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Sleep quality but not sleep quantity effects on cortisol responses to acute psychosocial stress

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Abstract

Given the well-documented deleterious health effects, poor sleep has become a serious public health concern and increasing efforts are directed towards understanding underlying pathways. One potential mechanism may be stress and its biological correlates; however, studies investigating the effects of poor sleep on a body's capacity to deal with challenges are lacking. The current study thus aimed at testing the effects of sleep quality and sleep quantity on cortisol responses to acute psychosocial stress. A total of 73 college-aged adults (44 females) were investigated. Self-reported sleep behavior was assessed via the Pittsburgh Sleep Quality Index and salivary cortisol responses to the Trier Social Stress Test (TSST) were measured. In terms of sleep quality, we found a significant three-way interaction, such that relative to bad sleep quality, men who reported fairly good or very good sleep quality showed blunted or exaggerated cortisol responses, respectively, while women's stress responses were less dependent on their self-reported sleep quality. Contrarily, average sleep duration did not appear to impact cortisol stress responses. Lastly, participants who reported daytime dysfunctions (i.e., having trouble staying awake or keeping up enthusiasm) also showed a trend to blunted cortisol stress responses compared to participants who did not experience these types of daytime dysfunctions. Overall, the current study suggests gender-specific stress reactivity dysfunctions as one mechanism linking poor sleep with detrimental physical health outcomes. Furthermore, the observed differential sleep effects may indicate that while the body may be unable to maintain normal HPA functioning in an acute psychosocial stress situation after falling prey to low sleep quality, it may retain capacities to deal with challenges during extended times of sleep deprivation.

Key terms

sleep quality; sleep quantity; daytime dysfunctions; psychosocial stress; cortisol; Trier Social Stress Test

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Declaration of Interest

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Introduction

Sleep is a naturally-occurring state of decreased consciousness as well as reduced sensory and motor activity (Hays and Stewart, 1992). Humans engage in sleep for an average of seven to eight hours per 24-hour period (Basner, Fomberstein, 2007). Poor sleep behavior occurring over a limited time period induces changes in mood as well as diminishes alertness and cognitive performance. However, the effects of poor sleep behavior are cumulative (Veasey, Raymond, 2002). More specifically, chronic poor sleep and chronic insomnia have been linked to elevated risks for negative physiological and mental health outcomes (Spiegel, Leproult and Van Cauter, 1999; Taylor, Lichstein and Durrence, 2003). Given the deleterious health effects of both chronic low sleep quality and chronic sleep restriction, it is crucial to increase our understanding of the pathways underlying these associations.

Several lines of evidence suggest stress and its related physiological changes as a possible link between poor sleep and detrimental health outcomes. Specifically, findings from both animal and human studies point towards bidirectional effects between sleep and the hypothalamic-pituitary-adrenal (HPA) axis (Buckley and Schatzberg, 2005; Steiger, 2002). HPA axis activity is thereby typically assessed by the circadian rhythm of its end hormone, cortisol (Wust, Wolf, 2000), and several studies exist linking variation in diurnal cortisol secretion patterns to sleep behavior (Backhaus, Junghanns and Hohagen, 2004; Fries, Dettenborn and Kirschbaum, 2009; Kumari, Badrick, 2009; Raikkonen, Matthews, 2010; Wust, Wolf, 2000). HPA axis dysfunctions, in turn, have consistently been shown to impact physiological and mental health (Chrousos and Gold, 1992), thus presenting a pathway contributing to the effects of poor sleep on health (Minkel, Moreta, 2014).

Importantly, HPA axis dysfunctions are thought to result from wear and tear due to repeated or chronic activation of the HPA axis (McEwen, 1998). As such, changes in acute HPA axis *re*activity patterns may precede HPA axis activity dysfunctions and serve as an early warning sign for elevated health risks. However, human studies testing the associations between sleep and HPA axis reactivity in the context of acute stress are scarce and, to date, we are aware of only three studies. One demonstrated that poor-quality sleepers show exacerbated cortisol responses to a physiological stressor in the form of a cold-pressor task (Goodin, Smith, 2012). The second and third study used a psychosocial stressor in children and adolescents, linking low sleep efficiency to increased cortisol reactivity (Pesonen, Kajantie, 2012; Raikkonen, Matthews, 2010). Thus, it is unknown whether sleep effects on stress responses in adults generalize from physiological stressors to psychosocial stressors, or whether associations between sleep and psychosocial stress responses observed in children and adolescents generalize to adults.

Hence, the current study aimed at investigating effects of sleep habits on acute responses to psychosocial stress in healthy adults. Furthermore, the findings above do not allow any inferences with regard to which sleep dimension may interfere the most with cortisol responses to acute stress. In more detail, measures included ‘sleep quality’ assessed via self-report over the last month (Goodin, Smith, 2012), ‘sleep efficiency’ and ‘sleep duration’ assessed via actigraphy over 7 days (Raikkonen, Matthews, 2010), and ‘sleep problems,’

such as disorders of initiating sleep and excessive daytime somnolence (Pesonen, Kajantie, 2012). The current study thus further aimed at exploring the effects of various sleep dimensions, specifically sleep duration, sleep quality, and daytime dysfunctions on cortisol responses to acute psychosocial stress. Lastly, given gender-dependent differences in sleep behavior (Burgard, 2010) as well as sleep-stress associations (Pesonen, Kajantie, 2012), gender was considered a moderator of links between sleep habits and cortisol stress responses.

Methods

Participants

A total of 85 undergraduates and community members were assessed. Complete data were available for analyses from 73 participants (44 females; 19.69±2.36 yrs.; BMI=23.32±4.08 kg/m^2). Participants thereby had to be excluded from analyses due to incomplete descriptive information (n=1), missing Pittsburgh Sleep Quality Index (PSQI) data (n=2), incomplete cortisol data (n=5), missing PSQI and cortisol data (n=1), or abnormally high cortisol values (> 2 SD above the mean; n=3) indicating potential illness or chronic stress.

Participants were recruited via a student subject pool, flyers around campus, and via newspaper advertisements geared toward general community members. Exclusion criteria were habitual smoking, acute diseases (e.g., a flu), current or a history of psychiatric or physiological chronic diseases (e.g., depression, atopic diseases, cardiovascular diseases), medication indicating psychiatric or physiological chronic diseases, or medication interfering with sleep or stress hormone responses (e.g., oral contraceptives) (Rohleder, Schommer, 2001). The local Institutional Review Board approved the protocol, and participants were compensated either monetarily or with study credits.

Procedure

After a phone screening, eligible participants were scheduled to visit the laboratory on a weekday afternoon to minimize inter-individual differences in basal cortisol levels (Kirschbaum and Hellhammmer, 2007). The study protocol was explained and informed consent was obtained. During a subsequent resting period of 30 minutes allowing participants to acclimate and recover from potential earlier stressors, participants completed self-report demographic and health questionnaires as well as the PSQI. Prior to exposure to a psychosocial stress test protocol (Trier Social Stress Test: TSST), a pre-stress baseline saliva sample was taken using the Salivette collection device, and participants were lead to a separate testing room to complete the TSST. Upon task completion, participants returned to their study rooms and provided a second saliva sample (1min post-TSST). Additional saliva samples were taken 10, 20, 30, and 45 minutes post-TSST. During this time, participants were debriefed and after collection of the last saliva sample, participants received either money or study credit and were free to leave.

Measures and Apparatus

Self-report assessments

Sleep behavior: The 19-item Pittsburgh Sleep Quality Index (PSQI) was used to measure sleep behavior over the past month (Buysse, Reynolds III, 1989). An overall sleep quality score is computed (range 0–21) by summing seven subscale scores (range 0–3), each representing responses or combinations of responses to items measuring various dimensions of sleep. Higher PSQI values thereby indicate worse sleep quality. To address the study aim concerning potential sleep dimension-specific effects on cortisol stress responses, subscales aligning with components identified by Cole et al. (2006) as a result of a principal component analysis of the PSQI were assessed (Cole, Motivala, 2006). These include 'average hours of sleep' (During the past month, how many hours of actual sleep did you get at night?), 'sleep quality' (During the past month, how would you rate your sleep quality overall?) and 'daytime dysfunction' (During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity; During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?).

Stress manipulation

Trier Social Stress Test (TSST): Stress was induced by the TSST, a validated and widely-used psychosocial laboratory stress protocol consisting of a five minute preparation period, a five minute speech in front of a two-member panel, and a five minute mental arithmetic task (Kirschbaum, Pirke and Hellhammer, 1993).

Physiological assessment

Cortisol: Saliva samples were stored at −20C until study completion, centrifuged for 15 minutes at 1800×g, and tested for cortisol levels using a commercially available chemiluminescence assay (IBL, Toronto, Canada). Inter- and intra-assay variability were below 8%.

Analytical Plan

First, chi^2 and *t*-tests were computed to assess gender-dependent differences in any of the sleep and cortisol variables. All subsequent analyses controlled for time of day the TSST was administered as well as for BMI due to reported cortisol effects (Champaneri, Xu, 2013). A repeated-measures ANCOVA was computed to test whether the TSST was successful at inducing a cortisol stress response in participants.

To assess how the various sleep dimensions are associated with cortisol stress responses, repeated-measures ANCOVAs examining effects of the overall PSQI scores as well as the above specified PSQI subscales on cortisol stress responses were computed. Furthermore, univariate ANCOVAs examined the effects of sleep dimensions on baseline, pre-TSST cortisol levels. Gender was included as a second between-subject factor in all analyses. The statistic software package SPSS v21 was used for all analyses and p-values < .05 were considered indicative of a significant effect. Where indicated, Greenhouse-Geisser corrected values are reported.

Results

Preliminary Results

As frequencies across the answer categories in each of the subscales were heavily skewed, responses to each subscale were re-categorized either according to the literature or to combine categories with too-low frequencies. More specifically, the overall PSQI score was dichotomized with a score of 5 or lower indicating good sleep (n=38, 21 women), and scores above 5 indicating poor sleep (n=35, 23 women). Furthermore, PSQI item 4 asking "During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)," was categorized in line with the current literature (Prather, Epel, 2013; Wright, Valdimarsdottir, 2007) into 'below six hours' (n=12), 'from six until but not including seven hours' (n=17), and 'seven hours and above' (n=44). For all other items or subscales assessed in the current study, see Table 1 for more details on response frequencies and descriptive statistics. Importantly, frequency distributions for all sleep variables were gender-independent (all $p \geq .46$) and age-independent (all $p \geq .22$).

As a manipulation check, we next examined cortisol stress responses via repeated-measures ANCOVA (covariates: BMI, TSST start time). Independent of gender (main effect: $F(1, 69)=.51$, $p=.48$, $\eta_p^2=.007$; gender*cortisol: $F(5, 345)=2.15$, $p=.11$, $\eta_p^2=.03$), cortisol levels changed significantly over time, $F(5, 345)=3.43$, $p=.03$, $\eta_p^2=.05$, with peak levels occurring on average 10 minutes post-TSST followed by a return to baseline by 45 minutes post-TSST, confirming that the TSST was successful in inducing a cortisol stress response.

Sleep effects on cortisol stress responses

Testing for associations between the dichotomized global PSQI score and cortisol stress responses revealed that good sleepers and poor sleepers did not differ in their cortisol responses to the TSST, $F(5, 335)=0.55$, $p=.60$, $\eta_p^2=.008$, nor in their overall cortisol levels, $F(1, 67)=0.12$, $p=.73$, $\eta_p^2=.002$. This was true for both genders (all $p \geq .42$). Similarly, when assessing average hours of sleep, no differences in overall cortisol levels, $F(2, 65)=0.12$, $p=.89$, $\eta_p^2=.004$, or in cortisol stress responses, $F(10, 325)=0.71$, $p=.60$, $\eta_p^2=.02$, were observed among the three groups for either gender (all $p \geq .45$; see Fig. 1).

Contrarily, examining effects of self-reported sleep quality on cortisol stress responses, a gender-dependent sleep quality main effect, $F(2, 65)=4.07$, $p=.022$, $\eta_p^2=.11$, as well as a three-way interaction was found, $F(10, 325)=2.71$, $p=.025$, $\eta_p^2=.08$. In more detail, relative to stress responses linked to fairly bad / bad sleep quality, men reporting high sleep quality showed the strongest cortisol stress responses, while those reporting fairly good sleep quality showed relatively weak responses. Contrarily, women's stress responses appeared less dependent on their self-reported sleep quality (see Table 2, Fig. 2).

When assessing the role of daytime dysfunctions, a two-way interaction between trouble staying awake and cortisol levels over time emerged. That is, independent of gender, those who had trouble staying awake during the day showed a trend for blunted cortisol stress responses, $F(5, 335)=2.67$, $p=.065$, $\eta_p^2=.04$ (see Table 2, Fig. 3a). Similarly, trouble keeping up enthusiasm was linked to weaker cortisol stress responses, $F(10, 325)=2.31$, $p=.052$, $\eta_p^2=.07$ (see Fig. 3b).

Sleep effects on pre-stress cortisol levels—To distinguish effects of sleep habits on acute stress responses from more chronic influences on basal cortisol levels, we additionally examined associations between sleep variables and pre-TSST cortisol samples via univariate ANCOVA. Neither dichotomized PSQI scores, nor average hours of sleep, sleep quality, or daytime dysfunctions were linked to baseline cortisol levels (all $p \geq .71$; gender interactions: all $p \geq .27$).

Discussion

The current study revealed differential effects of sleep dimensions on cortisol responses to acute psychosocial stress. While self-reported average sleep duration did not appear to affect cortisol stress responses, perceived sleep quality impacted cortisol stress responses in a gender-dependent manner. Furthermore, participants who reported having trouble staying awake or keeping up enthusiasm showed blunted cortisol responses compared to participants who did not experience such daytime dysfunctions. Of note, overall sleep behavior assessed by poor and good sleep via PSQI was not associated with cortisol stress responses, supporting the current approach of differentiating between sleep dimensions. In the following discussion, each dimension and its relation to HPA axis reactivity will be discussed.

The self-reported average of hours slept per night over the last month was neither related to cortisol stress responses, nor was it linked to baseline cortisol values. As such, our findings extend previous research on children undergoing the TSST-C that failed to find a link between sleep duration assessed via seven days of actigraphy and stress responses, to a young adult population and self-reported average sleep duration over the last month (Raikkonen, Matthews, 2010). It should be pointed out that research focusing on diurnal cortisol instead of cortisol reactivity in adults suggest a threshold for sleep deprivation effects on HPA axis output (e.g., (Leproult, Copinschi, 1997; Wust, Wolf, 2000). Hence, as participants in the current study obtained an average of 6.9 hours of sleep per night, their HPA axis's ability to respond to challenge may not or not yet be affected by sleep loss (Chrousos and Gold, 1992; Lund, Reider, 2010; McEwen, 1998).

Contrary to self-reported sleep duration, we observed associations between self-reported sleep quality and cortisol stress responses. This finding expands previous findings on physiological stress effects to a psychosocial stressor (Goodin, Smith, 2012), suggesting that sleep quality affects the body's HPA axis independently of type of stressor. Importantly, in the current study, this effect differed for men and women, such that relative to fairly good sleep, men who reported poor-quality sleep showed exaggerated cortisol stress responses. Contrarily, women reporting poor-quality sleep showed slightly reduced responses. As such, the current findings in men are in line with those reported in the study on children mentioned above by Raikkonen and colleagues (Raikkonen, Matthews, 2010), as well as with previous findings demonstrating that women who were poor sleepers as measured by nighttime awakenings via actigraphy showed blunted response to an acute cognitive stressor, i.e., the Stroop test (Wright, Valdimarsdottir, 2007). It should be noted that women's cortisol responses generally appeared less dependent on sleep quality as well as less strong overall, compared to men's responses. This suggests that formerly reported effects of sex hormones

on stress responsivity may outweigh potential effects of perceived sleep quality (Kirschbaum, Wüst and Hellhammer, 1992). Lastly, although not a frequent questionnaire response, men who reported very good sleep quality showed exaggerated cortisol stress responses. One potential pathway contributing to this pattern of sleep effects on stress responsivity may be differences in emotion regulation strategies employed by men and women. Between the two predominant types of emotion regulation strategies, reappraisal and suppression, suppression is employed more frequently by men than by women (Gross and John, 2003). Interestingly, using suppression as an emotion regulation strategy has been linked to exaggerated cortisol responses (Lam, Dickerson, 2009), while using a more accepting emotion approaching way of emotion regulation employed before sleep has been associated with better sleep quality than a more top-down analytical evaluative approach (Vandekerckhove, Kestemont, 2012). Therefore, it is possible that differences in emotion regulation strategy use may influence sleep quality or report thereof and thus contribute to the gender-dependent relationship between sleep quality and cortisol.

Lastly, we assessed the effects of daytime dysfunctions experienced over the last month on cortisol stress responses. We found that those who reported trouble staying awake during the day or problems keeping up enough enthusiasm to get things done showed a trend toward lower cortisol responses to the TSST. Interestingly, daytime sleepiness has recently been reported to be associated with poor health outcomes in diabetes self-management (Chasens, Korytkowski, 2013). As such, our findings suggest that daytime dysfunctions may be another way in which poor sleep contributes to an organism's impaired ability to cope with challenges and eventually, to negative health outcomes.

Contrary to sleep quality, we found no gender-dependent differences in the link between daytime dysfunctions and acute stress measures. This may be due to differences in factors motivating men's and women's self-report behavior. When examining self-report measures of sleep quality, men state that they do not sleep as poorly as women (Vitiello, Larsen and Moe, 2004) though the majority of studies indicate that men have lower quality sleep than women (Ancoli-Israel, Cole, 2003). Thus, while men may not accurately self-report their sleep quality, it may be socially more acceptable for men to self-report daytime dysfunctions.

From a broader perspective, the current study observed differential stress effects for sleep quality and sleep quantity. When exposed to chronic sub-optimal sleep duration, the HPA axis appears to maintain its ability to perform appropriately in an acute psychosocial stress situation. On the other hand, perceptions of chronic poor sleep quality had immediate effects. It could be speculated that the body may retain compensatory capacities to deal with challenges during times of being taxed with the physiological effects of non-severe sleep deprivation. Self-reported poor sleep quality, however, may represent a more integrated measure capturing various aspects and consequences of poor sleep habits, and thus a circumstance in which an organism is no longer able to adequately respond to challenges.

Overall, the current study suggests psychosocial stress and its endocrine effects as one mechanism linking poor sleep quality and daytime dysfunctions with detrimental physical health outcomes. More specifically, both blunted and exaggerated cortisol reactivity have

been shown to be insalubrious (Andrews and Walker, 1999; Buske-Kirschbaum, von Auer, 2003) and as such may be one physiological link underlying the well-documented detrimental health effects of poor sleep (Spiegel, Leproult and Van Cauter, 1999; Taylor, Lichstein and Durrence, 2003). For example, blunted cortisol responses, such as seen in participants reporting high levels of daytime dysfunctions, may represent an increased risk for diseases associated with a lack of immune inhibition, including asthma (Buske-Kirschbaum, von Auer, 2003; Fagan, Scheff, 2001). Contrarily, men's exaggerated cortisol responses observed in the context of self-reported very good sleep quality may put them at risk for problems related to chronically elevated cortisol, such as insulin resistance and hypertension (Andrews and Walker, 1999).

The findings of the current study have to be interpreted in light of several limitations. First, the study was conducted on a sample dominated by university students, thus raising generalizability concerns. However, college students have been shown to exhibit erratic sleep schedules (Lund, Reider, 2010), making this population an interesting target group for sleep-related research. Secondly, without measures corroborating the effectiveness of the employed stress protocol, it is difficult to differentiate between blunted cortisol stress responses as an indicator of HPA dysfunction versus blunted cortisol stress responses being the result of lack of a stress experience in the first place. Hence, future studies should include self-report measures of stress and cardiovascular stress response measures. Lastly, we observed small to medium effects sizes for links between sleep quality dimension measures (self-reported sleep quality, daytime dysfunctions) and cortisol measures (Cohen, 1988), cautioning against over-interpretation of the findings. However, given the complexity of factors determining an individual's response to psychosocial stress exposure, as well as considering the differential pattern across sleep dimensions, these effect sizes support sleep quality as one important determinant of cortisol stress responsivity.

Conclusions

Results of the current study indicate that perceptions of sleep quality and daytime dysfunction have consequences for the body's ability to respond to challenges. As such, the current study implicates stress and the associated physiological changes in HPA axis reactivity as one gender-dependent pathway linking poor sleep with negative health outcomes. However, the lack of sleep duration effects suggests that the body may retain some degree of resilience. Future studies will have to investigate at what point the costs of counteracting sleep deprivation contribute to sleep quality-related HPA axis dysfunctions and thus negative health outcomes.

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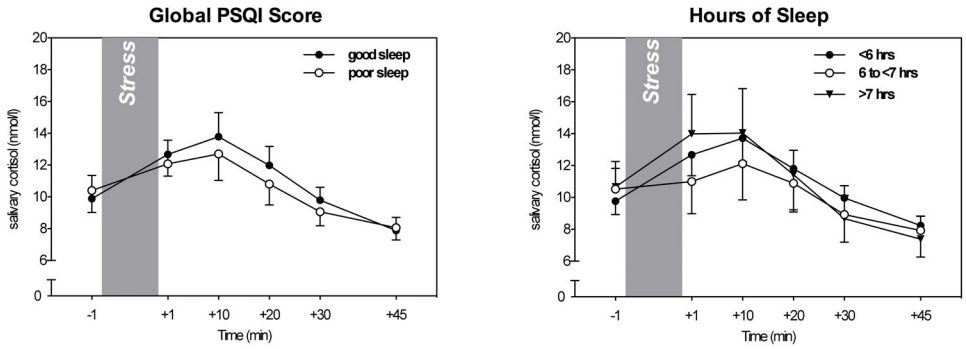

Figure 1.

Neither overall sleep assessed by global PSQI scores (left) nor hours of sleep (right) showed an effect on cortisol stress responses (mean, SE).

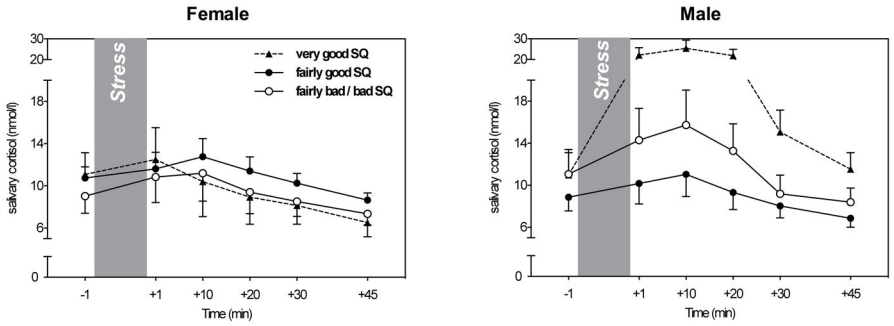

Figure 2.

The impact of sleep quality (SQ) on cortisol stress responses in females (left) and males (right).

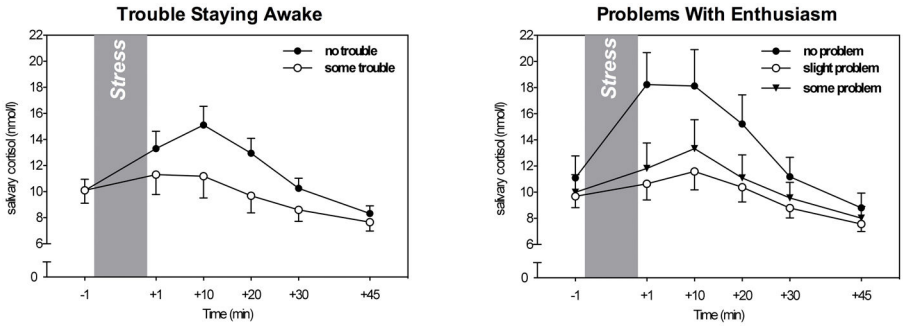

Figure 3.

Daytime dysfunctions: Trouble staying awake (left) as well as problems keeping up enthusiasm (right) were associated with blunted cortisol stress responses.

Table 1

Categorization of PSQI item scores by gender (mean and SD given for raw data).

Item Responses	**Males**	**Females**	**X^2**	**M**	**SD**
	Frequency				
	PSQI global score		.36	5.79	2.69
Good sleeper (≤5)	17	21			
Bad sleeper (>5)	12	23			
	Sleep Quality		.03	1.10	.67
Good sleep quality (0)	5	7			
Fairly good sleep quality (1)	17	26			
Bad sleep quality (2, 3)	7	11			
	Hours of Sleep		.61	6.88	1.09
7 hours or above (0)	16	28			
6 to 6.99 hours (1)	8	9			
0 to 5.99 hours (2, 3)	5	7			
	Trouble Staying Awake		.12	.56	.73
	Males	*Females*			
No trouble (0)	17	24			
At least some trouble (1, 2, 3)	12	20			
	Keep Up Enthusiasm		1.54	1.19	.76
No problem (0)	5	6			
A very slight problem (1)	18	23			
Somewhat of a problem (2, 3)	6	15			

Note. All X^2 were non-significant.

Table 2

Repeated-measures ANCOVAs testing gender-dependent sleep effects on cortisol stress responses (N=73).

	Good/bad sleep		Hours of sleep		Sleep quality		Staying awake		Enthusiasm	
	F	***p***	***F***	***p***	***F***	***p***	***F***	***p***	***F***	***p***
Between-subjects effects										
Gender	0.40	.53	0.69	.41	3.93	.052	0.75	.39	0.89	.35
IV	0.12	.73	0.12	.89	2.06	.14	1.82	.18	2.09	.13
IV * gender	0.01	.75	0.81	.45	4.07	.022*	1.30	.26	1.74	.18
Within-subjects effects										
Cortisol	3.47	.029*	3.16	.040*	4.31	.011*	2.82	.055†	4.73	.007*
Cortisol * gender	1.81	.16	1.52	.22	5.25	.004*	2.34	.092†	2.73	.060†
Cortisol * IV	0.55	.60	0.71	.60	1.99	.09†	2.67	.065†	2.31	.052†
Cortisol * IV * gender	0.90	.42	0.54	.73	2.71	.025*	0.55	.60	1.12	.35

Note.

† $p \leq .10$

* $p < .05$;

IV=independent variable