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Original Article

Effects of short sleep duration on hemodynamic and psychological responses under long working hours in healthy middle-aged men: an experimental study

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Abstract: This study examined the effects of short sleep duration (SSD) on hemodynamic and psychological responses under long working hours (LWH) in a laboratory experiment. Sixteen subjects participated in a crossover design experiment consisting of two conditions: normal (7-hours) sleep and short (5-hours) sleep. In each condition, participants engaged in simulated LWH (13 hours a day), comprising 12 task sessions. Hemodynamic and psychological responses were measured in each session. Results showed that there were significant main effects of condition and session but no interaction for hemodynamic and psychological responses. Systolic blood pressure and fatigue were higher in the later sessions than the first one. Stroke volume, sleepiness, fatigue, and stress were higher in the 5-hour than the 7-hour sleep condition (all $p<0.05$). These results suggest that although the combined effect of LWH and SSD was not significant, both LWH and SSD caused a hemodynamic and psychological burden.

Key words: Overtime work, Working long hours, Sleep restriction, Cardiovascular health, Mental health

Introduction

Long working hours (LWH) have several negative effects on workers' health. For FY2019, the Ministry of Health, Labour and Welfare, Japan reported 216 and 509 compensated cases of occupational cerebrovascular/cardiovascular diseases, and mental disorders, respectively, and 177 and 182 of these cases, respectively, were related to overwork of more than 80 hours per month[1)]. Previous studies reported that LWH are associated with cardiovascular and mental disorders. In particular, systematic reviews and meta-analyses have reported that workers exposed to LWH (≥ 55 hours per week) have higher risks of stroke[2)], ischemic heart disease[3)], and onset of depression symptoms[4)], than workers with standard working hours (35–40 hours per week). Other experimental studies that conducted one-day simulated LWH (13 hours) in a laboratory reported that LWH induced elevated blood pressure (BP) and a deterioration of psychological responses (stress and fatigue) during the working hours, especially during the afternoon and at night[5)]. These studies suggest that both chronic and

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one-day LWH are associated with increased cardiovascular and mental burden.

Sleep also influences workers' cardiovascular and mental health. Tochikubo *et al*. compared ambulatory BP in male technical workers during the day after a normal night's sleep (mean 8 hours) and the day after an insufficient night of sleep (mean 3.6 hours) and found that systolic and diastolic BP increased the day after a night of insufficient sleep relative to the day after a normal night[6]. Gangwisch *et al*. conducted a longitudinal study on the relationship between short sleep duration (SSD) and hypertension and reported that SSD (≤ 5 hours) was associated with increased risk of hypertension in subjects between the ages of 32 and 59 years, relative to 7 to 8 hours of sleep[7]. Cappuccio *et al*. conducted a systematic review and meta-analysis of 15 studies (24 cohorts) and found that SSD (≤ 5–6 hours) was associated with a greater risk of developing or dying of coronary heart disease and stroke but not total cardiovascular disease, relative to sufferers with 7 to 8 hours of sleep[8]. Zhai *et al*. conducted a meta-analysis of seven prospective studies and reported that SSD was significantly associated with the increased risk of depression in adults[9]. Thus, SSD may cause cardiovascular and mental burden.

Previous studies also reported that LWH could be a risk factor for SSD[10–12]; that is, LWH and SSD may be more likely to occur simultaneously. Consequently, workers with SSD might suffer more severe cardiovascular and mental health burden during LWH than workers with sufficient sleep. The clarification of these mutual effects is important for preventing negative effects on workers' cardiovascular and mental health. To the best of our knowledge, the combined effects of LWH and SSD have not been sufficiently examined. Liu *et al*. (2002) reported that although LWH (≥ 60 hours per week) and insufficient sleep (≥2 day per week of < 5 hours of sleep) were each related to an increased risk of acute myocardial infarction, there were no significant interaction[13]. Nakata (2011) reported that this combination of SSD (< 6 hours of sleep per day) and LWH (> 10 hours of work per day) had a synergistic effect on risk of depression[14]. These inconsistent results may be due to the duration and periods of the SSD. Previous studies have reported that more severe and/or chronic sleep restrictions have more severe impacts[15, 16]. Another possible reason was that many stress factors influence cardiovascular responses in the real workplace (e.g., occupational stress[17], low social support[18], and occupational noise[19, 20]). To control these confounding factors, we conducted our examination to within a controlled laboratory environment.

This study examined the effects of SSD on workers' cardiovascular (hemodynamic) and mental (psychological) responses under the LWH (13 hours) condition in an experimental laboratory. The study compared the recommended sleep duration (7 hours: 7–9 hours of sleep is recommended for adults[21]) to one day of SSD (5 hours: less than 6 hours is not recommended for adults[21]).

We tested the following three hypotheses. Hypothesis 1: Hemodynamic and psychological responses will gradually deteriorate with work time extension. Hypothesis 2: Hemodynamic and psychological responses will be worse in the 5-hour sleep condition than in the 7-hour sleep condition. Hypothesis 3: deterioration of hemodynamic and psychological responses due to LWH will be more noticeable in the 5-hour sleep condition than in the 7-hour sleep condition.

Methods

Participants

Participants who meet the following inclusion criteria were recruited through a temporary-employment provider: age (40–59 years), sex (men), smoking habit (ex- or non-smoker), health status in general (no history of cardiac disease, diabetes, asthma, cerebral stroke, chronic liver disorder, back problems, or mental disorders), usual sleep duration (from 6 hours 30 minutes to 7 hours 30 minutes), and BP (resting systolic BP < 140 mmHg and resting diastolic BP < 90 mmHg). The recruitment information was presented on the website of the employment provider, and e-mails were also sent to potential participants. After registering for an interview, potential participants had their resting BP confirmed on the day of the interview in the laboratory, and those with BPs outside the given range were excluded. After the interview, 22 participants were recruited for this study. The sample size was decided based on our previous study,[22] which examined the hemodynamic responses of normotensive (n=21) and untreated hypertensive men (n=13) under simulated LWH in an experimental laboratory and found these interaction effects. In contrast, this study conducted a counterbalance due to the within-subject design. We targeted 22 participants in the study, none of whom used antihypertensive drugs. They were required to abstain from substances that could affect the experiments (such as alcohol and caffeine) during the experimental period.

All participants gave written informed consent. The study protocol was approved by the Research Ethics Committee of the National Institute of Occupational Safety and

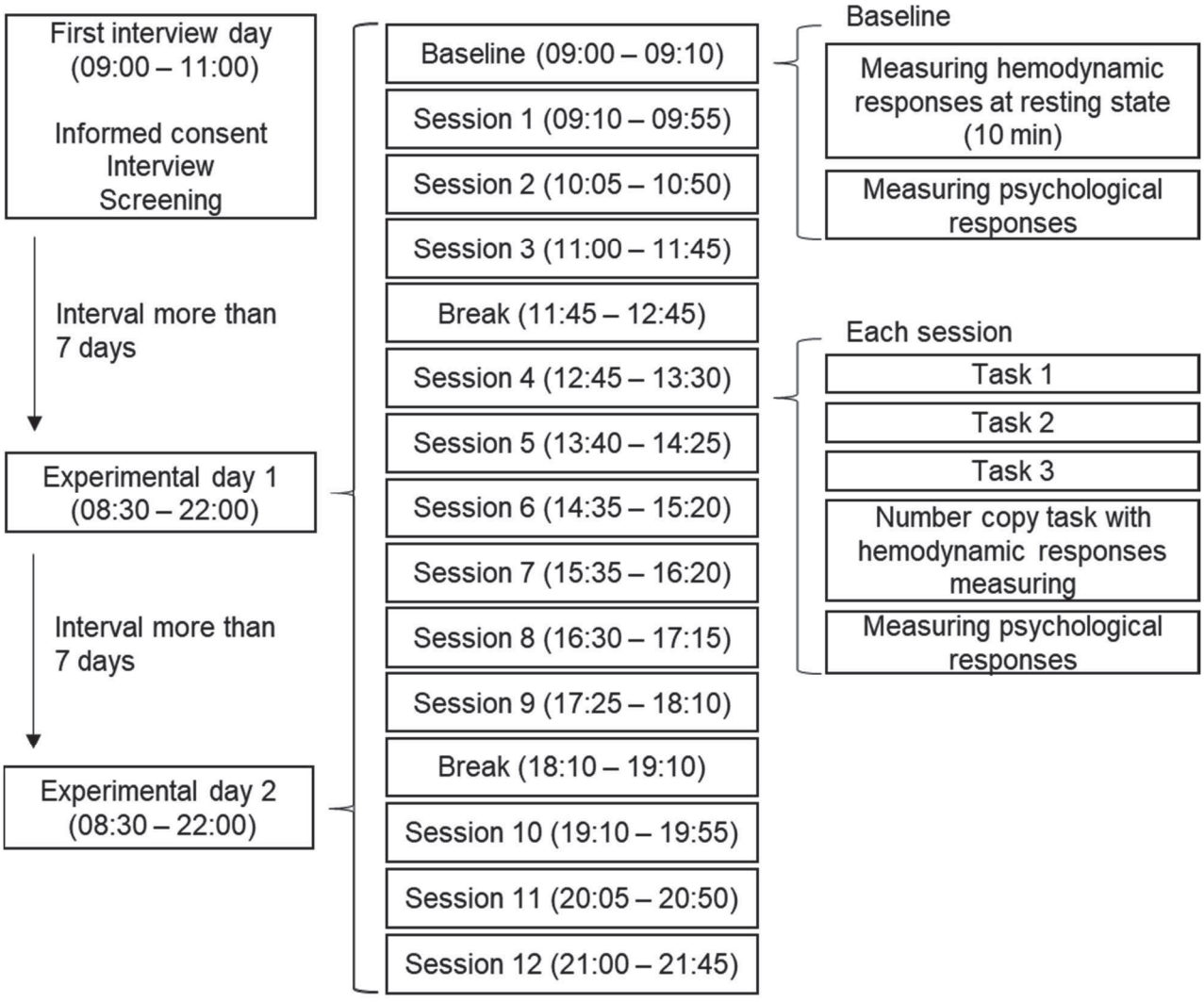

Fig. 1. Experimental protocol.

Health, Japan (H3013).

Experimental protocol

The participants took part in a crossover design experiment in two conditions with an interval of more than 7 days: normal and short sleep conditions. They were instructed to maintain their usual sleep–wake pattern during experimental periods where possible. In addition, in the normal sleep condition, the participants were required to sleep in their house for 7 hours (counting time in bed) and, in the SSD condition, for 5 hours, in both cases the night before the experimental day. The order of conditions was counterbalanced across participants. In each condition, the get-up time of the experimental day was set by each participant, according to his ordinary habit (ranging from 4:30 to 7:00), and bedtime before the experimental day was adjusted according to the sleep duration in each condition.

Procedures

The experiment was conducted over three nonconsecutive days (Fig. 1). On the first day, interviews were conducted from 09:00 to 11:00. The participants were informed of the experimental protocol, their characteristics, sleep quality (Japanese version of the Pittsburgh Sleep Quality Index: PSQI-J)[23], and resting BP were measured, and they practiced all experimental tasks. The PSQI-J presents 18 questions, and the sum of the PSQI-J scores (range: 0–21) evaluates sleep quality. Higher scores indicate more severe sleep complaints and the limit for primary insomnia was set at 5.5. The PSQI-J scores of all participants were below this limit. Resting systolic and diastolic BP was measured twice using an arm-cuff digital BP monitor (CH-463E; Citizen Systems Japan Co., Ltd., Tokyo, Japan), and the mean values were used to determine whether participants met the

inclusion criteria. The second and third days were the experimental days. The participants came to the laboratory by 8:30 a.m. The baseline measurements were conducted from 09:00 to 09:10. Then, participants engaged in the simulated LWH (almost 13 hours) from 09:10 to 22:00. Simulated LWH consisted of 12 task sessions (visual display terminals task [45 min] and breaks [10 min]; total 55 min) and long breaks (60 min) to eat lunch or dinner between sessions 3 and 4 and sessions 9 and 10. During each session, three tasks (psychomotor vigilance task [PVT], go/no-go task, and mental arithmetic task) were performed in randomized order. After this, to measure hemodynamic response, the participants wore a cuff, and calibration was performed. Then the number copy task and recording of the hemodynamic responses were conducted. Finally, psychological responses were measured.

Measurements

Sleep variables

Time in bed, sleep period time, total sleep time, sleep efficiency, sleep latency, wake after sleep onset, bed out latency, bedtime, time of sleep onset, time of wake onset, and get-up time before the experimental day were measured using an actigraph worn at the waist (FS-770; Kissei Comtec Co., Ltd. Nagano, Japan), and the measured data were analyzed by dedicated software (SleepSign Act ver. 2.0; Kissei Comtec Co., Ltd. Nagano, Japan). Time in bed is the total number of minutes between going to and getting out of bed. Sleep period time is the total number of minutes between initial sleep start and final sleep end. Total sleep time is the total number of minutes spent asleep during the sleep period (sleep period time minus the waking time after sleep onset). The sleep efficiency (%) is calculated as total sleep time/time in bed. The agreement rate between the actigraph and polysomnograph was high (85%)[24]. Participants were required to wear the actigraph from 7 days prior to the experimental days of each condition as possible. In addition, participants were required to wear the actigraph from 2 hours prior to the bedtime on the night before the experimental day, until their arrival at the laboratory on the experimental day.

Task performance

PVT was established following previous reports[25, 26]. The task presented a digital counter at irregular inter-stimulus intervals, ranging from 2 to 10 seconds on the computer screen. Participants responded as quickly as possible to the digital counter. The task duration was 10 minutes. Responses quicker than 100 ms were excluded from subsequent analyses[26]. The vigilance performance was evaluated by the 1/reaction time (i.e., response speed) and the number of lapses (lapses were defined as response times slower than 500 ms) because these variables were sensitive to sleep loss[26].

The go/no-go task, which reflects response inhibition, was based on previous reports[27, 28]. The task involved 120 stimuli (one letter of the alphabet) were presented sequentially on the computer screen. The stimulus duration was 500 ms, and the inter-stimulus interval was 1,359 ms. Participants were instructed to respond to any letter (target: 75%) except for "V" (non-target: 25%). The letters were presented in random order. Go/no-go task performance was evaluated as the correct response rate to the target and non-targets (i.e., no response) and the reaction time to the target.

The mental arithmetic task was used to induce psychological stress. The task presented two random two-digit numbers (ranging from 10 to 49) on the computer screen. Participants were asked to add them up mentally and type the result within 8 seconds using a 10-key number pad. The task duration was 8 minutes. The task performance was evaluated by correct response rate.

The number copy task was used to induce psychological stress. The task presented a random six-digit number on a computer screen. Participants were asked to type the same number within 8 seconds using a 10-key number pad. The task duration was 10 minutes. The task performance was evaluated by correct response rate.

All tasks were conducted using Presentation software (Neurobehavioral Systems, Inc.).

Hemodynamic responses

Hemodynamic responses were measured in a sitting position in a resting state at baseline (10 min) and in the working condition of the number copy task (10 min) at the end of each session. Hemodynamic responses included the systolic BP, diastolic BP, mean arterial pressure, stroke volume, heart rate, cardiac output, and total peripheral resistance. These were measured continuously over 10 minutes from the middle finger using a noninvasive monitor (Finometer Pro; Finapres Medical Systems, Amsterdam, the Netherlands). The validation of the device was confirmed following methods presented in previous studies[29].

Psychological responses

Subjective sleepiness, fatigue, stress, motivation, and depression were measured using the visual analog scale be-

cause it is a common tool previously employed in similar studies[5, 30]. The score range was 0–100, and higher scores were associated with greater sleepiness, fatigue, stress, motivation, and depression.

Analysis

One participant could not complete all sessions (sessions 10 and 12 of the normal sleep condition were lacking) due to technical problems. One participant failed to achieve the 5 hours' time in bed in the short sleep condition (he slept for 6 hours). In addition, the four participants who did not have enough sleep during time in bed before the experimental days were also excluded based on the following criteria: sleep efficiency was <50%, sleep latency was ≥ 20 mins, and/or wake after sleep onset was ≥ 20 min (two participants had ≥ 20 min of wake after sleep onset, one participant had <50% sleep efficiency, and one participant had ≥ 120 min of wake after sleep onset and sleep latency and <50% sleep efficiency. These six participants' data were excluded from the analyses. Table 1 shows the participants' demographic data (n=16).

To examine the different conditions of sleep variables, paired t-tests were conducted. To examine the condition differences in the baseline measurements (hemodynamic and psychological responses), paired t-tests were conducted. To explore the influences of LWH and SSD, two-way repeated ANOVAs (conditions [2] x sessions [12]) were conducted for hemodynamic responses, psychological responses, and task performances. Degrees of freedom were adjusted using the Greenhouse-Geisser epsilon calculation. All ANOVAs were followed by post-hoc tests with Bonferroni corrections. All statistical analyses were conducted using SPSS version 23.0 for Microsoft Windows (SPSS Software Inc., Tokyo, Japan).

Table 1. Demographics (n=16)

	Mean	(SD)
Age	46.8	(5.8)
BMI	22.4	(2.6)
Resting systolic blood pressure on first day	117.0	(10.5)
Resting diastolic blood pressure on first day	79.8	(5.8)
Resting heart rate on first day	75.3	(13.1)
PSQI-J[1] score	3.1	(1.1)
Usual bedtime[1]	23.9	(1.0)
Usual rise time[1]	6.7	(1.3)
Usual sleep duration[1]	6.7	(0.7)

[1]Measured by the Japanese version of the Pittsburgh Sleep Quality Index.

Results

Sleep variables before experimental day

Table 2 shows the sleep variables before the experimental days. Paired t-tests revealed that although time in bed, sleep period time, and total sleep time were significantly longer during the normal sleep condition than the short sleep condition (all $p<0.001$), there was no significant dif-

Table 2. Sleep variables the night before the experimental day (n=16)

Sleep variables	Normal sleep		Short sleep		*t-test*	
	Mean	(SD)	Mean	(SD)	t-value	*p*-value
Time in bed (min)	418.5	(12.6)	302.1	(6.9)	30.0	0.000
Sleep period time (min)	389.0	(22.7)	273.5	(22.1)	16.3	0.000
Total sleep time (min)	340.8	(46.6)	247.0	(28.7)	8.6	0.000
Sleep efficiency (%)	81.5	(11.4)	81.7	(9.2)	0.1	0.931
Sleep latency (min)	22.4	(21.0)	21.0	(15.7)	0.2	0.815
Wake after sleep onset (min)	48.3	(36.1)	26.5	(17.8)	2.7	0.016
Bed out latency (min)	7.1	(8.7)	7.6	(12.3)	0.1	0.884
Bedtime (time of day)	22.9	(0.6)	24.7	(0.9)	16.8	0.000
Time of sleep onset (time of day)	23.3	(0.8)	25.1	(0.8)	16.5	0.000
Time of wake onset (time of day)	5.8	(0.8)	5.6	(1.0)	1.2	0.263
Get-up time (time of day)	5.9	(0.7)	5.7	(0.9)	1.7	0.115

Table 3. Hemodynamic and psychological responses at baseline (n=16)

Variables	Normal sleep		Short sleep		*t-test*	
	Mean	(SD)	Mean	(SD)	t-value	*p*-value
Hemodynamic responses						
Systolic blood pressure (mmHg)	121.5	(7.9)	124.7	(6.7)	2.1	0.049
Diastolic blood pressure (mmHg)	75.2	(6.3)	76.3	(4.6)	0.7	0.478
Mean arterial pressure (mmHg)	94.3	(6.8)	95.9	(5.1)	1.2	0.242
Cardiac output (l/min)	5.9	(1.6)	6.1	(1.6)	0.7	0.493
Stroke volume (ml)	76.8	(14.6)	80.1	(13.1)	1.2	0.252
Heart rate (bpm)	77.0	(11.0)	76.0	(13.9)	0.6	0.543
Total peripheral resistance (MU)	1.0	(0.3)	1.0	(0.2)	0.4	0.669
Psychological responses (VAS)						
Sleepiness	27.1	(25.3)	37.9	(27.6)	1.2	0.234
Fatigue	10.3	(13.1)	16.8	(19.3)	1.1	0.281
Stress	11.9	(14.1)	22.3	(23.9)	2.0	0.063
Motivation	49.3	(34.5)	51.1	(27.9)	0.4	0.712
Depression	9.3	(11.3)	16.1	(17.1)	1.8	0.092

ference in sleep efficiency (p=0.931). Although sleep latency did not differ between conditions (p=0.931), the waking time after sleep onset was longer in the normal sleep condition than in the short sleep condition (p<0.05). In addition, although time of day of bedtime and sleep onset were significantly earlier in the normal sleep condition than in the short sleep condition (all p<0.01), the time of day of wake onset and the get-up time did not significantly differ between conditions (all p>0.05). In addition, the two-way (2 conditions x 6 day [2–7 days before the experiment]) repeated ANOVA was conducted for total sleep time to examine that the accumulated sleep loss (i.e., sleep debt) had occurred. The results showed that there were no significant main effects and interaction (all ps>0.05).

Hemodynamic responses

Table 3 shows the hemodynamic and psychological responses at the baseline (i.e., resting state at 09:00). The paired t-tests revealed that although systolic BP was significantly higher in the short sleep condition than in the normal sleep condition (p=0.049), there were no significant differences between conditions in other hemodynamic responses at baseline (all p>0.05). Fig. 2 shows hemodynamic responses in both conditions under simulated LWH. The two-way (condition x session) repeated ANOVA revealed a non-significant interaction in all hemodynamic responses (all p>0.05). On the other hand, ANOVA revealed significant main effects of condition for stroke volume (F(1.000, 15.000) = 4.887, p=0.043, partial η^2=0.246); that is, the stroke volume was significantly higher in the short sleep condition than in the normal sleep condition. In addition, ANOVA for all hemodynamic responses revealed a significant main effect for session (all p<0.05). Post-hoc tests showed that systolic BP was higher in session 12; the heart rate was lower in sessions 2, 3, 8, and 9; and total peripheral resistance was higher in session 3 than in session 1 (all p<0.05).

Psychological responses

The paired t-tests showed no significant differences between conditions in any psychological responses at the baseline (i.e., resting state at 09:00; all p>0.05; Table 3). Fig. 3 shows the psychological responses in both conditions under simulated LWH. The two-way repeated ANOVA showed a non-significant interaction in all psychological responses (all p>0.05). However, ANOVA showed significant main effects of condition for sleepiness (F(1.000, 15.000) = 9.658, p=0.007, partial η^2=0.392), fatigue

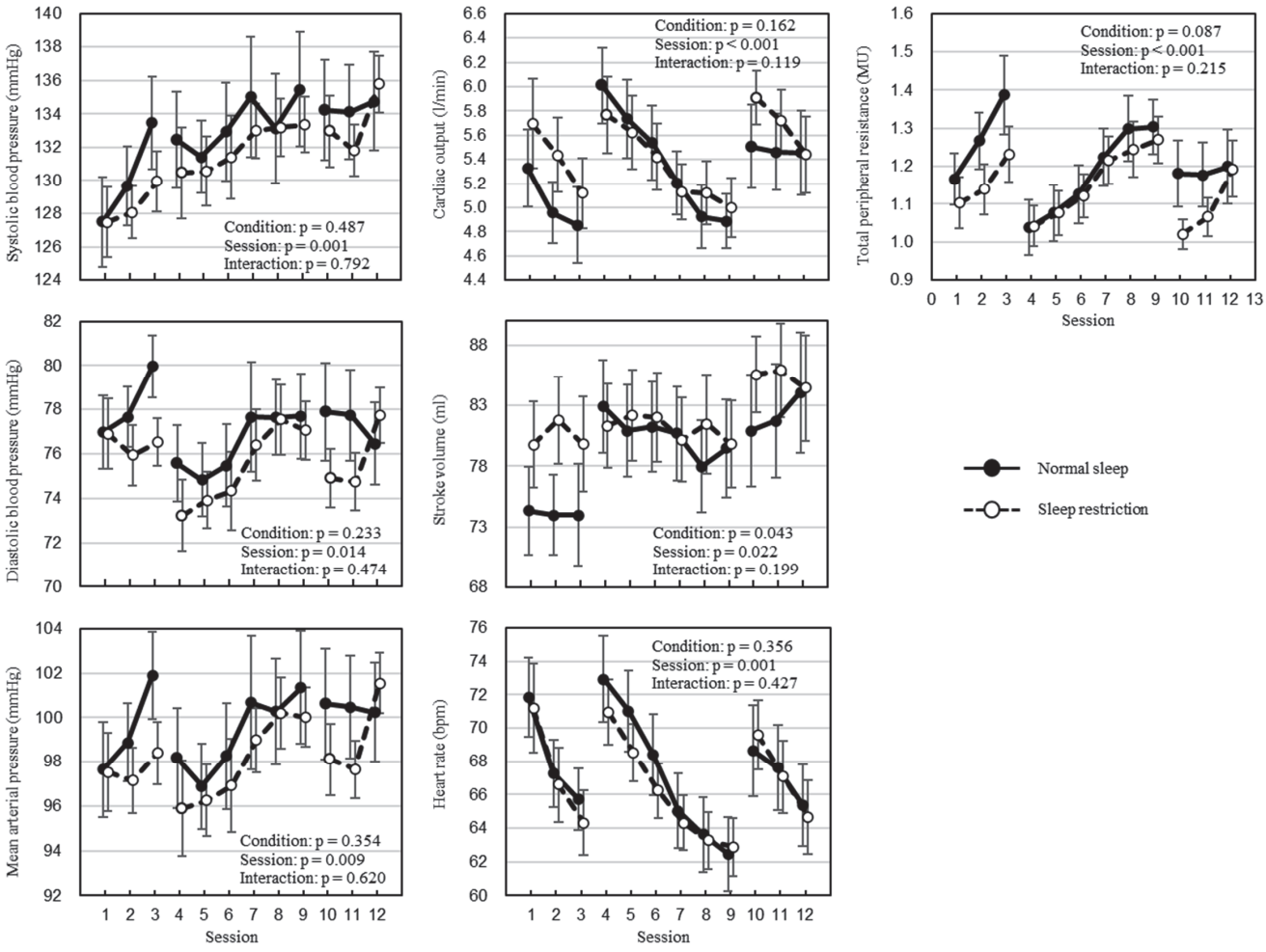

Fig. 2. Hemodynamic responses in both conditions under simulated long working hours.

(F[1.00, 15.00] = 4.684, p=0.047, partial η^2=0.238), and stress (F[1.00, 15.00] = 5.862, p=0.029, partial η^2=0.281). That is, sleepiness, fatigue, and stress were significantly deteriorated in the short sleep condition compared with the normal sleep condition. In addition, the ANOVA showed a significant main effect of session for sleepiness (F(3.728, 55.922) = 3.427, p=0.016, partial η^2=0.186), fatigue (F(3.645, 54.673) = 14.457, p<0.001, partial η^2=0.491), and stress (F(3.373, 50.588) = 3.842, p=0.012, partial η^2=0.204). Post-hoc tests showed that sleepiness was worse in sessions 5, and fatigue was worse in sessions 6–12 than in session 1 (all p<0.05); however, there were no significant differences in stress between session 1 and the other sessions (all p>0.05).

Task performances

Fig. 4 shows the task performances in both conditions under simulated LWH. Two-way repeated ANOVA revealed no significant interactions in all task performances (all p>0.05). On the other hand, ANOVA revealed significant main effects of condition for PVT speed (F[1.000, 15.000] = 8.664, p=0.010, partial η^2=0.366), reaction time to the target of the go/no-go task (F[1.000, 14.000] = 7.438, p=0.016, partial η2=0.347), correct response rate of mental arithmetic task (F[1.000, 15.000] = 7.364, p=0.016, partial η^2=0.329), and number copy task (F[1.000, 15.000] = 5.396, p=0.035, partial η^2=0.265). That is, PVT speed and reaction time to the target of the go/no-go task were significantly slower, and the mental arithmetic and number copy task performance was significantly lower in the short sleep condition than in the normal sleep condition. In addition, the ANOVA revealed a significant main effect of session for the PVT speed (F[5.234, 78.507] = 9.986, p<0.001, partial η^2=0.400), and lapse (F[4.548, 68.225] = 3.257, p=0.013, partial η^2=0.178). Post-hoc tests showed that although the PVT speed was slower in sessions 2 to 12 than

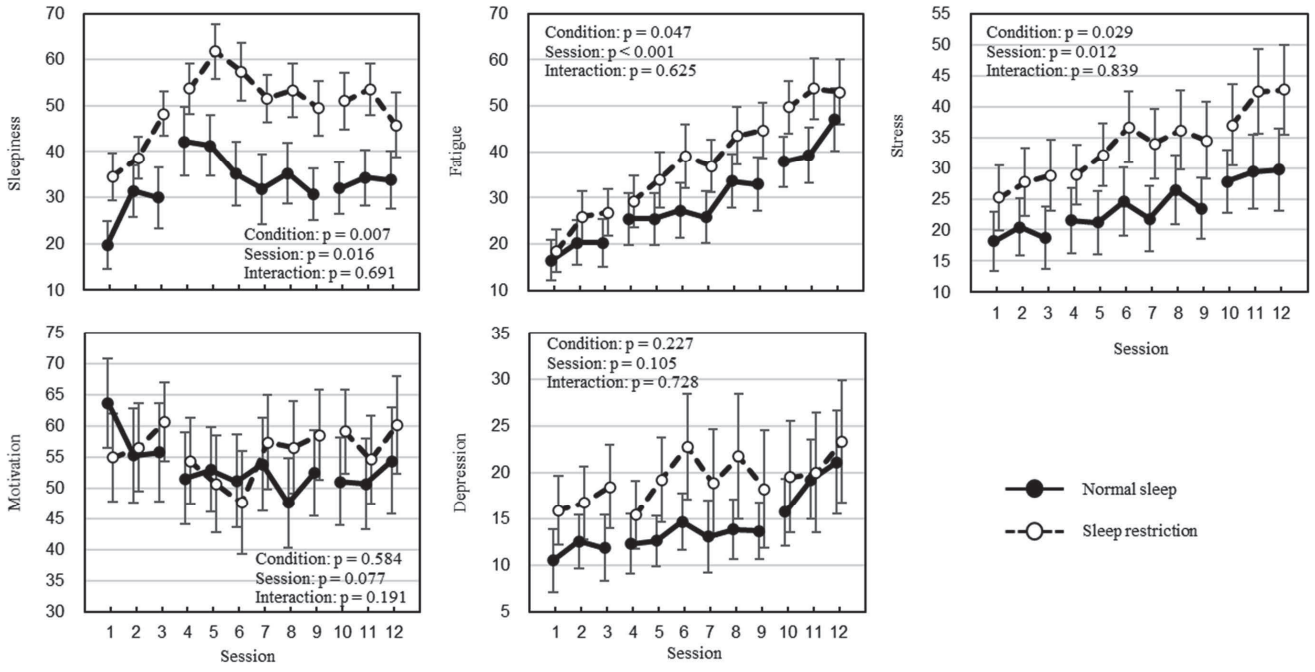

Fig. 3. Psychological responses in both conditions under simulated long working hours.

in session 1 (all $p<0.05$), the PVT lapse did not significantly differ between session 1 and the other sessions (all $p>0.05$).

Discussion

This study examined the effects of SSD on hemodynamic and psychological response under LWH in an experimental laboratory. Time in bed before experimental days was significantly shorter in the short sleep condition (mean = 5 hours 2 minutes) than in the normal sleep condition (mean = 6 hours 58 minutes). The results showed that there were no significant interactions between session and condition for all hemodynamic and psychological responses. On the other hand, systolic BP was higher in the last session, sleepiness was higher in session 5, and fatigue was higher in sessions 6 to 12 than in session 1 (all $p<0.05$). In addition, stroke volume was higher, and sleepiness, fatigue, and stress were all worse in the short sleep condition than in the normal sleep condition (all $p<0.05$). These results suggest that although the interactive effect between LWH and SSD was not significant, both LWH and SSD caused cardiovascular and mental burden.

Simulated LWH caused an increase in systolic BP, deterioration of sleepiness and fatigue, and delay of vigilance task speed; this is consistent with Hypothesis 1. Previous studies also reported that one-day LWH induced an increase in systolic BP[5], and our results agree with this finding. BP has a circadian rhythm (i.e., BP increases in the early morning hours, reaches high levels during the day and peak values between mid-morning and noon, decreases progressively throughout the day, reaches a low point during the night[31, 32]). However, our results showed that systolic BP gradually increased with work time extension and was highest in the final session (21:45). This result is considered may be due to the LWH rather than circadian rhythms. Fatigue also deteriorated from session 6 to the final session (15:20 to 21:45) and was worst in the last session. This suggests that the deterioration of fatigue occurred in the normal working hours, and LWH induced more severe fatigue. The results suggest that LWH induce psychological burden for workers. On the other hand, subjective sleepiness deteriorated in session 5 (14:25). This sleepiness might have been caused to a post-lunch dip, a phenomenon unrelated to food intake that results in a peak of sleep propensity in the afternoon; it is caused by the circasemidian rhythm[33]. Our results also showed that sleepiness peaked in the afternoon and decreased after that. Therefore, sleepiness might be caused by the post-lunch dip rather than the LWH. Moreover, PVT speed was slower in sessions 2 to 12 than in session 1, and the peak of deterioration occurred during session 6 (15:20). Because the speed was associated with sleep loss[26], the deterioration of sleepiness may be due to the behavioral measurements. Otherwise, there was

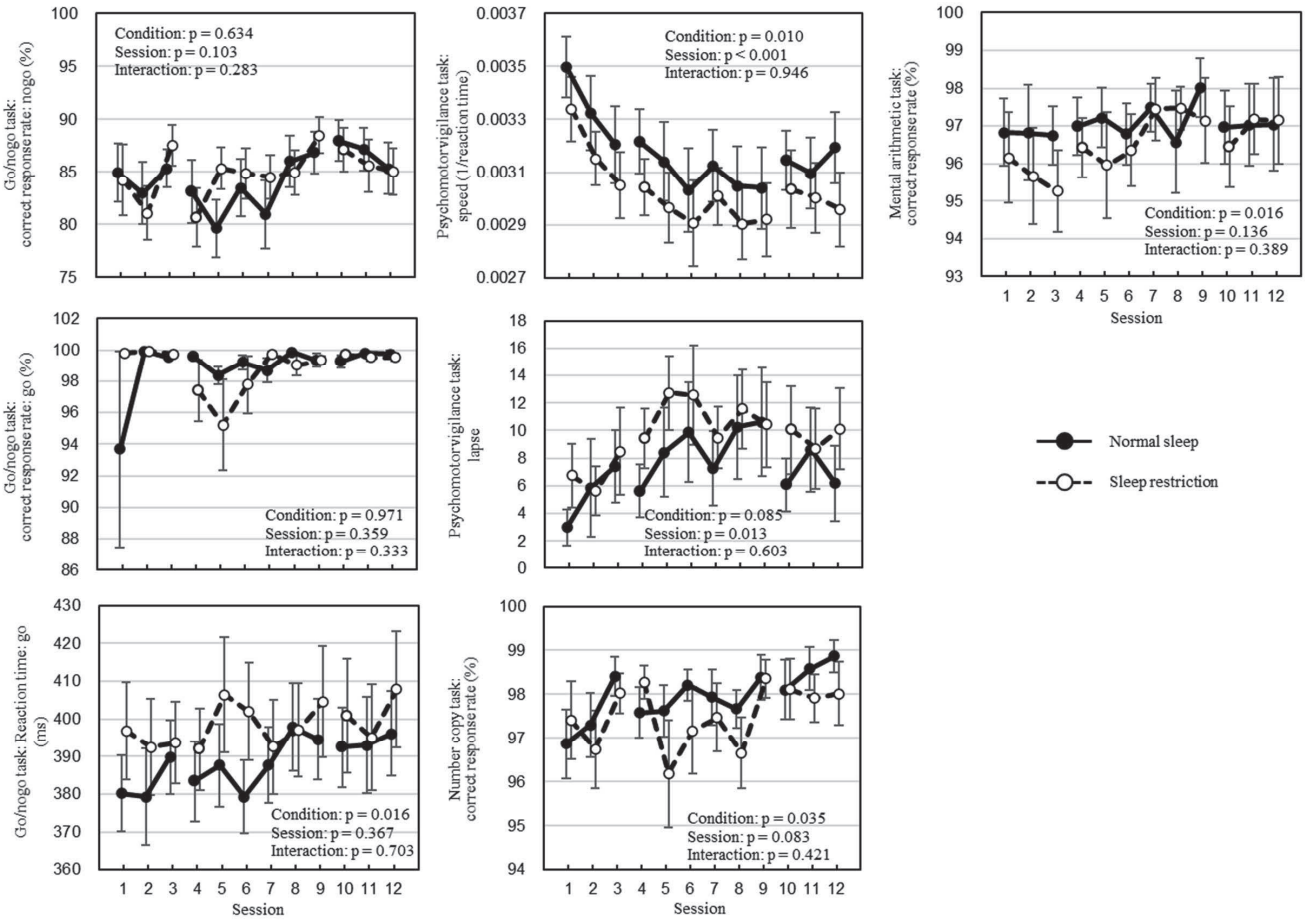

Fig. 4. Task performances in both conditions under simulated long working hours.

no deterioration of depression due to LWH. A previous epidemiological survey reported that LWH was associated with depression symptoms[4]. Because, in this study, LWH occurred only on one day, participants might not have experienced depression. Further studies focused on the effects of translation from one-day to chronic LWH on depression are needed to explore these relationships.

In the task performances, there was a main effect of session for PVT speed but not for the other performances. In addition, the main effects of condition were shown in PVT speed, the correct response rate of the mental arithmetic task and number copy task, and reaction time to the target of the go/no-go task but not the correct response rate to the target and non-target of the go/no-go task. One possible cause of the difference in the results among tasks may derive from the task characteristics. For example, PVT performance reflects the general arousal level, fatigue, and sensitivity to sleep loss. However, the go/no-go task—especially a correct response rate in the non-target—reflects the response inhibition of frontal lobe functioning. The previous study also reported that the temporary increase in sleepiness affects arousal level (vigilance task performance) but does not have an appreciable effect on frontal lobe functions[34].

The results showed that SSD caused an increase in stroke volume; deterioration of sleepiness, fatigue, and stress; deterioration of performance in task performance for number copy and mental arithmetic; and delay of PVT speed. These results are consistent with Hypothesis 2. Although systolic BP at baseline was significantly higher in the short sleep condition than in the normal sleep condition, BP during LWH was not significantly different between conditions. These results may suggest that on day of SSD (5 hours) increases systolic BP in the resting state, and the increase is masked by the increased BP due to working state. Tochikubo *et al*.[6] investigated the ambulatory BP of male technical workers the day after a normal night's sleep (mean 8 hours) and the day after an insufficient night's sleep (mean 3.6 hours) and found that systolic and diastolic BP increased the day after a night of insufficient sleep compared with the

day after a normal night. By contrast, this study controlled for real workplace stress, which influenced cardiovascular responses such as occupational stress[17], social support[18], and occupational noise[19, 20]. Additionally, sleep duration in the Tochikubo's study[6] was slightly longer than in this study. Thus, more severe stressful occasions and/or SSD could cause increased BP during LWH. In addition, habitual SSD could lead to the development and maintenance of hypertension due to extended exposure to hemodynamic load due to habitual SSD could lead to structural adaptations that gradually entrains the entire cardiovascular system to operate at an elevated pressure equilibrium[7, 35]. Therefore, it is possible that chronic SSD (5 hours of sleep) could increase BP during LWH. Further studies are needed to clarify these issues.

This study measured the hemodynamic response underlying changes of BP, such as cardiac response (cardiac output, stroke volume, and heart rate) and vascular responses (total peripheral resistance). BP is dependent on cardiac output and total peripheral resistance, and cardiac output is dependent on stroke volume and heart rate. The results showed that although BP during LWH did not differ between groups, stroke volume during LWH was significantly higher in the short sleep condition than in the normal sleep condition. The increased stroke volume may indicate that SSD may lead to cardiovascular burden. Three patterns of background hemodynamic are known to increase BP, as follows: the cardiac pattern, which increases cardiac output and decreases total peripheral resistance; the vascular pattern, which increases total peripheral resistance and decreases cardiac output; and the mixed pattern, which moderately increases both cardiac output and total peripheral resistance[36, 37]. In the present study, the differences did not reach the level of significance (p=0.087), and total peripheral resistance during LWH was lower in the sleep restriction condition than in the normal sleep condition. Therefore, although the SSD (5 hours) might reinforce the cardiac pattern, 5 hours of sleep per night might not be low enough to increase BP. Further studies should focus on the effects of more severe sleep restrictions on hemodynamic responses.

Both subjective sleepiness and PVT speed, which reflects sleep loss, deteriorated in the short sleep condition relative to the normal sleep condition. These results suggest that SSD causes subjective and objective sleepiness, which is consistent with previous studies[38]. In addition, stress and fatigue deteriorated in the short sleep condition relative to the normal sleep condition. Previous studies also reported that SSD was associated with stress and fatigue[39, 40]. These results suggest that SSD causes several negative psychological responses. Meanwhile, depression did not differ between conditions. Previous meta-analysis studies focused on the longitudinal study of sleep duration and depression and reported that SSD was associated with increased risk of depression[9]. Because LWH in this study only occurred on one day, there may not have been enough time for participants to have experienced depression.

The present results showed that although both the LWH and the SSD affected the hemodynamic and psychological responses, SSD followed by simulated LWH did not influence these responses. These results did not support Hypothesis 3. The LWH and SSD may be more likely to occur simultaneously[10–12], and each LWH and SSD may contribute independently to cardiovascular and mental burden in the real occupational place. Therefore, the workers who scheduled LWH might need to obtain sufficient sleep duration (e.g., recommended 7–9 hours of sleep[21]) to reduce these burdens due to SSD during LWH. On the other hand, the participants in the present study had only one day of LWH (13 hour) following one day of SSD (5 hours). Previous studies reported that more severe and/or chronic sleep restrictions have more severe impacts[15, 16]. Therefore, an experimental design that includes more days of LWH and SSD may reveal a significant mutual effect due to non-restored fatigue. To resolve the issue, further studies are needed.

This study had some limitations. First, only male workers participated because the menstrual cycle affects cardiovascular responses to stress[41]. Therefore, the results are only valid on men, and it is unknown whether the same results would be found in female workers. Second, it is unclear whether the outcomes and experimental findings of the present study are linked to the actual cardiovascular and mental disease events. Third, participants in this study performed tasks on visual display terminals during LWH, so it is not clear whether similar results would be obtained through physical tasks. Fourth, wake after sleep onset was significantly longer in the normal sleep condition than in the short sleep condition. On the other hand, total sleep time was significantly longer (almost 94 min) in the normal sleep condition than in the short sleep condition, suggesting that sleep duration was well controlled. Fifth, although this study used criteria of sample selection (i.e., age, sex, smoking habit, health status, usual sleep duration, and BP), the other conditions were not controlled, such as their usual work. Therefore, the participants in this study may be adequately selected; however, they may not be regarded as a representative sample. Sixth, although the present study

conducted the simulated LWH (12 session; 9:10–21:45) to examine the effects of LWH, there was no short working hour condition as a control. Therefore, the effects of the sessions may contain the effects of not only LWH but also circadian rhythms. To clarify the issue, further studies, which set the short working hours condition, are needed. Seventh, there might be confounders (e.g., the influences of habitual caffeine intake were unknown since the participants were requested to refrain from caffeinated beverages intake only during the experimental periods). Eighth, a statistical power analysis was not conducted to determine the sample size, and the sample size may be small. This potential weakness may have yielded unreliable results. Finally, this study focused on the immediate and direct effects of LWH following SSD on health, however, that may be one minor pathway for the health effects of LWH. Therefore, further studies examining the accumulated and indirect effects of LWH and SSD are needed.

In conclusion, this study found that simulated one-day LWH caused an increase in systolic BP and deterioration of fatigue, and SSD (5 hours of sleep) caused an increase in stroke volume, deterioration of sleepiness, fatigue, and stress. However, these interactions were not influenced by hemodynamic and psychological responses. These findings provide an improved understanding of the changes that occur in hemodynamic and psychological responses with SSD under simulated LWH. They may contribute to the body of evidence on the relationship between SSD and LWH. However, these effects may reflect a minor aspect of the negative effects of SSD and LWH. Further studies are needed to explore the effects of LWH and SSD on workers' health.

Conflict of Interest

The authors declare that there are no conflicts of interest.

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