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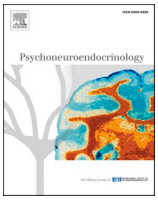

Separating the influences of means and daily variations of sleep on the stress-induced salivary cortisol response

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ABSTRACT

Background: Previous research regarding the effects of sleep quality and quantity on the acute stress response has yielded inconsistent findings. This may be attributed to various factors, including composite sleep components (i.e., means and daily variations) and mixed cortisol stress response (i.e., reactivity and recovery). Thus, this study aimed to separate the effects of means and daily variations of sleep on the reactivity and recovery of cortisol responses to psychological challenges.

Methods: In study 1, we recruited 41 healthy participants (24 women; age range, 18–23 years), monitored their sleep during seven consecutive days via wrist actigraphy and sleep diaries, and adopted the Trier Social Stress Test (TSST) paradigm to induce acute stress. Study 2 consisted of a validation experiment using the ScanSTRESS paradigm, which included 77 additional healthy individuals (35 women; age range, 18–26 years). Similarly to the TSST, the ScanSTRESS induces acute stress using uncontrollability and social evaluation. In both studies, saliva samples from the participants were collected before, during, and after the acute stress task.

Results: Using residual dynamic structural equation modeling, both study 1 and study 2 demonstrated that higher means of objective sleep efficiency, and longer means of objective sleep duration were related to greater cortisol recovery. In addition, fewer daily variations in objective sleep duration were associated with greater cortisol recovery. However, there was no correlation between sleep variables and cortisol reactivity, except for the daily variations in objective sleep duration in study 2. No correlation was observed between subjective sleep and cortisol response to stress.

Conclusions: The present study separated two features of multi-day sleep patterns and two components of cortisol stress response, providing a more comprehensive picture of the effect of sleep on the stress-induced salivary cortisol response, and contributing to the future development of targeted interventions for stress-related disorders.

1. Introduction

The hypothalamic-pituitary-adrenal (HPA) axis is a core stress system that plays an important role in enabling individuals to deal with acute psychological challenges (Dickerson and Kemeny, 2004). Cortisol, the end-product of the HPA axis, is an effective indicator of stress due to its sensitivity to acute stress (Dickerson and Kemeny, 2004; Hellhammer et al., 2009). Numerous factors, including sleep, influence the cortisol stress response. Recent studies reported that poor sleep (i.e., low sleep quality or short sleep duration) enhances (Bassett et al., 2015; Raikkonen et al., 2010), attenuates (Jackowska et al., 2017), or has no effect (Wright et al., 2007) on the cortisol stress response. These conflicting results may be attributed to various factors (Zhao et al., 2021), such as composite sleep components (i.e., means and daily variations) and mixed cortisol stress response (i.e., reactivity and recovery).

The relationship between sleep quantity and quality and the cortisol stress response may be complicated by the presence of composite sleep components. Two components describe the sleep/wake patterns across multiple days: the mean (i.e., the overall level across the entire period) and the variability (i.e., the daily variations around the mean) (Bei et al., 2016). The existing literature regarding the influence of sleep on the cortisol stress response focuses primarily on mean sleep. For example, previous studies measured sleep for several consecutive days via actigraphy and found that poor mean sleep efficiency increased or had no

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effect on the cortisol stress response (Hatzinger et al., 2014; Massar et al., 2017; Raikkonen et al., 2010). However, to date, no research has examined the relationship between daily variations in sleep and cortisol stress responses. Daily variations in sleep may also play a significant role in the response to stress. For instance, greater daily variations in sleep duration measured by actigraphy were independently correlated with higher perceived stress (Veeramachaneni et al., 2019). Further, even after controlling for mean of sleep, greater daily variations in sleep were associated with a higher risk of experiencing the stress-related disease (Slavish et al., 2019; Vidal Bustamante et al., 2020). Thus, this study aimed to separate the effects of means and daily variations of sleep on stress-induced salivary cortisol response to provide a comprehensive profile.

In addition to the composite sleep components, the mixed cortisol stress response was a confounding factor. The cortisol stress response showed a temporal trajectory, with cortisol levels peaking approximately 20–30 min after the onset of the acute stressor (cortisol reactivity), and then declining to baseline levels (cortisol recovery) (Dickerson and Kemeny, 2004). In view of mounting evidence showing that both extremes (exaggerated and diminished stress reactivity) are associated with unhealthy behaviors and adverse health outcomes, it would appear that the optimal response to stress is a moderate reaction (Miller et al., 2007; Phillips et al., 2013). In addition, the recovery of cortisol levels from peak to baseline when needed is essential. Failed recovery can disrupt homeostasis, leading to long-term negative health consequences (Fiksdal et al., 2019; Holsboer and Ising, 2010). Accordingly, sufficient, but not excessive, reactivity paired with adequate recovery may be considered as an efficient cortisol stress response pattern (He et al., 2021). Notably, previous studies have only examined the influence of sleep on overall or cortisol stress reactivity (e.g., Jackowska et al., 2017; Raikkonen et al., 2010). However, sleep may have a differential effect on cortisol reactivity and on recovery, which may lead to an ambivalent relationship between sleep and the overall cortisol stress response. In fact, earlier studies found that poor sleepers showed higher or similar levels of cardiovascular reactivity but consistently lower recovery from psychological stress compared to good sleepers (Brindle and Conklin, 2012; Massar et al., 2017; Mezick et al., 2014). Accordingly, we may hypothesize that good sleepers will have a more efficient cortisol stress response pattern, especially a greater recovery (i.e., post-peak decrease) in cortisol.

The present study aimed to explore the influence of means and daily variations of sleep on the reactivity and recovery of cortisol stress responses. Separating the differences between means and daily variations of sleep and those between cortisol reactivity and recovery will clarify the impact of sleep on stress-induced salivary cortisol response and contribute to subsequent targeted interventions for stress-related disorders. Notably, this study focused on sleep efficiency and duration, which are commonly used as indicators when exploring the effects of multi-day sleep on the HPA axis function (Massar et al., 2017; Raikkonen et al., 2010; Van Lenten and Doane, 2016). According to the method suggested in this field (Zhao et al., 2021), we measured sleep for 7 consecutive days via wrist actigraphy and sleep diaries. In study 1, we adopted The Trier Social Stress Test (TSST) paradigm to induce acute stress. The magnitude of cortisol stress response depends on the chosen stress-inducing task (Dickerson and Kemeny, 2004), which may further influence the association between sleep and stress response (Zhao et al., 2021). Therefore, in study 2, we used the ScanSTRESS paradigm with an independent sample to assess reliability. The ScanSTRESS paradigm triggers acute stress in the same way (uncontrollability and social evaluation) but induces a weaker HPA axis response compared to that of the TSST paradigm (Duan et al., 2017).

2. Material and methods

This study was approved by the review board of the Faculty of Psychology of Southwest University (no. H22008).

2.1. Study 1

2.1.1. Participants

A total of 43 healthy university students from Southwest University, Chongqing, China, were recruited as paid volunteers through Internet advertising. Among them, two participants were excluded for failing to complete all measures, leaving a final sample of 41 participants (24 females; age: mean [± standard deviation (SD), 20.12 (± 1.60) years, and range, 18–23 y). Because the post-stress cortisol levels of women in the luteal phase approach those of men, the female participants were all in their luteal phase (Kajantie and Phillips, 2006). Participants involved in the study were free of any psychiatric, neurological, or sleep disorders; were not taking psychotropic or glucocorticoid medications; and were not abusing alcohol or other substances.

2.1.2. Measurements

2.1.2.1. Sleep measures. Sleep duration (total sleep time, TST) is the time between trying to sleep and getting up, minus the time of latency (time between trying to sleep and actually falling asleep) and the time of awake after sleep onset. Sleep efficiency (SE) is sleep duration divided by the time between trying to sleep and getting up * 100% (Van Den Berg et al., 2008; Wright et al., 2007).

Objective sleep parameters were obtained via actigraphy (wGT3X-BT, Pensacola, USA), a non-invasive method for measuring sleep. The sampling intervals of actigraphy were set at 60 s to obtain summary statistics via the ActiLife software (6.13.4) and manufacturer algorithms. Sleep diary data were used to establish the scoring interval for actigraphic sleep (Acebo et al., 1999). The Cole-Kripke algorithm (Cole et al., 1992) was used to calculate the objective TST and objective SE automatically.

Subjective sleep parameters were measured using sleep diaries comprising the following questions: (1) When did you turn off your phone, close your eyes, and try to sleep last night? (2) How many minutes did it take to fall asleep the previous night? (3) When did you wake up today (eyes open, ready to get up)? (4) After falling asleep, how many minutes did you wake up last night in total?

2.1.2.2. Acute stress induction. The TSST paradigm, which contains uncontrollable and social-evaluative elements, is a widely used acute stress paradigm that activates the HPA axis response (Kirschbaum et al., 1993). The TSST consisted of an unrehearsed speech task (5 min), where participants were asked to elaborate on their abilities that would make them the best candidate for a chosen job, and a verbal subtraction task (5 min) in front of a camera and two judges (a male and a female).

2.1.2.3. Salivary cortisol. Saliva samples were collected using Salivette sampling devices (Salivette, SARSTEDT, Germany) and stored in a −20°C freezer until assay. Cortisol concentrations were measured by ELISA (IBL, Hamburg, Germany) following the manufacturer's instructions.

2.1.3. Procedure

The study consisted of three sessions. During session 1, the participants provided written informed consent and demographic details. Participants were instructed on how to use the wrist actigraph and how to complete the sleep diary. During session 2, sleep was monitored at home for 7 consecutive days via wrist actigraphy and sleep diaries. Following session 2, participants entered the laboratory where they were subjected to an acute stress task. All acute stress tasks were conducted between 1:30 pm and 5:00 pm to control the circadian rhythm of cortisol. Participants were asked not to smoke or engage in any strenuous exercise, drink alcohol or caffeine, eat, or brush their teeth for at least 1 h before the acute stress task. Upon arrival, we required the participants to rest for 30 min and then collected the baseline cortisol

(cort1). After 10 min, the TSST task began. After finishing the task, participants were instructed to rest. Participants were allowed to sit and read geography magazines during the rest period but were prohibited from using their phones. During the entire experiment, cortisol samples were collected at six timepoints: 0 min (baseline, cort1), + 10 min (TSST start, cort2), + 20 min (TSST end, cort3), + 30 min (cort4), + 50 min (cort5), and + 60 min (cort6).

2.2. Study 2

2.2.1. Participants

Eighty healthy college students from Southwest University in Chongqing, China, were recruited as paid volunteers through Internet advertising. Among them, three participants were excluded because they did not complete the acute stress task, leaving a final sample of 77 (35 females; age: mean (± SD) 20.18 (± 1.97) and range, 18–26 y). The female participants in the current study were all in their luteal phase (Kajantie and Phillips, 2006).

2.2.2. Acute stress induction

The ScanSTRESS paradigm was adopted in this study (Henze et al., 2020; Streit et al., 2014). It included both socially evaluated components (i.e., verbal and non-verbal feedback from the experimenters) and uncontrollable components (i.e., task difficulty and time constraints), which were core components inducing the HPA axis responses to acute stressors (Dickerson and Kemeny, 2004). Specifically, the stress condition involved participants completing challenging serial subtraction and mental rotation tasks under time pressure as displayed on a countdown timer. The speed and difficulty of the stress tasks were adapted to the individual performance to ensure a low success rate. Furthermore, participants were constantly presented with a live video feed of the two experimenters (one female and one male), who gave unsatisfactory visual feedback when participants answered incorrectly or slowly. Under the control condition, participants were asked to match numbers and figures with no time pressure or feedback. The live video stream was overlaid by a grey diagonal cross, and the two experimenters did not observe the participants. In addition, participants were given negative verbal feedback in the middle of the stress task to increase their sense of social evaluation. ScanSTRESS was performed in the magnetic resonance imaging environment; the brain data were not reported here because they were collected to assess different hypotheses from those in this study.

2.2.3. Procedure

All procedures in study 2 were identical to those of study 1, except for the acute stress task. Following 7 consecutive days of sleep measurement, the participants came to the laboratory to complete the acute stress task. Five saliva samples were collected, including the cort1 sample obtained 30 min after the participant arrived and had rested; the cort2, cort3, and cort4 samples were obtained at mid-ScanSTRESS, at the end of ScanSTRESS, and after a 20-min rest, respectively. The cort5 sample was obtained after a further 10-min rest. Participants were allowed to sit and read geography magazines during the rest period but were prohibited from using their phones.

2.3. Statistical analyses

Descriptive statistics were computed using SPSS version 25 (IBM Corp., Armonk, NY, USA). Saliva cortisol values were significantly skewed; thus, log10 transformation was used to ensure a normal distribution of cortisol values. We then subtracted the baseline value from the peak value as the measure of cortisol reactivity to acute stress. Cortisol recovery was calculated as the peak cortisol level minus the last measured level.

Residual Dynamic Structural Equation Modeling (RDSEM) using Mplus version 8.3 was adopted to model the relationship between sleep and acute stress response. RDSEM is a framework that combines multilevel, structural equation, time-series, and time-varying effects modeling (Asparouhov et al., 2018; McNeish and Hamaker, 2020). The RDSEM model can be expressed as follows (Fig. 1):

Level 1:

$$Sleep_{ti} = \alpha_i + \beta_i Time_{ti} + e_{ti}$$

$$e_{ti} = \varphi_i e_{(t-1)i} + \delta_{ti}$$

Level 2:

$$\alpha_i = \gamma_{00} + \mu_{0i}$$

$$\beta_i = \gamma_{10} + \mu_{1i}$$

$$\varphi_i = \gamma_{20} + \mu_{2i}$$

$$cortisol \quad reactivity_i = \gamma_{30} + \gamma_{31}\alpha_i + \gamma_{32}\varphi_i + \mu_{3i}$$

$$cortisol \quad recovery_i = \gamma_{40} + \gamma_{41}\alpha_i + \gamma_{42}\varphi_i + \mu_{4i}$$

$$\sigma_i^2 = \exp(\omega + \mu_{5i})$$

The Level 1 sleep ($Sleep_{ti}$) for individual i in day t ($t = 1, 2, \ldots, 7$) is broken down to a mean sleep (intercept value, α_i), time trends (β_i), daily variation (autoregressive coefficient, φ_i), and error (δ_{ti}) components, where $\delta_{ti} \sim (0, \sigma_i^2)$. Specifically, the φ_i represents the extent to which one variable can predict its future status (McNeish and Hamaker, 2020), i.e., a higher φ_i indicates fewer daily variations (or more stability) during the period (Blunden et al., 2019; Moen et al., 2021; Ten Brink et al., 2021). Level 1 components were regarded as random effects, where Level 2 residuals were considered (μ_{0i}, μ_{1i}, μ_{2i}, μ_{3i}, μ_{4i}, and μ_{5i}). Level 2 estimated the effect of sleep parameters on the cortisol responses to acute stress.

3. Results

3.1. Study 1

3.1.1. Descriptive results

Table 1 shows the correlation between the 7-day mean sleep and cortisol stress responses. Specifically, the objective (r=0.43, p=0.005) and subjective (r=0.42, p=0.006) TSTs were positively correlated with cortisol recovery. However, objective (r=0.26, p=0.095) and subjective (r=0.16, p=0.322) SE were not related to cortisol recovery. In addition, none of the sleep parameters were associated with cortisol reactivity (p>0.05).

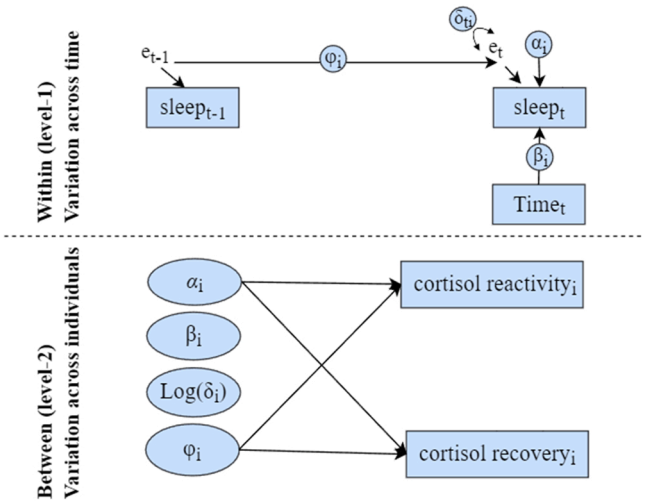

Fig. 1. Path diagram of the residual dynamic structural equation modeling.

Table 1
Means, standard deviations, and correlations between the variables in study 1.

	Mean	SD	1	2	3	4	5	6
Objective SE	88.52	5.51	1.00					
Objective TST	389.50	46.74	.68***	1.00				
Subjective SE	96.66	1.63	0.19	0.05	1.00			
Subjective TST	424.72	37.53	0.24	.86***	0.13	1.00		
cortisol reactivity	0.36	0.29	-0.20	-0.03	-0.04	0.08	1.00	
cortisol recovery	0.11	0.11	0.26	.43**	0.16	.42**	0.27	1.00

Note: Sleep data were averaged across 7 days. *** $p < 0.001$; ** $p < 0.01$. SE = Sleep Efficiency, TST = Total Sleep Time, SD = Standard Deviation.

3.1.2. The effect of objective sleep on the cortisol stress response

The results of the estimates and 95% confidence intervals (CI) for RDSEM are shown in Fig. 2A and Table 2. Longer means of objective TST ($\gamma 41 = 0.003$, 95% CI [0.00, 0.01]) and the fewer daily variations in objective TST ($\gamma 42 = 0.20$, 95% CI [0.00, 0.47]) were associated with greater cortisol recovery. However, there was no correlation between objective TST and stress reactivity.

Moreover, the mean objective SE ($\gamma 41 = 0.03$, 95% CI[0.01, 0.07]), but not the daily variation ($\gamma 42 = 0.19$, 95% CI[−0.12, 0.76]), affected stress recovery (Fig. 2B and Table 2). A one-unit increase in mean SE predicted a 0.03 increase in the amount of cortisol recovery. However, no objective SE component was related to stress reactivity.

3.1.3. The effect of subjective sleep on the cortisol stress response

As described in Table 2 and Fig. 2C and 2D, none of the subjective TST components were associated with cortisol reactivity or recovery. No subjective SE components were related to the acute stress response.

By employing an RDSEM, study 1 showed that longer means of objective TST and higher means of objective SE were linked to greater cortisol recovery. Moreover, individuals with fewer daily variations in TST had greater cortisol recovery. In addition, none of the subjective sleep components was associated with cortisol reactivity and recovery. Considering that the relationship between sleep and cortisol stress response is influenced by the stress-inducing paradigm chosen (Zhao et al., 2021), the ScanSTRESS paradigm, which elicits an attenuated cortisol stress response compared to the TSST paradigm, was applied to an independent sample in study 2 to assess the reliability of the results.

3.2. Study 2

3.2.1. Descriptive results

Table 3 shows the correlation between the 7-day mean sleep and cortisol stress responses. Consistent with the results of study 1, study 2 found that the objective (r=0.41, p<0.001) and subjective (r=0.30, p=0.008) TSTs were positively correlated with cortisol recovery. In addition, none of the sleep parameters were associated with cortisol reactivity (ps>0.05). However, in contrast to study 1, objective SE and cortisol recovery were correlated (r=0.29, p=0.011).

3.2.2. The effect of objective sleep on the cortisol stress response

The estimates and 95% confidence intervals (CI) for RDSEM are shown in Fig. 3 and Table 4. Similar to study 1, study 2 found that longer mean objective TST ($\gamma 41 = 0.01$, 95% CI[0.00, 0.02]) and fewer daily variations in objective TST ($\gamma 42 = 0.51$, 95% CI[0.03, 1.64]) were associated with greater cortisol recovery (Fig. 3 A and Table 4). However, the mean objective TST and stress reactivity were not correlated. In contrast with study 1, study 2 found that daily variations in objective TST were correlated with cortisol reactivity ($\gamma 32 = 0.71$, 95% CI [0.01,

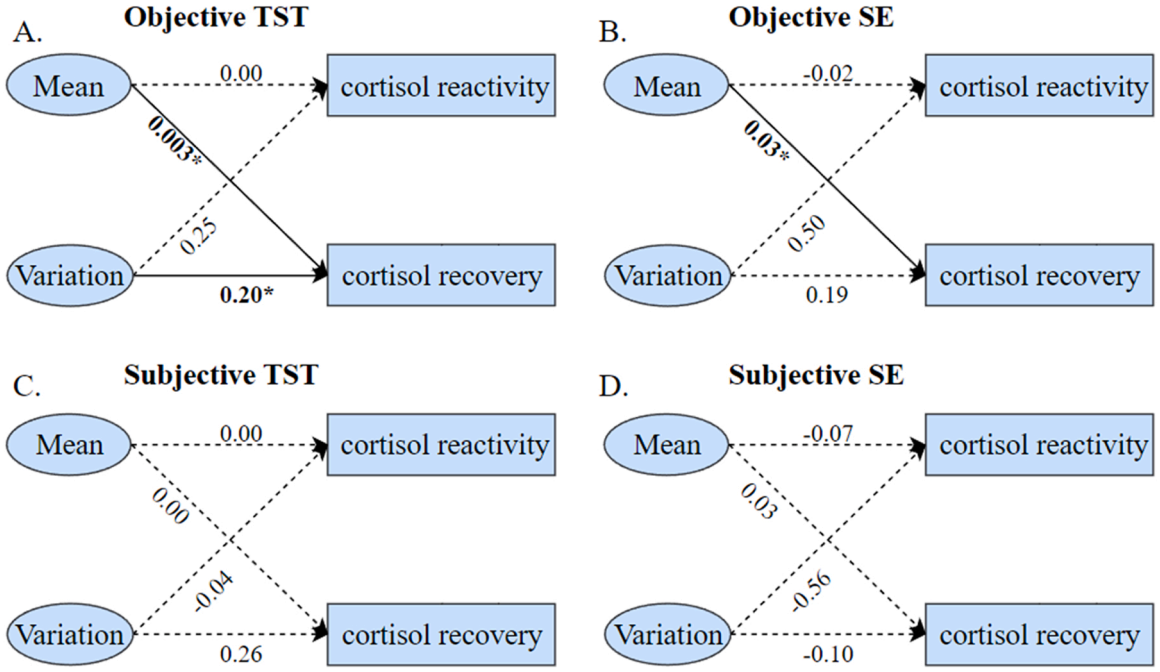

Fig. 2. Estimates for the effect of sleep on the cortisol response in study 1. (A) Estimates for the effect of objective total sleep time on cortisol response. (B) Estimates for the effect of objective sleep efficiency on cortisol response. (C) Estimates for the effect of subjective total sleep time on cortisol response. (D) Estimates for the effect of subjective sleep efficiency on cortisol response. Pathways depicted in bold solid lines with a "* " symbol indicate estimates for which the 95% confidence interval (CI) does not include the zero value (significant). Dashed lines indicate non-significant pathways, which contain the zero value within the CI. The mean (α) and variation (φ) of sleep; greater φ values reflect less daily variation; i.e., more stability. TST = Total Sleep Time, SE = Sleep Efficiency.

Table 2
Estimates and 95% confidence intervals for RDSEM of objective sleep parameters and cortisol response in study 1.

		Objective TST		Objective SE		Subjective TST		Subjective SE	
	Notion	Posterior Median	95% CI	Posterior Median	95% CI	Posterior Median	95% CI	Posterior Median	95% CI
Intercept (alpha)	γ00	388.86*	[366.27, 411.34]	90.24*	[88.34, 92.09]	420.22*	[403.75, 437.13]	97.22*	[96.4, 98.11]
Intercept (beta)	γ10	0.05	[− 4.06, 4.23]	-0.11	[− 0.40, 0.17]	1.52	[− 2.19, 4.94]	0.02	[− 0.11, 0.14]
Intercept (phi)	γ20	0.21*	[0.01, 0.41]	0.41*	[0.15, 0.63]	0.12	[− 0.10, 0.34]	0.29*	[0.03, 0.56]
Intercept (cortisol reactivity)	γ30	0.71	[− 2.87, 7.07]	2.02	[− 3.12, 8.25]	-0.10	[− 358.89, 305.84]	6.93	[− 8.63, 55.49]
Intercept (cortisol recovery)	γ40	-1.03*	[− 3.49, − 0.30]	-2.55*	[− 5.92, − 0.95]	-1.11	[− 118.22, 61.60]	-2.36	[− 15.14, 8.08]
Intercept (logv)	ω	8.01*	[7.68, 8.33]	2.71*	[2.33, 3.09]	7.97*	[7.56, 8.34]	1.33*	[0.80, 1.86]
cortisol reactivity on alpha	γ31	0.00	[− 0.02, 0.01]	-0.02	[− 0.09, 0.03]	0.00	[− 0.74, 0.84]	-0.07	[− 0.56, 0.09]
cortisol recovery on alpha	γ41	0.00*	[0.00, 0.01]	0.03*	[0.01, 0.07]	0.00	[− 0.15, 0.28]	0.03	[− 0.08, 0.16]
cortisol reactivity on phi	γ32	0.25	[− 0.51, 1.01]	0.50	[− 0.42, 2.34]	-0.04	[− 3.68, 3.23]	-0.56	[− 4.52, 2.72]
cortisol recovery on phi	γ42	0.20*	[0.00, 0.47]	0.19	[− 0.12, 0.76]	0.26	[− 0.84, 1.30]	-0.10	[− 1.51, 1.26]
Var. (alpha)	τ00	730.23*	[33.43, 2101.17]	8.18*	[1.59, 20.34]	369.37*	[0, 1560.59]	1.12*	[0.07, 3.03]
Var. (beta)	τ11	15.84*	[0.94, 54.41]	0.07*	[0.00, 0.31]	16.88*	[1.27, 52.42]	0.01*	[0.00, 0.06]
Var. (phi)	τ22	0.09*	[0.02, 0.23]	0.06*	[0.00, 0.21]	0.03*	[0.00, 0.18]	0.04*	[0.00, 0.21]
Var. (logv)	τ55	0.52*	[0.19, 1.12]	0.91*	[0.45, 1.78]	0.88*	[0.41, 1.80]	2.28*	[1.28, 4.09]
Res. Var. (cortisol reactivity)	τ33	0.08*	[0.03, 0.15]	0.07*	[0.01, 0.14]	0.08*	[0.02, 0.15]	0.06*	[0.01, 0.13]
Res. Var. (cortisol recovery)	τ44	0.00*	[0.00, 0.01]	0.01*	[0.00, 0.01]	0.01*	[0.00, 0.02]	0.01*	[0.00, 0.02]

Note: * $p < 0.05$, RDSEM = Residual Dynamic Structural Equation Modeling, SE = Sleep Efficiency, TST = Total Sleep Time, CI= Confidence Interval.

Table 3
Means, standard deviations, and correlations between the variables in study 2.

	Mean	SD	1	2	3	4	5	6
Objective SE	87.35	5.12	1.00					
Objective TST	372.61	38.27	.42**	1.00				
Subjective SE	96.61	2.11	0.09	0.14	1.00			
Subjective TST	414.23	43.20	-0.16	.79**	.30**	1.00		
cortisol reactivity	0.10	0.27	0.02	0.01	-0.13	-0.03	1.00	
cortisol recovery	0.09	0.25	.29*	.41***	0.10	.30**	0.20	1.00

Note: Sleep data were averaged across 7 days. *** $p < 0.001$; ** $p < 0.01$, * $p < 0.05$. SE = Sleep Efficiency, TST = Total Sleep Time, SD = Standard Deviation.

2.07]).

Moreover, consistent with the findings from study 1, study 2 showed that higher mean objective SE (Fig. 3B and Table 4) correlated with greater stress recovery (γ41 = 0.03, 95% CI [0.01, 0.05]). No other objective SE component was related to stress reactivity or recovery.

3.2.3. The effect of subjective sleep on the cortisol stress response

As described in Fig. 3C and 3D, and in Table 4, none of the subjective TST and SE components were associated with cortisol reactivity and recovery, in agreement with the results from study 1.

Overall, study 2 replicated study 1, indicating that the means and daily variations of sleep reliably influenced the cortisol stress response. Furthermore, study 2 found that few daily variations in objective TST were associated with cortisol reactivity.

4. Discussion

To our knowledge, this is the first study to separate the influence of means and daily variations of sleep on the reactivity and recovery of cortisol responses to psychological challenges. Using different acute stress paradigms and different samples, both studies found that objective, rather than subjective, sleep affects HPA axis responses. Specifically, higher mean objective sleep efficiency, longer mean objective sleep duration, and fewer daily variations in objective sleep duration were related to greater cortisol recovery. In addition, sleep and cortisol reactivity were not correlated except for fewer daily variations in sleep duration correlated with greater cortisol reactivity in study 2. Our study provides a novel insight into the relationship between sleep and the HPA axis response to acute stress, which will provide a more comprehensive profile of sleep and health maintenance.

4.1. Association between means of sleep and cortisol stress response

By employing RDSEM, our study determined means of sleep, and found that individuals with better mean sleep (i.e., higher objective sleep efficiency and longer objective sleep duration) exhibited a greater cortisol recovery response. Previous studies have also linked better sleep with greater cardiovascular recovery in responses to acute stress (Brindle and Conklin, 2012; Massar et al., 2017). These suggest both adequate sleep efficiency and sufficient sleep time protect against stress. One potential explanation is that long-term poor sleep efficiency and inadequate sleep duration are forms of chronic stress that lead to chronic activation of the HPA axis. The cumulative model of stress indicates that the chronic activation of the HPA axis leads to dysfunction of stress-mediating systems and eventually to physiological wear and tear of the body (Karatsoreos and McEwen, 2013; Young et al., 2019). Specifically, the HPA axis is not only triggered by acute psychological stressors but also exhibits a distinct diurnal rhythm, with levels peaking

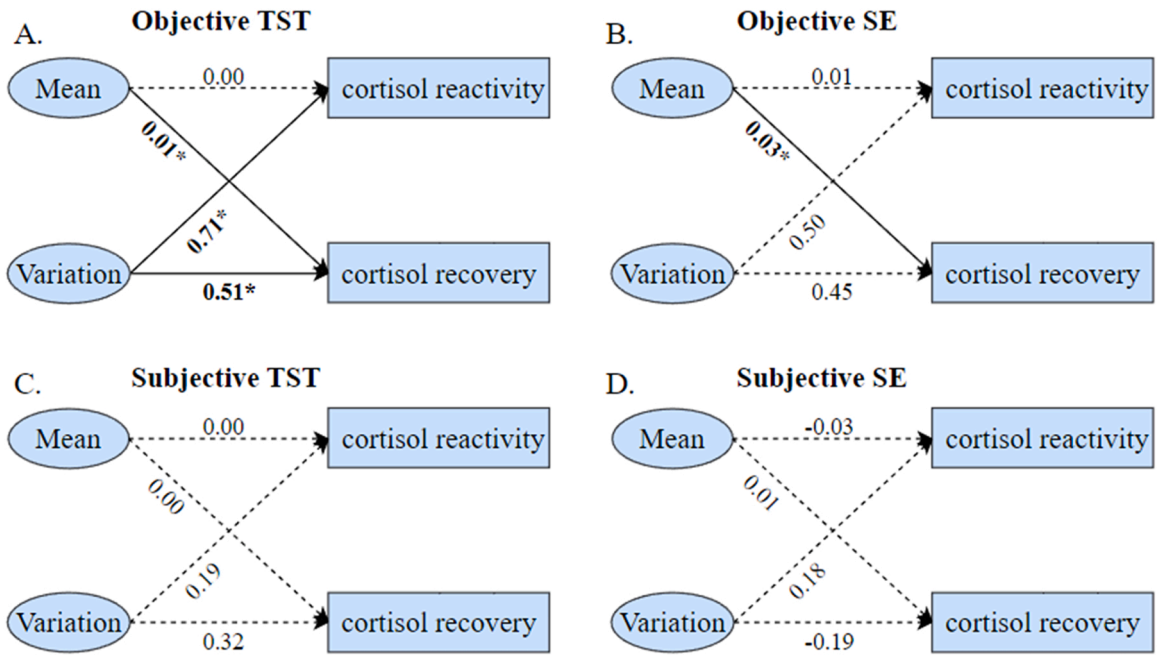

Fig. 3. Estimates for the effect of sleep on cortisol response in study 2. (A) Estimates for the effect of objective total sleep time on cortisol response. (B) Estimates for the effect of objective sleep efficiency on cortisol response. (C) Estimates for the effect of subjective total sleep time on cortisol response. (D) Estimates for the effect of subjective sleep efficiency on cortisol response. Pathways depicted in bold solid lines with a "* " symbol indicate estimates for which the 95% confidence interval (CI) does not include the zero value. Dashed lines indicate non-significant pathways, which contain the zero value within the CI. The mean (α) and variation (φ) of sleep; greater φ values reflect less daily variation; i.e., more stability. TST = Total Sleep Time, SE = Sleep Efficiency.

Table 4
Estimates and 95% confidence intervals for RDSEM of objective sleep parameters and cortisol response in study 2.

		Objective TST		Objective SE					
	Notion	Posterior Median	95% CI	Posterior Median	95% CI	Posterior Median	95% CI	Posterior Median	95% CI
Intercept (alpha)	γ00	361.14*	[348.31, 373.33]	88.3*	[86.86, 89.73]	395.81*	[381.89, 410.14]	97.13*	[96.49, 97.67]
Intercept (beta)	γ10	2.94*	[0.51, 5.56]	-0.04	[− 0.24, 0.14]	4.21*	[1.52, 7.12]	0.08*	[0.01, 0.15]
Intercept (phi)	γ20	0.12	[− 0.01, 0.25]	0.19*	[0.03, 0.37]	0.19*	[0.03, 0.34]	0.28*	[0.08, 0.49]
Intercept (cortisol reactivity)	γ30	-0.04	[− 2.76, 2.28]	-0.44	[− 2.71, 1.48]	0.24	[− 40.9, 8.76]	2.70	[− 130.11, 158.75]
Intercept (cortisol recovery)	γ40	-2.97*	[− 6.75, − 1.1]	-2.38*	[− 4.62, − 0.70]	-1.67	[− 21.66, 2.04]	-0.91	[− 119.92, 97.12]
Intercept (logv)	ω	8.11*	[7.87, 8.35]	3.03*	[2.74, 3.32]	8.19*	[7.96, 8.42]	1.20*	[0.78, 1.61]
cortisol reactivity on alpha	γ31	0.00	[− 0.01, 0.01]	0.01	[− 0.02, 0.03]	0.00	[− 0.02, 0.1]	-0.03	[− 1.63, 1.34]
cortisol recovery on alpha	γ41	0.01*	[0.00, 0.02]	0.03*	[0.01, 0.05]	0.00	[− 0.01, 0.05]	0.01	[− 1, 1.22]
cortisol reactivity on phi	γ32	0.71*	[0.01, 2.07]	0.50	[− 0.11, 1.69]	0.19	[− 0.48, 1.93]	0.18	[− 1.86, 3.3]
cortisol recovery on phi	γ42	0.51*	[0.03, 1.64]	0.45	[− 0.06, 1.34]	0.32	[− 0.22, 1.89]	-0.19	[− 2.57, 1.46]
Var. (alpha)	τ00	398.25*	[114.14, 1013.91]	15.63*	[7.53, 26.28]	512.08*	[0.60, 1481.86]	1.02*	[0.00, 2.76]
Var. (beta)	τ11	12.84*	[0.92, 34.18]	0.02*	[0.00, 0.12]	16.47*	[0.84, 49.06]	0.01*	[0.00, 0.03]
Var. (phi)	τ22	0.05*	[0.01, 0.14]	0.08*	[0.01, 0.18]	0.08*	[0.01, 0.20]	0.05*	[0.00, 0.15]
Var. (logv)	τ55	0.58*	[0.26, 0.97]	1.07*	[0.68, 1.64]	0.40*	[0.14, 0.70]	2.59*	[1.77, 3.87]
Res. Var. (cortisol reactivity)	τ33	0.05*	[0.01, 0.09]	0.06*	[0.02, 0.10]	0.07*	[0.03, 0.11]	0.07*	[0.02, 0.1]
Res. Var. (cortisol recovery)	τ44	0.02*	[0.00, 0.05]	0.04*	[0.01, 0.07]	0.04*	[0.01, 0.07]	0.06*	[0.02, 0.09]

Note: * $p < 0.05$, RDSEM = Residual Dynamic Structural Equation Modeling, SE = Sleep Efficiency, TST = Total Sleep Time, CI= Confidence Interval.

approximately 30–45 min after waking in the morning and declining thereafter, reaching their nadir at midnight (Clow et al., 2010; Pruessner et al., 1997). The extent to which cortisol declines throughout the day, or the diurnal cortisol slope, may be indicative of an intact HPA axis negative feedback loop and is assumed to represent the ability to disengage and recover at the end of the day from stressful events (Dmitrieva et al., 2013; Kumari et al., 2010; Miller et al., 2009). Numerous studies have found that poor sleep was associated with a flattened cortisol diurnal slope and smaller decreases (Abell et al., 2016; Rotenberg et al., 2012; Zeiders et al., 2011), reflecting an impaired

negative feedback function in the HPA axis, which may further hinder recovery from acute stress.

4.2. Association between daily variations in sleep and cortisol stress response

Unlike mean sleep, sleep variability captures the regularity of sleep from day to day (Nicholson et al., 2022). Daily variation in sleep is increasingly considered an essential feature of sleep health, beyond mean sleep (Bei et al., 2016; Chaput and Shiau, 2019; Chaput et al., 2020). Greater sleep variability is linked to worse health outcomes (Becker et al., 2017; Bei et al., 2016; Chaput et al., 2020). To the best of our knowledge, this is the first study to expand on existing studies to highlight the effect of daily variations in sleep on the cortisol stress response. Our results showed that less variability in objective sleep duration was associated with greater stress reactivity (study 2) and recovery (studies 1 and 2), suggesting that individuals with stable sleep duration had more efficient cortisol stress patterns, particularly stress recovery.

Recovery from stress is a process of self-regulation (Beckmann and Kellmann, 2004). The stability of sleep duration facilitates stress recovery, possibly because it enhances self-regulation through self-control (i.e., individuals with stable sleep often have to resist temptations and impulses to sleep at a fixed time) or as a behavioral indication of individual differences in self-regulatory capacity (i.e., highly self-regulated individuals may be more effective in sustaining a stable sleep practice) (Barber and Munz, 2011; Barber et al., 2009; Hagger, 2009). An alternative explanation is the physiological adaptation to a circadian rhythm. Greater variability in sleep duration increases an individual's risk of circadian misalignment (e.g., wakefulness occurs when the internal circadian clock is facilitating sleep, and/or sleep occurs when the internal clock is facilitating wakefulness). Circadian misalignment has been linked to adverse changes in stress regulation, cognitive performance, and mood (Baron and Reid, 2014; Chellappa et al., 2018). Moreover, repeated activation of the allostatic process may be linked to greater variability in sleep duration and less stress recovery. Allostasis is a process in which a physiological system maintains stability under changing demands, and it is pivotal in maintaining homeostasis (McEwen, 1998). Highly variable sleep duration requires the system to adapt to changing demands, which, if frequent, could cause the dysfunction of the HPA axis (Bei et al., 2017). Indeed, previous studies showed that individuals with greater sleep duration variability exhibited consistently lower recovery of the basal HPA axis (flatter diurnal slopes) (Bei et al., 2017; Van Lenten and Doane, 2016).

We found that the relationship between daily variation in sleep and cortisol stress response involved sleep duration but not sleep efficiency. This is consistent with earlier studies which showed that greater variability in sleep duration, but not in sleep efficiency, was associated with greater perceived stress (Veeramachaneni et al., 2019). Furthermore, after controlling for mean sleep, greater variability in sleep duration, rather than sleep efficiency, was associated with increased odds of developing stress-related disease (e.g., breathing, neurological, and gastrointestinal problems, as well as depression and pain) (Slavish et al., 2019). These findings may suggest that stability of sleep duration is more relevant from a health perspective than that of sleep efficiency. More research is needed to validate these results before definitive conclusions can be reached concerning the relative importance of variability in sleep duration versus in sleep efficiency. On the other hand, sleep efficiency provides a more precise measure of sleep deficiencies induced by internal cognitive or bio-behavioral processes than does sleep duration, which is subject to restrictions by daily routines (Forner-Cordero et al., 2018). These may lead to a relatively narrow range of variability in sleep efficiency, which may contribute to the null findings. Indeed, sleep efficiency is relatively stable across multiple years, whereas sleep duration is more labile (Knutson et al., 2007). Concordant with these, our two studies consistently showed sleep duration to be more varied than sleep efficiency over the 7-day study period (Tables 2 and 4).

4.3. Differential effects of sleep on cortisol reactivity and recovery

In agreement with previous studies, we found no significant correlation between cortisol reactivity and recovery. Cortisol reactivity and recovery appear to be separate and independent dimensions of the stress response (Ji et al., 2016; Ramsay and Lewis, 2003; Schuetze et al., 2008; Tackett et al., 2014). Moreover, using different acute stress paradigms and different samples, our two studies showed that better sleep predicted higher cortisol recovery. There was no correlation between sleep and cortisol reactivity, except for the variability in sleep duration associated with cortisol reactivity in study 2. Previous studies found that poor sleep is associated with either higher or lower stress reactivity, with lower cardiovascular recovery, and with delayed mood recovery from stressors (Brindle and Conklin, 2012; Capaldi et al., 2005; Hamilton et al., 2008; Massar et al., 2017; Mezick et al., 2014; Raikkonen et al., 2010). These studies indicate that good sleepers do not have a lower reactivity to acute stressors compared to poor sleepers; what distinguishes sleepers is the promptness of recovery. Given the high incidence of chronic cardiovascular disease, burnout, and other stress-related diseases, recovery from stressors is crucial and was identified as a pivotal indicator for long-term health (Chida and Steptoe, 2010; Freeman, 1939; Lauer and Froelicher, 2002). Some authors even maintain that failure to recover from stress responses may be a more important predictor of long-term health than the initial magnitude of reactivity (Hamilton et al., 2008; Linden et al., 1997; Roy et al., 1998). Our research offers a possible mechanism for the link between poor sleep and stress-related diseases. Also, this highlights the importance of distinguishing between stress reactivity and recovery in sleep studies.

4.4. Association between objective-subjective sleep and cortisol stress response

Actigraphy and sleep diaries are widely regarded as complementary but somewhat distinct measurements (Bauer and Blunden, 2008; Tryon, 2004; Williams et al., 2018). Actigraphy captures quiescence of behavior, whereas a sleep diary captures a person's perceptual awareness of sleep. Using different acute stress paradigms and different samples, both studies revealed that it is variables detected by actigraphy, not by a sleep diary, the ones that affect HPA axis responses to acute stressors, consistently with previous studies (Wright et al., 2007). Greater variability in sleep duration detected by actigraphy (but not by a sleep diary) was related to higher perceived stress (Veeramachaneni et al., 2019). One potential explanation lies in that sleep diaries are more biased than actigraphy in the estimation of sleep variables (Bauer and Blunden, 2008; Williams et al., 2018). Specifically, individuals may underestimate or overestimate sleep in diaries owing to personality traits and physiological-psychological factors (e.g., depression) (Jackowska et al., 2011; Rotenberg et al., 2000; Tsuchiyama et al., 2003). For example, a person with high levels of work-related stress may underestimate subjective sleep efficiency (Jackowska et al., 2011), confounding the relationship between sleep and stress responses. Our results suggest that objective sleep is a better predictor of the HPA axis response to acute stressors compared to subjective sleep. Moreover, previous studies have shown that poor subjective sleep is correlated with slower affective recovery from negative events (Hamilton et al., 2008) and an increased risk of stress-related disorders such as depression and anxiety (Do et al., 2013; Ojio et al., 2016). This may imply an essential role of subjective sleep on affective responses to acute stressors, which needs to be explored in future studies.

4.5. Limitations

The correlational research design prevented drawing causal

conclusions regarding the relationship between sleep and cortisol response to acute stressors. We also did not measure the levels of background stress and basal function of the HPA axis, both of which are related to sleep and the cortisol stress response (Ren et al., 2022; Sandner et al., 2020; Slavish et al., 2021; Xin et al., 2020), and the levels of background stress and basal function of the HPA axis may have confounded our results. Nevertheless, such relationships could be investigated using different acute stress paradigms and samples. Moreover, although actigraphy is a widely accepted, valid and objective measure of sleep, it does not detect wake time when no movement occurs (i.e., it may overestimate sleep duration) (Blood et al., 1997; Lockley et al., 1999; Tryon, 2004). This study focused on the mean and daily variation of sleep, which are challenging to measure using polysomnography. Future studies using polysomnography can examine the relationship between specific sleep stages (e.g., rapid eye movement or slow-wave sleep) and cortisol stress responses. Lastly, the relatively small number of participants is a limitation of the current research.

5. Conclusions

The current study explored the influence of means and daily variations in sleep on the reactivity and recovery of the cortisol stress response. We found that a higher mean of objective sleep efficiency, a longer mean objective sleep duration, and fewer daily variations in objective sleep duration were related to greater cortisol recovery. This study paints a more comprehensive picture of sleep and the stress response, which in turn provides a new perspective on the key role of sleep in the development of stress-related psychopathologies.

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CRediT authorship contribution statement

Xiaolin Zhao: Conceptualization, Methodology, Formal analysis, Investigation, Writing – original draft preparation, Writing – review & editing. **Weiyu Hu:** Conceptualization, Investigation, Formal analysis. **Yadong Liu:** Investigation, Formal analysis. **Kaige Guo:** Formal analysis. **Yuan Liu:** Formal analysis, Methodology, Writing – review & editing. **Juan Yang:** Conceptualization, Writing – review & editing, Supervision, Funding acquisition.

Conflict of interest

All authors have no conflict of interest relevant to this article.

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Disclosure statement

None declared.

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