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Sleep Deprivation and Stressors: Evidence for Elevated Negative Affect in Response to Mild Stressors When Sleep Deprived

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Stress often co-occurs with inadequate sleep duration, and both are believed to impact mood and emotion. It is not yet known whether inadequate sleep simply increases the intensity of subsequent stress responses or interacts with stressors in more complicated ways. To address this issue, we investigated the effects of one night of total sleep deprivation on subjective stress and mood in response to low-stress and high-stress cognitive testing conditions in healthy adult volunteers in two separate experiments (total $N = 53$). Sleep was manipulated in a controlled, laboratory setting and stressor intensity was manipulated by changing difficulty of cognitive tasks, time pressure, and feedback about performance. Sleep-deprived participants reported greater subjective stress, anxiety, and anger than rested controls following exposure to the low-stressor condition, but not in response to the high-stressor condition, which elevated negative mood and stress about equally for both sleep conditions. These results suggest that sleep deprivation lowers the psychological threshold for the perception of stress from cognitive demands but does not selectively increase the magnitude of negative affect in response to high-stress performance demands.

Keywords: sleep deprivation, stress, stressors, mood

Stress responses are essential for survival, but can also interfere with proper behavioral and biological functioning. Physiological responses to stressors (such as increased heart rate and respiration and pupil dilation) facilitate the detection of and escape from threat, but chronically elevated and excessive stress can damage bodily systems and reduce well-being and positive affect (Korte, Koolhaas, Wingfield, & McEwen, 2005). Threats to psychological integrity, such as to one's social standing or future earning potential, can activate the same physiological responses as physical threats (Gunnar & Quevedo, 2007). These responses can be maladaptive, interfering with attention (e.g., rapid heart rate during an important test can be distracting) and decreasing interpersonal effectiveness (e.g., during public speaking). When chronic, the physiological responses to stressors can lead to poor sleep quality and maladaptive behaviors that damage the nervous system over time (McEwen, 2008).

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It is believed that the relationship between stress and sleep is bidirectional—that is, stress can disrupt sleep and sleep loss can increase subsequent stress levels (Vgontzas & Chrousos, 2002; Vgontzas et al., 1998). While there is considerable scientific work on the disruption of sleep by prior stress (Akerstedt, 2006; Akerstedt et al., 2002; Akerstedt, Kecklund, & Axelsson, 2007), much less is known about the manner in which sleep loss affects subsequent responses to stressors.

Animal studies have found evidence that sleep deprivation alters both baseline activity of the stress system and physiological responses to subsequent stress (see Meerlo, Sgoifo, & Suchecki, 2008 for review), but data in humans are lacking. To date there are no published studies that have experimentally manipulated both sleep and stress exposure. Studies that have measured physiological markers of stress (such as cortisol) during sleep deprivation experiments have found inconsistent results (see Leproult, Copinschi, Buxton, & Van Cauter, 1997; Redwine, Hauger, Gillin, & Irwin, 2000). Nevertheless, indirect evidence from neuroimaging studies supports the hypothesis that sleep deprivation is likely to decrease the efficiency of top-down inhibition circuitry. Yoo, Gujar, Hu, Jolesz, and Walker (2007) reported that sleep deprivation was associated with reduced functional connectivity between the amygdala and medial prefrontal cortex, as well as greater overall amygdala reactivity, in response to passive viewing of negative photographs. Similar deficits in prefrontal control regions induced by sleep loss have been reported using a go/no-go task (Chuah, Venkatraman, Dinges, & Chee, 2006), gambling paradigm (Venkatraman, Chuah, Huettel, & Chee, 2007), working memory task (Chee et al., 2006), and passive viewing of positive photo-

graphs (Gujar, Yoo, Hu, & Walker, 2011). We reasoned that because the prefrontal control regions that are sensitive to sleep loss and involved in top-down inhibition are also implicated in stress induction (Wang et al., 2005), sleep deprivation would potentiate the subjective effects of exposure to a stressor. Furthermore, we predicted that the effect would be larger for a more intense stressor than for a milder stressor. If this were found, it would suggest sleep loss potentiates affective responses to stressors in proportion to the severity of the stressor.

Overview of the Present Studies

Two experiments were conducted to investigate the influence of sleep deprivation on subjective affective responses to cognitive performance stressors in healthy adult volunteers. To the best of our knowledge, these are the first studies to experimentally manipulate both exposure to stressors and sleep deprivation in humans. We used a previously validated stress-induction procedure (Dinges et al., 2005) that altered task difficulty and feedback about performance to create relatively low-stress and high-stress experimental conditions. The study design included random assignment to sleep condition and repeated exposure to stressors on two consecutive days of testing. This experimental design allowed for both within- and between-groups comparisons where nuisance variables (such as mood changes due to experimental conditions) could be evaluated and statistically controlled. Participants were randomly assigned to a night of total sleep deprivation or a control condition (full night of sleep), and completed stress-induction procedures in a controlled laboratory setting. The methods of each study (presented as Study 1 and Study 2) are explained below. The results from Study 2 independently replicated the results of Study 1 and were therefore combined to increase statistical power. Results from these combined analyses are reported with Cohen *d* effect sizes (Cohen, 1988). Results were inconsistent with our hypotheses, thus alternative explanations and theoretical models are discussed.

Method—Study 1

Participants and Procedure

Participants were 30 healthy adults (15 women, 15 men; 13 African American, 14 Caucasian, 3 other or declined to state) between the ages of 22 and 43 years (mean age = 29.0 +/- 7.0) recruited from the community. Volunteers were screened to ensure they had no medical, psychiatric, or sleep-related disorders, were nonsmokers, and were drug-free. This was determined by comprehensive self-reported medical history and the Structured Clinical Interview for *DSM–IV* Axis I disorders (SCID I; First, Spitzer, Gibbon, & Williams, 1997), which was conducted by a Masters-level doctoral student in clinical psychology. Interested participants were not included if they had engaged in any regular night or rotating shift work within the last 6 months, or had traveled across times zones in the last 3 months. Participants were also excluded immediately prior to study participation if they were physically ill, had poor sleep the previous night, indicated suicidal ideation, and/or reported Beck Depression Inventory (BDI-II; Beck, 1996) scores at or above 14 prior to the first day of testing.

Measures

Subjective stress was measured before and after each stressor condition by a computer-administered visual analog scale (0–8) with a range of *not stressed at all* to *extremely stressed*. Mood was measured after each condition by a computerized version of the Profile of Mood States (POMS; McNair, Lorr & Droppleman, 1971; McNair & Heuchert, 2005), a 65-item adjective checklist where each item is rated on a 5-point scale that ranges from *not at all* to *extremely*. The POMS was chosen because it has been used extensively in sleep deprivation research (see Goel, Rao, Durmer, & Dinges, 2009 for review). To facilitate comparisons among self-report instruments, raw scores were converted to a 0–100 scale, which adjusts for differences in the number of items included in each subscale. Analyses of the affective consequences of stressor exposure focused on Depression-Dejection, Tension-Anxiety, and Anger-Hostility subscales of the POMS. Responses to the Fatigue-Inertia subscale were analyzed as a manipulation check to confirm that sleep deprived participants reported more fatigue than the rested control group.

Sleep Deprivation

Participants completed a 48-hr laboratory-based protocol that included two continuous days of testing. The study was approved by the Institutional Review Board of the University of Pennsylvania and all participants provided informed consent and were compensated for their participation. Participants entered the laboratory at 9:00 a.m. (Day 1) and completed consenting procedures. Testing was completed throughout the day (procedures described below), and participants remained in the laboratory and either received no sleep opportunity (sleep deprivation group) or 9-hr of sleep opportunity (control group) from 11:00 p.m. to 8:00 a.m. Fourteen participants were randomized to the sleep deprivation protocol (6 women) and 16 participants (9 women) were randomized to the control condition. In order to prevent bias between sleep deprivation and control groups, participants were not informed of their condition until 8:00 p.m. on Day 1 (after low and high-stressor testing conditions had been completed). At night, participants in the sleep-deprived condition completed 10 minute reaction time (RT) tasks every two hours. Outside of testing, sleep-deprived participants were allowed to read magazines, complete puzzles, and interact with staff to maintain wakefulness, but no TV or electronics were available. Although two subjects at a time were enrolled in the study (always in the same sleep condition), contact between participants was strictly prohibited. A second day of testing (Day 2) was then completed followed by 10 hours of recovery sleep opportunity for all participants.

At all scheduled wake times, participants were kept awake in the laboratory under continuous behavioral monitoring and were exposed to artificial lighting only (levels were not strictly controlled during wake times). During scheduled sleep times, all lights were turned off (less than 1 lux) and trained staff members monitored participants using infrared cameras from an adjacent room. Adherence to sleep condition was further validated by actigraphy, an objective method for estimating sleep based on movement (Ancoli-Israel et al., 2003). Participants in both conditions remained in the laboratory for the full duration of the experiment.

Stress Induction

Participants completed cognitive performance test batteries under conditions designed and previously validated to induce relatively low and high subjective stress (Dinges et al., 2005). Participants completed both high-stressor and low-stressor conditions on both days of the experiment. Testing lasted approximately 20 minutes and occurred at the same time on both days (between 4:00 p.m. and 6:00 p.m.) to control for circadian effects on mood and alertness. All participants first completed the low-stressor condition, then returned to their rooms to complete questionnaires. Approximately 1 hour later, they completed the high-stressor condition. The lack of counterbalancing of order of stressor condition was deliberate to prevent responses to the more intense stressors from contaminating subsequent responses to mild stressors.

Testing environment. Participants completed all stressor tasks alone in a brightly lit room that contained minimal furniture, giving it an austere appearance. A trained staff member, unfamiliar to the participants, delivered instructions for the tasks by intercom. Cameras were clearly visible in front of and above the participant. Participants were not told that the tasks were intended to produce stress, but rather that they were tests of cognition and performance that were very important for the study.

Stressor tasks. Mental arithmetic tasks were used as the primary stressor. In the high-stressor condition, participants responded orally to a serial subtraction task (i.e., repeated subtraction of 13 from a four-digit number, Kirschbaum, Pirke, & Hellhammer, 1993; Wang et al., 2005) and a difficult descending subtraction task (i.e., repeated subtraction of 9, then 8, then 7 etc. from a four-digit number; Dinges, Orne, Evans, & Orne, 1981). In the low-stressor condition, participants counted backward by 2. The high-stressor condition also included a difficult version of the Stroop color-naming task (i.e., names and colors were discordant; Stroop, 1935) and a difficult divided attention task involving the concurrent presentation of memory, arithmetic, visual monitoring and auditory monitoring tasks (Synwork; Elsmore, 1994), while the low-stressor condition included an easy version of the Stroop (i.e., font colors and names were concordant) and no divided attention task. The high-stressor condition also included negative feedback about performance given both orally and by computer, and greater time pressure than the low-stressor condition. Negative feedback included presentation on the participant's monitor of his or her performance as a percentile score relative to other participants. All participants were shown a percentile score that was in the bottom 25th percentile (regardless of their actual performance) and an experimenter read a script over the intercom stating that their performance was "well below average." Participants were informed of the true nature of the experiment and the performance feedback immediately following the final stress induction procedure (on Day 2). Debrief interviews confirmed that participants were unaware that the procedures were primarily intended to elicit stress.

Additional Procedures

Although the primary aim of this experiment was to evaluate the influence of sleep deprivation on stress responses, additional procedures were completed when time allowed. These secondary measures included additional questionnaires and tests of executive function and other cognitive abilities (results not reported here).

Study 2

Study 2 was designed as a replication of Study 1 but it also included a higher probability of randomization to the sleep deprivation condition to maximize power for within-subjects comparisons. Methods are reported with an emphasis on ways in which the two studies differed. One participant in the sleep deprivation condition failed to complete questionnaires following exposure to the high-stressor condition on Day 2.

Method

Participants and Procedure

Participants were 23 healthy adults (9 women, 14 men; 13 Caucasian, 8 African American, 2 other or declined to state) between the ages of 22 and 45 years (mean age = 30.8 +/- 6.8) recruited from the community. The same inclusion criteria were used as in Study 1, but psychological problems were assessed by self-report only (i.e., no clinical interviews were conducted). Participants reporting suicidal ideation and/or BDI-II scores greater than 14 were disqualified from the study and referred to the study psychiatrist.

Measures

The same self-report measures were used as in Study 1. Subjective stress was measured before and after each induction procedure; mood was measured after each induction procedure.

Sleep Deprivation

The same sleep deprivation procedures were used in Study 2 as in Study 1 (see Sleep Deprivation section above). Fifteen participants were randomized to the sleep deprivation protocol (6 women) and 8 participants (3 women) were randomized to the control condition.

Stress Induction

Study 2 included the same basic approach to stress induction as Study 1 (see Stress Induction section above). All tasks were the same, with the exception of the difficult divided attention task (i.e., Synwork), which was removed from the high-stressor condition. The conditions differentiating the low-stressor from high-stressor conditions were also identical between Study 1 and Study 2, with the exception of negative feedback about performance, which was given by computer only, rather than both by computer and by the experimenter. These changes were made based on feedback from participants and staff about which tasks were most effective in eliciting subjective stress.

Results (Combined Sample)

Participants

The combined sample included 53 healthy adults (29 men and 24 women; 21 African American, 26 Caucasian, and 6 other or declined to state) between the ages of 22 and 45 years (mean age 29.8 +/- 6.9). The combined sample included 29 sleep-deprived

participants, 12 female, 15 African American, 10 Caucasian, and 4 other) and 24 control participants (12 female, 6 African American, 17 Caucasian, 1 other).

Manipulation Checks

The effects of the high-stressor versus low-stressor condition were compared using within-subjects analyses of affective responses on Day 1, prior to the sleep manipulation. In the combined sample, the high-stressor condition elicited significantly greater subjective stress than the low-stressor condition, $t(52) = 6.44$, $p < .001$, $d = 0.93$. The manipulation also had significant effects on negative affect. The Anger-Hostility, $t(52) = 2.51$, $p = .02$, $d = 0.35$; Tension-Anxiety, $t(52) = 4.73$, $p < .001$, $d = 0.65$; and Depression-Dejection, $t(52) = 2.83$, $p = .007$, $d = 0.50$, subscale scores were significantly higher following the high-stressor condition compared to the low-stressor condition.

The effects of sleep deprivation on subjective feelings of fatigue were assessed using between-subjects analyses of mood responses on Day 2, following the sleep manipulation. Sleep deprivation was associated with significantly greater Fatigue-Inertia subscale scores after both the low-stressor condition, $F(1, 51) = 54.01$, $p < .001$, $d = 2.33$, and high-stressor condition, $F(1, 50) = 32.56$, $p < .001$, $d = 1.67$.

Influence of Sleep Deprivation on Subjective Responses to Stressors

Sleep deprivation and low-stressor condition. ANCOVA analyses were used to compare subjective stress and negative affect (i.e., anger, anxiety, and depression) between sleep-deprived participants and rested controls on Day 2 (after the sleep manipulation) while controlling for these responses on Day 1 (before the sleep manipulation). Following the low-stressor condition, sleep-deprived participants reported significantly higher levels of subjective stress, $F(1, 52) = 5.48$, $p = .02$, $d = 0.61$ than control participants. Negative mood states were also elevated. Anger-Hostility, $F(1, 52) = 16.63$, $p < .001$, $d = 0.91$, and Tension-Anxiety, $F(1, 52) = 10.80$, $p = .002$, $d = 0.81$, subscale scores were greater for sleep deprived participants than for rested participants. There was also a marginally significant trend toward sleep-deprived participants reporting greater Depression-Dejection subscale scores, $F(1, 52) = 3.34$, $p = .07$, $d = 0.56$. These results are reported in Table 1.

Sleep deprivation and high-stressor condition. Following the high-stressor induction procedure, there were no significant differences between sleep-deprived and control participants on subjective stress, $F(1, 50) = 0.78$, $p = .38$. Similarly, there were no differences between sleep deprived and rested control participants on Anger-Hostility, Tension-Anxiety, or Depression-Dejection, all $F(1, 51) < 1.19$, all p values $> .28$, all d values <0.27 following the high-stressor condition.

Within-subjects analyses, postmanipulation. Paired-samples t tests comparing subjective stress and mood responses between the low-stressor and high-stressor conditions on Day 2 revealed that participants in the control condition reported greater Subjective Stress, $t(23) = 4.40$, $p < .001$, following the high-stressor condition than following the low-stressor condition. Negative mood states were also elevated. Tension-Anxiety, $t(23) = 3.10$, $p = .005$, and Anger-Hostility, $t(23) = 2.48$, $p = .02$, were significantly greater following the high-stressor condition, but the Depression-Dejection subscale was not significantly affected, $t(23) = 1.44$, $p = .17$.

Participants in the sleep deprivation condition reported significantly greater subjective stress following the high-stressor task than following the low-stressor task as well, $t(28) = 3.77$, $p = .001$. Analyses of mood subscales revealed a nonsignificant trend toward greater Tension-Anxiety scores, $t(27) = 1.93$, $p = .06$, but differences between the low-stressor and high-stressor condition were not significant for Anger-Hostility, $t(27) = 1.00$, $p = .33$, or Depression-Dejection, $t(27) = 0.27$, $p = .79$.

Interactions of sleep-condition and stressors. A repeated measures ANOVA was conducted where stress and mood responses on Day 2 to the low stressor condition and high stressor condition were entered as repeated measures and sleep condition (deprivation vs. control) was entered as a between-groups measure. Subjective stress and mood responses to stressors on Day 1 were entered as covariates in these analyses. The interaction of sleep condition and stressor level was not significant for subjective stress, $F(1, 48) = 0.42$, $p = .52$. A significant interaction of affective response by sleep condition was found for the Anger-

Table 1
Self-Reported Stress and Mood After Stressors for Sleep-Deprived and Control Participants (Both Experiments Combined)

Condition	Mood subscale	Rested ($n = 24$)	Sleep-deprived ($n = 28$)	Cohen d	p
Low Stressor	Subjective Stress	8.3 (10.9)	17.6 (24.3)	0.61	.02
	Anger-Hostility	0.9 (1.9)	4.0 (4.7)	0.91	<.01
	Depression-Dejection	1.0 (1.8)	2.8 (4.0)	0.56	.07
	Tension-Anxiety	8.6 (5.4)	14.0 (7.8)	0.81	<.01
	Fatigue-Inertia	4.8 (6.3)	32.4 (18.2)	2.33	<.001
High Stressor	Subjective Stress	29.2 (25.2)	33.8 (31.5)	0.17	.38
	Anger-Hostility	5.8 (9.8)	5.2 (8.0)	−0.09	.45
	Depression-Dejection	3.5 (8.6)	3.0 (5.0)	−0.10	.80
	Tension-Anxiety	14.4 (9.0)	16.7 (12.2)	0.26	.28
	Fatigue-Inertia	8.5 (10.3)	31.6 (17.9)	1.67	<.001

Note. A negative effect size indicates less negative mood in the sleep-deprived group than the rested control group. To facilitate comparisons, subscale scores were converted to a 0–100 scale.

Hostility subscale, $F(1, 48) = 6.08$, $p = .02$, but not for the Tension-Anxiety, $F(1, 48) = 2.10$, $p = .15$, or Depression-Dejection $F(1, 48) = 1.40$, $p = .24$.

Discussion

The purpose of the experiments reported was to investigate the effects of sleep deprivation on subjective responses to cognitive performance stressors in healthy adult volunteers. Based on previous findings that sleep deprivation is associated with impaired inhibitory control during both cognitive (Chuah et al., 2006) and emotional (Yoo et al., 2007) tasks, we expected to find an interaction where the effect of sleep deprivation increased as the stressor increased (i.e., there would be a small difference between groups in response to low-stressor conditions, but a larger difference in response to the high-stressor condition). In fact, our findings suggest the opposite occurred. Relative to participants who had a full night's sleep, sleep-deprived participants reported significantly greater subjective stress, anger, and anxiety in response to the low-stressor condition, but differences between the sleep-deprived and control groups in response to the high-stressor condition were not significant.

There are two important limitations to keep in mind when interpreting these results. First, all participants completed the low-stressor condition prior to the high-stressor condition. It is therefore possible that order effects influenced responses such that there may not be a unique relationship between mild stressors and sleep deprivation. Additional studies are needed to more fully disentangle these effects, but we believe the manipulation check provided by the control group (namely that they reported greater subjective stress in response to the high-stressor condition than the low-stress condition on Day 2) partially attenuates this concern. Second, the group-by-stressor-condition interactions were mostly not significant (with the exception of anger). As recently emphasized by Nieuwenhuis, Forstmann, & Wagenmakers. (2011), it would be incorrect to conclude that the differences between the responses to the low- and high-stressor conditions were significantly different between sleep-deprived and control participants. Nevertheless, we believe the failure to detect significant interactions likely reflects a relative lack of statistical power and cautiously suggest that there is probably a unique relationship between sleep deprivation and relatively minor stressors.

We interpret our results as suggesting that sleep loss does not necessarily potentiate subjective responses to cognitive performance stressors in proportion to the severity of the stressor. Instead, an alternative hypothesis was supported by both experiments. Rather than increasing subjective responses to more intense stressors, it appears that sleep loss primarily lowers the threshold at which a person experiences an event as a stressful. Additional studies are needed before a satisfactory mechanism for this effect can be identified, but we believe that cognitive appraisals are likely to be involved. A simple explanation for these findings is that sleep-deprived participants experienced unexpected difficulty on relatively easy tasks (e.g., counting by 2) during the low-stressor condition. In contrast, the high-stressor condition included tasks that were so challenging (e.g., subtracting by 13) that all participants experienced similar levels of subjective stress. We recommend that future studies in this area also assess cognitive variables (such as attributions for poor performance) that might mediate associations between sleep loss and negative affective responses to stressors.

An alternative explanation for these findings is that sleep-deprived participants would have hyper-responded to both low-stressor and high-stressor conditions, but the responses to the high-stressor condition were masked by ceiling effects. We believe this explanation is unlikely for two reasons. First, responses to the high-stressor condition were not near the top of the scale, suggesting that participants were at least able to report greater stress and mood changes if they chose. Second, and perhaps more important, responses to the high-stressor condition showed relatively more variability than responses in the low-stressor condition, even in the sleep-deprivation condition. Ceiling effects are associated with a restriction in range that was not observed here. Nevertheless, future studies could address this alternative explanation more directly by quantifying responses in other domains (such as behavioral or neural responses) and/or by exposing participants to a greater variety of stressor intensities.

The unexpected finding that sleep loss was associated with increased negative affective responses to relatively mild cognitive performance stressors represents an important contribution to understanding the relationship between stress and sleep in real-world settings. Previous research has established that subjectively perceived stress during the day interferes with sleep at night (Akerstedt, 2006). Our findings complement this research by showing that inadequate sleep at night may then increase subjective stress and negative mood in response to relatively minor stressors encountered the following day. These findings may also explain how sleep deprivation may contribute to the experience of people feeling overwhelmed (e.g., "I can't take it anymore") or overreacting (e.g., "flying off the handle") in the presence of relatively modest cognitive demands.

Our findings appear robust (given that they were largely replicated in two independent samples) for the effects of one night of total sleep deprivation, which can be experienced in real world settings, such as extended-duty shift work and students "pulling all nighters." It remains uncertain however, whether the results can be generalized to the even more common experience of chronic partial sleep deprivation, which occurs in association with many medical and psychological disorders that shorten sleep, as well as in lifestyles of increased work and commute times (Basner et al., 2007). There is ample evidence that chronic partial sleep deprivation produces cumulative cognitive deficits of the same type and degree as those found for total sleep deprivation (Dinges et al., 1997; Van Dongen, Maislin, Mullington, & Dinges, 2003; Belenky et al., 2003; see Banks & Dinges, 2007 for review), therefore, it is likely that chronic partial sleep deprivation also lowers the threshold for stress reactions to relatively mild performance demands. Nevertheless, studies using chronic partial sleep restriction and recovery sleep opportunities (e.g., Banks, Van Dongen, Maislin, & Dinges, 2010) must be expanded to include evaluation of responses to stressors before the findings presented here can be generalized with confidence beyond total acute sleep deprivation.

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