ingestive behavior (43). The appetitive and hormonal measures of appetite and satiety in the current study are in agreement with our recent systematic review that showed modest support for the consumption of breakfast (43). In addition to these findings, we also showed reduced activation in key cortico-limbic regions of the brain that modulate (food) reward and memory after a high-protein breakfast compared with breakfast skipping, which has been previously confirmed in other breakfast studies (28, 29). Although these data lend support for the consumption of breakfast, there continues to be a large gap in the intervention literature as to whether breakfast consumption affects

TABLE 2 Food intake throughout the day 7 testing day in healthy young adults[1,2]

	Breakfast	Skip
Required foods consumed		
Controlled breakfast		
Energy, kcal	343 ± 0*	0 ± 0
Protein, g	30.1 ± 0*	0 ± 0
Carbohydrate, g	35.5 ± 0*	0 ± 0
Fat, g	9.4 ± 0*	0 ± 0
Sugar, g	20.5 ± 0*	0 ± 0
Controlled lunch		
Energy, kcal	516 ± 0	516 ± 0
Protein, g	24.1 ± 0	24.1 ± 0
Carbohydrate, g	63.7 ± 0	63.7 ± 0
Fat, g	19.7 ± 0	19.7 ± 0
Sugar, g	5.7 ± 0	5.7 ± 0
Ad libitum foods consumed		
Free-living evening intake		
Energy, kcal	1938 ± 263	2252 ± 366
Weight, g	1005 ± 128*	1190 ± 151
Protein, g	62.9 ± 8.3	68.3 ± 10.0
Carbohydrate, g	274.0 ± 40.1	330.0 ± 52.8
Fat, g	68.5 ± 11.0	76.0 ± 14.3
Sugar, g	143.0 ± 22.6†	173.0 ± 26.6
High-carbohydrate and high-fat foods		
kcal	650 ± 123*	938 ± 180
g	389 ± 56*	520 ± 81
Total daily intake		
Energy, kcal	2798 ± 262.9	2768 ± 365.5
Protein, g	117 ± 8.3*	92.4 ± 10.0
Carbohydrate, g	373 ± 36.5	393 ± 52.8
Fat, g	97.6 ± 11.0	95.7 ± 14.3
Sugar, g	169 ± 22.6	179 ± 26.6

[1]Values are means ± SEMs, $n = 13$. *Different from Skip, $P < 0.05$. †Trend for difference compared with Skip, $P = 0.085$. Breakfast, consumption of a high-protein breakfast; Skip, skipping breakfast.
[2]Repeated-measures ANOVA, $P < 0.05$.

TABLE 3 Sleep outcomes from measures conducted across the 7-d testing period in healthy young adults[1,2]

	Breakfast	Skip
Measured		
TST, min	381 ± 13*	417 ± 14
Sleep efficiency, %	99 ± 0	98 ± 0
Orexin-A, pg/mL		
Fasting	0.89 ± 0.07	1.05 ± 0.18
Afternoon	1.31 ± 0.3	0.89 ± 0.06
Perceived		
Sleep quality, mm	73 ± 3†	63 ± 5
Sleep onset, min	12 ± 2†	16 ± 4
Number of awakenings, min	5.2 ± 1.5	6.3 ± 1.5
AUC, mm × 420 min		
Daytime sleepiness	−968 ± 1848	−797 ± 2324
Daytime energy	2570 ± 1909	2022 ± 2111

[1]Values are means ± SEMs, $n = 13$. *Different from Skip, $P < 0.05$. †Trend for difference compared with Skip, $P = 0.060$–0.077. Breakfast, consumption of a high-protein breakfast; Skip, skipping breakfast; TST, total sleep time.
[2]Repeated-measures ANOVA, $P < 0.05$.

potentially more meaningful outcomes, including daily food intake and diet quality.

The majority of published breakfast studies only measure food intake at the next eating occasion (i.e., lunch) in a laboratory-based, controlled setting or use estimates through dietary recalls or food records (26). Assessments of only the subsequent eating occasion fail to capture potential breakfast effects on eating occasions that occur later in the day, particularly during times when snacking and overeating are prominent (44). Furthermore, the use of food-intake estimations have been viewed as unreliable and inaccurate due to human error and the high prevalence of underreporting (45). The current study included an ad libitum evening dinner and snack packout approach to examine energy intake and food choice at dinner and during afternoon and evening snacking occasions. The consumption of a high-protein breakfast did not reduce total daily energy intake compared with breakfast skipping, which is consistent with other acute trials (27, 29, 46). However, our previous 12-wk study showed a reduction of ~400 kcal in daily intake after a high-protein breakfast compared with skipping the morning meal (26). Thus, it is possible that reductions in daily intake may have occurred within the current study if the study duration was extended.

Breakfast is considered a positive modulator of diet quality by increasing the consumption of shortfall nutrients and reducing the consumption of energy-dense foods, which are both factors related to reducing the risk of chronic disease (47, 48). The current study also showed improvements in diet quality as shown by the reduction in evening snacking behavior of carbohydrate- and fat-rich foods, which occurred after the consumption of breakfast compared with breakfast skipping. There was also an increase in total daily protein intake. Thus, although breakfast did not result in decreased daily calories, breakfast consumption may be an important strategy to improve diet quality.

Sleep health is regarded as a key component in the regulation of ingestive behavior; however, the question of whether there is a bidirectional relation such that eating behaviors modulate sleep is garnering increased interest (49). Food intake or omission at the wrong biological time (i.e., during typical wake-sleep times for most individuals) leads to peripheral circadian clock–driven changes in metabolism related to

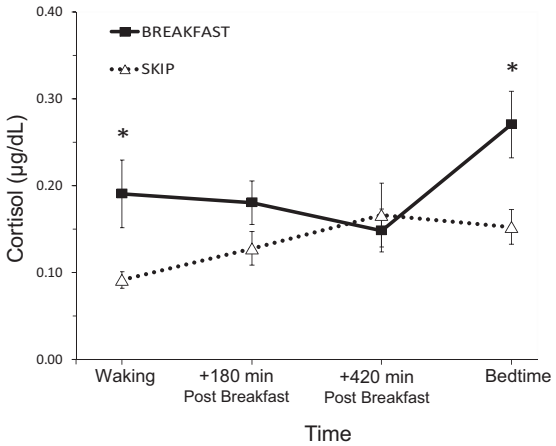


FIGURE 2 Salivary cortisol responses across day 7 in healthy young adults: *Difference between Breakfast and Skip, $P < 0.05$. Error bars denote means ± SEMs. Breakfast, consumption of a high-protein breakfast; Skip, skipping breakfast.

sleep-wake disturbances and circadian misalignment (50). It has been speculated that breakfast is an optimal eating occasion to establish ingestive behavior throughout the day, which, downstream, may affect sleep-wake cycles and overall sleep health.

With regard to the breakfast treatments, measured TST was shorter when breakfast was consumed than when it was skipped, with no differences in sleep efficiency. It is possible that the reduction in TST was not deleterious because sleep efficiency was not different and perceived sleep quality and sleep onset tended to improve when breakfast was consumed. However, improvements in sleep efficiency are often reported as a result of reduced sleep time and increased sleep pressure, typically in populations exhibiting clinically diagnosed sleep disturbances (51). Further investigation examining the effects of reduced sleep time in healthy adults is required. It is also important to note that the reductions in TST measured by actigraphy do not reflect sleep staging. We did not use polysomnography to measure sleep and therefore cannot determine alterations in sleep architecture (52). St-Onge et al. (51) examined whether eating behavior affects sleep and, more specifically, sleep architecture. Measures were taken during a tightly controlled feeding period as well as with ad libitum periods. Although TST did not differ between feeding periods, a curtailment of slow-wave sleep occurred and longer sleep-onset time occurred after the ad libitum period. Similar to our study, increased energy intake from sugar and carbohydrate-rich foods was associated with poor sleep.

As previously stated, the participants in the current study tended to report improved perceived sleep quality and shorter sleep-onset time after breakfast compared with breakfast skipping. These findings indicate potentially promising improvements because perceived sleep quality is an important marker of overall sleep quality and equally valued alongside measured sleep (53). A recent examination compared which determinants, either self-reported sleep or measured sleep, more strongly contribute to overall perceived sleep quality (53). The findings showed that self-reported sleep measures were primary drivers of perceived sleep quality rather than directly measured sleep-quality indexes. In short, perceived sleep-quality measures (i.e., perceived ratings of sleep, mood upon awakening) were better indicators of sleep quality than measured indicators of sleep quality (i.e., measured number of arousals, measured sleep efficiency). Collectively, these findings suggest that the consumption of breakfast may be helpful in improving some meaningful aspects of sleep quality.

As part of this investigation we sought to explore potential associations between ad libitum food intake and indexes of sleep health. A number of sleep outcomes were inversely associated with evening snacking. These findings suggest that evening intakes of carbohydrate- and fat-rich foods as well as sugar may negatively affect indexes of sleep quality.

Last, a number of peripheral or centrally derived neurochemicals, neurotransmitters, and hormones exist that modulate metabolism, ingestive behavior, and sleep-wake cycles. These substances include cortisol and hypocretins (orexin-A) (54). Thus, we were interested in whether breakfast consumption would alter these signals. Diurnal cortisol rhythms are normally characterized by high concentrations at waking with a steady decline throughout the day (55). In the current study, breakfast consumption elicited a typical diurnal cortisol pattern, whereas the pattern was blunted (i.e., the cortisol diurnal decline appeared to be diminished) when breakfast was skipped. We examined the effects of breakfast intake on the neuropeptide orexin-A due to its involvement in sleep, appetite, and feeding behaviors (21, 56). To our knowledge, this is the first nutrition intervention to examine whether plasma orexin-A is modulated by breakfast. However, orexin-A concentrations were not different between treatments, indicating no effect of breakfast or continued morning fasting on orexin-A.

Limitations

The current findings are limited to interpretation because all study participants were healthy, normal-weight individuals. Thus, the impact that breakfast consumption may have on sleep health in overweight, unhealthy individuals requires further study. Moreover, the study population did not report clinically poor sleep according to the Pittsburgh Sleep Quality Index. Perhaps the effect of nutrient timing is too modest to alter sleep behavior detectable by the current methods in healthy individuals without severe sleep impairments. Although this study was powered for the primary outcomes of appetite and satiety, it may have been underpowered for the food-intake and sleep measures. However, sample sizes of $n \leq 12$ are used frequently in sleep studies (9). Future breakfast studies including larger sample sizes over a longer duration are required to determine whether a breakfast intervention is efficacious in inducing more definitive changes in evening food intake or sleep. In addition, the current study did not include assessments of snacking or dinner timing during the ad libitum eating period. Consequently, we were unable to report the time of the latest meal of the day, the specific nutrient compositions of these final foods, or the relations of these factors and sleep. A limiting aspect to consider regarding the daily cortisol response patterns is the collection of the last cortisol measure of the day. Because the participants were asked to complete their last sample before a self-selected bedtime and bedtimes were not held constant, these samples varied by time of day (clock time). Thus, the latter portion of the curve could indicate an abnormal cortisol concentration relative to normal concentration curves if bedtimes are much later in the day than those utilized for standard cortisol response curves. Although circadian measures were not included in this trial, it is necessary to highlight that due to recent discoveries linking sleep perturbations to health status, the interactions between food intake, sleep health, and circadian physiology are next frontiers for investigation (50).

Conclusions

The daily consumption of breakfast improved appetite, satiety, and diet quality and may support improvements in some aspects of sleep health in healthy young adults.

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