SLEEPJ, 2020, 1–9

doi: 10.1093/sleep/zsaa062
Advance Access Publication Date: 1 April 2020
Original Article

ORIGINAL ARTICLE

Exercising before a nap benefits memory better than napping or exercising alone

Melodee Mograss[1,2,3,4,*], Monica Crosetta[2], Joanne Abi-Jaoude[2], Elizaveta Frolova[2,4], Edwin M. Robertson[5], Veronique Pepin[1,3,6] and Thien Thanh Dang-Vu[1,3,4*]

[1]Department of Health, Kinesiology and Applied Physiology, Concordia University, Montreal, QC, Canada, [2]Department of Psychology, Concordia University, Montreal, QC, Canada, [3]PERFORM Centre, Concordia University, Montreal, QC, Canada, [4]Centre de Recherche de l'Institut Universitaire de Gériatrie de Montréal, Montreal, QC, Canada, [5]Institute of Neuroscience & Psychology, University of Glasgow, Glasgow, UK and [6]Centre de recherche, Hôpital du Sacré-Cœur de Montréal, Montréal, QC, Canada

*Corresponding authors. Melodee Mograss, Department of Health, Kinesiology & Applied Physiology, PERFORM Centre, Concordia University, 7141 Sherbrooke St W, SP 165-27, Montreal, QC H4B 1R6, Canada. Email: melodee.mograss@concordia.ca; Thien Thanh Dang-Vu, Department of Health, Kinesiology & Applied Physiology, PERFORM Centre, Concordia University, 7141 Sherbrooke St W, SP 165-27, Montreal, QC H4B 1R6, Canada. Email: tt.dangvu@concordia.ca.

Abstract

Sleep leads to the enhancement of memory, and physical exercise also improves memory along with beneficial effects on sleep quality. Potentially, sleep and exercise may operate independently upon memory; alternatively, they may operate synergistically to boost memory above and beyond exercise or sleep alone. We tested this hypothesis in 115 young healthy adults (23 ± 3.9 years) randomly allocated to one of the four conditions in a 2 (exercise vs. no exercise) × 2 (nap vs. no nap) design. The exercise intervention consisted of a 40-minute, moderate intensity cycling, while the no exercise condition was an equivalent period of rest. This was followed by a learning session in which participants memorized a set of 45 neutral pictures for a later test. Subsequently, participants were exposed to either a 60-minute sleep period (nap) or an equivalent time of resting wakefulness, followed by a visual recognition test. We found a significant interaction between the effects of exercise and nap ($p = 0.014$, $\eta_p^2 = 0.053$), without significant main effects of exercise or nap conditions. Participants who experienced both exercise plus nap were significantly more accurate (83.8 ± 2.9) than those who only napped (81.1 ± 5.4, $p = 0.027$) and those who only exercised (78.6 ± 10.3, $p = 0.012$). Within the combined nap plus exercise group, higher recognition accuracies were associated with higher sleep spindle densities ($r = 0.46$, $p = 0.015$). Our results demonstrate that short-term exercise and a nap improve recognition memory over a nap or exercise alone. Exercise and sleep are not independent factors operating separately upon memory but work together to enhance long-term memory.

Statement of Significance

Both sleep and exercise have been shown to benefit memory, but little is known about how sleep and exercise interact to enhance memory consolidation. Here, we show that the combined effects of a nap and an acute exercise session upon human memory are substantially greater than the effects of each of them alone, in a sample of young healthy adults. Future studies should investigate the synergistic effects of sleep and exercise on cognition across the lifespan and in clinical populations. Our findings may have important implications for the design of future interventions aimed at promoting memory performance in populations vulnerable to cognitive impairments.

Key words: memory; exercise; sleep; nap; cognition

Submitted: 30 November, 2019; **Revised:** 3 March, 2020

Introduction

Physical exercise is becoming increasingly recognized as an important factor that can benefit human cognition and improve the quality of sleep [1, 2]. Exercise has been shown to help maintain or enhance different aspects of cognition [1, 2]. In two recent meta-analyses, a single bout of exercise typically lasting between 10 and 40 minutes led to *moderate-to-large* improvements in long-term memory in contrast to longer repeated bouts [1, 3]. The largest effects were seen when the participants were young adults of average fitness [1]. On the other hand, occasionally very little or no effect on cognition has been reported in the literature after high-intensity exercise or when the exercise type is running [4–6]. These beneficial effects of acute cardiovascular exercise on long-term memory were the greatest when the *exercise was completed prior to encoding*, possibly priming the appropriate mechanisms needed for learning and consolidation [1].

Studies have also shown that **exercise benefits sleep.** Self-reported studies have indicated that both moderate and regular physical activity have sleep-promoting benefits whereas exhaustive exercise of high intensity and long duration may at times be disruptive to sleep [4]. Kredlow *et al.* [5] showed significant changes in sleep architecture following a single bout of exercise. These authors reported that exercise improved sleep quality by increasing sleep duration, decreasing the time it takes to fall asleep, and increasing the amount of deep sleep. Similar results were found in a meta-analysis by Youngstedt *et al.* [6], who reviewed 38 studies exploring the relationship between acute exercise and nighttime sleep.

Sleep, like exercise, has been shown to **benefit cognitive** performance [7]. Non-Rapid Eye Movement (NonREM) sleep, particularly deep NonREM sleep (N3), has been shown to actively facilitate declarative memory, the type of long-term memory that relates to facts and events explicitly and intentionally learned and retrieved [8, 9]. Consistent with previous studies that employed a full night of sleep, it has been shown that even a brief NonREM nap compared with a period of wakefulness demonstrated improved performance for declarative memory tasks [8, 10]. For example, Alger *et al.* [11] found that those who achieved N3 sleep during a 60-minute nap had superior memory performance in a bimodal-paired association task compared with those who did not achieve N3 or those who remained awake.

In the last decade, increasing evidence has indicated a role for **brain oscillations** during sleep, measured by quantitative electroencephalography (EEG), in cognitive functioning. During NonREM sleep, brain activity is shaped by specific brainwaves produced by thalamocortical interactions [12]: spindles, which are 11–16 Hz waxing-and-waning oscillations, and large-amplitude, low-frequency slow waves (>75 microV, 0.5–4 Hz). In young adults, increases in sleep spindle activity were observed after learning a variety of memory tasks [13], including declarative tasks [14, 15]. In particular, the density of sleep spindles was significantly higher during sleep after learning the task compared with the non-learning control sleep [15].

While existing data show that both exercise and sleep have separate beneficial effects on declarative memory, it remains to be tested whether those effects are synergistic. In addition, what remains unclear is the mechanistic connection between exercise and cognition. We seek to test a largely overlooked potential mechanistic connection between physical exercise and cognition, namely the interaction with sleep. Given the paucity of literature on their combined effects, the current study aimed at assessing if the joint effects of sleep and physical activity go above and beyond the improvement conveyed by sleep or exercise alone.

Based on the literature, we hypothesized that: (1) an exercise intervention prior to memory encoding would enhance recognition memory after a nap, as compared with a nap or an exercise alone and (2) differences in spindle density during the post-learning nap would predict memory performance.

Methods

Participants

A final sample of 115 (45 males) healthy, yet sedentary, participants between the ages of 18 and 39 years (M_{age} = 23.31, *SD* = 3.9, see Table 1) was recruited. There were six participants who were excluded before the final sample due to the following reasons: taking nonprescription drugs (antihistamine, melatonin) (*N* = 2), noncompliance with the protocol (*N* = 1), extraneous lab noises during the nap (*N* = 2), and technical problems with the equipment (*N* = 1). Participants were recruited through local flyers and advertisements on the university campus and in the community. Exclusion criteria were a past or current history of psychiatric, neurological, or other medical condition; shift work and/or having traveled traveled through more than one time zone during the last month; sleep disorder; and highly active individuals (see screening assessments below). Any participants with uncorrected visual problems were excluded. None were using medications with a known effect on sleep or cognition.

Those potentially eligible after this prescreening were invited to our lab for a brief, in-person interview to further assess eligibility. During the interview, participants were asked to maintain regular sleep–wake schedules and to wear an actigraphy motion watch while completing a sleep diary for 10 days. Once recruited, the participants were randomly allocated to one of four separate groups: (1) Exercise plus nap **(ExNap group)** (2); Nap only **(NoExNap group)** (3) Exercise only **(ExNoNap group)**, and (4) neither exercise nor nap **(NoExNoNap group)** (see Figure 1). The Institutional Ethics Review Board at Concordia University approved this study, and all participants signed an informed consent form.

Procedure

All participants completed screening questionnaires evaluating the presence of sleep and mood disorders, level of physical activity, and subjective sleep quality to screen for the study's exclusion criteria (see the Assessments section below). Moreover, the questionnaires also collected basic demographic information (Table 1).

The morning before testing, the participants were asked to drink plenty of liquids, abstain from drinking caffeinated products, excessive smoking, or engaging in vigorous physical activity. They were also instructed to bring comfortable clothes and shoes as well as work to do between testing. On the testing day, participants arrived at 10:00 am, remained in the laboratory throughout the testing, and were instructed that the procedure would end at about ~05:30 pm. The actigraphy along with the sleep diary was reviewed for consistency of sleep periods prior

Table 1. Demographic variables and screening questionnaires (mean ± *SD*)

	ExNap (*N* =27)	NoExNap (*N* = 29)	ExNoNap (*N* = 30)	NoExNoNap (*N* = 29)
Sex F (M)	16 (11)	17 (12)	18 (12)	19 (10)
Age (yrs)	22.6 (3.5)	23.6 (4.0)	23.4 (3.8)	23.5 (4.4)
BMI (kg/m^2)	23.9 (3.7)	23.4 (4.7)	22.9 (3.0)	23.5 (4.0)
ESS	7.8 (3.1)	8.3 (3.5)	6.3 (3.9)	5.5 (2.6)
PSQI	4.4 (2.7)	4.5 (2.0)	4.5 (1.7)	4.2 (2.7)
ISI	4.4 (3.7)	6.2 (3.8)	4.2 (2.8)	3.6 (3.3)
SBQ	1.0 (0.7)	1.0 (0.7)	0.9 (0.6)	0.8 (0.6)
UNS	8.7 (3.4)	7.9 (3.1)	6.0 (2.8)	6.4 (2.8)
BAI	5.8 (5.6)	6.8 (6.8)	5.9 (5.2)	5.1 (4.7)
BDI-II	3.1 (2.7)	5.5 (5.7)	4.7 (4.8)	3.0 (4.1)
IPAQ	1619.2 (886.8)	1337.8 (828.4)	1470.1 (856.0)	1546.8 (870.3)
StudyVAS$_{arousal}$	2.6 (2.1)	2.5 (1.7)	1.8 (1.7)	1.9 (2.0)
TestVAS$_{arousal}$	2.3 (1.9)	2.4 (1.9)	1.7 (1.6)	1.6 (1.4)
StudyVAS$_{fatigue}$	4.7 (2.3)	3.6 (1.7)	4.6 (2.2)	4.5 (2.3)
TestVAS$_{fatigue}$	5.8 (2.5)	4.8 (2.4)	4.0 (2.4)	3.5 (2.7)
MEQ	47.2 (9.0)	45.7 (7.5)	48.9 (7.6)	50.7 (8.9)

N = 115. All comparisons between groups were nonsignificant.
BMI, Body Mass Index; BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; ESS, Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index; ISI = Insomnia Severity Index; SBQ,= Stop-Bang screening for sleep apnea; UNS, Ullanlinna Narcolepsy Scale; IPAQ, International Physical Activity Questionnaire; VAS, Visual Analog Scales for arousal and fatigue ratings given prior to the study and test sessions; MEQ, Morning Eveningness Questionnaire.

to the experimental procedure. On the day of the study, participants were randomly allocated to one of four groups. The participants were always monitored by a research assistant.

During the experimental procedure, participants underwent a 40-minute, moderate intensity cycling or sedentary procedure (e.g. sitting) at 11:00 am, followed by a light lunch at noon. This was followed by a study session (01:30 pm) and then a 60-minute nap or no nap (02:30 pm). At the test session (05:00 pm), participants completed a recognition memory task. Visual analog scales were administered 5 minutes before the study session and memory test to assess participants' level of arousal and fatigue. To control for time of day effects, the study session and the test session were kept constant +/−30 minutes (Figure 1).

Assessments

Sleep measures

Various aspects of subjective sleep quality were assessed using the Epworth Sleepiness Scale (ESS) [16] and the Pittsburgh Sleep Quality Index (PSQI) [17]. The ESS contains eight items assessing the likelihood that one would fall asleep in different situations and provides an assessment of daytime sleepiness [16]. The PSQI contains 19 items pertaining to sleep quality in the past month, with scores ranging from 0 (no difficulty sleeping) to 21 (severe difficulty sleeping) [17]. Higher scores indicate worse sleep quality. In order to assess sleep disorders in our participants, the Insomnia Severity Index (ISI) [18], the STOP-BANG Questionnaire (SBQ) [19], and the Ullanlinna Narcolepsy Scale (UNS) [20] were used to screen out participants with a high risk of insomnia disorder, obstructive sleep apnea, and narcolepsy, respectively. The ISI is a 7-item self-report questionnaire that assesses the severity of insomnia: scores less than 14 indicate mild or no clinically significant insomnia, whereas severe insomnia would be indicated by scores in the range of 22–28. An ISI > 14 was used as a cutoff score for exclusion [18]. The SBQ contains eight items assessing the risk of obstructive sleep apnea: answering "yes" to two questions or less indicates low risk, while answering "yes" to five or more indicates very high risk. Those with an SBQ score of ≥5 were excluded. The UNS includes four items evaluating the degree of narcoleptic symptoms and the risk of narcolepsy [19]: a total score greater than 14 is indicative of high suspicion of narcolepsy and was used as a cutoff score for exclusion.

Psychological questionnaires

To ensure that our participants were free of mood disorders, we used the Beck Anxiety Inventory (BAI) [21] and the Beck Depression Inventory (BDI-II) [22]. The BAI contains 21 items measuring the severity of anxiety symptoms. Items reflect the extent to which anxiety symptoms have bothered them in the past month, ranging from 0 (not at all) to 3 (severely). A BAI ≥ 36 was used as a cutoff score. The BDI-II contains 21 questions measuring the level of depressive symptoms. Responses for each item range from 0 (not indicative of depression) to 3 (highly indicative of depression). A cutoff score BDI-II > 20 was used for exclusion.

Physical activity

We used the short form of the International Physical Activity Questionnaire (IPAQ) [23] to assess participants' fitness levels. Questions on the IPAQ ask about latency and frequency of physical activity. Participants can be classified as being low, moderately active, or highly active. Only those who engaged in moderate levels of physical activity or less were included in the study. To meet each criterion, participants had to have reached a certain value of metabolic-equivalents-minutes (MET-minutes) in the past week. MET-minutes reflect the total energy cost of a person's physical activities. Those with MET-minutes scores under 600 were deemed low or inactive and those with scores from 600 to 3,000 were deemed moderately active. Those who scored higher than 3,000 METs were excluded.

Other measures

Visual Analog Scales (VAS) [24, 25] and the Morning-Eveningness Questionnaire (MEQ) for chronotype [26] were also obtained. Two VAS scales were used to assess differences in the participants' level of subjective arousal (VAS$_{arousal}$) and

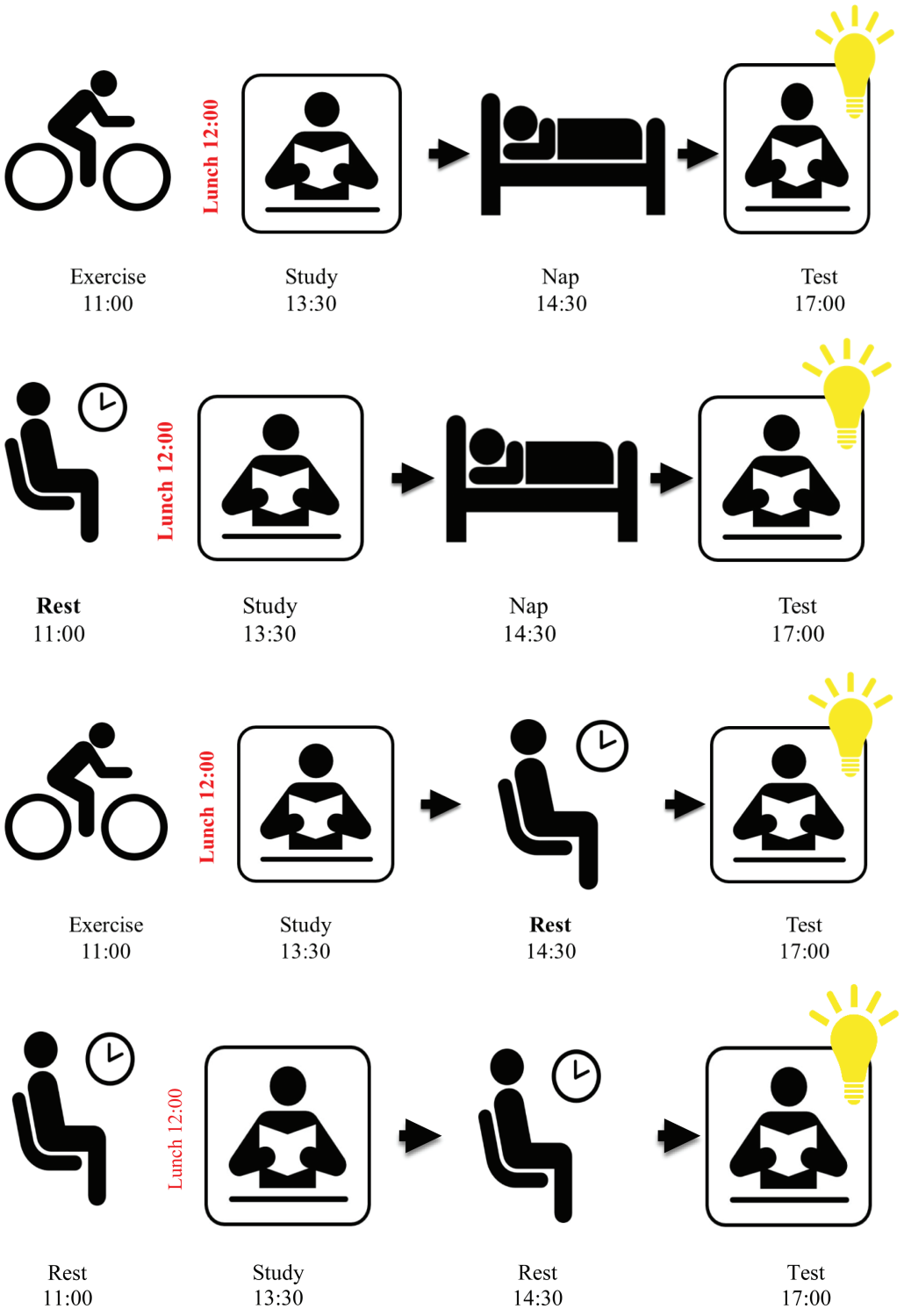

Figure 1. Experimental protocol.

fatigue ($VAS_{fatigue}$). Participants were asked to draw a slash on a 10-cm line at the point corresponding to their subjective mood 5 minutes prior to the study and test sessions. The $VAS_{arousal}$ consists of a 10-cm line ranging from "very calm" to "very excited." The results are computed as "calm" (0–2 cm), "within normal limits" (3–7 cm), and "excited" (8–10 cm). The $VAS_{fatigue}$ ranged from "very sleepy" at the left end to "very alert" at the right end. The results are computed as "sleepy" (0–2 cm), "normal" (3–7 cm), and "alert" (8–10 cm). The MEQ assesses whether someone is a morning or an evening chronotype or neither. It contains 19 questions, and a sum score of 41 and below indicates "evening types," scores of 59 and above indicate "morning types," and scores between 42 and 58 indicate "neither types."

Actigraphy screening.

For at least 10 days before coming in for the experimental procedure, participants were required to complete a sleep diary while wearing a motion wristwatch (Actiwatch Respironics). The watch measures motor activity and light exposure. Actigraphy was used to exclude those participants who did not adhere to consistent sleep/wake periods prior to the experimental procedure. The sleep diary was used in tandem, to resolve any ambiguity in the actiwatch histogram.

Exercise intervention

Based on previous research, it was determined that the exercise intervention needed to be of moderate intensity, in close proximity to learning, lasting ~30–40 minutes [6, 27, 28] and be performed >3 hours before sleep [5]. Moderate intensity exercise can be defined as achieving a rating of perceived exertion of 12 to 13 ("somewhat hard") on a 6–20 Borg Scale. For instance, walking at a brisk pace is considered a moderate intensity exercise.

Participants who had the exercise intervention (ExNap and ExNoNap groups) were required to cycle at moderate intensity on a stationary bike (Excite Med bike by Technogym). All those participants in the exercise conditions were able to complete the exercise protocol and mentioned that the exercise was "a little" challenging but not strenuous or impossible. Specifically, each participant's target heart rate was determined by using the following formula: (220 – age) × 0.6, corresponding to 60% of the age-predicted HR_{max} [29]. The exercise intervention was carried out by a licensed exercise physiologist and consisted of the following sequence: 5 minutes warming up, then 40 minutes at moderate intensity cycling, and finally 5 minutes cooling down [27]. Throughout the 40 minutes of moderate intensity cycling, participants were encouraged to maintain 60% of their maximum heart rate as determined by their age. Participants wore a heart monitor to keep track of their heart rate on the bike's digital display and were safely monitored by the exercise physiologist. A Borg rating scale of perceived exertion was completed throughout the procedure [30]. Those in the no exercise groups (NoExNap and NoExNoNap) were given a 40-minute sedentary procedure (sitting) instead.

Visual recognition task

We used a declarative memory task that was previously shown to benefit from NonREM sleep [31, 32]. There were two stages of the memory task: (1) the study session and (2) the test session. During the study (learning) session, participants were presented with 45 neutral photographs from the International Affective Pictures System (IAPS) [33]. The pictures were presented in the center of the screen at 1.5 m from the participant, sustaining a visual angle of 5 degrees. Each picture remained for 1,000 ms with an inter-stimulus interval of 7,000 ms. Participants were explicitly asked to study these photographs for a later memory test. These photographs were presented on a standard laptop computer using E-Prime (Psychology Software Tools, Pittsburgh, PA). During the study session, the participants were asked to rate the pictures as to how they felt (e.g. calm, neutral, or excited) when viewing the neutral scenes. This was to ensure that the participant attended to the stimuli. On average, this stage took approximately 5 minutes.

During the test session following the nap (or wake period), participants were presented with a set of 90 neutral photographs. Forty-five of these photographs were from the study session while the other 45 were "foils." Participants rated these stimuli as "old" (i.e. previously seen) or "foils" (i.e. not previously seen) by pressing a key on the keyboard (1 = old and 2 = foils). This stage took approximately 10 minutes to complete. Performance (accuracy) was calculated as the absolute number of correctly recognized items (maximum of 90 for the complete test session).

Nap/polysomnography

Participants in the nap groups (ExNap and NoExNap groups) were given a 60-minute NonREM nap, i.e. they were awoken ~60 minutes after sleep onset. Sleep onset latency (SOL) was defined as the period between lights out and beginning of N1 sleep (minimum three epochs of N1) or one epoch of any other sleep stage. EEG was referenced to the mastoids (0.3–100 Hz filter, sampling rate 512 Hz) and included frontal (F_3/F_4), central (C_3/C_4), occipital (O_1/O_2), and midline (F_{pz}, F_z, C_z, P_z, and O_z) sites. Other recorded signals in the polysomnography (PSG) included electrooculography, electromyography (submental, EMG_{chin}), and electrocardiography (ECG). Audiovisual recordings were also obtained. All participants, even those without a nap, underwent electrode application to ensure consistency in exposure between groups. Participants in the no-nap groups (ExNoNap and NoExNoNap) were instructed to sit upright at a table and were monitored closely by a research assistant. They were permitted to do their course work or read quietly. For the nap groups, a registered, experienced scorer monitored the *online* recording and scored the separate sleep stages according to standard scoring criteria from the American Academy of Sleep Medicine (AASM) [34]. The PSG variables during the nap included: total sleep time (TST), sleep stage percentage of TST (N1, N2, and N3), sleep efficiency (ratio between TST and time in bed after lights off), SOL (time to fall asleep after lights off), wake after sleep onset (WASO, total wake time after sleep onset), and number of awakenings.

Sleep spindle activity (11–16 Hz) on the central and frontal sites (C3-M2, C4-M1, and F3-M2) were automatically detected using the Domino version 2.6.0 (SOMNOscreen) system. Spindle data were extracted from artifact-free, NonREM sleep epochs (N2 and N3). For spindle detection, an adaptive and relative threshold was used based on standard settings. The parameters for spindle detection consisted of the following: 8-second time window for calculating a reference "baseline," relative threshold (250%) for the increased activity within the frequency range for spindle detection (11–16 Hz), min/max of 0.5 to 5.0 second duration of the spindle, and min/max amplitude of 5 to 300 μV of the spindle. Once detected, spindles were then visually reviewed and confirmed by an experienced, registered polysomnographer (M.M.). Spindles were quantified according to density (number per 30-second epoch) during NonREM N2 and N3 sleep. To quantify NonREM slow-wave activity (SWA), the EEG power in the SWA band (0.5–3.5 Hz) was computed automatically (Domino version 2.6.0, Somnomedics). For microarousals detection, the standard AASM criteria were used (e.g. ≥3 seconds of duration) [34], and microarousals were visually reviewed and confirmed by a registered polysomnographer (M.M.).

Data analysis

Statistical analyses were conducted with SPSS (version 17). We conducted a 2 × 2 ANOVA with the presence or absence of exercise and nap as factors, and performance accuracy as the main outcome variable. The main effects were reported first. When significant interaction occurred, follow-up post hoc *t*-tests were used to determine the differences between groups. In addition, independent *t*-tests were performed as exploratory analyses to determine if there were any differences in our sleep variables (TST, sleep efficiency, WASO, awakenings, SOL, microarousal index, sleep stages, SWA, and spindle density) between the two nap groups (ExNap and NoExNap). Pearson product–moment correlations were calculated between sleep spindle density and memory performance (accuracy) in the groups who napped (ExNap and NoExNap), combined and then separately for each group. For completeness, correlations were also calculated between performance accuracy and other sleep variables (TST, sleep efficiency, WASO, SOL, number of awakenings, microarousal index, % N1, N2, and N3, and SWA). When significant correlations were observed, we evaluated group differences in the correlation coefficients by using Fisher *r* to z transformations and comparing the z-values between the groups who slept (z test). In all tests, statistical significance was assumed at $p \leq 0.05$.

Results

Table 1 shows the demographic information, screening questionnaires (ESS, PSQI, ISI, SBQ, UNS, BAI, and BDI-II), and VAS and MEQ information of those who participated in the study. A total of 115 healthy, young adults with sedentary characteristics were divided into four groups: ExNap (n = 27), NoExNap (n = 29), ExNoNap (n = 30), and ExNoNap (n = 29). There were no group differences in the demographic and screening questionnaires. The participants were on average within the intermediate chronotype range (M = 48.1+/– SD = 8.3). The VAS scores were calculated for the study and test sessions, to provide subjective levels of arousal and fatigue. The $VAS_{arousal}$ ratings across the groups were characterized as "calm" with an average intensity of 2.2 cm +/– 1.8 and 2.0 cm +/– 1.8 for the study session and test session, respectively. The $VAS_{fatigue}$ ratings were characterized as within a normal range for fatigue (4.4 cm +/– 2.2 and 4.5 cm +/– 2.6 for study and test, respectively).

For our main analysis, a 2 × 2 ANOVA with exercise (yes/no) × nap (yes/no) was conducted to assess whether exercise, nap, or their interaction had an effect on memory performance accuracy. Results indicated a significant nap × exercise interaction on performance accuracy [F (1, 111) = 6.2; p = 0.014, η_p^2 = 0.053], yet no significant main effects for exercise or nap [F (1,111) = 0.09, p = 0.764 η_p^2 = 0.001; F (1,111) = 3.7, p = 0.069, η_p^2 = 0.029, respectively]. Post hoc tests showed that the ExNap group was more accurate than the NoExNap (M = 83.8 +/– SEM 0.56 vs. 81.1 +/– 1.0, t (54) = 2.47; p = 0.027, Cohen's d = 0.61) and the ExNoNap groups (M = 78.6 +/– 1.9, t (55) = 2.52; p = 0.012, d = 0.68). In addition, there was a strong *trend* toward a difference in accuracy between the ExNap group and the NoExNoNap group (81.9 +/–0.77, t (54) = 1.91; p = 0.058, d = 0.51) (Figure 2). There were no other significant differences in accuracy between groups: NoExNap versus ExNoNap, p = 0.23, d = 0.31; NoExNap versus NoExNoNap, p = 0.53, d = 0.16; and ExNoNap versus NoExNoNap, p = 0.12 and d = 0.42.

For the secondary exploratory analysis, we examined the effects of administering exercise prior to a NonREM nap on sleep variables. The sleep stage durations for each group were: ExNap group, Wake = 22.6 +/– 16.5 minutes, N1 = 3.9 +/– 2.5 minutes, N2 = 22.7 +/– 9.5 minutes, N3 = 20.4 +/– 14.1 minutes; NoExNap group, Wake = 22.8 +/– 14.1 minutes, N1 = 3.4 +/– 2.5 minutes, N2 = 24.3 +/– 9.2 minutes, and N3 = 20.2 +/– 13.4 minutes (see values in % of TST in Table 2). An independent *t*-test showed that there was no difference in sleep stages between the two nap groups (Table 2).

We conducted Pearson correlations between NonREM spindle density and performance. For the combined nap groups (N = 56, ExNap and NoExNap), there were no significant

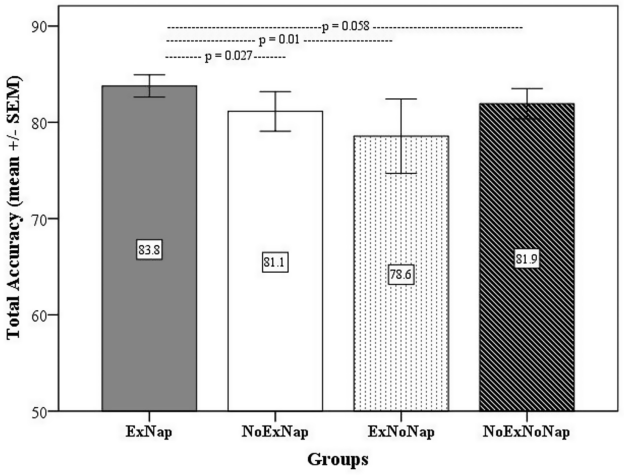

Figure 2. A 2 × 2 ANOVA revealed significant nap × exercise interaction on memory performance accuracy (mean number of stimuli including studied and foils), p < 0.02. Significant differences using post hoc *t*-tests are shown here (see main text for more details). Gray bar—ExNap, exercise plus nap group, N = 27; white bar—NoExNap, nap only group, N = 29; dotted bar—ExNoNap, exercise only group, N = 30; stripped bar—NoExNoNap, group without nap and without exercise, N = 29.

Table 2. Polysomnographic nap data (mean ± *SD*)

	ExNap (N = 27)	NoExNap (N = 29)
TST (min)	46.7 (13.1)	47.8 (12.4)
SE (%)	68.4 (21.3)	68.4 (18.6)
SOL (min)	8.1 (5.6)	10.1 (5.3)
AWK (number)	4.5 (1.9)	5.3 (2.6)
WASO (min)	22.6 (16.6)	22.8 (14.1)
Microarousal (nb/hr)	20.3 (13.4)	22.4 (15.7)
N1 (%)	9.47 (6.1)	8.4 (8.9)
N2 (%)	51.4 (19.9)	52.9 (18.5)
N3 (%)	39.9 (22.2)	39.0 (22.5)
SWA	47.0 (7.3)	48.4 (6.5)
Spindle density (nb/epoch)	3.1 (1.0)	3.3 (1.3)

N = 56. Independent *t*-tests: all p-values > 0.05.
Groups: ExNap, exercise plus nap, NoExNap, nap only.
TST, total sleep time; SE, sleep efficiency; SOL, sleep onset latency; AWK, number of wake periods during time in bed; WASO, wake after sleep onset; Microarousal index = number of awakenings ≥3sec but < 15sec per hour; Sleep stage N1, N2, and N3; SWA = slow-wave activity during NonREM (N2 and N3); Spindle density = spindle number per 30-sec epoch during NonREM (N2 and N3).

correlations between performance improvement and NonREM spindle density during N2 and N3 (nb per 30-second epoch) (Pearson: $r = 0.063$, $p = 0.65$). When separating the two groups, the *exercise plus nap* group ($N = 27$, ExNap) showed a positive correlation between spindle density and performance accuracy ($r = 0.46$, $p = 0.015$) (Figure 3). For the *nap only* group ($N = 29$, NoExNap), we did not find a significant correlation between performance and spindle density ($r = -0.04$, $p = 0.83$). We compared these correlation coefficients between ExNap and NoExNap groups and found a strong *trend* for a group difference in the correlation between spindle density and memory performance ($z = -1.63$, $p = 0.0516$). Finally, there were no significant correlations between performance and any of the other sleep variables (TST, sleep efficiency, WASO, SOL, number of awakenings, microarousal index, % N1, N2, and N3, and SWA), both in groups combined and in each group separately (see Supplementary Tables S1–S6).

Discussion

The primary aim of the present study was to decipher if the joint effects of physical activity and sleep go above and beyond the improvement conveyed by exercise or sleep alone. We found support for our hypothesis that a 60-minute NonREM nap and a single aerobic exercise session have synergistic, complementary effects on long-term memory, in healthy young sedentary adults. The accuracy of the exercise plus nap group was higher than that of the other groups, in agreement with our hypothesis. Our results are in line with the work of Wilckens *et al.* [35] who investigated sleep and exercise's influence on cognitive functioning in older adults. The results showed that sleep was a statistically significant mediator between METs, an indicator of physical activity, and cognitive functioning [35]. A limitation in Wilckens *et al.*'s [35] study was that sleep was not measured directly. Instead of PSG, both physical activity and sleep were measured by an accelerometer armband that monitors motor activity [35]. Our study adds to the existing literature by using a polysomnographic nap to investigate the joint influence of exercise and sleep on recognition memory. To our knowledge, this study is the first investigation of the combined benefits of short-term exercise and a brief polysomnographic nap on long-term memory in young adults controlling for level of fitness.

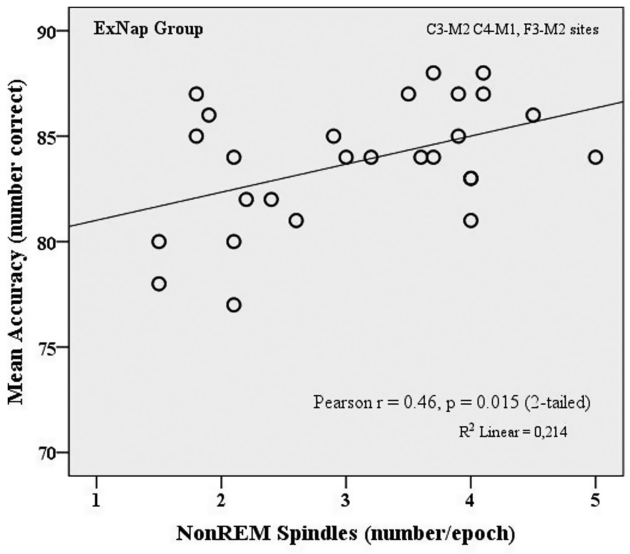

Figure 3. Pearson correlation between memory performance (accuracy) and sleep spindle density (number/30-second epoch during N2 and N3 sleep) in the group who exercised and napped (ExNap).

As mentioned in the Introduction section, a meta-analysis on the effects of cardiovascular exercise on memory showed that acute exercise has a small but beneficial effect on verbal, auditory, and procedural memory [1]. The data in our study do not support the idea that short-term exercise alone result in improvements in memory performance. This contrasts with a study by van Dongen *et al.* [36] who showed that a single bout of exercise performed after learning improved picture recall 48 hours later. However, these authors did not look at sleep in their respective study.

The literature on studies investigating the effectiveness of a nap in comparison with a wake control on declarative tasks have shown contradictory results [10, 37]. We found no difference between the nap only group (NoExNap) and those who remained awake on memory performance. This contrasts with a study by Lahl *et al.* [38] showing that even shorter daytime nap significantly enhances recall. However, Backhaus *et al.* [37] reported no difference between a 60-minute nap group and a wake control group on recall performance. Although Tucker *et al.* [10] found significant memory improvements following a 60-minute nap compared with a wake control, this effect was only found for one of the three memory tasks. Subsequently, when these authors divided up the group into low and high performers, their results showed that the low performers' improvements were similar across the nap versus wake, whereas the high performers did significantly better after napping compared with wake [10]. Memory performance benefits obtained during sleep are thus complex, as performance gains may be tied to task difficulty, learning strategies, attentional processes, or even the type of memory task (recall vs. recognition).

Our study shows that a nap alone is not sufficient to benefit recognition memory as compared with a wake interval. In contrast, we observed that a nap may benefit recognition memory when learning is preceded by exercise. What could make this combination of exercise and sleep so unique for memory performance? A possibility might reside in the effects of exercise on the encoding processes during learning, which might, in turn, trigger memory consolidation processes during subsequent sleep. There is some evidence that exercise impacts memory encoding. For example, Schmidt-Kassow *et al.* [39] showed that light-to-moderate intensity exercise during vocabulary learning increased retrieval performance. In line with our data, Salas *et al.* [40] showed that exercise such as brisk walking prior to learning but not prior to retrieval significantly increased recall performance scores. These findings suggest that exercise might have a direct effect on encoding processes, leading to better memory recall. However, some findings also suggest that exercise may impact memory consolidation processes taking place after learning. For instance, Coles *et al.* [41] demonstrated that moderate intensity cycling after encoding also facilitates consolidation. After learning word lists, participants who exercised maintained their recall performance, while those who did not showed a performance decrease. These authors suggested that exercise-induced arousal may facilitate the consolidation of information into long-term memory [41]. This is, however, hard to reconcile with our data in which there was no significant

difference in subjective arousal scores across the groups when the $VAS_{arousal}$ was given prior to the study session or prior to the test retrieval session (see Table 1).

Another line of explanation for the synergistic effects of exercise and sleep might reside in the impact of exercise on sleep architecture. We indeed expected that those participants who exercised plus napped compared with those who only napped (ExNap vs. NoExNap) would have significant improvements in sleep. This was, however, not the case. We found that the amount of N3 sleep was not different between those who exercised plus napped compared with the nap only group (ExNap vs. NoExNap). This observation is consistent with an early review by Trinder *et al.* [42] who reported that most studies looking at the effects of exercise on sleep failed to show a significant increase in N3 following exercise. These results are also comparable to more recent work of Wong *et al.*, [43] who found that those who ran for 40 minutes at moderate intensity were not significantly different on N3 sleep during the night compared with those who did not. Others have attributed the lack of effects of exercise in improving sleep perhaps due to the fact that aspects of sleep expressed in daytime naps differ from those of a full night (reduced sleep cycles in naps) and may not be fully adequate to achieve robust results [6]. It may also be that in most of these studies the focus was on good sleepers with little or no room for improvement (Youngstedt *et al.* [6]). Future studies are needed to look at the effect of exercise on sleep quality in populations with disturbed or reduced sleep.

A third possibility consists in the potential effects of exercise on the neural processes underlying memory consolidation during sleep. Specific electrophysiological phenomena (e.g. spindle oscillations) in NonREM sleep are believed to be involved in sleep-related memory consolidation [44]. Despite the absence of significant change in spindle density during the nap after exercise compared with the nap with no prior exercise, we did find a positive correlation between spindle activity and recognition memory accuracy in those who exercised plus napped. Recent data have shown that memory consolidation during sleep may involve spindle-related processes, such as changes in brain responses to spindles, without a change in the number or the density of sleep spindles [45]. Thus, it appears that sleep spindles may contribute to the higher accuracy in the exercise-plus-nap group. Schabus *et al.* [46] found that sleep spindle density in NonREM sleep accounted for superior memory performance post learning. In fact, sleep spindle density has been directly correlated with recall performance after sleep in several studies [13, 15]. As to why it was not observed in those who only napped without exercise remains unclear. It is possible that such a lack of association between spindle density and performance in the nap only group was due to our visual recognition task being too easy for some participants. Schmidt *et al.* [47] investigated the effects of previous declarative learning on sleep architecture and spindle oscillations during a daytime nap, and whether the encoding difficulty modulated the impact. They showed evidence that learning a declarative memory task before a nap leads to increased post-training sleep spindles, but only after the difficult encoding condition.

A limitation of our study is that we did not evaluate the effects of exercise on encoding processes or on the immediate recall performance after learning. Our study was indeed focused on evaluating whether exercise before learning followed by a nap would improve recognition memory compared with exercise or nap alone. Future studies should evaluate the effects of exercise, sleep, and their combination on other memory tasks. For instance, using a declarative memory protocol in which post-exercise learning would be followed by an immediate recall (before sleep or wake) and a delayed recall (after sleep or wake) would allow to evaluate the effects of exercise on both the early stages following encoding and the later memory consolidation processes. Our study may also pave the way for protocols adding event-related potentials or functional brain imaging during the learning task in order to provide insight into the effects of exercise on the encoding processes and their neural signatures. Finally, our study was not designed to differentiate between the effects of exercise on memory encoding and those of exercise on memory consolidation processes during sleep. Future studies may include groups in which exercise would take place after learning (and before sleep) as compared with before learning and sleep in order to further evaluate whether exercise affects encoding processes, sleep-related processes, or both.

In conclusion, the combination of acute cardiovascular exercise and a NonREM nap benefits recognition memory over a nap or exercise alone. These data support our hypothesis and demonstrate a synergistic effect between exercise and sleep. Exercise and sleep are not independent factors operating upon memory individually but rather work together to enhance long-term memory. Further studies are needed to improve our knowledge of the complex physiological mechanisms underlying these effects and to investigate the benefits of exercise in the promotion of sleep and memory in those whose sleep is compromised.

Supplementary Material

Supplementary material is available at *SLEEP* online.

Acknowledgments

We wish to thank our participants for their participation in this study, as well as our sleep lab volunteers, interns, and students (Cassandra Duguay, Elisabeth Houle, Shira Azoulay, Arshi Kaffash, Anthony Thorburn, Alex Nguyen, Steven Bouganim, Brent Rosenstein, Umberto Moretto, Joelle Ducharme, Julianna Ricchiuti, Jennifer Suliteanu, Katherine Walker, Brian Hodhod, Agata Kasprzyk, Tiffany Samaan, Teresa Johnson, Tehila Zvionow, Christy Lo, Matthew Kaba Aboud, Corina Moraru, Lukia Tarelli, Milan Barade, Diaa Chalati, and Kirsten Ka Mon Gong) for their technical support.

Funding

This research was supported by the American Sleep Medicine Foundation Focus Project Award (MM, TDV), the Natural Sciences and Engineering Research Council of Canada (TDV), and the Canada Foundation for Innovation (TDV). TDV is also supported by the Canadian Institutes of Health Research (MOP 142191, PJT 153115, PJT 156125, and PJT 166167), the Fonds de Recherche du Québec—Santé and Concordia University.

Conflict of interest statement. TDV received consultant fees from Eisai. The other authors report no financial or nonfinancial disclosure.

References

1. Roig M, *et al*. The effects of cardiovascular exercise on human memory: a review with meta-analysis. *Neurosci Biobehav Rev*. 2013;**37**:1645–1666.
2. Smith PJ, *et al*. Aerobic exercise and neurocognitive performance: a meta-analytic review of randomized controlled trials. *Psychosom Med*. 2010;**72**:239–252.
3. Verburgh L, *et al*. Physical exercise and executive functions in preadolescent children, adolescents and young adults: a meta-analysis. *Br J Sports Med*. 2014;**48**:973–979.
4. Driver HS, *et al*. Exercise and sleep. *Sleep Med Rev*. 2000;**4**:387–402.
5. Kredlow MA, *et al*. The effects of physical activity on sleep: a meta-analytic review. *J Behav Med*. 2015;**38**:427–449.
6. Youngstedt SD, *et al*. The effects of acute exercise on sleep: a quantitative synthesis. *Sleep*. 1997;**20**:203–214.
7. Rasch B, *et al*. About sleep's role in memory. *Physiol Rev*. 2013;**93**:681–766.
8. Gais S, *et al*. Sleep after learning aids memory recall. *Learn Mem*. 2006;**13**:259–262.
9. Marshall L, *et al*. The contribution of sleep to hippocampus-dependent memory consolidation. *Trends Cogn Sci*. 2007;**11**:442–450.
10. Tucker MA, *et al*. Enhancement of declarative memory performance following a daytime nap is contingent on strength of initial task acquisition. *Sleep*. 2008;**31**:197–203.
11. Alger SE, *et al*. Slow wave sleep during a daytime nap is necessary for protection from subsequent interference and long-term retention. *Neurobiol Learn Mem*. 2012;**98**:188–196.
12. Steriade M. Grouping of brain rhythms in corticothalamic systems. *Neuroscience*. 2006;**137**:1087–1106.
13. Clemens Z, *et al*. Overnight verbal memory retention correlates with the number of sleep spindles. *Neuroscience*. 2005;**132**:529–535.
14. Fogel SM, *et al*. Learning-dependent changes in sleep spindles and Stage 2 sleep. *J Sleep Res*. 2006;**15**:250–255.
15. Gais S, *et al*. Learning-dependent increases in sleep spindle density. *J Neurosci*. 2002;**22**:6830–6834.
16. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*. 1991;**14**:540–545.
17. Buysse DJ, *et al*. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;**28**:193–213.
18. Morin CM, *et al*. The Insomnia Severity Index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*. 2011;**34**:601–608.
19. Chung F, *et al*. High STOP-Bang score indicates a high probability of obstructive sleep apnoea. *Br J Anaesth*. 2012;**108**:768–775.
20. Hublin C, *et al*. The Ullanlinna Narcolepsy Scale: validation of a measure of symptoms in the narcoleptic syndrome. *J Sleep Res*. 1994;**3**:52–59.
21. Beck AT, *et al*. An inventory for measuring clinical anxiety: psychometric properties. *J Consult Clin Psychol*. 1988;**56**:893–897.
22. Beck AT, *et al*. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *J Pers Assess*. 1996;**67**:588–597.
23. Craig CL, *et al*. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc*. 2003;**35**:1381–1395.
24. Lee KA, *et al*. Validity and reliability of a scale to assess fatigue. *Psychiatry Res*. 1991;**36**:291–298.
25. Wright KP Jr, *et al*. Relationship between alertness, performance, and body temperature in humans. *Am J Physiol Regul Integr Comp Physiol*. 2002;**283**:R1370–R1377.
26. Horne JA, *et al*. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *Int J Chronobiol*. 1976;**4**:97–110.
27. Kashihara K, *et al*. Positive effects of acute and moderate physical exercise on cognitive function. *J Physiol Anthropol*. 2009;**28**:155–164.
28. Kubitz KA, *et al*. The effects of acute and chronic exercise on sleep. A meta-analytic review. *Sports Med*. 1996;**21**:277–291.
29. Gellish RL, *et al*. Longitudinal modeling of the relationship between age and maximal heart rate. *Med Sci Sports Exerc*. 2007;**39**:822–829.
30. Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehabil Med*. 1970;**2**:92–98.
31. Mograss M, *et al*. The ERP old-new effect: a useful indicator in studying the effects of sleep on memory retrieval processes. *Sleep*. 2006;**29**:1491–1500.
32. Mograss MA, *et al*. Event-related potentials differentiates the processes involved in the effects of sleep on recognition memory. *Psychophysiology*. 2008;**45**:420–434.
33. Lang PJ, *et al*. Emotion and motivation: measuring affective perception. *J Clin Neurophysiol*. 1998;**15**:397–408.
34. Iber C, *et al*. *The AASM Manual for the Scoring of Sleep and Associated Events*. Westchester: American Academy of Sleep Medicine; 2007.
35. Wilckens KA, *et al*. Physical activity and cognition: a mediating role of efficient sleep. *Behav Sleep Med*. 2018;**16**:569–586.
36. van Dongen EV, *et al*. Physical exercise performed four hours after learning improves memory retention and increases hippocampal pattern similarity during retrieval. *Curr Biol*. 2016;**26**:1722–1727.
37. Backhaus J, *et al*. Daytime naps improve procedural motor memory. *Sleep Med*. 2006;**7**:508–512.
38. Lahl O, *et al*. An ultra short episode of sleep is sufficient to promote declarative memory performance. *J Sleep Res*. 2008;**17**:3–10.
39. Schmidt-Kassow M, *et al*. Exercising during learning improves vocabulary acquisition: behavioral and ERP evidence. *Neurosci Lett*. 2010;**482**:40–44.
40. Salas CR, *et al*. Walking before study enhances free recall but not judgement-of-learning magnitude. *J Cogn Psychol*. 2011;**23**:507–513.
41. Coles K, *et al*. Effects of acute exercise on executive processing, short-term and long-term memory. *J Sports Sci*. 2008;**26**:333–344.
42. Trinder J, *et al*. The effect of exercise on sleep: the negative view. *Acta Physiol Scand Suppl*. 1988;**574**:14–20.
43. Wong SN, *et al*. The effects of moderate to vigorous aerobic exercise on the sleep need of sedentary young adults. *J Sports Sci*. 2013;**31**:381–386.
44. Gais S, *et al*. Declarative memory consolidation: mechanisms acting during human sleep. *Learn Mem*. 2004;**11**:679–685.
45. Jegou A, *et al*. Cortical reactivations during sleep spindles following declarative learning. *Neuroimage*. 2019;**195**:104–112.
46. Schabus M, *et al*. Sleep spindles and their significance for declarative memory consolidation. *Sleep*. 2004;**27**:1479–1485.
47. Schmidt C, *et al*. Encoding difficulty promotes postlearning changes in sleep spindle activity during napping. *J Neurosci*. 2006;**26**:8976–8982.