夕方に行う 10km 走が午後の昼寝によって悪化した夜間睡眠に及ぼす影響
Effects of an Early Evening 10-km Run on Nocturnal Sleep Homeostatically Degraded by an Afternoon Nap

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Key Words: exercise, sleep, PSG, ceiling and floor effect

Abstract

We examined the effect of acute exercise performed after an afternoon nap intended to homeostatically degrade the following night's sleep quality. Nine healthy young people unaccustomed to regular exercise ran for 10-km after an afternoon nap. Polysomnographic (PSG) measurements were carried out during night sleep and during naps in three different conditions: Baseline night (Nt0), Nap (Np1) + night after no run (Nt1), and Nap (Np2) + night after 10 km run (Nt2). Standard visual sleep stage scoring and Fast Fourier Transformation (FFT) analysis of the electroencephalogram (EEG) were performed on 30 sec epochs. For the seven subjects who met sleep parameter inclusion criteria, sleep efficiency (SE), total sleep time (TST), and duration of stage REM for Nt1 condition within sleep variables were significantly less than those of the Nt0 condition by 7.54 ± 6.57%, 36.21 ± 31.54 min, and 23.07 ± 21.63 min, respectively. There were no significant differences between Nt1 and Nt2 in duration of each sleep stage except the point that the total delta power of Nt2 was significantly higher than that of Nt1. In conclusion, the total delta power of Nt2 condition was higher than that of Nt1, suggesting that 10 km run improved following night sleep. The necessity of computerized quantitative analysis in such fine changes were also confirmed.

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Ⅰ.Introduction

Exercise is commonly believed to have favorable effects on night-time sleep quality. In a survey that solicited public opinions of the best behaviors for improving the quality of nightly sleep, exercise was top-ranked (Urponen, Vuori, Hasan, & Partinen, 1988).

There are, however, experimental investigations of the relations between exercise and sleep which have not consistently shown benefits of exercise on sleep. Many researchers have examined the effects of acute exercise to explore their impact on sleep. To the best of our knowledge, Baekeland and Lasky (1966) were among the first to study the relationship between exercise and subsequent sleep. They concluded that strenuous exercise resulted in an increased amount of slow-wave sleep (SWS) on the following night. Walker et al. (1978) tested the SWS-exercise hypothesis that slow EEG activity would be increased with exercise. Although they failed to support the hypothesis, they showed that athletes (runners) showed more non-rapid eye

movement (NREM) sleep and less eye movement activity during REM sleep than non-athletes. Kubitz et al. (1996) conducted a meta-analytic survey of 32 studies and concluded that acute exercise increases the total sleep time (TST) of sleep stages 3 and 4, reduces sleep onset latency (SOL), decreases the sleep stage REM, and prolongs REM latency (RL). Nevertheless, some reports contradict such positive expectations for exercise benefits on sleep (O'Connor, Breus, & Youngstedt, 1998; Youngstedt, Kripke, & Elliott, 1999). Youngstedt et al. (1997) conducted a meta-analysis of 38 studies and reported only slight overall effects of acute exercise on following sleep. Thus, exercise has not been clearly shown to improve sleep to any great degree. However, that meta-analysis included data from many studies that were not computer analyzed, but only visually scored. Visual scoring is, by design, a data reduction method for tabulating uniform length analysis epochs in which the distinctions among NREM sleep stages 2, 3, and 4 are arbitrary categorizations based on the amplitude and abundance of visually recognizable slow-waves in the EEG (Uchida, Feinberg, March, Atsumi, & Maloney, 1999).

In the present article, we define Slow Wave Sleep (SWS) as the summation of uniform 30-second sleep stage epochs scored as stages 3 and 4, while we define Slow Wave Activity (SWA) as the summation of delta band EEG activity of sleep stages 2, 3, and 4 within those epochs by Fast Fourier Transformation (FFT) analysis. Note that the inclusion of stages 2, 3, and 4 in SWA reflects the continuous nature of slow-wave expression that can be captured using computer analysis, rather than the cutoff imposed for stage 2 by visual analysis when less than 20% of an epoch contains slow-waveforms. Therefore, to assess subtle changes in SWS accurately, we evaluated sleep quality using both visual sleep stage scoring and Fast Fourier Transformation (FFT) analysis of the electroencephalogram (EEG). A closer examination of prior experiments reveals that past research focused on healthy young people, which makes the conflicting findings controversial. Because young subjects intrinsically sleep well, the effects of acute exercise might be evident as only slight positive (or negative) effects on intrinsically good sleep (ceiling and floor effects) (Youngstedt, 2003; Youngstedt, 2005). Therefore, experiments designed to minimize the ceiling and floor effects are needed. For this reason, we conducted a unique experiment in which the nightly sleep of participants was degraded by an afternoon nap. Thus, we measured the effect of acute exercise on participants' quality of sleep, using polysomnography (PSG) and computer analysis to examine both SWS and SWA changes during degraded night-time sleep after substantial exercise.

Driver et al.(2000) reported that "the duration of exercise was a more consistent variable on the acute effects of exercise on sleep than other factors considered including fitness and time of day, with the most reliable effects only observed following exercise lasting more than 1 h in physically fit individuals". Thus we adopted 10 km self-paced run as an approximately aerobic exercise in this study.

Ⅱ.Methods

1. Participants

The participants were nine males. Some of the participants used to participate in athletic teams in high school days, but none of them had regular exercise in past three years. Their physical characteristics (mean ± standard deviation) were 23 ± 1 years old, height 170.8 ± 6.8 cm, weight 61.2 ± 4.8 kg, and body mass index (BMI) 21 ± 3. However, two participants were excluded as bad sleepers (discussed

below). Hence, the physical characteristics of seven participants were: 23 ± 1 years old, height 172.6 ± 4.6 cm, weight 61.4 ± 4.9 kg, and body mass index (BMI) 21 ± 2. The participants were instructed to maintain a regular sleep wake schedule (23:00-07:00) for one week prior to the experiment. For the three prior days and during the experimental period, they were told not to consume alcohol or caffeine and were prohibited from sleeping at any time outside the scheduled period. The Ethics Board of the Faculty of Sport Sciences at Waseda University reviewed and approved the experimental program used for this research. The participant candidates acknowledged receiving sufficient information about this experiment and gave their informed consent to participate.

2. Experimental design

The participants slept in an electromagnetically and sound shielded room maintained at 26°C and 50% relative humidity. The first experimental day was reserved for adaptation to night sleep in the laboratory with electrodes mounted, scheduled as presented in **Figure 1**. Data from the second day were used as a baseline condition (Nt0). On the third day, participants took an afternoon nap but did not run (nap+no run condition: Np1+Nt1). Participants then took a one-week break to recover the standard sleep schedule. On the final day of experiments, they took an afternoon nap and ran 10 km (nap+run condition: Np2+Nt2). The acute exercise they performed was a 10 km self-paced run (four laps on a 2.5 km course) at their own pace starting at 17:00. From 23:00 to 7:00, they were in bed for night sleep. Two hours were allocated for a nap during 14:00–16:00, which had been shown to degrade the following night's sleep (Feinberg, Maloney, & March, 1992). Experiments were performed in August and September 2006.

The experiment was not counterbalanced to exclude any possible long term effects of the unaccustomed 10 km run on sleep one week after the run.

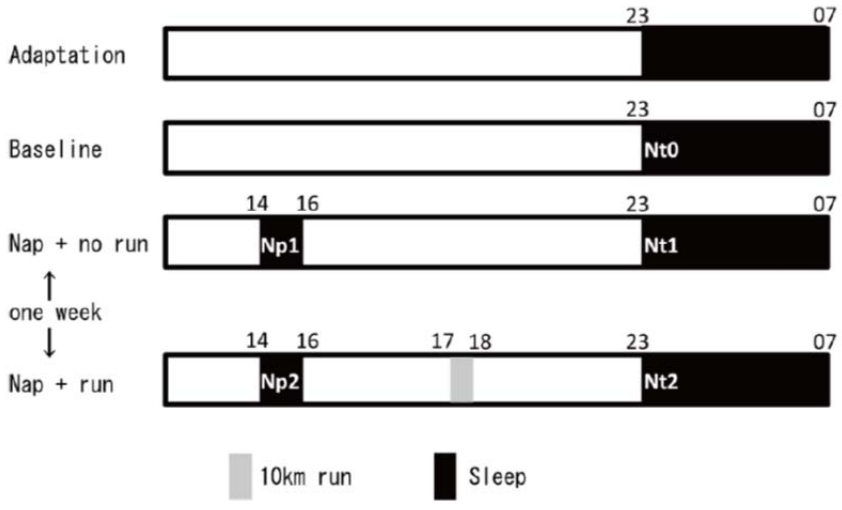

Figure 1. Experimental schedule

3. Polysomnographic (PSG) measurements

PSG measurements were made of nightly sleep for four days, and of afternoon naps for two days, using a portable device (Polymate; TEAC Corp., Japan). Electrodes for recording EEG were placed according to the international 10/20 system, at C3 and C4 and at O1 and O2 referred to contralateral earlobe electrodes (A1 and A2). For recording the electrooculogram (EOG), electrodes were

mounted above, below, left, and right of the both eyes to monitor vertical and horizontal eye movements. Electromyography (EMG) was conducted using electrodes on both sides of the chin. A single lead electrocardiogram (ECG) was recorded from electrodes affixed to the skin above the upper part of the both sides of clavicle.

The PSG acquisition sampling frequency was 200 Hz using time constants of 0.3 s for EEG and EOG, 0.01 s for EMG, and 1.50 s for ECG. High-cut filters were applied to suppress frequencies above 100 Hz for EEG, EOG, and ECG, and 200 Hz for EMG.

4. Data analysis: polysomnography

For each participant, visual sleep stage scoring was performed on the baseline night's sleep (Nt0), nap (Np1) and night sleep (Nt1) of no-run days, and nap (Np2), and night sleep (Nt2) of run days. Visual scoring was carried out on 30-second epochs using the original criteria standardized by Rechtschaffen and Kales (1968). Scorings were done by three authors (Kohei Shioda, Teruyo Mitsuyama, and Kouji Yoshikawa) and data conditions were blinded to the scorers.

Using Fast Fourier Transformation (FFT) analysis, we determined the delta power of the C3 EEG in the delta frequency band (0.3–3 Hz). We used our original software tested in past studies (Hirai, Uchida, Maehara, Okubo, & Shimizu, 1999; Nishida et al., 2004; Uchida, Maehara, Hirai, Okubo, & Shimizu, 2001). FFT analyses were performed on six 1024-point (5.12 s) epochs. A Hanning window was used for each epoch prior to FFT analysis. The alternate C4 EEG was analyzed for one participant whose C3 signal was contaminated with excessive continuous artifact. We defined total delta power as the sum of delta power from all epochs scored as stages 2, 3, and 4, excluding epochs contaminated byartifacts. We then calculated the average delta power as the total delta power divided by the number of epochs that were free of artifacts.

5. Subjective sleepiness and heart rate during exercise

To measure subjective sleepiness, we asked participants to fill in the OSA inventory survey (Oguri, Shirakawa, & Azumi, 1985) after waking from the nap and after waking in the morning, as well as visual analogue scales before nap and before night sleep. For visual analogue scales, participants were instructed to place a tick mark on each scale to show their feelings in terms of both arousal and sleepiness. The visual analogue scales were presented as a straight

line of 100 mm without any tick marks. The term “very much” was presented on the far right of the scale, and the term “not at all” was presented on the far left. Heart rate was recorded during the 10 km run using a heart rate monitor (S810i; Polar Electro JP Ltd.).

6. Statistical analysis

We excluded two participants as bad sleepers based on the following criteria. Those with sleep efficiency on the baseline below 80% and those with sleep efficiency on either experimental night's sleep below 60% were eliminated from analyses. After the adjustment to these criteria, seven participants from the original nine participants remained in the study and were used in statistical analyses.

Nap data were compared using paired *t*-tests. Night sleep data were compared using repeated measures analysis of variance and Tukey's HSD for multiple comparisons. The Huynh-Feldt correction (ε) to degrees of freedom was reported for all repeated measures analysis of variance. Eta-squared (η2), a measure of effect size, was also reported.

Statistical significance for all analyses was inferred for results where $p<0.05$.

Ⅲ.Results

All of the original 9 volunteers completed the full experimental schedule including the 10 km run. However, upon analysis of the PSG recordings, only the data for those seven participants who meet criteria were analyzed in the present results.

1. Heart rate during exercise

The predicted estimated maximum heart rate, HRmax commonly calculated as 204 -0.69 * years of age, was 188 beats·min-1 for our group of participants. The obtained averaged heart rate during the 10 km run was 172 ± 8 beats·min-1; participants ran 10 km in 72 ± 10 min, on average, for the seven participants.

2. Polysomnographic measurements

Visually scored, averaged sleep stage variables and computer analyzed delta power for the three nights' sleep (Nt0, Nt1, Nt2) are presented in **Table 1**.

Table 1 . Sleep Variables of nights

	Nt0	Nt1	Nt2
Sleep efficiency (%) †	94.39 ± 5.28	86.85 ± 7.41	91.56 ± 6.50
Time in bed (min)	480.00 ± 0.00	480.00 ± 0.00	480.00 ± 0.00
Total sleep time (min) †	453.07 ± 25.37	416.86 ± 35.55	439.50 ± 31.19
Sleep onset latency (min)	18.64 ± 20.85	45.64 ± 29.82	22.14 ± 18.32
REM latency (min)	78.86 ± 33.27	72.86 ± 23.24	99.64 ± 47.88
Total wake time (min)	27.86 ± 24.31	59.86 ± 40.16	40.57 ± 31.09
stage REM (min) †§	114.00 ± 15.60	90.93 ± 20.86	78.07 ± 17.71
stage 1 (min)	31.00 ± 7.00	43.14 ± 20.00	31.57 ± 4.85
stage 2 (min) §	229.36 ± 37.44	226.14 ± 18.56	259.43 ± 25.90
stage 3 (min)	34.93 ± 14.88	27.07 ± 7.86	27.71 ± 6.07
stage 4 (min)	38.29 ± 25.22	29.50 ± 14.70	36.57 ± 18.81
stage 3+4 (min)	73.21 ± 27.67	56.57 ± 17.02	64.29 ± 17.79
total δ power ($*10^5$ μV^2) ¶	1.22 ± 0.46	0.87 ± 0.27	1.38 ± 0.69
δ power/30 s (μV^2)	193.10 ± 54.39	149.88 ± 49.61	211.48 ± 101.39

Mean±s Significant difference by Tukey HSD post hoc analysis, †$p<0.05$: Nt0 vs Nt1, §$p<0.05$: Nt0 vs Nt2, ¶$p<0.05$: Nt1 vs Nt2.

The inserted naps were successful in altering the composition of the subsequent night's sleep. Comparison of Nt1 with Nt0 showed that the sleep efficiency (SE) of Nt1 decreased significantly after Np1 [ε= 1.000, F(2.000, 12.000) = 4.288, p = 0.039, η2 = 0.72], reflecting the significant 36.21 ± 31.53 min decrease in total sleep time (TST) on Nt1 [ε= 1.000, F(2.000, 12.000) = 4.288, p = 0.039, η2 = 0.72]. Furthermore, REM sleep duration on Nt1 decreased significantly by 23.07 ± 21.63 min [ε= 0.767, F(2.000, 12.000) = 8.979, p = 0.009, η2 = 1.50]. Comparison of Nt1 with Nt2, each of which followed comparable nap periods, showed that the total delta power of Nt2 was significantly greater than that of Nt1 [ε= 1.000, F(2.000, 12.000) = 5.349, p = 0.022, η2 = 0.89]. However, no significant difference in other sleep variables was found between the three nights' sleep (Sleep Onset Latency [ε= 0.706, F(2.000, 12.000) = 4.453, p = 0.057, η2 = 0.74], REM Latency [ε= 0.697, F(2.000, 12.000) = 1.529, p = 0.262, η2 = 0.26], Total Wake Time [ε= 1.000, F(2.000, 12.000) = 3.059, p = 0.084, η2 = 0.51], stage 1 [ε= 0.609, F(2.000, 12.000) = 1.611, p = 0.251 η2 = 0.27],

stage 2 [ε= 1.000, F(2.000, 12.000) = 6.574, p = 0.012, η2 = 1.10], stage 3 [ε= 0.919, F(2.000, 12.000) = 1.388, p = 0.288 η2 = 0.23], stage 4 [ε= 1.000, F(2.000, 12.000) = 0.582, p = 0.574, η2 = 0.10], stage 3+4 [ε= 1.000, F(2.000, 12.000) = 1.312, p = 0.305, η2 = 0.22], average delta power [ε= 0.991, F(2.000, 12.000) = 3.193, p = 0.078, η2 = 0.53]).

Comparison of sleep variables for the two naps that preceded the no running (Np1) and running (Np2) conditions are presented in **Table 2**. The REM latency of Np1 was significantly shorter than that of Np2 by 23.43 ± 24.89 min, while the duration of sleep stage 4 on Np1 was significantly longer than that of Np2 by 9.86 ± 7.85 min. No significant difference was found for other sleep variables. There were no significant differences in the EEG power spectra, including delta power, between the two naps.

Table 2 .Sleep Variables of naps

	Np1	Np2
Sleep efficiency (%)	91.55 ± 5.19	90.12 ± 8.05
Time in bed (min)	120.00 ± 0.00	120.00 ± 0.00
Total sleep time (min)	109.86 ± 6.22	108.14 ± 9.66
Sleep onset latency (min)	7.79 ± 4.04	10.14 ± 8.22
REM latency (min) *	45.64 ± 21.04	69.07 ± 25.81
Total wake time (min)	9.86 ± 6.69	11.64 ± 9.92
stage REM (min)	20.29 ± 12.68	17.71 ± 6.66
stage 1 (min)	10.57 ± 4.38	9.71 ± 4.32
stage 2 (min)	46.86 ± 15.39	54.50 ± 14.09
stage 3 (min)	9.64 ± 5.05	12.00 ± 4.09
stage 4 (min) *	23.14 ± 10.33	13.29 ± 6.24
stage 3+4 (min)	32.79 ± 8.14	25.29 ± 5.00
total δ power ($*10^5$ μV^2)	0.41 ± 0.28	0.28 ± 0.14
δ power/30 s (μV^2)	248.67 ± 97.87	183.95 ± 107.42

Mean±s

Significant difference by paired Student's t-test between conditions: *p<0.05.

3. Subjective sleepiness

Comparison of participants' ratings of nights showed no significant differences in scores on the OSA inventory or on the visual analog scales, as seen in **Table 3**. Participants did tend to report, through the integrated sleep feeling measure on the OSA inventory, that they felt Np2 was worse than Np1. However, no corresponding significant differences were found in the visual analog scales of Np1 and Np2.

Table 3 .OSA Inventory and VAS

	sleepiness	sleep maintenance	worries	integrated sleep feeling	sleep initiation	arousal (VAS)	sleepiness (VAS)
Nt0	22.26 ±4.78	18.33 ±6.96	25.33 ±5.01	23.81 ±6.03	22.76 ±5.58	47.71 ±21.26	53.57 ±26.14
Nt1	22.24 ±6.04	17.95 ±2.97	23.30 ±4.75	19.00 ±6.66	15.67 ±3.50	63.86 ±21.96	40.43 ±27.93
Nt2	20.92 ±6.78	17.21 ±5.40	21.60 ±5.39	21.24 ±8.15	22.52 ±8.16	41.71 ±17.65	69.43 ±14.37
Np1	30.27 ±6.45	24.37 ±3.11	29.80 ±5.17	28.24 ±6.83	27.81 ±5.21	52.57 ±24.30	48.29 ±26.74
Np2	26.02 ±5.48	21.57 ±5.46	26.06 ±6.88	19.95 ±6.39*	22.19 ±6.73	51.14 ±13.97	58.43 ±13.15

Mean ± *s* Significant difference by paired Student's *t*-test: *$p<0.05$ vs Np1.

Ⅳ.Discussion

In the present study, we examined the effect of an acute 10 km run on the quality of night sleep, after reducing the likely ceiling and floor effects of studying healthy young sleepers. One goal of the experimental design was to degrade the quality of night sleep using an afternoon nap. Overall, as seen in Table 1, the sleep of Nt1 (after an afternoon nap) was worse than that of Nt0 (no nap), indicating the success of the nap in degrading the following night's sleep. The sleep efficiency (SE) and total sleep time (TST) of Nt1 were both significantly lower than those of Nt0. The exercise intensity of our participants during the 10 km run in our experiment corresponded to 92%HRmax, indicating substantial compliance and effort within the group. That substantial effort at unaccustomed physical exercise did not produce a significant difference in the duration of visually tabulated sleep stages between Nt1 and Nt2. However, the computer analyzed total EEG delta band power of Nt2 was significantly higher than that of Nt1, reflecting the increased EEG power expressed in the delta frequency band after acute exercise, a significant physiological change undetected by the traditional method of visually scoring sleep stages. This finding confirms the report by Youngstedt et al. (1997), suggesting a minor effect of exercise on night sleep. Our findings reinforce the importance of spectrum analysis for examining the effects of acute exercise on sleep. Because sleep stage duration has been the main tool in investigations of the effects of exercise on sleep in most earlier experiments (Bunnell, Bevier, & Horvath, 1983; Driver et al., 1994; Horne & Staff, 1983; Horne & Moore, 1985; Netzer, Kristo, Steinle, Lehmann, & Strohl, 2001; Shirakawa & Oda, 2007), we propose the incorporation of spectral analysis in research conducted within this field.

Contrary to our expectation, a comparison of the two naps Np1 and Np2 revealed a significant difference. The Np1 REM latency was significantly shorter, and duration of sleep stage 4 was significantly longer than in Np2. One reason may have been the participants' awareness of the post-nap schedules. For Nt2, an unaccustomed and possibly worrisome 10 km run was scheduled. The exercise might have made some participants nervous because they had reported no fitness habits and might have felt that the 10 km run was heavy exercise. Anxiety and stress in anticipation of the 10 km run later that evening might have disturbed the Np2 nap that preceded Nt2.

Improvement of sleep on Nt2, therefore, might have been brought about by this

difference in the composition of the earlier nap. However, there was no significant difference in total delta power between Np1 and Np2, despite the lower number of epochs of visually recognized sleep stage 4. Therefore, it is more likely that the exercise after Np2 affected the following night's sleep.

Some improvements might raise the quality of experiments of this kind. Exercise intensity could be made more subjectively relevant by adjusting levels for each participant. Exercise intensity could also be monitored for each participant to obtain equivalent levels of maximal oxygen consumption (VO2max), which might provide better control than the standard 10 km run exercise used for this experiment. Such an adjustment of exercise intensity might contribute to more precise elucidation of the exercise effects than that of the present study.

The potentially stimulating and disruptive effect of exposure to light is another factor which should be controlled with accuracy. The 10 km run in our experiment was conducted outdoors at dusk. However, Nt2 might also have been exposed participants to more light than on Nt0 or Nt1. An experimental protocol that controls light exposure should be used since earlier studies suggest that light stimulation has a considerable effect on sleep (Driver & Taylor, 2000; Youngstedt & Kline, 2006). In this study, we did not examine no-nap exercise condition, since this design did not necessarily result significant effect in past studies (Paxton, Montgomery, Trinder, Newman, & Bowling, 1982; Walker et al., 1978). However, in order to clearly elucidate ceiling and floor effect, this condition also should have been examined.

The present results underscore the necessity of using computerized (here, spectral) analysis in addition to standard sleep stage criteria in studying the effects of exercise on sleep. Although the sleep stage criteria failed to detect the effect of exercise on slow wave sleep, a significant effect of the exercise was clearly revealed in the total delta power of the sleeping EEG. If a small effect of exercise is to be studied, as described by Youngstedt et al. (1997) , then computer analysis is indispensable for elucidate the effects of exercise on sleep, in addition to the sleep stage criteria.

Finally, to study sleep effects in healthy young people, effective removal of ceiling and floor effects is necessary for proper evaluation of the effectiveness of exercise on sleep. We have confirmed the usefulness of enforced napping to degrade sleep on the following night. Through improvements in precise control of exercise intensity and other suggested refinements, we will be able to further elucidate details of the effects of exercise on sleep in future studies.

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