



Associations of testosterone and cortisol concentrations with sleep quality in Japanese male workers

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ABSTRACT

Low testosterone concentrations are associated with disrupted sleep, and high levels of cortisol, which is elevated in response to stress, lead to insomnia. This study aimed to investigate the associations of testosterone and cortisol concentrations with sleep quality and to examine potential interactions between them in Japanese working men. This study was a cross-sectional design, and testosterone and cortisol concentrations in blood were the exposure variables and sleep parameters were the outcome variables. The Japanese version of the Pittsburgh Sleep Quality Index was used to measure sleep quality, and it included the total duration of sleep, time in bed (TIB), and sleep efficacy. We included 178 men (mean age = 49.1 years, standard deviation = 9.0) who completed all components in the questionnaire related to sleep and provided blood samples. Testosterone and cortisol concentrations were negatively associated with TIB (standardized beta = -0.15 and -0.24 , $p < 0.05$, respectively), while only testosterone concentrations were positively associated with sleep efficacy (standardized beta = 0.15 , $p < 0.05$). An interaction effect of testosterone and cortisol was significant for TIB and sleep efficacy (standardized beta for interaction term = 0.40 , $p < 0.001$ and -0.22 , $p = 0.012$, respectively). When stratified by cortisol concentrations, the associations between testosterone concentrations and sleep parameters were modified. Our findings suggest that associations between testosterone concentrations and sleep parameters are stronger at low cortisol concentrations, but not at high cortisol concentrations. High cortisol concentrations may diminish associations between low testosterone concentrations and diminished sleep efficacy.

1. Introduction

Insomnia is a common problem in daytime workers [1,2]. A meta-analysis study showed that psychosocial job stress was associated with a greater risk of insomnia [3]. Hormonal changes due to psychological stress are responsible for shorter sleep duration [4]. Recent studies have focused on associations between the endocrine system and sleep disorders [5–7]. The hypothalamic–pituitary–adrenal (HPA) axis is a mediator of the body's response to stress that helps maintain homeostasis [5], and cortisol concentrations are elevated in response to stress. Deep sleep has an inhibitory effect on the HPA axis, while activation of the HPA axis leads to arousal and sleeplessness, with cortisol secretion being related to insomnia [8]. Furthermore, cortisol concentrations

increase with age [9] and are related to diminished sleep quality in aging [10].

Aging is related to decreased sleep quality, including frequent awakening, a shorter total sleep duration, and decreased rapid eye movement (REM) sleep [7,11]. Testosterone concentrations decrease with aging, particularly after the age of 50 years [12]. Plasma testosterone concentrations are correlated with REM-non-REM cycles [7,13], and they can affect the ability of stress to alter sleep in mice [14]. Low testosterone concentrations are associated with sleep deprivation [15], sleep fragmentation [16], and decreased sleep duration [17].

According to a hypothetical discussion by Miller et al. [7]; testosterone and cortisol concentrations are in inverse proportion, such as decreased testosterone and increased cortisol concentrations with aging.

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Because testosterone production is often inhibited by cortisol, cortisol could be part of a mechanistic link explaining the associations between decreased testosterone concentrations and diminished sleep quality. A previous study showed that the ratio of testosterone and cortisol was negatively associated with severe obstructive sleep apnea [5]. Although researchers have hypothesized that there is an interaction effect of testosterone and cortisol on sleep quality, few studies have investigated this possibility and the interaction between cortisol and testosterone in relation to sleep quality is poorly understood.

This study aimed to investigate the associations between testosterone and cortisol concentrations and sleep quality in Japanese working men. We hypothesized that testosterone concentrations are positively associated and cortisol concentrations are negatively associated with sleep quality. An interaction effect of testosterone and cortisol concentrations may be found in sleep quality. Specifically, we hypothesized that cortisol concentrations modify the associations between testosterone concentrations and sleep quality. Therefore, a positive association between testosterone concentrations and sleep quality may be stronger at low cortisol concentrations, but not at high cortisol concentrations.

2. Material and methods

2.1. Participants

To enroll participants in the study, a physician working in a middle-sized ship building company's occupational health services department provided a questionnaire about stress to employees in June 2013. One week before the company's annual health check-up, this self-administered questionnaire was distributed to all employees aged 34 or older to be completed on a voluntary basis and returned at the time of their health check-up. Among 485 distributed questionnaires, 368 (338 men and 30 women) were collected (response rate = 75.9%), and blood samples were collected from 219 employees (199 men and 20 women). Participants were limited to 178 men who completed all components of the questionnaire related to sleep and provided blood samples.

Blood samples were collected between 11:30 a.m. and 1:00 p.m. because of the varied schedules of the individual participants. Serum testosterone (ng/dL) and cortisol concentrations ($\mu\text{g/dL}$) were estimated using a chemiluminescent immunoassay technique. These estimations were conducted by BML, Inc. (Japan).

Previous study methods related to job stress and blood sample collection have been reported in detail elsewhere [18–20].

2.2. Questionnaire

The Japanese version of the Pittsburgh Sleep Quality Index ([PSQI] Buysse et al., 1989 [21]); was used to measure sleep quality. The PSQI is widely used for clinical and epidemiological research [21,22]. The PSQI consists of 18 items, which comprise sleep habits and quality during the past 1 month. The scores range from 0 to 21, and a higher score indicates worse sleep quality. Sleep parameters comprising slept hours (total time spent asleep), time in bed (TIB: actual time spent in bed), and sleep efficiency (ratio of the total time spent asleep to the actual time spent in bed multiplied by 100) were measured by items adopted from the PSQI. Additionally, sleep-disordered breathing was asked about in the questionnaire. Sleep-disordered breathing was considered present if the participants' family pointed out that their breathing had stopped while they were sleeping and whether they snored once a week or more.

The questionnaire requested background information, such as age, weight, height, marital status (married = 1 or not married = 0), smoking status (current smoker or past smoker = 1, or never smoked = 0), alcohol consumption (drinking every day = 1 or drinking 6 days per week or less = 0), and exercise habits (habitually exercise with sweating once a week or more = 1 or no exercise habit = 0). The body mass index was calculated in kg/m^2 .

For the work environment, participants were asked about

occupational class (engaging in management tasks as a manager = 1, or none as a general worker = 0), work hours per week, and whether they had a night shift (having a night shift = 1 or not having = 0). A Japanese version of the Job Content Questionnaire (JCQ) [23,24] was used to measure work environment characteristics. This questionnaire comprises scales related to job control (nine items), job demand (five items), and support at work (eight items). The Japanese version of the JCQ has been validated and tested for reliability [24]. Job control assesses the ability to make decisions, be creative on the job, and develop one's ability. Job demand assesses the quantity of work, intellectual requirements, and time constraints of the job. Support at work assesses the participants' perceived support from supervisors (four items) and colleagues (four items). A 4-point Likert scale from 1 (completely disagree) to 4 (completely agree) is used. The scores of job control, job demand, support from supervisors, and support from colleagues were calculated by summing the scores according to the JCQ guidelines (<http://www.jcqcenter.org/>). Cronbach's alpha coefficients in the study participants were 0.76 for job control and 0.77 for job demand, 0.92 for support from a supervisor, and 0.84 for support from coworkers.

Depressive symptoms were assessed using the Japanese translation of the K10 scale [25,26]. The K10 scale consists of 10 items with a choice from 0 (not at all) to 4 (>5 days). Cronbach's alpha coefficient was 0.96. The total score (range: 0–40) has been used as an indicator of severe mental disorders or mood and anxiety disorders [27].

2.3. Ethical treatment

This study was conducted in accordance with the guidelines laid down in the Declaration of Helsinki and approved by the Institutional Review Board of Baika Women's University (approval number: 0013-0001). Written informed consent was obtained from all participants.

2.4. Statistical analyses

Natural log transformation for testosterone and cortisol was applied before statistical analyses. A Shapiro-Wilk test was performed and measured variables departed from normality ($p < 0.05$) except for job demand and body mass index. The median with interquartile range was used as well as the mean and SD. Pearson's and Spearman's correlation coefficients were calculated for continuous variables. A logistic regression analysis was performed to investigate the associations of testosterone and cortisol concentrations with sleep-disordered breathing, with adjustment for age, body mass index (dummy variable: 1 for ≥ 25 and 0 for $< 25 \text{ kg/m}^2$), marital status, night shift, job demand, job control, support from a supervisor, support from coworkers, and depressive symptoms. These potential confounding factors were taken into account based on the results of correlational analyses for continuous variables, and associations with testosterone, cortisol, and sleep parameters were investigated with the *t*-test and Mann Whitney's *U* test for categorical variables. The association of the ratio of testosterone to cortisol concentrations with sleep-disordered breathing was also investigated based on a previous study [5]. Multiple regression analyses were performed to examine the associations between testosterone and cortisol concentrations and sleep parameters, with adjustment for potential confounding factors. An interaction term involving testosterone and cortisol concentrations was also inserted into the model. For multiple regression analyses stratified by cortisol concentrations, the associations between testosterone concentrations and sleep parameters were investigated, with adjustment for potential confounding factors by low and high cortisol concentrations separately. Cortisol concentrations were categorized using the median cortisol concentration (median = 11.10 $\mu\text{g/dL}$). Missing variables were taken into the model as dummy variables. All tests were two-tailed, and $p < 0.05$ was considered statistically significant. Statistical analyses were carried out using IBM SPSS v27.0 (IBM Corp., Armonk, NY, USA).

3. Results

The participants' mean age was 49.1 years (standard deviation [SD] = 9.0), and their age ranged from 34 to 64 years. The participants' characteristics are detailed in Table 1.

Men who were married and working night shifts showed lower cortisol concentrations compared with their counterparts (night shift $M = 6.6 \mu\text{g/dL}$, $SD = 1.9$, not night shift $M = 10.7 \mu\text{g/dL}$, $SD = 1.4$, $t(176) = 3.08$, $p = 0.002$ and $U = 202.5$, $Z = -2.03$, $p = 0.043$; married $M = 10.2 \mu\text{g/dL}$, $SD = 1.4$, not married $M = 11.6 \mu\text{g/dL}$, $SD = 1.5$, $t(175) = 2.21$, $p = 0.029$ and $U = 2388$, $Z = -2.34$, $p = 0.019$). Men with sleep disordered breathing showed longer TIB ($M = 7.8$, $SD = 4.2$) and lower sleep efficacy ($M = 91.0\%$, $SD = 21.2$) than their counterparts (TIB $M = 7.0$, $SD = 1.4$; sleep efficacy $M = 95.3\%$, $SD = 9.5$); however, this difference did not reach statistical significance (TIB $p = 0.057$ for t -test and $p = 0.47$ for U test; sleep efficacy $p = 0.074$ for t -test and $p = 0.19$ for U test).

Table 2 shows Pearson's correlation coefficients between testosterone and cortisol concentrations, measured lifestyle factors, and variables of sleep quality. While testosterone concentrations were not significantly correlated with variables of sleep quality, cortisol concentrations were negatively correlated with TIB ($r = -0.32$, $p < 0.01$); however, Spearman's correlation coefficient showed no significant correlation. We then determined Pearson's and Spearman's correlation coefficients stratified by lower and higher cortisol levels. For lower cortisol levels, Pearson's correlation coefficients showed significant negative correlations of testosterone and cortisol with TIB ($r = -0.23$ and -0.52 , respectively, $p < 0.05$) and a positive correlation of cortisol

Table 1
Participants' characteristics (N = 178).

	Mean	SD	Median	Interquartile range
Slept hours (hours)	6.5	1.0	6.5	1.0
Time in bed (hours)	7.1	2.3	7.0	1.5
Sleep efficacy (%)	94.4	12.9	100.0	7.7
Sleep quality	4.0	2.7	3.0	4.0
Testosterone (ng/dL)#	535.0	179.8	6.3	0.4
Cortisol ($\mu\text{g/dL}$)#	11.2	3.7	2.4	0.5
Age (years)	49.1	9.0	50.5	17.0
Depressive symptoms	17.1	8.2	40.0	8.0
Working time (hours/week)	43.0	17.1	14.0	9.0
Job demand	32.3	6.5	32.5	8.3
Job control	67.4	11.3	68.0	14.5
Support from supervisor	11.1	2.5	12.0	2.0
Support from coworkers	11.1	2.0	11.0	2.0
Body mass index	24.3	3.4	24.1	4.2
	%			
Managers	34.3			
Night shift	2.8			
Married	72.5			
Alcohol drinking habits‡	57.3			
Exercise habits‡	42.7			
Sleep disordered breathing‡	20.2			
Smoking status				
Non smoking	31.5			
Currently smoking	41.0			
Past smoking	27.5			
Education attainment				
Junior high school	1.4			
High school	50.4			
Vocational school	12.8			
Junior college	2.1			
University	29.1			
Graduate university	3.5			
Others	0.7			

#Geometric mean.

‡Alcohol consumption defined as drinking every day, and exercise habits defined as habitual exercise with sweating once a week or more.

‡Sleep-disordered breathing was considered present if the participants' family pointed out that their breathing had stopped while they were sleeping and whether they snored once a week or more.

with sleep efficacy ($r = 0.41$, $p < 0.01$), but not for higher cortisol levels. Spearman's correlation coefficients showed no significant correlation.

Table 3 shows the odds ratios and 95% confidence intervals (CIs) of testosterone and cortisol concentrations and potential confounding factors in relation to variables of sleep-disordered breathing. Testosterone and cortisol concentrations were not associated with the risk of sleep-disordered breathing, whereas obesity (body mass index $\geq 25 \text{ kg/m}^2$) was significantly associated with an increased risk (odds ratio = 3.66, 95% CI: 1.60 to 8.37). When an interaction term of testosterone and cortisol concentrations was taken into the model, the association with sleep disordered breathing was not significant. Furthermore, when the ratio of testosterone to cortisol concentrations was included in the model, it was not significantly associated with sleep-disordered breathing (odds ratio = 1.61, 95% CI: 0.79 to 3.27).

The results of multiple regression analyses between testosterone and cortisol concentrations and measured variables of sleep are shown in Table 4. Testosterone and cortisol concentrations were negatively associated with TIB (standardized beta = -0.15 and -0.24 , $p < 0.05$, respectively), while testosterone concentrations were also positively associated with sleep efficacy (standardized beta = 0.15 , $p < 0.05$) (Model 1). When an interaction of testosterone and cortisol concentrations was included in the model, the interaction effect was significant for TIB (standardized beta = 0.40 , $p < 0.001$) and sleep efficacy (standardized beta = -0.22 , $p < 0.05$) (Model 2).

When stratified by cortisol concentrations, testosterone concentrations were negatively associated with TIB when cortisol concentrations were low (standardized beta = -0.23 , $p < 0.05$) (Model 3), while no significant association was found at high cortisol concentrations (standardized beta = 0.10 , $p = 0.42$) (Model 4). The positive association between testosterone concentration and sleep efficacy did not reach statistical significance for low cortisol concentrations (standardized beta = 0.19 , $p = 0.057$) (Model 3) or high cortisol concentrations (standardized beta = 0.05 , $p = 0.70$) (Model 4). However, the standardized beta for low cortisol concentrations indicated a higher value compared with that for high cortisol concentrations.

4. Discussion

The present study showed that testosterone and cortisol concentrations were negatively associated with TIB, while testosterone concentrations were positively associated with sleep efficacy. An interaction effect of testosterone and cortisol concentrations was also significant for TIB and sleep efficacy. Cortisol concentrations affected the associations between testosterone concentrations and these sleep parameters. These findings suggested that the associations between testosterone concentrations and sleep parameters were stronger at low cortisol concentrations, but not at high cortisol concentrations.

The results of the present study support the hypothesis proposed in a previous study [7] that cortisol affects the associations between testosterone and sleep quality. Low testosterone concentrations are associated with sleep problems, such as nocturnal awakenings and lower sleep efficiency [28,29]. Testosterone concentrations can decline as a result of shortened or fragmented sleep, possibly due to altered nocturnal testosterone secretion [30,31]. However, cortisol concentrations are positively associated with wakefulness and negatively associated with slow wave sleep [32]. During slow wave sleep, sympathetic nervous activity is decreased and cortisol secretion is inhibited [33]. Sleep loss prevents the usual fall in cortisol concentrations that occur during the late afternoon and early evening [34]. HPA axis activation is a primary stress-mediating pathway leading to increased peripheral cortisol concentrations [35]. HPA activation disrupts sleep and promotes wakefulness [36]. The stress-related suppression of testosterone is thought to be a consequence of an elevation in cortisol concentrations [5]. In male workers subjected to high work stress, HPA activation increases cortisol secretion, which could have an effect on testosterone and sleep quality.

With regard to associations between testosterone and sleep,

Table 2
Pearson's and Spearman's correlation coefficients of testosterone, cortisol, and measured factors in relation to sleep parameters.

	Slept hours		Hours in bed		Sleep efficacy (%)		Sleep quality	
	Pearson's r	Spearman's r	Pearson's r	Spearman's r	Pearson's r	Spearman's r	Pearson's r	Spearman's r
Testosterone (ng/dL) ^a	0.13	0.10	-0.13	0.05	0.12	0.05	-0.10	-0.09
Cortisol (µg/dL) ^a	-0.11	-0.11	-0.32**	-0.10	0.13	-0.07	0.08	0.13
Age (years)	0.00	-0.01	0.14	0.15*	-0.18*	-0.13	0.10	0.06
Body mass index	-0.17*	-0.16*	-0.03	-0.19*	0.03	0.03	0.08	0.06
Depressive symptoms	-0.17*	-0.12	-0.11	-0.07	-0.05	-0.1	0.51**	0.53**
Working time (hours/week)	-0.07	-0.14	-0.07	-0.16*	0.05	0.05	-0.05	0.02
Job demand	-0.18*	-0.16*	-0.08	-0.13	-0.02	-0.1	0.16*	0.18*
Job control	0.03	0.04	-0.25**	-0.06	0.21**	0.10	-0.23**	-0.21**
Support from supervisor	0.11	0.10	0.16*	0.09	-0.03	0.00	-0.21**	-0.19*
Support from coworkers	0.12	0.11	-0.09	0.00	0.13	0.11	-0.32**	-0.31**

*p < 0.05; **p < 0.01.
a log transformed.

Table 3
Odds ratios and 95% CI of associations with sleep disordered breathing[†].

	OR#	95% CI	
		Lower	Upper
Testosterone (continuous)	3.04	0.86	10.75
Cortisol (continuous)	0.61	0.20	1.89
Age (continuous)	0.99	0.94	1.03
Body mass index ≥25 (reference = BMI < 25)	3.66	1.60	8.37
Married (reference = not married)	3.51	1.08	11.39
Support from supervisor (continuous)	1.08	0.92	1.28

#ORs are for 1-unit increase, and fully adjusted with variables in the model.
†Sleep-disordered breathing was considered present if the participants' family pointed out that their breathing had stopped while they were sleeping and whether they snored once a week or more.

testosterone concentrations are reduced after sleep curtailment and deprivation [31]. In the present study, testosterone concentrations were not significantly correlated with sleep parameters according to correlation analyses. However, when stratified by shorter and longer sleep duration based on the median, testosterone concentrations were negatively correlated with TIB ($r = -0.32, p < 0.01$) for shorter sleep duration but not for longer sleep duration, according to Pearson's correlation coefficients [31]. found a reduction in serum testosterone concentrations after total sleep deprivation and sleep restriction with early awakening due to a reduced sensitivity of gonadal Leydig cells, which are stimulated by luteinizing hormone. However, large epidemiological cohort studies in adult men have not consistently shown a

Table 4
Results of multiple regression analyses of testosterone and cortisol with sleep parameters.

	Slept hours		Hours in bed		Sleep efficacy (%)		Sleep quality	
	Standardized-beta#	p	Standardized-beta#	p	Standardized-beta#	p	Standardized-beta#	p
Model 1								
Testosterone (ng/dL)	0.13	0.081	-0.15	0.034	0.15	0.047	-0.09	0.16
Cortisol (µg/dL)	-0.12	0.15	-0.24	0.001	0.10	0.205	0.09	0.18
Model 2								
Testosterone (ng/dL)	0.09	0.26	-0.02	0.73	0.08	0.30	-0.08	0.27
Cortisol (µg/dL)	-0.15	0.071	-0.14	0.042	0.05	0.55	0.10	0.15
Testosterone × Cortisol	-0.14	0.12	0.40	<0.001	-0.22	0.012	0.05	0.51
Model 3								
Testosterone (ng/dL)	0.17	0.12	-0.23	0.013	0.19	0.057	-0.07	0.46
Model 4								
Testosterone (ng/dL)	0.14	0.26	0.10	0.42	0.05	0.70	-0.07	0.49

Standardized-beta was adjusted for age, body mass index, marital status, night shift, sleep disordered breathing, job demand, job control, support from supervisor, support from coworkers, and depressive symptoms.

Model 1: sleep measure is an outcome and testosterone and cortisol are explanation variables adjusted for covariates.

Model 2: sleep measure is an outcome and testosterone, cortisol, and testosterone × cortisol are explanation variables adjusted for covariates.

Model 3: sleep measure is an outcome and testosterone is an explanation variable adjusted for covariates in the low cortisol group.

Model 4: sleep measure is an outcome and testosterone is an explanation variable adjusted for covariates in the high cortisol group.

cross-sectional association between the sleep duration and testosterone concentrations [28,33,37]. Interestingly, testosterone concentrations are related to sleep quality, but not to the total sleep duration. Circulating testosterone concentrations strongly depend on sleep efficacy and quality [16,38,39]. Similarly, the present study showed that testosterone concentrations were associated with sleep efficacy, but not with the duration of sleep. Disrupted sleep quality arising from obstructive sleep apnea is associated with frequent awakenings, fragmented sleep, and less REM sleep [33]. Severe obstructive sleep apnea is associated with lower blood testosterone concentrations [33].

In the present study, testosterone concentrations were not significantly associated with sleep-disordered breathing and the total score of sleep quality [5]. showed that the ratio of testosterone to cortisol concentrations was significantly lower in patients with severe sleep-disordered breathing. However, the ratio of testosterone to cortisol was not associated with a risk of sleep-disordered breathing in the present study. The reasons for this discrepancy between studies may be due to the measurement of sleep-disordered breathing, which was estimated by self-reporting, and it could not be determined whether it was severe. Although the association between testosterone concentrations and sleep-disordered breathing did not reach statistical significance in the present study, testosterone showed a tendency to increase the risk of sleep-disordered breathing. However, the high body mass index showed an increased risk of sleep-disordered breathing, and higher testosterone concentrations were negatively correlated with the body mass index in the present study (data not shown). Obesity is a risk factor of obstructive sleep apnea [40], and obesity may affect testosterone concentrations, which also have significant effects on protein metabolism and lipolysis

[41]. Decreased testosterone concentrations may lead to an increase in obesity and disruption of sleep quality in men. Further studies are necessary to investigate the effects of endocrine changes on metabolic control and sleep.

Some limitations to the present study should be discussed. First, all participants were workers at a target company and they participated in the present study on a voluntary basis. Therefore, the study population was not representative of the Japanese male working population, and self-selection bias could have effects on the results. When we compared the analyzed participants with the excluded male participants, the analyzed participants were older, had lower support from coworkers, and there were higher percentage of managers, married men, an alcohol drinking habit, and sleep-disordered breathing, and a lower percentage of non-smokers (Supplemental Table A). The sample size was also small and measured data were departed from normality, even after log transformation. When mean centered variables (measured data – mean) were used, the results were the same (data were not shown). Second, the cross-sectional design did not permit causal interpretation. Third, blood samples were collected during daytime at approximately lunch time (between 11:30 a.m. and 1:00 p.m.) at the workplace because the study was conducted at the annual health check. Testosterone and cortisol concentrations change with circadian rhythms and seasonal variations [42,43]; therefore, time of blood sampling should be controlled. Finally, confounding factors, such as aging, chronic illness, and health behavior, may affect testosterone and cortisol concentrations [44–48]. In the present study, some health-related lifestyle factors were adjusted for, and lifestyle habits including smoking status, alcohol habits, and physical activity were not associated with testosterone concentrations or sleep parameters. When smoking status, and physical activity were included in the model, the results were not altered. However, history of chronic illness, including diabetes mellitus and hypertension, were not measured in the present study. With regard to taking sleep medication, 5% of the participants had this habit as shown by the PSQI. Although depressive symptoms were also asked about using the K10, there was no question about a history of mental health. Because there are many potential confounding factors, controlling for the effect of these variables is impossible.

5. Conclusions

Testosterone concentrations are significantly associated with sleep efficacy, and there is an interaction effect of testosterone and cortisol concentrations on sleep efficacy. Cortisol concentrations affect the associations between testosterone concentrations and sleep parameters, and high cortisol concentrations may diminish the association between decreased testosterone concentrations and diminished sleep efficacy. Cortisol secretion can increase when workers are subjected to job stress, which also leads to adverse sleep quality; therefore, stress management at work should be an important countermeasure. For future studies, the mechanism of better sleep quality that is related to testosterone and cortisol should be clarified while taking into account aging in male workers.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.cpnec.2022.100158>.

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