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Temporal patterns of sleep disturbance, anxiety, and depressed mood in generalized anxiety disorder

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TEMPORAL PATTERNS OF SLEEP DISTURBANCE, ANXIETY, AND DEPRESSED MOOD IN GENERALIZED ANXIETY DISORDER

by

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TEMPORAL PATTERNS OF SLEEP DISTURBANCE, ANXIETY, AND DEPRESSED MOOD IN GENERALIZED ANXIETY DISORDER

JACQUELINE BULLIS

Boston University Graduate School of Arts and Sciences, 2016

Major Professor: David H. Barlow, Professor of Psychology

ABSTRACT

Studies suggest that sleep disturbance may be an important etiological factor in the development of comorbid anxiety and depressive disorders, whereby anxiety leads to sleep difficulties, which in turn increase the vulnerability for depression. The primary aim of this study was to determine whether the sequential comorbidity patterns observed at the disorder level (i.e., where anxiety disorders most often precede insomnia, and insomnia most often precedes depression) were also present in daily fluctuations of symptoms. The secondary aim was to explore possible moderators of any observed temporal associations. Participants were 15 patients with generalized anxiety disorder (GAD; mean age = 28.9 years, SD = 9.8) and 15 good sleeper controls (mean age = 27.1 years, SD = 8.3) who were comparable in female:male ratio (73% female vs. 67% female). For 14 days, participants wore an actigraph to objectively assess sleep quality (sleep onset latency, total sleep time, wake after sleep onset, sleep efficiency) and completed daily symptom ratings multiple times each day using their smartphones to assess symptoms of anxiety, depressed mood, and subjective sleep quality.
Study aims were assessed using multilevel modeling, with daily symptoms nested within individuals. Many of the analyses were lagged such that the time-varying predictor variable preceded the time-varying outcome variable temporally. Consistent with hypotheses, results demonstrated that anxious mood was predictive of later subjective and objective sleep disturbance in individuals with GAD, and this effect was strongest among individuals with higher levels of neuroticism, negative affect, and dysfunctional beliefs about sleep. Anxious mood was not associated with later subsequent sleep disturbance in healthy controls. In the GAD group, subjective and objective sleep disturbance predicted later depressed mood; this effect was moderated by temperament and dysfunctional beliefs about sleep. For the control group, the effect of subjective sleep disturbance on later depressed mood was moderated by neuroticism and the effect of objective sleep disturbance was moderated by dysfunctional beliefs about sleep, suggesting that sleep disturbance may increase vulnerability for depressed mood even in healthy individuals. These results suggest that explicitly targeting sleep disturbance during the treatment of GAD may attenuate the experience of depressive symptoms.
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<th>Description</th>
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<tr>
<td>ADIS-5-L</td>
<td>Anxiety Disorder Interview Schedule for DSM-5, Lifetime Version</td>
</tr>
<tr>
<td>AR1</td>
<td>Autoregressive Covariance Type</td>
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<td>ASI</td>
<td>Anxiety Sensitivity Index</td>
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<tr>
<td>BAI</td>
<td>Beck Anxiety Inventory</td>
</tr>
<tr>
<td>BDI</td>
<td>Beck Depression Inventory</td>
</tr>
<tr>
<td>CARD</td>
<td>Center for Anxiety and Related Disorders</td>
</tr>
<tr>
<td>CBT</td>
<td>Cognitive Behavior Therapy</td>
</tr>
<tr>
<td>CSD</td>
<td>Consensus Sleep Diary</td>
</tr>
<tr>
<td>DBAS</td>
<td>Dysfunctional Beliefs About Sleep</td>
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<tr>
<td>DISS</td>
<td>Daytime Insomnia Symptom Scale</td>
</tr>
<tr>
<td>DSM5</td>
<td>Diagnostic and Statistic Manual, 5th Edition</td>
</tr>
<tr>
<td>EMA</td>
<td>Ecological Momentary Assessment</td>
</tr>
<tr>
<td>FSS</td>
<td>Fatigue Severity Scale</td>
</tr>
<tr>
<td>GAD</td>
<td>Generalized Anxiety Disorder</td>
</tr>
<tr>
<td>ISI</td>
<td>Insomnia Severity Index</td>
</tr>
<tr>
<td>MECS</td>
<td>Morningness-Eveningness Composite Scale</td>
</tr>
<tr>
<td>MLM</td>
<td>Multilevel Modeling</td>
</tr>
<tr>
<td>NFFI</td>
<td>NEO Five-Factor Inventory</td>
</tr>
<tr>
<td>OR</td>
<td>Odds Ratio</td>
</tr>
<tr>
<td>PANAS</td>
<td>Positive and Negative Affect Scale</td>
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<tr>
<td>PSQI</td>
<td>Pittsburgh Sleep Quality Index</td>
</tr>
<tr>
<td>Acronym</td>
<td>Description</td>
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<tr>
<td>PSWQ</td>
<td>Penn State Worry Questionnaire</td>
</tr>
<tr>
<td>PTSD</td>
<td>Posttraumatic Stress Disorder</td>
</tr>
<tr>
<td>Q-LES-Q</td>
<td>Quality of Life Enjoyment and Satisfaction Questionnaire</td>
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<tr>
<td>SD</td>
<td>Standard Deviation</td>
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<tr>
<td>SHHC</td>
<td>Sleep Habits and Hygiene Checklist</td>
</tr>
<tr>
<td>SRBQ</td>
<td>Sleep-Related Behaviours Questionnaire</td>
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<tr>
<td>STAI-T</td>
<td>State-Trait Anxiety Inventory, Trait Version</td>
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<tr>
<td>WSAS</td>
<td>Work and Social Adjustment Scale</td>
</tr>
<tr>
<td>WSI-SF</td>
<td>Weekly Stress Inventory, Short-Form</td>
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Introduction

Epidemiological studies suggest that approximately 40 million American adults currently suffer from an anxiety disorder, with a 12-month prevalence rate of 18.1% and a lifetime prevalence rate of 28.8% (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Anxiety disorders are the most commonly diagnosed mental disorder, and are associated with significant distress and functional impairment in social, occupational, and health-related domains, resulting in considerable economic and societal costs each year (Aderka et al., 2012; Barrera & Norton, 2009; Whiteford, Ferrari, Degenhardt, Feigin, & Vos, 2015). When anxiety is comorbid with a depressive disorder, consequences include more severe and persistent symptoms, poorer quality of life, increased economic costs in health service utilization and losses in work productivity, greater risk of suicide, and a blunted response to treatment (Wittchen, Beesdo, Bittner, & Goodwin, 2003; Wittchen, Carter, Pfister, Montgomery, & Kessler, 2000).

Sleep disturbance is often conceptualized as a symptom of other disorders or as secondary to another disorder, particularly anxiety and depressive disorders (Harvey, 2001; Mason & Harvey, 2014; Reynolds et al., 2010; Stepanski & Rybarczyk, 2006). It is often assumed that diagnosis and treatment of the primary disorder or underlying psychological disorder will result in the remission of sleep difficulties as well. However, in a study where patients with posttraumatic stress disorder (PTSD) were treated with cognitive behavioral therapy (CBT) for PTSD, over half of patients continued to report symptoms of insomnia after remission of PTSD (Zayfert & DeViva, 2004). Another study demonstrated similar findings; following CBT for major depression, 92% of
patients who longer met depressive criteria continued to report symptoms of sleep disturbance (Carney, Harris, Friedman, & Segal, 2011).

Prior to a recurrent depressive episode, insomnia and fatigue are the most frequently reported symptoms (Perlman, Johnson, & Mellman, 2006), with insomnia preceding approximately half of new onset or recurrent depressive episodes (Jackson, Cavanagh, & Scott, 2003). Symptoms of insomnia are also the most persistent and treatment-resistant symptoms in depression (Israel, 2010; McClintock et al., 2011; Nierenberg et al., 2010; Taylor, Walters, Vittengl, Krebaum, & Jarrett, 2010). Among a group of patients who met objective criteria for remission of depression, patients who did not feel that they had achieved full remission reported distress related to sleep disturbance (Zimmerman et al., 2005; 2012), suggesting that sleep disturbance may play a significant role in an individual’s subjective perception of functional impairment.

The emergence of transdiagnostic models of psychopathology, which aim to identify common processes or risk factors across multiple forms of psychopathology, represent a significant advancement in our understanding of comorbidity across disorders (Barlow, Allen, & Choate, 2004; Harvey, Watkins, Mansell, & Shafran, 2004; Hofmann, Sawyer, Fang, & Asnaani, 2012). More than half of individuals with one psychological disorder meet criteria for at least one other disorder (Kessler et al., 2005), and continued exploration of transdiagnostic processes may help us better understand the mechanisms that contribute to such high rates of comorbidity. If sleep disturbance mediates the causal pathway by which anxiety disorders increase the risk for incident depression, the implications for both comorbidity models and clinical practice would be significant.
Wittchen and colleagues (2003) estimate that if all anxiety disorders were successfully treated in individuals between the ages of 14 and 24, up to 43% of depressive episodes in early adulthood would be prevented. Other studies of sequential comorbidity suggest that childhood anxiety is a strong predictor of adolescent depression and advocate for further exploration of a causal anxiety-depression link (e.g., Schleider, Krause, & Gillham, 2014).

Concurrent and Sequential Comorbidity of Anxiety and Depression

Extensive comorbidity also exists between anxiety and depressive disorders. Comorbidity patterns assessed in a large sample of treatment-seeking adults using a semi-structured, diagnostic interview at an anxiety and related disorders clinic indicated that over half (63%) of patients with a principal diagnosis of a depressive disorder also received a current diagnosis of an anxiety disorder (Brown, Campbell, Lehman, Grisham, & Mancill, 2001). In another epidemiological study of over 1,700 adults, 67% of individuals with a depressive disorder had a current comorbid anxiety disorder and 63% of individuals with an anxiety disorder also met criteria for a current depressive disorder (Lamers et al., 2011). Extant studies suggest that comorbid anxiety and depressive disorders are associated with greater symptom severity, increased disability, greater chronicity, and greater risk of suicide (Bruce et al., 2005; Dolnak, 2006; Hirschfeld, 2001). Comorbid anxiety and depressive disorders are also associated with the highest productivity loss-related costs of all chronic illness, more days spent in bed due to illness, poorer performance at work, more somatic complaints, and high utilization of health services (Marciniak et al., 2005; McLaughlin, Khandker, Kruzikas, & Tummala, 2006; Wittchen, 2002), with GAD resulting in more economic costs than any other anxiety
disorder (Bereza, Machado, & Einarson, 2009). Perhaps of particular importance, comorbid depression results in poorer treatment outcome for anxiety disorders (Bruce et al., 2005; Campbell-Sills et al., 2012; Wittchen, Kessler, Pfister, & Lieb, 2000). The same is true in the reverse direction – comorbid anxiety results in a delayed or blunted response to both pharmacological (Andreescu et al., 2007; Fava et al., 2004) and psychological treatments for depression (Hirschfeld, 2001; Overbeek, Schruers, Vermetten, & Griez, 2002; Westen & Morrison, 2001).

Whereas the concurrent comorbidity of anxiety and depressive disorders is well documented (e.g., Burke et al., 2005; Kessler et al., 2005a, 2005b), the data on sequential comorbidity (i.e., when one disorder reliably predicts the onset of another disorder) is less straightforward due to some evidence of bidirectional relationships. However, both longitudinal (e.g., Burke et al., 2005; Merikangas et al., 2003; Wittchen, Kessler, et al., 2000) and retrospective studies (e.g., de Graaf, Bijl, Spijker, Beekman, & Vollebergh, 2003; Essau, 2003; Lamers et al., 2011) show that anxiety disorders most often precede depression. Indeed, while anxiety disorders are frequently present in the absence of depression, depression in the absence of comorbid anxiety is less common (e.g., Almeida et al., 2012).

Temporal Sequence of Anxious and Depressive Mood Symptoms

In addition to the high rates of concurrent and sequential comorbidity seen in anxiety and depressive disorders, anxiety symptoms are also significantly correlated with depressive symptoms, with greater overlap between these constructs observed at the symptom level than at syndrome or disorder levels (Hiller, Zaudig, & von Bose, 1989;
Preisig, Merikangas, & Angst, 2001). In a recent daily diary study, Starr and Davila (2012a) found that anxious and depressed mood were not only concurrently associated, but that daily anxious mood predicted later depressed mood; depressed mood did not reliably predict later anxious mood. These findings suggest that the temporal precedence of anxiety disorders over depression that has been observed both longitudinally and retrospectively may also present at the symptom level and over much shorter periods of time.

Given the temporal precedence of anxiety over depression at both the symptom and disorder level, it has been proposed that anxiety may serve as a risk factor for depression (e.g., Wittchen et al., 2003). Whether anxiety disorders truly represent a causal pathway by which vulnerability for depression develops requires further investigation, but a few studies have begun to explore the role of various transdiagnostic processes that may mediate the temporal precedence of anxiety over depression. In a prospective study that assessed undergraduate students over two time points, Grant and colleagues (2007) found that social anxiety at intake was associated with emotional avoidance, which predicted depressive symptoms one year later, even after controlling for depressive symptoms present at intake. Among a sample of treatment-seeking socially anxious adults, behavioral avoidance was shown to account for 41.9% of the effect of social anxiety symptoms on depressive symptoms at baseline, and changes in behavioral avoidance during treatment were significantly related to subsequent changes in depressive symptoms (Moitra, Herbert, & Forman, 2008). In a daily diary study of patients with GAD, daily cognitions about anxious mood (i.e., rumination about anxiety
symptoms, negative attributions about anxiety symptoms) moderated the concurrent association of daily anxious and depressed mood, such that anxious and depressed mood were more likely to co-occur on days when patients engaged in rumination or viewed their anxiety symptoms as more difficult to control (Starr & Davila, 2012b).

Before the temporal sequencing of anxiety and depression can be conceptually integrated into existing comorbidity models and used to inform treatment approaches, further research is needed on the mechanisms by which these temporal patterns occur. There is currently a discrepancy between the processes that have been hypothesized to mediate the relationship between anxiety and depression, and the methodology used to assess these relationships. Most of the proposed mediators discussed above (e.g., emotional avoidance, rumination, behavioral avoidance) are processes that occur over the course of very short time intervals (i.e., days or weeks), yet longitudinal studies of sequential comorbidity often involve annual assessments over the course of multiple years (e.g., Burke et al., 2005; Wittchen et al., 2003). Indeed, Mineka, Watson, and Clark (1998) proposed that the study of anxiety-depression comorbidity should begin with exploration of the co-occurrence of the symptoms that define the disorders. By evaluating mediators of daily symptom co-occurrence, we may be able to gain a more refined understanding of the microprocesses involved in the presentation of concurrent anxiety and depression, which may lead to more macro-level changes in our conceptualization of their comorbidity (Starr & Davila, 2012a).

Sleep Disturbance as a Transdiagnostic Process in Anxiety and Depression
Sleep disturbance is highly comorbid across many disorders and is consistently recognized as a core symptom in anxiety and depressive disorders (Buckner, Bernert, Cromer, Joiner, & Schmidt, 2008; Dolsen, Asarnow, & Harvey, 2014; Peterson & Benca, 2006). Up to 70% of patients with depression report experiencing sleep disturbance (e.g., insomnia, hypersomnia) that persists even in periods of remission and is associated with poorer treatment outcome (Medina, Lechuga, Escandon, & Moctezuma, 2014; Murphy & Peterson, 2015; Plante, 2015). Individuals with anxiety disorders commonly report sleep disturbance, including insomnia, nonrestorative sleep, and excessive daytime fatigue, that appears to exacerbate symptom severity (Cox & Olatunji, 2016). Comorbid anxiety and depression are associated with more severe insomnia complaints than depressive or anxiety disorders alone, and individuals with anxiety or depression and clinical levels of insomnia report more severe symptoms of anxiety, depression, and disability than those without insomnia (Mason & Harvey, 2014; Soehner & Harvey, 2012; Soehner, Kaplan, & Harvey, 2014).

Temporal sequencing of anxiety disorders, depression, and insomnia suggest that anxiety disorders most often precede insomnia, while insomnia tends to precede depression. In a retrospective study exploring the direction of risk between these constructs, Johnson and colleagues (2006) found that anxiety disorders preceded insomnia 73% of the time, while insomnia preceded the development of depression in 69% of cases; prior insomnia was not associated with the onset of anxiety disorders and prior depression was not associated with the onset of insomnia. The presence of any anxiety disorder was associated with increased risk of subsequent insomnia (OR: 3.5),
and prior insomnia was associated with onset of depression (OR: 3.8), even after adjusting for any prior anxiety disorders (Johnson et al., 2006). Other epidemiological studies of insomnia suggest that individuals presenting with symptoms of insomnia are between 2.6 (Baglioni et al., 2011) and 9.8 times (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005) more likely to develop depression than those without insomnia. Longitudinal studies demonstrate a similar relationship between anxiety disorders, insomnia, and depression (e.g., Jansson & Linton, 2006; Jansson-Frojmark & Lindblom, 2008). Taken together, these studies suggest that sleep disturbance may be an important etiological factor in the development of comorbid anxiety and depressive disorders, whereby anxiety leads to sleep difficulties, which in turn increase the vulnerability for depression.

The Role of Sleep Disturbance in Daily Anxious and Depressed Mood

There is evidence to suggest that even short-term manipulation of sleep impacts mood symptoms the next day. For example, sleep loss has been shown to both amplify negative emotions in response to disruptive events and to reduce positive emotions in response to goal-enhancing events experienced the following day in medical school residents (Zohar, Tzischinsky, Epstein, & Lavie, 2005). Healthy adults allowed only 4.5 hours in bed report more sleepiness and greater symptoms of depression than those who spent 7.5 hours in bed (De Valck & Cluydts, 2001). Following 35 hours of sleep deprivation, healthy adults demonstrate amplified amygdala reactivity to emotionally negative stimuli, suggesting that even acute sleep deprivation can result in poor emotion regulation the following day (Yoo, Gujar, Hu, Jolesz, & Walker, 2007). In an experience sampling study, poor subjective sleep quality predicted low positive affect and subjective
daytime dysfunction the next day in both patients with clinical levels of depression and healthy controls (Bower, Bylsma, Morris, & Rottenberg, 2010). In patients with bipolar disorder, the amount of time spent awake after sleep onset has been shown to predict negative mood the next morning (Talbot et al., 2012). These studies propose that disturbances in sleep quality, either subjectively reported or experimentally manipulated, result in reduced pleasurable experiences the following day, which over time may result in an increased risk for depression.

While it is more difficult to experimentally manipulate daily anxiety symptoms to assess the acute impact on sleep quality, some studies have explored the effects of cognitive arousal on sleep onset latency. Tang and Harvey (2004) randomized good sleeper controls to either an anxious cognitive arousal, neutral cognitive arousal, or a no manipulation condition prior to napping, and found that individuals in both arousal conditions overestimated their sleep onset latency compared to individuals in the no manipulation condition (i.e., they reported greater sleep onset latency that was not associated with objective sleep-onset delay as assessed by actigraphy). Another study found that worrying about the anticipated consequences of poor sleep before bed was the strongest predictor of delayed sleep onset measured with actigraphy (Wicklow & Espie, 2000). Unsurprisingly, individuals high in trait rumination tend to experience the most sleep interference when instructed to ruminate following a stressful life event (Guastella & Moulds, 2007).

In a sample of healthy controls, variability in sleep timing perceptions was most closely related to current symptoms of anxiety, whereas sleep satisfaction perceptions
demonstrated the strongest relationships with depression (Mayers, Grabau, Campbell, & Baldwin, 2009). In a comparison of patients with major depressive disorder and healthy controls, depressed patients reported poorer quality of life, poorer sleep quality, and lower mood, despite comparable sleep quality to healthy controls (Mayers, van Hooff, & Baldwin, 2003). It is particularly noteworthy that the presence of depressed mood exerted the strongest influence on sleep satisfaction in the presence of co-occurring anxiety, even though the symptoms of depression were sub-threshold (Mayers et al., 2009), implicating a unique interaction between symptoms of anxiety, depression, and sleep disturbance. These studies suggest that processes that contribute to the onset and maintenance of anxiety disorders (e.g., rumination or excessive worry, safety behaviors) may also contribute to sleep difficulties, which result in low mood and daytime dysfunction the following day. However, it remains unclear whether actual sleep disturbance (i.e., objectively measured sleep parameters) or perceived sleep disturbance, or a combination of both, is most strongly related to low mood and daytime dysfunction the following day.

**Rationale for the Proposed Study**

GAD is the anxiety disorder most strongly associated with depression (Kessler et al., 2005b; Wittchen et al., 2003). GAD is also one of only two anxiety disorders where sleep difficulty is explicitly recognized as a diagnostic criterion (American Psychiatric Association, 2013). Among individuals with insomnia and comorbid anxiety disorders, GAD is the most commonly reported anxiety disorder in both primary care patients (Marcks, Weisberg, Edelen, & Keller, 2010) and community samples (Ramsawh, Stein, Belik, Jacobi, & Sareen, 2009). Patients with GAD report decreased total sleep time,
poorer sleep efficiency, and more frequent early morning awakenings (Guille, Cortese, & Uhde, 2012). Across six different polysomnography studies, a diagnosis of GAD was associated with an average of 32.8 more minutes spent awake after falling asleep and a decrease of 54.8 minutes in total sleep time (Monti & Monti, 2000).

While all anxiety disorders share commonalities with one another and high rates of comorbidity with depression, it is certainly possible that different causal pathways are associated with different anxiety disorders. Research on temporal sequencing of symptoms within disorders is exceptionally rare, and the relationship between nightly sleep difficulties and daily symptoms of anxious and depressed mood has yet to be systematically evaluated. Given that there is already preliminary evidence that daily anxious mood predicts later depressed mood in patients with GAD and a history of depressive symptoms (Starr & Davila, 2012a), exploring the relationship between anxiety, depressed mood, and sleep disturbance in a analogous sample (i.e., patients with GAD and a history of depressive symptoms) is a reasonable starting point.

**Proposed Moderators**

As discussed earlier, a few studies have begun to explore possible moderators of the comorbidity between anxiety and depressive disorders, including rumination (McEvoy, Watson, Watkins, & Nathan, 2013; Spinhoven, Drost, van Hemert, & Penninx, 2015; Starr & Davila, 2012a) and behavioral avoidance (Moitra et al., 2008). Exploring potential moderators of the functional relationships between anxiety, sleep disturbance, and depressed mood at the within-subject level is important because it may shed greater
light on the co-occurrence of these symptoms, particularly with regard to markers of vulnerability for symptom co-occurrence and the identification of treatment targets.

One proposed moderator of the relationship between sleep disturbance and anxiety and depressive disorders is the presence of faulty sleep-related cognitions (i.e., maladaptive beliefs and appraisals about sleep, unrealistic expectations). Dysfunctional beliefs about sleep are considered a perpetuating factor in the cognitive model of insomnia (Harvey, 2002a), and have demonstrated significant relationships with subjective report of insomnia symptoms, anxiety, and depression, but not with specific sleep parameters (i.e., sleep diary data or polysomnography) or specific insomnia subtypes (Carney et al., 2010a; Morin, Vallieres, & Ivers, 2007; Park, An, Jang, & Chung, 2012). High levels of maladaptive beliefs about sleep have been shown to differentiate between individuals with insomnia and individuals with either depression or comorbid insomnia and depression, with much of the variability in dysfunctional beliefs accounted for by depression severity (Carney, Edinger, Manber, Garson, & Segal, 2007). These studies suggest that dysfunctional beliefs about sleep may represent a salient treatment target for individuals with anxiety or depression who are also reporting sleep disturbance.

Another perpetuating factor in insomnia is the presence of sleep-related safety behaviors (Harvey, 2002a, 2002b); examples include attempts to catch up on sleep by napping, going to bed early, or conserving energy during the day (Ree & Harvey, 2004). Safety behaviors, which are strategies employed to prevent the occurrence of a feared outcome, are seen in psychological disorders, particularly in individuals with anxiety
disorders. Safety behaviors contribute to the maintenance of symptoms because they prevent disconfirmation of maladaptive beliefs (Salkovskis, 1991). Sleep-related safety behaviors are associated with both sleep disturbance and daytime dysfunction, and have been shown to mediate the efficacy of CBT for insomnia (Lancee, Eisma, van Straten, & Kamphuis, 2015). In an outpatient sample of adults with anxiety and depressive disorders, sleep-related safety behaviors were strongly associated with subjective reports of sleep disturbance and fatigue (Fairholme & Manber, 2014). There is also evidence that individuals with higher levels of depression tend to report greater utilization of sleep-related safety behaviors (Woodley & Smith, 2006), again suggesting that safety behaviors related to sleep influence symptoms of sleep disturbance in individuals with anxiety or depression.

While maladaptive beliefs about sleep and sleep-related safety behaviors represent specific cognitive and behavioral processes that may influence the functional relationship between anxiety, sleep disturbance, and depressed mood, more generalized vulnerabilities may also contribute to the co-occurrence of these symptoms. Neuroticism is a temperamental style associated with an enduring tendency to experience negative emotions, and appears to be a generalized biological vulnerability to experience anxiety or depression (Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014). There is also evidence to suggest that individuals higher in neuroticism are more predisposed to experience sleep disturbance (Duggan, Friedman, McDevitt, & Mednick, 2014; Gurtman, McNicol, & McGillivray, 2014). In an undergraduate sample, anxiety sensitivity, dysfunctional beliefs about sleep, and neuroticism were all significantly related to sleep

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disturbance, but neuroticism emerged as the most significant predictor in a stepwise regression (Calkins, Hearon, Capozzoli, & Otto, 2013). A longitudinal study that followed participants from birth to midlife found that although neuroticism assessed in adolescence did not predict the onset of sleep difficulties, sleep difficulties in adolescence and into young adulthood predicted midlife neuroticism levels and continued sleep difficulties (Danielsson, Jansson-Frojmark, Linton, Jutengren, & Stattin, 2010). Despite ample empirical evidence supporting the contribution of neuroticism as a generalized biological vulnerability to emotional disorders, very few studies to date have explored the contribution of neuroticism to sleep difficulties, and the vast majority of existing studies have relied on cross-sectional or retrospective data.

**Hypotheses**

The current study aimed to determine whether the sequential comorbidity patterns observed at the disorder-level are also present at the symptom-level, as well as to explore possible moderators of this relationship, which would not only provide support for the inclusion of sleep disturbance as an explicit target in existing treatment protocols, but also inform approaches for addressing sleep disturbance from a prevention framework.

**Aim 1.** To examine the temporal associations among anxiety, sleep disturbance, and depressed mood as assessed by daily symptom ratings and actigraphy over a monitoring period of 14 days.

**Hypothesis 1.1.** Anxiety at $t-1$ will be significantly related to prospective sleep disturbance at $t$ as assessed by daily symptom ratings and actigraphy.
Hypothesis 1.2. Sleep disturbance at $t$-1 will be significantly related to depressed mood at $t$ as assessed by daily symptom ratings during the following day.

Aim 2. To examine potential moderators of any temporal associations among daily symptoms of anxiety, sleep disturbance, and depressed mood.

Hypothesis 2.1. Dysfunctional beliefs about sleep will predict stronger associations among anxiety, sleep disturbance, and depressed mood.

Hypothesis 2.2. Sleep-related safety behaviors will predict stronger associations among anxiety, sleep disturbance, and depressed mood.

Hypothesis 2.3. Neuroticism and negative affect will predict stronger associations among anxiety, sleep disturbance, and depressed mood.

Methods

Participants

Participants for the current study consisted of a clinical sample of adults between the ages of 18 and 65 with a principal diagnosis of GAD and a history of depressive symptoms, and a control sample of good sleeper, healthy subjects. A total of 33 participants were enrolled in the study to obtain sufficient data for 30 participants (15 healthy controls, 15 patients with GAD). Recruitment flow is presented in Figure 1 and Figure 2 for the control and clinical samples, respectively. Individuals for the clinical sample were recruited through the Center for Anxiety and Related Disorders (CARD) and healthy control participants were recruited through the community via online advertisements. Participants recruited through CARD were treatment-seeking adults who completed a diagnostic assessment as part of routine Center practices.
Exclusion criteria for all participants included: 1) inability to speak and understand English; 2) inability or unwillingness to provide informed consent; 3) serious medical illness for which hospitalization may be likely within the next three months; 4) active suicidal or homicidal ideation, or suicide attempts within the past six months requiring hospitalization; 5) history of serious head injury or unexplained loss of consciousness; 6) seizure disorder; 7) use of prescription sleep medications (e.g., Lunesta, Ambien), anxiolytics used as sleep aids (e.g., Ativan) or dependence on over-the-counter sleep aids (e.g., Tylenol PM, Benadryl, ZzzQuil, melatonin dietary supplements); 8) unwillingness to maintain stable medication dosage throughout 14-day monitoring period; 9) coffee consumption (or equivalent) of > four cups in a 24 hour period for the duration of the 14-day monitoring period; and 10) alcohol consumption of >14 drinks per week for the duration of the 14-day monitoring period. Participants were asked to abstain from marijuana use for the duration of the 14-day monitoring period, minimize caffeine intake after 4:00 PM, and to avoid consuming alcohol within two hours of bedtime.

The following additional exclusion criteria applied to healthy participants: 1) any current depressive disorder, including seasonal major depressive episodes, anxiety disorder, obsessive-compulsive or related disorder, trauma- or stressor-related disorder, somatic symptom or related disorder, and substance-related or addictive disorders, or schizophrenia spectrum or other psychotic disorder; 2) total score of 5 or greater on the insomnia screening measure; and 3) current symptoms consistent with diagnosis of sleep disorder. The following additional exclusion criteria applied to participants with GAD: 1)
schizophrenia spectrum or other psychotic disorder, bipolar or related disorder, mental
disorder due to a general medical condition or substance; 2) no hospitalization for
depression within the past year; and 3) current symptoms consistent with diagnosis of
sleep disorder other than insomnia. In addition, participants with GAD had to endorse a
history of at least one of the two cardinal symptoms of depression (i.e., significant
depressed mood or loss of interest in almost all usual activities). They were also required
to meet the following stabilization criteria for psychotropic medications: 1) six weeks for
initiation or dosage change and four weeks for discontinuation of antidepressants or
stimulants; and 2) four weeks for initiation and two weeks for dosage change or
discontinuation of benzodiazepines or beta-blockers. Comorbid, current diagnoses of
major depression, persistent depressive disorder, or any anxiety disorder were permitted
as long as GAD was the principal diagnosis.

Measures

Diagnostic Assessment

After obtaining informed consent, the following measures were administered to
determine study eligibility.

Anxiety and Related Disorders Schedule for DSM-5: Lifetime version

(ADIS-5-L; Brown & Barlow, 2014). The ADIS5-L is a semi-structured interview
designed to assess for DSM-5 diagnoses of anxiety disorders, obsessive-compulsive and
related disorders, trauma- and stressor-related disorders, depressive disorders, somatic
symptom and related disorders, and substance-related and addictive disorders. The
investigator administered a modified version of the ADIS-5 that assessed for current
diagnoses to patients and healthy controls, unless a patient had already undergone the diagnostic assessment through CARD. The ADIS-5 allowed the investigator to assess all diagnostic inclusion and exclusion criteria for both GAD patients and healthy controls.

**Insomnia Severity Index** (ISI; Bastien, Vallieres, & Morin, 2001). The ISI is a 7-item self-report instrument that measures sleep impairment, distress, and daytime impairment. ISI scores converge closely with polysomnography and are frequently used as a screening tool or outcome measure in behavioral sleep medicine. A clinical cut-off score of 10 was determined to be optimal for the detection of significant insomnia symptoms in a community sample (Morin, Belleville, Belanger, & Ivers, 2011).

**Sleep Disorder Assessment.** The Sleep Disorder Assessment is a 38-item self-report measure designed to quickly and efficiently screen for the presence of sleep disorders, including sleep apnea, insomnia, narcolepsy, gastroesophageal reflux, and periodic limb movement disorder.

**Self-Report Measures**

**Dysfunctional Beliefs and Attitudes about Sleep Scale, Brief Version** (DBAS-16; Morin et al., 2007). The DBAS-16 is a 16-item self-report measure of sleep-related beliefs comprised of four factors: medication, worry/helplessness about insomnia, expectations for sleep, and perceived consequences of insomnia. The DBAS-16 has demonstrated acceptable internal consistency and temporal stability.

**Pittsburgh Sleep Quality Index** (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is a 19-item self-report measure designed to assess sleep quality and disturbances in a clinical population retrospectively over the past month. It yields one
global score and seven component scores: sleep latency, subjective sleep quality, sleep duration, habitual sleep efficiency, use of sleeping medication, sleep disturbances, and daytime dysfunction. The PSQI has demonstrated adequate internal consistency and construct validity (Carpenter & Andrykowski, 1998).

**Sleep-Related Behaviours Questionnaire** (SRBQ; Ree & Harvey, 2004). The SRBQ is a 32-item self-report measure that assesses the use of behavioral strategies used to cope with daytime fatigue and to improve sleep. The SRBQ has demonstrated strong reliability and has been shown to successfully differentiate between poor and good sleepers (Ree & Harvey, 2004).

**Fatigue Severity Scale** (FSS; Krupp, LaRocca, Muir-Nash, & Steinberg, 1989). The FSS is a 9-item self-report measure of perceived fatigue severity and associated interference. It is the most commonly used fatigue specific questionnaire (Hjollund, Andersen, & Bech, 2007), and has demonstrated excellent internal consistency and reliability (Valko, Bassetti, Bloch, Held, & Baumann, 2008).

**Sleep Habits and Hygiene Checklist** (SHHC). The SHHC is an unpublished self-report checklist designed to assess the frequency with which an individuals engages in poor sleep hygiene behaviors (e.g., daytime napping, watching television in bed).

**The Morningness-Eveningness Composite Scale** (MECS; Horne & Ostberg, 1976). The MECS is a brief, 13-item self-report measure designed to assess individual variability in preferences for morning or evening activities (i.e., morning and evening chronotypes). Evening chronotypes have demonstrated stronger relationships with depression severity and sleep variability than morning chronotypes (Suh et al., 2012).
Beck Anxiety Inventory (BAI; Beck & Steer, 1990). The BAI is a 21-item, brief self-report inventory that measures the severity of anxiety symptoms and it has demonstrated strong psychometric validity and reliability. The BAI is especially effective for differentiating between symptoms of anxiety and depression.

Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). The BDI is a 21-item self-report inventory that measures the severity of depressive symptoms and it has shown good convergent and discriminant validity, and high internal consistency (Coles, Gibb, & Heimberg, 2001).

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a 16-item measure designed to assess the trait of worry. It has been shown to differentiate between GAD-diagnosed clinical subjects and healthy controls, and between GAD and other anxiety disorders (Brown, Antony, & Barlow, 1992).

Work and Social Adjustment Scale (WSAS; Marks, Connolly, & Hallam, 1973; Mundt, Marks, Shear, & Greist, 2002). The WSAS is a 5-item self-report measure of symptom interference in the domains of work, home management, private leisure, social leisure, and family relationships.

Positive Affect and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a self-report measure comprised of two 10-item mood scales that assess positive and negative affectivity. The scales have demonstrated adequate internal consistency and stability over a two-month period.

Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI is a 16-item self-report measure designed to assess fear of anxiety-related sensations, which consists
of three lower-order factors: fear of physical symptoms, mental incapacitation, and social concerns (Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004). The ASI has demonstrated strong internal consistency (Peterson & Reiss, 1992) and discriminant validity with measures of trait anxiety (Sandin, Chorot, & McNally, 2001).

**Quality of Life Enjoyment and Satisfaction Questionnaire - Short Form** (Q-LES-Q-SF; Endicott, Nee, Harrison, & Blumenthal, 1993). The Q-LES-Q is a brief, 15-item measure designed to assess enjoyment and satisfaction in a number of life domains. It is one of the most commonly used outcomes measures in psychological research and has demonstrated good internal consistency, test-retest reliability, and validity (Stevanovic, 2011).

**State-Trait Anxiety Inventory, Trait Version** (STAI-T; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The STAI-T is a 20-item self-report measure that assesses more stable, trait-level anxiety using a 4-point Likert scale. The STAI-T is the most widely used self-report measure of anxiety and has well-supported construct and concurrent validity (Spielberger, 1989).

**NEO Five-Factor Inventory** (NFFI; Costa & McCrae, 1992). The NFFI is a 60-item self-report measure of the five-factor model of personality. Only the 12-item Neuroticism scale will be administered, and it will be used to assess levels of neuroticism, which have been linked to sleep disturbance (Danielsson et al., 2010; Duggan et al., 2014; Gurtman et al., 2014).

**Weekly Stress Inventory, Short Form** (WSI-SF; Brantley et al., 2007). The WSI-SF is a 25-item self-report scale designed to measure minor stressors. Participants
rank items on an 8-point Likert scale to indicate whether a particular stressful event occurred within the past week, and if so, how much distress it caused; the WSI-SF yields an event score (i.e., occurrence) and an impact score (i.e., associated distress). The WSI-SF has demonstrated good internal consistency for both scores (Brantley et al., 2007). The WSI-SF will be administered at baseline, the end of week 1, and the end of week 2 to capture psychosocial stressors experienced during the past week.

**Daily Symptom Ratings**

Daily symptom ratings were completed using ecological momentary assessment (EMA), which is an alternative to retrospective self-report assessment, that collects data in real-time, in real-world settings, and typically across multiple repeated time points (Shiffman, Stone, & Hufford, 2008; Trull & Ebner-Priemer, 2009). EMA is particularly well suited to capture data on individual differences, relationships between multiple factors, and how these relationships change over time (Shiffman et al., 2008). Participants were prompted to provide symptom ratings six times a day (i.e., upon awakening, mid-morning, noon, mid-afternoon, evening, prior to bedtime); prompts were programmed based on the participant’s typical bedtime and wakeup times. The remaining four ratings were delivered at regular intervals throughout the day.

In order to facilitate compliance with daily symptom ratings, questions were designed to assess the relevant variables quickly and efficiently. Based on previous daily diary studies (e.g., Starr & Davila, 2012a, 2012b), brief, face-valid items (e.g., “How (anxious/depressed) do you feel right now?”) were used to assess anxious and depressed mood on a 10-point Likert scale (1 = not at all, 10 = extremely). In order to capture both
cardinal symptoms of depression (i.e., low mood, anhedonia), participants were also asked once at the end of the day whether they experienced diminished interested or pleasure in the activities they usually enjoy.

**Subjective Sleep Measures**

Subjective sleep quality was assessed using items from the Consensus Sleep Diary (CSD; Carney et al., 2012). The CSD is a standardized sleep diary that assesses the time of getting into bed, the time at which the participant attempted to fall asleep, sleep onset latency, number and duration of awakenings, time of final awakening, final rise time, and perceived sleep quality. It also includes items to be completed in the evening, including napping or use of alcohol, caffeine, or medications.

Daytime dysfunction related to sleep difficulty was assessed using the Daytime Insomnia Symptom Scale (DISS; Buysse et al., 2007). The DISS is a 20-item self-report measure that assesses symptoms of daytime dysfunction related to insomnia, and yields four principal component scores: alert/cognition, negative mood, positive mood, and sleepiness/fatigue. The DISS was designed and piloted using EMA, and thus was appropriate for the aims of the current study.

**Objective Sleep Measures**

Participants wore an actigraph, the Actiwatch 2, continuously on the wrist of the non-dominant hand during the 14-day period. Actigraphy is a relatively non-invasive method for the monitoring of sleep/wake cycles. Actigraphs resemble wristwatches and are water-resistant to allow for continuous use for weeks at a time. Most research supports actigraphy as highly appropriate for the assessment of sleep variability from
night-to-night in patients with insomnia (e.g., Ancoli-Israel et al., 2003). Activity data were sampled in 30-second epochs using a medium wake threshold selection and analyzed with the Actiware software program. Participants were instructed to press an event marker button to signify when they began trying to fall asleep and when they awoke for the final time in the morning. In the absence of event markers, daily sleep diary data were utilized to determine the start and end of each rest interval. Each actigraphy record was visually inspected to ensure that participants’ reported bed and rise times were consistent with observed activity and ambient light patterns. Using these parameters, the Actiware software provided ratings of sleep onset latency, total sleep time, and wake after sleep onset. Sleep efficiency was calculated using total sleep time and time spent in bed.

**Procedure**

Potential participants completed a brief phone screen to determine preliminary eligibility criteria as reflected by the inclusion and exclusion criteria listed above. Participants received an overview of study procedures, including length of monitoring period (14 days), use of an actigraph to monitor sleep, and daily symptom ratings. Participants who met preliminary eligibility criteria were then scheduled for an enrollment appointment (Study Visit 1).

For GAD patients referred through CARD, Study Visit 1 was approximately 45 minutes to one hour. Participants then completed a measure of insomnia symptom severity to ensure that sufficient sleep interference was present, as well as a screening measure to determine whether a sleep disorder may be present. Study Visit 1 for healthy
controls lasted approximately 90 minutes due the administration of a semi-structured diagnostic interview in addition to the sleep-related screening measures. All participants completed a self-report battery and then were oriented to the actigraph. Next, participants downloaded the SymTrend application and were instructed how to use the application to complete daily symptom ratings on their smartphones. Participants were given a choice between completing the daily symptom ratings on their own smartphone or a smartphone provided by the study.

Following completion of the 14-day monitoring period, participants were scheduled to return to the Center for Study Visit 2, which lasted approximately 15 minutes. During Study Visit 2, participants returned the actigraph and smartphone if applicable and were given an opportunity to report any deviations from the study protocol. Study adherence data were reviewed with each participant and a preferred mailing address for receipt of payment was obtained. Participants were compensated $15 for each day where compliance with symptom ratings reaches or exceeds 85% for a maximum compensation of $210. All participants provided written informed consent and all study procedures were approved by the Institutional Review Board at Boston University.

Results

Adherence to Daily Symptom Ratings

Participants were prompted via text message or email to complete symptom ratings at six points throughout the day: morning, mid-morning, lunch, afternoon, evening, and bedtime. At each of these time points, participants were asked to complete the daily symptom ratings (i.e., the DISS and single-item, face-valid measures of anxiety
and depression), which consisted of 22 items. At bedtime, the daily symptom ratings included two additional questions that assessed 1) level of anhedonia experienced over the course of the day, and 2) participants’ rating of how much the prior evening’s sleep interfered with their functioning over the course of the day. Participants were also prompted to complete the morning and evening sleep diaries upon awakening and prior to bed, respectively. Therefore, adherence was calculated based on completion of eight questionnaires throughout the day and 112 questionnaires over the course of 14-day study. Participants completed, on average, 93.24% ($SD = 7.72$, range = 62 – 100) of the daily symptom ratings and earned an average of $191.50 ($SD = 28.32$, range = 75 – 210) of the $210 maximum compensation. There were no differences between the clinical sample and healthy controls on adherence to daily symptom ratings or amount of compensation received.

**Demographic and Clinical Data**

Demographic information is presented for each sample in Table 1. The average age of participants, collapsed across groups was 28.00 years ($SD = 8.97$, range = 18 – 53). Both groups were primarily female ($n = 21$), Caucasian ($n = 22$), and single ($n = 22$). Participants were well educated, with the vast majority completing at least some college education ($n = 28$). There were no significant differences between groups on any of the demographic variables, although the relationships between group condition and both marital status ($\chi^2 = 10.91, p = .012$) and level of education ($\chi^2 = 12.80, p = .012$) approached statistical significance.
Clinical characteristics of each sample are presented in Table 2. For the clinical sample, the average clinical severity rating (CSR) for the principal diagnosis of GAD was 5.27 (SD = .80, range = 4 - 7) and participants had an average of 1.2 additional diagnoses (SD = .86, range = 0 - 2). The three most common comorbid diagnoses were social anxiety disorder (n = 5), persistent depressive disorder (n = 5), and major depressive disorder (n = 2), with approximately half (46.7%) of the participants meeting criteria for an additional current diagnosis of a depressive disorder (i.e., major depressive disorder, persistent depressive disorder). Consequently, although a principal diagnosis of GAD was a primary inclusion criterion for the study, the clinical sample contained diagnostic heterogeneity that paralleled the comorbidity patterns typically observed in patients with GAD (e.g., Kessler at al., 2005).

Only two participants from the GAD sample were on psychotropic medication; both participants were prescribed stimulants. The clinical sample reported symptoms consistent with moderate levels of depression, anxiety, and anxiety sensitivity, as well as severe pathological worry and trait anxiety. Levels of negative affect were similar to reported norms for psychiatric samples, but levels of positive affect were over a full standard deviation lower than published psychiatric norms (e.g., Clark & Watson, 1991). As expected, participants in the clinical sample scored significantly higher than healthy controls across all measures of depression, anxiety, and functional impairment.

Sleep data for each sample are presented in Table 3. On the screening measure of insomnia severity (ISI), more than half of GAD participants (n = 9) reported moderate (or more severe) difficulty with sleep onset latency, the majority (n = 13) endorsed difficulty
with sleep maintenance, and approximately half \( n = 8 \) reported regularly waking up earlier than intended. Global scores on the PSQI, a measure of subjective sleep quality, were well over the clinical threshold of 5 \( (M = 10.67, SD = 1.88) \) for GAD participants. Across each of the PSQI component scores, the GAD sample reported significantly greater impairment than healthy controls. Average scores for perceived daytime fatigue \( (FSS; \text{mean} = 41.40, SD = 10.25) \) were also elevated above the suggested clinical threshold of 36. Participants in the clinical sample reported average levels of dysfunctional beliefs about sleep \( (DBAS) \) and sleep-related safety behaviors \( (SRBQ) \) that were comparable to those observed in insomnia samples. Compared to the control group, participants with GAD endorsed engaging in more behaviors associated with poor sleep hygiene and engaging in those behaviors more frequently within the past month. Across both groups, the majority of participants \( n = 26 \) identified as intermediate type, which reflects a circadian typology that is neither morning- nor evening-dominant.

**Daily Symptom Ratings**

Table 4 presents the daily averages of each group for each subscale of the DISS (alert/cognition, sleepiness/fatigue, positive mood, negative mood) and the face-valid items \( \text{e.g., “How (anxious/depressed) do you feel right now?”} \), as well as the end-of-day questions that assessed experience of anhedonia and the degree to which the previous evening’s sleep interfered with functioning throughout the day. As expected, the GAD sample reported significantly greater levels of daily negative mood, anxiety, depression, anhedonia, daytime fatigue, and sleep-related interference than the control group. GAD
participants also reported significantly lower levels of daily positive mood and alertness compared to healthy controls.

Sleep Disturbance

Table 5 presents the averages for the daily self-reported ratings of sleep disturbance and actigraphy outcomes across the 14-day study by group. Consistent with expectations, the GAD sample reported poorer quality of sleep and feeling less rested than the control sample. For the actigraphy outcomes, there were no differences between groups in the amount of sleep received each evening or how long it took to fall asleep. However, the control group appeared to spend significantly more time awake after sleep onset and demonstrated lower sleep efficiency than the GAD group. Specifically, healthy controls spent approximately 9 more minutes awake after sleep onset (mean = 37.96 minutes, SD = 28.08 vs mean = 29.05, SD = 16.67) and slept approximately 3% less efficiently than GAD participants (mean = 83.17, SD = 11.13 vs 86.33, SD = 7.89).

Table 6 presents correlational analyses between subjective and objective sleep ratings. Both measures of subjective sleep were highly correlated with each other for the control group (r = .68, p < .001) and the GAD group (r = .60, p < .001). For the control group, self-reported ratings of sleep quality were significantly related to greater total sleep time, more efficient sleep, and lower sleep onset latency, while self-reported ratings of feeling rested were associated solely with greater total sleep time. In comparison, subjective sleep measures were not related to any of the objective sleep measures for the GAD group.

Temporal Instability
A unique aspect of time series or intensive longitudinal data is the ability to explore the temporal instability of time-varying phenomena, as extreme and frequent fluctuations in symptomology can be characteristic of psychopathology in and of itself. Temporal instability reflects not only high variability, but also low temporal dependency of data (Jahng, Wood, & Trull, 2008). To explore the temporal instability of sleep parameters and daily symptom ratings, mean square successive differences (MSSDs) were computed by squaring the difference between consecutive measurements. Since there were 14 days of monitoring in the current study, 13 square successive differences (SSDs) were computed in total and then a mean value was derived (MSSD). Since the MSSD is computed using successive change, the resulting value reflects both variability and temporal dependency and is thus recommended as the gold-standard for capturing temporal instability in both affective states and sleep behaviors (Sanchez-Ortuno, Carney, Edinger, Wyatt, & Harris, 2011; Suh et al., 2012).

Contrary to expectations, there were no between-group differences observed in temporal instability for any of the subjective sleep ratings (i.e., overall sleep quality, feeling rested upon awakening) or the actigraphy-derived sleep parameters (i.e., sleep onset latency, wake after sleep onset, sleep efficiency, total sleep time). However, the clinical sample demonstrated significantly greater instability than the control sample on daily depressed mood ($t(15.97) = -2.30, p = .035$; Figure 3), daily anxious mood ($t(23.75) = -2.68, p = .013$; Figure 4), anhedonia ($t(27.12) = -3.42, p = .003$; Figure 5), interference in functioning attributed to sleep ($t(14.47) = -2.72, p = .016$; Figure 6), and the cognition/alertness scale of the DISS ($t(18.63) = -2.13, p = .047$; Figure 7). Group
differences in temporal instability approached statistical significance for the sleepiness/fatigue ($t(21.62) = -2.01, p = .057$; Figure 8) and negative mood ($t(25.34) = -2.01, p = .055$; Figure 9) scales of the DISS.

It is noteworthy that in comparison to healthy controls, participants with GAD experienced both higher overall levels of symptoms and greater day-to-day variability in the level of symptoms experienced. Moreover, there was less temporal dependency between days, suggesting that the levels of symptoms experienced at $t-1$ were not strongly correlated with the level of symptoms experienced at $t$.

**Statistical Analysis**

To examine study aims, multi-level models (MLM), with daily symptoms nested within individuals, were constructed for each group. MLM is well equipped to handle data that is missing at random, which makes it an ideal approach for intensive longitudinal data (Fitzmaurice, Laird, & Ware, 2004). For the present study, adherence to daily symptom ratings was not predicted by group status, predictor variables, or outcomes, suggesting that data were indeed missing at random and that an MLM framework was appropriate. In addition, full maximum likelihood estimation was specified for analyses, which utilizes all available data to calculate between- and within-person level parameters and the associated standard errors.

Models for the temporal analyses were built in a series of steps. Level 2 equations were specified to model *between-person* variability and level 1 equations were specified to model *within-person* variability. Accordingly, level 2 effects were estimated using each participant’s mean of the predictor variable across 14 days (i.e., one value) and level
1 effects were estimated using each participant’s centered daily score (i.e., daily score – participant’s mean; 14 values), and therefore reflected an individual participant’s deviation from his or her mean. Next, lagged predictor variables were computed to examine associations among contemporaneous time-varying constructs. Time lags were computed for up to four days to determine peak significance of associations, where a one-day lag reflected a predictor variable at \( t-1 \) predicting an outcome at \( t \), a two-day lag reflected a predictor variable at \( t-2 \) predicting an outcome at \( t \), and so on. Multiday lags were aggregated so that the lag represented a cumulative effect of the predictor variable over time (i.e., four-day lag predicting outcome, \( t = \Sigma (predictor_{t-4}, predictor_{t-3}, predictor_{t-2}, predictor_{t-1}) \)).

An autoregressive (AR[1]) repeated covariance type was specified to control for auto-correlations of residuals (Rovine & Walls, 2006). Autoregressive modeling treats the value of an outcome at each measurement occasion as a separate dependent variable and the prior measurement as the predictor, along with any other specified covariates, in the regression model. Time was represented by each day of the study, where day 1 of the study represented time 0, which anchored all level 2 effects at the first day of the study. Time was entered as both a fixed and random effect; the fixed effect of day estimated the mean association of time at the between-person level and the random effect estimated individual differences in the effect of time on the outcome of interest. The inclusion of time as a fixed effect in the model provided a more robust assessment of study aims since it prevents the model from estimating spurious temporal relationships between the predictor variables and the dependent variable. Therefore, time was retained as a fixed
effect in subsequent models regardless of significance. Due to the lagged nature of the analyses, only the intercept and time were entered as random effects.

**Concurrent Associations Between Anxious and Depressed Moods**

Anxious and depressed moods were concurrently associated for both the GAD group \( (B = .36, SE = .05, p < .001) \) and the control group \( (B = .30, SE = .04, p < .001) \), suggesting that individuals typically experienced greater depressed mood on days when they also felt greater anxiety.

**Does Anxious Mood Predict Later Sleep Disturbance?**

To evaluate whether anxious mood was associated with prospective sleep disturbance, level 1 and level 2 fixed effects were estimated for both subjective sleep ratings (i.e., overall sleep quality, how rested the participant felt upon awakening) and actigraphy sleep parameters (i.e., total sleep time, sleep onset latency, sleep efficiency, wake after sleep onset). The level 1 effect assessed the association between participants’ day-to-day deviations from their mean level of anxiety and day-to-day variation in sleep (i.e., do participants experience greater sleep disturbance following days that they experience higher-than-average anxiety?). The level 2 effect assessed the association between participants’ means levels of anxiety and sleep (i.e., do participants with higher levels of anxiety experience greater sleep disturbance than participants with lower levels of anxiety?).

Table 7 displays the results for anxious mood predicting subsequent subjective sleep disturbance at different time lags for each group. For participants with GAD, there were no level 2 (between-person) effects observed for mean levels of anxious mood on
prospective sleep disturbance. Level 1 (within-person) results demonstrated that anxious mood was associated with subjective ratings of both overall sleep quality and feeling rested upon awakening at both a one- and two-day lag. However, the magnitude of the effect and statistical significance of the association between anxious mood and these subjective ratings were highest at a one-day lag, suggesting that when GAD participants experienced above-average levels of anxious mood, they rated their sleep quality as poorer and reported feeling less rested upon awakening the following morning. In comparison, there were no level 1 effects observed in the control group. There was a level 2 effect of anxious mood on ratings of feeling rested upon awakening, such that healthy controls who reported higher levels of mean anxious mood also reported feeling less rested.

Table 8 displays the results for anxious mood predicting actigraphy sleep parameters at different time lags for each group. There were no level 2 (between-person) effects of anxious mood on sleep parameters for either group. For the GAD group, above-average anxious mood at $t{-}1$ was associated with lower sleep efficiency, more time spent awake after initial sleep onset, and less total sleep time at $t$. Among healthy controls, there was a negative linear temporal association for sleep onset latency, suggesting that these individuals experienced a decrease in the amount of time it took to fall asleep over the course of the study.

To determine whether the significant associations observed between anxious mood at $t{-}1$ and sleep outcomes at $t$ were independent of any associations with depressed mood, additional models were constructed for the GAD group. Mean levels of depressed

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mood were included as a level 2 (between-person) effect and depressed mood values at \(t-1, t-2, t-3,\) and \(t-4\) were included as a level 1 (within-person) effect. Anxiety at \(t-1\) remained significantly associated with self-reported sleep quality at \(t (B = -.35, SE = .13, p = .008)\), subjective ratings of feeling rested at \(t (B = -.28, SE = .13, p = .026)\), total sleep time at \(t (B = -35.40, SE = 12.63, p = .006)\), and time spent awake after sleep onset at \(t (B = -4.50, SE = 2.26, p = .049)\); there was no effect of depressed mood at level 1 or level 2. Although the effect of anxious mood at \(t-1\) on sleep efficiency at \(t\) was no longer significant once depressed mood was included in the model \((B = -.02, SE = .01, p = .115)\), depressed mood was independently associated with sleep efficiency.

**Does Sleep Disturbance Predict Later Depressed Mood?**

To evaluate whether sleep disturbance was associated with depressed mood, level 1 and level 2 fixed effects were estimated using both subjective sleep ratings (i.e., overall sleep quality, how rested the participant felt upon awakening) and actigraphy parameters (i.e., total sleep time, sleep onset latency, sleep efficiency, wake after sleep onset) as lagged predictor variables. Each model contained the four lagged predictor variables as level 1 (within-person) effects and the 14-day mean of the predictor variable as a level 2 (between-person) effect. Based on previous analyses that demonstrated a significant effect of anxious mood at \(t-1\) on sleep disturbance at \(t\), anxious mood with a one-day lag was included as residual variance in the models estimating the effect of sleep disturbance at \(t-1\) on depressed mood at \(t\).

Table 9 displays the results for subjective sleep disturbance predicting later depressed mood at different time lags for each group. Average sleep quality was
associated with depressed mood at the between-person level for both the GAD ($B = -2.23$, $SE = .94$, $p = .025$) and control group ($B = -.16$, $SE = .04$, $p < .001$), reflecting that individuals who reported better sleep quality relative to their cohort also reported lower levels of depressed mood at the onset of the study. For the GAD group, there was a significant level 1 (within-person) effect of sleep quality at $t-1$ on depressed mood at $t$, suggesting that on days when individuals in the GAD group rated the prior evening’s sleep quality as above-average, they also experienced lower levels of depressed mood ($B = -.03$, $SE = .01$, $p = .048$). The inverse relationship was observed for healthy controls, such that below-average levels of sleep quality across a two-day lag was associated with lower levels of depression ($B = .04$, $SE = .02$, $p = .019$). For the GAD group, there was a trend for the effect of feeling rested upon awakening at $t-3$ on depressed mood at $t$ ($B = .03$, $SE = .02$, $p = .052$), indicating that the cumulative effect of feeling less rested than average over the course of three days was associated with higher levels of later depressed mood at $t$. For healthy controls, there was a level 2 (between-person) effect of feeling rested upon awakening ($B = -.17$, $SE = .07$, $p = .020$), reflecting that individuals who reported higher mean levels of feeling rested also reported higher levels of depression at the beginning of the study.

For the actigraphy sleep parameters in the GAD group, there was a significant fixed effect of time spent awake after initial sleep onset at both level 1 and level 2 (see Table 10). These results suggest that at the between-person level, individuals with GAD who reported greater mean time spent awake after initial sleep onset also reported greater depressed mood at the onset of the study ($B = -.05$, $SE = .02$, $p = .030$). Additionally,
individuals with GAD who reported above-average time spent awake after sleep onset across a three-day lag also reported higher levels of depressed mood at \(t\) \((B = .01, SE = .003, p = .033)\). There were no significant associations observed between lagged actigraphy-assessed sleep parameters and depressed mood among healthy controls.

**Does Temperament Moderate the Associations Among Anxiety, Sleep Disturbance, and Depression?**

In order to determine whether the associations between anxiety and later sleep disturbance were moderated by temperament, specifically neuroticism and negative affectivity, cross-level interaction effects were computed. Each temperament predictor was group-mean centered and was added to the model to estimate the level 2 (between-person) effect of the variable. Then equations reflecting the interactions between the level 2 temperament predictor and the level 1 (within-person) lagged anxious mood predictors were added to the multilevel model. To interpret the interaction effect, simple slope tests were conducted (Jaccard & Turrisi, 2003).

For the GAD sample, there was no level 2 effect or cross-level effects of neuroticism or negative affectivity on lagged anxious mood in the prediction of self-reported ratings of sleep quality. In the model predicting subjective ratings of feeling rested upon awakening, the interaction between neuroticism and anxious mood at \(t-1\) was significant \((B = -.06, SE = .03, p = .033;\ Figure 10)\). For the sleep parameters assessed via actigraphy, the only significant cross-level interaction was between negative affectivity and lagged anxious mood in the prediction of sleep onset latency \((B = 1.68, SE = .42, p < .001;\ Figure 11)\). However, a similar trend was observed for the interaction between...
neuroticism and lagged anxious mood at $t-1$ on sleep onset latency ($B = 1.57, SE = .87, p = .074$; Figure 12), as well as the interaction between negative affectivity and lagged anxious mood at $t-1$ on sleep efficiency ($B = -.002, SE = .001, p = .063$; Figure 13). These results suggest that the effect of anxious mood on later sleep disturbance is strongest for individuals higher in neuroticism and negative affectivity (i.e., one SD above group mean). For healthy controls, there were no cross-level interaction effects between temperament and lagged anxious mood on later sleep disturbance as assessed by either self-report or actigraphy. Temperament was also not associated with sleep disturbance as a level 2 (between-person) predictor.

To evaluate whether temperament moderated the association between sleep disturbance and later depressed mood, cross-level interactions effects were computed in the same manner as described above. For participants with GAD, there was a trend toward a cross-level effect of neuroticism and subjective sense of feeling rested upon awakening at $t-1$ ($B = -.02, SE = .01, p = .072$; Figure 14) on depressed mood at $t$. Both neuroticism ($B = -.001, SE = .001, p = .028$; Figure 15) and negative affectivity ($B = -.01, SE = .0003, p = .022$; Figure 16) moderated the effect of sleep onset latency at $t-1$ on depressed mood at $t$. Negative affectivity also moderated the effect of time spent awake after sleep onset at a two-day lag ($B = -.001, SE = .001, p = .022$; Figure 17) and the effect of total sleep time at a three-day lag ($B = .001, SE = .0003, p = .013$; Figure 18) on daily depressed mood. These cross-level interactions reflect that for the GAD group, the effect of sleep disturbance on later depressed mood is strongest for individuals higher in neuroticism and negative affectivity.
Among healthy controls, significant cross-level interactions were observed for neuroticism and self-reported sleep quality at $t-2 (B = .01, SE = .003, p = .012; \text{Figure 19})$, as well as neuroticism and subjective rating of feeling rested upon awakening at $t-1 (B = .01, SE = .003, p = .002; \text{Figure 20})$, on later depressed mood. Similarly to the associations observed in the clinical sample, lagged sleep disturbance exerted the strongest effect on later depressed mood for healthy individuals with above-average levels of neuroticism.

**Do Dysfunctional Beliefs About Sleep or Sleep-Related Safety Behaviors Moderate the Associations Among Anxiety, Sleep Disturbance, and Depression?**

In order to explore whether levels of dysfunctional beliefs about sleep or sleep-related safety behaviors, both assessed at baseline, moderated the lagged associations between anxiety and later depressed mood, cross-level interaction effects were computed and added to the multilevel model.

For the GAD group, there was a significant cross-level interaction between anxious mood at $t-4$ and dysfunctional beliefs about sleep on self-reported rating of feeling rested upon awakening at $t (B = -.14, SE = .05, p = .007; \text{Figure 21})$; anxious mood at $t-1$ and dysfunctional beliefs about sleep on subjective ratings of sleep quality at $t (B = -.20, SE = .10, p = .044; \text{Figure 22})$; and, anxious mood at $t-3$ and dysfunctional beliefs about sleep on sleep efficiency at $t (B = -.02, SE = .01, p = .048; \text{Figure 23})$. With regard to sleep-related safety behaviors, there was a significant cross-level interaction between anxious mood at $t-4$ and sleep-related safety behaviors on self-reported rating of feeling rested upon awakening at $t (B = -.01, SE = .01, p = .042; \text{Figure 24})$. A trend was
observed for the cross-level interaction between anxious mood at t-1 and sleep-related safety behaviors on amount of time spent awake after initial sleep onset at t \((B = -.38, SE = .21, p = .079; \text{Figure 25})\). These findings suggest that the effect of lagged anxious mood on subsequent sleep disturbance is strongest among GAD participants who also report higher levels of dysfunctional beliefs about sleep and engage in more sleep-related safety behaviors. For the control group, there were no cross-level interaction effects between either dysfunctional beliefs about sleep or sleep-related safety behaviors and sleep disturbance as assessed by self-report or actigraphy, and neither baseline predictor was associated with sleep disturbance as a level 2 effect.

For the association between sleep disturbance and depressed mood in the GAD group, there was a trend toward a moderation effect of dysfunctional beliefs about sleep on both total sleep time at t-4 \((B = .002, SE = .001, p = .081; \text{Figure 26})\) and subjective ratings of feeling rested upon awakening at t-4 \((B = -.02, SE = .01, p = .065; \text{Figure 27})\) in the prediction of depressed mood at t. In the control group, significant cross-level interactions were observed between dysfunctional beliefs about sleep and time spent awake after sleep onset at t-3 \((B = .01, SE = .002, p = .006; \text{Figure 28})\), as well as subjective ratings of feeling rested upon awakening at t-3 \((B = -.05, SE = .02, p = .031; \text{Figure 29})\), on depressed mood at t. Across both groups, higher levels of dysfunctional beliefs about sleep were associated with a stronger impact of lagged sleep disturbance on current depressed mood. Safety-related behaviors, on the other hand, did not moderate any of the associations between sleep disturbance and later depressed mood for either group.
Discussion

The primary aim of the present study was to explore the temporal relationships between symptoms of anxiety, depressed mood, and sleep disturbance at both the within-person and between-person level. The study improved on prior studies of temporal patterns of anxious and depressed mood in GAD in several important ways. First, the present study utilized EMA procedures, which confer the advantage of time-stamped entries over daily diary studies and collected multiple ratings throughout the day that were then averaged to control for any diurnal effects on mood. Participants completed daily ratings of sleep satisfaction and also wore actigraphs, which facilitated the comparison of temporal associations with both subjective and objective measures of sleep. The present study also assessed study aims in both a clinical sample and a control group to ascertain whether observed temporal relationships were specific to individuals with GAD or also present in healthy controls.

Sleep Disturbance

Compared to healthy controls, individuals with GAD reported greater sleep disturbance across all domains and more daytime dysfunction related to sleep difficulty at baseline. Throughout the 14-day study, GAD individuals also reported more sleep disturbance on a daily basis as reflected by lower ratings of sleep quality and reports of feeling less rested upon awakening. However, their subjective experience of sleep disturbance did not converge with actigraphy-derived sleep parameters. These findings are consistent with prior studies that have found weak relationships between objective and subjective sleep data in individuals with insomnia or mood disturbances (e.g.,
Mayers et al., 2003; Means, Edinger, Glenn, & Fins, 2003). It has also been suggested that fatigue, a daytime symptom of insomnia, is more of a subjective sleep complaint than an objective finding in patients with depressive disorders (Dauvilliers, Lopez, Ohayon, & Bayard, 2013).

Contrary to expectations, between-group differences in objective sleep disturbance (i.e., wake after sleep onset and sleep efficiency) were indicative of greater sleep disturbance in healthy controls. It is possible that these differences, while statistically significant, do not reflect clinically meaningful differences, although at least one other study found that healthy adults who denied any sleep problems demonstrated poorer sleep quality as assessed by actigraphy than those who identified as poor sleepers (McCrae et al., 2005). Unfortunately, there is a paucity of studies directly comparing objective and subjective sleep measures in individuals with anxiety disorders generally and no known studies in adults with GAD specifically, which limits the context in which these findings may be interpreted.

Nevertheless, the absence of objective sleep impairment in the GAD group suggests that these individuals experience sleep disturbance and associated daytime-interference based on variables that are not captured by more objective assessments of sleep (i.e., actigraphy). Qualitative data collected by Harvey and colleagues (2008) on the subjective meaning of sleep quality in insomnia and normal sleeper groups found that individuals with insomnia listed more required criteria for rating sleep as good quality. Although both groups reported fatigue upon awakening or throughout the day, nighttime awakenings, and sleep onset latency as the most important factors in their subjective
assessment of sleep quality, the insomnia group also listed motivation to get up in the morning and feeling alert, clear-headed, and able to concentrate throughout the day as critical aspects of sleep quality. Given that a lack of motivation and cognitive impairment (e.g., memory, processing speed, concentration) are commonly associated with depression and anxiety, it is not surprising that GAD participants in the present study rated their subjective sleep quality as poor. These findings are bolstered by other studies that have demonstrated that perceptions of sleep quality in individuals with insomnia are most closely related to daytime functioning and are not accounted for by quantitative sleep assessments or demographic factors (Ustinov et al., 2010).

It is also common for patients with insomnia to misperceive their sleep as more impaired relative to objective measures, and this discrepancy appears to be most strongly related to worry, selective attention to sleep-related threats (e.g., noise), and the presence of brief awakenings (Harvey & Tang, 2012). Other studies have found that while worry is not associated with specific self-reported sleep parameters (e.g., sleep onset latency, wakefulness after sleep onset), it does appear to predict subjective ratings of interference and distress associated with sleep disturbance in individuals with insomnia (Carney, Harris, Moss, & Edinger, 2010b; O’Kearney & Pech, 2014; Takano, Iijima, & Tanno, 2012). In a study comparing patients with GAD to healthy controls, emotion dysregulation was found to mediate the relationship between GAD and subjective sleep disturbance independently from the effect of comorbid diagnoses of depression or additional anxiety disorders (Tsypes, Aldao, & Mennin, 2013).
Although few studies have compared objective and subjective sleep indices in anxiety disorders, findings from the insomnia and depression literature provide strong evidence that the subjective experience of sleep disturbance may be distinct from objective sleep measures. Additionally, subjective sleep disturbance appears to be strongly tied to both dissatisfaction with daytime functioning (e.g., fatigue, alertness) and worry, which may represent a mutually reinforcing relationship that perpetuates distress about sleep.

**Affective Instability**

In comparison to healthy controls, individuals with GAD demonstrated significantly more variability and less temporal dependency in their day-to-day ratings of anxious and depressed mood, anhedonia, sleep-related interference, and alertness. Although some disorders are characterized by temporal instability of affect, such as borderline personality disorder, anxiety and depressive disorders are most often conceptualized as pervasive affective states. However, results from the present study suggest that symptoms fluctuate significantly on a daily basis and that an individual’s level of symptoms on a given day may not be similar to the prior day’s level of the same symptom. Intolerance of uncertainty is considered a defining feature of GAD, so it is possible that the distress associated with the disorder is tied not only to the average level of anxiety experienced, but also to the unpredictability of which days will be characterized by above-average levels of anxiety (Gentes & Ruscio, 2011).

Prior research has shown that insomnia is often characterized by night-to-night instability (Buysse et al., 2010; Suh et al., 2012), and that this instability may be more
pronounced when symptoms of insomnia are related to another mental disorder (Sanchez-Ortuno et al., 2011). Therefore, it is surprising that there were no differences between the control and GAD groups in temporal instability of either objective or subjective sleep disturbance. Given the small sample sizes, the degree of between-subject temporal instability in measures of sleep may have eclipsed any between-group differences.

Temporal Associations Between Anxiety and Sleep

Consistent with hypotheses, above-average levels of anxious mood at $t-1$ were predictive of poorer self-reported sleep quality and feeling less rested at $t$ in participants with GAD. This relationship was moderated by both neuroticism and dysfunctional beliefs about sleep, such that the effect of anxious mood on subsequent subjective sleep disturbance was strongest in individuals with above-average levels of these baseline covariates. Dysfunctional beliefs about sleep reflect concern over meeting specific sleep requirements (e.g., getting 8 hours of sleep) and worry about the consequences of insomnia the following day, which puts considerable pressure on the individual to fall asleep and often produces a paradoxical effect. Prior studies have demonstrated that anxious cognitive arousal prior to bedtime is associated with both subjective and objective assessments of delayed sleep onset (Tang & Harvey, 2004; Wicklow & Espie, 2000), so it is possible that higher-than-average levels of anxiety throughout the day interfere with sleep onset, which is especially distressing for individuals who tend to overestimate the effect of sleep quality on their functioning the following day. These results are supported by a recent cross-sectional comparison of patients with insomnia to
healthy controls matched in age and sex that found that the impact of neuroticism on insomnia was mediated by dysfunctional beliefs about sleep (Chauvin et al., 2015).

An investigation of “folk theories” about the causes of insomnia conducted by Harvey and colleagues (2013) found that individuals most often attributed their insomnia to emotions (e.g., stress, anxiety, excitement), thinking patterns (e.g., can’t shut off thoughts), and sleep-related emotions (e.g., stress or anxiety about not falling asleep). If emotionality is perceived as a threat to sleep, it is not surprising that GAD participants in the current study who were both higher in neuroticism (and therefore more likely to experience negative affect and view themselves as unable to cope with stress) and overvalued the causal relationship between sleep and daytime functioning would be most affected by experiencing above-average anxiety on a particular day. This finding is consistent with prior research that has demonstrated that while cognitive and somatic arousal at bedtime are significant predictors of transient insomnia, dysfunctional beliefs about sleep and worry are the most important factors for distinguishing chronic insomnia from transient insomnia (Yang, Lin, & Cheng, 2013).

Elevated anxious mood at $t-1$ was also predictive of objective sleep disturbance at $t$ as characterized by lower sleep efficiency, more time spent awake after sleep onset, and shorter total sleep time in the GAD group, although the association between lagged anxious mood and sleep efficiency was no longer significant when depressed mood was included in the multilevel model. There was a trend for a moderation effect of sleep-related safety behaviors on the association between anxious mood at $t-1$ and time spent awake after sleep onset at $t$, which suggests that individuals who report engaging in
safety behaviors related to sleep (e.g., going to bed early to allow plenty of time to fall asleep) more frequently may be particularly motivated to do so on days that they experience above-average levels of anxiety, which then results in greater sleep disturbance.

For healthy controls, there were no significant within-person temporal relationships between anxiety and sleep disturbance. Individuals who reported higher mean levels of anxious mood did report feeling less rested upon awakening in the morning on day 1 of the study, which is consistent with the within-person temporal association observed in GAD participants. In other words, healthy controls did not appear to experience day-to-day fluctuations in anxious mood that influence subsequent sleep disturbance, but there was a between-person effect that reflected a significant association between greater average anxiety and perceptions of sleep as less restorative.

**Temporal Associations Between Sleep and Depression**

Consistent with hypotheses, the associations between subjective sleep disturbance and later depressed mood were significant among GAD participants at both the between-person and within-person level. In other words, higher average ratings of self-reported sleep quality predicted lower levels of depression and above-average sleep quality on a particular evening predicted less depressed mood the following day. Healthy controls demonstrated the same between-person association between sleep quality and depressed mood as the GAD group (i.e., higher average ratings of sleep quality were associated with lower mean levels of depressed mood). These results are consistent with prior research demonstrating that self-reported sleep disturbance is commonly associated with
depression (e.g., Babson, Trainor, Feldner, & Blumenthal, 2010; Mayers et al., 2009; Mayers et al., 2003; Yaugher & Alexander, 2015), and that poor subjective sleep quality is predictive of low mood the following day in both depressed individuals and healthy controls (Bower et al., 2010). Surprisingly, below-average sleep quality across a two-day lag was associated with lower levels of depression in healthy controls. This finding may reflect an antidepressant effect of acute sleep deprivation, although such an effect is typically most pronounced in depressed individuals (McEwen & Karatsoreos, 2015; Wolf et al., 2016). It is also possible that in the context of the very low levels of depressed mood that were reported in the control group, this effect, albeit statistically significant, does not capture clinically meaningful temporal associations between subjective sleep disturbance and mood.

With regard to objective sleep measures, greater time spent awake after sleep onset across a three-day lag was predictive of greater depressed mood in the GAD group, suggesting that a cumulative effect (i.e., more than one night) of objective sleep disturbance may be necessary to affect levels of depressed mood the following the day in GAD individuals. Neuroticism and negative affectivity did moderate the effect of objective sleep disturbance on subsequent depressed mood, consistent with study hypotheses and prior research supporting the role of neuroticism as a predisposing factor for both insomnia (Gurtman, McNicol, & McGillivray, 2014) and depression (Naragon-Gainey, Gallagher, & Brown, 2013). There were no associations between objective measures of sleep disturbance and subsequent depressed mood in healthy controls, which may reflect greater overall stability in these constructs. The absence of an association
may also indicate a between-group difference in how objective sleep disturbance affects mood the following day in healthy controls; for example, Van mill and colleagues (2013) found that short sleep duration was only associated with impaired work performance in individuals with psychopathology.

Although sleep-related safety behaviors did not moderate any of the relationships between sleep measures and later depressed mood, dysfunctional beliefs about sleep moderated the effect of the sleep disturbance on subsequent depressed mood in both GAD individuals and healthy controls. Since there was some evidence that sleep-related safety behaviors moderate the effect of anxious mood on objective sleep quality in the GAD group, it is possible that sleep-related safety behaviors exert a more indirect effect on later depressed mood. It is also possible that dysfunctional beliefs about sleep are simply more proximally related to the association between sleep disturbance and depressed mood. In a previous study that discriminated between perceived need to use safety behaviors and actual use of safety behaviors, only ratings of safety behavior utility were associated with insomnia symptoms (Hood, Carney, & Harris, 2011). In addition, greater change in dysfunctional beliefs about sleep during treatment for insomnia has also been found to be associated with greater symptom remission for both insomnia and depression, further supporting the importance of this construct as a perpetuating or maintaining factor (Ashworth et al., 2015; Eidelman et al., 2016; Harvey et al., 2015).

The questionnaire that assesses dysfunctional beliefs about sleep includes items such as, “when I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before,” so that the same interpretation bias may be present in
the reverse direction. Research suggests that negative cognitive biases are associated with depressive symptoms (Duque & Vazquez, 2015; Korn, Sharot, Walter, Heekeren, & Dolan, 2014; Wisco & Nolen-Hoeksema, 2010) and dysfunctional beliefs about sleep may represent a specific negative cognitive bias regarding the relationship between sleep and functioning. Consistent with this hypothesis, a recent study of undergraduates found that poor sleep quality was uniquely associated with enhanced memory for negative stimuli and a deficit in sustained attention to non-emotional stimuli (Gobin, Banks, Fins, & Tartar, 2015). Taken together, these findings support prior research that has suggested that individuals with insomnia engage in ineffective cognitive control strategies at bedtime that results in difficulty down-regulating negative mood states the following day (Schmidt, Harvey, & Van der Linden, 2011).

Clinical Implications

Findings from the present study may have important clinical implications. First, it provided insight into the naturalistic patterns of fluctuations in mood in individuals with GAD and either current or past history of depressed mood. It also characterized differences in subjective and objective sleep disturbance in adults with GAD complaining of insomnia compared to good sleeper, healthy controls. Although GAD individuals’ subjective perceptions of sleep disturbance were not correlated with objective sleep parameters, time-lagged multilevel analyses revealed that anxious mood predicted both subjective and objective sleep disturbance. These findings suggest that clinical interventions that target anxiety may be more effective at reducing sleep disturbance than interventions geared toward the treatment of insomnia. Moreover, to the extent that an
intervention is able to modify temperamental risk factors, such as neuroticism and negative affect, it may be possible to significantly attenuate the negative effect of anxiety on sleep. There is also some evidence from the present study to suggest that directly targeting dysfunctional beliefs about sleep during treatment may similarly reduce depressive symptoms through attenuation of the effect of sleep disturbance on subsequent depressed mood.

Study Limitations and Directions for Future Research

Results from the present study should be interpreted in the context of several limitations. First, it was not possible to assess whether sleep disturbance mediated day-to-day variation in anxious and depressed mood. Although the present study was able to identify temporal associations among time-varying variables, it was not able to definitively infer causal relationships amongst them or provide insight into why the observed temporal relationships exist. The inclusion of both subjective and objective (i.e., actigraphy) measures of sleep disturbance improves on previous studies that relied on only method, but even actigraphy is not fully objective; the scoring algorithms utilize the participant’s reported bedtime and waketime to calculate sleep parameters. The study samples, while well matched in sex, were predominantly female, which limits some of the generalizability of study findings. For the GAD sample, comorbid diagnoses of additional anxiety disorders were allowed provided that GAD was primary, which strengthens external validity, but also limits the ability to draw conclusions about whether the observed associations are specific to GAD or anxiety disorders more generally. Lastly,
although the use of brief, face-valid items facilitates compliance with EMA procedures, it may be desirable to utilize longer measures of mood that are validated.

Future research should explore the significance of affective instability in individuals with GAD and other anxiety disorders, and assess whether instability uniquely contributes to distress above and beyond mean levels of dysregulation. Even though multilevel modeling substantially magnifies the power of analyses, it will be important to replicate these findings in larger samples with more equitable sex ratios. Visual inspection of variability figures that suggest that some individuals experience more affective instability than others, and it may be informative to utilize sub-classes within clinical and control samples to determine whether variability is an important within-person predictor of symptomatology. Although the present study did assess moderation effects using baseline covariates, future studies should incorporate the assessment of concurrent moderators to facilitate more nuanced understanding of temporal associations. Finally, it will be important to assess the temporal associations in the context of treatment, which will also provide greater insight into the casual mechanisms driving these observed relationships.

**Conclusion**

Sleep is a complex physiological process that intersects with affective states, such as anxious or depressed mood, in dynamic and multifaceted ways. For example, sleep deprivation can precipitate, exacerbate, or attenuate negative mood states (Wiebe, Cassoff, & Gruber, 2012). Results from the present study provide new insight into the associations between daily symptoms of sleep disturbance, anxiety, and depressed mood.
These findings emphasize the importance of concurrent assessment of both subjective and objective sleep disturbance, as objective sleep impairment appears to be only peripherally related to self-reported insomnia symptoms in individuals with GAD. In the context of recent research demonstrating that the presence of comorbid anxiety and depressive disorders does not negatively impact treatment outcomes for insomnia (e.g., Belanger et al., 2016; Hamoen, Redlich, & de Weerd, 2014), the present study provides a strong rationale for explicitly targeting sleep disturbance during treatment of emotional disorders.
Table 1. Demographic characteristics for each group.

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</table>

Note. GAD = Generalized Anxiety Disorder; CSR = Clinical Severity Rating.

Table 2. Clinical characteristics for each group.
<table>
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<th>GAD $(n = 15)$</th>
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<th>p-value</th>
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<td>M</td>
<td>SD</td>
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<td>Q-LES-Q (%)a</td>
<td>84.40</td>
<td>10.68</td>
<td>47.86</td>
<td>16.29</td>
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</table>

Note. GAD = Generalized Anxiety Disorder; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; ASI = Anxiety Sensitivity Index; STAI-T = State-Trait Anxiety Inventory, Trait Version; PSWQ = Penn State Worry Questionnaire; PANAS-NA = Positive and Negative Affect Schedule - Negative Affectivity; Positive and Negative Affect Schedule – Positive Affectivity; NFFI-N = NEO Five-Factor Inventory, Neuroticism Scale; WSI-E = Weekly Stress Inventory - Event Scale; WSI-I = Weekly Stress Inventory - Impact Scale; WSAS = Work and Social Adjustment Scale; Q-LES-Q = Quality of Life Enjoyment and Satisfaction Questionnaire.

aFor these measures, higher scores are reflective of less impairment.
Table 3. Sleep measures for each group.

<table>
<thead>
<tr>
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<th>$p$-value</th>
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<td>$M$</td>
<td>$SD$</td>
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<td>7.57</td>
<td>41.40</td>
<td>10.25</td>
</tr>
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<td>SRBQ</td>
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<td>16.70</td>
<td>52.80</td>
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<td>SHHC</td>
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<tr>
<td>PSQI</td>
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<td>Sleep Quality</td>
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<tr>
<td>Sleep Latency</td>
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<td>Sleep Disturbances</td>
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<td>0.33</td>
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<tr>
<td>Daytime Dysfunction</td>
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</tr>
<tr>
<td>Global Score</td>
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<td>10.67</td>
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<table>
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<th></th>
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<td>0.48</td>
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<td>Intermediate Type</td>
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<td>Evening Type</td>
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<td>0.00</td>
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</table>

Note. GAD = Generalized Anxiety Disorder; ISI = Insomnia Severity Index; PSQI = Pittsburgh Sleep Quality Index; FSS = Fatigue Severity Scale; SRBQ = Sleep-Related Behaviours Questionnaire; DBAS = Dysfunctional Beliefs About Sleep Scale, Brief Version; SHHC = Sleep Habits and Hygiene Checklist; MECS = Morningness-Eveningness Composite Scale.
Table 4. Daily symptom ratings for each group.

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<th>p-value</th>
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<td></td>
<td>(n = 15)</td>
<td>(n = 15)</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>DISS Alert/Cognition (0-100)</td>
<td>77.45</td>
<td>12.65</td>
<td>51.96</td>
<td>13.12</td>
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<tr>
<td>DISS Sleepiness/Fatigue (0-100)</td>
<td>21.69</td>
<td>14.12</td>
<td>53.71</td>
<td>15.36</td>
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<tr>
<td>DISS Positive Mood (0-100)*</td>
<td>72.05</td>
<td>13.81</td>
<td>45.48</td>
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<td>DISS Negative Mood (0-100)</td>
<td>9.81</td>
<td>9.61</td>
<td>38.37</td>
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<tr>
<td>Anxious Mood (1-10)</td>
<td>1.51</td>
<td>0.74</td>
<td>4.82</td>
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<td>Depressed Mood (1-10)</td>
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<td>Anhedonia (1-10)</td>
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<tr>
<td>Sleep-Related Interference (1-10)</td>
<td>1.75</td>
<td>1.27</td>
<td>4.88</td>
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</table>

Note. GAD = Generalized Anxiety Disorder; DISS = Daytime Insomnia Symptom Scale. Descriptive statistics were computed by first taking within-person means across all time points, and then computing descriptive statistics across participants. *For these measures, higher scores are reflective of less impairment.
Table 5. Subjective and objective sleep data for each group.

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<th>p-value</th>
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</thead>
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<tr>
<td></td>
<td>(n = 15)</td>
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**Subjective Ratings**

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<td>Sleep Quality a</td>
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<td>2.95</td>
<td>0.80</td>
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<td>Rested Upon Awakening a</td>
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**Actigraphy Parameters**

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<th>p-value</th>
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<tbody>
<tr>
<td>Sleep Onset Latency (mins)</td>
<td>23.07</td>
<td>33.35</td>
<td>18.35</td>
<td>27.66</td>
<td>1.64</td>
<td>.103</td>
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<tr>
<td>Total Sleep Time (mins)</td>
<td>390.76</td>
<td>94.83</td>
<td>389.18</td>
<td>89.77</td>
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<td>Sleep Efficiency (%)</td>
<td>83.17</td>
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<tr>
<td>Wake After Sleep Onset (mins)</td>
<td>37.96</td>
<td>28.08</td>
<td>29.05</td>
<td>16.67</td>
<td>3.93</td>
<td>&lt;.001</td>
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</table>

*Note.* GAD = Generalized Anxiety Disorder. Descriptive statistics were computed by first taking within-person means across all time points, and then computing descriptive statistics across participants.

*For these measures, higher scores are reflective of less impairment.*
Table 6. Correlation coefficients of subjective and objective sleep measures for each group.

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<td></td>
<td>Sleep Quality(^a)</td>
<td>Rested(^a)</td>
</tr>
<tr>
<td>Sleep Onset Latency</td>
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<td>-.03</td>
</tr>
<tr>
<td>Wake After Sleep Onset</td>
<td>-.07</td>
<td>-.06</td>
</tr>
<tr>
<td>Sleep Efficiency</td>
<td>.23(^**)</td>
<td>.12</td>
</tr>
<tr>
<td>Total Sleep Time</td>
<td>.28(^***)</td>
<td>.32(^**)</td>
</tr>
</tbody>
</table>

Note. GAD = Generalized Anxiety Disorder. \(^*\)p < .05; \(^**\)p < .01; \(^***\)p < .001 (2-tailed). \(^a\)For these measures, higher scores are reflective of less impairment.
Table 7. Results of multilevel modeling analysis simultaneously predicting subjective sleep disturbance from anxious mood at four time lags for each group.

<table>
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<td></td>
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<tr>
<td></td>
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<td>(n = 15)</td>
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<tr>
<td><strong>Between-Person</strong></td>
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<td></td>
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<tr>
<td>Average Anxious Mood</td>
<td>-0.32 0.29 62.01</td>
<td>0.01 0.10 57.92</td>
</tr>
<tr>
<td>Day</td>
<td>-0.01 0.02 57.13</td>
<td>-0.02 0.02 63.39</td>
</tr>
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<td>-0.43 0.11 110.37</td>
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<td>Anxious Mood t-2</td>
<td>-0.16 0.18 92.57</td>
<td>0.38 0.11 93.58</td>
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<td>Anxious Mood t-3</td>
<td>0.06 0.16 109.23</td>
<td>-0.13 0.10 101.85</td>
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<td><strong>Within-Person</strong></td>
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</tr>
<tr>
<td>Day</td>
<td>0.01 0.02 63.99</td>
<td>0.02 0.02 56.98</td>
</tr>
<tr>
<td>Anxious Mood t-1</td>
<td>-0.10 0.16 110.28</td>
<td>-0.37 0.11 105.86</td>
</tr>
<tr>
<td>Anxious Mood t-2</td>
<td>0.00 0.15 110.33</td>
<td>0.24 0.11 94.55</td>
</tr>
<tr>
<td>Anxious Mood t-3</td>
<td>0.02 0.14 108.37</td>
<td>-0.16 0.10 98.53</td>
</tr>
<tr>
<td>Anxious Mood t-4</td>
<td>0.02 0.09 149.69</td>
<td>0.07 0.06 146.26</td>
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</table>

Dependent variable = Refreshed

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<th>t</th>
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<td></td>
</tr>
<tr>
<td>Average Anxious Mood</td>
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<td>-0.11 0.10 52.32</td>
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<td></td>
</tr>
<tr>
<td><strong>Within-Person</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day</td>
<td>0.01 0.02 63.99</td>
<td>0.02 0.02 56.98</td>
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<td></td>
</tr>
<tr>
<td>Anxious Mood t-1</td>
<td>-0.10 0.16 110.28</td>
<td>-0.37 0.11 105.86</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxious Mood t-2</td>
<td>0.00 0.15 110.33</td>
<td>0.24 0.11 94.55</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxious Mood t-3</td>
<td>0.02 0.14 108.37</td>
<td>-0.16 0.10 98.53</td>
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<td></td>
</tr>
<tr>
<td>Anxious Mood t-4</td>
<td>0.02 0.09 149.69</td>
<td>0.07 0.06 146.26</td>
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<td></td>
</tr>
</tbody>
</table>
Note. GAD = Generalized Anxiety Disorder; B = unstandardized coefficient; SE = standard error; df = degrees of freedom; t = t statistic; p = p-value.

Table 8. Results of multilevel modeling analysis simultaneously predicting objective sleep disturbance from anxious mood at four time lags for each group.

<table>
<thead>
<tr>
<th>Dependent variable =</th>
<th>Control (n = 15)</th>
<th>GAD (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sleep Efficiency</strong></td>
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<td></td>
</tr>
<tr>
<td><strong>Between-Person</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average Anxious Mood</td>
<td>-0.02 0.05 52.48 -0.33 .746</td>
<td>0.01 0.01 76.36 0.89 .378</td>
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<tr>
<td><strong>Within-Person</strong></td>
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<td></td>
</tr>
<tr>
<td>Day</td>
<td>0.00 0.00 55.89 1.33 .188</td>
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*Note.* GAD = Generalized Anxiety Disorder; B = unstandardized coefficient; SE = standard error; df = degrees of freedom; t = t statistic; p = p-value.
Table 9. Results of multilevel modeling analysis simultaneously predicting depressed mood from subjective sleep disturbance at four time lags for each group.

<table>
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<tr>
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<th>Control ($n = 15$)</th>
<th>GAD ($n = 15$)</th>
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Note. GAD = Generalized Anxiety Disorder; B = unstandardized coefficient; SE = standard error; df = degrees of freedom; t = t statistic; p = p-value.

Table 10. Results of multilevel modeling analysis simultaneously predicting depressed mood from objective sleep disturbance at four time lags for each group.

<table>
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<th>t</th>
<th>p</th>
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Dependent variable = Depressed Mood

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Note. GAD = Generalized Anxiety Disorder; B = unstandardized coefficient; SE = standard error; df = degrees of freedom; t = t statistic; p = p-value; WASO = Wake After Sleep Onset.
Figure 1. Participant flow chart for healthy controls.

- **Contacted**  
  $n = 19$

- **Completed Phone Screen**  
  $n = 19$
  - Currently in treatment ($n = 1$)
  - Elevated sleep disturbance ($n = 1$)

- **Enrolled**  
  $n = 17$
  - Actigraph malfunctioned ($n = 1$)
  - Unable to use SymTrend ($n = 1$)

- **Completed Study**  
  $n = 15$
Figure 2. Participant flow chart for recruitment of clinical sample.

- **Contacted**
  - $n = 41$
  - Unresponsive ($n = 2$)
  - Uninterested in research ($n = 5$)
  - Unwilling to delay treatment ($n = 4$)

- **Completed Phone Screen**
  - $n = 30$
  - Initiated treatment ($n = 2$)
  - No history of low mood ($n = 1$)
  - Change in diagnosis ($n = 1$)
  - Currently in treatment ($n = 1$)
  - Declined participation ($n = 1$)
  - Insufficient sleep disturbance ($n = 8$)

- **Enrolled**
  - $n = 16$

- **Completed Study**
  - $n = 15$
  - Did not wear actigraph ($n = 1$)
**Figure 3.** Variability across 14 days for daily average of depressed mood for the control group (a) and the GAD group (b).

**A. CONTROL**

**B. GAD**

*Note.* GAD = Generalized Anxiety Disorder. Each individual’s data across 14 days are represented by a single line.
Figure 4. Variability across 14 days for daily average of anxious mood for the control group (a) and the GAD group (b).

Note. GAD = Generalized Anxiety Disorder. Each individual’s data across 14 days are represented by a single line.
Figure 5. Variability across 14 days for end-of-day rating of anhedonia for the control group (a) and the GAD group (b).

Note. GAD = Generalized Anxiety Disorder. Each individual’s data across 14 days are represented by a single line.
Figure 6. Variability across 14 days for end-of-day rating of interference in functioning attributed to sleep for the control group (a) and the GAD group (b).

Note. GAD = Generalized Anxiety Disorder. Each individual’s data across 14 days are represented by a single line.
Figure 7. Variability across 14 days for daily average of alert/cognition scale of DISS for the control group (a) and the GAD group (b).

Note. GAD = Generalized Anxiety Disorder; DISS = Daytime Insomnia Symptom Scale. Each individual’s data across 14 days are represented by a single line.
**Figure 8.** Variability across 14 days for daily average of fatigue/sleepiness scale of DISS for the control group (a) and the GAD group (b).

Note. GAD = Generalized Anxiety Disorder; DISS = Daytime Insomnia Symptom Scale. Each individual’s data across 14 days are represented by a single line.
**Figure 9.** Variability across 14 days for daily average of negative mood scale of DISS for the control group (a) and the GAD group (b).

**Note:** GAD = Generalized Anxiety Disorder; DISS = Daytime Insomnia Symptom Scale. Each individual’s data across 14 days are represented by a single line.
Figure 10. Cross-level interaction between anxious mood at $t-1$ and neuroticism on subjective rating of feeling rested upon awakening at $t$ in the GAD group.

Note. GAD = Generalized Anxiety Disorder; NFFI = NEO Five-Factor Inventory, Neuroticism Scale.

$^a$For these measures, higher scores are reflective of less impairment.
Figure 11. Cross-level interaction between anxious mood at $t-1$ and negative affectivity on sleep onset latency at $t$ in the GAD group.

Note. GAD = Generalized Anxiety Disorder; NA = Positive and Negative Affect Schedule – Negative Affectivity.
Figure 12. Cross-level interaction between anxious mood at t-1 and neuroticism on sleep onset latency at t in the GAD group.

Note. GAD = Generalized Anxiety Disorder; NFFI = NEO Five-Factor Inventory, Neuroticism Scale.
Figure 13. Cross-level interaction between anxious mood at $t-1$ and neuroticism on sleep efficiency in the GAD group.

Note. GAD = Generalized Anxiety Disorder; NFFI = NEO Five-Factor Inventory, Neuroticism Scale.
Figure 14. Cross-level interaction between subjective rating of feeling rested upon awakening at $t-1$ and neuroticism on depressed mood at $t$ in the GAD group.

Note. GAD = Generalized Anxiety Disorder; NFFI = NEO Five-Factor Inventory, Neuroticism Scale.
Figure 15. Cross-level interaction between anxious mood at $t-1$ and neuroticism on depressed mood at $t$ in the GAD group.

Note. GAD = Generalized Anxiety Disorder; NFFI = NEO Five-Factor Inventory, Neuroticism Scale.
**Figure 16.** Cross-level interaction between sleep onset latency at $t-1$ and negative affectivity on depressed mood at $t$ in the GAD group.

*Note.* GAD = Generalized Anxiety Disorder; NA = Positive and Negative Affect Schedule – Negative Affectivity.
**Figure 17.** Cross-level interaction between wake after sleep onset at \(t-2\) and negative affectivity on depressed mood at \(t\) in the GAD group.

*Note.* GAD = Generalized Anxiety Disorder; NA = Positive and Negative Affect Schedule – Negative Affectivity.
**Figure 18.** Cross-level interaction between total sleep time at \( t-3 \) and negative affectivity on depressed mood at \( t \) in the GAD group.

*Note.* GAD = Generalized Anxiety Disorder; NA = Positive and Negative Affect Schedule – Negative Affectivity.
Figure 19. Cross-level interaction between subjective sleep quality at $t-2$ and neuroticism on depressed mood at $t$ in the control group.

Note. NFFI = NEO Five-Factor Inventory, Neuroticism Scale.
Figure 20. Cross-level interaction between subjective rating of feeling rested upon awakening at $t-1$ and neuroticism on depressed mood at $t$ in the control group.

CONTROL ($n = 15$)

Note. NFFI = NEO Five-Factor Inventory, Neuroticism Scale.
**Figure 21.** Cross-level interaction between anxious mood at \( t-4 \) and dysfunctional beliefs about sleep on subjective rating of feeling rested upon awakening at \( t \) in the GAD group.

\[ \text{GAD (n = 15)} \]

\[ \begin{array}{c|c}
\text{Low DBAS} & \text{High DBAS} \\
\hline
\text{Anxious Mood } t-4 & \\
\end{array} \]

*Note.* GAD = Generalized Anxiety Disorder; DBAS = Dysfunctional Beliefs About Sleep Scale.

*For these measures, higher scores are reflective of less impairment.*
Figure 22. Cross-level interaction between anxious mood at \( t-1 \) and dysfunctional beliefs about sleep on subjective sleep quality at \( t \) in the GAD group.

Note. GAD = Generalized Anxiety Disorder; DBAS = Dysfunctional Beliefs About Sleep Scale.

\(^a\)For these measures, higher scores are reflective of less impairment.
**Figure 23.** Cross-level interaction between anxious mood at $t-3$ and dysfunctional beliefs about sleep on sleep efficiency at $t$ in the GAD group.

Note. GAD = Generalized Anxiety Disorder; DBAS = Dysfunctional Beliefs About Sleep Scale.
Figure 24. Cross-level interaction between anxious mood at $t-4$ and sleep-related behaviors on subjective rating of feeling rested upon awakening at $t$ in the GAD group.

Note. GAD = Generalized Anxiety Disorder; SRBQ = Sleep-Related Behaviors Questionnaire.
**Figure 25.** Cross-level interaction between anxious mood $t-1$ and sleep-related behaviors on time spent awake after sleep onset at $t$ in the GAD group.

*Note.* GAD = Generalized Anxiety Disorder; SRBQ = Sleep-Related Behaviors Questionnaire.
Figure 26. Cross-level interaction between total sleep time at $t-4$ and dysfunctional beliefs about sleep on depressed mood at $t$ in the GAD group.

Note. GAD = Generalized Anxiety Disorder; DBAS = Dysfunctional Beliefs About Sleep Scale.
Figure 27. Cross-level interaction between subjective rating of feeling rested upon awakening at t-4 and dysfunctional beliefs about sleep on depressed mood at t in the GAD group.

Note. GAD = Generalized Anxiety Disorder; DBAS = Dysfunctional Beliefs About Sleep Scale.
Figure 28. Cross-level interaction between time spent awake after sleep onset at $t-3$ and dysfunctional beliefs about sleep on depressed mood at $t$ in the control group.

CONTROL (n = 15)

Note. DBAS = Dysfunctional Beliefs About Sleep Scale.
Figure 29. Cross-level interaction between subjective rating of feeling rested upon awakening at \( t-3 \) and dysfunctional beliefs about sleep on depressed mood at \( t \) in the control group.

Note. DBAS = Dysfunctional Beliefs About Sleep Scale.
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Vita

Jacqueline Bullis earned her B.A. in psychology from Boston University and her M.A. in psychology from Boston University. She is currently a doctoral candidate in clinical psychology at Boston University and is expected to receive her doctoral degree in 2016. During her matriculation at Boston University, Ms. Bullis has received training at the Center for Anxiety and Related Disorders and the Eating Disorders Program at Boston University, as well as at the Freedom Trail Clinic and Massachusetts General Hospital. Ms. Bullis has been extensively involved in research during her time at Boston University. She has received research funding from the Barlow Foundation. Ms. Bullis has authored multiple peer-reviewed research papers and presented at various national conferences. Her research interests largely revolve around treatment, including its development, outcome, augmentation, dissemination, and implementation. She is most passionate about research aimed at increasing the public health impact of existing psychological treatments by applying a transdiagnostic, interdisciplinary approach to the diagnosis, treatment, and prevention of emotional disorders.