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NATURALISTIC PARTIAL SLEEP DEPRIVATION LEADS TO GREATER NEXT-DAY

ANXIETY: THE MODERATING ROLE OF BASELINE ANXIETY AND DEPRESSION

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The detrimental effects of sleep deprivation on emotional functioning have been well-established. In particular, insufficient sleep has been linked to both anxiety and depression. At the daily level, total sleep deprivation usually leads to increased anxiety and depressive symptoms the following day. Although partial sleep deprivation has been suggested to have detrimental effects similar to that of total sleep deprivation, relatively few studies have examined the relationships between partial sleep deprivation and next-day symptoms of anxiety and depression in everyday life, which this study sought to characterize. Participants ($N = 94$) completed daily diary surveys twice per day for two weeks without instructions to alter their sleep in any way. Nights of spontaneous, naturally-occurring partial sleep deprivation were followed by increased levels of self-reported symptoms of anxiety the next day, but were unrelated to next-day symptoms of depression or general distress. The relationship between partial sleep deprivation and next-day anxiety was found to be moderated by both baseline depressive symptoms and anxiety such that individuals reporting higher levels of depression or anxiety at baseline showed relatively greater increases in symptoms of anxiety following partial sleep deprivation. These results suggest that partial sleep deprivation occurring in everyday life

can lead to higher levels of next-day anxiety, a relationship that is particularly deleterious for individuals with higher overall levels of anxiety or depressive symptoms.

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Introduction

Psychological research has repeatedly confirmed that receiving an adequate amount of sleep each night is critical for optimal human functioning. For example, in a comprehensive meta-analytic review, Pilcher and Huffcutt (1996) found that sleep deprivation adversely impacts mood and cognitive and motor responses, with the greatest effect on mood. Sleep deprivation has also been linked to impaired decision-making abilities (Harrison & Horne, 2000), increased physiological arousal (Bonnet & Donna, 2006), emotional dysregulation (Kahn, Sheppes, & Sadeh, 2013), and a reduced ability to experience positive mood and emotions coupled with an increased susceptibility to negative emotional experiences (Franzen, Siegle, & Buysse, 2008; Minkel et al., 2012; Palmer & Alfano, 2017; Paterson et al., 2011). Given the link between sleep and emotional health, it comes as no surprise that sleep disturbances are strongly associated with both anxiety and depression (Jackson, Sztendur, Diamond, Byles, & Bruck, 2014; Hertenstein et al., 2019; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). While generally considered symptoms of mood disorders (American Psychiatric Association, 2013), sleep disturbances have been linked to worse clinical outcomes in depression (Buysse et al., 1999). Furthermore, they have also been shown to persist following remission of a depressive episode and predict depressive recurrence (Perlis, Giles, Buysse, Tu, & Kupfer, 1997), highlighting the importance of understanding the mechanisms of disrupted sleep in relation to psychopathology.

Longitudinal studies examining the specific relationships between insufficient sleep, anxiety, and depression over time have produced conflicting findings. Some studies have found chronic sleep deprivation to predict the later development of anxiety disorders (Neckelmann, Mykletun, & Dahl, 2007; Roberts & Duong, 2017), while others have found that anxiety disorders predicted later insomnia but not vice-versa (e.g., Johnson, Roth, & Breslau, 2006). Insomnia has generally been shown to be a significant risk factor for the development of later depression (see Baglioni et al., 2011 for a meta-analysis; Riemann & Voderholzer, 2003; Szklon-Coxe, Young, Peppard, Finn, & Benca, 2010), although depressive symptoms have also been found to be more likely to precede symptoms of insomnia (Ohayon & Roth, 2003). Still other studies have argued for a bidirectional relationship between insomnia and mood disorders, in which disrupted sleep increases the risk for developing subsequent anxiety or depression, and the presence of anxious or depressive symptoms increases risk for later insomnia (Buysse et al., 2008; Jansson-Fröjmark & Lindblom, 2008).

In order to better understand these complex relationships, researchers have increasingly turned to examining the interplay between sleep and mood on a daily level. For example, Fuligni and Hardway (2006) found that receiving less sleep at night predicted higher levels of anxiety, depressed mood, and fatigue the following day in a sample of adolescents. Similarly, lower total sleep time relative to the person's average has been shown to predict greater anxiety the following day (Cox, Sterba, Cole, Upender, & Olatunji, 2018). Recent evidence suggests that the relationships between sleep disturbance, anxiety, and depression may be bidirectional (Peltz, Rogge, Pugach, & Strang, 2017; Kalmbach, Arnedt, Swanson, Rapier, & Ciesla, 2017). Interestingly, Kalmbach and colleagues (2017) found that the influence of total sleep time on depressive symptoms was complex, such that a single night of short sleep led to less anhedonic

depression the next day, whereas women who obtained shorter sleep across a two-week period reported greater anhedonia. Thus, it is likely that the nature of the relationships between sleep, anxiety, and depression may depend in part on the timescale over which they are observed.

The tripartite model of anxiety and depression (Clark & Watson, 1991) has been successfully used as a theoretical tool to parse out the differential relationships between sleep disturbance, anxiety, and depression on a daily level (e.g., Babson, Trainor, Feldner, & Blumenthal, 2010; Kalmbach et al., 2017). The tripartite model posits that anxiety and depression share a core set of symptoms contributing to general affective distress such as feeling hopeless, depressed, or uneasy but can be distinguished by a set of symptoms specific to each disorder. Depression can be uniquely characterized by symptoms pertaining to low positive emotions and a lack of interest, while anxiety is characterized by symptoms of physiological hyperarousal such as psychomotor agitation and shortness of breath. Longitudinal studies have suggested a degree of specificity between anxiety and depression in their relationships with impaired sleep, which the tripartite model is especially suited to teasing apart on the daily level.

Historically, much attention has been given to the effects of total sleep deprivation on emotional functioning the following day, almost exclusively in a laboratory or experimental setting. Such deprivation usually presages increases in next-day symptoms of anxiety and depression (Babson et al., 2010; Franzen et al., 2008; Paterson et al., 2011). However, highly depressed individuals often report an antidepressant effect of total sleep deprivation wherein their mood improves the day after sleep deprivation (Pflug & Tolle, 1971; Wirz-Justice & Van den Hoofdakker, 1999). Thus, paradoxically, chronic insufficient sleep predicts and exacerbates depression (Buysse et al., 1999; Perlis et al., 1997), yet a single night of acute sleep loss leads to a temporary improvement in depressive symptoms the following day. Unfortunately, the

antidepressant properties of acute sleep deprivation are generally lost after the next instance of sleep (Wirz-Justice & Van den Hoofdakker, 1999).

The temporary antidepressant effect of total sleep deprivation is not well understood, but it serves to highlight the importance of studying anxiety and depression separately in relation to sleep, as there does not appear to be any condition under which lack of sleep reliably has anything other than a detrimental effect on anxiety (Wirz-Justice & Van den Hoofdakker, 1999). The antidepressant effect of sleep deprivation has also been found in depressed inpatients who spent 4.5 hours in bed (Giedke, Klingberg, Schwärzler, & Schweinsberg, 2003; Neumeister et al., 1996), and it has been suggested that partial sleep deprivation has deleterious effects comparable to that of total sleep deprivation (Pilcher & Huffcutt, 1996). Thus, it is critical to also examine the effects of partial sleep deprivation on next-day symptoms of anxiety and depression, especially given that a night of short sleep in which an individual sleeps for only a few hours is intuitively more likely to occur in everyday life than forgoing sleep entirely.

Aims and Hypotheses

The present study sought to examine the relationship between naturally occurring (i.e., unmanipulated) partial sleep deprivation and next-day symptoms of anxiety and depression in an undergraduate sample using a daily diary design. Total sleep deprivation, being the complete lack of sleep, is easy to define, but partial sleep deprivation has proven to be a more nebulous concept, without an established standard. However, a variety of literatures assessing the effects of partial sleep deprivation on next-day drowsiness and cognitive, emotional, physical, and metabolic functioning have converged on a widely accepted definition of partial sleep deprivation as a night during which participants received 4 hours of sleep or less (e.g., Åkerstedt et al., 2010; Brondel, Romer, Nougues, Touyarou, & Davenne, 2010; Donga et al., 2010;

Gosselin, De Koninck, & Campbell, 2017; McMakin et al., 2016; Schmid et al., 2009; Schwarz et al., 2013; Spiegel, Tasali, Penev, & van Cauter, 2004). In line with convention, partial sleep deprivation was operationalized in this study as a night for which participants reported receiving 4 hours of sleep or less.

The use of a cut-off of 4 hours of sleep or less, as opposed to determining the occurrence of partial sleep deprivation by some deviation below the participant's average number of hours slept across the study, offers the additional advantage of isolating the effects of insufficient sleep on next-day affective experience. Given that depression can be accompanied by insomnia, hypersomnia, or both (Geoffroy et al., 2018), depressed individuals who suffer from hypersomnia may routinely sleep for 10 hours or more per night. For such an individual, a definition of sleep deprivation based on less sleep than average might consider a night of 7 or 8 hours of sleep to be an instance of partial sleep deprivation, although that is generally regarded as an optimal amount of sleep for young adults and adults (Hirshkowitz et al., 2015).

It was hypothesized that, following a night of self-reported partial sleep deprivation, participants would report higher levels of next-day anxious arousal, anhedonic depression, and general distress. It was further hypothesized that these relationships would be moderated by baseline levels of anxiety and depression such that individuals who report higher overall levels of anxiety or depression at baseline will report a greater increase in anxiety and general distress relative to their peers. Further, individuals with higher levels of anxiety or depression were additionally hypothesized to experience less of an increase in anhedonic depression following partial sleep deprivation. In other words, greater levels of baseline anxiety and depression were posited to potentiate the effect of partial sleep deprivation on next-day symptoms of anxiety and general affective distress and dampen its effect on next-day depressed mood given findings of an

improvement in mood following partial sleep deprivation in highly depressed individuals (e.g, Giedke et al., 2003).

Method

Participants and Procedure

Ninety-four undergraduate students between the ages of 18 and 34 ($M = 19.60$, $SD = 1.99$, 77.7% female) were recruited from psychology courses at a large, Midwestern university. Undergraduates are especially suited for an investigation into the relationship between partial sleep deprivation and next-day anxiety and depression, as they are a population characterized by high rates of comorbid symptoms of depression and insomnia (Gress-Smith, Roubinov, Andreotti, Compas, & Luecken, 2015), irregular sleep-wake patterns, and insufficient sleep (Lund, Reider, Whiting, & Prichard, 2010). Study participants were 87.2% Caucasian/White, 7.5% African American/Black, 3.2% Multiracial, 1.1% Hispanic/Latino, and 1.1% Other. Broken down by year in school, participants were 41.5% Freshmen, 27.7% Sophomores, 14.9% Juniors, 14.9% Seniors, and 1.1% Other. Fourteen participants reported having taken a prescribed antidepressant during the past month. Rule-out criteria included a self-reported history of bipolar mood disorders and lack of a smartphone on which to access the daily, online questionnaires. Two individuals were excluded from all analyses due to self-reporting a history of bipolar mood disorders. In order to ensure adequate variability in depressive symptoms, oversampling was used to recruit participants who took the Center for Epidemiologic Studies Depression Scale Revised (CESD-R; Eaton, Smith, Ybarra, Muntaner, & Tien, 2004) administered as part of the university's mass testing protocol for undergraduate psychology students and scored above the cut-off score of 16, which indicates possible clinical significance and elevated risk for depression (Radloff, 1977). Based on their CESD-R scores, 131 potential participants were invited to

participate, 16 of which were enrolled in the study. Analyses comparing the 16 participants who were invited and included in the final sample to individuals who were invited but declined to participate in the study showed no significant differences in CESD-R scores, gender, age, or year in school (all p 's > .13). The remainder of the study sample consisted of unselected undergraduates ($n = 78$).

Once informed consent was provided, participants completed a battery of baseline questionnaires and were asked to complete daily questionnaires that were sent to their smartphones twice per day for the next 14 days. The morning surveys were sent at 6 am with instructions to complete as soon as possible after waking up, while the evening surveys were sent at 8 pm with instructions to complete approximately 2 hours before they planned to go to bed for the night. Participants were compensated with course credit, as well as an entry into a raffle to win one of eight \$25 gift cards for every daily survey they completed, up to a maximum of 28 entries. The study received ethics approval the university Institutional Review Board.

Baseline Measures

Depressive Symptoms. The Center for Epidemiologic Studies Depression Scale Revised (CESD-R; Eaton et al., 2004) is a 20-item self-report inventory that asks participants to rate how frequently in the past two weeks they have experienced depressive symptoms, both physical (e.g., "My appetite was poor") and emotional (e.g., "I could not shake off the blues"), on a scale of 0 (not at all or less than 1 day last week) to 3 (nearly every day for two weeks). Possible scores on the CESD-R range from zero to 60. The CESD-R has been shown to possess high internal consistency and theoretically consistent convergent and divergent validity (Van Dam & Earleywine, 2011). All study analyses utilized the CESD-R scores gathered at baseline; the CESD-R scores produced during mass testing were used for the purposes of oversampling only.

The final study sample included 49 participants scoring at or above the clinical cut-off score of 16 (Radloff, 1977), 22 of whom scored at or above the more conservative proposed cut-off score of 27 (Gotlib, Lewinsohn, & Seeley, 1995). In the current study, the CES-D demonstrated excellent internal consistency ($\alpha = 0.92$).

Anxiety. The State-Trait Anxiety Inventory Form X – State (STAI-XS; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1970) is a 20-item self-report inventory intended to assess levels of anxiety. Participants indicate their agreement with statements describing current symptoms of anxiety (e.g., “I feel tense”) on a scale from 1 (not at all) to 4 (very much so). Possible scores on the STAI-XS range from 20 to 80, with scores of 54 and higher indicating clinically significant anxiety. In this sample, 12 participants scored above the clinical cut-off. The State subscale of the STAI-X was used due to findings that the Trait subscale measures nonspecific general negative affect (Balsamo et al., 2013). The STAI-XS has demonstrated acceptable convergent and discriminant validity (Fydrich, Dowdall, & Chambless, 1992). In the current study, the STAI-XS also demonstrated excellent internal consistency ($\alpha = 0.93$).

Daily Measures

Anhedonic Depression, Anxious Arousal, and General Distress. The evening survey included the Mini Mood and Anxiety Symptom Questionnaire (Mini-MASQ; Casillas & Clark, 2000), a 26-item self-report measure of depression and anxiety that is an abbreviated version of the original, 90-item MASQ (Watson, Clark, et al., 1995; Watson, Weber, et al., 1995) modified to ask about experiences during the past day. Three main factors assess general distress (i.e., overlapping features of depression and anxiety; e.g., “felt keyed up, on edge,” “felt depressed”), anxious arousal (i.e., anxiety-specific symptoms; e.g., “was short of breath,” “was trembling or shaky”), and anhedonia (i.e., depression-specific symptoms; e.g., “felt like I had a lot to look

forward to,” “felt really happy” (both reverse coded)). Participants respond to each item with an assessment of how much they have felt or experienced each that day on a scale from 1 (not at all) to 5 (extremely). The general distress and anhedonic depression subscales are made up of 8 items each, while the anxious arousal subscale is composed of 10 items. Thus, possible scores on the general distress and anhedonic depression subscales range from 8 to 40, while possible scores on the anxious arousal subscale range from 10 to 50. The original MASQ has been validated for use in students and nonclinical samples (Watson, Clark, et. al, 1995) and has been shown to have good convergent and discriminant validity (Keogh & Reidy, 2000). The Mini-MASQ has also shown good convergent and discriminant validity in young adult populations (Lin et al., 2014).

In order to account for the nested structure of the daily diary data, a three-level unconditional, multilevel model was created, with items nested within days, and days nested within person (Nezlek, 2017). This allowed the calculation of both within-person and between-person reliability estimates. The within-person reliability estimate is the functional equivalent of a Cronbach’s alpha that takes the nested structure of the data into account by estimating the consistency of responses across the items of each subscale. The between-person reliability estimate does not assess internal consistency, but rather provides an index of how reliable the mean response is for each individual within the sample. At the between-person level, the Mini-MASQ demonstrated good reliability for the general distress ($\alpha = 0.85$) and anxious arousal ($\alpha = 0.75$) subscales and acceptable reliability for the anhedonic depression subscale ($\alpha = 0.63$). At the within-person level, internal consistency for the general distress subscale was good ($\alpha = 0.80$), although internal consistency was relatively low for the anxious arousal ($\alpha = 0.55$) and anhedonic depression ($\alpha = 0.40$) subscales. However, Nezlek (2017) notes that within-person reliability “tends to be lower (often much lower)” (p. 152) than between-person reliability, and

suggests relatively relaxed standards for evaluating the within-person reliability of a scale due to the tendency for scales administered daily to have fewer items than trait-level scales and the greater ability of multilevel modeling to take unreliability into account than traditional OLS regression.

Sleep Items. Sleep duration was assessed in the morning survey with the item, “How many hours of sleep did you get last night?” drawn from the Pittsburgh Sleep Quality Index (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989) and modified to assess the prior night. Participants responded to the item via an interactive slider on a scale from 0 to 16 hours reported to the nearest tenth of an hour. Participants were also asked if they had taken a nap during the previous day. In order to account for the possible influence of substance use on sleep, participants also reported the total number of alcoholic drinks they had consumed and the total number of times they had used tobacco or cannabis during both the past 24 hours overall and specifically in the 4 hours before getting in bed. They further reported the number of caffeinated beverages they had consumed in the 4 hours before getting in bed. All questions assessing substance use were delivered during the morning survey in order to capture any use that may have occurred after participants had completed the evening survey but before they went to bed for the night.

Results

Preliminary Analyses

We first explored whether reported amount of sleep was associated with demographic variables, baseline anxiety or depressive symptoms, or substance use. The average number of hours slept per night across the study was 7.52 ($SD = 1.07$), which was unrelated to age, gender, ethnicity, year in school, baseline anxiety or depressive symptoms, and average alcohol, nicotine, or cannabis use (all p 's $>.051$). However, average caffeine use ($r = -.28, p < .01$) and taking naps ($r = -.30, p < .01$) predicted significantly lower average hours of sleep, while antidepressant use predicted significantly higher average hours of sleep ($r = .34, p < .001$). Participants completed an average of 24.33 of the online diaries ($SD = 4.41$, 86.89% compliance), which was unrelated to average hours of sleep per night, demographic variables, or baseline anxiety or depressive symptoms (all p 's $>.41$). Additionally, the associations between demographic variables and average levels of anxious arousal, anhedonic depression, and general distress were examined, and no relationship was found (all p 's $>.14$). Bivariate correlations between average number of hours slept per night and all affective indices are presented in Table 1.

We next examined how frequently participants reported experiencing a night of partial sleep deprivation, operationally defined as 4 hours of sleep or less. Thirty-four participants reported experiencing at least one night of partial sleep deprivation during the study (11 of whom reported 2 or more nights). A total of 57 nights of partial sleep deprivation was reported, representing 4.9% of all observations.

Outliers were handled conservatively by taking them in. First, data points greater than 3 standard deviations above the group mean were identified, and then data points within that subset that were also greater than 3 standard deviations above the person mean were replaced with a value equal to three standard deviations above the person mean. Across the entire dataset, only 1 observation of daily anhedonic depression was adjusted in this way.

Data Analytic Plan

Multilevel modeling was used to account for the time-nested structure of the daily data, with repeated observations nested within persons. Multilevel modeling has the additional advantage of being robust to missing data, which is common in daily diary studies (Singer & Willett, 2003). The indices of the Mini-MASQ (anxious arousal, anhedonic depression, and general distress) represented the dependent variables in these models, all of which were estimated using restricted maximum likelihood approximation. A two-level (time within person) multilevel model with no predictors was created in order to estimate the intraclass correlation coefficient, which estimates the degree to which the outcome variable is truly nested. This model revealed that 58.1% of the variability in daily anxious arousal scores was due to between-subjects differences and 41.9% was due to within-subjects differences. Similarly, only 49.9% of the variability in daily anhedonic depression scores and 54.2% of the variability in general distress scores was due to between-subjects differences, justifying the use of a multilevel analytic framework.

The specific hypotheses of the study involve the relationship between partial sleep deprivation, operationalized as a night of 4 hours of sleep or less, and next-day anxious arousal and anhedonic depression. Within the multilevel model framework, each individual conceptually has a separate Level 1 equation fitted to their data that estimates an intercept (π_{0j}) and a slope

determined by the relationship between each Mini-MASQ subscale and the day-level predictor of sleep deprivation (π_{1j}). These coefficients can also be predicted by person-level variables such as baseline symptoms of anxiety. For example, in the following equation, a significant effect for β_{10} would indicate that daily levels of anxious arousal are significantly predicted by whether the previous night was one of partial sleep deprivation, while a significant effect for β_{11} would signify that the relationship between partial sleep deprivation and next-day anxious arousal is moderated by overall levels of anxiety assessed at baseline.

$$\text{Level 1: } MASQ_AA_{ij} = \pi_{0j} + \pi_{1j}(\text{Sleep Deprivation}_{t-1j}) + e_{ij}$$

$$\text{Level 2: } \pi_{0j} = \beta_{00} + \beta_{01}(STAIXS_j) + r_{0j}$$

$$\pi_{1j} = \beta_{10} + \beta_{11}(STAIXS_j)$$

In order to test for the possibility of participant reactivity, which occurs when repeated measurements of a variable influence the participant's responses over time, models were run in which time was a predictor of the daily Mini-MASQ subscales. These analyses revealed that time was a significant predictor of anxious arousal ($\pi_{1j} = -.066, z = -3.94, p < .001, 95\% \text{ CI } [-.099, -.033]$) and general distress ($\pi_{1j} = -.130, z = -4.47, p < .001, 95\% \text{ CI } [-.187, -.073]$) but not anhedonic depression ($\pi_{1j} = .065, z = 1.92, p = .054$). Thus, there was a trend within the sample as a whole for anxious arousal and general distress scores to decrease over the course of the assessment period. In order to account for these trends, time of measurement was entered into all following analyses as a covariate.

Main Analyses

The next set of analyses examined the prediction of anxious arousal, anhedonic depression, and general distress following a night of naturally occurring partial sleep deprivation. In addition to time, the following covariates were included in these models: baseline depressive

and anxious symptoms, gender, the previous day's level of the dependent variable, whether the participant had napped the previous day, antidepressant use, and before-bed caffeine use.

Multilevel modeling revealed that anxious arousal was significantly higher the day following a night of partial sleep deprivation ($\pi_{1j}=.944$, $z=2.40$, $p=.016$, 95% CI [.174, 1.71]). Furthermore, this relationship was found to be moderated by both baseline anxiety ($\beta_{1j}=.084$, $z=2.74$, $p=.006$, 95% CI [.024, .144]) and baseline depressive symptoms ($\beta_{1j}=.087$, $z=3.28$, $p=.001$, 95% CI [.035, .139]). Thus, partial sleep deprivation was followed by relatively higher next-day anxious arousal for individuals reporting a greater number of anxious and depressive symptoms.¹

The form of these interactions was probed at one standard deviation above and below the mean for both baseline anxiety and baseline depressive symptoms. At one standard deviation above the mean of baseline anxiety, the effect of partial sleep deprivation on next-day anxious arousal was highly significant ($\beta_{1j}=1.90$, $z=3.63$, $p<.001$, 95% CI [.873, 2.92]). In contrast, at one standard deviation below the mean of baseline anxiety, the relationship between partial sleep deprivation and next-day anxious arousal was nonsignificant ($\beta_{1j}=-.088$, $z=-0.16$, $p=.87$; see Table 2 and Figure 1). Similarly, partial sleep deprivation was a significant predictor of anxious arousal the following day at one standard deviation above the mean of baseline depressive symptoms ($\beta_{1j}=1.76$, $z=3.81$, $p<.001$, 95% CI [.855, 2.67]) but was a nonsignificant predictor at one standard deviation below the mean ($\beta_{1j}=-.386$, $z=-0.69$, $p=.49$; see Table 3 and Figure 2).

Finally, the relationships between partial sleep deprivation and next-day anhedonic depression and general distress were examined. Unlike anxious arousal, neither anhedonic

¹ To explore the robustness of these effects, analyses were repeated with alternative partial sleep deprivation cut-offs of 5 or 3 hours of reported sleep. These results were replicated in significance and direction using the more lenient cut-off of 5 hours and became marginally significant using the more stringent cut-off of 3 hours. Additionally, the main effects held even if analyses were restricted only to nights of partial sleep deprivation (defined using the 4-hour cut-off) preceded by a night of at least 6 hours of sleep, although the interaction with baseline anxiety became marginally significant.

depression ($\pi_{1j}=.163$, $z=0.20$, $p=.843$) nor general distress ($\pi_{1j}=.024$, $z=0.04$, $p=.972$) were significantly different following a night of partial sleep deprivation. Further, neither of these relationships were moderated by either baseline anxiety or baseline depressive symptoms.

Discussion

The present study aimed to characterize the relationships between naturally-occurring partial sleep deprivation and next-day symptoms of anxiety and depression in a non-clinical sample using a 14-day daily diary design. The results were partially supportive of hypotheses; participants reported higher levels of anxious arousal the day after partial sleep deprivation, as predicted, although there were no significant differences in levels of anhedonic depression or general distress. Furthermore, as predicted, the relationship between partial sleep deprivation and next-day anxious arousal was moderated by both baseline anxiety and baseline depressive symptoms. Greater overall levels of anxious or depressive symptoms were associated with a sharper increase in anxious arousal the day following partial sleep deprivation.

One of the study strengths was the ability to tease apart the unique relationships between partial sleep deprivation and symptoms specific to anxiety and depression as posited by the tripartite model of anxiety and depression (Clark & Watson, 1991). Partial sleep deprivation led to increased levels of anxiety-specific symptoms such as physiological hyperarousal and somatic tension but did not show a significant increase in general distress, which captures the affective components shared between anxiety and depression, such as feeling “keyed up,” on edge, or uneasy. Thus, it may be that partial sleep deprivation, as it occurs naturally in ordinary life, is most strongly related to the physical symptoms of anxiety. In support of this hypothesis, Babson, Feldner, Trainor, and Smith (2009) found that a single night of total sleep deprivation led to an increase in the intensity of next-day physical, but not cognitive, symptoms of panic, suggesting

that physical symptoms of anxiety may be relatively more sensitive to the effects of acute sleep loss.

In addition to establishing a relationship between naturally-occurring partial sleep deprivation and next-day anxious arousal, this study was also the first to find the relationship was moderated by both baseline anxiety and baseline depressive symptoms. These results suggest that anxious or depressed individuals may be especially susceptible to the deleterious effects of sleep loss. Such individuals possess cognitive (e.g., Beck & Clark, 1988) and biological (e.g., Sham et al., 2000) vulnerabilities to experiencing anxiety and depressed mood on a daily basis that may be exacerbated by partial sleep deprivation. Thus, it may be that a lack of adequate sleep in individuals already predisposed to experiencing anxiety may further increase their risk, while individuals without these predispositions may be less impacted by acute sleep disruptions. It is possible that this represents one mechanism by which sleep disturbances and sleep loss may develop into clinical levels of anxiety and depression later in life, as occasional nights of short sleep may lead to an increase in anxious symptoms, which may in turn magnify the anxiolytic effects of further sleep restriction. Longitudinal research is needed to establish the role of partial sleep deprivation in the development of clinical syndromes over time.

The finding that partial sleep deprivation is especially damaging for individuals already predisposed to experience anxiety and depressed mood holds important implications for clinical interventions. While improving sleep is not typically a focus of traditional cognitive-behavioral therapies for anxiety and/or depression (Clarke et al., 2015), these results suggest that receiving regular, adequate sleep is crucial for all individuals suffering from anxiety and/or depression, even if sleep disturbances are not a primary complaint. Practitioners would do well to consider the importance of receiving adequate sleep in reducing risk for the development of physical

symptoms of anxiety in all their clients, but especially those with clinically significant levels of anxiety or depression. Failure to ensure clients are maintaining a regular sleep schedule may sabotage other efforts to improve anxiety.

The daily diary design employed in this study offers several advantages over laboratory studies in the assessment of the effects of partial sleep deprivation. Participants were allowed to go about their lives without any alterations to their routines or having their sleep manipulated in any way by researchers, maximizing ecological validity. Most laboratory sleep studies require participants to spend the night in a new environment, often connected to several measurement devices if polysomnography is being used, and afford relatively few measurement instances (generally a few days or less). Such studies must contend with the possibility of experiencing a first night effect, which refers to the tendency for participants to sleep worse during the first night of a laboratory sleep study (Agnew, Webb, & Williams, 1966; Hauri & Olmstead, 1989; Riedel, Winfield, & Lichstein, 2001). Experiencing a first night effect has been posited to be related to spending the night in a new environment and is associated with higher levels of depression and anxiety (Hauri & Olmstead, 1989; Riedel et al., 2001), making it an especially problematic phenomenon when assessing anxious or depressed persons. Because participants are allowed to sleep in their own beds and follow their usual routines, the risk of experiencing a first night effect is reduced, and the potential ability of such effects to distort the true relationship between amount of sleep and symptoms of anxiety and depression are diminished by obtaining up to 14 observations for each individual across the study period. Furthermore, daily diary studies likely attract a more representative sample, as completing a survey assessing the prior night's sleep is less daunting and disruptive than spending an entire night or more inside a laboratory.

The lack of a significant relationship between partial sleep deprivation and next-day depression-specific symptoms, such as low positive affect, was surprising given previous studies that have shown experimental, total sleep deprivation to lead to reduced positive affect in healthy populations (Franzen et al., 2008; Paterson et al., 2011) and increased positive affect in depressed populations (Pflug & Tolle, 1971; Wirz-Justice & Van den Hoofdakker, 1999). Two studies have used the tripartite model of anxiety and depression (Clark & Watson, 1991) to assess the effects of sleep duration on anxious arousal, anhedonic depression, and general distress, as was done in this study. Babson and colleagues (2010) found that all three constructs increased in a non-clinical sample the day after experimental total sleep deprivation, while Kalmbach and colleagues (2017) observed a decrease in anhedonic depression on days following relatively shorter sleep duration in a daily diary study, also using a non-clinical sample.

It is possible that the lack of significant relationships between partial sleep deprivation and anhedonic depression or general distress as expected based on theory and past research can be explained by our use of an observational, daily diary design that emphasizes general patterns of sleep over time. Studies which utilize laboratory sessions to induce sleep deprivation may produce larger (or even different) effects, but the external validity of such studies is debatable. Intentional partial sleep deprivation is a fairly rare phenomenon, and even in an undergraduate sample known to sleep less than other groups (Lund et al., 2010), only 4.9% of all nights observed in this study qualified as instances of partial sleep deprivation (defined as 4 hours of sleep or less). Thus, it may be that findings observed in sleep deprivation laboratory studies simply do not generalize to real world experiences of sleep deprivation. It is also possible that the lack of significant results regarding anhedonic depression may be attributable to the relatively poor within-person internal consistency of the anhedonic depression subscale of the

Mini-MASQ observed in this sample, although multilevel modeling is relatively robust to such unreliability (Nezlek, 2017). Furthermore, improvements in mood following a night of sleep deprivation may be specific to laboratory sleep deprivation, clinical samples, or other aspects such as the activities the participant engages in overnight in lieu of sleep. Much research is needed to fully integrate the relationship between findings produced in laboratory sleep studies and sleep deprivation as it occurs naturally in everyday life.

There are additional limitations to the study warranting mention. In the process of maximizing ecological validity, we also had to sacrifice the rigorous control over conditions that experimental studies possess and as such, we cannot rule out the possibility that unmeasured variables, such as whether the night of partial sleep deprivation was intentional (e.g., choosing to stay up late to study for an exam) or unintentional (e.g., trying but being unable to fall asleep) are related to next-day anxious arousal, anhedonic depression, or general distress. Future studies should take into account the context of naturalistic sleep deprivation, as increased anxious arousal may be experienced differently based on whether a night of total or partial sleep deprivation was intentional and how pleasant or stressful the activities individual engaged in during the night. Acute loss of sleep due to a long night of bonding with close friends may impact next-day mood differently than lack of sleep due to insomnia or a looming project deadline.

Additionally, this study relied on self-report for assessments of both total sleep time and symptoms of anxiety and depression. There is evidence to suggest that self-reported sleep duration is systematically greater than objectively assessed sleep duration (e.g., Cespedes et al., 2016; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008), although this concern is mitigated by the focus on within-person changes of self-reported sleep duration across time and the

conservatively set cut-off for partial sleep deprivation. Still, future studies that seek to examine the influence of sleep deprivation in everyday life would benefit from the increased precision offered by the use of objective measures of total sleep time such as actigraphy in addition to self-report. In a similar vein, more nuanced investigations of the effects of sleep deprivation on next-day anxious arousal and the physical symptoms of anxiety would be made possible by the measurement of biological markers of physiological arousal such as changes in cortisol levels. Finally, replication of the study in larger samples with greater power to detect effects is warranted, as is an examination of the effects of naturalistic partial sleep deprivation in clinical samples.

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Figure 1. Moderation of the Relationship Between Partial Sleep Deprivation and Next-Day Anxious Arousal by Baseline Anxiety Symptoms. High and Low represent ± 1 SD from the mean. Sleep Dep indicates that the previous night qualified as an instance of partial sleep deprivation, while Normal Night indicated that the prior night did not qualify. STAIXS = State-Trait Anxiety Inventory Form X – State.

Figure 2. Moderation of the Relationship Between Partial Sleep Deprivation and Next-Day Anxious Arousal by Baseline Depressive Symptoms. High and Low represent ± 1 SD from the mean. Sleep Dep indicates that the previous night qualified as an instance of partial sleep deprivation, while Normal Night indicated that the prior night did not qualify. CESD-R = Center for Epidemiologic Studies Depression Scale Revised.

Table 1

Descriptive Statistics and Bivariate Correlations Among Hours of Sleep and Symptoms of Anxiety and Depression (N = 94).

	M (SD)	1	2	3	4	5
1. Hours	7.52 (1.07)	–				
2. CESD-R	19.03 (12.38)	-.08	–			
3. STAI-XS	39.42 (11.89)	-.16	.69*	–		
4. AA	12.12 (2.79)	.03	.56*	.58*	–	
5. AD	23.55 (4.77)	.01	.46*	.52*	.37*	–
6. GD	12.43 (4.48)	-.02	.73*	.65*	.63*	.47*

Note: Means and standard deviations were calculated using each subject's mean rating across the study for each variable. These values were used in the correlation analyses to preserve independence of observations.

Hours = average number of hours slept per night; CESD-R = Center for Epidemiologic Studies Depression Scale Revised; STAI-XS = State-Trait Anxiety Inventory Form X – State; AA = anxious arousal subscale of the Mini Mood and Anxiety Symptom Questionnaire (Mini-MASQ); AD = anhedonic depression subscale of the Mini-MASQ; GD = general distress subscale of the Mini-MASQ.

* $p < .001$

Table 2

Interaction of Baseline Anxiety Symptoms and Partial Sleep Deprivation Predicting Current Anxious Arousal.

Outcome	Predictor	β	Z	p-value
Anxious Arousal	Intercept	5.85	8.13	<.001
	Anxious Arousal _{t-1}	.24	7.53	<.001
	Sleep Dep _{t-1}	-2.41	-1.88	.06
	STAI-XS	.05	2.63	<.01
	STAI-XS*Sleep Dep _{t-1}	.08	2.74	<.01
	CESD-R	.04	1.97	<.05
	Gender	.57	1.44	.15
	Time	-.009	-.44	.66
	Antidepressant Use	.63	1.23	.22
	Napping Behavior _{t-1}	.02	.12	.90
	Caffeine Use _{t-1}	-.02	-.20	.85

Note: β = unstandardized regression coefficient; Sleep Dep = whether the previous night was an instance of partial sleep deprivation; STAI-XS = State-Trait Anxiety Inventory Form X – State; CESD-R = Center for Epidemiologic Studies Depression Scale Revised.

Table 3

Interaction of Baseline Depressive Symptoms and Partial Sleep Deprivation Predicting Current Anxious Arousal.

Outcome	Predictor	β	Z	p-value
Anxious Arousal	Intercept	5.81	8.09	<.001
	Anxious Arousal _{t-1}	.24	7.54	<.001
	Sleep Dep _{t-1}	-.97	-1.38	.17
	CESD-R	.04	1.81	.07
	CESD-R*Sleep Dep _{t-1}	.09	3.28	.001
	STAI-XS	.06	2.74	<.01
	Gender	.57	1.44	.15
	Time	-.006	-.33	.74
	Antidepressant Use	.63	1.24	.21
	Napping Behavior _{t-1}	.03	.17	.87
	Caffeine Use _{t-1}	-.03	-.24	.81

Note: β = unstandardized regression coefficient; Sleep Dep = whether the previous night was an instance of partial sleep deprivation; CESD-R = Center for Epidemiologic Studies Depression Scale Revised; STAI-XS = State-Trait Anxiety Inventory Form X – State.