Sleep Quality but Not Sleep Quantity Effects on Cortisol Responses to Acute Psychosocial Stress

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ABSTRACT

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A thesis presented to the Department of Psychology

Graduate School of Arts and Sciences
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Waltham, Massachusetts

By Sarah Bassett

Objective: Poor sleep is a widespread problem and given the well-documented deleterious health effects, has become a serious public health concern. As such, increasing efforts are directed towards understanding the underlying mechanisms. One potential mechanism may be cortisol dysfunctions, 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.

Methods: N=73 adults (44 females, 19.7±2.4 yrs.) self-reported sleep behavior, and underwent the Trier Social Stress Test (TSST). Saliva samples for cortisol assessment were taken immediately before the TSST as well as repeatedly afterwards.

Results: Average sleep duration did not appear to impact cortisol stress responses (\(p=.66\)). Contrarily, men who reported poor sleep quality showed exaggerated cortisol responses, while women exhibited blunted responses (\(F=3.17\),
Lastly, participants who reported having trouble staying awake or keeping up enthusiasm also showed blunted cortisol responses compared to participants who did not experience such daytime dysfunctions ($F=2.72, p=.06; F=1.99, p=.09$, respectively).

**Conclusions:** Overall, the current study suggests stress reactivity dysfunctions as one mechanism linking poor sleep with detrimental physical health outcomes in a gender-dependent manner. Furthermore, it can be speculated that the differential sleep effects may indicate that while the body is 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 times of sleep deprivation.
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Introduction

Sleep is a naturally occurring state of decreased consciousness as well as reduced sensory and motor activity (1,2). Humans engage in sleep for an average of seven to eight hours per 24-hour period (3). Poor acute sleep behavior (poor sleep quality and low sleep quantity) 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, getting worse as one goes for a longer period of time with restricted sleep (4-6). Unfortunately, chronic sleep restriction is a widespread problem and has become a serious public health concern. For example, individuals suffering from chronic insomnia have significantly higher rates of cardiovascular disease, depression, gastrointestinal problems, and chronic pain than their better-sleeping counterparts (7,8). Given the deleterious health effects of both chronic low sleep quality and sleep quantity, it is crucial to increase our understanding of the pathways underlying these associations. One potential pathway in this regard is stress with its related physiological changes.

Sleep quality versus sleep quantity

The two major facets of sleep behavior are sleep quantity and sleep quality. Sleep quality can be captured by a combination of factors including sleep efficiency (time asleep relative to time in bed), number of nighttime awakenings, daytime fatigue, and personal satisfaction with sleep. Sleep quantity refers to the combination of sleep
duration, sleep latency, and number of nighttime awakenings (9). Importantly, sleep quality and sleep quantity are differentially associated with health problems. For example, low sleep duration has been associated with obesity and type II diabetes, whereas poor sleep quality has been associated with increased susceptibility to pain as well as with low life satisfaction, anger, and depression (9-11).

The health-relevance of self-reported perceived sleep behavior is emphasized by its use as a criterion in diagnosing insomnia, a sleep quantity and quality-related disease (12-14). Self-reported sleep behavior can thereby be assessed in multiple ways (15). A post-sleep inventory allows the individual to draw upon recent memories regarding their night of sleep. However, questionnaires assessing average behavior over longer time-periods are better suited to capture sleep habits, including those indicative of sleep behavior problems. One example is the Pittsburgh Sleep Quality Index (PSQI, 15), which assesses sleep behavior over the past month. The latter will be the focus of the current study.

**Stress – a potential link between sleep and health**

Several lines of evidence exist suggesting stress as a possible link between poor sleep and detrimental health outcomes. For example, sleep loss has been implicated as a physiological stressor, indicated by increased cortisol levels the subsequent evening (16). It has been suggested that due to this dysregulation, chronic poor sleep can increase the risk for diseases such as cardiovascular disease and diabetes (17). Stress, in turn, has been found to be a catalyst for insomnia (18), suggesting a vicious cycle between poor sleep and stress.
**Stress systems**

Stress systems, which include the hypothalamus-pituitary-adrenal (HPA) axis with its end hormone cortisol and the sympathetic-adrenal-medullary (SAM) system with its end hormones norepinephrine and epinephrine, work in a well-orchestrated and self-regulatory manner, inhibiting non-essential behaviors and increasing physiological and psychological behaviors helpful when undergoing short-term, acute stress (19). However, repeated activation of stress systems can lead to wear and tear and eventually to dysfunctions with clinical relevance (McEwen, 1998). For example, basal stress system over-activity has been linked to hypervigilance and insomnia, anxiety, immunosuppression, and melancholic depression. On the other hand, basal stress system hypoactivity has been observed in the context of prolonged exposure to a significant stressor and has been linked to health relevant changes such as a greater risk of inflammation (19-21) as well as atypical depression, which differs from melancholic depression in that it entails hypersomnia (20, 22).

**Sleep and acute stress**

Given that the dysfunctions in basal stress system activity described above are thought to be in large parts consequences of dysfunctions in acute stress system reactivity, it is important to understand the effects of sleep on stress reactivity. Looking at the effects of poor sleep habits as opposed to recent sleep behavior will allow linkage of decrements in sleep quality and quantity to health-relevant acute differences in stress responses, thus providing insights into mechanisms underlying negative health effects of poor sleep.
To date, we are aware of only two studies with human participants that have examined the effects of sleep habits on acute biological stress responses through the employment of stress manipulations. One study found that adults reporting poor sleep quality (assessed by PSQI) not only showed higher cortisol levels pre-stress, but also exaggerated cortisol reactivity and stronger pain responses when undergoing an acute physiological stressor in the form of a cold-pressor task (11). The second study found that children who displayed low sleep efficiency (assessed by actigraphy), showed increased cortisol reactivity when undergoing a psychosocial stressor in the form of the Trier Social Stress Test for Children (TSST-C) (23).

While the first study demonstrated that poor quality sleepers show exacerbated cortisol response to a physiological stressor, it is not yet known whether the same holds true for a psychosocial stressor. While the second study used a psychosocial stressor, it is unclear whether its findings for adolescents will generalize to adults with poor sleep behavior. Hence, the current study aimed at investigating effects of sleep habits on acute responses to psychosocial stress in healthy adults.

Gender as a moderator of the link between sleep and stress

When examining associations between sleep habits and acute stress responses, it is important to consider gender as a potential moderator. For example, according to the American Time Use Surveys, although men sleep for a shorter period of time than women, they have overall higher sleep quality and less fatigue and report lower rates of insomnia (24). With regard to stress, men typically show greater salivary cortisol reactivity than do women; however, this difference vanishes when menstrual cycle phase is taken into account (25-29).
Hypotheses

Assuming that the above findings in adolescents will generalize to young adults and that the findings for a physiological stressor will generalize to a psychosocial stressor, we hypothesized that lower sleep quality will be associated with stronger cortisol stress responses. In line with studies reporting differential effects on health outcomes, we further hypothesized to find differential effects of sleep quantity and sleep quality on cortisol stress responses. Lastly, given the paucity in studies assessing the role of gender in the link between sleep and stress, analyses including gender are considered exploratory.
Methods

Participants

A total of 85 undergraduates and community members were assessed (53 females, 19.89±2.78 yrs.). However, complete data were available for analyses from 73 participants (44 females; 19.69±2.36 yrs.; BMI = 23.34±4.06 kg/m²). One participant was excluded from analyses due to incomplete descriptive information, three due to missing PSQI data, four due to incomplete cortisol data and one due to both, missing PSQI and missing cortisol data. Lastly, five participants with abnormally high cortisol values indicating potential illness or chronic stress were excluded as well.

Participants were recruited via a student subject pool, flyers around campus, and via newspaper advertisements geared toward general community members. Participants were excluded if they reported current or a history of psychiatric disease or were taking any psychiatric medication or medication that would interfere with their stress hormone response (including oral contraceptives). The local Institutional Review Board approved the protocols 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. The study protocol was explained and informed
consent was obtained. During a subsequent resting period (30min), 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 room 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. Multiple subscales measure either sleep quantity or quality. Subscales relevant to the current study measuring sleep quality include ‘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?), and ‘sleep quality’ (During the past month, how would you rate your sleep quality overall?), while the subscale ‘sleep duration’ (During the past month, how many hours of actual sleep did you get at night?) measures sleep quantity. Subscale responses, or combinations thereof, are categorized into scores between 0 and 3 (15). In addition to the PSQI sleep quantity question, duration was computed from self-
reported bedtime and wake-up times.

Stress manipulation.

**Trier Social Stress Test (TSST):** Stress was induced by the TSST, a validated and widely used psychosocial laboratory stress protocol (30). In more detail, participants were asked to give a speech explaining why they are qualified for their dream job, using only qualifications based upon their personality rather than academic or professional qualifications. Participants were then given five minutes to prepare a speech. This was followed by participants being asked to get up and give a five-minute speech in front of a panel of two confederates wearing white lab coats and behaving in a neutral manner. Participants were told that their responses would be both judged and recorded for later body language and voice modulation analyses. Immediately post-speech, participants were asked to count backwards from 2043 in increments of 17. Each time they voiced an incorrect number over the course of those five minutes, they were asked to start over.

**Physiological assessment.**

**Cortisol:** Saliva samples were stored at -20C until study completion, centrifuged for 15 minutes at 1800 x g, and tested for cortisol levels using a commercially available chemiluminescence assay (IBL, Toronto, Canada). Maximum cortisol increases were computed by subtracting for each participant individually the pre-TSST level from their highest post-TSST level.
Analytical Plan

All analyses controlled for BMI, as it has been shown to have a significant effect on cortisol production (31). Chi$^2$ and t-tests for gender-dependent differences in any of the sleep and cortisol variables were conducted. A repeated-measures ANCOVA was computed to test whether the TSST was successful at inducing a cortisol stress response in participants.

To test our hypothesis that lower sleep quality and quantity would be associated with stronger cortisol stress responses, we computed several repeated-measures ANCOVAs examining effects of the various PSQI subscales on cortisol stress responses. We also conducted univariate ANCOVAs to examine the effects of these subscales on maximum cortisol increase. 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 of < .05 were considered indicative of a significant effect.
Results

Preliminary Results

As frequencies across the 0 to 3 categories in each of the subscales were heavily skewed, we re-categorized the answers in each subscale either according to the literature or aiming at combining categories with too low frequencies. More specifically, for sleep duration, we broke down the PSQI sleep duration subscale (PSQI items 1 and 2: wake-up time minus bed-time) into three categories in line with the current literature (32-34): below six hours (n=12), from six until but not including seven hours (n=16), and seven hours and above (n=45). Answers on the one-item sleep duration subscale (PSQI item 3) 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.),” were categorized similarly into ‘severe sleep duration problems’ (n=9), ‘minimal sleep duration problems’ (n=18), and ‘no sleep duration problems’ (n=46). Of note, twenty-one participants changed sleep duration categories when comparing sleep-duration computed based on self-reported bedtime and wake-up times (PSQI items 1 and 2) with self-reported sleep duration (PSQI item 3). For all other items assessed in the current study, see Table 1 for more details on response frequencies and descriptive statistics. For none of the sleep variables did we find differences in gender distributions across answer categories (all $p \geq .46$). The same was true for age (all $p \geq .40$).
When examining cortisol stress responses, we found that cortisol responses changed significantly over time in a gender-dependent manner such that although no overall gender main effect was observed, $F(1, 69) = .86, p = .36$, men showed a trend to stronger responses compared to women, $F(5, 345) = 2.48, p = .08$.

**Testing Hypotheses**

*Effects of sleep duration on cortisol stress responses.*

When assessing the effects of sleep duration on cortisol stress responses via a repeated-measures ANCOVA, no effect was found for either gender. This was true whether sleep duration was examined via PSQI self-reported average sleep duration, $F(10, 325) = .63, p = .66$, or sleep duration computed based on self-reported average bedtimes and wake-times, $F(10, 325) = .43, p = .81$.

*Effects of sleep quality on cortisol stress responses.*

Examining sleep quality effects on cortisol stress responses, a gender-dependent three-way interaction was found, $F(5, 335) = 3.17, p = .04$, such that both men and women reporting good sleep quality showed comparable cortisol stress responses in the expected intensity. However, women with low sleep quality exhibited a blunted cortisol response after undergoing a psychosocial stressor, while men with low sleep quality showed an exaggerated cortisol response to the TSST (see Table 2 and Figure 1).

*Effects of daytime dysfunction on cortisol stress responses.*

When examining the impact of participants’ report on trouble staying awake via repeated-measures ANCOVA, we found a two-way interaction between trouble staying
awake and cortisol levels over time, $F(10, 365) = 2.72, p = .06$, indicating that independent of gender, those who had trouble staying awake during the day showed a trend toward blunted cortisol stress responses (see Table 2 and Figure 2a).

In terms of responses to the question about ‘trouble keeping up enthusiasm’, we observed a gender-independent trend for those participants who said that they never had any trouble keeping up enthusiasm whatsoever in the past month to exhibit a low cortisol response, while those who indicated that they had either somewhat of a problem or a very big problem keeping up enthusiasm showed a stronger cortisol response when compared with the other groups, $F(5, 325) = 1.99, p = .09$ (see Table 2 and Figure 2b).

*Impact of maximum cortisol increase and sleep quality.*

The above pattern of findings stayed the same when assessing maximum cortisol increases rather than cortisol stress response profiles. However, to distinguish effects of sleep habits on acute stress responses from more chronic influences on basal cortisol levels, we additionally examined the effects of sleep variables on baseline cortisol samples via univariate ANCOVA. While neither sleep duration nor daytime dysfunctions were linked to baseline cortisol level differences (all $p \geq .53$), subjective sleep quality over the previous month impacted participants’ baseline cortisol responses, $F(1, 72) = 12.24, p < .001$, with poor sleep quality being associated with blunted baseline cortisol levels compared to good sleep quality.
**Discussion**

The current study revealed differential effects of sleep facets on cortisol responses to acute psychosocial stress. More specifically, self-reported average sleep duration did not appear to impact cortisol stress responses. Contrarily, perceived sleep quality not only impacted baseline cortisol levels for both men and women, but also cortisol stress responses. However, this effect was gender-dependent, such that men who reported poor sleep quality showed exaggerated cortisol responses, while women exhibited blunted responses. Lastly, participants who reported having trouble staying awake or keeping up enthusiasm also showed blunted cortisol responses compared to participants who did not experience such daytime dysfunctions.

**Sleep duration**

In previous research on children, short sleep duration has been linked to higher levels of diurnal cortisol as measured by area under the curve, as well as toward a higher cortisol awakening response (CAR), which is a spike in cortisol after awakening hypothesized to come from anticipation of the coming day (23,35). Interestingly, in adults sleep duration does not appear to have the same effect on CAR (36,37). The current study’s finding in young adults showing a lack of sleep duration impact on afternoon baseline cortisol levels extends those previous reports. However, it should be pointed out that the above observations are in contrast to findings in participants who are severely sleep deprived. More specifically, severe sleep deprivation (e.g. two hours
a night) has been associated with significantly blunted or exaggerated cortisol levels the next day (38,39). However, participants in the current study obtained an average of 6.89 hours per night, thus not experiencing a comparable level of sleep deprivation. This discrepancy suggests a threshold beyond which sleep deprivation affects the HPA axis.

Our findings also replicate previous research on children undergoing the TSST-C, showing that sleep duration was not associated with differences in cortisol stress responses (23). Hence, while sleep duration may affect basal cortisol levels if lowered below a certain threshold, it appears that the stress system’s ability to react is not or not yet affected in young adults and children. However, short sleep duration may contribute to future allostatic load (40). More specifically, it is possible that negative effects of abnormal sleep patterns, such as typically seen in undergraduate university students (41), will accumulate over time and thus become health-relevant later in life (19,20).

**Sleep quality**

Contrary to sleep duration, we observed associations with baseline cortisol levels for sleep quality. This finding is similar to Goodin et al.’s (11) finding showing that poor sleep quality increases basal cortisol levels. However, our observation that sleep quality also affects cortisol responses to acute psychosocial stress expands those previous findings (11). More specifically, Goodin and colleagues found that poor sleep quality increased cortisol responses to a physiological stressor. Together, those observations point towards poor sleep quality having a stressor type-independent effect on the body’s stress system. Furthermore, sleep quality’s impact on pre-stress cortisol levels indicate that a body already under strain due to the physiological stress of not
obtaining high-quality sleep is at risk for increased dysregulation when further challenged, in this case with a psychosocial stressor. Coupled with the finding that poor sleep quality adversely impacts baseline cortisol levels, it is possible that poor-quality sleepers can enter into a viscous cycle in which an already chronically taxed system is further taxed by repeated and acute stress responses. These responses then can cause increased wear-and-tear to the body, i.e., adding allostatic load, which in turn may then increase the risk for basal stress system dysfunction (40).

Importantly, the effect of sleep quality on cortisol stress responses differed for men and women. As such, the current findings extend those reported in the study on children mentioned above by Raikkonen et al. 2010 (23), who found that low sleep efficiency, another measure of sleep quality, led to increased cortisol reactivity after undergoing the TSST in both genders. More specifically, those effects observed in children appear to lead into gender-dependent findings in adults: men who are poor-quality sleepers showed exaggerated cortisol stress responses and women who are poor-quality sleepers showed blunted responses. This replicates previous findings demonstrating that women who were objectively poor sleepers, as measured by nighttime awakenings via actigraphy, showed blunted response to an acute mental stressor, i.e., the Stroop test (42). Furthermore, Prather and colleagues found that women, but not men, who reported baseline sleep quality disturbances had higher interleukin-6 levels over a five-year period (43). As hypocorticolism is linked to increased proinflammatory cytokines, it is possible that blunted HPA axis reactivity as a result of low sleep quality could be a mechanism by which inflammatory markers are increased in women (44). Contrarily, in a study by Vgontzas et al. (45), participants with
low sleep efficiency exhibited significantly higher 24-hour plasma cortisol than their better-sleeping peers. Interestingly, the majority of participants in the low-sleep efficiency group were male, thus being in line with the pattern found for men in the current study. Vgontzas et al. (45) hypothesized that the exaggerated cortisol levels in low-efficiency sleepers were due to HPA system hyperarousal.

It is intriguing to speculate that the observed gender differences in sleep quality effects on stress measures may present a mechanism underlying some of the gender-specific differences in disease incidence and prevalence. In more detail, women’s blunted cortisol responses resulting from poor sleep quality may put them at increased risk for diseases associated with a lack of immune inhibition including autoimmune diseases and allergies (46,47) as well as asthma and rhinitis (48). Men’s exaggerated cortisol response due to poor sleep quality may put them at risk for problems related to chronically exaggerated cortisol such as insulin resistance, hypertension, and infectious disease such as community-acquired pneumonia (49,50).

In summary, sleep quality shows strong gender-dependent associations with the way an individual’s body is responding to acute psychosocial stress. Although opposite in direction, both types of dysfunctions – blunted cortisol stress responses seen in women and exaggerated stress response seen in men – can be linked to gender-specific increased risks for negative health outcomes (e.g., 46,49).

Looking at the above findings from a methodological angle, it should be noted that although we observed overall stronger cortisol stress responses in men compared to women, this difference vanished when assessing only participants reporting high sleep quality, such that both men and women who were high-quality sleepers showed
healthy, robust cortisol responses. This observation suggest that future studies examining cortisol responses to acute psychosocial stress may benefit from assessing sleep quality as a covariate, since the often observed weaker cortisol stress responses in women (e.g., 25) may be a function of their sleeping pattern rather than simply the effect of sex hormones.

**Daytime dysfunction**

Lastly, we assessed the effects of daytime dysfunctions on cortisol stress responses and found that those who had trouble staying awake during the day showed a trend toward lower cortisol responses to the TSST. Interestingly, both poor perceived sleep quality and daytime sleepiness have recently been found to be associated with poor health outcomes in diabetes self-management (51). As such, our findings suggest that an organism’s impaired ability to cope with challenges may be an important mechanism linking daytime dysfunction and negative health outcomes. The same is true for trouble keeping up enthusiasm, another component of daytime dysfunctions associated with poor sleep. Specifically, we observed that trouble keeping up enthusiasm over the past month was linked to blunted cortisol responses to the TSST. One closely related area of research in this context is burnout. Indeed, previous literature has indicated that burnout is associated with dysregulated cortisol awakening responses, however, results regarding the directionality of this dysregulation are mixed (i.e. blunted or exaggerated cortisol responses) (52,53).

From a more broader perspective, blunted cortisol stress responses in association with both components of daytime dysfunction also point toward the existence of gender-dependent pathways by which poor nighttime sleep quality may
exert its negative health effects. More specifically, blunted cortisol responses were only observed when participants experienced high daytime dysfunction or when female participants indicated low sleep quality. One potential pathway contributing to these gender-dependent patterns 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 (54,55). Interestingly, using suppression as an emotion regulation strategy leads to exaggerated cortisol responses, and using suppression before bed leads to worse sleep quality than employing reappraisal (54,55). Therefore, it is possible that men’s use of suppression as an emotion regulation strategy may modulate the relationship between sleep quality and cortisol. Another promising pathway underlying the observed gender-differences may be sleep effects of self-regulation. Self-regulation is often measured by heart rate variability (HRV) (56). Men were found to exhibit increased high frequency (HF) power, a component of HRV and measure of sympathetic activity, in stage two and stage four sleep when compared to wakefulness (57) and a higher low frequency/high frequency (LF/HF) ratio during stage four sleep, indicating sympathetic, rather than parasympathetic, dominance (57). Contrarily, women did not show increased HF power or a significantly higher LF/HF ratio during any sleep stages when compared to wakefulness (57). Hence, it can be speculated that, given men having more trouble with self-regulation during sleep than women, it should be more difficult for men’s already-taxed bodies to deal with poor sleep quality than women’s. Lastly, gender-dependent differences in the pattern of links between sleep and acute stress measures
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 (58), though the majority of studies indicate that men have lower quality sleep than women (13). Thus, while men may not accurately self-report their sleep quality, it may be socially more acceptable for men to self-report daytime dysfunctions. As such, differences in self-report accuracy may help explain different physiological effects of sleep quality and daytime dysfunction in men.

Besides the above gender-dependent patterns, the current findings also differentiated between 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. However, it is possible that while the body is unable to maintain normal HPA functioning in an acute psychosocial stress situation after falling prey to low sleep quality, it may retain compensatory capacities to deal with challenges during times of being taxed with the physiological effects of shorter, non-severe sleep duration.

Overall, the current study suggests psychosocial stress and its endocrine effects as one mechanism linking poor sleep quality and daytime dysfunction with detrimental physical health outcomes. More specifically, both blunted and exaggerated cortisol reactivity have been shown to be insalubrious (e.g., 46,49) and as such may be one physiological link underlying the well-documented detrimental health effects of poor sleep (33,59,60)
Limitations

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 irregular sleeping patterns (9), 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, it would be interesting for future studies to extend the current study by including relevant health outcomes.

Summary and Outlook

Results of the current study indicate that perceptions of sleep quality and daytime dysfunction have consequences to the body’s ability to respond to challenges. As such, the current study implicates stress and the associated physiological stress systems as one gender-dependent pathway linking poor sleep with negative health outcomes. However, the lack of sleep duration effects indicates that the body may retain some degree of resilience. Future studies will have to investigate if this resilience persists into later stages of life or whether over time, will contribute to sleep quality-related stress system dysfunctions and thus negative health outcome.
References


11. Goodin BR, Smith MT, Quinn NB, King CD, McGuire L. Poor sleep quality and exaggerated salivary cortisol reactivity to the cold pressor task predict greater acute pain severity in a non-clinical sample. Biol Psychol 2012;91:36-41.


42. Wright CE, Valdimarsdottir HB, Erblich J, Bovbjerg DH. Poor sleep the night before an experimental stress task is associated with reduced cortisol reactivity in healthy women. Biol Psychol 2007;74:319-27.


sleep: Breaking the link between negative events and sleep disturbance. Emotion 2012;12:1415.


Table 1. *Categorization of PSQI subscale scores by gender.*

<table>
<thead>
<tr>
<th>Item Responses</th>
<th>Item Response</th>
<th>(X^2)</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(PSQI category scores)</strong></td>
<td><strong>Frequency</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Trouble Staying Awake</strong></td>
<td>Males</td>
<td>Females</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No trouble (0)</td>
<td>18</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At least some trouble (1, 2, 3)</td>
<td>11</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Trouble With Enthusiasm</strong></td>
<td>Males</td>
<td>Females</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No problem (0)</td>
<td>5</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A very slight problem (1)</td>
<td>18</td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somewhat of a problem (2, 3)</td>
<td>6</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sleep Duration (PSQI)</strong></td>
<td>Males</td>
<td>Females</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 hours or above (0)</td>
<td>17</td>
<td>28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 to 6.99 hours (1)</td>
<td>7</td>
<td>9</td>
<td></td>
<td></td>
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<tr>
<td>0 to 5.99 hours (2, 3)</td>
<td>5</td>
<td>7</td>
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<td><strong>Sleep Duration (computed)</strong></td>
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<td>Females</td>
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<td>7 hours or above (0)</td>
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<td>Females</td>
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<td></td>
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<tr>
<td>Good sleep quality (2, 3)</td>
<td>16</td>
<td>23</td>
<td></td>
<td></td>
</tr>
</tbody>
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*Note. All \(X^2\) were non-significant.*
Table 2. Regression analysis findings of gender-dependent daytime dysfunction and sleep quality effects on cortisol stress responses.

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<thead>
<tr>
<th>IV</th>
<th>Sleep Quality</th>
<th>Staying Awake</th>
<th>Enthusiasm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$F$ (df, df)</td>
<td>$p$</td>
<td>$F$ (df, df)</td>
</tr>
<tr>
<td><strong>Between-subject effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>0.04 (1,67)</td>
<td>.85</td>
<td>0.89 (1, 73)</td>
</tr>
<tr>
<td>Gender</td>
<td>0.75 (1,67)</td>
<td>.39</td>
<td>1.06 (1, 73)</td>
</tr>
<tr>
<td>IV</td>
<td>3.43 (1,67)</td>
<td>.07†</td>
<td>3.11 (1, 73)</td>
</tr>
<tr>
<td>IV * gender</td>
<td>3.24 (1,67)</td>
<td>.08†</td>
<td>1.31 (1, 73)</td>
</tr>
<tr>
<td><strong>Within-subject effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisol</td>
<td>0.94 (5,335)</td>
<td>.40</td>
<td>0.87 (5, 365)</td>
</tr>
<tr>
<td>Cortisol * BMI</td>
<td>0.61 (5,335)</td>
<td>.56</td>
<td>1.59 (5, 365)</td>
</tr>
<tr>
<td>Cortisol * gender</td>
<td>2.24 (5,335)</td>
<td>.10†</td>
<td>2.51 (5, 365)</td>
</tr>
<tr>
<td>Cortisol * IV</td>
<td>1.37 (5,335)</td>
<td>.26</td>
<td>2.72 (10, 365)</td>
</tr>
<tr>
<td>Cortisol * IV * gender</td>
<td>3.17 (5,335)</td>
<td>.04*</td>
<td>0.60 (10,365)</td>
</tr>
</tbody>
</table>

*Note. *$p < .05$. **$p < .01$. ***$p < .001$
Figures

Figure 1. *The impact of sleep quality on cortisol stress responses in females (left) and males (right).*

Figure 2. *The impact of daytime dysfunction on cortisol stress responses (left: trouble staying awake, right: trouble with enthusiasm).*