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ORIGINAL ARTICLE

The Effects of Poor Sleep on Cognitive, Affective, and Physiological Responses to a Laboratory Stressor

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Abstract
Background Recent research suggests that poor sleep may be associated with altered stress regulation.
Purpose This study aims to examine the associations between prior-night and prior-month sleep measures and affective, cognitive, and physiological responses to a laboratory stressor.
Methods Ninety-eight (50 % female) young adults completed measures of sleep quality in the context of a laboratory stress study. Measures included positive (PA) and negative affects (NA) and blood pressure (BP) reactivity, as well as change in pre-sleep arousal.
Results Prior-month poor sleep *quality* and sleep *disturbances* predicted dampened BP reactivity. Both prior-night and prior-month sleep *quality* predicted greater decrease in PA. *Sleep-associated monitoring* predicted NA reactivity and prolonged cognitive and affective activation. Prior-month sleep *continuity* predicted greater cognitive pre-sleep arousal change, and prior-month sleep *quality, daytime dysfunction, and disturbances* predicted prolonged cognitive and affective activation.
Conclusion Findings suggest that inadequate sleep confers vulnerability to poor cognitive, affective, and physiological responses to stress.

Keywords Sleep quality · Stress · Negative affect · Positive affect · Pre-sleep arousal

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Introduction

As many as 40 million Americans may suffer from chronic or intermittent disorders of sleep and sleep deprivation, adding an estimated $15.9 billion to the national health care bill [1]. Almost a quarter of the adult population meets criteria for insomnia [2]. Poor sleep is associated with increased health care use, work absenteeism, and reduced work productivity[3], along with a growing list of adverse health outcomes, including impaired immune functioning [4], susceptibility to infectious illness [5], metabolic syndrome [6, 7], inflammation [8, 9], coronary artery calcification [10], and all-cause mortality [11, 12]. In short, understanding the development of sleep problems, as well as the mechanisms by which poor sleep affects health, are major public health concerns.

Models of the development of sleep disturbance strongly implicate stress as a precipitating factor (e.g., [13–17]). Yet, recent multiprocess models of stress regulation posit that poor sleep both derives from *and* in turn disrupts the other stress processes in feed-forward fashion [18–22]. Importantly, there is little research on the associations between naturally occurring poor sleep and stress regulation in nonclinical samples (i.e., not diagnosed with insomnia)—a research focus that would extend the generalizability of experimental studies in which sleep is manipulated and clinical studies of established sleep disorders. The current study examined the associations between (a) prior-night and prior-month self-reported sleep duration, continuity, and quality, as well as ratings of physical symptoms attributed to prior-night poor sleep (i.e., "sleep-associated monitoring") and (b) affective and physiological responses to a laboratory stressor and changes in pre-sleep arousal—an established marker of current and future sleep difficulties.

Sleep in the Context of Stress Regulation

Stress regulation is a broad term that refers to cognitive, behavioral, and physiological processes that serve to alter, foster adaptation to, or transform psychological stress. Stress, however, is not a unitary construct; rather, it consists of several component processes including stress exposure, stress reactivity, stress recovery, and restoration. These processes are central to recent theoretical frameworks for understanding the mechanisms by which stress affects health [18–21]. Within a multiprocess framework, sleep is considered a central aspect of stress restoration—operating to repair cellular damage and return an individual to baseline levels of physiological activity [23]. Importantly, process models of stress regulation hypothesize feedback loops among the stress processes. Relevant to the current study, inadequate restoration is hypothesized to disrupt the other stress regulation processes. For example, poor sleep may lead to enhanced affective or physiological responses to stressful circumstances, poorer recovery (including prolonged cognitive activation), and further decrements in restoration.

It is also important to consider that sleep itself is not a unitary construct but can be characterized by dimensions that include duration, continuity, architecture, and quality [24], each of which may confer risk for poor stress regulation. In addition, monitoring the internal (e.g., physical sensations) and external environment for sleep-related threats and perception of daytime deficits related to poor perceived sleep (i.e., "sleep-associated monitoring") appears to be central to the development and maintenance of sleep disturbance [13, 15]. Indeed, greater sleep-associated monitoring is associated with higher negative affect during the day [25]. Although associations with affective response to stressful events have not yet been examined, these findings suggest that this sleep-related variable may play a role in stress regulation.

Evidence that disturbed sleep is associated with poor stress regulation comes, in part, from prior research on the effects of sleep loss on emotional states and neural activation to emotion-relevant information. For example, experimental restriction of sleep has been found to result in increased negative affect and decreased positive affect [26, 27]. Whereas prior research has only examined mean levels of negative mood throughout the day, these findings suggest that examination of affective reactivity to stress may be warranted. Furthermore, sleep deprivation is associated with greater pupil dilation to negative versus positive or neutral emotional stimuli [28]. Sleep-deprived individuals (i.e., normal sleepers forced to stay awake overnight) also exhibit greatly enhanced amygdala activity in response to emotional imagery measured with functional magnetic resonant imaging but decreased activity of the medial–prefrontal cortex, suggesting a disruption of the emotional modulation circuitry [29]. That is, sleep-deprived individuals appear to evidence greater central nervous system responses to threat (i.e., amygdala activity to viewing negative/aversive pictures) and less ability to modulate their threat responses, as evidenced by less activity in brain regions that serve that function. These findings are consistent with research indicating that even minor sleep restriction (i.e., 6 h/night for 2 weeks) in normal sleepers has a cumulative negative effect on executive functioning [30], the constellation of cognitive processes that reflects prefrontal cortex functioning and has been implicated in the recently proposed Neurovisceral Integration Model of self-regulation [31]. Specifically, the prefrontal cortex is hypothesized to support not only adaptive self-regulatory behavior but also stress-dampening physiological activity through parasympathetic nervous system mechanisms (via connections to the vagus nerve). Thus, associations between sleep and executive functioning suggest a potential mechanism by which poor sleep may influence cardiovascular responses to stress. Taken together, prior experimental research on the effects of poor sleep on emotional states and cognitive functioning suggests that there may also be adverse effects on cognitive, affective, and physiological responses to stress.

The association between poor sleep quality and affective and physiological responses to life stress has been less studied. Zohar, Tzischinsky, Epstein, and Lavie [32] found that poor sleep among medical residents predicted greater negative affect in response to disruptive daily events. Furthermore, poor sleep (night awakenings and sleep efficiency measured with actigraphy) among adolescents prospectively predicts compromised emotional information processing (emotional expression identification) [33]. Recently, Hasler and Troxler [34] found that, among men, better sleep efficiency predicted fewer next-day negative interactions with romantic partners, suggesting that poor sleep may lead to greater interpersonal stress. Associations between better sleep duration and consistency and reduced perceived stress over time have also been demonstrated [35]. In addition, poor sleep quality the night before a laboratory stressor has been found to predict dampened cortisol responses, interpreted as poor stress adaptation [36], as well as reduced immune cell proliferation [37] in response to the stressor. Indeed, blunted reactivity of both the HPA axis and sympathetic nervous system are increasingly considered important indicators of poor stress adaptation [38]. Furthermore, sleep deprivation studies, as well as studies of individuals with sleep disorders, suggest that poor sleep is associated with increased pro-inflammatory cytokines [39]. Given the established relationship between pro-inflammatory cytokines and negative mood, these findings suggest a potential pathway between poor sleep and affective response to stress.

In summary, prior research suggests that experimentally induced sleep loss predicts increases in negative affect, decreases in positive affect, and disruption of emotion circuitry in the brain. Furthermore, prior-night poor sleep quality may adversely affect stress exposure, as well as physiological and affective responses to stress, and may moderate the

association between stress and health outcomes (e.g., stress-immune functioning associations). Findings of blunted HPA axis reactivity (i.e., cortisol) in relation to poor sleep, suggest the need to examine associations between recent sleep quality and sympathetic nervous system responses to stress (i.e., cardiovascular reactivity).

The Current Study

The current study extends prior research by examining associations between naturally occurring variations in both prior-night and prior-month sleep and affective and cardiovascular reactivity to a laboratory stressor, as well as post-stress cognitive, affective, and somatic pre-sleep arousal in a sample of young adults. Cardiovascular reactivity is an important laboratory stress measure, given that it is implicated in the development of cardiovascular disease [40] and both high and low reactivity may signal homeostatic imbalance [38]. Furthermore, stress exposure may also occur in anticipation of a stressor (i.e., worry) and when a prior stressor is mentally re-imagined (i.e., rumination)—collectively termed perseverative cognition, leading to the proposal for a stress-related construct termed *prolonged activation* [41, 42]. Prolonged activation in response to stressful events may manifest in higher pre-sleep arousal, a key vulnerability factor in the development of chronic sleep problems [43] and perseverative cognition (rumination) has been shown to mediate concurrent associations between stress and poor sleep [44]. Finally, demonstrating that poor sleep confers risk for poor responses to laboratory stress induction will inform both etiological models of the development of insomnia, as well as multiprocess stress regulation models. The present study examined the extent to which prior-night and prior-month measures of sleep predicted changes in NA, PA, HR, and blood pressure, along with nighttime prolonged activation and pre-sleep arousal, in response to a well-validated laboratory stress task.

Although the prior research on associations between sleep quality and stress responses has largely been experimental (i.e., sleep duration is manipulated), several tentative hypotheses are offered. First, prior research suggests that both prior-night sleep duration and sleep-associated monitoring will be associated with greater affective reactivity (i.e., increased NA and decreased PA) to stress. Thus, it is expected that reports of fewer hours slept in the prior night and symptoms in response to prior-night sleep (i.e., sleep-associated monitoring) will be associated with ratings of higher NA and lower PA immediately following the laboratory stressor relative to baseline levels. Second, given evidence that pre-sleep arousal is associated with self-reported vulnerability to stress-related sleep problems [43], it is hypothesized that reports of poorer prior-month sleep continuity and quality will be associated with increases in pre-sleep arousal in response to the laboratory stressor (i.e., nighttime ratings of continued cognitive and affective response to the stressor, ratings of higher pre-sleep arousal the night following the stressor relative to ratings for the prior night). Finally, although the prior literature is limited and mixed, it is tentatively predicted that reports of both prior-night and prior-month poor sleep duration and continuity will be associated with greater blood pressure reactivity (i.e., higher blood pressure during the laboratory stressor relative to baseline levels). The hypothesized associations for the current study are depicted in Fig. 1.

Method

Participants

Participants were 49 male and 49 female undergraduate students who received credit for their participation. Mean age was 23.0 years, SD=5.8. The ethnicity of the sample was 74 % Caucasian, 7 % Latino/Latina, 6 % Asian Pacific, 3 % African American, and 7 % other. Exclusionary criteria included current use of medication known to influence cardiac functioning (e.g., beta blockers). Additionally, participants were asked to avoid consumption of caffeine, use of nicotine, and to refrain from exercise 2 h prior to the experimental session.

Procedure

Following informed consent, participants were screened for exclusionary criteria and compliance with pre-experimental requirements (noncompliance resulted in re-scheduling the lab session). Before beginning the experimental protocol, participants completed ratings of the prior night's sleep and pre-sleep arousal. Participants were then instructed to relax quietly while breathing normally for a period of 10 min, during which baseline blood pressure readings were collected at 90-s intervals. At the end of baseline, participants completed a measure of positive and negative state affect (PA and NA). Following the baseline period, participants rank ordered a list of 21 stressors commonly experienced by college students (derived from the Inventory of College Students' Recent Life Experiences [45]; e.g., *conflicts with friends* and *financial burden*) from least to most stressful. The top-ranked stressful event (that the participant was willing to discuss) was selected for the focus of the Social Competence Interview [46] (described below). Blood pressure readings were obtained every 90 s during the 8- to 10-min stressor period (as well as during a 7-min recovery period). Immediately following the stressor period, participants provided NA and PA ratings related to the prior task. Following the laboratory stress portion of the study, participants completed questionnaire measures. Participants were

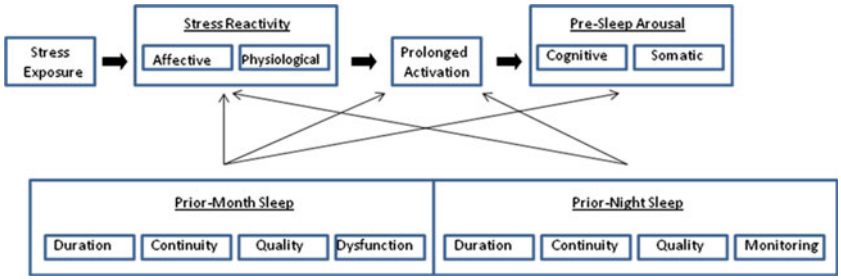

Fig. 1 Hypothesized associations between sleep measures and stress responses in the current study

given instructions to complete an online rating of pre-sleep arousal, as well as ratings of continued cognition and distress regarding the laboratory stressor, the evening after their laboratory session. Participants also received an e-mail prompt in the late afternoon/early evening to complete the survey at bedtime.

Measures—Interviews

Social Competence Interview [46]

The Social Competence Interview is a 10-min semi-structured interview designed to assess socio-emotional responses to a real-life event. The reliability and validity of this interview as a standard laboratory stressor have been well-documented. Blood pressure and HR responses to the interview match or exceed those elicited by stressors such as mental arithmetic, mirror tracing, and video games; moreover, responses to the Social Competence Interview show stronger associations to ambulatory monitoring during daily activities than those stressors (see [46] for review) suggesting that it is has enhanced external validity compared with most laboratory stressors. Furthermore, use of this method of induced stress exposure is in line with recent research implicating mental activation of stress (i.e., anticipation of future stress or re-living past stress) as central to prolonged stress activation [42, 47].

The interview is structured such that the first 4 to 6 min are devoted to re-experiencing the stressful event (specific people, places, what the participant said, and felt). The remainder of the interview focuses on coping with the event, as well as preferred resolution of the problem. Consistent with prior research, reactivity to the first four minutes of the interview was examined.

Measures-Laboratory Physiology and Affect

Blood Pressure and Heart Rate

Dinamap 8100 monitors (Critikon; Tampa, FL) were used to measure heart rate (HR), systolic (SBP), and diastolic blood pressure (DBP). Blood pressure readings were taken at 90-s intervals by attaching an occluding cuff to the upper portion of the nondominant arm.

Positive and Negative Affect Schedule (State Version)

The Positive and Negative Affect Schedule is a self-report measure designed to assess both positive affect (PA; 10 items, e.g., *excited*, *inspired*) and negative affect (NA; 10 items, e.g., *afraid*, *nervous*) [48]. Participants were asked "to what extent did you experience each of the following?" Responses were made on a Likert scale ranging from 1 (very slightly or not at all) to 5 (extremely).

Measures—Questionnaires

Pittsburgh Sleep Quality Index (PSQI)

The PSQI assesses sleep quality disturbances during the previous month [49]. The scale is composed of 19 items which are used to derive seven component scores: Sleep Quality, Sleep Latency, Sleep Duration, Habitual Sleep Efficiency, Sleep Disturbances, Sleep Medication, and Daytime Dysfunction. Component scores are summed to produce a global PSQI score with higher scores indicating poorer sleep quality. This instrument has demonstrated good reliability (alpha=0.83) and validity [50].

Pre-sleep Arousal Scale

The Pre-sleep Arousal Scale is a self-report measure containing 16 items that assess both cognitive (e.g., racing thoughts and worries) and somatic (e.g., heart racing and muscle tension) states of arousal at bedtime [51], with item scores ranging from 1 (not at all) to 5 (extremely). Participants completed this measure for the night prior to the laboratory session and as part of an online survey the evening after completion of the laboratory session. This scale has demonstrated internal consistency (alphas=0.67 to 0.88), and scores reliably discriminate normal sleepers from those with insomnia [50].

Prior-Night Sleep

At the beginning of the laboratory session (i.e., prior to the stress induction paradigm), participants responded to a number of questions regarding their sleep in the prior night. Four measures of prior-night sleep were obtained (1) sleep-onset

latency (*How long did it take you to fall asleep*? (in minutes)); (2) total sleep time (*About how long did you sleep altogether* (*in hours per minute*)? *In other words*, *how much actual sleep did you get last night*?); (3) state sleep quality (*How satisfied/dissatisfied are you with your sleep last night*? *How worried/distressed are you about the quality of your sleep last night*? *How rested or refreshed do you feel*? (5-point Likert scales)); (4) Sleep-Associated Monitoring Index [52] (feelings of tiredness or heaviness in body; heaviness, soreness or itchiness in eyes; arms and/or legs feeling tired or heavy; fatigue (5-point Likert scale)). This latter rating does not assess prior night sleep quality per se, but the extent to which physical symptoms are attributed to poor sleep (i.e., sleep-related "threat" [52]).

Prolonged Activation

Participants were asked to rate cognitive and affective prolonged activation in relation to the stressor task as part of an online survey the evening after completion of the laboratory session. Items included: (1) *To what extent are you thinking about issues discussed in the research session right now* (*before bedtime*)? (2) *To what extent are you distressed by issues discussed in the research session right now* (*before bedtime*)? Each item was rated on a 5-point Likert scale.

Statistical Analysis

To characterize the sample with respect to the sleep measures, means and standard deviations for the prior-month and prior-night sleep measures and the zero-order correlations among them were calculated. Correlations between the sleep measures and baseline affect (NA and PA ratings) and physiology (resting HR and blood pressure) were then calculated. To confirm that the laboratory stress task resulted in expected changes in affect and physiology, paired-sample t tests examined change in these measures. To examine the hypothesized associations between reports of prior-month and prior-night sleep and reactivity to the laboratory stressor, separate regression models predicting affective (NA and PA) and physiological (HR, SBP, and DBP) measures during the stressor, controlling for baseline levels, were conducted. To examine hypothesized associations between prior-month sleep quality and post-stressor prolonged activation and pre-sleep arousal, separate regression analyses with PSQI global scores predicting nighttime ratings of cognitive and affective activation, as well as nighttime ratings of pre-sleep arousal (controlling for pre-sleep arousal ratings for the prior night) were conducted. To reduce the overall number of analyses, associations with prior-month sleep were examined initially using the global PSQI score. For outcomes in which the global PSQI score was a significant predictor, PSQI component scores were examined to characterize the specific aspects of global sleep quality that were associated.

Results

Sleep Quality Descriptive Statistics and Relation to Baseline Measures

The PSQI mean was 6.2, SD=3.7. Because number of hours of sleep per night has been associated with a variety of negative health outcomes, descriptive statistics on the sample were calculated. Thirty-nine percent reported getting less than 7 h of sleep/night on average during the prior month and 13 % reported getting less than 6 h/night. For the prior night, 41 % of the sample reported getting less than 7 h of sleep.

Global PSQI scores were significantly related to baseline ratings of NA and PA, r values=0.28 and −0.24, respectively, p values of <0.05, ratings of the prior night's (i.e., pre-stress) pre-sleep arousal, r=0.57, p<0.05.and marginally associated with resting HR, r=0.17, p=0.10. PSQI global scores were not significantly associated with baseline blood pressure, −0.11>r values of <0.00, p values of >0.10.

With respect to prior-night sleep measures, both the Sleep Associated Monitoring Index scores and State Sleep Quality scores were correlated with baseline NA, r values=0.24 and 0.34, respectively, p values of <0.05, and PA, r values=−0.35 and −0.33, respectively, p values of <0.05. Neither prior-night total sleep time nor prior-night sleep-onset latency was significantly associated with baseline NA or PA, p values of >0.10. None of the prior-night sleep measures were associated with resting physiology, p values of >0.10.

Manipulation Check: Stressor Effects on Physiology and Affect

Means and standard deviations for baseline and stressor (or immediate post-stressor in the case of affect ratings) levels of DBP, SBP, HR, NA, PA, and pre-sleep arousal are shown in Table 1. Paired sample t tests indicated that average HR, DBP, and SBP increased from baseline to the stressor period. Ratings of NA also increased from baseline to stressor and ratings of PA decreased. Finally, pre-sleep arousal ratings on the night following the stressor were significantly higher than the ratings for the prior night, indicating an increase in cognitive and somatic arousal prior to bedtime. As shown in Table 2, cognitive pre-sleep arousal change was associated with ratings of prolonged cognitive and affective activation in relation to the stressor (assessed at bedtime after the lab session), suggesting that the increase in cognitive pre-sleep arousal was, at least in part, attributable to reactions to the laboratory stressor. These findings

Table 1 Means, standard deviations, and paired samples *t* tests for physiological and affective measures prior to and during (physiology measures), immediately after the stressor (NA and PA), or the evening after the stressor (pre-sleep arousal)

	Baseline	Stressor	*t*
DBP	64.5 (8.3)	72.7 (9.7)	11.2*
SBP	114.9 (14.0)	123.9 (15.2)	8.5*
HR	68.8 (10.7)	77.6 (10.8)	12.2*
NA	14.0 (4.7)	15.3 (4.7)	3.2*
PA	27.4 (7.0)	25.3 (7.9)	−4.1*
Pre-sleep arousal	24.2 (8.7)	27.3 (8.3)	3.8*

DBP diastolic blood pressure (in millimeters of mercury), *SBP* systolic blood pressure (in millimeters of mercury), *HR* heart rate (beats per minute), *NA* negative affect, *PA* positive affect

*$p<0.05$

indicate that the stressor served to increase physiological arousal and subjective distress.

Associations Between Sleep Measures, Stress Reactivity, Prolonged Activation, and Pre-sleep Arousal

Data Analytic Approach

To examine the associations between recent sleep measures and stress reactivity, stressor period measures of physiological (HR, SBP, and DBP) and affective (NA and PA) responses were regressed on measures of prior-night and prior-month sleep in separate regression models, controlling for the appropriate baseline levels of each measure. Thus, stress reactivity was operationalized as stressor period physiology and affect controlling for baseline levels. Correlations among all sleep measures and residualized change scores reflecting stress reactivity are shown in Table 2. Regression analyses similarly tested the association between prior-month sleep (PSQI global scores) and change in pre-sleep arousal (i.e., post-stress evening ratings, controlling for ratings of prior night). Because prior-night sleep ratings would be largely confounded with ratings of pre-sleep arousal for the prior night, associations with stress-related change in pre-sleep arousal were not examined; however, correlations between prior-night sleep measures and prolonged activation measures are shown in Table 2.

In instances in which the PSQI global scores were related to a stress outcome, PSQI component scores were examined to clarify which aspects of prior-month sleep were associated. All reported betas are standardized. *t* tests were conducted to examine potential sex differences in stress responses and sleep. There were no sex differences on any stress response measure, *p* values of >0.10. The only statistically-significant difference on sleep measures was on the sleep-associated monitoring index; women reported significantly more physical symptoms related to prior night sleep than did men, $t=-2.09$, $p=0.04$. Given these collective findings, all analyses were conducted collapsed across sex.

Stress Reactivity

As shown in Tables 2 and 3, in regression analyses predicting stress-related change in physiology and affect, PSQI global scores were associated with lower DBP reactivity. Individuals with poorer overall sleep quality evidenced dampened DBP reactivity. Prior-night Total Sleep Time (hours slept in prior night) was (marginally) negatively associated with SBP responses—individuals who slept fewer hours evidenced greater reactivity. In regression analyses examining stress-related change in affect, Sleep-Associated Monitoring Index scores predicted change in negative affect and State Sleep Quality scores predicted change in positive affect. PSQI Global scores predicted change from baseline to stressor in positive affect. There were no other significant associations between either state or prior-month sleep quality and affective or cardiovascular reactivity.

In summary, greater prior-night sleep associated monitoring (i.e., perceived physical symptoms attributed to poor sleep) predicted increases in negative affect. Ratings of poor global sleep quality over the prior month were associated with dampened DBP reactivity and decreases in positive affect. Prior-night sleep quality ratings also predicted decreases in positive affect. Fewer hours slept in the prior night (marginally) predicted higher SBP reactivity.

Pre- to Post-stress Pre-sleep Arousal Change

PSQI global scores significantly predicted post-stress pre-sleep arousal (evening ratings), $\beta=0.32$, $p<0.05$, $\Delta R^2=0.06$, controlling for the pre-sleep arousal ratings for the prior night, $\beta=0.47$, $p<0.0001$. Examination of the Pre-sleep Arousal Scale subscales indicated that this effect was significant for cognitive pre-sleep arousal (see Tables 2 and 3) but not somatic arousal. Thus, individuals who reported poorer overall sleep quality in the prior month evidenced greater increases in nighttime worry and mental alertness following the stressor.

Prolonged Activation

As shown in Table 2, PSQI Global scores were significantly associated with ratings of cognitive and affective prolonged activation to the stressor (i.e., thinking about and distress in relation to the stressor task). With respect to prior-night sleep measures, the Sleep-Associated Monitoring Index and State Sleep Quality scores were positively related to both ratings, and Sleep-Onset Latency was marginally

Table 2 Zero-order correlations among study variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. NA change[a]	–	0.12	0.02	0.03	0.06	0.10	−0.17	−0.15	0.09	0.22*	−0.12	0.03	0.06	−0.00
2. PA change[a]		–	0.00	0.07	0.06	0.11	−0.08	−0.30*	−0.25*	−0.17**	−0.14	0.14	−0.13	−0.09
3. SBP change[a]			–	0.36*	0.16	−0.23*	−0.07	−0.15	−0.07	−0.15	−0.05	−0.17**	−0.08	−0.19**
4. DBP change[a]				–	0.05	−0.02	−0.20**	−0.24*	−0.01	−0.01	−0.19**	0.00	−0.22*	−0.06
5. HR change[a]					–	−0.18**	−0.09	−0.07	0.03	−0.05	−0.04	−0.09	−0.08	0.17
6. PSA change[a] somatic						–	0.30*	0.13	N/A	N/A	N/A	N/A	−0.04	0.17
7. PSA change[a] cognitive							–	0.28*	N/A	N/A	N/A	N/A	0.31*	0.34*
8. PSQI global score								–	0.67*	0.57*	0.49*	−0.09	0.31*	0.37*
9. SSQ total									–	0.72*	0.23*	−0.21*	0.30*	0.40*
10. SAMI total										–	0.15	−0.20*	0.37*	0.42*
11. Prior night SOL											–	0.05	0.20**	0.16
12. Prior night TST												–	−0.13	0.05
13. Nighttime cognition													–	0.62*
14. Nighttime distress														–

NA negative affect, *PA* positive affect, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, *PSA* pre-sleep arousal, *PSQI* Pittsburgh Sleep Quality Index, *SOL* sleep onset latency, *SE* habitual sleep efficiency, *SSQ* state sleep quality, *SAMI* Sleep-Associated Monitoring Index, *TST* total sleep time

*$p<0.05$; **$p<0.10$

[a] Change refers to residualized change scores

Table 3 Significant associations between prior-night and prior-month sleep quality predicting rank order change in physiological and affect responses to stress

	β	*t*	*p*	ΔR^2
DV: DBP-stressor				
DBP baseline	0.77	12.1	<0.0001	
PSQI global	−0.15	−02.4	0.02	0.02
Total R^2	0.61			
DBP baseline	0.77	12.1	<0.0001	
PSQI sleep quality	−0.14	−2.2	0.03	0.02
Total R^2	0.61			
DBP baseline	0.77	12.1	<.0001	
PSQI-sleep disturbances	−0.19	−3.0	0.003	0.03
Total R^2	0.62			
DV: NA-stressor				
NA baseline	63	83	<0.0001	
SAMI-prior night	0.17	2.2	0.03	0.03
Total R^2	0.48			
DV: PA-stressor				
PA baseline	0.78	12.4	<0.0001	
SSQ-prior night	−0.16	−2.6	0.01	0.02
Total R^2	0.70			
PA baseline	0.79	13.1	<0.0001	
PSQI global	−0.18	−3.0	0.003	0.03
Total R^2	0.71			
PA baseline	0.79	13.1	<0.0001	
PSQI sleep quality	−0.17	−2.9	0.004	0.02
Total R^2	0.70			
DV: cognitive PSA post-stress				
Cognitive PSA prior night	0.42	4.2	<0.0001	
PSQI global	0.35	3.5	0.001	0.07
Total R^2	0.48			
Cognitive PSA prior night	0.58	7.1	<0.0001	
PSQI latency	0.18	2.3	0.03	0.03
Total R^2	0.44			
Cognitive PSA-prior night	0.61	7.7	<.0001	
PSQI habitual sleep efficiency	−0.39	−2.3	0.02	0.03
Total R^2	0.44			
Cognitive PSA-prior night	0.50	5.2	<0.0001	
PSQI sleep disturbances	0.22	2.2	0.03	0.03
Total R^2	0.44			

SBP systolic blood pressure, *DBP* diastolic blood pressure, *NA* negative affect, *PA* positive affect, *PSQI* Pittsburgh Sleep Quality Index, *PSA* pre-sleep arousal, *SAMI* Sleep-Associated Monitoring Index, *SSQ* state sleep quality

associated with prolonged cognitive activation. Thus, individuals who reported poorer overall sleep quality in the prior month, greater physical symptoms in relation to the prior night, and poorer sleep quality in the prior night indicated that they were continuing to think about the stressor task and rated themselves as more distressed about the laboratory task before bedtime.

PSQI Component Scales

In order to discern which components of prior-month sleep quality explained the associations of the global score, analyses were repeated using the component scales of the PSQI. Because only a small percentage reported any sleep medication use in the prior month (18 %), that component scale was not examined. With respect to associations with baseline affect and physiology: sleep disturbances, $r=0.23$, $p<0.05$, were related to resting HR; sleep duration, $r=-0.20$, $p<0.05$, sleep quality, $r=0.38$, $p<0.05$, and daytime dysfunction, $r=0.32$, $p<0.05$, were related to baseline NA; sleep disturbances, $r=-0.21$, $p<0.05$, and daytime dysfunction, $r=-0.21$, $p<0.05$, were related to baseline PA.

For stress-related change in positive affect, PSQI Sleep Quality was the only component score that was significantly associated (see Table 3). As shown in Table 3, sleep latency, habitual sleep efficiency, and sleep disturbances were associated with stress-related change in cognitive pre-sleep arousal. With respect to associations with nighttime cognition and distress ratings, respectively, sleep quality, *r* values=0.31 and 0.31; daytime dysfunction, *r* values=0.29 and 0.40; and sleep disturbances, *r* values=0.27 and 0.28, were significantly associated, *p* values of <0.05. Finally, dampened DBP reactivity was associated with sleep quality and sleep disturbances (Table 3).

In summary, stress-related decrease in positive affect was associated with prior-month ratings of sleep *quality*. Change in cognitive pre-sleep arousal was associated with components reflecting poor sleep *continuity* (sleep latency, sleep disturbances, and habitual sleep efficiency) in the prior month. Dampened blood pressure reactivity and prolonged cognitive and affective activation were associated with both *continuity* and *quality*.

Discussion

The current study examined associations between naturally occurring variations in sleep in the prior night and in the prior month on cognitive, affective and physiological responses to a laboratory stressor in young adults. Results indicated that both prior-night and prior-month ratings of sleep duration, continuity, and quality, as well as perception of sleep-related threat (i.e., physical symptoms attributed to prior-night poor sleep) are associated with poorer responses to the laboratory stress induction.

Sleep, Prolonged Activation, and Pre-sleep Arousal Change

Overall, the strongest associations between sleep and stress responses were on increases in cognitive pre-sleep arousal. Prior-month ratings of global sleep quality were associated with increases in cognitive pre-sleep arousal (i.e., mental alertness and worry), as well as nighttime ratings of continued thinking about and distress in relation to the laboratory stressor. Examination of the PSQI component scales indicated that prior-month associations were primarily attributable to poor sleep *continuity* (i.e., sleep latency, sleep disturbances, and habitual sleep efficiency). These findings are noteworthy because pre-sleep arousal has been implicated as a specific vulnerability in the development of chronic sleep disturbance [43] and worry at night is a central component of cognitive models of insomnia [13, 15]. Thus, findings of the current study are consistent with a reciprocal, feed-forward association between stress and sleep: poor sleep may lead to further sleep disruption via prolonged activation (i.e., pre-sleep arousal, perseverative cognition) in response to stressful circumstances. From a component process view of stress, associations between poor sleep and perseverative cognition (e.g., rumination about prior stressors) are consistent with a feedback loop between sleep and stress exposure, since mental re-activation of stress has similar physiological sequelae as direct exposure to external events.

Sleep and Affective Reactivity to Stress

Results of the current study also suggest that aspects of both prior-night and prior-month sleep were associated with poorer affective responses to stress. *Sleep-associated monitoring* ratings (i.e., physical symptoms attributed to poor sleep) for the prior night were related to stress-related increases in negative affect. These findings add to prior research indicating that, among individuals with insomnia, monitoring for sleep-related threat increases negative thoughts throughout the day and serves to maintain sleep difficulties [25]. Results of the current study suggest that monitoring for physical signs of poor sleep may also be detrimental *prior to* the development of chronic sleep disturbance.

Greater decreases in positive affect during laboratory stress induction were associated with ratings of poorer sleep *quality* for both the prior month and for the prior night. These findings suggest that the subjective perception of poor sleep quality (i.e., nonrestorative sleep) is associated with a lack of engagement (e.g., decreased interest, enthusiasm, determination, and attentiveness). Although associations between sleep and PA reactivity to stressors have not been studied previously, the current findings are consistent with recent research indicating less positive emotion expressiveness in sleep-deprived individuals [53]. Although less considered in stress reactivity literature, lack of engagement, particularly low motivation and fatigue, has important implications for self-regulation broadly [54] and positive affect may improve self-regulatory abilities under conditions of depletion [55], suggesting that changes in PA may be important in overall stress regulation. Because low positive affect is a defining component of depressed mood, current findings may also add to our understanding of the prospective association between sleep disturbance and depression [56], with inflammation potentially serving as a mediating pathway [39].

Sleep and Cardiovascular Reactivity to and Recovery from Stress

With respect to cardiovascular responses to stress, prior-month global sleep ratings (especially sleep quality, daytime dysfunction, and sleep disturbances) were associated with dampened DBP reactivity. Although this finding is counter to initial prediction, it is consistent with prior findings of poor sleep associations with dampened HPA-axis responses to laboratory stress [36] and with the recent conceptualization of low cardiovascular reactivity as an indicator of homeostatic imbalance [38]. On the other hand, individuals who slept fewer hours in the prior night (i.e., poorer sleep *duration*) evidenced modestly higher SBP reactivity. Current findings are consistent with prior research indicating that sleep duration and quality should be examined separately in relation to stress responses (e.g., [57]). In addition, reported sleep disturbances in the prior month (i.e., frequency of nighttime awakenings) were associated with higher resting HR. Although the effect sizes indicate relatively minor shifts in cardiovascular stress responses, repeated over time, they may confer risk for poor health outcomes [40]. For example, greater blood pressure reactivity is associated with risk of cardiovascular disease [58–60]. Indeed, poor sleep duration and continuity are associated with adverse changes in blood pressure over 5 years in adults [61].

Prevalence of Inadequate Sleep in Young Adults

Although there are individual differences in the optimal number of hours of sleep, guidelines from the National Sleep Foundation suggest that most adults should get 7–9 h of sleep/night [62] and research indicates a cumulative effect of getting 6 or fewer hours of sleep/night on cognitive functioning [30]. Notably, more than a third of the young adults in the current study indicated that they got less than 7 h of sleep, on average, in the prior month (39 %). Furthermore, a relatively high proportion (41 %) reported getting less than 7 h of sleep in the prior night, with the physical effects evident in greater blood pressure reactivity to stress. These findings are consistent with recent reports from the Centers for Disease Control indicating that approximately 35 % of adults get less than 7 h of sleep [63],

indicating that insufficient sleep is a major public health concern.

Summary of Findings

Overall, current findings build on recent research examining experimentally-induced sleep deprivation and emotional states (e.g. [27]). In general, there appears to be both an immediate and a cumulative effect of naturally occurring poor sleep on cognitive, affective, and physiological responses to stress. Indeed, the combination of dampened blood pressure responses and decreases in positive affect among individuals who reported poor sleep in the prior month are consistent with homeostatic imbalance and lack of engagement, suggesting potential difficulties with self-regulation, generally. Furthermore, individuals who are already experiencing poor sleep continuity (i.e., difficulty initiating and maintaining sleep) may show increases in cognitive pre-sleep arousal, even when baseline levels are controlled. Although reciprocal effects were not tested in the current study, these findings are consistent with models of stress regulation positing that sleep (i.e., stress *restoration*) and the other stress processes (i.e., *exposure*, *reactivity*, and *recovery*) may interact in a feed-forward fashion to place vulnerable individuals on a trajectory of increasingly disturbed sleep and, potentially, negative mental and physical health outcomes.

Limitations, Conclusions, and Future Directions

The current study provides important evidence for the association between poor sleep and stress responses. Because prior research suggests that subjective reports of sleep and objective indicators may differentially predict important outcomes [24], generalization of current findings should be limited to self-reported sleep dimensions. Self-reported sleep dimensions are important to examine—subjective perceptions of poor sleep, regardless of objective indicators, figure prominently in the development of insomnia; furthermore, there is no agreed-upon objective method for assessing "restorative" sleep. Nevertheless, extending the current research to objective assessment of sleep (e.g., actigraphy and polysomnography) is an important future direction. In addition, there are individual differences in the effects of poor sleep (e.g., [21]); thus, examination of potential moderators of the reported sleep-stress regulation associations, such as personality factors and cognitive functioning, will be an important future direction.

Future research using daily sampling methodology that includes sleep measures will allow for direct testing of hypothesized reciprocal associations between stress and sleep (see [34] for a recent example) and would confirm that the associations between poor sleep and subsequent changes in pre-sleep arousal are attributable to response to daily stress. Given the strong association between recent sleep continuity and stress-related increases in pre-sleep arousal, future research on this construct seems particularly important. Findings from the current study should be also placed in developmental context. The sample was young, presumably healthy, and potentially "choosing" to sleep less because of academic and social demands—the negative effects of sleep disruption, particularly effects on physiological responses, may be more pronounced in older adults and individuals with existing sleep disorders.

In conclusion, individuals who reported recent poor sleep evidenced poor cognitive, affective, and physiological responses to stress, particularly increases in cognitive pre-sleep arousal—a key vulnerability factor in the development of chronic sleep disturbance. Given the health consequences of poor sleep, targeted interventions which operate to break this cycle before sleep disturbance becomes chronic seem particularly important.

Conflict of Interest Statement The authors have no conflict of interest to disclose.

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