INTRODUCTION

Athletes are required to perform strenuous exercise on consecutive days to improve their performance. Therefore, it is essential for them to promote recovery of physical function (e.g., maximal strength and power output) during the post-exercise period to increase the quality of their next training session. Among the various “post-exercise treatments” that have been established, including massage, cryotherapy, nutrient intake, and sleep\(^1\)\(^{-3}\), sleep stands out as it is indispensable for life, physiological growth, and repair\(^4\). In general, 9–10 h of sleep per night is recommended to facilitate appropriate recovery following an exercise session\(^5\). However, a survey of 890 elite athletes revealed that 41% of the athletes experienced sleep problems (e.g., falling asleep at night) and 11% slept less than 6 h per night\(^6\). Furthermore, the onset of sleep among athletes can be delayed due to stress derived either from competition, daily strenuous exercise, or training and travel occurring late in the evening\(^7\)\(^{-8}\). According to Oda and Shirakawa\(^8\), high-intensity exercise before bedtime delayed sleep onset relative to moderate-intensity exercise.

Total sleep deprivation (TSD) and partial sleep deprivation (PSD) have been shown to impair exercise performance\(^9\)\(^{-13}\). Oliver et al.\(^12\) showed that a single night of sleep deprivation decreased performance during an endurance running test on the following day. Moreover, maximal aerobic power decreased by 50% after PSD\(^11\), while heart rate and minute ventilation during submaximal exercise increased\(^16\). Peak oxygen consumption during exercise also decreased significantly, although the maximal workload at exhaustion was not affected. In addition to impairing endurance performance, PSD may also reduce anaerobic performance. It was found that 4 h of PSD significantly reduced both peak and mean power outputs during a 30 s maximal sprint\(^9\). Furthermore, PSD augmented exercise-induced elevations in plasma interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-\(\alpha\)) following repeated sprint training\(^14\), and a single night of PSD increased the transcription of IL-6 and TNF-\(\alpha\)\(^15\). However, these experiments were designed to determine the impact of reduced sleep duration itself, without exercise. Considering
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that athletes perform consecutive days of high-intensity exercise, it is necessary to determine the impact of PSD after an exercise session. Therefore, the present study examined the effect of PSD after exercise on metabolic responses and exercise performance on the following morning. We hypothesized that a single night of PSD after exercise would reduce exercise performance when compared with a normal sleep duration.

METHODS

Subjects

Eleven male athletes participated in the study. The mean ± standard error (SE) age, height, and body mass of the study subjects were 20.8 ± 0.8 years, 168.9 ± 6.9 cm, 67.4 ± 9.8 kg, respectively. They all followed a regular sleep cycle, with an average sleep duration of approximately 6 h and 7 min. Prior to the study, all subjects were informed about the experimental procedures and possible risks involved in this study, and they subsequently provided informed consent. The present study was approved by the Ethics Committee for Human Experiments at Ritsumeikan University, Shiga, Japan.

Experimental design

Habitual sleep duration was monitored for 7 days before the experiment. Sleep duration was recorded using an accelerometer (ActiGraph; Ambulatory Monitoring, Inc., Ardsley, NY, USA), and the individual sleep duration and magnitude of sleep deprivation were determined. Prior to the experiment, an incremental running test was performed to evaluate the maximal oxygen uptake (VO\textsubscript{2max}) using a treadmill (Valient; Lode, Groningen, the Netherlands). On day 1 of the experiment, the subjects arrived at the laboratory at 16:00 and performed an exercise session. They then completed one of the two trials in which they were allowed different amounts of sleep. For the control (CON) trial, the subjects slept from 23:00 to 7:00, whereas for the PSD trial, the subjects’ sleep duration was shortened to 40% of their individual sleep duration (a 60% reduction of the normal sleep duration). In the PSD trial, the early phase of nighttime sleep was deprived. During this period of sleep deprivation, the subjects were not allowed to sleep, and they spent this time watching television, reading books, or engaging in other non-exercise activities. On the following morning (day 2) of both trials, exercise performance and subjective scores of fatigue, leg muscle soreness, sleepiness, quality of sleep, and falling asleep were evaluated (Figure 1). The two trials were separated by a 3-week interval using a randomized crossover design. The subjects’ diet and physical activity levels were controlled to match in the two trials.

Exercise protocol

On day 1, the exercise protocol was started with a 5 min warm-up consisting of running at 60% VO\textsubscript{2max}. Then, the subjects completed 90 min of continuous running at 75% VO\textsubscript{2max} followed by drop jumps (10 × 10 sets, 100 drop jumps in total). They were allowed a 5 min rest between the 90 min of continuous running and the drop jumps. The maximal voluntary isometric contraction (MVC) of knee extension was evaluated before and after completion of the drop jumps. On the following morning (day 2), the MVC was evaluated again, followed by 20 min of submaximal running at 75% VO\textsubscript{2max} and a time to exhaustion (TTE) test at 85% VO\textsubscript{2max}.

Measurements

VO\textsubscript{2max}

The running velocity was initially set at 6 km/h, and then was increased by 2 km/h every 4 min until it reached 10 km/h. Once the running velocity reached 10 km/h, it was increased by 2 km/h every 3 min until it reached 14 km/h. Once the running velocity reached 14 km/h, it was increased by 0.6 km/h every minute until volitional exhaustion. During the test, expired gases were collected and analyzed with an automatic gas analyzer (AE300S; Minato Medical Science Co., Ltd., Tokyo, Japan). The collected data were
averaged every 30 s. Heart rate (HR) was measured continuously during the test with a wireless HR monitor (Acculex Plus; Polar Electro Oy, Kempele, Finland).

**Maximal voluntary isometric contraction of knee extension**

The MVC with the knee positioned at 70° of extension (the fully extended position was defined as 0°) was measured using an isokinetic dynamometer (Biodex System 4; SAKAI Medical Co., Tokyo, Japan). The subjects exerted their maximal strength (3 s) knee extension twice, and the highest value was included in the analysis. A 60 s rest period was provided between extensions.

**Subjective assessments**

Subjective fatigue, leg muscle soreness, sleepiness, quality of sleep, and falling asleep were evaluated using a 100 mm visual analog scale. The subjects provided ratings for respiratory strain (RPE-R) and leg strain (RPE-L) every 15 min during the running sessions on days 1 and 2 using a 10-point scale of perceived exertion.

**Physiological responses**

On day 2, oxygen uptake (\(\dot{V}O_2\)), carbon dioxide output (\(VCO_2\)), respiratory exchange ratio (RER), and expired minute ventilation (VE) were determined during 20 min of submaximal running at 75% \(VO_2\)\(_{max}\). Respiratory samples were collected and analyzed by the breath-by-breath method using a metabolic cart (AE300S; Minato Medical Science Co., Ltd.). The collected data were averaged every 30 s. HR was recorded at 1 min intervals.

**Time to exhaustion**

An all-out running test at 85% \(VO_2\)\(_{max}\) to determine TTE was performed after 20 min of submaximal running at 75% \(VO_2\)\(_{max}\) and a 2 min rest. After this rest, the subjects started running again at 85% \(VO_2\)\(_{max}\) until exhaustion. No information about the elapsed time was provided to the subject during the test. The TTE was defined as the time when the subjects touched the rope twice at the prescribed position on the treadmill.

**Statistical analysis**

All data are expressed as the mean ± SE. Two-way analysis of variance (ANOVA) with repeated measures was used to confirm the interaction (trial × time) and main effects (trial and time). When the ANOVA revealed a significant interaction or main effect, the Tukey-Kramer test was performed to identify differences. For all tests, a \(p\)-value less than 0.05 was considered statistically significant.

**RESULTS**

**Subjective variables**

Subjective scores of fatigue, leg muscle soreness, sleepiness, and sleep quality were evaluated on day 2. The sleepiness score was significantly higher in the PSD trial than in the CON trial (\(p < 0.001\)), whereas the vitality score was significantly lower in the PSD trial than in the CON trial (\(p < 0.05\)). The scores of subjective fatigue, muscle soreness, and sleep were not significantly different between the two trials.

**Cardiorespiratory variables and time to exhaustion during the submaximal running test**

Figure 2 shows the change in respiratory variables during the 20 min of submaximal running on day 2. The results show that \(VO_2\) did not differ significantly between the CON and PSD trials (Figure 2A). However, as shown in Figure 2B and C, the PSD trial showed significantly lower \(VO_2\) and RER during running (\(p < 0.05\)). The average RER during 20 min of running was significantly lower in the PSD trial (0.86 ± 0.01) than in the CON trial (0.89 ± 0.01, \(p < 0.05\)). In addition, no significant interaction between trial and time or the main effect of trial was observed for VE. The HR during running did not differ significantly at any point between the two trials.

The TTE during running at 85% \(VO_2\)\(_{max}\) was significantly shorter in the PSD trial (665.73 ± 100.3 s) than in the CON trial (887.64 ± 162.2 s, \(p < 0.05\), Figure 3). During the TTE test, the HR did not significantly differ between the CON (180 ± 5 bpm) and PSD trials (177 ± 4 bpm, \(p > 0.05\)).

**Maximal voluntary isometric contraction (MVC) of knee extension**

The changes in the MVC of knee extension are shown in Table 1. In the PSD trial, a significant decrease in MVC values was observed on day 2 when compared to day 1 (main effect for time, \(p < 0.05\)), whereas no significant change was observed in the CON trial. However, no significant difference between the PSD and CON trials was observed at any time point.

| Day 1 | Day 2 |
|-------|-------|
| (Before exercise) | (After exercise) | CON | PSD |
| 225 ± 8 | 188 ± 10* | 206 ± 9 |
| 224 ± 11 | 199 ± 9 | 187 ± 9* |

Values are means ± SE. Unit: Nm. *; \(p < 0.05\) vs. before exercise on Day 1.

**DISCUSSION**

The present study investigated the effect of PSD after a prolonged exercise session on exercise performance on the following morning. The results showed that neither the \(VO_2\) (running economy) during 20 min of running at 75% \(VO_2\)\(_{max}\) nor the MVC differed significantly between the PSD and CON trials. A novel finding of this study is that the RER during running (at 75% \(VO_2\)\(_{max}\)) was significantly lower in the PSD trial than in the CON trial. In addition, the TTE during subsequent running at 85% \(VO_2\)\(_{max}\) was significantly shortened in the PSD trial when compared to the CON trial.
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These findings indicate that PSD after an exercise session reduces endurance performance, but not maximal muscular strength, on the following morning.

The RER during 20 min of submaximal running at 75% \(V\text{\textsubscript{O}}_{\text{2max}}\) was significantly lower in the PSD trial, suggesting augmented fat utilization during the exercise. In general, augmented fat utilization during submaximal exercise decreases muscle glycogen utilization (i.e., muscle glycogen is spared due to augmented fat utilization), leading to an increase in TTE during exercise\(^{15}\). In contrast, the PSD trial revealed a significantly shorter TTE at 85% \(V\text{\textsubscript{O}}_{\text{2max}}\) after 20 min of submaximal running at 75% \(V\text{\textsubscript{O}}_{\text{2max}}\). A plausible reason for the impaired endurance performance in the PSD trial is reduced muscle glycogen content at the start of the exercise on day 2. Skein et al.\(^{18}\) investigated the influence of a single night of sleep deprivation on muscle glycogen content and intermittent sprint performance on the following morning. In that study, team sport athletes conducted a 30 min incremental run followed by intermittent sprint exercises after 30 h of sleep deprivation. Muscle biopsy samples were collected before exercise. Sleep deprivation decreased intermittent sprint performance along with a concomitant reduction in muscle glycogen content before exercise. Moreover, in another study, performance of a 3 km time trial (3 km TT) was impaired (-4% vs. the baseline on the previous day) following PSD (sleep was restricted from 22:30 to 2:00) compared with control sleep conditions (-0.5% vs. baseline)\(^{19}\). The authors suggested that the impaired 3 km TT performance was attributed to incomplete muscle glycogen replenishment following exercise in the previous evening. Sleep deprivation may also affect glucose metabolism during exercise. As mentioned previously, the RER during 20 min of submaximal exercise was significantly lower in the PSD trial. Exercise with decreased glycogen content impairs exercise tolerance\(^{20}\), but a compensatory increase in fat utilization (shown as a lower RER) would be induced. Recovery of muscle glycogen content during the post-exercise period is strongly influenced by energy intake after the completion of exercise\(^{21}\). In the present study, diet during post-exercise recovery was carefully controlled and was kept constant between the two trials. Therefore, it is unlikely that the difference in energy intake affected the results. However, because energy expenditure during sleep is lower than that during waking\(^{22}\), energy expenditure during the night was considered to be higher in the PSD trial than in the CON trial. It is also interesting that the VE during the initial part of the 20 min run was significantly lower in the PSD trial than in the CON trial. Therefore, PSD appeared to attenuate the initial increase in the cardiorespiratory response during exercise\(^{23}\). In contrast, no change in HR was observed during 20 min of submaximal running. This is consistent with previous findings of no change in HR during exercise following sleep deprivation\(^{12,24-26}\).
Despite a marked reduction in endurance performance in the PSD trial, the influence of PSD on MVC was small, and there was no significant difference in MVC between the CON and PSD trials. In a previous study, sleep restriction after strenuous exercise did not attenuate peak isokinetic torque on the following morning. Bambaei et al. demonstrated that PSD (2.5 h of sleep) did not significantly alter maximal strength or core temperature in the morning (6:00) or evening (18:00). Moreover, 60 h of sleep deprivation did not attenuate MVC compared to that under normal sleep conditions. These findings are consistent with the results of our PSD trial.

Some limitations must be carefully considered when interpreting these results. First, we were not able to evaluate muscle glycogen content, which might explain the lower endurance performance in the PSD trial. Second, we measured the effect of PSD after an exercise session only on the following morning, thus it is unclear how long the impairment of endurance performance lasts. Third, although we focused on the impact of reduced sleep duration, sleep quality is also important for promoting recovery following exercise. Therefore, it would be meaningful to evaluate sleep quality after exercise in future studies. Finally, the present PSD trial deprived the subjects of sleep during the first part of the night. However, in some previous studies, sleep deprivation during the latter part of the night would be interesting. Moreover, 60 h of sleep deprivation during the latter part of the night would be interesting. Therefore, it would be meaningful to evaluate sleep quality after exercise in future studies. Finally, the present PSD trial deprived the subjects of sleep during the first part of the night. However, in some previous studies, sleep deprivation during the latter part of the night.

In conclusion, a single night of PSD after exercise did not markedly affect the strength or VO₂ kinetics during submaximal running on the following morning. However, the TTE during running was significantly decreased after PSD.

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