

**Examining the acute effects of sleep restriction and timing on energy balance,
satiety efficiency and food reward in adults**

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THESIS ABSTRACT

The main objective of this thesis was to examine the independent effects of sleep duration and timing on appetite, food reward and energy balance. Study 1 investigated the associations between satiety quotient (SQ) with habitual, self-reported sleep duration, quality and timing. No significant associations were noted between SQ and sleep parameters. Short-duration sleepers had a lower mean SQ *vs.* those with ≥ 7 h sleep/night ($P=0.04$). Study 2 evaluated associations between changes in sleep duration, efficiency and timing with changes in next day food reward. Greater sleep duration and earlier wake-times were associated with greater food reward ($P=0.001$). However, these associations were no longer significant after controlling for elapsed time between awakening and completion of the food reward task. Study 3 examined the effects of 50% sleep restriction (SR) anchored during the first (delayed bedtime) or second (advanced wake-time) half of the night on appetite, SQ, food reward, energy intake (EI) and energy expenditure (EE). Greater appetite ratings and explicit high-fat food reward were noted following SR with an advanced wake-time *vs.* control and SR with a delayed bedtime ($P=0.03-0.01$). No difference in SQ was noted between sessions. Energy and carbohydrate intakes were greater on day 2 and over 36h in the delayed bedtime *vs.* control session ($P=0.03$). Activity EE and moderate-intensity physical activity (PA) time were greater following delayed bedtime *vs.* control and advanced wake-time on day 1, whereas vigorous-intensity PA time was greater following advanced wake-time *vs.* delayed bedtime on day 1 ($P=0.01-0.04$). Greater sleep quality and slow-wave sleep duration between SR sessions were associated with lower EI and increased vigorous-intensity PA time, respectively ($P=0.01-0.04$). Collectively, these findings suggest that appetite, SQ and food reward are influenced by sleep parameters, but these changes may not alter EI. These findings also suggest that individuals with greater sleep quality in response to SR had greater vigorous-intensity activity time and lower EI.

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PREFACE

The work presented herein is my own, and I take full responsibility for its content. All thesis manuscripts in Chapter 3 were co-authored by Dr Éric Doucet. Dr Geneviève Forest was a co-author on manuscripts 3 and 4. Data collection for thesis manuscript 1 was completed by a separate research group at the University of Laval; this study lead by Drs Angelo Tremblay and Vicky Drapeau. Dr Jean-Philippe Chaput was also the senior author on this manuscript. Manuscript 1 was published in the *European Journal of Clinical Nutrition*, and manuscript 2 in the *Journal of Sleep Research*. Manuscripts 3 and 4 will be submitted for publication in the *Journal of Sleep Research* and the *American Journal of Clinical Nutrition*, soon after the submission of the thesis. Ethical approval was required for all studies, and certificates of ethical approval from *Le Comité d'éthique de la recherche de l'Université Laval* and the University of Ottawa Health Sciences Research Ethics Board are included in Appendix A. The published versions of manuscripts 1 and 2 can be found in Appendix B and C, respectively.

In addition to the thesis manuscripts, a list of non-thesis published abstracts and peer-reviewed scientific articles during my PhD tenure can be found in Appendix D. Permission for reproduction of manuscripts 1 and 2 in a thesis was not required from the respective Journals as I am an author of this content (see Appendix E). Additionally, the reproduction of Figures 1 and 2 presented in Chapter 2, as originally published by Waterhouse *et al.* ⁽⁵⁵⁾ in the *Journal of Physiological Anthropology* does not require reproduction permission, as this Journal is part of BioMed Central Open Access (see Appendix E).

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LIST OF ABBREVIATIONS

Term	Description	Units
AASM	American Academy of Sleep Medicine	N/A
AUC	Area under the curve	N/A
EE	Energy expenditure	kilocalorie
EEG	Electroencephalography	Hertz
EI	Energy Intake	kilocalorie
EMG	Electromyography	Hertz
EOG	Electrooculography	Hertz
ExEE	Exercise energy expenditure	kilocalorie
HPA	hypothalamic-pituitary-adrenal	N/A
LFPQ	Leeds Food Preference Questionnaire	N/A
MVPA	Moderate-to-vigorous physical activity	Time (minutes)
NREM	Non-rapid eye movement	N/A
PFC	Prospective food consumption	millimeter
PSG	Polysomnography	Hertz
PSQI	Pittsburgh Sleep Quality Index	N/A
RMR	Resting metabolic rate	kilocalorie
REM	Rapid eye movement	N/A
RRV	Relative-reinforcing value	N/A
SCN	Suprachiasmatic nucleus	N/A
SQ	Satiety quotient	millimeter/100 kilocalories
SWS	Slow-wave sleep	N/A
VAS	Visual analogue scale	mm
WASO	Wake after sleep onset	Time (minutes)

LIST OF DEFINITIONS

1) *Ad libitum*: This latin term signifies "at liberty" or "without restraint". In feeding studies, it signifies that a relatively large quantity of food (often 2-3 portions of each item) is available to the participant and they may eat "as much or as little as they want", which does not restrain the quantity and frequency of their food intake.

2) Behavioral Choice Task to assess the relative-reinforcing value of a preferred food: This task is described in more detail in Temple *et al.* ⁽⁴⁷⁾. This task is a measure of food wanting, by assessing the amount of work a participant is willing to do in order to obtain a preferred food item.

3) Circadian rhythm (Process "C"): The circadian rhythms represents an \approx 24-hour clock that is self-sustained by an endogenous circadian oscillator, but it can be entrained to external stimuli such as light, food or activity. Sleep is facilitated when exposure to a preferential environment to sleeping (*e.g.* reduced light exposure, fasting and reduced activity participation) that is centrally driven by an endogenous circadian oscillator occurs.

4) Dopamine: A neurotransmitter in the brain that plays a key role in reward-motivated behaviors. More specifically, dopamine is released following a rewarding experience (*e.g.* ingesting a novel food item) and acts on the forebrain to provide an index of the rewarding value of this experience (*e.g.* the nutritional value of the consumed food item). The release of dopamine then acts as a learning signal that associates this experience (consumption of a food item) with its reward (its nutrients). Once this association has been strengthened over repeated experiences, anticipatory dopamine activity occurs when exposure to external cues linked with the experience of receiving this reward (*e.g.* the sight and smell of the food item often leads to the consumption of this item) takes place. At this time, dopamine activity acts as a motivational drive to receive a reward (*e.g.* seek and consume the food item) in addition to being a learning signal.

5) Epworth sleepiness scale: This questionnaire, created and validated by Dr Murray Johns ⁽⁹³⁾, determines the degree of daytime sleepiness when faced with different situations (*e.g.* sitting and reading, watching television). A score \geq 10 indicates excessive amounts of daytime sleepiness. Participants with scores \geq 10 were excluded from further participation in Study #3.

6) Food liking: Incorporates the sensory properties of a food item and forms hedonic or aversive behavioral reactions to this item. This affective reaction is assessed the moment food is ingested, thus reflecting the acute hedonic impact of consuming this food item.

7) Food reward: The rewarding value associated with consuming a food item, which is based on the taste and nutritional/caloric content of the food (*i.e.* a rewarding value is associated with consuming food to encourage food seeking behavior and avoid starvation).

8) Food wanting: The objective, and sometimes implicit, drive to seek and consume a targeted food item. This component of food reward reflects the changes in the propensity to consume a food item and can be influenced by physiological need and/or desire to eat. However, food wanting may also be modulated by higher cognitive processes (*e.g.* choosing not to consume a certain food due to high cognitive dietary restraint).

9) Homeostatic sleep drive (Process "S"): The homeostatic sleep drive builds up with increasing time spent awake and declines during sleep. Hence, prolonged wakefulness promotes sleep onset.

10) Leeds Food Preference Questionnaire: This task is described in more detail in Finlayson *et al.* ⁽³⁵⁾. This task measures food wanting and liking, by asking the participants to rate 16 randomized visual food cues in turn on a 100-mm visual analogue scale (anchored by Not at all-Extremely), based on the following questions: “how much do you want some of this food now?” and “How pleasant would it be to experience a mouthful of this food now”, respectively. These 16 visual food cues are divided into 4 different categories, based on fat content and taste (high-fat savory, low-fat savory, high-fat sweet and low-fat sweet).

11) Morning-Eveningness Questionnaire: This questionnaire, created and validated by Drs. James Horne and Olov Ostberg (1976) ⁽⁹⁴⁾, provides an indication of an individual's circadian rhythm chronotype (*e.g.* early riser or night owl). Scores below 30 and above 70 indicate extreme morning and evening types; participants with these scores were excluded from participating in Study #3.

12) Non-rapid eye movement sleep: In humans, non-rapid eye movement sleep contains 3 distinct stages of sleep: stage 1 and 2 (termed light sleep), as well as stage 3 (termed slow-wave sleep). During these sleep stages, brain wave activity becomes larger in amplitude and decreases in frequency when compared to a waking state. Contrary to rapid eye movement sleep, there is little to no eye movement during these sleep stages. Many theories suggest that non-rapid eye movement sleep plays a critical role in energy conservation and nervous system/cellular recovery through the removal of deleterious byproducts (*e.g.* reduced oxidative stress) and/or the restoration of essential metabolites (*e.g.* protein synthesis).

13) Partial sleep restriction: Having a sleep duration that is less than what is usually received/needed, without exceeding a total wake time of 24 hours. For instance, only sleeping 4 hours in 1 night when you usually require at least 8 hours of sleep per night in order to not feel drowsy or tired the following day (among other factors/potential consequences).

14) Physical activity time (via accelerometry): The biaxial accelerometer used in Studies #2 and 3 (SenseWear Pro 3 Armbands©, HealthWear Bodymedia, Pittsburgh, PA) provides information on total daily energy expenditure (kcal) and active energy expenditure (kcal; ≥ 3 METs). It also provides information on the amount of sedentary time (minutes; < 3 METs), as well as the amount of time spent performing moderate (minutes; 3-6 METs) and vigorous (minutes; > 6 METs) intensity physical activity.

15) Polysomnography: This measurement offers a comprehensive reading of the biophysiological changes that occur during sleep. This measurement monitors many body functions, such as brain wave activity (via electroencephalogram), muscle activity (via electromyogram) and retina activity and eye movement (via electrooculogram) during sleep. The results from these different measurements can be used to evaluate many sleep parameters, such as the absolute and relative time spent in different sleep stages, total sleep duration, sleep efficiency, sleep latencies and the number and duration of awakenings throughout the night. These different measurements also allow for the identification of certain sleep-related disorders, such as sleep apnea (with added nasal and oral airflow monitoring) and restless leg syndrome.

16) Rapid eye movement sleep: During this sleep stage, high-frequency and low-amplitude activity ($\approx 4-12$ Hertz-alpha and theta waves), which resembles the desynchronized pattern of stage 1 sleep or wakefulness, dominates the EEG tracing. However, contrasting a state of wakefulness, there is increased and desynchronized rapid eye movement, coupled with greatly reduced (or even non-existent) skeletal muscle activity. Many theories suggest that rapid eye movement sleep plays a crucial role in localized recuperative/development processes within the brain, memory consolidation and emotion regulation.

17) Satiety efficiency: The extent to which a food item or meal can reduce subjective appetite sensations per unit of intake (kilocalories).

18) Satiety quotient: A calculation that can be used as a valid marker of the satiety efficiency (Δ fasting and post-meal appetite sensations divided by energy content of the food item or meal consumed).

19) Sleep: A natural recurring state characterized by reduced sensory and brain wave activity, a decrease in voluntary motor activity and a slower response to stimulation/greater arousal threshold. Although the functions of sleep in humans remain unclear, many theories propose that sleep plays a critical role in energy conservation, nervous system/cellular recovery, brain development/recovery and/or memory consolidation.

20) Sleep architecture: Encompasses the different sleep stages which compose a sleep cycle.

21) Sleep duration: The total amount of time spent sleeping over a 24-hour period.

22) Sleep efficiency: Determined as: (total sleep duration/time lying down in bed) X 100.

23) Sleep onset latency: The amount of time required to fall asleep, or reach a certain sleep stage (maintain stage 1 sleep for 10 minutes or any other sleep stage for 20 seconds).

24) Sleep quality: Generally speaking, sleep quality can be measured by the question: "How well did you sleep?". The quality of sleep can be affected by many factors, such as the time spent in slow-wave sleep, sleep duration, sleep efficiency, sleep onset latency, sleep disturbances (*e.g.* loud noises outside your room at night) and the use of sleeping aids or consuming other products before bed (*e.g.* alcohol, caffeine). A poor sleep quality may also lead to daytime dysfunction the following day (*e.g.* feeling drowsy or tired, having lower psychomotor vigilance).

25) Sleep timing: A combination of bedtime and wake-time, independently of sleep duration. The sleep timing midpoint (wake up time - 1/2 total sleep duration) can also be used as an indicator of sleep timing.

26) Stage 1 sleep: During this sleep stage, conscious awareness of the external environment gradually decreases. Brain wave activity slows, with activity ranging from $\approx 4-7$ Hertz (termed theta waves). The disappearance of alpha waves ($\approx 8-12$ Hz) also mark the beginning of this sleep stage.

27) Stage 2 sleep: During this sleep stage, complete loss of conscious awareness occurs. Theta waves predominant the EEG tracing, coupled with "sleep spindles" and "K-complexes". There may also be some delta waves present (1-3 Hertz).

28) Stage 3 (or slow-wave) sleep: Can also be referred to as "deep sleep". During this sleep stage, at least 20% of the tracing illustrates delta waves (1-3 Hertz; low-frequency, high-amplitude activity).

29) Suprachiasmatic nucleus (SCN): The SCN is located in the anterior hypothalamus and acts as the "pacemaker" for a number endocrine (*e.g.* HPA axis activity and cortisol release),

physiological (*e.g.* core temperature, heart rate) and behavioral parameters (*e.g.* activity and eating patterns) that vary according to an \approx 24-hour clock.

30) Total sleep deprivation: Remaining awake for 24+ hours.

31) Wake after sleep onset: The amount of wake-time following sleep onset.

32) Wakefulness: The cortical EEG typically contains desynchronized high-frequency, low-amplitude waves within the 14-30 Hertz range (termed beta waves). This activity presumably reflects cognitive processing, motor and perceptual function activities. When resting with eyes closed (but remaining awake), the EEG activity ranges from \approx 8-12 Hertz (termed alpha waves).

33) Visual analogue scale: A measurement that can be used to quantify subjective characteristics or attitudes, such as appetite (*e.g.* feelings of fullness). When responding to an item on a visual analogue scale, participants are asked to answer the question asked by indicating a vertical line on a continuous horizontal line denoted by 2 extremes (*e.g.* Not full at all-Very full when asked about their feelings of fullness).

CHAPTER 1: INTRODUCTION

The decision to initiate feeding, the amount of food consumed and when to terminate it involve both homeostatic and non-homeostatic factors, such as learned and motivated behaviors, social context, as well as external and internal sensory cues ⁽¹⁾. Feeding is also driven by the willingness to expend effort and work towards obtaining a food item ⁽²⁾. However, in a modern environment where food is plentiful, not only is the energy or amount of work required to obtain food greatly diminished, but the abundance of readily available food and its associative cues (*e.g.* sight, smell) may trigger a desire to initiate feeding, even when satiated ⁽³⁾.

The conditions of modern living have also shaped a society that promotes sleep curtailment in many individuals in order to accomplish different work-related tasks, social and/or family demands ⁽⁴⁾. Certain individuals may also have short sleep duration and/or poor sleep quality due to mental distress (*e.g.* depression, anxiety) ⁽⁵⁾. Sleep clearly serves an important daily function in mammals, as it was conserved by evolution over time, and a lack of sleep leads to severe physical and cognitive discomfort/consequences, followed by a strong "rebound" in sleep recovery ⁽⁶⁾. Simply put, sleep can be described as a natural state of reduced voluntary motor activity, lower response to stimulation/greater arousal threshold, as well as a stereotypical posture allocation of lying down ⁽⁷⁾. The functions of sleep in humans remain unclear, however many theories have been proposed to partially explain the daily need in sleep ⁽⁸⁾. Most theories suggest that non-rapid eye movement (NREM) sleep plays a critical role in energy conservation and nervous system/cellular recovery through the removal of deleterious byproducts (*e.g.* reduced oxidative stress) and/or the restoration of essential metabolites (*e.g.* protein synthesis). Conversely, rapid eye movement (REM) sleep plays a crucial role in localized recuperative/development processes within the brain, memory consolidation and emotional regulation through high brain wave activity ⁽⁸⁾.

Sleep is also traditionally said to be regulated by 2 overlapping processes: the homeostatic process (or process "S") and the circadian rhythm (or process "C")⁽⁹⁾. Briefly, homeostatic sleep drive builds up with increasing time spent awake and declines during sleep⁽¹⁰⁾, whereas the circadian rhythm represents a \approx 24-hour clock that is self-sustained by an endogenous circadian oscillator but can be entrained to external stimuli such as light, food or activity⁽¹¹⁾. Hence, sleep is facilitated following prolonged wakefulness, combined with exposure to a preferential environment to sleeping (*e.g.* reduced light exposure, fasting and reduced activity participation) that is centrally driven by an endogenous circadian oscillator⁽¹²⁾. These processes may affect total sleep time, but they primarily affect sleep architecture⁽¹⁰⁾. More specifically, the homeostatic sleep drive increases the propensity of slow-wave sleep (SWS), the deepest sleep stage when whole-body oxygen consumption is at its lowest⁽¹³⁾, during the first part of the night as the occurrence of this sleep stage is greatly influenced by the length of prior wakefulness⁽¹⁴⁾. On the other hand, REM sleep is mainly influenced by the circadian oscillator and is more common during the second part of the night, a time during which hypothalamic-pituitary-adrenal (HPA) axis activity and cortisol release are greater⁽¹⁵⁾.

A number of experimental studies evaluated the effects of an imposed sleep restriction (\approx 4-6 hours in bed/night *vs.* \approx 8-12 hours in bed/night in a control session) on energy intake (EI)⁽¹⁶⁻²³⁾, components of energy expenditure (EE)^(16-21, 23, 24) and food reward⁽²⁵⁻²⁷⁾. However, these protocols often differ in the timing of the imposed sleep restriction period, even though the degree of sleep restriction is relatively similar (\approx 4-6 hours in bed/night); some studies impose a later bedtime coupled with an earlier wake-time, whereas others induce a later bedtime only. Considering that the homeostatic drive to sleep and the circadian rhythm influence sleep architecture^(10, 28), it is possible that changing the timing of the sleep restriction protocol will alter sleep architecture, which may then affect food reward and components of the energy

balance differently. Rutters *et al.* ⁽²⁹⁾ previously demonstrated that habitually lower amounts of SWS, independently of sleep duration, were associated with a higher wanting for food and greater *ad libitum* EI. Additionally, Shechter *et al.* ⁽³⁰⁾ noted negative associations between the amount of REM and stage 2 sleep with hunger ratings and *ad libitum* EI, respectively, as well as a negative association between the quantity of SWS and REM sleep with fat and carbohydrate intakes, when assessed under experimental sleep restriction and habitual sleep conditions. Although these associations cannot draw cause-and-effect relations, it can be hypothesized that differing sleep architecture may exert an effect on energy balance and food preference.

1.1 Rationale and statement of the problem

Under conditions of sleep restriction, sleep efficiency is said to increase, as the quantity of SWS is expected to be preserved, whereas light sleep (stages 1 and 2 sleep) and REM sleep may be proportionally reduced ^(31, 32). However, REM sleep duration increases during early morning hours, a time when the circadian rhythm promotes greater HPA-axis activity ⁽¹⁵⁾. Hence, altering bed- or wake-time, in addition to reducing sleep duration, is expected to affect sleep architecture. More specifically, sleep restriction protocols ^(31, 32) comparing differences in sleep architecture when anchoring the sleep period during the first or second half of the night reported no differences in SWS between sleep restriction protocols, whereas REM sleep duration was greater during sleep held in the second half of the night. Stage 2 sleep duration was consequently reduced during the second half of the night as a result of maintained SWS and increased REM sleep durations during this time.

Considering the combined effects of altered sleep timing (combined bedtime and wake-time) and sleep restriction on sleep architecture ^(31, 32), as well as the previous associations reported between the time spent in different sleep stages with EI and food reward ^(29, 30), the

effects of imposed sleep restriction coupled with altered sleep timing on measures of food reward, satiety efficiency and the energy balance require further investigation.

The studies outlined below are designed to evaluate (i) whether measures of satiety efficiency and food reward vary in response to self-reported and objectively-measured sleep duration, sleep efficiency and sleep timing assessed under free-living conditions, (ii) whether anchoring a sleep restriction period at the beginning or later in the night affects food reward, satiety efficiency, EI and EE the following day, and (iii) whether changes in sleep architecture related to reductions in sleep duration coupled with alterations in sleep timing are associated with changes in food reward, satiety efficiency, EI and EE.

1.2 Objectives

This thesis aims to answer the following questions:

1) Does the satiety quotient (SQ) in response to a standard meal vary according to habitual, self-reported sleep duration, sleep quality and sleep timing? Do individuals with habitual short sleep duration (< 7 hours of sleep/night), poor sleep quality (score ≥ 5 on the Pittsburgh Sleep Quality Index; PSQI) and a later bedtime (sleep timing midpoint > 2h30) have a lower SQ and, consequently, greater EI?

These research questions will be answered with a cross-sectional design employed in Study 1.

2) Are variations in sleep duration, sleep efficiency and sleep timing related to changes in next day food reward?

This research question will be answered with a randomized, counterbalanced crossover design employed in Study 2.

3) Does a 50% sleep restriction anchored during the first or second half of the night alter appetite sensations, satiety efficiency, food reward and energy balance (EI and EE) differently? Are changes in these outcomes related to changes in sleep architecture between sessions?

These research questions will be answered with a randomized, counterbalanced crossover design employed in Study 3.

1.3 Hypotheses

1) The SQ will be associated with sleep duration, sleep quality and sleep timing. Additionally, individuals with short sleep duration (< 7 hours of sleep/night), poor sleep quality (score ≥ 5 on the PSQI) and a later bedtime (sleep timing midpoint > 2h30) will have a lower SQ and greater EI during an *ad libitum* lunch (**Study 1**).

2) Changes in sleep duration, sleep efficiency and sleep timing will be associated with changes in next day food reward (**Study 2**).

3) A 50% sleep episode anchored during the first half of the night (*i.e.* habitual bedtime and advanced wake-time) will lead to greater EI and lower activity EE and moderate-to-vigorous PA. These changes in EI and EE will be associated with changes in REM sleep between the control and sleep restriction with an advanced wake-time sessions (**Study 3**).

4) A 50% sleep episode anchored during the first half of the night (*i.e.* habitual bedtime and advanced wake-time) will lead to greater appetite sensations and food reward. These changes in appetite sensations and food reward will be associated with changes in REM sleep between the control and sleep restriction with an advanced wake-time sessions (**Study 3**).

1.4 Implications

These studies will allow us to determine whether alterations in sleep timing, independently of sleep duration, may be associated with changes in appetite, satiety efficiency, food reward and energy balance (EI and EE). Furthermore, these studies will help determine whether satiety efficiency and food reward may be relevant factors in explaining potential variations in EI in response to habitual or imposed sleep protocols. Lastly, the correlations between changes in crude sleep parameters (sleep duration, efficiency/quality and timing) and sleep architecture with changes in appetite, satiety efficiency, food reward and energy balance (EI and EE) between sessions will allow us to determine whether variations in sleep parameters are associated with changes in appetite, food reward and energy balance parameters the following days.

1.5 Limitations and delimitations

The findings from Studies #2 and #3 are limited to a relatively small sample size of healthy men and women (*i.e.* 14 and 18 men and women in Studies #2 and #3, respectively), which limits generalizability to other populations; especially individuals with sleep complaints or disorders. The cross-sectional design employed in Study #1 and the correlations drawn in Studies #2 and #3 cannot infer causality. There are inherent limitations with the use of self-reported sleep measurements in Study #1. Similar limitations exist with the use of accelerometry to estimate sleep-wake activities and EE. The assessment of sleep and all outcome variables in Studies #2 and #3 were only conducted for 1 night and \approx 36 h post-intervention in each condition, which does not account for day-to-day variations in these variables, nor can they be compared to studies imposing prolonged sleep restriction protocols. Differences in a homeostatic need for sleep between the sleep restriction protocols in Study #3 (*i.e.* no degree of sleep restriction had occurred when wake-time was advanced *vs.* when bedtime was delayed) may influence the sleep

architecture results. Likewise, meal and appetite measurement times were fixed for each participant across sessions, meaning that the time spent awake was much greater in the sleep restriction with an advanced wake-time protocol.

CHAPTER 2: REVIEW OF THE LITERATURE

This literature review will summarize research evaluating the associations between sleep duration and sleep timing with food reward, satiety efficiency and components of the energy balance (EI and EE). The concepts of food reward, satiety efficiency and sleep regulation will first be discussed, followed by the effects of reduced sleep duration and sleep timing on sleep architecture, appetite, food reward and/or the energy balance. Accordingly, the studies presented in this thesis will investigate the independent effects of sleep duration and sleep timing on satiety efficiency, food reward and energy balance components (EI and EE). Many terms found throughout this document are defined on **Page xii**.

2.1 "Eating to live or living to eat?". The "wanting" and "liking" components of food reward

Affect and motivation, or liking and wanting, can be seen as major forces in directing human feeding behavior⁽³³⁾. More specifically, food "liking" is a process that incorporates the sensory properties of a food item and forms hedonic and aversive behavioral reactions to this item⁽³⁴⁾. Food "liking" is the affective reaction that is assessed the moment food is ingested, thus reflecting the acute hedonic impact of consuming this food item⁽³⁵⁾. On the other hand, food "wanting" can be defined as the objective, and sometimes implicit, drive to seek and consume a targeted food⁽³⁵⁾. The "wanting" component of food seeking behavior reflects the changes in the propensity to consume a food item, independently of liking, and can be influenced by physiological need and/or desire to eat⁽³⁴⁾. However, food "wanting" is not entirely dependent on physiological or caloric needs (termed food "needing") due to its ability to be modulated by higher cognitive processes (*e.g.* seeking and eating food when satiated or choosing not to consume a certain food due to high cognitive dietary restraint). It has been previously hypothesized that food "wanting" may be influenced by the active process of assigning

perceptual value to events, sensory or cognitive inputs (*e.g.* the sight and smell of a preferred food, eating lunch because it is 12h00), which then become cues linked to the consumption of a food through operant conditioning ⁽³³⁾.

Following the ingestion of a novel food item, dopamine is released and acts on the forebrain, which provides an index of the nutritional (or reward) value of the food item, and acts as a learning signal that associates this food item with its nutrients (*i.e.* reward value of the food) ⁽³⁶⁾. Once this association has been strengthened, anticipatory dopamine activity occurs when exposure to external food cues (*e.g.* sight and smell) linked to the consumption of this food item takes place, thus acting as a motivational drive to obtain and consume that food in addition to being a learning signal ⁽²⁾. This notion is supported by studies that noted decreases in EI to a significantly lesser degree during a palatable *vs.* bland test-meal ⁽³⁷⁾, as well as greater desire to eat ratings ⁽³⁸⁾, portion size selection and intake ⁽³⁹⁾ of a palatable food item following the exposure to the sight and smell of this item. These results thus suggest that internal sensory signals (*i.e.* satiety signals) may be overridden when palatable foods are available, or when exposed to previously associated food cues (*e.g.* sight and smell).

2.2 *Is there a clear dissociation between food "wanting" and food "liking"?*

Although it may be difficult to clearly dissociate wanting and liking responses to food (*e.g.* I may choose to consume a food because it has a pleasant taste, or my perception of the pleasant taste may increase because I really want this item), Salamone *et al.* ⁽⁴⁰⁾ demonstrated that low-to-moderate doses of dopamine antagonist administration in rats led to a lower number of lever presses in order to obtain a preferred food item. However, the intake of a less preferred food item (lab chow), which was concurrently available in the chamber, was greater in these rats. Hence, even though the “wanting” (or amount of work willing to put forth to obtain a preferred

food item) was lower in these rats following low-to-moderate doses of dopamine antagonist administration, no differences in total food intake, or preference for the well-liked pellets compared to the lab chow, was noted ⁽⁴⁰⁾. Based on these results, it may be hypothesized that a lower wanting of a food item does not necessarily imply that the taste of this food item will be less pleasurable. These results are further supported by recent studies conducted in humans ^(41, 42). More specifically, a study that assessed the liking and wanting of chocolate following the consumption of individual pieces noted a faster and greater decrease in the wanting for chocolate vs. liking ratings with greater chocolate consumption ⁽⁴¹⁾. The daily consumption of chocolate for 15 consecutive days in a different study ⁽⁴²⁾ led to a decrease in the reported liking for chocolate, whereas *ad libitum* chocolate intake significantly increased over time, which once again suggests that the pleasantness rating (liking) and intake (wanting) are distinct entities. Despite these results, there is some overlap in the wanting and liking components, where the palatability of a food item may directly affect the desire to consume this item ^(43, 44) or its intake ^(37, 39, 45). The repeated intake of a food item may also decrease the hedonic rating of this item ^(46, 47).

Taken together, food wanting and liking seem to play an integral role in directing human feeding behavior, and this especially in a modern environment where food is plentiful and readily accessible. More studies are needed to assess potential changes in food wanting and liking in response to common external stressors, such as sleep restriction, to better comprehend the influence that these factors may have on human feeding behavior.

2.3 The satiety efficiency and its link with energy intake

The satiety efficiency was first proposed as a measure of the effectiveness of a meal at decreasing appetite per unit of EI (*i.e.* slope between EI in kilocalories and the changes in appetite ratings across time following meal consumption) ⁽⁴⁸⁾. The SQ, a derivative of the

changes in subjective appetite sensations in relation to the energy content of a meal ⁽⁴⁹⁾, was later introduced as a valid marker of satiety efficiency in response to a standardized meal ⁽⁵⁰⁾.

Consequently, a lower SQ, or a smaller change between pre- and post-meal appetite scores (*i.e.* desire to eat, fullness, hunger and prospective food consumption ratings (PFC)) in relation to the energy content of the meal consumed, is associated with a lower satiety efficiency ⁽⁴⁹⁾.

Conversely, a higher SQ is associated with greater satiety efficiency in response to a standardized meal. A lower fullness SQ, or smaller changes in subjective fullness ratings in response to a standardized meal, has been associated with higher EI in normal-weight, obese and weight-reduced individuals ^(49, 51). Furthermore, individuals characterized with a low satiety phenotype (*i.e.* individuals with a mean SQ < 8mm/100kcal) had lower cortisol responses to a standardized meal ⁽⁵²⁾, which was previously associated with higher EI and potential weight gain over time ^(53, 54). It is, however, unknown whether the SQ may differ according to different sleep parameters, such as duration, timing and quality. Hence, future studies are needed to assess whether the SQ varies according to these different sleep parameters, and whether it is related to EI and food reward under these conditions.

2.4 "The rewards of shut-eye...or consequences due to lack of". The roles of the homeostatic process and the circadian rhythm in sleep regulation

As briefly mentioned, sleep is said to be regulated by 2 overlapping processes: the homeostatic process (or process "S") and the circadian rhythm (or process "C") ⁽⁹⁾. **Figure 1** illustrates the variations in these 2 processes over an entire sleep-wake cycle; a model that was first introduced by Borbély in 1982 ⁽⁹⁾, and reproduced by Waterhouse *et al.* ⁽⁵⁵⁾.

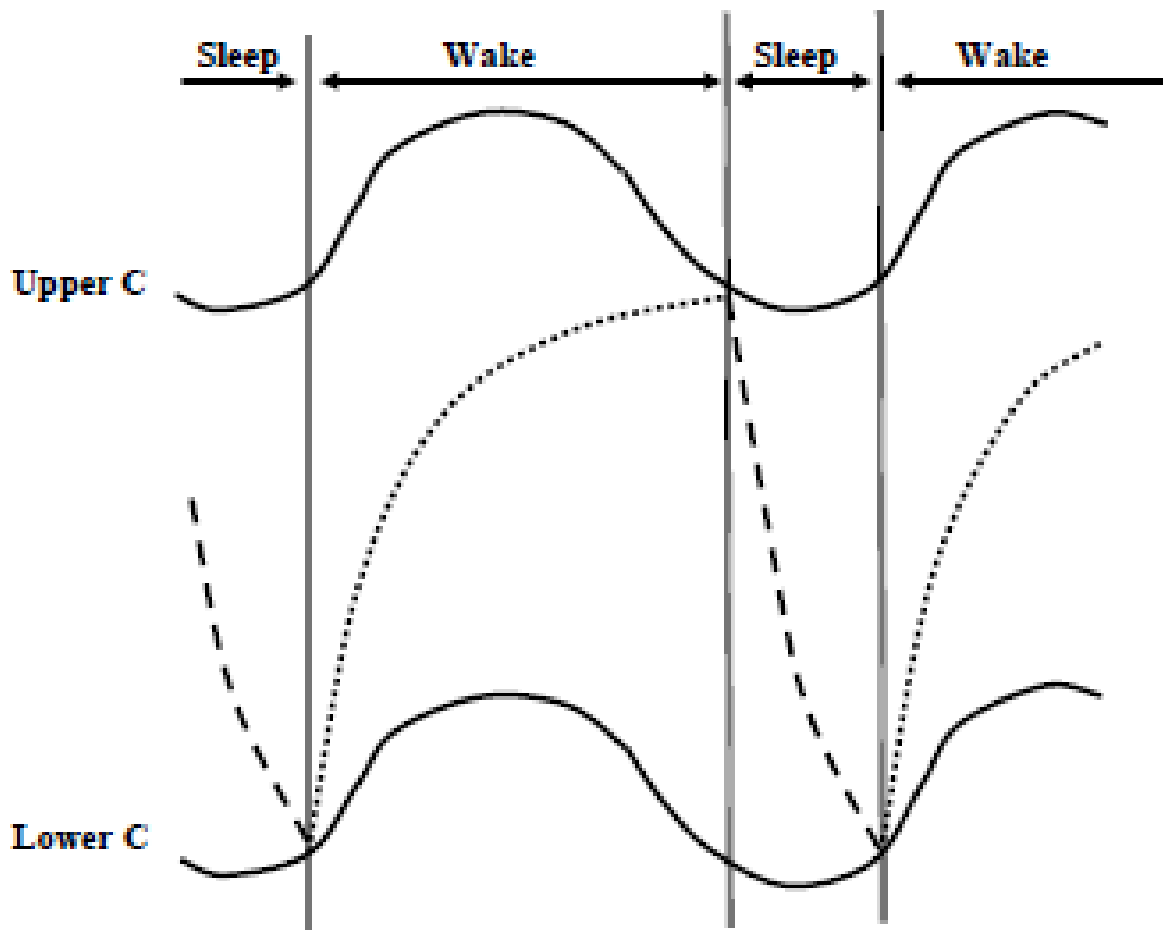


Figure 1. The sleep-wake cycle model initially proposed by Borbély in 1982⁽⁹⁾. This Figure is presented by Waterhouse *et al.*⁽⁵⁵⁾ (BioMed Central Open Access). The homeostatic sleep drive (process "S") is represented by a dotted line. The circadian rhythm (process "C") is represented by 2 components: Upper C drive and Lower C drive.

This model suggests that the sleep drive (process "S") increases exponentially during waking, but then decreases exponentially during sleep; its variations in line with the need for sleep. The circadian rhythm (process "C") varies according to a ≈ 24 -hour period that is mainly controlled by the circadian oscillator (or "pacemaker") located in the suprachiasmatic nucleus (SCN) in the anterior hypothalamus. The SCN orchestrates the ≈ 24 -hour variations of a number of endocrine

(*e.g.* HPA axis activity and cortisol release), physiological (*e.g.* core temperature, heart rate) and behavioral (*e.g.* activity and eating patterns) parameters ⁽⁵⁶⁾. These variations (*e.g.* reduced sympathetic nervous system activity, reduced core temperature and greater melatonin release) create an environment that is said to be conducive to sleep initiation and maintenance ⁽⁵⁵⁾. Waterhouse *et al.* ⁽⁵⁵⁾ suggested that sleep onset occurs when the rising value of the "S" function intercepts with the upper "C" function, which normally occurs during the rapid decline of the upper "C" function but before it reaches its nadir. During sleep, the "S" function decreases until it intercepts with the lower "C" function, at which time waking occurs (**Figure 1**).

Inter- and intra-variations in the sleep-wake cycle do occur ⁽⁵⁵⁾. For instance, it is common for individuals to delay their bedtime over the weekend because of increased social activity participation. Although it is often difficult to determine whether the need for sleep, and the timing of the sleep period are mainly driven by internal or external cues, individuals with a delayed sleep onset and/or need for a smaller amount of sleep will experience an increase in the "S" function that is less rapid (smaller slope), whereas individuals who have a rapidly increasing "S" function may require more sleep and/or have an earlier bedtime (greater slope) ⁽⁵⁵⁾. Even though the circadian rhythm is self-sustained by the SCN, a number of external stimuli (*e.g.* light exposure, physical activity and eating patterns) may influence its variations. These variations are often present in shift workers, who may experience circadian misalignment when their internal circadian rhythm functions according to a ≈ 24 -hour pattern that is different than their behavioral or external pattern (*i.e.* staying awake and eating during a phase of the circadian rhythm that is suited for sleeping and fasting) ⁽¹¹⁾. Although the internal circadian rhythm will promote habitual variations in physiological parameters during the correct circadian phase, a circadian misalignment induced by exposure to external stimuli (*e.g.* exposure to light during the night) will induce a certain degree (or lack of) variation in these physiological parameters (*e.g.* smaller

melatonin release, greater cortisol and glucose release)⁽¹¹⁾. An imposed 28-h environmental cycle, aimed at disrupting the circadian rhythm, coupled with a reduction in sleep duration (5.6 h in bed/night), for 3 weeks led to a reduction in insulin secretion in response to a standard meal, as well as a reduction in resting energy expenditure (REE)⁽⁵⁷⁾. A different study also reported a reduction in total daily EE following 3 days of imposed night-shift work, which corroborates these results⁽⁵⁸⁾. Taken together, these imposed circadian misalignments often cause a certain degree of mental or physical discomfort (*e.g.* gastro-intestinal complaints, reduced cognitive performance) in the short-term, and can also lead to cardio-metabolic complications if sustained over time (*e.g.* insulin resistance)^(11, 59-61).

The interaction between these 2 processes also influence sleep architecture. More specifically, the homeostatic sleep drive (process "S") promotes the occurrence SWS during the first part of the night as the amount of this sleep stage is greatly influenced by the length of prior wakefulness⁽¹⁴⁾. On the other hand, REM sleep is mainly influenced by the circadian rhythm (process "C") and is more common during the second part of the night when core temperature is reduced and HPA axis activity and cortisol release are greater⁽¹⁵⁾. NREM sleep, which includes light sleep (stage 1 and 2 sleep) and SWS, occurs when the cells in the preoptic and basal forebrain regions are maximally stimulated⁽⁸⁾. As for REM sleep, this sleep stage is generated by the activation of neurons located in the pons and midbrain region, which are located in the brainstem⁽⁸⁾. Additionally, the activation of these neurons decreases muscle tone activity within the postural muscles of the body by simultaneously inhibiting the stimulation of motoneurons. NREM sleep is characterized by a decrease in neocortical and brainstem neurons activation⁽⁸⁾ which is more pronounced during SWS, and creates low frequency, high amplitude brain wave activity⁽⁷⁾. Conversely, REM sleep is characterized by high frequency, low amplitude brain wave activity which resembles stage 1 sleep or a waking state⁽⁷⁾, in addition to an inhibition in the

discharge of motoneurons and neurons linked to noradrenaline, hypocretin, epinephrine, serotonin and histamine activity, which cause a loss of consciousness and muscle tone activity⁽⁸⁾.

2.5 Alterations in sleep architecture in response to sleep restriction

Reductions in the absolute time spent in different sleep stages would be expected to occur when decreasing total sleep time. However, deep sleep is minimally affected by partial sleep restriction, with reductions mostly occurring in light (stage 1 and 2) and REM sleep⁽⁶²⁻⁶⁴⁾. These effects are more pronounced during sustained sleep restriction protocols (*i.e.* ≥ 2 consecutive nights of partial sleep restriction)^(62, 65). Although SWS is immediately preserved/compensated in situations of sustained sleep restriction, REM sleep time seems to only increase after $\approx 3-4$ nights of partial sleep restriction, thus suggesting that a delay in REM sleep rebound may be sustained over the short-term⁽⁶²⁾. Furthermore, the SWS and REM sleep latency periods are reduced in response to sustained sleep restriction⁽⁶⁴⁾, which may in part explain the greater sleep efficiency observed in response to this type of sleep restriction protocol^(62, 66, 67).

As briefly mentioned, the quantity of SWS is expected to be preserved when sleep is reduced, whereas light sleep (stages 1 and 2) and REM sleep durations may be proportionally reduced^(31, 32). SWS is mainly driven by an homeostatic need for sleep⁽¹³⁾, whereas REM sleep duration increases with a circadian rhythm which promotes greater HPA-axis activity^(14, 15). Hence, it is expected that SWS be preserved/compensated when sleep debt accumulates, independently of bedtime, whereas REM sleep duration be predominant during the early morning hours⁽¹⁴⁾. Indeed, sleep restriction protocols comparing differences in sleep architecture when anchoring the sleep period during the first or second half of the night reported no differences in SWS between sleep held during the first or second half of the night^(31, 32). The main differences lied in the amount of REM and stage 2 sleep; REM being greater during sleep

held in the second half of the night, whereas sleep held during the first half of the night contained more stage 2 sleep. Furthermore, lower sleep efficiency was reported in individuals randomized to an early wake-time or delayed bedtime protocol (79% vs. 96%)⁽³¹⁾. This difference in sleep efficiency may be explained by the amount of time spent awake prior to sleep, which is greater when bedtime is delayed comparatively to when wake-time is advanced^(62, 68). Taken together, imposing a 50% sleep period during the first or second half of the night is expected to alter REM sleep duration, as SWS duration tends to be preserved/compensated in sleep restriction protocols. However, it is unknown whether these changes in sleep stage durations are related to changes in appetite, food reward and energy balance outcomes the following day.

2.6 The effects of sleep restriction on energy intake and energy expenditure

The energy, macronutrient and snack intakes, as well as EE (*i.e.* REE and activity energy expenditure) measurements following imposed partial sleep restriction protocols in adults are presented in **Table 1**.

Table 1. Variations in energy, macronutrient and snack intake, and energy expenditure following imposed sleep restriction in adults.

Study	Participants	Design	Intervention	Energy intake and Energy expenditure	Macronutrient intake	Snack intake
Bosy-Westphal <i>et al.</i> ⁽¹⁷⁾	14 women	Controlled intervention	2 nights >8h in bed/night (BL), 4 nights of increasing SR (1 night at 7h in bed/night, 2 nights at 6h in bed/night and 1 night at 4h in bed/night) and 2 R nights (>8h in bed/night).	24-hour daily EI was greater during SR vs. BL (10.57±2.12 vs. 8.83±1.07 MJ/day; <i>P</i> <0.05). No difference in EI between SR and R, but EI did return to near BL values during R. No difference in REE and activity EE between BL, SR and R.	No difference in relative macronutrient intake between conditions.	Not reported.
Brondel <i>et al.</i> ⁽¹⁸⁾	12 men	Randomized crossover	4h in bed/night and 8h in bed/night for 48 hours each.	Greater EI during an <i>ad libitum</i> breakfast (703±262 vs. 485±149 kcal; <i>P</i> <0.01) and dinner (982±470 vs. 630±320 kcal; <i>P</i> <0.001) following SR. During SR, activity EE was greater during night 1 and the next day (<i>P</i> <0.01), but lower during night 2 (<i>P</i> <0.05).	Greater relative fat intake during <i>ad libitum</i> dinner following SR (48±13 vs. 37±14%; <i>P</i> <0.001).	Not reported.
Calvin <i>et al.</i> ⁽¹⁶⁾	17 men and women	Controlled intervention	2 groups: <i>ad libitum</i> sleep for 3 nights (acclimatization) followed by 1) 6:00am wake-time and bedtime calculated to give an in-bed time equal to two-thirds of usual sleep time or 2) 6:00am wake-time and <i>ad libitum</i> bedtime. Lastly, 3 nights of sleep recovery (6:00am wake-time and <i>ad libitum</i> bedtime).	24-hour EI is greater during sleep restriction vs. baseline in the experimental group (2382±578 vs. 2942±978 kcal; <i>P</i> <0.01). A non-significant decrease in 24-hour EI occurred in the control group across time (3060±835 vs. 2942±794 kcal; <i>P</i> >0.05); resulting in a net significant difference of 677 kcal/day between groups during the intervention (<i>P</i> <0.05). No differences in total activity accelerations between groups or across time.	Not reported.	Not reported.
Hursel <i>et al.</i> ⁽⁶⁹⁾	15 men	Randomized crossover	Sleep fragmentation (hourly wake-up calls) and non-fragmented sleep for 2 nights each. *Sleep fragmentation led to lower TST, SWS and REM sleep.	Changes in EI were negatively correlated (<i>r</i> = -0.56; <i>P</i> <0.05) with changes in sleep quality during the disturbed sleep session (<i>i.e.</i> greater EI in participants with a greater decrease in their sleep quality).	Not reported.	Not reported.
Markwald <i>et al.</i> ⁽¹⁹⁾	8 men and 8 women	Randomized crossover	3 nights of 9h in bed/night (BL) followed by 5h in bed/night and 9h in bed/night for 5	24-hour daily <i>ad libitum</i> EI was 6% greater (<i>P</i> <0.05) during SR vs. CON. *There was an influence of condition order (<i>i.e.</i> reduced EI when	24-hour daily <i>ad libitum</i> carbohydrate intake was greater during SR vs. CON (394±119 vs. 356±109 g;	No difference in pre-dinner snack intake. Carbohydrate (118±60 vs. 75±44 g; <i>P</i> =0.001),

			days each.	transitioning from 5h to 9h in bed/night). Total EE was 5% greater in SR vs. CON, and 9% greater in SR vs. BL ($P<0.01$).	$P<0.001$).	protein (22 ± 11 vs. 15 ± 10 g; $P<0.001$) and fiber (5.4 ± 1.9 vs. 3.2 ± 1.6 g; $P<0.001$) intakes were greater after dinner during SR vs. CON.
Nedeltcheva <i>et al.</i> ⁽²⁰⁾	5 women and 6 men	Randomized crossover	5.5h in bed/night and 8.5h in bed/night for 14 days each.	No difference in 24-hour EI and EE (including REE and activity EE) between conditions.	No difference between conditions.	Greater snack intake following SR vs. CON (1087 ± 541 vs. 866 ± 365 kcal/day; $P<0.05$), which occurred particularly between 7PM-7AM.
Schmid <i>et al.</i> ⁽²¹⁾	15 men	Randomized crossover	4h in bed/night and 8h in bed/night for 48 hours each.	No difference in 24-hour EI between conditions. Lower activity counts during day 1 post-SR ($43\ 622\pm 4713$ vs. $50\ 190\pm 4554$; $P=0.01$), but no difference on day 2 vs. CON. During SR, greater time spent in low-intensity activity ($57.6\pm 4.5\%$ vs. $52.3\pm 3.5\%$; $P=0.02$) and less time spent in high-intensity activity ($22.6\pm 3.5\%$ vs. $25.4\pm 3.4\%$; $P=0.04$).	Trend (35.7 ± 1.1 vs. $34.0\pm 1.3\%$; $P = 0.05$) towards greater relative fat intake after SR.	No difference in sweet and salty food consumption between conditions.
Shechter <i>et al.</i> ⁽²⁴⁾	10 women	Randomized crossover	4h in bed/night and 8h in bed/night for 3 nights each.	Greater 24-hour total EE during SR vs. CON (1914.0 ± 62.4 vs. 1822.1 ± 43.8 kcal; $P = 0.01$).	Not reported.	Not reported.
Spaeth <i>et al.</i> ⁽²²⁾	225 men and women	Controlled intervention	2 groups: 10h or 12h in bed/night for 2 nights (BL) followed by 1) 5 nights of SR (4h in bed/night or 2) 10h in bed/night for 5 nights. A subset of SR participants underwent 2 nights of recovery sleep (12h in bed/night).	Note: EI was only measured in 31 SR and 6 CON participants. 24-hour daily <i>ad libitum</i> EI was greater during SR vs. CON (130 ± 43 vs. $101\pm 11\%$ of daily caloric requirements; $P=0.003$). Greater 24-hour EI during SR vs. BL ($P<0.05$) and R ($P<0.01$).	Greater intake of all macronutrients during SR vs. BL and R ($P<0.001$ for all). However, during day 4 of SR, there was only greater fat intake following SR vs. BL ($P<0.05$).	+553±266 kcal between 10:00PM-4:00AM during SR. The % of kcal from fat was greater during late-night hours vs. daytime and evening hours ($P<0.05$).
St-Onge <i>et al.</i> ⁽²³⁾	15 men and 15 women	Randomized crossover	4h in bed/night and 9h in bed/night for 5 nights each.	Greater 24-hour <i>ad libitum</i> EI following SR vs. CON (2814 ± 593 vs. 2518 ± 593 ; $P=0.02$). No differences in REE, activity EE and total EE between protocols.	Greater 24-hour <i>ad libitum</i> fat intake following SR vs. CON (112 ± 35 vs. 92 ± 35 ; $P=0.01$)	Not reported.

Note: SR, sleep restriction; BL, baseline; R, recovery; CON, control; TST, total sleep time; SWS, slow-wave sleep; REM, rapid eye movement; EI, energy intake; MJ, Megajoules; kcal, kilocalories; g, grams; REE, resting energy expenditure.

Most studies noted greater 24-h EI following imposed sleep restriction protocols, which was \approx 200-500 kcal higher during the imposed sleep restriction condition *vs.* the control condition (*i.e.* habitual sleep duration) ^(17-19, 22, 23). Furthermore, these increases were often characterized by greater late-night or post-dinner snack intake during the sleep restriction condition ^(19, 20, 22). As expected, no differences in REE were noted between sleep restriction protocols ^(17, 20, 23).

However, overall conflicting results were noted when assessing the effects of sleep restriction on activity EE; some studies reported no differences in activity EE between sleep protocols inside a lab/inpatient clinic ^(16, 20) and under free-living conditions ⁽²³⁾, one study noted greater activity EE following 48 h of sleep restriction (4 h in bed/night) ⁽¹⁸⁾, and another study noted a lower activity EE following 48 h of sleep restriction (4 h in bed/night) ⁽²¹⁾. Similarly, certain studies noted no differences in total EE between sleep restriction protocols ^(17, 20, 23), while others observed greater total EE following sleep restriction (5 h in bed for 5 nights; 4 h in bed for 3 nights), compared to a control session (9 h in bed for 5 nights; 8 h in bed for 3 nights) ^(19, 24). Taken together, many experimental studies reported greater 24-h EI following imposed sleep restriction *vs.* control ^(17-19, 22, 23). Some studies also reported greater total or activity EE post-sleep restriction ^(18, 19, 24), whereas others reported no differences ^(16, 20, 23), or lower EE ⁽²¹⁾. However, these studies often differ in the timing of the imposed sleep restriction period; some imposed a later bedtime coupled with an earlier wake-time, whereas others induced a later bedtime only. Considering that the homeostatic drive to sleep and the circadian rhythm influence sleep architecture ^(10, 28), utilizing sleep restriction protocols with different bed or wake-times will affect sleep architecture. Studies are thus needed to assess the effects of altered sleep timing, independently of sleep duration, on measures of EI and EE.

2.7 The effects of sleep restriction on appetite and food reward

Some recent findings suggest that appetite and food reward could be a relevant factor in explaining these variations in EI and food preference following imposed partial sleep restriction (18, 25-27, 70). Spiegel *et al.* (70) were among the first to demonstrate greater feelings of hunger and appetite ratings for calorie-dense, high-carbohydrate foods following 2 days of partial sleep restriction. Some recent findings corroborate these results (18). However, others reported either no differences in appetite ratings and EI following sleep restriction *vs.* control (21), or no changes in feelings of hunger and appetite sensations despite significantly greater EI post-sleep restriction (17, 23). In regards to food reward, a functional MRI study noted greater orbitofrontal cortex activation in response to visual food cues following partial sleep restriction (4 h in bed/night) *vs.* habitual sleep duration (9 h in bed/night) (25). An enhanced activation in reward and food-sensitive centers of the brain in response to unhealthy *vs.* healthy food cues was also noted following the sleep restriction protocol in these same participants (26). A different functional MRI study corroborates these results by demonstrating greater right anterior cingulate cortex activation in response to visual food cues following one night of total sleep deprivation (27). These participants also rated high calorie foods as being 24% more appetizing following sleep deprivation. Finally, participants made riskier selections, despite the long-term losses that resulted, when faced with a gambling scenario following 48 h of sleep deprivation (71). These results suggest that total sleep deprivation may lead to a decrease in impulse control and delayed gratification. Taken together, these results suggest that individuals who are subjected to sleep restriction have an enhanced susceptibility to food cues. Despite these results, no cause-and-effect inferences can be made as far as the relationship between functional MRI results and EI is

concerned, thus implying that studies are needed to assess the potential changes in food reward (*i.e.* food wanting and liking) in response to sleep restriction.

2.8 The effects of sleep timing on food preference, energy intake and energy expenditure

Recent studies have suggested that sleep timing (*i.e.* combination of bedtime and wake-time) may also have a marked impact on EI and macronutrient preference^(22, 72). For instance, a study in adults⁽⁷²⁾ noted that participants with a later sleep-wake timing (sleep timing midpoint \geq 5h30) consumed more kilocalories after 20h00, ate less fruits and vegetables and more fast food items, compared to those with a sleep timing midpoint $<$ 5h30. Furthermore, a study⁽²²⁾ indicated that participants who were subjected to a later bedtime combined with a sleep restriction protocol (*i.e.* 4 h in bed/night from 4h00-8h00) had greater 24h EI and late-night (22h00-3h00) energy and fat intakes, compared to participants who were part of a control condition of 10 h in bed/night. As for EE, Shechter & St-Onge⁽⁷³⁾ recently reported that a later bedtime, wake-time and sleep midpoint were all associated with more sedentary time and less light and moderate-to-vigorous physical activity time when measured with accelerometry for 7-18 days. Opposite results were noted in individuals with earlier bedtimes, wake-times and sleep midpoints. These results were independent of sleep duration; however sleep duration was negatively associated with sedentary time and moderate-to-vigorous physical activity time in the regression model, which included wake-time. Although studies suggest that a later bedtime, wake-time and/or sleep midpoint may lead to greater EI and preferences for energy-dense foods, in addition to reduced physical activity participation, the factors related to sleep timing that may in part explain these variations in energy balance parameters are unknown.

As previously discussed, altering bed- or wake-time within a sleep restriction protocol may impact sleep architecture; more specifically, the amount of stage 2 and REM sleep^(31, 32). Although the effects of these alterations in sleep architecture on EI and EE has yet to be assessed, Rutters *et al.*⁽²⁹⁾ reported that participants with habitually lower amounts of SWS, independently of sleep duration, had a higher wanting for food and reported feeling hungrier and less full the following day, which led to greater *ad libitum* EI at this time. Additionally, St-Onge *et al.*⁽²⁶⁾ reported that individuals with smaller reductions in REM sleep in response to partial sleep restriction tend to have smaller differences in insula activation between partial sleep restriction and habitual sleep duration conditions. Shechter *et al.*⁽³⁰⁾ also noted negative associations between the amount of REM and stage 2 sleep with hunger ratings and *ad libitum* EI between habitual and partial sleep restriction conditions. This study also reported negative associations between the quantity of SWS and REM with fat and carbohydrate intakes. Taken together, these studies suggest that sleep architecture may affect appetite and EI the following day. However, it is unknown whether imposed alterations in sleep timing, in addition to reduced sleep duration, has an effect on EI and EE the following day. Although the present thesis aims to assess the effects of altered sleep architecture induced by a reduction in sleep duration coupled with altered sleep timing on energy balance parameters, it is important to keep in mind that this type of study protocol does not provide evidence as to the effects of habitual differences in sleep timing and sleep architecture on energy balance parameters.

2.9 Literature review summary and conclusions

In summary, many experimental studies are consistent in suggesting that imposed sleep restriction leads to an increase in 24-h EI^(17-19, 22, 23). However, the results on EE^(16, 18-21, 23, 24) are conflicting, and may require further investigation. Furthermore, recent findings suggest that appetite sensations, as well as food reward assessed with functional MRI, could be relevant factors in explaining increased EI following imposed partial sleep restriction⁽²⁵⁻²⁷⁾. Although the effects of reduced sleep duration on EI and EE have been extensively investigated, the effects of altered sleep timing, independently of sleep duration, on measures of the energy balance require further investigation. Studies have reported that a delayed bedtime, or sleep midpoint, is associated with greater late-night EI⁽⁷²⁾ or reduced EE⁽⁷³⁾ under free-living conditions, which may be associated with habitual differences in chronotype and sleep architecture. However, it is unknown whether imposed alterations in sleep timing, combined with the same degree of sleep restriction, may affect appetite, satiety efficiency, food reward and components of the energy balance (EI and EE). Furthermore, it is unknown whether the changes in sleep architecture associated with changing the bed- or wake-time within a sleep restriction protocol are associated with potential changes in these same outcome variables. Therefore, the present thesis sought to investigate the independent effects of sleep duration and sleep timing on measures of appetite, satiety efficiency, food reward and/or components of energy balance by conducting 3 different studies utilizing different methods and procedures.

CHAPTER 3: METHODS AND RESULTS

3.1 Thesis article #1

Mean Satiety Quotient is Lower in Overweight/Obese Men with a Shorter Sleep Duration

This article was accepted for publication as a short communication on September 12th 2013 by the *European Journal of Clinical Nutrition*, and has been formatted accordingly.

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Abstract

We examined satiety quotient (SQ) and energy intake (EI) according to sleep duration, quality and timing. Seventy-five overweight/obese men (age: 41.1 ± 5.8 years; BMI: 33.6 ± 2.9 kg/m²) completed visual analogue scales for appetite sensations before, immediately after, and every 10 minutes for 1h following a standardized breakfast. Mean SQ (primary outcome of study) was calculated from 4 appetite sensations. The Pittsburgh Sleep Quality Index identified short-duration (<7 hours/night) and "recommended sleep duration" (≥ 7 hours/night) sleepers, poor (score ≥ 5) and good (score <5) quality sleepers, late (midpoint of sleep >2:30AM) and early (midpoint of sleep $\leq 2:30$ AM) sleepers. A 3-day food record and buffet-style meal assessed EI. No associations were noted between sleep parameters and SQ variables. Short sleepers had a lower mean SQ compared to adequate sleepers (6.5 ± 4.9 vs. 8.8 ± 4.3 mm/100 kcal; $P=0.04$). Mean SQ between poor and good (6.9 ± 4.6 vs. 8.7 ± 4.6 mm/100 kcal; $P=0.11$), and between early and late (8.99 ± 5.10 vs. 9.32 ± 4.02 mm/100 kcal; $P=0.78$) sleepers were not significantly different. EI did not differ between sleep groups. Thus, short sleepers had a lower mean SQ compared to adequate sleepers. However, this did not coincide with greater EI.

Keywords: satiety quotient, sleep duration, sleep quality, sleep timing, energy intake

Introduction

Current evidence associates short sleep duration with the development of obesity⁽¹⁾. The satiety quotient (SQ), expressed according to energy intake (EI), determines the extent to which a meal can reduce subjective appetite sensations⁽²⁾. A lower fullness SQ, or smaller changes in subjective fullness ratings in response to a meal, were associated with higher EI in obese individuals⁽²⁾. It is, however, unknown whether changes in SQ may differ according to sleep parameters.

The present study evaluated the SQ in response to a standardized meal in overweight/obese men according to sleep duration, sleep quality and sleep timing. The mean SQ, based on responses to 4 different appetite sensations, was the main outcome of this study. We hypothesized that short sleep duration, poor sleep quality and later bedtimes would be associated with a lower mean SQ, and greater EI.

Methods

Participants

Seventy-five overweight/obese, healthy Caucasian men completed an in-laboratory assessment at Laval University (Quebec, Canada). The inclusion criteria were: aged 30-50 years, body mass index between 28-40 kg/m², non-smokers, not taking medications which could influence appetite, non-diabetic with no insulin treatment, weight stable (± 4 kg within the past 2 months), <3x 30 minutes/week of physical activity, and low dietary restraint (score <10 on the Three Factor Eating Questionnaire). This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures were approved by the Laval University ethics committee. Participants provided written informed consent.

Procedure and measurements

Participants arrived at the laboratory at 8:00AM following a 12-h overnight fast. They were instructed not to consume alcohol or engage in structured physical activity (*e.g.*, playing sports) for at least 24h prior to testing, and to follow habitual sleeping habits the previous night. Upon arrival, height, weight and waist circumference were measured according to standardized procedures⁽³⁾ and fat mass was measured by dual-energy X-ray absorptiometry (GE Medical Systems Lunar, Diegem, Belgium).

A standardized breakfast and *ad libitum* lunch were served at 8:30AM and 12PM, respectively. The composition of these meals are described in more detail in **Appendix 1**. The breakfast had a food quotient (global indicator of meal macronutrient composition) of 0.85, and was entirely consumed within 20 minutes. The participants' appetite sensations were recorded using visual analogue scales (VAS) before, immediately after, and every 10 minutes for 1h following breakfast consumption. The 150-mm VAS were used to answer 4 questions that

quantify subjective appetite sensations: desire to eat, hunger, fullness, and prospective food consumption. The SQ was calculated for each appetite sensation using the following equation⁽²⁾:

$$\text{SQ (mm/100kcal)} = \frac{[\text{fasting appetite sensation (mm)} - \text{mean post meal appetite sensation (mm)}]}{\text{energy content of the test meal (kcal)}} \times 100$$

It is important to note that the SQ calculation for fullness is reversed (mean post-meal rating - fasting rating). The mean SQ represents the mean value of the 4 individual SQ scores. This was selected as the primary outcome of the study since it provides a composite indication of the changes in appetite sensations in response to the meal. A lower SQ indicates a weaker satiety response to a meal⁽³⁾.

The Pittsburgh Sleep Quality Index⁽⁴⁾ determined sleep duration (self-reported item), sleep quality (total score) and sleep timing (midpoint of sleep based on reported wake time and sleep duration) over the last month. The calculations for sleep timing are described elsewhere⁽⁵⁾.

Three-day food records and physical activity diaries, including 2 weekdays and 1 weekend day, assessed habitual EI and moderate-to-vigorous physical activity participation, respectively, following the in-laboratory assessment.

Statistical analyses

Independent *t*-tests compared variables between sleep duration/quality/timing groups. Statistical significance was set at $P < 0.05$. Statistical analyses were performed using JMP (version 10; SAS Institute, Cary, NC).

Results

Table 1 presents participants' characteristics according to sleep groups. There were no differences in these variables between groups, except for 3-day carbohydrate intake between sleep quality groups ($P = 0.03$). There were no significant associations between SQ scores and all sleep parameters, nor were there differences in specific SQ for desire to eat, hunger, fullness or prospective food consumption between groups (data not shown). Short-duration sleepers had a lower mean SQ compared to sleepers with recommended sleep durations, while no significant differences in mean SQ between sleep quality and sleep timing groups were noted (**Figure 1**).

Discussion

To our knowledge, this is the first study to examine measures of SQ according to sleep duration, sleep quality and sleep timing in overweight/obese men. There were no significant associations between sleep parameters and SQ variables. Short sleep duration was associated with a weaker mean SQ, despite no significant differences in body weight, fat mass and EI between sleep duration groups. There were no differences in mean SQ between sleep quality and sleep timing groups, despite a greater 3-day carbohydrate intake in good vs. poor sleepers. The SQ is a more valid indicator of potential changes in subjective appetite ratings in response to a standardized meal compared to 1h post-prandial area under the curve calculations because it considers pre-meal appetite sensations and meal caloric content⁽³⁾.

The greater mean SQ in short sleepers did not coincide with greater EI in this study. These results suggest that appetite ratings may not be consistently related to measured or reported EI⁽⁶⁾. Furthermore, despite noting greater EI following imposed sleep restrictions^(7, 8), one study saw no differences in appetite ratings between sleep conditions⁽⁷⁾, while another only noted greater pre-prandial hunger ratings following sleep restriction⁽⁸⁾. Taken together, changes in appetite ratings, or SQ, may not be consistently related to changes in EI, and *vice versa*.

Studies have also shown that a later sleep timing may lead to greater EI after 8PM⁽⁹⁾ and total EI⁽⁵⁾ in adults and obese children/adolescents, respectively. Conversely, the current study did not observe a significant difference in mean SQ and EI between sleep timing groups. This lack of association may be due to differences in participant characteristics and calculated sleep midpoint between this study and others^(5, 9).

Lastly, reductions in stage 2, REM and slow-wave sleep were associated with greater hunger ratings and EI⁽¹⁰⁾, while the occurrence of sleep fragmentation led to lower fullness and

greater desire to eat ratings compared to a non-fragmented sleep condition⁽¹¹⁾. These results suggest that alterations in specific sleep stages following imposed sleep fragmentation, rather than self-reported habitual sleep quality, may alter appetite ratings.

The present findings are limited to a small sample size of overweight/obese men, which limits generalizability to other populations. The cross-sectional design employed does not allow for causal relationships. Lastly, the use of self-reported measurements and the possibility of residual confounding factors cannot be overlooked (*e.g.* we are unable to determine whether differences in sleep timing are related to biological predispositions or social circumstances).

Although exploratory, we observed a lower mean SQ in short sleepers. The mean SQ between sleep quality and sleep timing groups was not statistically different. Lastly, no difference in EI was noted between sleep groups. Future studies are needed to confirm these preliminary findings.

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Conflicts of Interest

The authors have no conflicts of interest to disclose.

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Table 1. Characteristics of participants according to sleep duration, sleep quality and sleep timing groups.

	Sleep duration		Sleep quality		Sleep timing	
	Short-duration sleepers (< 7 h/night) (n=34)	Sleepers with recommended sleep durations (\geq 7 h/night) (n=41)	Poor sleepers (PSQI score \geq 5) (n=33)	Good sleepers (PSQI score < 5) (n=42)	Late sleepers (midpoint of sleep > 2:30 AM) (n=37)	Early sleepers (midpoint of sleep \leq 2:30 AM) (n=38)
Mean \pm SD	5.7 \pm 0.9 h/night	7.5 \pm 0.5 h/night	PSQI score 6.6 \pm 1.9	PSQI score 2.9 \pm 1.2	3:12AM \pm 36 minutes	2:00AM \pm 30 minutes
Range	3-6.75 h/night	7-9 h/night	PSQI score 5-11	PSQI score 1-4		
Age (years)	41.6 \pm 6.6	40.4 \pm 4.6	41.0 \pm 6.4	40.9 \pm 5	39.3 \pm 5.7	41.8 \pm 5.0
Height (cm)	174.0 \pm 5.7	174.6 \pm 7.0	174.2 \pm 5.3	174.3 \pm 7.3	175.3 \pm 7.2	173.0 \pm 5.6
Body weight (kg)	101.4 \pm 9.5	103.2 \pm 12.6	101.5 \pm 9.5	103.1 \pm 12.9	101.3 \pm 11.2	104.0 \pm 11.9
BMI (kg/m ²)	33.5 \pm 2.9	33.8 \pm 3.0	33.4 \pm 2.9	33.9 \pm 3.1	33.8 \pm 2.9	33.8 \pm 3.2
Fat mass (kg)	34.2 \pm 6.4	35.2 \pm 7.6	34.6 \pm 6.2	34.8 \pm 8.1	34.7 \pm 7.9	34.2 \pm 6.9
Fat mass (%)	33.8 \pm 4.8	34.2 \pm 4.7	34.2 \pm 4.7	33.8 \pm 4.9	33.4 \pm 5.1	33.8 \pm 4.4
Waist circumference (cm)	109.9 \pm 6.9	112.5 \pm 8.6	110.9 \pm 6.8	111.5 \pm 9.0	109.6 \pm 7.1	112.2 \pm 8.8
Lunch EI (kJ)	4987 \pm 1501	5323 \pm 1710	4984 \pm 1360	5411 \pm 1808	4891 \pm 1629	5580 \pm 1638
Carb intake (kJ)	2146 \pm 649	2265 \pm 766	2127 \pm 589	2304 \pm 811	2115 \pm 631	2344 \pm 841
Fat intake (kJ)	2018 \pm 831	2153 \pm 907	1999 \pm 732	2214 \pm 980	1938 \pm 935	2290 \pm 812
Protein intake (kJ)	824 \pm 297	905 \pm 276	858 \pm 288	893 \pm 282	839 \pm 309	947 \pm 241
EI (kJ/day)	11816 \pm 2335	11715 \pm 2464	11259 \pm 2251	12029 \pm 2439	11919 \pm 2653	11610 \pm 2168
Carb intake (kJ/day)	5206 \pm 1368	5365 \pm 1212	4887 \pm 1357	5588 \pm 1146*	5397 \pm 1491	5255 \pm 1104
Fat intake (kJ/day)	4238 \pm 1136	3986 \pm 1084	3938 \pm 989	4131 \pm 1129	4179 \pm 1187	3969 \pm 962
Protein intake (kJ/day)	2000 \pm 425	1924 \pm 445	1954 \pm 390	1945 \pm 472	1972 \pm 403	1936 \pm 465
Alcohol intake (kJ/day)						
MVPA (min/day)	385 \pm 469	530 \pm 826	516 \pm 525	425 \pm 802	418 \pm 522	497 \pm 818
	12.6 \pm 22.7	22.2 \pm 69.8	12.2 \pm 23.8	23.6 \pm 71.8	13.3 \pm 21.3	23.1 \pm 76.5

Note: SD, standard deviation; BMI, body mass index; EI, energy intake; MVPA, moderate-to-vigorous physical activity; PSQI, Pittsburgh Sleep Quality Index

All variables were not significantly different between groups with the use of independent t-tests.

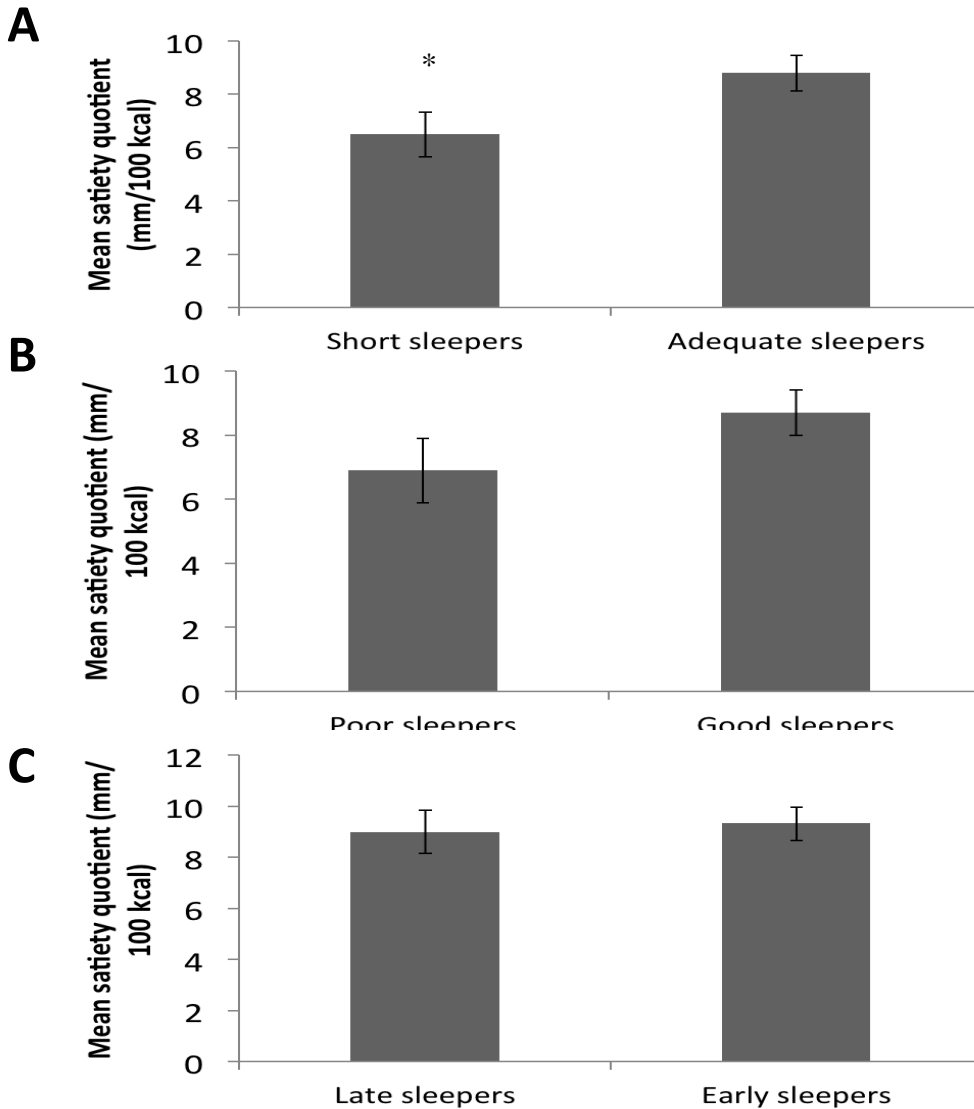


Figure 1. Mean satiety quotient between short-duration sleepers (< 7 hours of sleep/night) and sleepers with a recommended sleep duration (≥ 7 hours of sleep/night) (A), between poor sleepers (PSQI score < 5) and good sleepers (PSQI score ≥ 5) (B), and between late sleepers (midpoint of sleep > 2:30AM) and early sleepers (midpoint of sleep $\leq 2:30$ AM) (C). Values are presented as means for 75 participants with standard errors of the mean represented by vertical bars. * $P = 0.04$ when compared to adequate sleepers. $P = 0.11$ between poor and good sleepers. $P = 0.78$ between early and late sleepers.

Note: PSQI, Pittsburgh Sleep Quality Index

Appendix 1

Composition of the standardized breakfast and *ad libitum* lunch.

	Quantity provided (grams)	Energy content (kJ)
<i>Standardized breakfast</i>		
White bread	100	1092
Butter	12	371
Peanut butter	16	429
Cheddar cheese	40	688
Orange juice	250	486
Total	418	3066
	Initial quantity provided (grams)	Energy content (kJ/kg)
<i>Ad libitum lunch</i>		
Sliced turkey	130	3930
Sliced ham	150	5480
Salmon mousse	90	10833
Liver pâté	70	13350
Gruyere cheese (28% fat)	100	17286
Mozzarella cheese (17% fat)	100	11718
Cottage cheese (2% fat)	100	3384
White bread	150	11300
Whole-wheat bread	150	10170
Soda crackers	100	18400
Butter	40	29990
Mayonnaise	60	30630
Ketchup	40	4350
Italian dressing	60	26110
Mustard	30	3140
Lettuce	60	670
Tomatoes	100	880
Carrots	150	1800
Butter biscuits	70	20840
Chocolate fudge cookies	100	19700
Strawberry yoghurt (1.5% fat)	250	4050
Regular crisps	60	23214
Apples	100	2470
Oranges	100	1970
Milk (2% fat)	1000	2095
Orange juice	1000	1826
Coca-cola	355	1720
7-up	355	1674
Water	1000	0

3.2 Thesis article #2

Associations between sleep parameters and food reward

This article was accepted for publication as a short paper on December 4th 2014 by the *Journal of Sleep Research*, and has been formatted accordingly.

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Conflict of interest disclosure: The authors of this paper declare no conflict of interest.

Contributions from each author: JM, SC and ÉD formulated the research questions, designed the study and carried out the experiment. JM, GF and ÉD analyzed the data. All authors were involved in writing the paper and had final approval of the submitted and published version.

Summary

We examined the effects of acute, isocaloric aerobic and resistance exercise on different sleep parameters, and whether changes in these sleep parameters between sessions were related to next morning food reward. Fourteen men and women (age: 21.9 ± 2.7 years; BMI: 22.7 ± 1.9 kg/m²) participated in 3 randomized crossover sessions: aerobic exercise, resistance exercise and sedentary control. Target exercise energy expenditure was clamped at 4 kilocalories/kilogram of body weight, and performed at 70% of VO_{2peak} or 70% of 1 repetition-maximal. Sleep was measured (accelerometry) for 22 hours following each session. The "wanting" for visual food cues (validated computer task) was assessed the next morning. There were no differences in sleep parameters and food "wanting" between conditions. Lower sleep duration and earlier wake-times were significantly associated with greater food "wanting" between sessions ($\rho = -0.78$ and 0.77 ; $P=0.001$). However, these associations were no longer significant after controlling for elapsed time between wake-time and the food reward task. These findings suggest that shorter sleep durations and earlier wake-times are associated with greater food reward, but these associations are driven by elapsed time between awakening and completion of the food reward task.

Keywords: food reward, exercise modality, sleep patterns

Introduction

Acute bouts of exercise may have beneficial effects on sleep by increasing sleep duration and slow-wave sleep (Bunnell *et al.*, 1983; Youngstedt *et al.*, 1997). However, some studies noted no significant differences in self-reported (Porter & Horne, 1981), and objectively-measured (King *et al.*, 2008) sleep duration and sleep quality between exercise and non-exercise days. Passos *et al.* (2010) reported greater sleep duration and sleep efficiency following an acute bout of moderate-intensity aerobic exercise, but not following high-intensity aerobic and moderate-intensity resistance exercise, in insomniac patients. These results are however limited by a lack of control over exercise energy expenditure (ExEE), which is lower during resistance *vs.* aerobic exercises per unit of time (Donnelly *et al.*, 2004). Hence, the investigation of sleep following acute, isocaloric aerobic and resistance exercise is warranted.

Studies also reported greater neuronal responsiveness to food *vs.* non-food stimuli following imposed sleep restrictions (St-Onge *et al.*, 2012; Benedict *et al.*, 2012). It is however unknown whether habitual changes in sleep parameters under free-living conditions are associated with changes in food reward.

The objective of the current study was twofold. First, we examined the effects of an acute bout of isocaloric aerobic and resistance exercise on different sleep parameters (sleep duration, sleep efficiency, sleep efficiency after sleep onset, sleep onset latency, bedtime and wake-time). Secondly, we investigated whether changes in these sleep parameters between sessions were related to next day food "wanting" through secondary analyses.

Methods

Seven men and 7 women (age: 21.9 ± 2.7 years; BMI: 22.7 ± 1.9 kg/m²; body fat percentage: $21.0 \pm 7.9\%$; $VO_{2\text{peak}}$: 52.6 ± 9.0 ml/kg/min) completed all required measurements. They were between 18-45 years, non-smokers, weight stable (± 4 kg) within the last 6 months, did not have heart problems or diabetes, and participated in <150 minutes of physical activity/week. Two participants had ratings ≥ 10 on the Pittsburgh Sleep Quality Index (PSQI), which classifies them as being poor sleepers according to this questionnaire. Only non-pregnant, premenopausal women were recruited. This study was conducted according to the guidelines laid down in the Declaration of Helsinki. The University of Ottawa ethics committee approved all procedures involving human participants. Written informed consent was obtained from all participants.

Participants took part in 3 randomized crossover sessions: aerobic exercise, resistance exercise and sedentary control. Exercise energy expenditure (ExEE) was matched at 4 kilocalories/kilogram of body weight. The mean ExEE during the aerobic and resistance exercise sessions were 274.5 ± 50.6 and 270.4 ± 56.3 kilocalories, respectively ($P=NS$; Cadieux *et al.*, 2014). A washout period of at least 7 days separated each session for men and at least 1 month for women because they were always tested between days 1 and 8 of the menstrual cycle, as cortisol responses to sleep restriction (LeRoux *et al.*, 2014) and brain activation to food cues (Alonso-Alonso *et al.*, 2011) have both been shown to vary across the menstrual cycle. For each session, participants arrived at the laboratory at 8:00A.M. following a 12-hour overnight fast. They were instructed not to consume alcohol or engage in structured physical activity (*e.g.* training and playing sports) for at least 24 hours prior to each session, and during the data collection period. Participants were also instructed not to consume caffeine during the data

collection period. Upon arrival, participants were weighed to the nearest 0.1 kg with a BWB-800AS digital scale and served a standard breakfast. At 10:30A.M. (10:00A.M. for the resistance exercise), participants completed the aerobic (running at 70% of VO_{2peak}) or resistance (supersets at 70% of 1-maximal repetition) exercise interventions, which ended when target ExEE was reached (\approx 11:00A.M. and 11:30A.M. for the aerobic and resistance exercises, respectively), or the sedentary control session (recreational reading for 45 minutes). ExEE during each intervention was measured with a portable indirect calorimetry unit (model K4b², COSMED, Chicago, IL), as previously described (Cadieux *et al.*, 2014).

After each intervention (\sim 2:00P.M.), participants wore a biaxial accelerometer (SenseWear Pro 3 Armbands©, HealthWear Bodymedia, Pittsburgh, PA) around the upper arm to assess habitual sleep parameters over 22 hours. Sharif *et al.* (2013) noted no significant differences in sleep duration, wake-time and sleep efficiency assessed with this biaxial accelerometer or polysomnography. The overall intraclass correlation between both tools was above 0.8, indicating good agreement in assessing sleep parameters (Sharif *et al.* 2013). These results were also verified with sleep diaries. Participants were asked to rate their sleep quality the following morning by choosing an answer on a 5-point Likert scale that best describes their sleep quality the preceding night (1-much better than normal, 2-better than normal, 3-normal, 4-worse than normal and 5-much worse than normal). Participants also rated their feelings of sleepiness on a 7-point Likert scale (1-Alert/Not Tired and 7-Sleep onset soon/Very tired) within 15 minutes prior to going to bed, and within 15 minutes of waking up the following morning.

The following morning, between 10:00A.M. and 12:00P.M., participants completed a validated computer-based behavioral procedure (Leeds Food Preference Questionnaire).

Participants rated the extent to which they "wanted" 16 pre-determined and randomly presented

visual food cue with a 100-millimeter visual analogue scale. A mean “wanting” score, which combines the ratings for all 16 visual food cues was calculated, and compared between sessions. Additional details on the questions and scoring methods used are described elsewhere (Finlayson *et al.*, 2008).

Statistical analyses were performed using SPSS (version 17.0; SPSS Inc, Chicago, IL). One-way repeated measures ANOVA tests determined the effects of exercise modality on sleep parameters and next morning food "wanting". Bivariate Spearman correlations with Bonferroni corrections assessed the strength of relationships between sleep parameters with food "wanting" at each session, as well as the changes in these parameters between sessions (Δ control-aerobic; Δ control-resistance; Δ aerobic-resistance). Data are presented as mean \pm standard deviation. Differences with *P*-values <0.05 and ≤ 0.004 were considered statistically significant for the ANOVA analyses and Spearman correlations, respectively.

Results

No differences were noted between sessions (**Table 1**), or across time for all sleep outcomes, and food “wanting” (results not shown). Sleep duration was negatively associated with food "wanting" in the aerobic exercise session ($\rho = -0.83$; $P=0.0001$). Changes in sleep duration ($\rho = -0.78$; $P=0.001$) and wake-time ($\rho = -0.77$; $P=0.001$) were negatively correlated with Δ food "wanting" in the Δ aerobic-resistance exercise condition (**Figure 1**). However, these associations were no longer significant after correcting for the time elapsed between wake-time and completion of the food reward task (results not shown).

No other significant correlations were noted between sleep parameters with food "wanting", or between the changes in these variables (results not shown). Lastly, no significant correlations were noted between naptime (minutes) and PSQI scores with sleep efficiency and sleepiness measurements (results not shown).

Discussion

This is the first study to investigate acute effects of isocaloric aerobic and resistance exercise on sleep parameters, and whether habitual changes in sleep parameters are associated with next morning food “wanting”.

Our results indicated no significant effects of exercise modality on sleep, which supports studies that subjectively (Porter & Horne 1981) and objectively (King *et al.*, 2008) assessed sleep parameters on exercise and non-exercise days. A study that noted greater sleep duration and slow-wave sleep time did so following acute aerobic exercise to exhaustion (Bunnell *et al.*, 1983). The latter is further supported by a meta-analysis (Youngstedt *et al.*, 1997), which reported significant median increases in sleep duration and slow-wave sleep of 10 and 1.4 minutes, respectively, following an acute bout of aerobic exercise; with effects being greatest for aerobic exercises that exceeded 1 hour. Hence, the shorter aerobic exercise session (mean duration of 24 minutes) sustained at 70% of VO_{2peak} in the present study may not be sufficient to alter sleep. Conversely, the resistance exercise lasted on average 86 minutes, and had no significant impact on sleep. It may be hypothesized that ExEE, which was matched in this study, may have a greater impact on sleep rather than exercise duration *per se*. Future studies are needed to evaluate this hypothesis, and assess the effects of exercise modality performed at different times of day on sleep, as advancements in melatonin release following evening exercise (Buxton *et al.*, 2003) may affect sleep differently.

Secondary analyses revealed that lower sleep duration and earlier wake-times were associated with greater food reward. These results add to studies reporting greater neuronal responsiveness to food vs. non-food stimuli following imposed sleep restriction (St-Onge *et al.*, 2012; Benedict *et al.*, 2012). Although other cross-sectional studies reported associations

between later sleep timing midpoints with poorer diet qualities (*e.g.* higher fast food and lower fruit/vegetable intakes) (Baron *et al.*, 2011; Sato-Mito *et al.*, 2011), this is the first study to suggest that an earlier wake-time is associated with greater food reward. An important confounding factor in the present study is the elapsed time between wake-time and completion of the food reward task (between 10:00A.M. and 12:00P.M.), as participants with an earlier wake-time may express greater drives towards food in the morning because of greater elapsed time spent awake prior to completing the task, comparatively to individuals who usually wake later.

The present findings are limited by a small sample size of normal-weight men and women. Only 1-day assessments of outcomes, and acute exercise interventions, were performed, which may not account for normal day-to-day variability, or potential additive effects. The food reward task represents a proxy of actual food intake. Differences in exposures to environmental factors in the evening and/or overnight between conditions were not evaluated and may alter sleep (*e.g.* exposure to blue-light from technological devices). The strength of associations noted in women may be in part influenced by the menstrual cycle, as all women were tested between days 1 and 8 of the follicular phase. Lastly, correlations cannot infer causality. However, the temporal order of events (*i.e.* sleep preceding food reward measurements) reinforces current results.

These findings suggest that exercise modality does not acutely alter sleep. Shorter sleep durations and earlier wake-times were associated with greater food “wanting”, but these associations were driven by elapsed time between awakening and completion of the food reward task. Future studies are needed to confirm these preliminary findings, and further assess the effects of sleep timing, or individual circadian rhythms, on food reward. Future studies should

also consider the elapsed time between awakening and completion of food reward measurements, as this may be an important cofounder driving food seeking behavior.

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Table 1. Objectively- and subjectively-measured sleep parameters, food "wanting" and food intake assessed following each exercise session.

	Sedentary control		Aerobic exercise		Resistance exercise		Session effect
	Mean	SD	Mean	SD	Mean	SD	
<i>Sleep parameters (accelerometry)</i>							
Sleep duration (min)	416	88	432	59	407	83	<i>P</i> = 0.62
Sleep efficiency (%)	81.2	8.2	82.8	8.7	78.8	6.8	<i>P</i> = 0.21
Sleep efficiency after sleep onset (%)	86.7	7.7	86.9	8.4	86.6	6.7	<i>P</i> = 0.99
Sleep onset latency (min)	13	7	9	8	19	23	<i>P</i> = 0.23
Bedtime (<i>i.e.</i> sleep onset)	12:03A.M.	1h22min	12:22A.M.	2h16min	12:03A.M.	1h12min	<i>P</i> = 0.77
Wake-time	7:51A.M.	1h17min	7:47A.M.	1h30min	7:54A.M.	1h43min	<i>P</i> = 0.97
Daytime nap time (minutes)	24	45	11	23	22	44	<i>P</i> = 0.44
<i>Sleep parameters (self-reported)</i>							
Sleep quality	3.2	0.9	3.4	0.6	3.0	0.6	<i>P</i> = 0.25
Evening sleepiness rating	5.0	1.5	5.0	1.0	5.4	1.1	<i>P</i> = 0.44
Morning sleepiness rating	3.2	1.0	2.8	1.1	3.4	1.2	<i>P</i> = 0.37
<i>Food reward measurement</i>							
Food "wanting" (millimeters)	40.6	15.7	35.7	15.9	36.3	15.2	<i>P</i> = 0.34

Note: Food "wanting" scores were measured with a 100-millimeter visual analogue scale. Self-reported sleep quality was measured with a 5-point Likert scale (*i.e.* score of 3 is indicative of an habitual, or "normal", sleep quality), and sleepiness ratings were measured with a 7-point Likert scale. Sleep efficiency (%) is defined as sleep duration/time lying down in bed, and sleep efficiency after sleep onset (%) is defined as sleep efficiency (%) after sleep onset and before wake-time. SD, standard deviation.

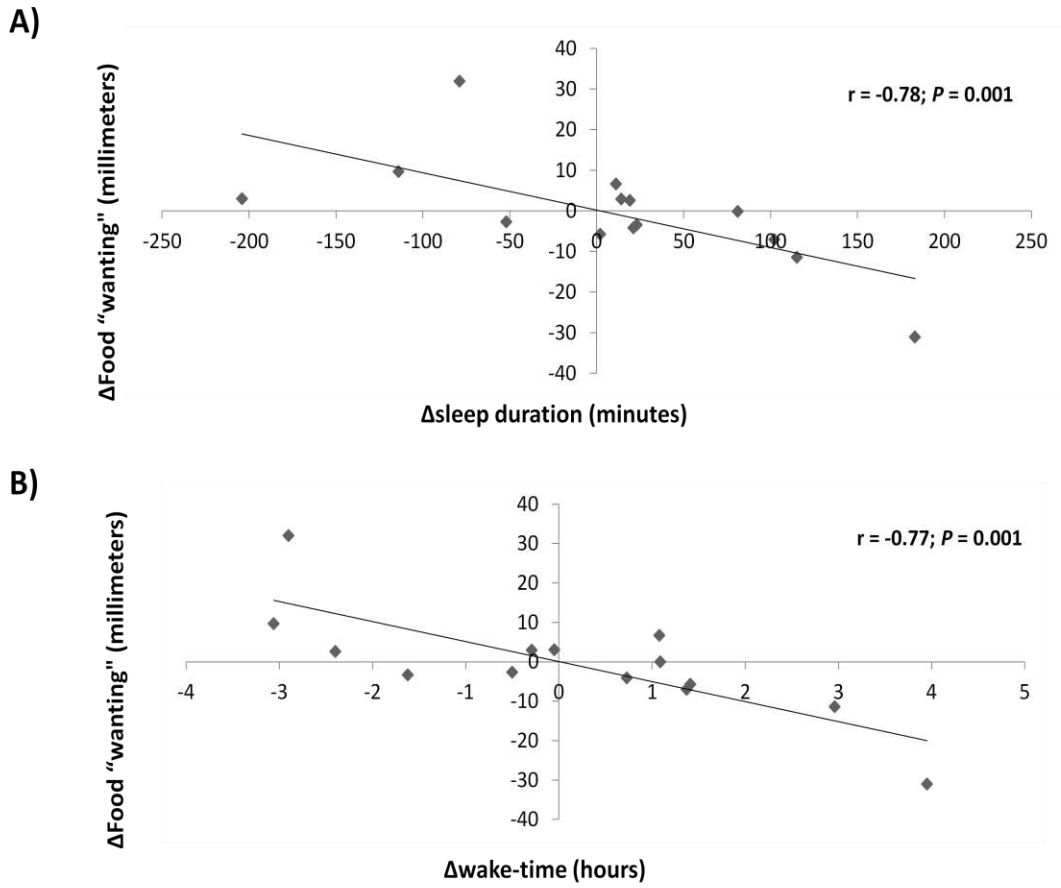


Figure 1. Associations between changes in sleep duration (A) and wake-time (B) with changes in food "wanting" between the aerobic and resistance exercise sessions. Values are presented as means for 14 participants with standard errors of the mean represented by vertical bars.

3.3 Thesis article #3

The effects of sleep restriction and altered sleep timing on energy intake and activity energy expenditure

This article was submitted for publication as a regular research paper on November 20th 2015 in the *Journal of Sleep Research*, and has been formatted accordingly.

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Author contributorship: JM, ÉD and GF formulated the research questions and designed the study. JM, J-FB, LJH, IC, ÉL, RM and AR carried out the experiment. JM, ÉD, J-FB and GF analyzed the data. All authors were involved in writing the paper and had final approval of the submitted and published version.

Abstract

It is unknown whether alterations in sleep timing, independently of sleep duration, impact energy intake (EI) and energy expenditure (EE). Hence, we examined the effects of sleep restriction with an advanced wake-time or delayed bedtime on EI and total EE over 36h. Twelve men and 6 women (age: 23 ± 4 years, body fat: $18.8\pm 10.1\%$) participated in 3 randomized crossover sessions: control (habitual bed- and wake-time), 50% sleep restriction with an advanced wake-time, and 50% sleep restriction with a delayed bedtime. Outcome variables included sleep architecture (polysomnography), EI (food menu), total and activity EE and activity times (accelerometry). Energy and carbohydrate intakes were greater over 36h in the delayed bedtime vs. control session (EI: 5502 ± 1592 vs. 5031 ± 1470 kcal, $P=0.03$; Carbohydrate intake: 3202 ± 1018 vs. 2893 ± 859 kcal, $P=0.03$). Activity EE was greater in the delayed bedtime session vs. control and advanced wake-time sessions on day 1 (1.3 ± 1.1 vs. 0.9 ± 0.6 and 1.0 ± 0.6 kcal/min, $P=0.03$). Moderate-intensity physical activity (PA) was also greater in the delayed bedtime session vs. control and advanced wake-time sessions on day 1 (27 ± 20 vs. 16 ± 11 and $18\pm 12\%$, $P=0.01$) and over 36 h (22 ± 14 vs. 14 ± 9 and $15\pm 6\%$, $P=0.01$), whereas vigorous-intensity PA time was greater following advanced wake-time vs. delayed bedtime on day 1 (3 ± 3 vs. $1.3\pm 2.4\%$, $P=0.02$). Greater sleep quality and slow-wave sleep (SWS) duration between sleep restriction sessions were associated with reduced fat intake ($r = -0.59$; $P=0.01$) and greater vigorous-intensity PA time ($r = 0.52$; $P=0.03$), respectively. These findings suggest that sleep restriction with a delayed bedtime leads to greater activity EE and EI. Additionally, individuals with greater sleep efficiency and SWS in response to sleep restriction had greater vigorous-intensity PA and lower energy and macronutrient intakes the next day.

Keywords: food intake, physical activity, sleep architecture, bedtime, wake-time

Introduction

Borbély (1982) suggested that sleep is regulated by 2 overlapping processes: the homeostatic sleep drive (or process "S") and the circadian rhythm (or process "C"). The homeostatic sleep drive (process "S") promotes the occurrence of slow-wave sleep (SWS) as the amount of this sleep stage is greatly influenced by the length of prior wakefulness (Gonnissen *et al.* 2013). Conversely, REM sleep is mainly influenced by the circadian rhythm (process "C") and is more common during the second part of the night when core temperature is reduced and hypothalamic-pituitary-adrenal (HPA) axis activity and cortisol release are greater (Wu *et al.* 2008). Sleep restriction protocols (Tilley & Wilkinson 1984; Wu *et al.* 2010) comparing sleep architecture when anchoring the sleep period during the first or second half of the night reported no differences in SWS between sleep restriction protocols, whereas REM sleep was greater during sleep held the second half of the night. Stage 2 sleep duration was consequently reduced as a result of maintained SWS and greater REM sleep durations during this time.

Studies have reported mean increases of ≈ 300 -500 kilocalories over 24h following an imposed sleep restriction condition *vs.* a control condition (habitual sleep duration) (Bosy-Westphal *et al.* 2008; Brondel *et al.* 2010; Calvin *et al.* 2013; Markwald *et al.* 2013; Spaeth *et al.* 2013; St-Onge *et al.* 2011). However, the effects of imposed sleep restriction on energy expenditure (EE) are not as consistent, with some studies reporting no changes (St-Onge *et al.* 2011; Calvin *et al.* 2013; Nedeltcheva *et al.* 2009), greater (Brondel *et al.* 2010; Shechter *et al.* 2013) or lower (Schmid *et al.* 2009) in total and/or activity EE following similar sleep restriction protocols (2-3 nights of 4h in bed/night). Studies have also reported negative associations between SWS and energy intake (EI) the following day under habitual sleep conditions (Rutters *et al.* 2012), as well as negative associations between changes in SWS and REM sleep with

changes in carbohydrate and fat intakes between a habitual and partial sleep restriction condition (Shechter *et al.* 2012).

Taken together, these studies suggest that reduced sleep duration increases EI and may affect EE. However, it is unknown whether imposed alterations in sleep timing, in addition to reduced sleep duration, have an effect on EI and EE the following day. The primary objective of the present study was to evaluate the effects of a 50% sleep restriction with an advanced wake-time or delayed bedtime on EI, total and activity EE over 36h. The secondary objective was to assess the strength of the associations between changes in sleep architecture with changes in EI and EE between sessions. It was hypothesized that sleep restriction with an advanced wake-time would lead to greater EI, coupled with lower activity EE and moderate-to-vigorous physical activity (PA) time. It was also hypothesized that these changes in EI and EE would be associated with changes in REM sleep between the control and advanced wake-time sessions.

Materials and Methods

Participants

Eighteen participants (12 men and 6 women) completed all sessions. Participants were between the ages of 18-45 years, non-smokers, weight stable (± 4 kg) within the last 6 months, did not have heart problems or diabetes, did not take medication which may affect appetite or sleep, and reported not performing shift work nor taking regular daytime naps. All participants reported having habitual sleep duration of 7-9 h/night. Only women taking monophasic combined estrogen-progesterone birth control were recruited to control for sex-steroid hormone effects on sleep parameters (Baker *et al.* 2001). This study was conducted according to the guidelines laid down in the Declaration of Helsinki and the University of Ottawa ethics committee approved all procedures involving human participants. Written informed consent was obtained from all participants.

Design and Procedure

Participants took part in a preliminary session followed by 2 weeks of sleep-wake monitoring with accelerometry and sleep diaries, an in-lab habituation night followed by a recovery night at home, and 3 experimental sessions. **Figure 1** presents an overview of the sleep protocol for each experimental session. A washout period of at least 7 days separated each experimental session. Participants were instructed not to consume alcohol or exercise for at least 24h prior to the preliminary and experimental sessions. They were also asked not to consume caffeinated products after 12h00, and to wash their hair in order to facilitate electrode installation on the day of each experimental session. Compliance to these instructions was verified by self-report at the start of each session.

Preliminary session

Participants arrived at the lab at 8h00 following an imposed 12h overnight fast. At this time, height, body weight and composition were measured. Participants were then provided with *ad libitum* quantities of the following foods for breakfast: whole-wheat toast (*D'Italiano*[®]; 4 slices), strawberry jam (*Smuckers*[®]; 60 grams), peanut butter (*Kraft Smooth Peanut Butter*[®]; 60 grams), cheddar cheese (*Cracker Barrel Marble Cheddar Cheese*[®]; 42 grams) and orange juice (*Tropicana*[®]; 500 grams). They were given 15 minutes to eat as much or as little as they wanted. The measured quantity and composition of the consumed breakfast was provided to them during each experimental session, and they were instructed to consume the breakfast in its entirety during these sessions. Hence, the energy content and composition of the breakfast varied between participants, but were constant across sessions for the same participant. To assess habitual chronotype and degrees of daytime sleepiness, participants completed the Horne-Ostberg Morningness-Eveningness Questionnaire (Horne & Ostberg 1976) and the Epworth sleepiness scale (Johns 1991), respectively. Participants with extreme morning or evening chronotypes (scores ≥ 70 and ≤ 30 , respectively) (n=0) and/or frequent feelings of daytime sleepiness (score ≥ 10) (n=1) were excluded from further study participation. Lastly, participants were asked to wear an accelerometer (SenseWear Pro 3 Armbands[®], HealthWear Bodymedia, Pittsburgh, PA, USA) and to complete a sleep diary over 2 weeks to assess habitual sleep patterns (bedtime, wake-time, sleep duration and efficiency). Participants were excluded from further participation if they had a mean sleep efficiency over 2 weeks $< 80\%$ (n=1). These data were also used to tailor the experimental interventions to each participant, and to better capture inter-individual representations of habitual sleep patterns.

Experimental sessions

All participants took part in 2 habituation nights, which included an in-lab session during which the entire polysomnography (PSG) set-up was performed, followed by a recovery night at home. The first in-lab habituation night was used to exclude participants with sleep disorders (*e.g.* restless leg syndrome, sleep apnea), and to provide an adaptation to the experimental settings used for in-lab sleep assessment. No participants were excluded based on detected sleep disorders, but 1 participant was excluded from further testing because of very low sleep efficiency during this session (sleep efficiency in this instance was $\approx 20\%$).

Following this session, participants took part in 3 randomized crossover sessions: control (habitual bed- and wake-time), 50% sleep restriction with an habitual bedtime and advanced wake-time, and 50% sleep restriction with a delayed bedtime and habitual wake-time. Each session followed the same protocol and differed only in the amount of total time in bed, and the assigned bed- or wake-time. Since the assigned bed- and wake-times slightly differed between participants (range for bedtime: 22h16-24h40, wake-time: 6h18-8h37), the time at which each test was administered also differed according to this same range between participants, but remained the same for each participant across sessions. Participants were evenly randomized for the order of experimental sessions (*i.e.* 6 participants started with each of the 3 experimental sessions). Participants arrived at the lab 3h prior to their set bedtime to allow enough time to place the electrodes (≈ 90 min), set up the polysomnogram (≈ 30 min) and allow for some downtime before bedtime (≈ 60 min). When forced to remain awake during the night and the following morning, participants took part in sedentary activities (*e.g.* reading, watching movies), and remained inside the lab with the researchers. Upon awakening, participants took a shower. Prior to breakfast consumption, which was set at the same time during each session, body weight

was measured. Participants also had access to an *ad libitum* lunch, which was consumed inside the lab before being given *ad libitum* quantities of self-selected food items in packed containers for the remainder of the day (day 1; upon leaving the lab until midnight or ≈ 18 h) and the following day (day 2; midnight to midnight or 24h) (McNeil *et al.* 2012) along with an accelerometer to wear for this same time period. Lastly, they were asked to bring back the remaining food and all containers, along with the accelerometer, within the few days following the end of data collection.

Anthropometric measurements

Standing height was measured, without shoes, to the nearest centimeter using a Tanita HR-100 height rod (Tanita Corporation of America Inc, Arlington Heights, IL, USA). Body weight and composition (body fat %) were measured using a standard beam scale (HR-100; BWB-800AS, Tanita Corporation, Arlington Heights, IL, USA) and DXA scanner (Lunar Prodigy, General Electric, Madison, WI, USA), respectively. Standing height and body composition were only measured during the preliminary session, whereas body weight was measured prior to breakfast during the 3 experimental sessions.

Sleep assessment and sleepiness ratings

Sleep was recorded using EEG (C3, C4, O1, O2, F3 and F4), EMG (bipolar submental) and EOG on a Medipalm 22 (Braebon Medical Corporation, Kanata, Ontario, Canada), with the Pursuit Sleep Software (Braebon Medical Corporation, Kanata, Ontario, Canada) inside the lab. Inferior tibialis EMG and naso-oral thermistor signals were also recorded during the in-lab adaptation night. All PSG recordings were scored independently by 2 judges according to the AASM (2007) criteria using 30-second epochs; discrepancies were resolved by mutual agreement. The following sleep variables were extracted: total sleep duration in minutes (from

sleep onset to wake-time, minus wake after sleep onset), sleep latency (elapsed time between the set bedtime and 10 minutes of stage 1 or 20 seconds of any other sleep stage), % of sleep efficiency [(sleep time/time in bed) X 100], wake after sleep onset (WASO) in minutes, as well as the absolute amount of time spent in each sleep stage (stages 1 and 2 sleep, SWS and REM sleep).

Feelings of sleepiness were assessed prior to bedtime, immediately upon awakening and 30 minutes post-awakening with a 100-mm computerized visual analogue scale (Marsh-Richard *et al.* 2009) with the following question: "How sleepy do you feel?" (Alert/Not sleepy at all - very sleepy/sleep onset soon).

Ad libitum energy intake

Ad libitum energy and macronutrient intakes were measured with a validated food menu (McNeil *et al.* 2012). Participants completed separate menus for lunch (consumed inside the lab), the remainder of that day (end of the session to midnight; consumed outside the lab), and the entire following day (midnight to midnight; consumed outside the lab). Standard breakfast intake, in-lab *ad libitum* lunch and EI for the remainder of the day (end of session to midnight) were added, and provided energy and macronutrient intake values for day 1. Day 2 EI included the foods consumed during the following day (midnight to midnight). When completing the food menus, participants were instructed to select the items that they may want to consume over each time period. The selected food items were prepared and measured according to the guidelines previously described by McNeil *et al.* (2012). One participant did not bring back all food containers at the end of 1 session; hence, the results of 17 participants are presented herein for day 1, day 2 and 36h energy and macronutrient intakes.

Out-of-lab energy expenditure and activity times

Participants were given a biaxial accelerometer (SenseWear Pro 3 Armbands[®], HealthWear Bodymedia, Pittsburgh, PA, USA) prior to leaving the lab during each experimental session, and were instructed to wear the accelerometer at all times, including when sleeping. The accelerometer was placed around the upper arm (mid-distance between the acromion and the olecranon). The SenseWear Professional software (version 7.0, Bodymedia, Pittsburgh, PA, USA) was used to retrieve the data once the accelerometer was returned to the lab. Collected data included: total EE (kilocalories), activity EE (kilocalories; ≥ 3 METs), sedentary time (minutes; < 3 METs), moderate-intensity PA time (minutes; 3-6 METs), vigorous-intensity PA time (minutes; > 6 METs) and estimated sleep time (minutes).

Statistical analyses

Statistical analyses were performed using SPSS (version 17.0; SPSS Inc., Chicago, IL, USA). One-way repeated measures ANOVA tests were used to determine the main effects of sleep condition on body weight, in-lab sleep variables (sleep duration, sleep efficiency, sleep latency, WASO, and absolute sleep stage duration), energy and macronutrient intakes (day 1, day 2 and 36h), EE (total and activity EE; day 1, day 2 and 36h), activity time (sedentary, moderate- and vigorous-intensity; day 1, day 2 and 36h) and sleepiness ratings. Since significant differences in accelerometer wear time were noted on day 2 (1273 ± 173 , 1372 ± 78 , 1363 ± 97 ; $P=0.01$; partial $\eta^2=0.23$), the EE per minute of wear time (kilocalories/minute), as well as the relative activity times (%) were compared between sessions. The Wilcoxon Signed Ranks Test was used to assess potential differences between sessions for variables that were not normally distributed according to the Shapiro-Wilk test. For normally distributed data, *post-hoc* tests with LSD adjustments were used to determine where significant differences existed. Bivariate

Spearman correlations were computed between changes in sleep parameters (sleep latency, sleep efficiency and absolute sleep stage duration) with changes in energy and macronutrient intakes, activity EE and activity times over 36h between sessions. Values are presented as means \pm standard deviations. Differences with *P*-values < 0.05 were considered statistically significant.

Results

Participant Characteristics, in-lab sleep assessment and sleepiness ratings

Participant characteristics are presented in **Table 1**. No significant differences in body weight were noted between sessions (69.2±9.2, 69.4±9.3, 69.2±9.4; $P=0.72$; partial $\eta^2=0.02$), a crude indication of energy balance maintenance. As expected, sleep duration was reduced during both sleep restriction conditions (**Table 2**). Sleep duration was also lower during the advanced wake-time vs. delayed bedtime condition. Sleep efficiency was greater, and WASO lower, during the delayed bedtime session vs. the control and advanced wake-time sessions. WASO was lower during the advanced wake-time vs. control session. Stage 1, stage 2 and REM sleep durations were higher during the control vs. both sleep restriction conditions (**Figure 2**). Stage 1 and 2 sleep durations were higher, and REM sleep was lower, during the advanced wake-time vs. delayed bedtime condition. SWS was only higher during the control vs. advanced wake-time condition. Sleepiness ratings were higher prior to bedtime when it was delayed, and upon awakening in the advanced wake-time session.

Energy and macronutrient intakes

No differences in energy and macronutrient intakes were noted for day 1 (**Table 3**). Carbohydrate intake was greater during the delayed bedtime vs. control session on day 2. Greater 36h energy and carbohydrate intakes were noted during the delayed bedtime vs. control session (**Figure 3A**).

Energy expenditure and activity times

Table 3 presents data for relative total and activity EE from accelerometry, and relative activity times during each session for days 1 and 2. **Figure 3B** illustrates relative activity times over 36h. Activity EE was greater during the delayed bedtime compared to control on day 1. No

differences were noted in total and activity EE between sessions on day 2 and over 36h. Vigorous-intensity PA time was higher during the advanced wake-time vs. delayed bedtime session on day 1, but this difference was not observed on day 2 and over 36h. Conversely, moderate-intensity PA time was higher during the delayed bedtime compared to the advanced wake-time and control sessions on day 1 and over 36h. No differences in sedentary and sleep times were noted between sessions for days 1 and 2, and over 36h.

Delta correlation results

Lower sleep latency ($r = -0.50$; $P=0.03$) and higher SWS duration ($r = 0.50$; $P=0.04$) were correlated with greater activity EE between the control and advanced wake-time sessions. Greater sleep efficiency ($r = 0.57$; $P=0.02$) and lower sleep latency ($r = -0.59$; $P=0.01$) were associated with greater 36h protein intake between these sessions. When comparing both sleep restriction sessions, greater sleep efficiency was associated with lower fat intake ($r = -0.59$; $P=0.01$). Greater sleep latency was associated with greater energy ($r = 0.58$; $P=0.02$), carbohydrate ($r = 0.55$; $P=0.02$) and fat ($r = 0.64$; $P=0.01$) intakes. Lastly, greater SWS was associated with higher vigorous-intensity PA time ($r = 0.52$; $P=0.03$), whereas greater REM sleep was associated with higher sedentary time ($r = 0.49$; $P=0.04$). No other significant correlations were noted between changes in sleep parameters and changes in energy balance parameters (results not shown).

Discussion

To our knowledge, this is the first study to examine the effects of an imposed sleep restriction combined with altered bed- or wake-times on objective measures of EI and EE with a randomized crossover design. This study also used 2 weeks of accelerometry data for each participant to personalize the bed- and wake-times for the experimental sessions, which offers optimal control over inter-individual variations in circadian rhythms. This may in part explain the relatively high in-lab sleep efficiency values (93-97%) observed in this study. Collectively, our results suggest that carbohydrate intake on day 2 and over 36h was greater following sleep restriction with a delayed bedtime compared to the control session. Additionally, activity EE and moderate-intensity PA time were greater on day 1 and over 36h during the delayed bedtime *vs.* advanced wake-time and control sessions. These results do not support our initial hypothesis. The changes in EI and activity EE between the control and advanced wake-time sessions were not associated with changes in REM sleep duration, thus refuting our second hypothesis. However, when comparing both sleep restriction conditions, greater sleep quality and SWS duration were associated with decreased energy and macronutrient intakes, and greater vigorous-intensity PA time, respectively. This is the first study to report associations between changes in sleep architecture with next day EI and EE between sleep restriction conditions.

Differences in EI over 36h were mainly driven by greater carbohydrate intake during day 2 following sleep restriction with a delayed bedtime *vs.* control. Many experimental studies reported greater 24h EI following sleep restriction *vs.* control (Bosy-Westphal *et al.* 2008; Brondel *et al.* 2010; Calvin *et al.* 2013; Markwald *et al.* 2013; Spaeth *et al.* 2013; St-Onge *et al.* 2011). Although sleep timing was not evaluated in these studies, the majority employed longer-

term (\approx 2-14 nights) sleep restriction conditions, which may explain the greater differences in EI between conditions.

It is also possible that the greater activity EE observed during day 1 following the delayed bedtime session may be influencing these changes in carbohydrate intake on day 2. *Post-hoc* Spearman correlations revealed positive correlations between total EE on day 1 with EI on day 2 during the control and delayed bedtime sessions ($r = 0.49-58$; $P < 0.05$). Strong correlations between EI and EE in individuals with moderate-to-high levels of habitual PA participation were first demonstrated by Mayer *et al.* (1956). Considering that the participants in the present study were healthy, active individuals (\approx 15-23% of moderate-to-vigorous intensity PA time), increases in EI that would follow increases in EE are expected to occur. Experimental studies imposing partial sleep restrictions reported overall conflicting results for activity EE; some reporting no changes in activity EE inside a lab/inpatient clinic (Calvin *et al.* 2013; Nedeltcheva *et al.* 2009) and under free-living conditions (St-Onge *et al.* 2011), whereas others reported either greater (Brondel *et al.* 2010) or lower (Schmid *et al.* 2009) activity EE following sleep restriction. Differences in habitual PA levels may explain these discrepancies in study results, as individuals with habitually greater levels of PA, such as those recruited in the present study, may resort to greater PA participation to combat feelings of fatigue. Studies comparing energy balance measurements in individuals with habitually high- vs. low-levels of PA participation following sleep restriction and altered sleep timing are needed to further investigate this hypothesis.

Greater sleep efficiency and SWS duration were associated with higher protein intake and activity EE between the control and advanced wake-time sessions. Previous studies reported greater sleep quality and/or SWS following amino acid infusion (Lacey *et al.* 1978) and exercise

(King *et al.* 2008). Hence, these associations may be reciprocal, as greater sleep quality may exert an effect on PA participation and dietary choices or *vice versa*. When comparing both sleep restriction conditions, greater sleep quality and SWS duration were associated with smaller energy and macronutrient intakes, and greater vigorous-intensity PA time, respectively. Previous studies reported associations between SWS and REM sleep durations with energy balance parameters under habitual (control) conditions (Rutters *et al.* 2012), and when assessing changes in sleep architecture between a habitual and partial sleep restriction condition (Shechter *et al.* 2012). Although no cause-and-effect associations can be drawn from the present findings, it is possible that participants with greater SWS and overall sleep quality may be able to maintain (or increase) high-activity levels and exert greater control over food intake, even though total sleep time is reduced by 50%.

The present findings are limited to a small sample size of healthy, physically active adults with a high sleep efficiency. This limits generalizability to other populations, including individuals with sleep complaints or disorders. Accelerometry provides an estimation of sleep-wake activities. The assessment of sleep with PSG and all outcome variables were only conducted for 1 night and 36h post-intervention in each condition, which does not account for day-to-day variations, and limits the comparison of results with studies imposing prolonged sleep restrictions.

In conclusion, sleep restriction with a delayed bedtime led to greater activity EE, moderate-intensity PA time, and carbohydrate intake. However, the temporal order of effects suggests that greater activity EE may be associated with this greater carbohydrate intake. Additionally, greater sleep quality and SWS duration between sleep restriction sessions were associated with lower energy and macronutrient intakes, and greater vigorous-intensity PA time,

respectively. These novel findings suggest that individuals with higher levels of sleep efficiency and SWS and/or reduced sleep latency in response to sleep restriction had greater vigorous-intensity physical activity time and lower energy and macronutrient intakes.

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Table 1. Participant characteristics (n = 18)

	Mean ± SD
Age (years)	23 ± 4
Body mass index (kg/m ²)	22.7 ± 2.7
Body fat (%)	18.8 ± 10.1
Standard breakfast intake (grams)	500 ± 147
Standard breakfast intake (kcal)	779 ± 240
<i>Carbohydrate intake (kcal)</i>	406 ± 121
<i>Fat intake (kcal)</i>	292 ± 110
<i>Protein intake (kcal)</i>	109 ± 34
Total score on the Epworth Sleepiness scales (0-10)	6 ± 2
Total score on the Horne-Ostberg Morning-Eveningness Questionnaire (30-70)	54 ± 8
Habitual time in bed (min)*	490 ± 38
Habitual sleep efficiency (%)*	86 ± 4
Habitual bedtime (24-h clock)	23h27 ± 37 min
Habitual wake-time (24-h clock)	7h37 ± 38 min
Habitual sleep timing midpoint (24-h clock)	3h32 ± 32 min

*Based on data collected for 14 days with accelerometry.

Note: kcal, kilocalories; SD, standard deviation

Table 2. In-laboratory sleep parameters during each session (n = 18)

	Control	Advanced wake-time	Delayed bedtime	Main effect analysis
	<i>Mean ± SD</i>	<i>Mean ± SD</i>	<i>Mean ± SD</i>	<i>P value; partial η^2</i>
Sleep duration (min)	463 ± 30 ^a	229 ± 17 ^b	236 ± 17 ^c	<i>P</i> = 0.0001; partial η^2 = 0.99
Sleep efficiency (%)	95 ± 3 ^a	93 ± 4 ^a	97 ± 2 ^b	<i>P</i> = 0.001; partial η^2 = 0.32
Sleep latency (min)	10 ± 13 ^a	10 ± 8 ^a	4 ± 4 ^a	<i>P</i> = 0.07; partial η^2 = 0.16
WASO (min)	17 ± 10 ^a	8 ± 8 ^b	5 ± 3 ^c	<i>P</i> = 0.0001; partial η^2 = 0.57
Sleepiness ratings (0-100)				
<i>Prior to bedtime</i>	65 ± 14 ^a	63 ± 22 ^a	83 ± 16 ^b	<i>P</i> = 0.002; partial η^2 = 0.34
<i>Upon awakening</i>	44 ± 26 ^a	68 ± 20 ^b	60 ± 25 ^b	<i>P</i> = 0.001; partial η^2 = 0.32
<i>30 minutes post-awakening</i>	22 ± 16 ^a	51 ± 19 ^b	45 ± 26 ^b	<i>P</i> = 0.0001; partial η^2 = 0.46

Note: Means not sharing the same letter are significantly different from each other (*P* < 0.05).

REM, rapid eye movement; SWS, slow-wave sleep; SD, standard deviation; WASO, wake after sleep onset

Table 3. *Ad libitum* energy and macronutrient intakes (n = 17), as well as energy expenditure and activity times (n = 18) during each session

	Control	Advanced wake-time	Delayed bedtime	Main effect analysis
	Mean ± SD	Mean ± SD	Mean ± SD	P value; partial η^2
<i>Intake - Day 1</i>				
Energy intake (kcal)	2686 ± 765 ^a	2768 ± 825 ^a	2844 ± 735 ^a	P = 0.42; partial η^2 = 0.05
Carbohydrate intake (kcal)	1507 ± 442 ^a	1544 ± 496 ^a	1623 ± 483 ^a	P = 0.33; partial η^2 = 0.07
Fat intake (kcal)	854 ± 304 ^a	874 ± 313 ^a	870 ± 238 ^a	P = 0.92; partial η^2 = 0.01
Protein intake (kcal)	385 ± 139 ^a	401 ± 130 ^a	411 ± 126 ^a	P = 0.32; partial η^2 = 0.07
<i>Intake - Day 2</i>				
Energy intake (kcal)	2345 ± 816 ^a	2534 ± 783 ^a	2658 ± 899 ^a	P = 0.05; partial η^2 = 0.17
Carbohydrate intake (kcal)	1386 ± 513 ^a	1453 ± 440 ^{a,b}	1579 ± 571 ^b	P = 0.04; partial η^2 = 0.18
Fat intake (kcal)	649 ± 291 ^a	755 ± 316 ^a	743 ± 281 ^a	P = 0.10; partial η^2 = 0.13
Protein intake (kcal)	354 ± 138 ^a	377 ± 142 ^a	385 ± 145 ^a	P = 0.44; partial η^2 = 0.05
<i>Expenditure - Day 1</i>				
Total (kcal/min)	2.2 ± 0.6 ^a	2.3 ± 0.5 ^a	2.5 ± 0.9 ^a	P = 0.05; partial η^2 = 0.16
Active (kcal/min)	0.9 ± 0.6 ^a	1.0 ± 0.6 ^{a,b}	1.3 ± 1.1 ^b	P = 0.04; partial η^2 = 0.18
<i>Expenditure - Day 2</i>				
Total (kcal/min)	2.0 ± 0.5 ^a	2.0 ± 0.3 ^a	2.1 ± 0.5 ^a	P = 0.28; partial η^2 = 0.07
Active (kcal/min)	0.6 ± 0.5 ^a	0.7 ± 0.4 ^a	0.9 ± 0.6 ^a	P = 0.22; partial η^2 = 0.08
<i>Expenditure - 36 hours</i>				
Total (kcal/min)	2.0 ± 0.5 ^a	2.1 ± 0.3 ^a	2.2 ± 0.6 ^a	P = 0.10; partial η^2 = 0.13
Active (kcal/min)	0.7 ± 0.5 ^a	0.7 ± 0.4 ^a	1.0 ± 0.7 ^a	P = 0.06; partial η^2 = 0.16
<i>Activity time - Day 1*</i>				
Vigorous-intensity (%)	1.6 ± 2.3 ^{a,b}	2.7 ± 3.0 ^a	1.3 ± 2.4 ^b	P = 0.02; partial η^2 = 0.20
Moderate-intensity (%)	16.1 ± 10.6 ^a	17.5 ± 11.8 ^{a,b}	26.6 ± 19.9 ^b	P = 0.01; partial η^2 = 0.24
Sedentary (%)	73.0 ± 13.1 ^a	66.4 ± 16.9 ^a	62.3 ± 18.4 ^a	P = 0.06; partial η^2 = 0.07
Sleep (%)	9.3 ± 6.2 ^a	13.4 ± 10.9 ^a	9.8 ± 11.7 ^a	P = 0.43; partial η^2 = 0.05
<i>Activity time - Day 2*</i>				
Vigorous-intensity (%)	0.9 ± 1.2 ^a	1.1 ± 2.1 ^a	1.1 ± 1.5 ^a	P = 0.92; partial η^2 = 0.01
Moderate-intensity (%)	12.6 ± 9.3 ^a	12.6 ± 7.3 ^a	16.6 ± 10.0 ^a	P = 0.18; partial η^2 = 0.10
Sedentary (%)	51.6 ± 9.5 ^a	52.3 ± 6.0 ^a	48.6 ± 13.1 ^a	P = 0.45; partial η^2 = 0.05
Sleep (%)*	35.0 ± 7.8 ^a	34.0 ± 7.1 ^a	33.7 ± 8.7 ^a	P = 0.82; partial η^2 = 0.01

Note: Means not sharing the same letter are significantly different from each other ($P < 0.05$).

*Day 1 includes activities from the time the participants left the laboratory until midnight; a total of \approx 18 h. Day 2 includes activities from midnight to midnight the following day (total of 24 h).

kcal, kilocalories; SD, standard deviation

|-----Habitual Sleep Duration -----|

Based on 2 weeks of accelerometry data



Control session



50% sleep restriction-Delayed bedtime



50% sleep restriction-Advanced wake-time

■ Sleep Duration

□ Sleep Restriction

Figure 1. The sleep protocol applied during each experimental session.

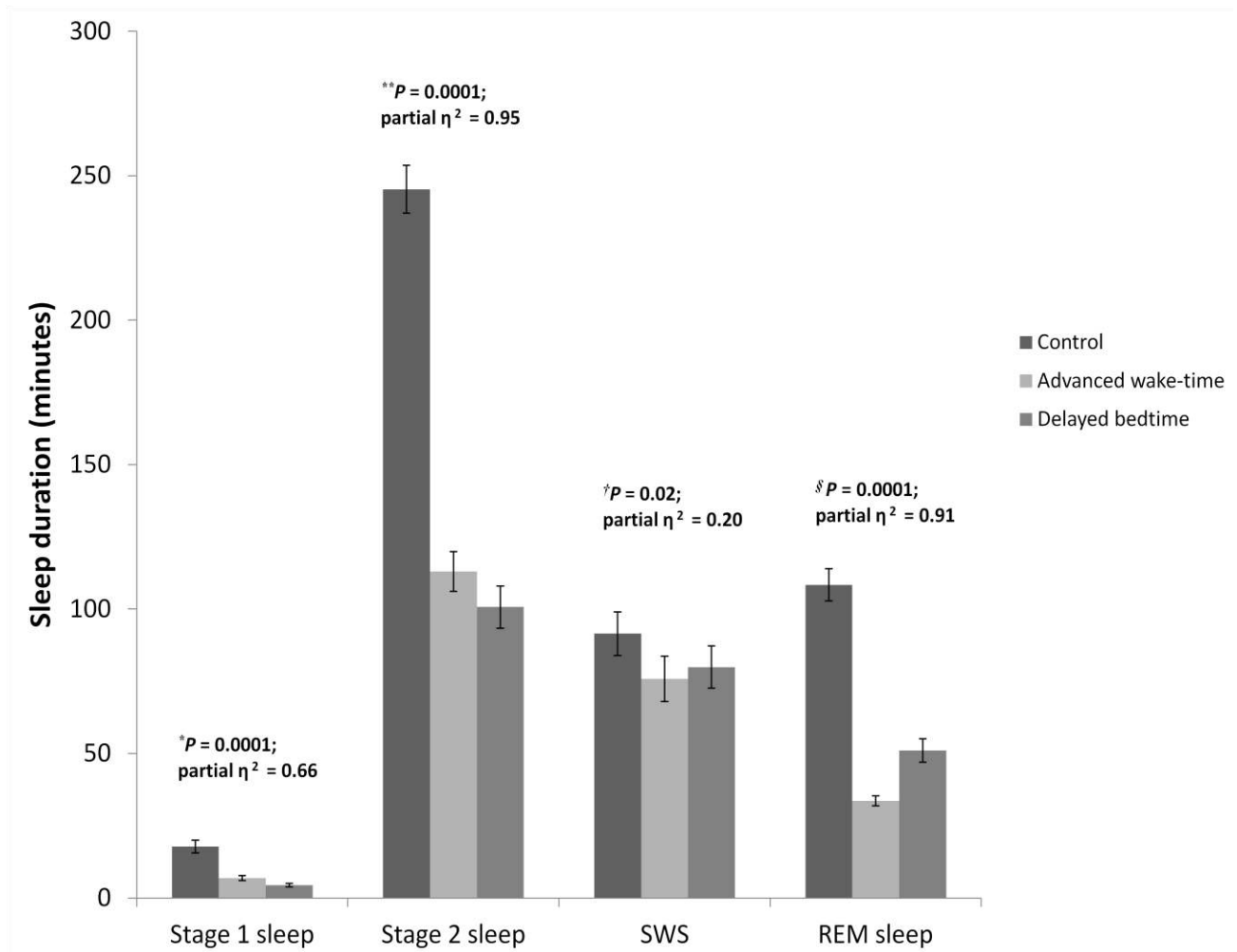


Figure 2. The absolute amount of time (minutes) spent in each sleep stage during the 3 experimental sessions. Values are presented for 18 participants. Means and SEM. *Stage 1 sleep: $P=0.0001$ for control vs. delayed bedtime and advanced wake-time, and $P=0.01$ for advanced wake-time vs. delayed bedtime. **Stage 2 sleep: $P=0.0001$ for control vs. delayed bedtime and advanced wake-time, and $P=0.04$ for advanced wake-time vs. delayed bedtime. †SWS: $P=0.01$ for control vs. advanced wake-time. §REM sleep: $P=0.0001$ for control vs. advanced wake-time and delayed bedtime, and $P=0.0001$ for delayed bedtime vs. advanced wake-time.

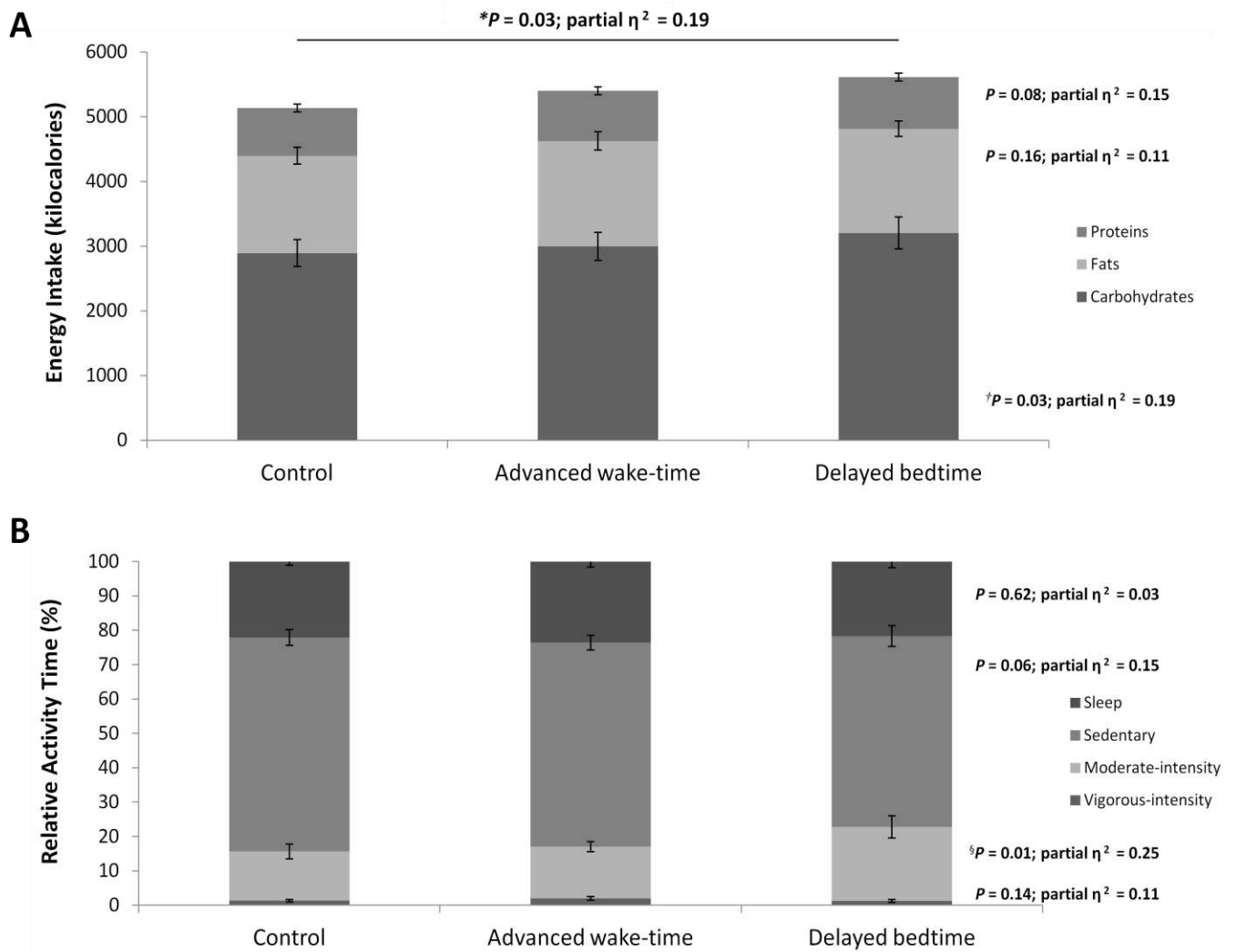


Figure 3. *Ad libitum* energy and macronutrient intakes (A) and the relative amount of activity time (%) measured over 36h (B) during the 3 experimental sessions. Values are presented for 17 and 18 participants for intake and activity time, respectively. Means and SEM. *Energy intake: $P=0.03$ for control vs. delayed bedtime and $P=0.05$ for control vs. advanced wake-time. † Carbohydrate intake: $P=0.03$ for control vs. delayed bedtime. § Moderate-intensity activity time: $P=0.02$ for control vs. delayed bedtime and $P=0.04$ for advanced wake-time vs. delayed bedtime.

3.4 Thesis article #4

The effects of partial sleep restriction and altered sleep timing on appetite and food reward

This article will be submitted to the *American Journal of Clinical Nutrition* for publication following the publication of thesis article #3.

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Abstract

Whether alterations in sleep timing, independently of sleep duration, impact measures of appetite, food reward and energy intake (EI) remains to be established. We examined the effects of partial sleep restriction (PSR) with an advanced wake-time or delayed bedtime on measures of appetite, food reward and subsequent EI. Twelve men and six women (age: 23 ± 4 years, body fat: $18.8\pm 10.1\%$) participated in 3 randomized crossover sessions: control (habitual bed- and wake-time), 50% PSR with an advanced wake-time, and 50% PSR with a delayed bedtime. Outcome variables included sleep architecture (polysomnography), *ad libitum* EI (validated food menu), appetite sensations (visual analogue scales), the satiety quotient (SQ; mm/100 kcal) and food reward (Leeds Food Preference Questionnaire and the relative-reinforcing value (RRV) of preferred food task). Greater appetite ratings were noted following PSR with an advanced wake-time compared to the control session (Hunger ratings: 39 ± 14 vs. 31 ± 15 ; $P = 0.01$). Greater explicit wanting and liking for high- relative to low-fat foods were also noted during the advanced wake-time vs. control session (Explicit wanting: -3.5 ± 12.5 vs. -9.3 ± 8.9 , $P = 0.01$; Explicit liking: -1.6 ± 8.5 vs. -7.8 ± 9.6 , $P = 0.002$). No differences in the RRV of preferred food, the SQ and *ad libitum* lunch intake were noted between sessions. These findings suggest that appetite sensations and food reward are greater when wake-time is advanced, independently of sleep duration. However, this did not translate into greater EI during a test meal. It is possible that the severity of the sleep intervention imposed in the present study may not be sufficient, or would have to be more prolonged in order to reduce executive control, so that in the presence of greater appetite sensations and food reward, increases in EI would actually take place.

Keywords: appetite, food reward, satiety quotient, sleep architecture, sleep deprivation

Introduction

Food reward, which encompasses measures of food "liking" (*i.e.* sensory properties of a food item) and food "wanting" (*i.e.* objective drive to seek and consume a targeted food item), and appetite sensations can be seen as major forces in directing human food selection and intake⁽¹⁻³⁾. Spiegel *et al.*⁽⁴⁾ were among the first to demonstrate greater feelings of hunger and appetite ratings for calorie-dense, high-carbohydrate foods following 2 days of partial sleep restriction. Furthermore, a recent functional MRI study observed enhanced activation in the orbitofrontal cortex in response to visual food cues following partial sleep restriction (4 vs. 9 h in bed/night)⁽⁵⁾. Activity in reward and food-sensitive areas of the brain was also greater in response to unhealthy vs. healthy food cues in these same participants following partial sleep restriction⁽⁶⁾. A second study, in which greater right anterior cingulate cortex activation in response to visual food cues was observed following 1 night of total sleep deprivation, corroborated these results⁽⁷⁾. Participants from this latter study also rated high-calorie foods as being 24% more appetizing following total sleep deprivation. It is possible that these effects may lead to decreases in impulse control and delayed gratification, which have been previously noted in participants who made riskier selections, despite the long-term losses that could ensue, when faced with a gambling scenario following 48 h of sleep deprivation⁽⁸⁾.

Sleep timing may also have a marked impact on food preference⁽⁹⁾, as adults with a later sleep timing midpoint consumed more kilocalories after 8:00PM, ate less fruits and vegetables and more fast food items, compared to those with an earlier sleep timing midpoint. Although no cause-and-effect associations can be drawn from these results, it may be hypothesized that changes in sleep architecture which occur in sleep restriction protocols with differing bed- or wake-times (*i.e.* preserved slow-wave sleep (SWS) coupled with greater stage 2 sleep during the

first half of the night *vs.* preserved SWS coupled with greater rapid eye movement (REM) sleep during the second half of the night^(10, 11)) may be related to changes in food reward and food preference. Indeed, Rutters *et al.*⁽¹²⁾ noted that participants with habitually lower amounts of SWS, independently of sleep duration, reported feeling hungrier and less full the following day, and had greater food wanting and *ad libitum* EI. Additionally, St-Onge *et al.*⁽⁶⁾ reported that individuals with smaller reductions in REM sleep in response to partial sleep restriction tend to have smaller differences in insula activation between partial sleep restriction and habitual sleep duration conditions. Shechter *et al.*⁽¹³⁾ also noted a negative association between the amount of REM sleep and next day hunger ratings. Finally, we recently showed a lower mean satiety quotient (SQ), indicative of smaller changes in appetite sensations to a standard meal, in men reporting an average of < 7 h of sleep/night *vs.* those reporting at least 7 h of sleep/night, with no differences in SQ between sleep timing groups (sleep timing midpoint > 2h30 *vs.* ≤ 2h30)⁽¹⁴⁾.

Taken together, these results suggest that individuals subjected to sleep restriction have an enhanced susceptibility to food cues. Furthermore, inter-individual variations in habitual sleep architecture, or changes in sleep stage durations to partial sleep restriction, may be linked to appetite and food reward. However, it is unknown whether partial sleep restriction combined with altered bed or wake-times, which are expected to alter sleep architecture^(10, 11), are associated with appetite and food reward. The objective of the present study is to evaluate the effects of a 50% sleep restriction held during the first or second half of an habitual sleep period on appetite sensations and food reward. It is hypothesized that sleep restriction with an advanced wake-time will lead to greater appetite and food reward. It is also hypothesized that these changes in appetite and food reward will be associated with changes in REM sleep duration between the control and advanced wake-time sessions.

Materials and Methods

Participants

Twenty-two participants who corresponded to all inclusion criteria were recruited. However, only 18 completed all sessions (12 men and 6 women; age: 23 ± 4 years; BMI: 22.7 ± 2.7 kg/m²; body fat percentage: $18.8 \pm 10.1\%$). Study methodologies are described in more detail elsewhere ⁽¹⁵⁾. Briefly, participants were between the ages of 18-45 years, non-smokers, weight stable (± 4 kg) within the last 6 months, did not have heart problems or diabetes, did not take medication which may affect appetite or sleep, and reported not performing shift work nor taking regular daytime naps. They also reported having a habitual sleep duration of 7-9 h/night. Only women taking monophasic, combined estrogen-progesterone birth control pills were recruited in order to control for the effects of menstrual cycle phase and sex-steroid hormones on sleep parameters ⁽¹⁶⁾ and food reward ⁽¹⁷⁾. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and the University of Ottawa ethics committee approved all procedures involving human participants. Written informed consent was obtained from all participants.

Design and Procedure

This study followed a randomized crossover design, which included a preliminary session, 2 weeks of sleep-wake monitoring with accelerometry (SenseWear Pro 3 Armbands[®], HealthWear Bodymedia, Pittsburgh, PA, USA) and sleep diaries under free-living conditions, 2 habituation nights (1 in-lab and 1 outside of the lab) and 3 randomized experimental sessions (control with an habitual bed- and wake-time, 50% sleep restriction with an habitual bedtime and advanced wake-time, and 50% sleep restriction with a delayed bedtime and habitual wake-time). During the preliminary session, anthropometric data were collected and participants were given

ad libitum access to a standard breakfast, which included whole-wheat toast, strawberry jam, peanut butter, cheddar cheese and orange juice. Participants were also asked to write down their favorite snack and fruit/vegetable that would be used in the relative-reinforcing value (RRV) of a preferred food task ⁽¹⁸⁾ during each of the experimental conditions. Lastly, participants rated 202 food images that were divided into 4 categories according to fat content and taste (high-fat savory, low-fat savory, high-fat sweet, low-fat sweet) based on the following question: "How often do you consume this food item?". The 4 highest-rated food items within each category were then used to personalize the computer-based behavioral procedure called the Leeds Food Preference Questionnaire (LFPQ) ⁽¹⁹⁾ that was administered during each experimental session. Hence, the food items presented during this task may have differed between participants, but were standardized across sessions for the same participant.

Also personalized to each participant was the prescribed bed- and wake-times for the 3 experimental sessions, which were based on the accelerometry data collected over 2 weeks following completion of the preliminary session. The mean bed- and wake-times for each participant was used to prescribe the time in bed for the control session, whereas the mean sleep midpoint was used to determine the advanced wake-time or delayed bedtime in the sleep restriction conditions. Hence, the assigned bed- and wake-times, as well as the timing of measurements the following morning, differed between participants but were standardized for each participant across sessions. A washout period of at least 7 days separated each experimental session.

Evening and overnight procedures and measurements

Each experimental session began 3 h prior to the set bedtime to allow enough time to place all the electrodes (≈ 90 min), set up the polysomnogram (≈ 30 min) and allow for some

downtime before going to bed (≈ 60 min). EEG (C3, C4, O1, O2, F3 and F4), EMG (bipolar submental) and EOG were recorded on a Medipalm 22 (Braebon Medical Corporation, Kanata, Ontario, Canada) with the Pursuit Sleep Software (Braebon Medical Corporation, Kanata, Ontario, Canada). This setting was used to assess sleep inside the lab during each experimental session. Recordings were scored independently by 2 researchers according to the AASM 2007 criteria⁽²⁰⁾ using 30-second epochs, and discrepancies were resolved by mutual agreement. When forced to remain awake during the night and the following morning, participants were allowed to take part in any type of sedentary activity (*e.g.* reading, watching movies) as long as they remained inside the lab with the evaluator.

Next morning procedures and measurements

Upon awakening, participants were given 1 h to shower. Body weight (HR-100; BWB-800AS, Tanita Corporation, Arlington Heights, IL, USA) and fasting appetite sensations were measured at the same clock time each morning. This took place prior to breakfast consumption, which contained the exact quantity and composition of the breakfast consumed during the preliminary session for each participant. Fasting and post-meal (measured every 30 minutes for 3 h following breakfast) appetite sensations were recorded with 100-mm computerized visual analogue scales (VAS)⁽²¹⁾. The following 4 questions were asked at every time point: desire to eat ("How strong is your desire to eat?": very weak - very strong), hunger ("How hungry do you feel?"; Not hungry at all - As hungry as I have ever felt), fullness ("How full do you feel?"; Not full at all - Very full), and prospective food consumption (PFC) ("How much food do you think you could eat?"; Nothing at all - A large amount). Post-meal area under the curve (AUC) was calculated with the trapezoid method, as previously described⁽²²⁾, and included appetite measurements taken at 0, 30, 60, 90, 120, 150 and 180 minutes post-breakfast intake.

Fasting and mean post-meal appetite sensations over 180 minutes were also used to calculate the SQ for each appetite sensation question using the following equation ⁽²³⁾:

$$\text{SQ (mm/100kcal)} = \frac{[\text{fasting appetite sensation (mm)} - \text{mean post meal appetite sensation (mm)}]}{\text{energy content of the breakfast (kcal)}} \times 100$$

The SQ calculation for the fullness rating is reversed (*i.e.* mean post-meal fullness rating - fasting fullness rating). A mean SQ score including the 4 appetite ratings was also calculated. This SQ calculation has shown good reliability when assessed under controlled lab conditions (intra-class correlation coefficient of $r = 0.7$ for mean SQ) ⁽²⁴⁾ over 60 minutes post-breakfast intake. A higher SQ score indicates a greater satiety response to the breakfast ⁽²⁴⁾.

The RRV of food task ⁽¹⁸⁾ was administered 180 minutes following breakfast intake. This task measures the number of responses for a preferred snack item *vs.* a preferred fruit/vegetable using a fixed ratio of reinforcement for each item, hence providing a measure of the participants' "wanting" to gain access to a preferred item. Before initiating the task, participants were given 10 grams of each preferred item to consume, which acted as a primer. Briefly, this computer-based task allows participants to earn points towards receiving the preferred snack or preferred fruit/vegetable with a slot machine game that contained 3 boxes with different colored shapes. There was 1 slot machine game associated with each item, and when the left button on the mouse was pressed, the shapes changed. When all of the colored shapes matched, the participants earned a point towards that item. For every 5 points earned, the participants received access to 25 grams of that item. This task ran for a total of 2 minutes. The food items earned were then given to the participants during their *ad libitum* lunch, and the amount of each item consumed was determined by weighing the food before and after lunch.

The LFPQ⁽¹⁹⁾ was completed at 60- and 180-minutes post-breakfast consumption, as well as following *ad libitum* lunch intake. This task provides measures of the wanting and liking for an array of food images divided into 4 categories according to fat content and taste (high-fat savoury, low-fat savoury, high-fat sweet and low-fat sweet). A total of 16 different food items (*e.g.* banana, cucumber, chocolate cake, beef slices) formed the array for this task for each participant, and were chosen according to personal preference/familiarity during the preliminary session. The questions and scoring methods used to assess the implicit wanting, explicit wanting and explicit liking for the different food images in this task are described elsewhere^(19, 25). Briefly, during the forced choice part of the test, each food image was presented with every other image in turn. The participants were instructed to select the food they “most want to eat now” during each trial. A standardized implicit wanting score for each food category was calculated as a function of the reaction time in selecting a certain food adjusted for the frequency of choice for each category⁽²⁵⁾. Participants were also asked to rate the extent to which they “liked” or “wanted” each randomly presented food item with a 100-mm visual analogue scale, which were used as a measure of explicit liking and wanting, respectively. Bias scores were calculated for all food reward measurements; the mean low-fat scores were subtracted from the mean high-fat scores (fat content bias) and the mean savory scores were subtracted from the mean sweet scores (taste bias). Positive scores indicate a preference for high-fat or sweet foods, negative values indicate a preference for low-fat or savory foods, and a score of 0 indicates an equal preference for both fat content and taste categories.

Lastly, an *ad libitum* lunch was selected by the participants from a validated food menu⁽²⁶⁾. Briefly, participants were instructed to consume "as much or as little as you want" from the foods that they selected from the menu. They were also told to take the time needed to consume

lunch, which was monitored. Energy and macronutrient intakes were assessed by weighing each food item before and after lunch consumption.

Statistical analyses

Statistical analyses were performed using SPSS (version 17.0; SPSS Inc., Chicago, IL, USA). Two-way repeated measures ANOVA tests were used to determine the main effects of sleep condition (control, 50% sleep restriction with advanced wake-time and 50% sleep restriction with delayed bedtime) and time (60 and 180 minutes post-breakfast consumption, and after lunch) on LFPQ food reward measurements. One-way repeated measures ANOVA tests were used to determine the main effects of sleep condition (control, 50% sleep restriction with advanced wake-time and 50% sleep restriction with delayed bedtime) on appetite sensations, the SQ, *ad libitum* energy and macronutrient intakes over lunch, and the RRV responses (button presses) to the preferred snack and fruit/vegetable and the intake of these items. The Wilcoxon Signed Ranks Test was used to assess potential differences between sessions for variables that were not normally distributed according to the Shapiro-Wilk test. For normally distributed data, *post-hoc* tests with LSD adjustments were used to determine where significant differences existed. Bivariate Spearman correlations assessed the strength of the associations between changes in absolute sleep stage durations (minutes) with changes in appetite, mean SQ, LFPQ food reward measurements assessed 60 minutes post-breakfast intake, *ad libitum* EI, and RRV of food button presses between sessions (delta control-advanced wake-time, control-delayed bedtime, advanced wake-time-delayed bedtime). Values are presented as means \pm standard deviations. Differences with *P*-values < 0.05 were considered statistically significant.

Results

As previously reported⁽¹⁵⁾, absolute stage 1 (18±9, 7±4, 4±3 min; $P = 0.0001$; partial $\eta^2 = 0.66$), stage 2 (245±35, 113±29, 101±31 min; $P = 0.0001$; partial $\eta^2 = 0.95$) and REM (108±24, 34±7, 51±17 min; $P = 0.0001$; partial $\eta^2 = 0.91$) sleep durations were greater during the control vs. both sleep restriction conditions. Stages 1 and 2 sleep durations were also greater, and REM sleep duration was lower, during the advanced wake-time vs. delayed bedtime session ($P = 0.01, 0.04$ and 0.0001 , respectively). SWS was only greater during the control vs. advanced wake-time session (92±32, 76±33, 80±31 min; $P = 0.02$; partial $\eta^2 = 0.20$). Lastly, there were no differences in body weight (69.2±9.2, 69.4±9.3, 69.2±9.4 kg; $P = 0.72$; partial $\eta^2 = 0.02$) between sessions⁽¹⁵⁾.

The fasting, post-meal AUC and SQ for each appetite sensation, as well as *ad libitum* energy and macronutrient intakes during lunch are presented in **Table 1**. Fasting desire to eat and hunger ratings were greater during the advanced wake-time compared to the control and delayed bedtime sessions. Greater hunger and PFC, and lower fullness AUC values were also noted during the advanced wake-time vs. control session. No other significant differences, including all SQ variables and *ad libitum* intakes, were noted between sessions.

Greater stage 1 and stage 2 sleep durations between the control and delayed bedtime sessions were associated with higher fasting fullness ratings ($r = -0.48$; $P = 0.04$) and lower mean SQ ($r = -0.58$; $P = 0.01$) scores, respectively. Greater stage 1, and lower REM, sleep durations were also correlated with greater post-meal hunger (stage 1 sleep: $r = 0.52$; $P = 0.03$, REM sleep: $r = -0.56$; $P = 0.02$) and PFC (stage 1 sleep: $r = 0.55$; $P = 0.02$, REM sleep: $r = -0.50$; $P = 0.04$), when comparing both sleep restriction conditions. No other significant correlations were noted

between changes in sleep stage durations with delta appetite, mean SQ and *ad libitum* EI between sessions (results not shown).

The fat and taste bias scores for the implicit wanting, explicit wanting and explicit liking for foods assessed at 60 and 180 minutes post-breakfast, as well as after lunch are presented in **Table 2**. Greater explicit liking and wanting for high-fat relative to low-fat foods were noted during the advanced wake-time compared to the control session (**Figure 1**). When analysing the effect of *time*, independently of sleep conditions, on LFPQ measures, the explicit wanting for high- relative to low-fat foods was greater after lunch compared to 60 minutes post-breakfast consumption ($P = 0.01$). The explicit liking ($P = 0.01$), explicit wanting ($P = 0.004$) and implicit wanting ($P = 0.0001$) for sweet relative to savory foods were greater at 60 vs. 180 minutes post-breakfast consumption. The explicit liking for sweet relative to savory foods was greater prior to lunch compared to following lunch ($P = 0.04$), whereas the implicit wanting for sweet relative to savory foods was greater after lunch compared to before lunch ($P = 0.03$). No significant correlations between changes in LFPQ at 60 minutes following breakfast and delta sleep stage durations were noted between sessions (results not shown).

Results from the RRV of food task are presented in **Table 3**. No differences in snack, fruit and total button presses, as well as in the intake of these preferred foods, were noted between sessions. No significant correlations between changes in RRV button presses and delta sleep stage durations were noted between sessions (results not shown).

Discussion

To our knowledge, this is the first study to investigate changes in appetite and food reward in response to partial sleep restriction combined with altered sleep timing. Furthermore, the present study assesses the associations between these outcome variables with sleep stage durations, in addition to exerting optimum control over inter-individual circadian rhythms by personalizing each participant's assigned bed- and wake-times. Collectively, our findings suggest that most fasting and post-meal appetite ratings are greater following partial sleep restriction with an advanced wake-time compared to the control and partial sleep restriction with a delayed bedtime conditions. The explicit liking and wanting for high- relative to low-fat foods were greater during the advanced wake-time compared to the control session. These results corroborate our initial hypothesis. However, these changes in appetite and food reward did not lead to greater EI during an *ad libitum* lunch during this session. No differences in SQ and RRV of preferred food responses were noted between sessions. Changes in REM sleep between the control and advanced wake-time sessions were not associated with changes in appetite and food reward, which rejects our second hypothesis. However, lower REM sleep duration was associated with greater post-meal appetite ratings between both sleep restriction conditions.

These results first suggest that partial sleep restriction with an advanced wake-time leads to greater subjective appetite sensations and explicit food reward for high- relative to low-fat foods. These results corroborate previous studies reporting greater hunger and/or greater activation in food-sensitive centers of the brain following partial sleep restriction⁽⁴⁻⁷⁾. These results also suggest that sleep timing may have an independent effect on appetite sensations and food reward. A different study completed in our lab assessed habitual sleep parameters under free-living conditions following acute exercise interventions, and revealed that lower sleep

duration and earlier wake-times were associated with greater food reward the next morning ⁽²⁷⁾. However, an important confounder in these findings was the elapsed time between measured wake-time and completion of the food reward task, which was standardized across sessions for all participants. It is thus possible that the greater elapsed time between the end of the sleep period and completion measurements, which was kept stable for the same participant across sessions, may have influenced the observed results.

The ability to modulate EI with higher cognitive processes, even when presented with a physiological "need" or greater "wanting" for food ⁽¹⁾, may in part explain the observed lack of association between appetite and food reward with actual EI during an *ad libitum* lunch. Hence, it is possible that the severity of the sleep interventions imposed in the present study (1 night of 50% sleep restriction) may not be sufficient, or prolonged enough (held over several days) to reduce executive control over EI, despite greater feelings of hunger and food reward, in these participants. Furthermore, the non-significant differences in the SQ and the RRV of preferred food responses do not corroborate the appetite and LFPQ explicit wanting findings, respectively. First, the calculation of the SQ is based on changes in appetite ratings between pre- and post-standard meal intake, which will be similar between sessions if the appetite scores are comparable pre- vs. post-meal intake, independently of the appetite rating value. Second, the differences in food "wanting" findings between tasks may be in part influenced by the forced- vs. voluntary-choice response protocols utilized by each tool. If the imposed intervention is not sufficient to alter executive control over voluntary food-selection responses and EI, it is possible that the protocol utilized by the RRV of food task may better reflect the voluntary choices (or changes) in EI following an intervention.

Greater stage 1 sleep duration between the control and delayed bedtime sessions was associated with greater fullness ratings. Despite no differences in the SQ between sessions, greater stage 2 sleep duration was also associated with lower mean SQ between the control and delayed bedtime sessions. These associations may be in part driven by the greater amount of sleep time during the control session. However, the negative association between changes in stage 2 sleep duration with changes in mean SQ are counterintuitive, as lower SQ scores are indicative of a reduced satiety efficiency to the standard meal. *Post-hoc* Spearman correlations revealed a negative association between stage 2 and SWS durations during the delayed bedtime session ($r = -0.59$; $P = 0.01$). Although a mere hypothesis, participants with greater SWS, and consequently lower stage 2 sleep, durations during this session may have a greater SQ the following morning. Hence, SWS would have an indirect effect on SQ through reductions in stage 2 sleep duration. In addition to these findings, greater stage 1, and lower REM, sleep durations were associated with greater appetite sensations between both sleep restriction conditions. These findings add to those previously reported by St-Onge *et al.* ⁽⁶⁾, a study noting that individuals with smaller reductions in REM sleep following partial sleep restriction had reduced changes in insula activation ⁽⁶⁾. Furthermore, Shechter *et al.* noted a negative association between REM sleep time and hunger ratings ⁽¹³⁾, which also supports our findings.

A shift in food preference occurred over time within each session, from a preference for low-fat sweet foods earlier in the morning towards a preference for high-fat savory foods around lunch. Appetite for savory foods varies according to changes in feelings of hunger, whereas preferences for sweet tasting foods tend to be more stable throughout the day ^(2, 3). Additionally, it is possible that a lower sensory specific satiety (*i.e.* lower pleasantness rating and subsequent intake of a specific food item consumed to satiety) ^(28, 29) for high-fat foods may have occurred

over lunch. In fact, lunch fat intake was $\approx 25\%$ during each session, whereas fat intake during the standard breakfast was $\approx 37\%$. Hence, the shift in preference for high-fat foods may reflect actual fat intake during these meals. These results corroborate a previous study performed in our lab, which showed a similar shift for fat preference across time (*i.e.* greater bias for high-fat, sweet foods following lunch compared to before lunch) ⁽³⁰⁾.

These findings are limited to a small sample size of healthy adults with a very high sleep efficiency ($\approx 93\text{-}97\%$ when assessed inside the lab). This limits generalizability of these findings to individuals with sleep complaints or disorders. All outcomes variables were assessed following 1 night of altered sleep duration and bed or wake-time, which does not account for day-to-day variations in these outcomes, nor can they be compared to studies imposing prolonged sleep restriction. The food images presented during the LFPQ were not the same as those offered on the menu. Likewise, the RRV task was administered prior to an *ad libitum* lunch. These limitations may influence the participants' responses on each of these tasks, and contribute to the observed dissociation between food reward and EI across sessions.

These findings suggest that appetite and food reward may be influenced by alterations in sleep timing, independently of sleep duration. However, a greater amount of time elapsed between awakening and completion of the outcome measurements may influence these results, and would require further investigation. These changes in appetite sensations and LFPQ food reward did not necessarily lead to greater EI during an *ad libitum* lunch. Future studies evaluating prolonged sleep restriction with altered sleep timing on appetite and food reward are needed. Studies are also needed to investigate appetite sensations and food reward in individuals with sleep disorders (*e.g.* insomnia, sleep apnea), as sustained poor sleep quality ($< 80\%$ sleep efficiency) and/or reduced sleep duration may increase their risk of weight gain over time ⁽³¹⁾.

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Conflicts of interest

The authors declare no conflict of interest.

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Table 1. Fasting, post-meal area under the curve and satiety quotient values for each appetite measurement, as well as *ad libitum* energy and macronutrient intakes during lunch (n = 18)

	Control	Advanced wake-time	Delayed bedtime	Main effect analysis
	Mean ± SD	Mean ± SD	Mean ± SD	P value; partial η^2
Fasting Appetite (mm)				
<i>Desire to eat</i>	67 ± 18 ^a	81 ± 18 ^b	68 ± 17 ^a	P = 0.03; partial η^2 = 0.19
<i>Hunger</i>	65 ± 18 ^a	77 ± 16 ^b	64 ± 16 ^a	P = 0.03; partial η^2 = 0.19
<i>Fullness</i>	15 ± 12 ^a	11 ± 13 ^a	19 ± 16 ^a	P = 0.08; partial η^2 = 0.14
<i>Prospective food consumption</i>	68 ± 15 ^a	77 ± 16 ^a	67 ± 11 ^a	P = 0.06; partial η^2 = 0.15
Post-meal Appetite AUC				
<i>Desire to eat</i>	4579 ± 2289 ^a	5991 ± 2045 ^a	5427 ± 2433 ^a	P = 0.05; partial η^2 = 0.17
<i>Hunger</i>	4508 ± 2136 ^a	5982 ± 1781 ^b	5198 ± 2201 ^{a, b}	P = 0.03; partial η^2 = 0.21
<i>Fullness</i>	11753 ± 2687 ^a	9820 ± 2873 ^b	10999 ± 2237 ^{a, b}	P = 0.03; partial η^2 = 0.20
<i>Prospective food consumption</i>	5393 ± 2321 ^a	6830 ± 1794 ^b	6244 ± 2638 ^{a, b}	P = 0.02; partial η^2 = 0.21
Satiety Quotient (mm/100 kcal)				
<i>Desire to eat</i>	5.6 ± 3.8 ^a	6.4 ± 4.0 ^a	5.3 ± 3.7 ^a	P = 0.64; partial η^2 = 0.03
<i>Hunger</i>	5.4 ± 3.5 ^a	5.6 ± 2.9 ^a	4.7 ± 2.8 ^a	P = 0.65; partial η^2 = 0.03
<i>Fullness</i>	7.0 ± 2.9 ^a	5.6 ± 2.4 ^a	5.6 ± 2.4 ^a	P = 0.12; partial η^2 = 0.12
<i>Prospective food consumption</i>	5.2 ± 2.1 ^a	5.2 ± 2.8 ^a	4.4 ± 1.9 ^a	P = 0.42; partial η^2 = 0.05
<i>Mean</i>	5.8 ± 2.8 ^a	5.7 ± 2.7 ^a	5.0 ± 2.4 ^a	P = 0.56; partial η^2 = 0.03
Lunch Intake				
<i>Energy intake (kcal)</i>	627 ± 258 ^a	682 ± 227 ^a	707 ± 323 ^a	P = 0.40; partial η^2 = 0.05
<i>Carbohydrate intake (kcal)</i>	383 ± 182 ^a	407 ± 151 ^a	430 ± 228 ^a	P = 0.54; partial η^2 = 0.04
<i>Fat intake (kcal)</i>	157 ± 99 ^a	169 ± 91 ^a	179 ± 78 ^a	P = 0.55; partial η^2 = 0.04
<i>Protein intake (kcal)</i>	95 ± 53 ^a	111 ± 52 ^a	108 ± 61 ^a	P = 0.26; partial η^2 = 0.08
Lunch intake time (minutes)	15 ± 6 ^a	18 ± 6 ^a	17 ± 6 ^a	P = 0.21; partial η^2 = 0.09

Note: Means not sharing the same letter are significantly different from each other ($P < 0.05$).

AUC, area under the curve; kcal, kilocalories; mm, millimeter; SD, standard deviation

Table 2. The implicit wanting, explicit wanting and explicit liking for high- relative to low-fat foods, and sweet relative to savoury foods between conditions, across time (60 and 180 minutes post-breakfast intake, and after lunch), and condition*time interactions (n = 18).

	Control	Advanced wake-time	Delayed bedtime	Condition effect	Time effect	Condition*Time interaction
	Mean ± SD	Mean ± SD	Mean ± SD	P value; partial η^2	P value; partial η^2	P value; partial η^2
<i>Implicit wanting</i>						
<u>Fat bias</u>				$P = 0.33$; partial $\eta^2 = 0.06$	$P = 0.52$; partial $\eta^2 = 0.04$	$P = 0.76$; partial $\eta^2 = 0.03$
60 min after breakfast	-33±25.9	-21.0±30.0	-26.4±34.6			
180 min after breakfast	-24.8±29.6	-20.7±45.2	-19.9±40.1			
After lunch	-33.6±28.1	-25.8±43.5	-24.1±35.2			
<u>Taste bias</u>				$P = 0.98$; partial $\eta^2 = 0.001$	$P = 0.01$; partial $\eta^2 = 0.27$	$P = 0.88$; partial $\eta^2 = 0.02$
60 min after breakfast	29.2±35.5	25.7±43.9	33.1±40.3			
180 min after breakfast	6.7±48.8	5.0±47.7	4.7±49.6			
After lunch	27.7±48.5	30.8±37.7	27.5±42.7			
<i>Explicit wanting</i>						
<u>Fat bias</u>				$P = 0.02$; partial $\eta^2 = 0.20$	$P = 0.01$; partial $\eta^2 = 0.24$	$P = 0.11$; partial $\eta^2 = 0.10$
60 min after breakfast	-13.2±14.1	-4.3±9.7	-9.3±15.5			
180 min after breakfast	-12.2±18.2	-4.9±13.9	-6.9±17.6			
After lunch	-2.4±5.6	-1.4±5.8	-1.5±8.3			
<u>Taste bias</u>				$P = 0.16$; partial $\eta^2 = 0.10$	$P = 0.03$; partial $\eta^2 = 0.19$	$P = 0.50$; partial $\eta^2 = 0.05$
60 min after breakfast	8.4±11.3	6.5±17.2	11.1±15.9			
180 min after breakfast	-1.9±15.4	1.9±20.6	3.3±18.6			
After lunch	1.9±6.6	5.5±8.8	5.8±12.3			
<i>Explicit liking</i>						
<u>Fat bias</u>				$P = 0.01$; partial $\eta^2 = 0.25$	$P = 0.08$; partial $\eta^2 = 0.14$	$P = 0.14$; partial $\eta^2 = 0.10$
60 min after breakfast	-10.6±13.1	-2.1±8.8	-7.7±15.6			
180 min after breakfast	-9.2±14.6	-1.6±13.8	-4±13.8			
After lunch	-3.7±7.2	-1.2±6.5	-1.3±9.9			
<u>Taste bias</u>				$P = 0.25$; partial $\eta^2 = 0.08$	$P = 0.03$; partial $\eta^2 = 0.18$	$P = 0.62$; partial $\eta^2 = 0.04$
60 min after breakfast	9.9±14.5	8.0±17.0	-2.1±8.8			
180 min after breakfast	2.1±15.8	4.1±20.0	-1.6±13.8			
After lunch	3.9±11.4	7.1±10.5	-1.2±6.5			

Note: A positive score indicates a relative preference for high- relative to low fat, or sweet relative to savoury, foods. A negative score indicates a relative preference for low- relative to high-fat, or savoury relative to sweet, foods. A score of 0 indicates an equal preference between fat and taste categories. SD, standard deviation.

Table 3. The relative reinforcing value of a preferred food (number of button presses), and the intake of each of these food items during each session (n = 18)

	Control	Advanced wake-time	Delayed bedtime	Main effect analysis
	<i>Mean ± SD</i>	<i>Mean ± SD</i>	<i>Mean ± SD</i>	<i>P value; partial η^2</i>
Preferred snack responses (button presses)	47 ± 69	48 ± 52	35 ± 40	<i>P = 0.57; partial $\eta^2 = 0.03$</i>
Preferred fruit responses (button presses)	92 ± 82	67 ± 52	62 ± 37	<i>P = 0.08; partial $\eta^2 = 0.14$</i>
Total responses (button presses)	139 ± 139	115 ± 95	97 ± 64	<i>P = 0.25; partial $\eta^2 = 0.08$</i>
Preferred snack intake (kcal)	80 ± 121	75 ± 90	55 ± 77	<i>P = 0.50; partial $\eta^2 = 0.04$</i>
Preferred fruit intake (kcal)	34 ± 30	27 ± 24	30 ± 28	<i>P = 0.68; partial $\eta^2 = 0.02$</i>
Total preferred food intake (kcal)	113 ± 144	102 ± 102	85 ± 86	<i>P = 0.58; partial $\eta^2 = 0.03$</i>

Note: kcal, kilocalories; SD, standard deviation

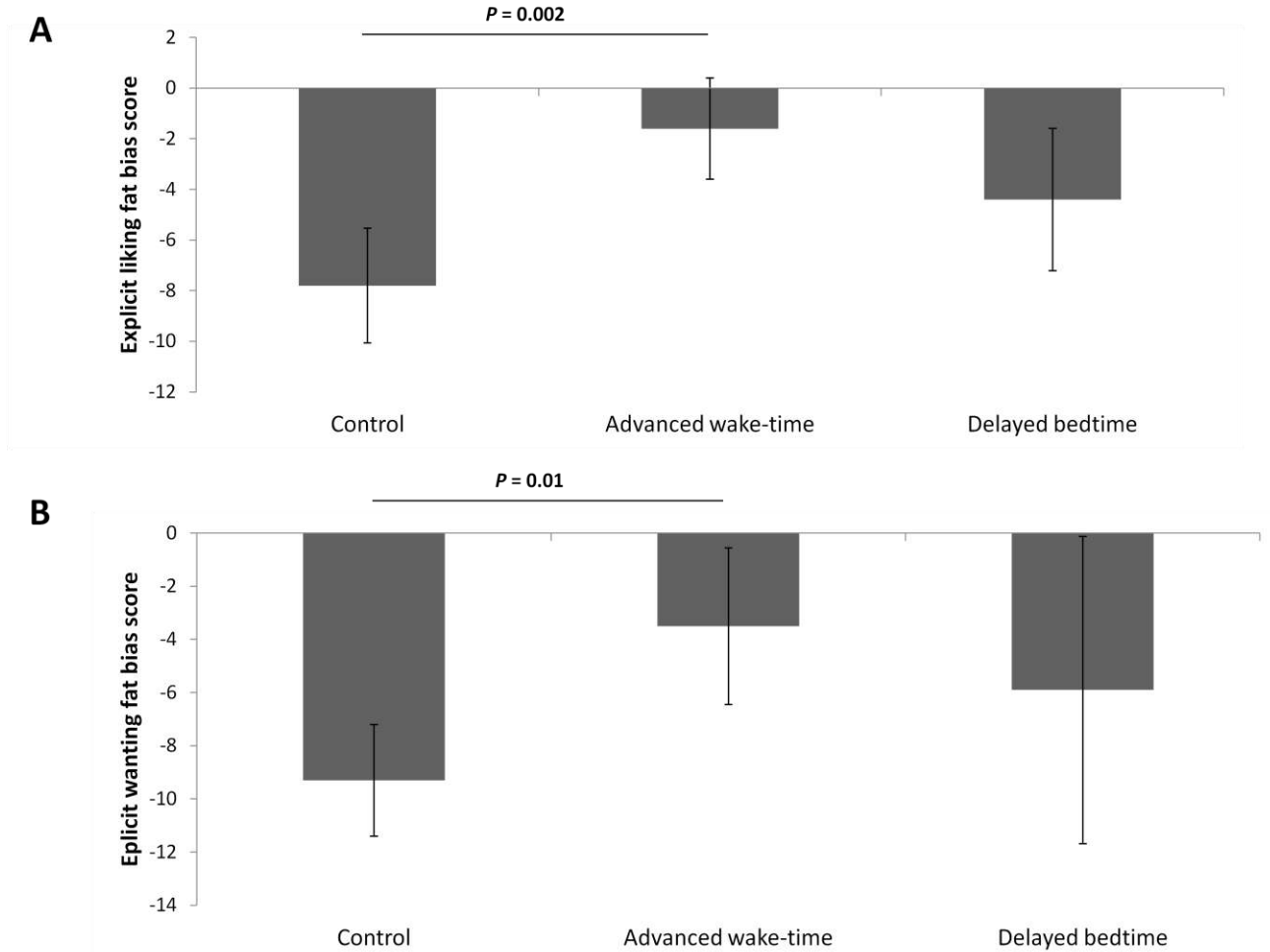


Figure 1. The explicit liking (A) and explicit wanting (B) for high- relative to low-fat foods during the 3 experimental sessions. Values are presented as means for 18 participants with standard errors of the mean represented by vertical bars.

Note: A positive score indicates relatively greater explicit liking/wanting for high vs. low- fat foods. A negative score indicates a relatively greater explicit liking/wanting for low- vs. high-fat foods. A score of 0 indicates an equal explicit liking/wanting score between fat categories.

CHAPTER 4: THESIS DISCUSSION

4.1 Summary

The majority of experimental studies^(16-20, 22, 23) suggest that imposed partial sleep restriction leads to greater 24 h EI, when compared to a control sleep duration condition. However, the effects of reduced sleep duration on EE are not as consistent, with some studies reporting no changes^(16, 20, 23), higher^(18, 24) or lower⁽²¹⁾ EE following imposed sleep restriction. Inter-individual differences in habitual PA participation have been previously suggested to be an important confounding factor when explaining these discrepancies in results⁽⁷⁴⁾. Hence, individuals with habitually high levels of PA participation may resort to exercise (or moderate-to-vigorous intensity PA) to combat fatigue in contexts where their PA participation is not limited.

Although the effects of reduced sleep duration on EI and EE have been extensively investigated, the effects of altered sleep timing, independently of sleep duration, on measures of the energy balance requires further investigation. It is unknown whether imposed alterations in sleep architecture associated with differing bed- or wake-times, independently of sleep duration, has an effect on appetite, food reward and the energy balance. Therefore, the present thesis sought to answer 3 specific questions on the independent effects of sleep duration and sleep timing on measures of appetite, food reward and/or the energy balance by conducting 3 different studies and utilizing different methods and procedures:

- 1) Do individuals with habitual short sleep duration, poor sleep quality and a later bedtime have a lower SQ, and consequently greater EI?
- 2) Are variations in sleep duration, sleep efficiency and sleep timing related to changes in next day food reward?

3) Does a 50% sleep restriction anchored during the first or second half of the night alter next day appetite ratings, SQ, food reward and energy balance (EI and EE)? Are potential changes in these outcomes related to changes in sleep architecture?

Table 1 summarizes the results of the studies performed to answer these research questions. These results are discussed in each of the four articles presented in Chapter 3. The following section will consider the broader implications of the sum of results presented in this thesis while also discussing future directions for this area of research.

Table 1. Summary of main thesis findings

Study 1 (Thesis article #1)	<ol style="list-style-type: none"> 1. There were no significant associations between SQ scores and all sleep parameters. 2. A lower mean SQ was noted in habitual short-duration sleepers. No differences in the SQ were noted between higher and lower sleep quality, and earlier and later sleep timing midpoint groups.
Study 2 (Thesis article #2)	<ol style="list-style-type: none"> 1. Short sleep durations and earlier wake-times were associated with greater food reward the next morning. However, these associations were driven by elapsed time between awakening and completion of the food reward task.
Study 3 (Thesis articles #3 and #4)	<ol style="list-style-type: none"> 1. Sleep restriction with a delayed bedtime led to greater active EE and moderate-intensity PA time on day 1, as well as greater energy and carbohydrate intakes on day 2 and over 36h. Vigorous-intensity PA time was greater following sleep restriction with an advanced wake-time <i>vs.</i> sleep restriction with a delayed bedtime. 3. Greater sleep quality and SWS duration were associated with lower energy and macronutrient intakes, and greater vigorous-intensity PA time, respectively, between sleep restriction conditions. 4. Greater appetite sensations and explicit liking and wanting for high- relative to low-fat foods were noted following partial sleep restriction with an advanced wake-time. No differences in <i>ad libitum</i> lunch EI. 5. Greater light sleep duration was associated with greater fasting fullness ratings and lower mean SQ. Greater stage 1 sleep, and lower REM sleep, durations were related to greater post-meal appetite ratings.

4.2 Implications of the present findings and future directions

Studies 1 and 2 provided preliminary findings that contributed to determining whether the SQ, a valid measure of satiety efficiency, and food reward may be altered by habitual sleep parameters. Furthermore, these findings provided an important rationale for the construct and design of Study 3.

Epidemiologic evidence suggests that short sleep duration (generally characterized as a habitual sleep duration < 7 h/night) is associated with weight gain over time⁽⁷⁵⁻⁷⁷⁾. Considering these associations between reduced sleep duration and weight gain over time in longitudinal studies, and assuming that sustained short sleep duration is the cause of this weight gain, it is expected that reduced sleep duration would lead to greater EI and/or lower EE, thus resulting in a positive energy balance. Experimental studies imposing partial sleep restrictions^(16-20, 22, 23), including Study 3 in the present thesis, do suggest that reducing sleep duration increases EI. However, these studies are often conducted in healthy individuals with habitual sleep durations within the recommended ranges of 7-9 hours per night, and are only sustained over the short-term (\approx 1- 14 nights). Imposing a long-term sleep restriction protocol in lean individuals to evaluate whether this intervention would lead to weight gain over time would not be feasible for ethical and logistical reasons⁽⁷⁴⁾. Consequently, the conclusions on the short sleep-obesity link must be based on short-term intervention studies and epidemiologic evidence. The accumulation of short-term intervention studies do suggest that sleep restriction is related to higher EI^(16-20, 22, 23), as well as short-term reductions in glucose tolerance, insulin sensitivity and/or variations in appetite-regulating hormones (*e.g.* ghrelin, leptin)^(59, 61, 78, 79). When these results are considered alongside consistent epidemiologic evidence of an association between short-sleep duration with obesity/weight gain^(75, 76, 77, 80) and weight loss success^(81, 82), it is plausible to suggest that sleep

plays an important role in weight regulation and cardio-metabolic risk factors. Although randomized controlled trials cannot be used to identify lack of sleep as a direct cause of obesity or weight gain, a randomized control trial that is currently underway is evaluating the effects of a 30-60 minute/night sleep extension on different endocrine, metabolic and psychological parameters in obese individuals with sleep durations that are habitually below 6.5 hours/night⁽⁸³⁾. Preliminary results from this study suggest that extending sleep duration by an average of 30 minutes per night in these individuals led to anecdotal reports of a better mood and ability to focus, reduced sleepiness during the day, more willingness to exercise, as well as a lower caffeine intake and less cravings for sweet and salty foods during the evening⁽⁸³⁾. Once completed, this study will provide meaningful clinical evidence as to the implementation or promotion of sleep extension as a means of potentially improving weight loss success or preventing weight gain over time.

Discrepancies in sleep duration findings according to the analyses performed were present in Study 1, as the SQ was only different between sleep duration groups but not linearly associated with sleep duration. These differences in findings are common in epidemiologic studies assessing the link between sleep duration with obesity and/or cardio-metabolic risk factors^(75, 80, 84-87), and may be in part explained by the U- or J-shaped associations often reported between sleep duration and fat mass^(75, 80) and cardio-metabolic risk factors⁽⁸⁴⁻⁸⁷⁾. However, in Study 1, a visual inspection of the association between mean SQ and sleep duration revealed no clear association between these variables (**Figure 1**). This may be in part explained by the small variance in sleep duration times within this population (*i.e.* 95% of participants reported a habitual sleep duration between 5 and 8 h/night).

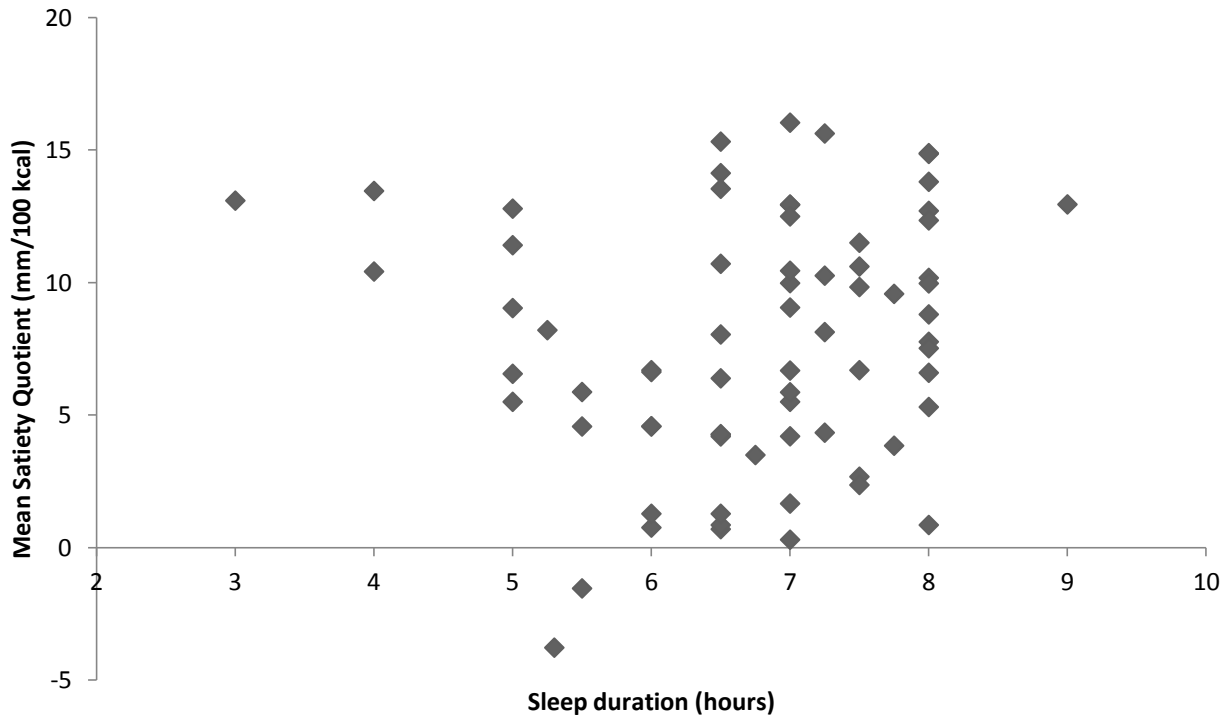


Figure 1. The association between self-reported sleep duration and mean satiety quotient from Study 1.

Future large-scale epidemiologic studies measuring sleep duration should consider this potentially U- or J-shaped association when conducting statistical analyses, as a lack of linear association between sleep duration with obesity, health outcomes and/or energy balance measurements does not necessarily imply that these variables do not vary according to this sleep parameter.

An important confounder in a number of in-laboratory studies, including those presented in the current thesis, could be the elapsed time between awakening and the completion of tasks or measurements the following morning (*e.g.* VAS, food reward tasks, or EI). It is possible that individuals with habitually earlier wake-times may express greater drives towards food in the morning, compared to individuals who usually wake later. Additionally, if comparing individuals

with similar daily routines/schedules, it may also be hypothesized that those with habitually earlier wake-times may not eat breakfast the moment they awake, as they have more time to accomplish different tasks in the morning, compared to those who wake later and may have adopted a habit of eating breakfast upon awakening because of lack of time. These effects may also be amplified when wake-time is advanced and/or the elapsed time between awakening and breakfast intake are greater, as was shown in the present thesis. Studies are needed to further investigate the independent effects of the elapsed time spent awake, independently of sleep parameters, on appetite and energy balance. If found to be an independent predictor of these outcomes, future studies should opt to control for the participants' wake-times, rather than standardize the time of outcome measurements across participants. Additionally, the studies presented in this thesis did not assess the distribution of EI throughout the day, and did not permit food intake during the hours that participants were forced to remain awake in Study 3. If *ad libitum* food intake would have been permitted during these overnight hours, EI would have most likely been higher. Indeed, greater 24-h EI following imposed sleep restriction in previous studies were often characterized by greater late-night and/or post-dinner snack intake during the sleep restriction condition^(19, 20, 22). Hence, the timing of food intake should be monitored in future studies, as this may influence, or help to further explain, the sleep-EI link⁽⁸⁸⁾.

An important feature of the present thesis is the independent assessment of sleep timing, which has been overlooked in previous sleep restriction studies^(16-21, 23, 24), as they have often imposed a sleep period with a combined delayed bedtime and advanced wake-time. Later sleep timing midpoints were previously associated with greater late-night EI⁽⁷²⁾ and reduced moderate-to-vigorous PA participation⁽⁷³⁾ under free-living conditions. Although a direct cause cannot be implied based on these associations, it may be hypothesized that a preference towards sedentary

activities and energy-dense food intake may be more common during evening hours as opposed to the morning. Taking into consideration the influence of the circadian rhythm on sleep architecture^(14, 31, 32), imposing changes in bed- and/or wake-time without necessarily altering the time spent in bed will influence sleep architecture. Indeed, results from Study 3 highlighted a small difference in SWS between both sleep restriction conditions (≈ 5 minutes), whereas REM sleep was ≈ 18 minutes greater during sleep restriction with a delayed bedtime *vs.* an advanced wake-time. Additionally, participants with greater sleep efficiency and SWS duration between sleep restriction conditions had higher vigorous-intensity PA time and smaller energy and macronutrient intake, whereas lower stage 1 sleep, in exchange for greater REM sleep, durations were associated with greater post-meal hunger ratings. Previous studies have shown strong associations between SWS and REM sleep durations with appetite and energy balance parameters under habitual (control) conditions⁽²⁹⁾, and when assessing changes in sleep architecture between habitual and partial sleep restriction conditions⁽³⁰⁾. Findings from the present thesis add to these previously published results, as participants who optimized their sleep quality during sleep restriction had higher activity levels, and exerted greater control over food intake the following day. These findings have important implications, as inter-individual differences in sleep quality in response to imposed sleep restriction may impact feelings of appetite and energy balance differently the next day. Simply put, not everyone will experience a decrease in cognitive inhibition, which may lead to changes in EI and/or EE, following sleep restriction⁽⁸⁹⁾. Further characterization of individuals based on personality traits (*e.g.* extraversion, neuroticism)⁽⁹⁰⁾ and cognitive responses (*e.g.* prefrontal cortex and insula activation; responses on psychomotor vigilance tasks)⁽⁸⁹⁾ following sleep restriction would

provide important evidence as to the inter-individual characteristics that may predispose certain individuals to weight gain over time if reduced sleep duration were to be sustained.

Another implication of these findings is whether the changes in SQ and food reward lead to alterations in EI. Overall, the findings from this thesis suggest that changes in food reward, appetite and SQ do not translate into changes in EI. Self-reported measurements of sleep parameters and food intake may not properly capture small changes in EI that could be related to changes in sleep parameters. Furthermore, individuals' abilities to modulate food intake with higher cognitive processes, even when presented with a physiological "need" or greater drive for food intake⁽³³⁾, may in part explain the observed lack of association between appetite, SQ and food reward with food intake. It is possible that a 1-night partial sleep restriction intervention in individuals with very high sleep efficiencies ($\approx 93-97\%$ when measured inside the lab in Study 3) may not be sufficient to reduce executive control over food intake in these individuals. Hence, prolonged sleep restriction protocols may be needed to induce greater feelings of hunger and EI in these individuals. Although the implementation of long-term sleep restriction protocols in healthy individuals with high sleep efficiencies to assess the risk of weight gain over time would not be ethically and logistically feasible, investigating energy balance parameters in individuals with sleep disorders and/or reduced sleep quality would provide clinically meaningful evidence, as these individuals may experience sustained changes in sleep architecture that could promote a positive energy balance and ultimately lead to weight gain over time⁽⁸⁸⁾. Future studies are thus needed to assess objective measures of sleep, EI and EE in individuals with sleep disorders and/or reduced sleep quality to further investigate this hypothesis.

A final implication of the present thesis was the assessment of active EE in Study 3, which provided additional information on how partial sleep restriction combined with altered

sleep timing may affect the energy balance. The lack of control over physical activity participation post-sleep restriction may be seen as a limitation, as variations in activity participation seemed to have had an independent effect on food intake. Conversely, not limiting active EE in Study 3 provided a more accurate, "real-life" measure of PA participation. More specifically, participants in Study 3 may have resorted to greater PA following sleep restriction to combat feelings of fatigue during the day following the intervention (day 1). This may be seen as an evident solution for healthy, active (*i.e.* \approx 15-23% of the time spent performing moderate-to-vigorous intensity PA) individuals, such as those recruited in Study 3. In addition to reporting a positive association between EI and EE in individuals with moderate-to-high levels of habitual PA participation, Mayer *et al.* ⁽⁹¹⁾ also reported a negative association between EE and EI in individuals with greater sedentary time (*i.e.* lower EE coupled with higher EI). Based on these results, it may be hypothesized that individuals with greater habitual sedentary time may have a similar EI as those with moderate-to-high levels of habitual PA, without necessarily increasing their PA time post-sleep restriction. If this were to occur, individuals with greater habitual sedentary time may be at an increased risk of weight gain over time if sleep restriction were to be sustained. Future studies with the aim of comparing energy balance measurements in individuals with different habitual activity levels following sleep restriction and altered sleep timing are needed to evaluate this hypothesis.

Taken together, the results from this thesis suggest that 1) sleep timing has an independent effect on EI and EE; 2) non-restricted PA participation under free-living conditions following sleep restriction influences EI when assessed in healthy and habitually active individuals; 3) appetite, SQ and food reward are influenced by crude sleep parameters (sleep duration, quality and/or timing) and/or sleep architecture, but these alterations do not necessarily

translate into greater EI; and 4) changes in sleep architecture in response to sleep restriction and/or altered sleep timing are related to changes in appetite and energy balance parameters the following day. Although not presented and discussed in the results of the present thesis, inter-individual differences in personality traits and/or cognitive inhibition to sleep restriction may be associated to changes in next day food intake and activity patterns in response to sleep restriction. Future studies are needed to further investigate sleep architecture and energy balance parameters in individuals with sleep disorders (*e.g.* insomnia, sleep apnea), as individuals with sustained poor sleep quality (< 80% sleep efficiency) and/or reduced sleep duration may be at an increased risk of weight gain over time ⁽⁸³⁾. This information would be invaluable given the increasing prevalence of individuals experiencing regular circadian misalignment as a result of shift work, in addition to an increase in the incidences of sleep disorders and/or voluntary sleep restriction as a result of a greater number of work, family and/or social demands ⁽⁹²⁾.

4.3 Thesis conclusions

The main objective was to assess the independent effects of sleep duration and sleep timing on measures of appetite, food reward and energy balance. The findings of greater active EE preceding greater EI following sleep restriction with a delayed bedtime compared to the control session suggest that sleep timing has an independent effect on energy balance parameters. However, the similar degree of change in these parameters (≈ 404 kcal increase in activity EE *vs.* ≈ 470 kcal increase in EI between the control and delayed bedtime sessions over 36 h) may not necessarily lead to changes in body weight over time in healthy active individuals with high degrees of sleep efficiency. Furthermore, the associations between sleep stage time and next day measures of appetite, EI and activity time add to the complexity of the sleep-energy balance link,

as greater SWS duration was associated with greater active EE and reduced EI, whereas greater amounts of REM sleep were correlated with lower post-meal appetite sensations. This area of research requires further investigation, as many studies assessing the association between sleep and obesity/weight gain often only consider crude indicators of sleep quality (*i.e.* sleep duration and sleep efficiency). These findings will inform future studies on the importance of assessing sleep timing and sleep architecture within sleep restriction protocols. Furthermore, these findings will provide evidence for the measurement of SQ, food reward and unrestricted PA participation within sleep and energy balance studies to be conducted in other populations who may be at greater risk of weight gain over time as a result of sustained short-sleep durations, sleep disorders and/or poor sleep quality.

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APPENDIX A: NOTICES OF ETHICAL APPROVAL FOR THESIS STUDIES #1, #2 AND #3



Vice-rectorat à la recherche
Comité d'éthique de la recherche

Sainte-Foy, le 30 août 2004

Monsieur Angelo Tremblay
Laboratoire des sciences de l'activité physique
PEPS
Université Laval

Objet : Projet intitulé Évaluation clinique pour améliorer la prescription d'intervention dans le traitement de l'obésité : les aliments fonctionnels comme une solution potentielle chez les individus obèses ayant des signaux de satiété altérés **(2004-137)**

Monsieur,

Le Comité d'éthique de la recherche de l'Université Laval a pris connaissance de votre réponse à sa lettre du 8 juin concernant le projet de recherche cité en objet. Le Comité considère que les modifications effectuées au formulaire de consentement satisfont à ses demandes. Par conséquent, le Comité approuve ledit projet, ainsi que la version datée du 25 août 2004 du formulaire de consentement, pour une période d'un an, soit jusqu'au 1^{er} septembre 2005.

Le Comité d'éthique devra être informé et devra réévaluer ce projet advenant toute modification ou l'obtention de toute nouvelle information qui surviendrait à une date ultérieure à celle de la présente approbation et qui comporterait des changements dans le choix des sujets, dans la manière d'obtenir leur consentement ou dans les risques encourus. De plus, toute complication imprévue et sérieuse concernant un participant inscrit à la présente étude devra être immédiatement rapportée par écrit au comité d'éthique peu importe si cet événement est survenu dans notre milieu ou ailleurs dans un autre centre. Le chercheur devra y joindre son évaluation personnelle de la situation en précisant si, selon lui, cet événement est relié à l'étude, s'il s'agit d'un risque jusque-là inconnu, si les participants déjà inscrits doivent être informés et si une modification du formulaire de consentement est nécessaire pour les nouveaux sujets.

Le projet devra être réévalué un an à partir de la date d'approbation, le chercheur indiquant brièvement l'évolution et le déroulement de sa recherche, le nombre de participants recrutés et si les perspectives de cette recherche se déroulent tel que prévu. Un formulaire de demande de renouvellement est disponible sur le site Internet du Comité à l'adresse suivante : <http://www.ulaval.ca/vrr/deontologie/cdr/CDR.html>

Veillez agréer, Monsieur, nos sentiments les meilleurs.

Édith Deleury
Présidente
Comité d'éthique de la recherche de l'Université Laval

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Université d'Ottawa **University of Ottawa**
Bureau d'éthique et d'intégrité de la recherche Office of Research Ethics and Integrity

Certificat d'approbation déontologique
CÉR Sciences et science de la santé

Chercheur principal / Superviseur / Co-chercheur(s) / Étudiant(s)

<u>Prénom</u>	<u>Nom de famille</u>	<u>Affiliation</u>	<u>Rôle</u>
Éric	Doucet	Sciences de la santé / Activité physique	Chercheur principal

Numéro du dossier: H 09-11-05

Type du projet: Professeur

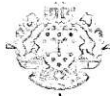
Titre: Les effets de l'intensité et du type d'entraînement sur la compensation post-entraînement

Date d'approbation (mm/jj/aaaa)	Date d'expiration (mm/jj/aaaa)	Approbation
01/17/2012	01/16/2013	Ia

(Ia: Approbation complète, Ib: Autorisation préliminaire de libération de fonds de recherche)

Conditions Spéciales / Commentaires:

N/A



Université d'Ottawa | University of Ottawa

Bureau d'éthique et d'intégrité de la recherche | Office of Ethics and Research Integrity

Le 26 mars, 2012

Éric Doucet
École des sciences de l'activité physique
Faculté des sciences de la santé
Université d'Ottawa
200 Lees Avenue
Ottawa ON K1N 6N5

Object: Les effets de l'intensité et du type d'entraînement sur la composition post-entraînement (Dossier H 09-11-05)

Cher chercheur,

Le Comité d'éthique de la recherche en Sciences de la santé et Sciences de l'Université d'Ottawa a étudié votre demande de modification de votre projet de recherche. Les modifications suivantes ont été acceptées:

- Le chercheur recrutera un plus grand nombre de participants afin d'avoir une plus grande puissance statistique.
- Désormais, le chercheur recrutera aussi des participants modérément actifs à sédentaires.
- Le chercheur ajoute l'aspect sommeil à la recherche afin de vérifier si la session d'exercice aura un impact sur la qualité et la durée du sommeil des participants. Pour ce faire, trois questionnaires (Pittsburgh Sleep Questionnaire, Post-sleep Questionnaire et Pre-sleep Questionnaire) ont été ajoutés aux outils de recherche.
- Pour des raisons de sécurité et de confort, le cathéter sera retiré avant les exercices et replacé après.
- Le chercheur a modifié la dépense énergétique cible de 350 kcals à 4 kcal/kg.
- Le nombre de séances expérimentales est réduit de 5 à 3.
- La mesure du rythme métabolique de repos (RMR) sera faite avant la première session expérimentale au lieu de lors de la visite préliminaires et le participant devra arriver au laboratoire à 7 h 10 au lieu de 8 h afin de faire le test.
- Il n'y aura plus de deuxième visite préliminaire.
- La compensation sera désormais de 25 \$ par session vu que le nombre de séances expérimentales et de séances préliminaires a été réduit.
- Jessica McNeil se joint à l'équipe de recherche et travaillera sur le volet sommeil de la recherche : Sébastien Cadieux devient le responsable du recrutement des participants.

L'approbation éthique, valide jusqu'au 10 juin 2012 couvre les modifications demandées.

Si vous avez des questions, vous pouvez me téléphoner au 613-562-5387.

Veuillez agréer mes considérations sincères.

Germain Zongo
Responsable de l'éthique à la recherche
Pour le Dr Daniel Lagarec, Président du CÉR en
Sciences de la santé et Sciences

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Office of Research Ethics and Integrity

Ethics Approval Notice

Health Sciences and Science REB

Principal Investigator / Supervisor / Co-investigator(s) / Student(s)

<u>First Name</u>	<u>Last Name</u>	<u>Affiliation</u>	<u>Role</u>
Éric	Doucet	Health Sciences / Human Kinetics	Supervisor
Geneviève	Forest	Social Sciences / Psychology	Co-Supervisor
Jessica	McNeil	Health Sciences / Human Kinetics	Student Researcher

File Number: H 04-14-04

Type of Project: PhD Thesis

Title: The effects of sleep restriction and sleep timing on the energy balance, food reward, satiety efficiency and olfactory capacity.

Approval Date (mm/dd/yyyy)	Expiry Date (mm/dd/yyyy)	Approval Type
05/21/2014	05/20/2015	Ia

(Ia: Approval, Ib: Approval for initial stage only)

Special Conditions / Comments:

N/A



Ethics Approval Notice
Health Sciences and Science REB

Principal Investigator / Supervisor / Co-investigator(s) / Student(s)

<u>First Name</u>	<u>Last Name</u>	<u>Affiliation</u>	<u>Role</u>
Éric	Doucet	Health Sciences / Human Kinetics	Supervisor
Geneviève	Forest	Social Sciences / Psychology	Co-Supervisor
Jessica	McNeil	Health Sciences / Human Kinetics	Student Researcher

File Number: h04-14-04

Type of Project: PhD Thesis

Title: The effects of sleep restriction and sleep timing on the energy balance, food reward, satiety efficiency and olfactory capacity.

Renewal Date (mm/dd/yyyy)	Expiry Date (mm/dd/yyyy)	Approval Type
05/21/2015	05/20/2016	Ia

(Ia: Approval, Ib: Approval for initial stage only)

Special Conditions / Comments:

N/A

APPENDIX B: FINAL PUBLISHED VERSION OF THESIS ARTICLE #1

European Journal of Clinical Nutrition (2013), 1–4
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www.nature.com/ejcn

SHORT COMMUNICATION

Short sleep duration is associated with a lower mean satiety quotient in overweight and obese men

J McNeil¹, V Drapeau², AR Gallant², A Tremblay³, É Doucet¹ and J-P Chaput⁴

We examined satiety quotient (SQ) and energy intake (EI) according to sleep duration, quality and timing. Seventy-five overweight/obese men (age: 41.1 ± 5.8 years; body mass index: 33.6 ± 2.9 kg/m²) completed visual analogue scales for appetite sensations before, immediately after and every 10 minutes for 1 hour following a standardized breakfast. The mean SQ (primary outcome of the study) was calculated from four appetite sensations. The Pittsburgh Sleep Quality Index identified short-duration (<7 h/night) and 'recommended sleep duration' (≥ 7 h/night) sleepers, poor (score ≥ 5) and good (score <5)-quality sleepers and late (midpoint of sleep >0230 hours) and early (midpoint of sleep ≤ 0230 hours) sleepers. A 3-day food record and buffet-style meal assessed the EI. Short-duration sleepers had a lower mean SQ compared with recommended sleep duration sleepers (6.5 ± 4.9 vs 8.8 ± 4.3 mm/100 kcal; $P = 0.04$). The mean SQ between poor and good (6.9 ± 4.6 vs 8.7 ± 4.6 mm/100 kcal; $P = 0.11$) and that between early and late (8.99 ± 5.10 vs 9.32 ± 4.02 mm/100 kcal; $P = 0.78$) sleepers were not significantly different. EI did not differ between the sleep groups. Thus, short-duration sleepers had a lower mean SQ compared with recommended sleep duration sleepers. However, this did not coincide with an increased EI.

European Journal of Clinical Nutrition advance online publication, 16 October 2013; doi:10.1038/ejcn.2013.204

Keywords: satiety quotient; sleep duration; sleep quality; sleep timing; energy intake

INTRODUCTION

Current evidence associates short sleep duration with the development of obesity.¹ The satiety quotient (SQ), expressed according to energy intake (EI), determines the extent to which a meal can reduce subjective appetite sensations.² A lower fullness SQ, or smaller changes in subjective fullness ratings in response to a meal, was associated with an increased EI in obese individuals.² It is, however, unknown whether changes in SQ may differ according to sleep parameters.

The present study evaluated the SQ in response to a standardized meal in overweight/obese men according to sleep duration, sleep quality and sleep timing. The mean SQ, based on responses to four different appetite sensations, was the main outcome of this study. We hypothesized that a short sleep duration, poor sleep quality and a later bedtime would be associated with a lower mean SQ and a greater EI.

METHODS

Participants

Seventy-five overweight/obese, healthy Caucasian men completed an in-laboratory assessment at Laval University (Quebec, Canada). The inclusion criteria were as follows: age between 30 and 50 years, body mass index between 28 and 40 kg/m², non-smokers, not taking medications that could influence appetite, non-diabetic with no insulin treatment, weight stable (± 4 kg within the past 2 months), <3 \times 30 min/week of physical activity and a low dietary restraint (score <10 on the Three-Factor Eating Questionnaire). This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures

were approved by the Laval University ethics committee. Participants provided written informed consent.

Procedure and measurements

Participants arrived at the laboratory at 0800 hours following a 12-h overnight fast. They were instructed not to consume alcohol or engage in structured physical activity (for example, playing sports) for at least 24 h before testing and to follow their usual sleeping habits the previous night. Upon arrival, height, weight and waist circumference were measured according to standardized procedures³, and fat mass was measured by dual-energy X-ray absorptiometry (GE Medical Systems Lunar, Diegem, Belgium).

A standardized breakfast and *ad libitum* lunch were served at 0830 and 1200 hours, respectively. The compositions of these meals are described in more detail in Appendix. The breakfast had a food quotient (global indicator of meal macronutrient composition) of 0.85 and was entirely consumed within 20 minutes. The participants' appetite sensations were recorded using visual analogue scales (VAS) before, immediately after, and at every 10 minutes for 1 hour following breakfast consumption. The 150-mm VAS were used to answer four questions that quantify subjective appetite sensations: desire to eat, hunger, fullness and prospective food consumption. The SQ was calculated for each appetite sensation using the following equation (2):

$$SQ \text{ (mm/100kcal)} = \frac{[\text{fasting appetite sensation (mm)} - \text{mean post meal appetite sensation (mm)}]}{\text{energy content of the test meal (kcal)}} \times 100$$

It is important to note that the SQ calculation for fullness is reversed (the mean post meal rating – fasting rating). The mean SQ represents the mean value of the four individual SQ scores. This was selected as the primary outcome of the study as it provides a composite indication of the changes in appetite sensations in response to the meal. A lower SQ indicates a weaker satiety response to a meal.³

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The Pittsburgh Sleep Quality Index⁴ determined sleep duration (self-reported item), sleep quality (total score) and sleep timing (midpoint of sleep based on reported wake time and sleep duration) over the last month. The calculations for sleep timing are described elsewhere.⁵

Three-day food records and physical activity diaries, including 2 weekdays and one weekend day, assessed habitual EI and moderate-to-vigorous physical activity participation, respectively, following the in-laboratory assessment.

Statistical analyses

Independent *t*-tests compared variables between the sleep duration, sleep quality and sleep timing groups. Statistical significance was set at $P < 0.05$. Statistical analyses were performed using JMP (version 10; SAS Institute, Cary, NC).

RESULTS

Table 1 presents participants' characteristics according to sleep groups. There were no differences in these variables between groups, except for 3-day carbohydrate intake between sleep quality groups ($P = 0.03$). There were no significant differences in specific SQ for desire to eat, hunger, fullness or prospective food consumption between groups (data not shown). Short-duration sleepers had a lower mean SQ compared to sleepers with recommended sleep durations, whereas no significant differences in the mean SQ between sleep quality and sleep timing groups were noted (Figure 1).

DISCUSSION

To our knowledge, this is the first study to examine measures of SQ according to sleep duration, sleep quality and sleep timing in overweight/obese men. Short sleep duration was associated with a weaker mean SQ, despite no significant differences in body weight, fat mass and EI between sleep duration groups. There

were no differences in the mean SQ between sleep quality and sleep timing groups, despite a greater 3-day carbohydrate intake in good vs poor sleepers. The SQ is a more valid indicator of potential changes in subjective appetite ratings in response to a standardized meal compared with 1 h post-prandial area under the curve calculations because it considers pre-meal appetite sensations and meal caloric content.³

The greater mean SQ in short-duration sleepers did not coincide with greater EI in this study. These results suggest that appetite ratings may not be consistently related to measured or reported EI.⁶ Furthermore, despite noting a greater EI following imposed sleep restrictions,^{7,8} one study found no differences in appetite ratings between sleep conditions,⁷ whereas another only noted increased pre-prandial hunger ratings following sleep restriction.⁸ Taken together, changes in appetite ratings, or SQ, may not be consistently related to changes in EI and *vice versa*.

Studies have also shown that a later sleep timing may lead to an increase in EI after 2000 hours,⁹ as well as a greater total EI⁵ in adults and obese children/adolescents, respectively. Conversely, the current study did not observe a significant difference in the mean SQ and EI between sleep timing groups. This lack of association may be due to differences in participant characteristics and calculated sleep timing midpoints between this study and others.^{5,9}

Finally, reductions in stage 2, rapid eye movement and slow-wave sleep were associated with greater hunger ratings and EI,¹⁰ whereas the occurrence of sleep fragmentation led to a lower fullness and greater desire to eat ratings compared with a non-fragmented sleep condition.¹¹ These results suggest that alterations in specific sleep stages following imposed sleep fragmentation, rather than self-reported habitual sleep quality, may alter appetite ratings.

The present findings are limited to a small sample size of overweight/obese men, which limits generalizability to other populations. The cross-sectional design used does not allow for

Table 1. Characteristics of participants according to sleep duration, sleep quality and sleep timing groups

	Sleep duration		Sleep quality		Sleep timing	
	Short-duration sleepers (< 7 h/night) (n = 34)	Sleepers with recommended sleep durations (≥ 7 h/night) (n = 41)	Poor sleepers (PSQI score ≥ 5) (n = 33)	Good sleepers (PSQI score < 5) (n = 42)	Late sleepers (midpoint of sleep > 0230 hours) (n = 37)	Early sleepers (midpoint of sleep ≤ 0230 hours) (n = 38)
Mean \pm s.d.	5.7 \pm 0.9 h/night	7.5 \pm 0.5 h/night	PSQI score 6.6 \pm 1.9	PSQI score 2.9 \pm 1.2	0312 hours \pm 36 min	0200 hours \pm 30 min
Range	3–6.75 h/night	7–9 h/night	PSQI score 5–11	PSQI score 1–4		
Age (years)	41.6 \pm 6.6	40.4 \pm 4.6	41.0 \pm 6.4	40.9 \pm 5	39.3 \pm 5.7	41.8 \pm 5.0
Height (cm)	174.0 \pm 5.7	174.6 \pm 7.0	174.2 \pm 5.3	174.3 \pm 7.3	175.3 \pm 7.2	173.0 \pm 5.6
Body weight (kg)	101.4 \pm 9.5	103.2 \pm 12.6	101.5 \pm 9.5	103.1 \pm 12.9	101.3 \pm 11.2	104.0 \pm 11.9
BMI (kg/m ²)	33.5 \pm 2.9	33.8 \pm 3.0	33.4 \pm 2.9	33.9 \pm 3.1	33.8 \pm 2.9	33.8 \pm 3.2
Fat mass (kg)	34.2 \pm 6.4	35.2 \pm 7.6	34.6 \pm 6.2	34.8 \pm 8.1	34.7 \pm 7.9	34.2 \pm 6.9
Fat mass (%)	33.8 \pm 4.8	34.2 \pm 4.7	34.2 \pm 4.7	33.8 \pm 4.9	33.4 \pm 5.1	33.8 \pm 4.4
Waist circumference (cm)	109.9 \pm 6.9	112.5 \pm 8.6	110.9 \pm 6.8	111.5 \pm 9.0	109.6 \pm 7.1	112.2 \pm 8.8
Lunch EI (kJ)	4987 \pm 1501	5323 \pm 1710	4984 \pm 1360	5411 \pm 1808	4891 \pm 1629	5580 \pm 1638
Carb intake (kJ)	2146 \pm 649	2265 \pm 766	2127 \pm 589	2304 \pm 811	2115 \pm 631	2344 \pm 841
Lipid intake (kJ)	2018 \pm 831	2153 \pm 907	1999 \pm 732	2214 \pm 980	1938 \pm 935	2290 \pm 812
Protein intake (kJ)	824 \pm 297	905 \pm 276	858 \pm 288	893 \pm 282	839 \pm 309	947 \pm 241
EI (kJ/day)	11 816 \pm 2335	11 715 \pm 2464	11 259 \pm 2251	12 029 \pm 2439	11 919 \pm 2653	11 610 \pm 2168
Carb intake (kJ/day)	5206 \pm 1368	5365 \pm 1212	4887 \pm 1357	5588 \pm 1146*	5397 \pm 1491	5255 \pm 1104
Lipid intake (kJ/day)	4238 \pm 1136	3986 \pm 1084	3938 \pm 989	4131 \pm 1129	4179 \pm 1187	3969 \pm 962
Protein intake (kJ/day)	2000 \pm 425	1924 \pm 445	1954 \pm 390	1945 \pm 472	1972 \pm 403	1936 \pm 465
Alcohol intake (kJ/day)	385 \pm 469	530 \pm 826	516 \pm 525	425 \pm 802	418 \pm 522	497 \pm 818
MVPA (min/day)	12.6 \pm 22.7	22.2 \pm 69.8	12.2 \pm 23.8	23.6 \pm 71.8	13.3 \pm 21.3	23.1 \pm 76.5

Abbreviations: BMI, body mass index; EI, energy intake; MVPA, moderate-to-vigorous physical activity; PSQI, Pittsburgh Sleep Quality Index. All variables were not significantly different between groups with the use of independent *t*-tests, except for daily carbohydrate intake between good and poor sleepers (indicated by the *).

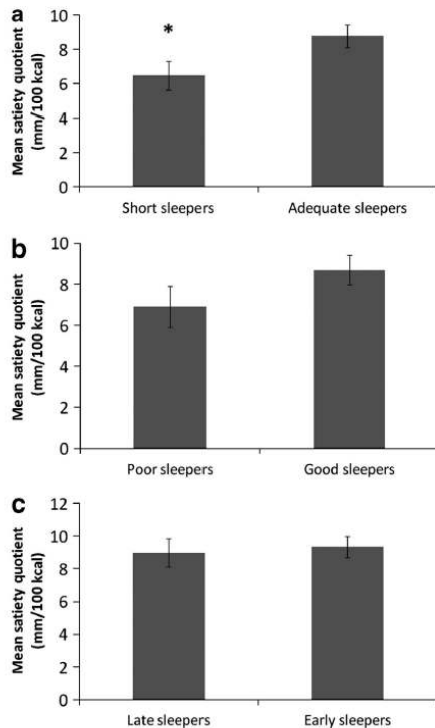


Figure 1. The mean satiety quotient between (a) short-duration sleepers (<7h of sleep/night) and sleepers with a recommended sleep duration (≥ 7 h of sleep/night, b), between poor sleepers (PSQI score <5) and good sleepers (PSQI score ≥ 5 , c) and between late sleepers (midpoint of sleep >0230 hours) and early sleepers (midpoint of sleep ≤ 0230 hours). Values are presented as means for 75 participants with standard errors of the mean represented by vertical bars. * $P=0.04$ when compared with recommended sleep duration sleepers. $P=0.11$ between poor and good sleepers. $P=0.78$ between early and late sleepers. PSQI, Pittsburgh Sleep Quality Index.

causal relationships to be drawn. Finally, the use of self-reported measurements and the possibility of residual confounding factors cannot be overlooked (for example, we are unable to determine whether differences in sleep timing are related to biological predispositions or social circumstances).

Although exploratory, we observed a lower mean SQ in short-duration sleepers. The mean SQ between sleep quality and sleep timing groups was not statistically different. Finally, no difference in EI was noted between sleep groups. Future studies are needed to confirm these preliminary findings.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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APPENDIX

TABLE A1. Characteristics of participants according to the sleep duration, sleep quality and sleep timing groups

	Quantity provided (grams)	Energy content (kJ)
<i>Standardized breakfast</i>		
White bread	100	1092
Butter	12	371
Peanut butter	16	429
Cheddar cheese	40	688
Orange juice	250	486
Total	418	3066
	Initial quantity provided (grams)	Energy content (kJ/kg)
<i>Ad libitum lunch</i>		
Sliced turkey	130	3930
Sliced ham	150	5480
Salmon mousse	90	10833
Liver pâté	70	13 350
Gruyere cheese (28% fat)	100	17 286
Mozzarella cheese (17% fat)	100	11 718
Cottage cheese (2% fat)	100	3384
White bread	150	11 300
Whole-wheat bread	150	10 170
Soda crackers	100	18 400
Butter	40	29 990
Mayonnaise	60	30 630
Ketchup	40	4350
Italian dressing	60	26 110
Mustard	30	3140
Lettuce	60	670
Tomatoes	100	880
Carrots	150	1800
Butter biscuits	70	20 840
Chocolate fudge cookies	100	19 700

Table A1. (Continued)

	<i>Quantity provided (grams)</i>	<i>Energy content (kJ)</i>
Strawberry yoghurt (1.5% fat)	250	4050
Regular crisps	60	23214
Apples	100	2470
Oranges	100	1970
Milk (2% fat)	1000	2095
Orange juice	1000	1826
Coca cola	355	1720
7-Up	355	1674
Water	1000	0

Associations between sleep parameters and food reward

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exercise modality, food seeking, sleep patterns

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SUMMARY

This study examined the effects of acute, isocaloric aerobic and resistance exercise on different sleep parameters, and whether changes in these sleep parameters between sessions were related to next morning food reward. Fourteen men and women (age: 21.9 ± 2.7 years; body mass index: 22.7 ± 1.9 kg m⁻²) participated in three randomized crossover sessions: aerobic exercise; resistance exercise; and sedentary control. Target exercise energy expenditure was matched at 4 kcal kg⁻¹ of body weight, and performed at 70% of VO_{2peak} or 70% of 1 repetition-maximal. Sleep was measured (accelerometry) for 22 h following each session. The 'wanting' for visual food cues (validated computer task) was assessed the next morning. There were no differences in sleep parameters and food 'wanting' between conditions. Decreases in sleep duration and earlier wake-times were significantly associated with increased food 'wanting' between sessions ($P = 0.001$). However, these associations were no longer significant after controlling for elapsed time between wake-time and the food reward task. These findings suggest that shorter sleep durations and earlier wake-times are associated with increased food reward, but these associations are driven by elapsed time between awakening and completion of the food reward task.

INTRODUCTION

Acute bouts of exercise may have beneficial effects on sleep by increasing sleep duration and slow-wave sleep (Bunnell *et al.*, 1983; Youngstedt *et al.*, 1997). However, some studies noted no significant differences in self-reported (Porter and Horne, 1981) and objectively-measured (King *et al.*, 2008) sleep duration and sleep quality between exercise and non-exercise days. Passos *et al.* (2010) reported increased sleep duration and sleep efficiency following an acute bout of moderate-intensity aerobic exercise, but not following high-intensity aerobic and moderate-intensity resistance exercise, in insomniac patients. These results are, however, limited by a lack of control over exercise energy expenditure (ExEE), which is lower during resistance versus aerobic exercises per unit of time (Donnelly *et al.*, 2004). Hence, the investigation of sleep following acute, isocaloric aerobic and resistance exercise is warranted.

Studies also reported greater neuronal responsiveness to food versus non-food stimuli following imposed sleep restrictions (Benedict *et al.*, 2012; St-Onge *et al.*, 2012). It is, however,

unknown whether habitual changes in sleep parameters under free-living conditions are associated with changes in food reward.

The objective of the current study was twofold. First, we examined the effects of an acute bout of isocaloric aerobic and resistance exercise on different sleep parameters (sleep duration, sleep efficiency, sleep efficiency after sleep onset, sleep-onset latency, bedtime and wake-time). Secondly, we investigated whether changes in these sleep parameters between sessions were related to next day food 'wanting' through secondary analyses.

MATERIALS AND METHODS

Seven men and seven women (age: 21.9 ± 2.7 years; body mass index: 22.7 ± 1.9 kg m⁻²; body fat percentage: $21.0 \pm 7.9\%$; VO_{2peak}: 52.6 ± 9.0 mL kg⁻¹ min⁻¹) completed all required measurements. They were between 18 and 45 years old, non-smokers, weight stable (± 4 kg) within the last 6 months, did not have heart problems or diabetes, and participated in <150 min of physical activity per week.

Two participants had ratings ≥ 10 on the Pittsburgh Sleep Quality Index (PSQI), which classifies them as being poor sleepers according to this questionnaire. Only non-pregnant, premenopausal women were recruited. This study was conducted according to the guidelines laid down in the Declaration of Helsinki. The University of Ottawa ethics committee approved all procedures involving human participants. Written informed consent was obtained from all participants.

Participants took part in three randomized crossover sessions: aerobic exercise; resistance exercise; and sedentary control. ExEE was matched at 4 kcal kg^{-1} of body weight. The mean ExEE during the aerobic and resistance exercise sessions were 274.5 ± 50.6 and $270.4 \pm 56.3 \text{ kcal}$, respectively ($P = \text{NS}$; Cadieux *et al.*, 2014). A washout period of at least 7 days separated each session for men, and at least 1 month for women because they were always tested between days 1 and 8 of the menstrual cycle as cortisol responses to sleep restriction (Leroux *et al.*, 2014) and brain activation to food cues (Alonso-Alonso *et al.*, 2011) have both been shown to vary across the menstrual cycle. For each session, participants arrived at the laboratory at 08:00 hours following a 12-h overnight fast. They were instructed not to consume alcohol or engage in structured physical activity (e.g. training and playing sports) for at least 24 h prior to each session, and during the data collection period. Participants were also instructed not to consume caffeine during the data collection period. Upon arrival, participants were weighed to the nearest 0.1 kg with a BWB-800AS digital scale and served a standard breakfast. At 10:30 hours (10:00 hours for the resistance exercise), participants completed the aerobic (running at 70% of $\text{VO}_{2\text{peak}}$) or resistance (supersets at 70% of 1-maximal repetition) exercise interventions, which ended when target ExEE was reached (~11:00 and 11:30 hours for the aerobic and resistance exercises, respectively), or the sedentary control session (recreational reading for 45 min). ExEE during each intervention was measured with a portable indirect calorimetry unit (model K4b²; COSMED, Chicago, IL, USA), as previously described (Cadieux *et al.*, 2014).

After each intervention (~14:00 hours), participants wore a biaxial accelerometer (SenseWear Pro 3 Ambands©; HealthWear Bodymedia, Pittsburgh, PA, USA) around the upper arm to assess habitual sleep parameters over 22 h. Sharif and Bahammam (2013) noted no significant differences in sleep duration, wake-time and sleep efficiency assessed with this biaxial accelerometer or polysomnography. The overall intraclass correlation between both tools was above 0.8, indicating good agreement in assessing sleep parameters (Sharif and Bahammam, 2013). These results were also verified with sleep diaries. Participants were asked to rate their sleep quality the following morning by choosing an answer on a five-point Likert scale that best described their sleep quality the preceding night (1 – much better than normal; 2 – better than normal; 3 – normal; 4 –

worse than normal; and 5 – much worse than normal). Participants also rated their feelings of sleepiness on a seven-point Likert scale (1 – alert/not tired and 7 – sleep onset soon/very tired) within 15 min prior to going to bed, and within 15 min of waking up the following morning.

The following morning, between 10:00 and 12:00 hours, participants completed a validated computer-based behavioural procedure (Leeds Food Preference Questionnaire). Participants rated the extent to which they 'wanted' each randomly presented visual food cue with a 100-mm visual analogue scale. The questions and scoring methods used are described elsewhere (Finlayson *et al.*, 2008).

Statistical analyses were performed using SPSS (version 17.0; SPSS, Chicago, IL, USA). One-way repeated-measures ANOVA tests determined the effects of exercise modality on sleep parameters and next morning food 'wanting'. Bivariate Spearman correlations with Bonferroni corrections assessed the strength of relationships between sleep parameters with food 'wanting' at each session, as well as the changes in these parameters between sessions ($\Delta_{\text{control}} - \text{aerobic}$; $\Delta_{\text{control}} - \text{resistance}$; $\Delta_{\text{aerobic}} - \text{resistance}$). Data are presented as mean \pm SD. Differences with P -values < 0.05 and < 0.004 were considered statistically significant for the ANOVA analyses and Spearman correlations, respectively.

RESULTS

No differences were noted between sessions (Table 1), or across time for all sleep and food reward outcomes (results not shown). Sleep duration was negatively associated with food 'wanting' in the aerobic exercise session ($\rho = -0.83$; $P = 0.0001$). Changes in sleep duration ($\rho = -0.78$; $P = 0.001$) and wake-time ($\rho = -0.77$; $P = 0.001$) were negatively correlated with $\Delta_{\text{food 'wanting'}}$ in the $\Delta_{\text{aerobic}} - \text{resistance}$ exercise condition. However, these associations were no longer significant after correcting for the time elapsed between wake-time and completion of the food reward task (results not shown).

No other significant correlations were noted between sleep parameters with food 'wanting', or between the changes in these variables (results not shown). Lastly, no significant correlations were noted between naptime (min) and PSQI scores with sleep efficiency and sleepiness measurements (results not shown).

DISCUSSION

This is the first study to investigate acute effects of isocaloric aerobic and resistance exercise on sleep parameters, and whether habitual changes in sleep parameters are associated with next morning food reward.

Our results indicated no significant effects of exercise modality on sleep, which supports studies that subjectively (Porter and Home, 1981) and objectively (King *et al.*, 2008) assessed sleep parameters on exercise and non-exercise days. A study that noted increases in sleep duration and

Table 1 Objectively- and subjectively-measured sleep parameters and food 'wanting' assessed following each session

	Sedentary control		Aerobic exercise		Resistance exercise		Session effect
	Mean	SD	Mean	SD	Mean	SD	
Sleep parameters (accelerometry)							
Sleep duration (min)	416	88	432	59	407	83	<i>P</i> = 0.62
Sleep efficiency (%)	81.2	8.2	82.8	8.7	78.8	6.8	<i>P</i> = 0.21
Sleep efficiency after sleep onset (%)	86.7	7.7	86.9	8.4	86.6	6.7	<i>P</i> = 0.99
Sleep-onset latency (min)	13	7	9	8	19	23	<i>P</i> = 0.23
Bedtime (i.e. sleep onset)	12:03 hours	1 h 22 min	12:22 hours	2 h 16 min	12:03 hours	1 h 12 min	<i>P</i> = 0.77
Wake-time	07:51 hours	1 h 17 min	07:47 hours	1 h 30 min	07:54 hours	1 h 43 min	<i>P</i> = 0.97
Daytime nap time (min)	24	45	11	23	22	44	<i>P</i> = 0.44
Sleep parameters (self-reported)							
Sleep quality	3.2	0.9	3.4	0.6	3.0	0.6	<i>P</i> = 0.25
Evening sleepiness rating	5.0	1.5	5.0	1.0	5.4	1.1	<i>P</i> = 0.44
Morning sleepiness rating	3.2	1.0	2.8	1.1	3.4	1.2	<i>P</i> = 0.37
Food reward measurement							
Food 'wanting' (mm)	40.6	15.7	35.7	15.9	36.3	15.2	<i>P</i> = 0.34

SD, standard deviation.

Food 'wanting' scores were measured with a 100-mm visual analogue scale. Self-reported sleep quality was measured with a five-point Likert scale (i.e. score of 3 is indicative of an habitual or normal, sleep quality), and sleepiness ratings were measured with a seven-point Likert scale. Sleep efficiency (%) is defined as sleep duration/time lying down in bed, and sleep efficiency after sleep onset (%) is defined as sleep efficiency (%) after sleep onset and before wake-time.

slow-wave sleep did so following acute aerobic exercise to exhaustion (Bunnell *et al.*, 1983). The latter is further supported by a meta-analysis (Youngstedt *et al.*, 1997), which reported significant median increases in sleep duration and slow-wave sleep of 10 and 1.4 min, respectively, following an acute bout of aerobic exercise; with effects being greatest for aerobic exercises that exceeded 1 h. Hence, the shorter aerobic exercise session (mean duration of 24 min) sustained at 70% of $\text{VO}_{2\text{peak}}$ in the present study may not be sufficient to alter sleep. Conversely, the resistance exercise lasted on average 86 min, and had no significant impact on sleep. It may be hypothesized that ExEE, which was matched in this study, may have a greater impact on sleep rather than exercise duration *per se*. Future studies are needed to evaluate this hypothesis, and assess the effects of exercise modality performed at different times of day on sleep, as advancements in melatonin release following evening exercise (Buxton *et al.*, 2003) may affect sleep differently.

Secondary analyses revealed that decreases in sleep duration and earlier wake-times were associated with increased food reward. These results add to studies reporting greater neuronal responsiveness to food versus non-food stimuli following imposed sleep restriction (Benedict *et al.*, 2012; St-Onge *et al.*, 2012). Although other cross-sectional studies reported associations between later sleep timing midpoints with poorer diet qualities (e.g. higher fast food and lower fruit/vegetable intakes; Baron *et al.*, 2011; Sato-Mito *et al.*, 2011), this is the first study to suggest that an earlier wake-time is associated with increased food reward. An important confounding factor in the present study is the elapsed time between wake-time and completion of the food reward task (between 10:00 and 12:00 hours), as participants with an earlier wake-time may express greater drives towards food in the morning because of greater elapsed time spent awake prior to completing the task, comparatively to individuals who usually wake later.

The present findings are limited by a small sample size of normal-weight men and women. Only 1-day assessments of outcomes, and acute exercise interventions, were performed, which may not account for normal day-to-day variability or potential additive effects. The food reward task represents a proxy of actual food intake. Differences in exposures to environmental factors in the evening and/or overnight between conditions were not evaluated and may alter sleep (e.g. exposure to blue-light from technological devices). The strength of associations noted in women may be in part influenced by the menstrual cycle, as all women were tested between days 1 and 8 of the follicular phase. Lastly, correlations cannot infer causality. However, the temporal order of events (i.e. sleep preceding food reward measurements) reinforces current results.

These findings suggest that exercise modality does not acutely alter sleep. Shorter sleep durations and earlier wake-times were associated with increased food reward, but these associations were driven by elapsed time between

awakening and completion of the food reward task. Future studies are needed to confirm these preliminary findings and further assess the effects of sleep timing, or individual circadian rhythms, on food reward. Future studies should also consider the elapsed time between awakening and completion of food reward measurements, as this may be an important cofounder driving food-seeking behaviour.

AUTHOR CONTRIBUTIONS

J. M., S. C. and É. D. formulated the research questions, designed the study and carried out the experiment. J. M., G. F. and É. D. analysed the data. All authors were involved in writing the paper, and had final approval of the submitted and published version.

CONFLICT OF INTEREST DISCLOSURE

The authors of this paper declare no conflict of interest.

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APPENDIX D: LIST OF PUBLISHED NON-THESIS ABSTRACTS AND PEER-REVIEWED SCIENTIFIC ARTICLES DURING PH.D. TENURE

Published peer-reviewed scientific articles

1. Jean-Philippe Chaput. Simon Jomphe-Tremblay. Jacynthe Lafrenière. Sean Patterson. **Jessica McNeil**. Zachary M Ferraro. 2015. Reliability of a Food Menu to Measure Energy and Macronutrient Intake in Adolescents. *European Journal of Clinical Nutrition*, 1-5.
2. Aidan Gribbon. **Jessica McNeil**. Ollie Jay. Mark S Tremblay. Jean-Philippe Chaput. 2015. Active video games and energy balance in male adolescents: a randomized crossover trial. *American Journal of Clinical Nutrition*, 101(6).1126-34.
3. **Jessica McNeil**. Sébastien Cadieux. Graham Finlayson. John E Blundell. Éric Doucet. 2015. The effects of a single bout of aerobic or resistance exercise on food reward. *Appetite*, 84. 264-270.
4. **Jessica McNeil**. Mark S Tremblay. Geneviève Leduc. Charles Boyer. Priscilla Bélanger. Allana G LeBlanc. Michael M Borghese. Jean-Philippe Chaput. 2015. Objectively-measured sleep and its association with adiposity, physical activity and screen time in a sample of Canadian children. *Journal of Sleep Research*, 24. 131-139.
5. Justin J Lang. **Jessica McNeil**. Mark S Tremblay. Travis J Saunders. 2015. Sit less, stand more: A randomized point-of-decision prompt intervention to reduce sedentary time. *Preventive Medicine*, 73. 67-69.
6. **Jessica McNeil**. Alexander Schwartz. Rémi Rabasa-Lhoret. Jean-Marc Lavoie. Martin Brochu. Éric Doucet. 2014. Changes in leptin and peptide YY do not explain the greater than predicted decreases in resting energy expenditure after weight loss. *Journal of Clinical Endocrinology and Metabolism*, 100(3). E443-52.
7. **Jessica McNeil**. Mohamed M Mamlouk. Karine Duval. Alexander Schwartz. Nelson Nardo Junior. Éric Doucet. 2014. Alterations in metabolic profile occur in normal-weight and obese men during the Ramadan fast despite no changes in anthropometry. *Journal of Obesity*, Article ID 482547. 9 pages.
8. **Jessica McNeil**. Denis Prud'homme. Irene Strychar. Rémi Rabasa-Lhoret. Martin Brochu. Jean-Marc Lavoie. Éric Doucet. 2014. Satiety quotient linked to food intake and changes in anthropometry during menopause. A MONET Study. *Climacteric*, 17. 1-7.
9. Josiane A Alves Bianchini. Danilo Fernandez Da Silva. Luzia Jaeger Hintze. Vanessa D Seron Antonini. Carlos A Lopera. **Jessica McNeil**. Nelson Nardo Junior. 2014. Obese adolescents who gained/maintained or lost weight have similar results in body composition and cardiometabolic risk factors following a multidisciplinary intervention. *Journal of Exercise Science & Fitness*, 12. 38-45. .
10. Sébastien Cadieux. **Jessica McNeil**. Mary P Lapierre. Marie-Ève Riou. Éric Doucet. 2014. Resistance and aerobic exercises do not affect post-exercise energy compensation in normal weight men and women. *Physiology & Behavior*, 130. 113-119.

11. Danilo Fernandez Da Silva. Josiane A Alves Bianchini. Vanessa D Seron Antonini. Danielle A Munhos Hermoso. Carlos A Lopera. Bruno G Morais Pagan. **Jessica McNeil**. Nelson Nardo Junior. 2014. Parasympathetic cardiac activity is associated with cardiorespiratory fitness in overweight and obese adolescents. *Pediatric Cardiology*, 35 (4). 684-690.
12. **Jessica McNeil**. Éric Doucet É. Jean-Philippe Chaput. 2013. Inadequate sleep as a contributor to obesity and type 2 diabetes. *Canadian Journal of Diabetes*, 37. 103-108.
13. Jean-Philippe Chaput. **Jessica McNeil**. Jean-Pierre Després. Claude Bouchard. Angelo Tremblay. 2013. Seven to eight hours of sleep a night is associated with a lower prevalence of the metabolic syndrome and reduced overall cardiometabolic in adults. *Plos One*, 8(9). 1-6.
14. Jean-Philippe Chaput. **Jessica McNeil**. Jean-Pierre Després. Claude Bouchard. Angelo Tremblay. 2013. Short sleep duration as a risk factor for the development of the metabolic syndrome in adults. *Preventive Medicine*, 57 (6). 872-877.
15. Jean-Philippe Chaput. Caroline Doyon. **Jessica McNeil**. Éric Doucet É. Angelo Tremblay. 2012. Influence of sleeping habits on adaptive thermogenesis during weight loss in adults. *Bioenergetics Open Access*, 1:103. doi:10.4172/beg.1000103.
16. Jean-Philippe Chaput. **Jessica McNeil**. Jean-Pierre Després. Claude Bouchard. Angelo Tremblay. 2012. Short sleep duration is associated with greater alcohol consumption in adults. *Appetite*, 59. 650-655.

Published book chapters

1. **Jessica McNeil**. Jean-Philippe Chaput. Geneviève Forest. Éric Doucet. 2013. Altered energy balance in response to sleep restriction. *Hormones and Behavior*, Nova Publishers. Chapter 5. 105-119.
Role in publication: Jessica McNeil compiled all relevant scientific articles and wrote the manuscript.
2. **Jessica McNeil**. Éric Doucet. Jean-Philippe Chaput. 2013. Sleep, sedentary activity, and weight gain. *Handbook of Nutrition, Diet and Sleep*, Wageningen academic publishers. Chapter 14. 210-224.
Role in publication: Jessica McNeil compiled all relevant scientific articles and wrote the manuscript.

Published abstracts

1. **Jessica McNeil**. Denis Prud'homme. Rémi Rabasa-Lhoret. Graham Finlayson. Éric Doucet. 2015. Changes in smell function, satiety quotient and food reward in response to a weight loss intervention. *Obesity Facts*. 8, suppl. 1: T3:PO.092. (The European Congress on Obesity; Prague, Czech Republic). Page 100.
2. Jacynthe Lafrenière. **Jessica McNeil**. Véronique Provencher. Éric Doucet. 2015. The effects of food perception on energy compensation following exercise in sedentary women. *Obesity Facts*. 8, suppl. 1: T3:PO.090 (The European Congress on Obesity; Prague, Czech Republic). Page 99.
3. **Jessica McNeil**. Gilles Lamothe. Jameason D Cameron. Marie-Ève Riou. Sébastien Cadieux. Jacynthe Lafrenière. Gary Goldfield. Stephanie Willbond. Éric Doucet. 2015. Investigating predictors of eating: Is resting metabolic rate really the strongest proxy of energy intake? *Canadian Journal of Diabetes*. 39, suppl. 1: P6.08 (The Canadian Obesity Network Summit Meeting; Toronto, Ontario, Canada). Page S59.

4. Josiane A Alves Bianchini. Danilo Fernandez Da Silva. Rafaela Pilegi Dada. Carlos A Lopera. **Jessica McNeil J.** Nelson Nardo Junior. The impact of BMI-Z score changes on the effects of a multidisciplinary programme of obesity treatment on health-related quality of life (HRQoL) in Brazilian Adolescents. *Canadian Journal of Diabetes*. 39, suppl. 1: P3.14 (The Canadian Obesity Network Summit Meeting; Toronto, Ontario, Canada). Page S42.
5. **Jessica McNeil.** Denis Prud'homme. Irene Strychar. Rémi Rabasa-Lhoret. Martin Brochu. Jean-Marc Lavoie. Éric Doucet. 2014. The satiety quotient is related to energy intake and changes in anthropometric variables across time in menopausal women. A MONET group study. *Applied Physiology, Nutrition and Metabolism*. 39, suppl. 1 (The Canadian Nutrition Society; St John's, Newfoundland, Canada). Page 630.
6. **Jessica McNeil.** Mark S Tremblay. Geneviève Leduc. Charles Boyer. Priscilla Bélanger. Allana G LeBlanc. Michael M Borghese. Jean-Philippe Chaput. 2014. Short sleep duration and later bedtime are associated with more time spent in sedentary activities and greater adiposity in a sample of Canadian children. *Journal of Physical Activity and Health*. 11, suppl. 1 (The Global Summit on the Physical Activity of Children; Toronto, Ontario, Canada). Page S175.
7. **Jessica McNeil.** Vicky Drapeau. Annette R Gallant. Angelo Tremblay. Éric Doucet. Jean-Philippe Chaput. 2013. Short sleep duration is associated with a lower satiety quotient in overweight men. *Canadian Journal of Diabetes*. 37, suppl.2: 643-OR (The Canadian Obesity Network Summit Meeting; Vancouver, British-Columbia, Canada). Page S246.
8. **Jessica McNeil.** Sébastien Cadieux. Graham Finlayson. John E Blundell. Éric Doucet. 2013. Short sleep duration is associated with next day food wanting and liking in adults. *Obesity Facts*. 6, suppl.1: T2:P.019 (The European Congress on Obesity; Liverpool, United Kingdom). Page 151.
9. Sébastien Cadieux. **Jessica McNeil.** Mary LaPierre. Dominique Pharand. Éric Doucet. Effects of exercise modality on short term post-exercise energy compensation in adults. *Obesity Facts*. 6, suppl.1: T2:P.089 (The European Congress on Obesity; Liverpool, United Kingdom). Page 170.
10. Jean-Philippe Chaput. **Jessica McNeil.** Jean-Pierre Després. Claude Bouchard. Angelo Tremblay. 2013. Short sleep duration as a risk factor for the development of the metabolic syndrome in adults. *Obesity* 21, suppl. 1: T-334-P (The Obesity Society Annual Meeting; Atlanta, Georgia, USA). Page S131.
11. Jean-Philippe Chaput. **Jessica McNeil.** Jean-Pierre Després. Claude Bouchard. Angelo Tremblay. 2013. Short sleep duration is associated with features of the metabolic syndrome and with overall cardiometabolic risk in adults. *Obesity* 21, suppl. 1: T-335-P (The Obesity Society Annual Meeting; Atlanta, Georgia, USA). Page S131.
12. Jean-Philippe Chaput. Caroline Doyon. **Jessica McNeil.** Éric Doucet É. Angelo Tremblay. 2012. Insufficient sleep is not associated with a greater adaptive reduction in thermogenesis during weight loss in adults. *Obesity*. 20, suppl. 1: 256-P (The Obesity Society Annual Meeting; San Antonio, Texas, USA). Page S110.
13. Jean-Philippe Chaput. **Jessica McNeil.** Jean-Pierre Després. Claude Bouchard. Angelo Tremblay. 2012. Short sleep duration is associated with greater alcohol consumption in adults. *Obesity*. 20, suppl. 1: 808-P (The Obesity Society Annual Meeting; San Antonio, Texas, USA). Page S233.

APPENDIX E: PERMISSIONS FOR REPUBLICATION

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