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

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HIGHLIGHTS

- Sleep restriction with a delayed bedtime led to greater carbohydrate intake
- Sleep restriction led to greater moderate-vigorous intensity physical activity time
- Stage 1 sleep was linked with energy intake between sleep restriction sessions

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ABSTRACT

Experimental evidence suggests that sleep restriction increases energy intake (EI) and may alter energy expenditure (EE). However, it is unknown whether the timing of a sleep restriction period impacts EI and EE the following day. Hence, we examined the effects of sleep restriction with an advanced wake-time or delayed bedtime on next day EI and EE. Twelve men and 6 women (age: 23 ± 4 years, body fat: 18.8 ± 10.1%) participated in 3 randomized crossover sessions: control (habitual bed- and wake-times), 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 EE and activity times (accelerometry). Carbohydrate intake was greater on day 2 in the delayed bedtime *vs.* control session (1386 ± 513 *vs.* 1579 ± 571 kcal; $P = 0.03$). Relative moderate-intensity physical activity (PA) time was greater in the delayed bedtime session *vs.* control and advanced wake-time sessions on day 1 (26.6 ± 19.9 *vs.* 16.1 ± 10.6 and 17.5 ± 11.8%; $P = 0.01$), whereas vigorous-intensity PA time was greater following advanced wake-time *vs.* delayed bedtime on day 1 (2.7 ± 3.0 *vs.* 1.3 ± 2.4%; $P = 0.004$). Greater stage 1 sleep ($\beta = 110$ kcal, 95% CI for $\beta = 42$ to 177 kcal; $P = 0.004$), and a trend for lower REM sleep ($\beta = -20$ kcal, 95% CI for $\beta = -41$ to 2 kcal; $P = 0.07$), durations were associated with greater EI between sleep restriction sessions. These findings suggest that the timing of a sleep restriction period impacts energy balance parameters. Additional studies are needed to corroborate these findings, given the increasing prevalence of shift workers and incidences of sleep disorders and voluntary sleep restriction.

1. Introduction

Borbély [1] 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 [2]. 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 [3]. Sleep restriction protocols [4,5] 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.

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Studies have reported mean increases of ≈300–500 kcal over 24 h following an imposed sleep restriction condition *vs.* a control condition (habitual sleep duration) [6–11]. However, the effects of imposed sleep restriction on energy expenditure (EE) are not as consistent; some studies reported no changes in EE inside a lab/inpatient clinic [8,12] and under free-living conditions [11], whereas others reported either greater [7] or lower [13] EE following similar sleep restriction protocols (1–2 nights of 4 h in bed/night). Studies have also reported negative associations between SWS duration and energy intake (EI) the following day under habitual sleep conditions [14], 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 [15]. Finally, Gonnissen et al. [16] reported greater post-dinner desire to eat ratings following 1 night of fragmented sleep, which caused a significant reduction in REM, but not SWS time, compared to 1 night of non-fragmented sleep (control condition).

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 and EE over 36 h. The secondary objective was to assess the strength of the associations between changes in sleep architecture with changes in next day EI and EE between sessions. It was hypothesized that sleep restriction with an advanced wake-time would lead to greater EI coupled with lower 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.

2. Materials and methods

2.1. 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 durations 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 [17] and EI [18]. 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.

2.2. Design and procedures

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. Fig. 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 24 h prior to the preliminary and experimental sessions. They were also asked not to consume caffeinated products after 12 h00 (noon), and to wash their hair in order to facilitate electrode installation on the day of each experimental session. Lastly, participants were asked if they felt well rested at the start of each experimental session. Compliance to these instructions was verified by self-report at the start of each session.

2.3. Preliminary session

Participants arrived at the lab at 8 h00 following an imposed 12 h overnight fast. At this time, height, body weight and body 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 g), peanut butter (*Kraft Smooth Peanut Butter®*; 60 g), cheddar cheese (*Cracker Barrel Marble Cheddar Cheese®*; 42 g) and orange juice (*Tropicana®*; 500 g). They were given 15 min 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

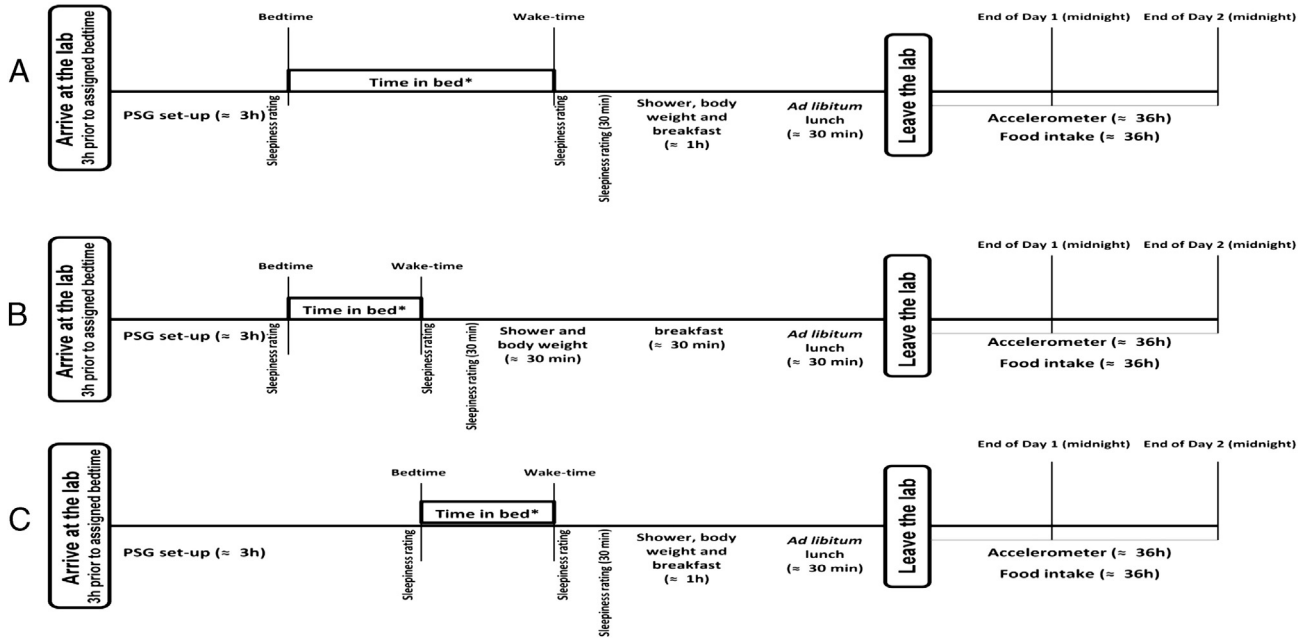

Fig. 1. Overview of the sleep protocol applied during each experimental session. A) Control session; B) Sleep restriction with an advanced wake-time; C) Sleep restriction with a delayed bedtime *Based on 2 weeks of accelerometry data.