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A Test of the Effects of Acute Sleep Deprivation on General and Specific Self-Reported Anxiety and Depressive Symptoms: An Experimental Extension

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Abstract

Evidence indicates acute sleep deprivation affects negative mood states. The present study experimentally tested the effects of acute sleep deprivation on self-reported symptoms of state anxiety and depression as well as general distress among 88 physically and psychologically healthy adults. As hypothesized, the effects of acute sleep deprivation increased state anxiety and depression, as well as general distress, relative to a normal night of sleep control condition. Based on the tripartite model of anxiety and depression, these findings replicate and extend prior research by suggesting sleep deprivation among individuals without current Axis I disorders increases both state symptoms of anxiety and depression specifically, and general distress more broadly. Extending this work to clinical samples and prospectively testing mechanisms underlying these effects are important future directions in this area of research.

Keywords

sleep; mood; depression; anxiety; sleep deprivation

1. Introduction

The World Health Organization's (WHO) 2004 Global Burden of Disease report indicated 3.6 million years of productive healthy life is lost worldwide as a result of primary insomnia (World Health Organization, 2008). Sleep deprivation results in an estimated \$65 billion dollars in health care costs and lost productivity in the United States alone (U.S. Surgeon General, 2004). In fact, as many as 70 million people in the United States report sleep deprivation, making this one of the most common health problems in the United States (U.S. Surgeon General, 2004). Approximately 30-35% of people meet diagnostic criteria for primary insomnia (Breslau, Roth, Rosenthal, & Andreski, 1996), which is characterized by impairment resulting from problems falling and staying asleep or nonrestorative sleep (American Psychiatric Association [APA], 2000; American Sleep Disorders Association, 1990). These data clearly highlight the importance of understanding the effects of sleep deprivation.

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Contemporary research coalesces to indicate sleep deprivation increases negative affect and decreases positive affect. For example, sleep deprivation increases negative reactions to challenging life events and decreases positive reactions to pleasant events (Zohar Tzischinsky, Epstein, & Lavie, 2005). Similarly, an experimental test of one night of sleep deprivation compared to sleeping normally decreased positive mood and increases negative mood (Franzen, Siegle, & Buysse, 2008). Comparably, 56 hours of continuous wakefulness increases somatic complaints, anxiety symptoms, depressive symptoms, and paranoia among healthy individuals (Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007). In addition to research linking sleep deprivation to broad affective consequences, studies also suggest sleep deprivation is associated with increased anxiety and depressive symptoms specifically.

Sleep deprivation is positively correlated with anxiety (Ford & Kamerow, 1989; Gregory, Eley, Moffitt, O'Connor, & Poulton, 2005; Gregory, Eley, O'Connor, & Plomin, 2004; Gregory & O'Connor, 2002; Roth et al., 2006), as well as, tension, nervousness, and irritability (Oginska & Pokorski, 2006). Moreover, laboratory-based work suggests acute sleep deprivation increases anxiety. For example, an experimentally manipulated 36-hour period of acute sleep deprivation increased state anxiety symptoms among a sample of 12 young, healthy, non-clinical participants (Sagaspe, Sanchez-Ortuno, & Charles, 2006). Emerging experimental research further suggests that anxious reactivity to acute sleep deprivation may be associated with vulnerability to panic spectrum problems (Babson, Feldner, Trainor, & Smith, 2009) and the development of posttraumatic stress symptoms [see Babson and Feldner, 2010 for a review].

Research on the effects of sleep deprivation on depressed mood is less consistent than work focused on anxiety. Sleep deprivation appears to reduce depressive symptoms among depressed individuals (Naylor et al., 1993; Pflug, 1976). For example, Naylor and colleagues (1993) induced 36 hours of sleep deprivation among clinically-depressed adolescents (12 to 17 years old). Depression ratings were acquired prior to, during, and after sleep deprivation. Results indicated that sleep deprivation decreased depression symptom severity. In contrast, evidence from studies examining the effects of acute sleep deprivation among healthy samples suggests acute sleep deprivation (i.e., 24 hours) *increases* depressive symptoms (Caldwell, Caldwell, Brown, & Smith, 2004; Cutler & Cohen, 1979; Rose, Manser, & Ware, 2008). For instance, the effects of sleep deprivation on depressed mood were examined among a sample of 64 healthy young adults (Campos-Morales, Valencia-Flores, & Castano-Meneses, 2005). Participants who reported greater levels of sleepiness reported higher levels of depressive symptoms. These results suggest that among a healthy sample, sleep loss may positively correlate with depressive symptoms. Collectively, it appears that sleep deprivation is related to increases in both anxiety and depressive symptoms among healthy participants. No single study conducted to date, however, has experimentally manipulated sleep to examine the effects of acute sleep deprivation on both anxiety and depressive symptoms. Importantly, examining the effects of sleep deprivation on both of these types of symptoms is important as distinctions have been made between anxiety and depression, despite the common overlap between the two.

The tripartite model (Brown, Chorpita, & Barlow, 1998; Clark & Watson, 1991; Watson et al., 1995) distinguishes between anxiety and depression. This model indicates negative affect is elevated in both anxiety and depression, but physiological arousal and positive affect differentiate between them. Anxiety and depression are characterized by elevated physiological arousal and decreased positive affect, respectively. Employing this theoretical approach to distinguish between anxiety and depression, the consequences of sleep deprivation should increase both anxiety and depressive symptoms. The physiological effects of sleep deprivation, such as jitteriness and agitation (Everson, 1998), likely would increase physiological arousal, which is a central component of anxiety according to the tripartite model. The affective

consequences of sleep deprivation, such as increased depressive thinking and loss of interest in pleasurable activities (Kahn-Greene et al., 2007) would likely decrease positive affect, which is central to depression. Furthermore, consequences of sleep loss that may not be specific to symptoms of state anxiety or depression, but rather common to both (e.g., fatigue, confusion, psychomotor retardation), are likely to increase general distress as defined by the tripartite model.

While past research has investigated the effects of sleep deprivation on mood, the current study extends this past research in multiple significant ways. First, to empirically test the effects of acute sleep deprivation on state anxiety and depressive symptoms independently, it is critical to employ an assessment strategy that has been designed to tease apart these highly correlated constructs. Indeed, many measures of anxiety and/or depressive symptoms, including those employed in prior studies in this domain (Kahn-Greene et al., 2007), do not sufficiently discriminate between anxiety and depression (Clark & Watson, 1991; Gotlib, 1984).

Accordingly, the current study employed the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995) to measure state anxiety and depression-specific symptoms, in addition to symptoms that are correlated with both (i.e., general distress). The MASQ is based upon the tripartite model of depression and anxiety and is well-established in mood and anxiety-related research. Second, past research has followed one to three nights of sleep deprivation with an assessment of mood or anxiety following the sleep deprivation (e.g., Campos-Morales et al., 2005). However, the absence of baseline measurements of anxiety and depressive symptoms prior to sleep deprivation makes it difficult to draw conclusions regarding the effects of acute sleep deprivation on these symptoms (e.g., Campos-Morales et al., 2005). The current study extends this work by implementing a pre- and post-sleep deprivation assessment of state anxious and depressive symptoms. Finally, past research on the effects of sleep deprivation on state anxiety and depressive symptoms (as opposed to mood; Franzen et al., 2008) has not used a sleep as normal control group (e.g., Kahn-Greene et al., 2007). For this reason, the current study included a sleep as normal control condition, which further strengthens confidence in conclusions that can be drawn (by, for instance, methodologically controlling for the effects of repeated assessment). It was hypothesized that in comparison to the normal sleep control group, the sleep deprivation group would report (1) an increase in general distress, (2) an increase in the intensity of state anxiety-specific symptoms, and (3) an increase in state depression-specific symptoms. Past research has suggested no significant difference in the magnitude of anxiety versus depressive symptom increases resulting from sleep deprivation (Kahn-Greene et al., 2007). For this reason, no specific hypotheses were made regarding the relative magnitude of symptom elevation between anxiety and depressive symptoms following sleep deprivation. Evidence supporting these hypotheses may help to inform the prevention of comorbid sleep problems and disorders of anxiety and depression by advancing currently limited understanding of the affective consequences of acute sleep deprivation.

2. Method

2.1 Participants

Recruitment was conducted via announcements and fliers placed around the University of Arkansas and the Northwest Arkansas community. Inclusion criteria included age of at least 18 years. Exclusionary criteria included evidence of: 1) a lifetime history of Axis I psychopathology, including sleep disorders and nonclinical panic attack histories; 2) limited mental competency and the inability to give informed, voluntary, written consent to participate; and 3) suicidality. Participants reporting current Axis I psychopathology, including sleep disorders, and/or suicidality were given referral information when appropriate. Fourteen people were excluded due to current insomnia or the use of prescription sleep medications and 54 individuals were excluded based on meeting at least one of the other exclusionary criteria.

One hundred and two (44 females) adults ($M_{\text{age}} = 23.19$ years, $SD = 8.2$) people were enrolled into the study after determining if they met inclusion and exclusion criteria. From the original sample of 102, 8 completed day 1 of the protocol but did not return for the day 2 assessment, and 6 were not included in analyses due to missing data. Overall, a final sample of 88 (37 females) adults ($M_{\text{age}} = 23.21$ years, $SD = 8.23$) completed all portions of the experimental protocol and were included in all analyses. Participants lost to attrition did not differ from those retained in the study in terms of any of the variables examined in the current study, including group assignment. Please see Table 1 for an overview of demographic information pertaining to the sample, including ethnicity and education.

2.2 Measures

Structured Clinical Interview-Non-Patient Version for DSM-IV (SCID-IV)—

Lifetime and current Axis I psychopathology and psychotropic medication use were indexed using the SCID-IV (First, Spitzer, Gibbon, & Williams, 1995). This instrument also was used to assess the presence of suicidality and nonclinical panic attack history. The non-patient version was used because participants were identified as non-clinical. Adequate reliability of the SCID has been demonstrated (Spitzer, Williams, Gibbon, & First, 1989). The SCID-IV was administered to all participants by the first author (K.B.). Training in SCID-IV administration was conducted by the second author (M.F.) until mastery of the interview was demonstrated. Thereafter, six administrations were audiotaped and diagnoses were compared with the second author. The first author began independently administering the interview subsequent to reaching 100% diagnostic agreement with the second author. Also, ongoing supervision to correct for rater drift was included throughout the project. Upon completion of the study a random selection of 10% of the administrations were checked by the third author (C.T.). Inter-rater agreement was 100%.

Sleep Disorders Questionnaire (SDQ)—The SDQ (Violani et al., 2000) is a brief insomnia questionnaire, consisting of 18 questions that allow for evaluation of insomnia based on Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (APA, 1994) criteria. Questions index excessive sleepiness, sleep apnea, parasomnias, and duration of sleep problems. Sample questions include: “In the last 30 days did you suffer from 1 or more of the following problems: did you take more than a half hour to fall asleep?” and “Did you have problems staying awake during the day?” Responses are given as *yes* or *no* answers. Questions focus on sleeping patterns within the last month as well as current sleeping habits. Adequate convergent validity has been demonstrated via correlations with scores on the Pittsburgh Sleep Quality Index, a well established measure of insomnia (Cohen’s kappa = .78; Violani et al., 2004). In the current study, the SDQ was utilized to identify, and exclude, participants endorsing a history of sleep disorders.

Positive and Negative Affect Schedule (PANAS)—The PANAS is a 20-item measure in which respondents indicate on a 5-point Likert-type scale (1 = *very slightly or not at all* to 5 = *extremely*) the extent to which they generally feel different feelings and emotions (e.g., *Hostile*). The PANAS is a well-established measure of individual differences in affect that is commonly used in psychopathology research (Watson, Clark, & Tellegen, 1988). Factor analysis indicates that it assesses two global dimensions of affect: negative and positive. Both subscales of the PANAS have demonstrated good convergent and discriminant validity. Additionally, both the negative affect as well as the positive affect scales of the PANAS have demonstrated high levels of internal consistency (range of alpha coefficients: .83 to .90 and .85 to .93, respectively). A large body of literature supports the validity of the PANAS (see Watson, 2000). In the current study, the negative affect subscale was utilized to compare groups in terms of baseline negative affectivity to ensure groups did not differ on baseline levels of this factor.

Mood and Anxiety Symptom Questionnaire (MASQ)—The MASQ (Watson et al., 1995) is a 62-item comprehensive measure of affective symptoms (e.g., cheerful, afraid, confused). This self-report instrument assesses mood dimensions important to the tripartite model of anxiety and depression (Watson et al., 1995). There are three main facets to this measure. The Anxious Arousal scale (MASQ-AA) measures the symptoms of somatic tension and arousal (e.g., “felt dizzy”). The Anhedonic Depression scale (MASQ-AD) measures a loss of interest in life (e.g., “felt nothing was enjoyable”) and reverse-keyed items measuring positive affect. The General Distress scale (MASQ-GD) measures symptoms of both anxiety and depression. This scale can be separated into two sub-factors. The General Distress: Depressive Symptoms scale measures depressed mood expected to be nondifferentiating relative to anxiety (e.g., “felt discouraged”). The General Distress: Anxious Symptoms scale indexes anxious mood expected to be nondifferentiating relative to depression (e.g., “felt nervous”). However, research has suggested the general distress factors do not demonstrate more discriminant or convergent validity compared to the combined General Distress scale (Watson et al., 1995). Therefore, consistent with past research (Keogh & Reidy, 2000) the combined General Distress scale (MASQ-GD) was used in the current study to measure nondifferentiating anxiety and depressive symptoms; the MASQ-AD and the MASQ-AA subscales were employed as measures of depression-specific and anxiety-specific symptoms, respectively. Consistent with previous research (Mustanski, 2007), participants indicated on a five-point Likert-type scale how much they were currently experiencing each symptom from 1 (*not at all*) to 5 (*extremely*). The MASQ shows excellent convergence with other measures of anxiety and depression (Watson et al., 1995).

Semi-Structured Substance Use and Sleep Interview—This semi-structured interview consists of two sections. Section one includes an assessment of substance use, including nicotine, stimulants, and prescription medication, during the past 24-hours. Questions are administered in two parts. Part I asks participants to respond with a yes or no if they have used each specific substance in the past 24 hours. Part II asks participants how much of each substance they have used in the past 24 hours. Section two of the interview includes an assessment of sleep during the past 24 hours. Participants are asked to provide the number of hours and minutes of sleep they obtained during the past 24 hours. This section also includes questions about napping behavior during the past 24 hours. This interview was administered by a pre-doctoral graduate student in clinical psychology.

2.3 Procedure

Interested participants who contacted the laboratory were invited to attend a laboratory-based individual baseline assessment session. At this session, participants provided written informed consent. The SDQ and SCID were then administered to evaluate exclusionary criteria. Participants not eligible for participation at this stage were compensated \$10 and thanked for their participation. Eligible participants then completed a battery of self-report measures including the PANAS and MASQ.

Participants were then randomly assigned to either an acute sleep deprivation (experimental) or a no sleep deprivation (control) group. Participants were asked to choose a day and time when losing sleep for an entire night would not interfere in their daily life and when they could attend the second laboratory session the morning after the night of acute sleep deprivation. Those assigned to the control group were instructed to sleep as normal and those in the experimental group were instructed to stay awake through the night. Participants were not informed of which group they were assigned to until the end of the day 1 procedures. This ensured that participants in the experimental group would not change their behavior during the day before coming to the lab for the first session (e.g., sleep all day prior to the first

appointment). In addition, individuals in the experimental group were given the following set of instructions:

“I am going to ask that you stay awake for the next 24-hours. Please refrain from sleeping, including any naps from this point until our next scheduled meeting time which will be tomorrow. Every hour I would like you to call into our laboratory digital monitoring system and provide your name and time of day. In addition, due to the effects of sleep loss, I am asking that you do not drive to or from the laboratory tomorrow, but instead have a friend drive you or take public transportation.”

All participants were instructed to not change drug use patterns, including alcohol and cigarette use patterns, relative to normal routines. In an effort to control for possible differences in drug-related withdrawal symptoms, they were not instructed to completely refrain from typical substance use (e.g., smoking) patterns. Participants then scheduled their day 2 appointment for the next day, and were compensated \$20 for completion of day 1. All participants completed day 1 between 12:00 P.M. and 5:00 P.M.

Participants then returned to the laboratory to complete day 2 of the protocol between 6:00 A.M. and 10:00 A.M. This one hour session included a brief semi-structured interview administered to assess the prior night's sleep pattern as well as 24 hour substance use patterns (i.e., cigarettes, caffeine, stimulants). Participants who reported changing use of these substances relative to typical use patterns and those who did not follow the protocol for acute sleep deprivation or a full night sleep, were given the option of either (1) refraining from sleep on a separate night and again returning to the laboratory to complete the protocol or (2) discontinuing participation in the study and receiving an additional \$10 compensation. All participants followed the protocol for acute sleep deprivation, therefore these alternative options were not employed. Participants completed a brief questionnaire packet including the MASQ. Participants were debriefed and compensated an additional \$60 for completion of day 2.

3. Results

3.1 Manipulation Checks

Random Assignment—Chi-squared analyses and one-way analyses of variance (ANOVAs) were employed to compare groups in terms of theoretically-relevant baseline characteristics to check the efficacy of random assignment. These analyses indicated no group differences on day 1 baseline indices, including negative affectivity, and gender. Groups also did not differ in terms of any of the day 1 MASQ subscales, including Anxious Arousal, Anhedonic Depression, or General Distress. Table 1 includes descriptive information regarding each of these factors. Together, groups did not differ prior to the experimental (sleep) manipulation in terms of any of the factors examined in the current study.

Sleep Manipulation—Consistent with the sleep deprivation protocol, all participants in the sleep deprivation group made hourly phone calls during the night. To further assess compliance with the sleep manipulation protocol, t-tests were conducted to compare groups on total hours of sleep and cigarette use during the night. These findings supported findings from our digital sleep diary. Specifically the control group ($M = 7.77$ hours, $SD = 1.10$ hours) obtained significantly more sleep during the night in comparison to the sleep deprivation group [$M = .14$ hours, $SD = 0.79$ hours; $t(85) = 36.47, p = .0001$]. Furthermore, among smokers, there were no group differences in cigarettes smoked during the night between the control ($M = 4.27$, $SD = 3.72$) and experimental ($M = 5.43$, $SD = 4.61$) groups [$t(16) = .56, p = .44$].

3.2 Primary Hypothesis Tests

To test the effect of sleep deprivation on state anxiety symptoms and depression symptoms, a series of repeated measures ANOVAs were conducted. Specifically, the within-subject variables of levels of General Distress, Anxious Arousal, and Anhedonic Depression, as measured by the MASQ (MASQ-GD, MASQ-AA, MASQ-AD; respectively) on day 1 and day 2, were entered into separate repeated measures ANOVAs to compare groups (control versus experimental) in terms of levels of all within-subject factors between day 1 and day 2 (pre- to post-sleep deprivation). In the case of significant interactions, planned interaction contrasts were conducted to analyze the interaction. Effect size was indexed via eta squared (η^2).

General Distress—Results indicated no main effects of either group [$F(1, 86) = 2.94, p = .09$] or day [$F(1, 86) = 0.02, p = .89$]. However, there was a significant interaction between group and day [$F(1, 86) = 7.69, p = .007; \eta^2 = .08$]. Interaction contrasts revealed that general distress significantly increased in the experimental group [$t(42) = 1.96, p = .05; \eta^2 = .09$] from day 1 to day 2. In comparison, the control group significantly decreased in general distress [$t(44) = 1.22, p = .05; \eta^2 = .03$] from day 1 to day 2. Figure 1 includes a graphic representation of this pattern and Table 1 presents the associated means and standard deviations.

Anxious Arousal—Results indicated a significant main effect of day [$F(1, 86) = 7.26, p = .008; \eta^2 = .08$]; anxious arousal was greater on day 2 in comparison to day 1 (see Table 1 for the associated means). In contrast, there was no main effect of group [$F(1, 86) = 0.00, p = .95$]. These findings, however, were qualified by a significant interaction between group and day [$F(1, 85) = 12.38, p = .01; \eta^2 = .05$]. Interaction contrasts suggested that Anxious Arousal scores increased in the experimental group [$t(44) = 3.48, p = .001; \eta^2 = .25$], but not in the control group [$t(44) = 0.43, p = .67; \eta^2 = .00$] from day 1 to day 2. Figure 2 presents a graphic representation of this interaction and Table 1 includes the associated means and standard deviations.

Anhedonic Depression—Results indicated no main effects of either group [$F(1, 86) = 0.00, ns$] or day [$F(1, 86) = 2.77, p = .10$]. Results did suggest a significant interaction between group and day in terms of Anhedonic Depression scores [$F(1, 85) = 74.96, p = .01; \eta^2 = .07$]. Interaction contrasts suggested that Anhedonic Depression levels increased in the experimental group [$t(42) = 3.55, p = .001; \eta^2 = .28$] from day 1 to day 2, but not in the control group [$t(44) = 0.48, p = .63; \eta^2 = .00$]. Figure 3 portrays this interaction and Table 1 includes the associated means and standard deviations.

3. Discussion

Research has demonstrated positive associations between sleep deprivation and negative mood states (Oginska & Pokorski, 2006), including anxiety (Sagaspe et al., 2006). Evidence pertaining to depressive symptoms has been less consistent, with sleep deprivation decreasing depressive symptoms among clinically-depressed individuals (Naylor et al., 1993; Pflug, 1976) and increasing symptoms among healthy individuals (Campos-Morales et al., 2005). Importantly, however, an experimental test of the effects of sleep deprivation on both state anxiety and mood symptoms that employed a symptom measurement strategy designed to tease these inter-related constructs apart had not been conducted. Accordingly, the current study experimentally tested the effects of acute sleep deprivation on both state anxiety and depression-specific symptoms as well as general distress among physically and psychologically healthy young adults.

Consistent with hypotheses, acute sleep deprivation increased specific symptoms of state anxiety and depression as well as general distress. In relation to anxious arousal, a significant interaction between group and day indicated that sleep deprived individuals, but not participants in the normal sleep condition, reported increases in anxious arousal from day 1 to day 2. The anxious arousal factor of the MASQ indexes symptoms specific to anxiety, with a focus on physiological arousal (e.g., racing heart, easily startled, shaky, and twitching muscles). This pattern generally suggests that acute sleep deprivation increases anxious arousal. A second significant group by day interaction suggested that sleep deprivation increased anhedonic depression. Consistent with the tripartite model of anxiety and depression, the anhedonic depression scale of the MASQ measures depression-specific symptoms focused on a relative absence of positive affect (e.g., feeling slowed down, bored, withdrawn, needing extra effort to get moving). Extrapolating from these data, the current results suggest that acute sleep deprivation, in part, reduces positive affect. Finally, among sleep-deprived individuals, general distress increased from day 1 to day 2, whereas participants with a full night of sleep reported a significant decrease in general distress from day 1 to day 2. As described previously, the general distress subscale of the MASQ measures non-differentiating symptoms of anxiety and depression (e.g., feeling tired, trouble concentrating, feeling confused, trouble remembering, worry). Thus the current findings suggest that sleep deprivation increases general distress (as well as anxious arousal and anhedonic depression). A normal night of sleep actually appeared to decrease general distress, which may reflect habituation to the experimental context such that participants were relatively more comfortable (i.e., less generally distressed) with the laboratory environment during day 2 of the procedure.

The current study's incorporation of a measure designed to differentiate between state anxiety and depression symptoms, along with the pre/post assessment of state anxiety and mood symptoms and the use of a control group, significantly strengthens the postulation that acute sleep deprivation increases both state anxiety and depression symptoms among nonclinical samples. Moreover, the current results suggest that sleep deprivation may increase state anxiety via increasing general distress broadly and anxious arousal specifically. Comparably, sleep deprivation may increase state depressive symptoms among healthy individuals by increasing general distress and decreasing positive affect/increasing anhedonia. Several future studies are now needed to further clarify the linkage between sleep deprivation and anxiety and mood symptoms. Longitudinal research has suggested that chronic sleep problems are positively correlated with the development of anxiety and depression (Breslau, Roth, Rosenthal, & Andreski, 1996; Ford & Kamerow, 1989; Gregory et al., 2004, 2005; Gregory & O'Connor, 2002). However, the degree to which results from studies of acute sleep deprivation generalize to these naturalistic examinations of chronic sleep deprivation remains unclear. While this study is a first step in this area, future research is needed to understand if different processes are involved in the relation between acute versus chronic sleep deprivation with state anxiety and depression. For example, a study could implement varying levels of sleep deprivation ranging from 8 to 72 hours while using a multimodal assessment of both mood and anxiety throughout the deprivation period. Such research would further advance our understanding of how relatively more protracted sleep loss may differentially affect mood and anxiety. Also, examining how individual differences that affect anxiety and depression development resulting from chronic sleep deprivation relate to responding to acute sleep deprivation would advance understanding of the generalizability of acute sleep deprivation designs to naturalistic examination of chronic sleep disturbance. For example, research has demonstrated that the relation between worry and sleep disturbance is dependent upon time and chronicity (Jansson & Linton, 2006; Morin, 2003). This work suggests that chronic sleep disturbance combined with worry may interact to influence the development of anxiety and depression. Linking worry to the effects of acute sleep deprivation would help to further understand the generalizability of findings from acute sleep deprivation designs to those focused on naturalistic chronic sleep disturbance.

The current findings also support further investigation into mechanisms of action that may account for why acute sleep deprivation may increase anxiety and depressive symptoms among healthy individuals, but reduce depression among clinically depressed people. For instance, neuroimaging studies have demonstrated that sleep deprivation increases synaptic dopamine, noradrenaline, and serotonin levels among individuals with major depression (Adrien, 2002; Ebert et al., 1996; Gerner, Post, & Bunney, 1976; Müller, Riemann, Berger, & Müller, 1993). These findings have led to suggestions that sleep deprivation acts in a similar manner to antidepressants (Adrien, 2002; Dieter & Berger, 1998). Indeed, sleep deprivation has been used as a treatment method for major depression, although the antidepressant effects appear to be short term (Kundermann, Hemminger-Spörl, Huber, Krieg, & Lautenbacher, 2008). It is not currently clear why these effects emerge among clinically-depressed people whereas depressive symptoms appear to increase among non-depressed individuals. It is possible that increases in synaptic serotonin, for example, among non-depressed individuals do not result in increases in positive affect because of saturation of serotonin receptor sites, but rather the effects of sleep deprivation (e.g., loss of interest in pleasurable activities) mimic depressive symptoms and therefore appear to increase depression among this population. Among depressed individuals, however, the increase in serotonin (for example) may provide an important increase in relatively lowered levels of this neurotransmitter, thereby alleviating depression temporarily. In addition, it is possible sleep deprivation may be correlated with changes in mood as a result of the overlapping role of the hypothalamus in the regulation of sleep and mood via regulation of the sympathetic system. For example, inhibition (via serotonin or noradrenaline) of the ventrolateral preoptic nucleus (VLPO; involved in the inhibition of neurons involved in wakefulness) may lead to disruptions in sleep (Saper, Scammell, & Lu, 2005). These same neurochemical inhibitors (serotonin, GABA, noradrenaline) are also involved in changes in mood states. In addition, non-human research has demonstrated VLPO neurons double in firing rate during recovery following sleep deprivation. Interestingly, these neurons do not increase as a result of sleepiness, but instead as a function of the production of sleep (Saper, Chou, & Scammell, 2001). Therefore, future research may benefit from investigating the role of sleep deprivation on these overlapping neural mechanisms among humans. Cognitive mediators also need to be explored. It is possible that the depression-relevant effects of sleep deprivation differ in salience between depressed (e.g., positively valenced change relative to depressed baseline) versus non-depressed (e.g., negatively valenced change relative to non-depressed baseline) individuals. Also, research has identified sleepiness (Campos-Morales et al., 2005; Franzen et al., 2008) as a factor that may be critical to consider in the link between sleep deprivation and anxiety and depression. Furthermore, emerging evidence suggests elevated reactivity to bodily arousal may be a factor involved in the relation between sleep deprivation and panic spectrum problems (Babson et al., 2009). Future research is needed to understand the underlying mechanisms of these patterns to better elaborate models of the consequences of sleep loss in terms of the etiology and maintenance of anxiety and mood disorders.

Limitations to the current study must be considered when interpreting these results. First, this sample consisted of mainly Caucasian, well-educated, healthy, young adults. While employing a sample of healthy individuals strengthens conclusions regarding the effects of sleep deprivation by methodologically controlling for confounds that may be introduced by pre-existing psychopathology (e.g., major depression), the generalizability of the current findings is limited. In a similar domain, even though the current sample included a relatively large age range (19-60 years old), it was comprised of a large proportion of young adults. Given evidence of age differences in sleep patterns, an important next step would be to extend this research to samples of individuals systematically varying in age. For example, given the elevated rates of sleep problems among children (Carskadon, 1990), investigating the effects of acute sleep deprivation on anxiety and depressive symptoms among children would improve our understanding of the generalizability of the current results to other developmental stages as

well as aid in advancing models implicating sleep loss in the development of psychopathology. A second limitation of the current study is the lack of direct observation of the sleep manipulation. The implementation of the phone-in digital sleep diary allowed for a certain degree of monitoring sleep loss and improved confidence in the validity of the sleep manipulation by eliminating “faked compliance” by last-minute completion of paper and pencil diaries, which is often observed in studies utilizing self-monitoring (Litt, Cooney, & Morse, 1998). Nonetheless, future research would benefit from conducting the sleep deprivation portion of the study within the laboratory context in order to increase confidence in the sleep manipulation. Furthermore, monitoring anxiety and depressive symptoms during the night would allow for comparability with research employing this type of design with clinically-depressed samples (Kundermann et al., 2008). Finally, the current study used self-report measures of both anxiety and depression. While the self-report measures were important in differentiating between the effects of sleep deprivation on anxiety versus depressive symptoms, a multimodal assessment of anxiety and depression would aid future research. For example, self-report measures could be supplemented with a laboratory-based assessment of depressive and anxious reactivity. This methodological extension would increase confidence in conclusions regarding the affective consequences of acute sleep deprivation.

In conclusion, these findings combine to suggest sleep deprivation increases general and specific symptoms of anxiety and depression. These preliminary findings have both theoretical and preventive implications. First, models of the pathogenesis of anxiety and depression may benefit from considering the role of sleep deprivation given the high prevalence of sleep disorders and the role sleep deprivation appears to play in increasing anxiety and depressive symptoms among healthy individuals. In terms of possible preventive implications, these results indicate that acute sleep deprivation increases the likelihood that an individual will experience elevations in both anxiety and depression. As such, sleep deprivation may set the stage for the future development of anxiety and depressive disorders. Therefore, successful alleviation of sleep problems among otherwise healthy individuals may help prevent the development of future anxiety and depressive disorders.

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Figure 1.
Interaction of group by day in terms of day 1 and day 2 General Distress.

Figure 2.
Interaction of group by day in terms of day 1 and day 2 Anxious Arousal levels.

Figure 3.
Interaction of group by day in terms of day 1 and day 2 Anhedonic Depression levels.

Table 1
Descriptive Data for Demographic and Main Variables Examined as a Function of Group

	Group	
	<i>Experimental</i> <i>M or n</i> (<i>SD or %</i>)	<i>Control</i> <i>M or n</i> (<i>SD or %</i>)
<i>Demographics</i>		
Gender	20 (46.5%)	17 (37%)
Ethnicity		
Caucasian	41 (95.3%)	42 (93.3%)
African-American	0 (0%)	3 (6.7%)
Asian	2 (4.7%)	0 (0%)
Other	0 (0%)	0 (0%)
Education Completed		
High school	1 (2.3%)	3 (6.5%)
Two-year college	1 (2.3%)	1 (2.2%)
Partial four-year college	41 (95.3%)	37 (80.4%)
Four-year college	0 (0%)	2 (4.3%)
Partial graduate	0 (0%)	2 (4.3%)
Complete graduate	0 (0%)	1 (2.2%)
Smoking Status	7 (16.3%)	11 (23.9%)
Negative Affect	17.06 (5.51)	15.10 (3.66)
<i>Within-Subject Factors (Day 1)</i>		
General Distress	35.55 (9.90)	33.58 (8.51)
Anxious Arousal	20.09 (2.95)	21.10 (5.35)
Anhedonic Depression	35.04 (11.27)	36.89 (10.25)
<i>Within-Subject Factors (Day 2)</i>		
General Distress	36.93 (9.71)	32.42 (7.87)
Anxious Arousal	22.46 (5.04)	21.48 (4.62)
Anhedonic Depression	39.04 (11.48)	36.75 (13.29)

Note: Total $n = 88$ (experimental group $n = 43$). n for gender reflects women.