Examining the Relationship between Worry and Sleep: A Daily Process Approach

BY

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THESIS

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LIST OF ABBREVIATIONS

B	Unstandardized beta
β	Standardized beta
CBT	Cognitive Behavioral Therapy
CBT-I	Cognitive Behavioral Therapy for Insomnia
CI	Confidence Interval
D	Deviance
DSM-IV	Diagnostic and Statistical Manual 4 th Edition
FW	Focused Worry
GAD	Generalized Anxiety Disorder
ISI	Insomnia Severity Index
M	Mean
n	Number in Subgroup
N	Number in Total Group
NA	Negative Affect
ns	Nonsignificant
p	Probability
PA	Positive Affect

PANAS	Positive and Negative Affect Scale
PDA	Personal Data Assistant
PSWQ	Penn State Worry Questionnaire
SC	Stimulus Control
<i>SD</i>	Standard Deviation
χ^2	Chi-Square

SUMMARY

There is growing evidence suggesting that worry and sleep are intimately linked. However, the way that these two phenomena influence each other over the course of a day is largely unknown. Specifically, it is possible that (1) worry contributes to sleep disturbance, (2) sleep disturbance contributes to worry, or (3) there is a bidirectional relationship between worry and sleep. The present study examined the daily relationship between worry and sleep in 53 high trait worriers who were randomly assigned to one of two interventions aimed at reducing worry. A daily process approach was utilized wherein participants completed daily reports of sleep and worry during a 7-day baseline period, as well as daily reports of sleep, worry, and emotion during a 14-day intervention period. Results of repeated measures multilevel modeling analyses indicated that worry experienced on a particular day predicted increased sleep disturbance that night during both the baseline and intervention weeks. However, there was no evidence of a bidirectional relationship as sleep characteristics did not predict worry the following day. Additionally, neither intervention nor daily emotional functioning affected the daily relationship between worry and sleep. Results of the present study are consistent with the cognitive model of insomnia (Harvey, 2002) and suggest that worry experienced throughout the day and prior to sleep onset has negative effects on sleep. Contrary to our hypotheses, there was no evidence suggesting that sleep disturbance affects worry symptoms. Results highlight the importance of addressing and treating worry among individuals with high trait worry and sleep disturbance.

I. INTRODUCTION

Worry and sleep share a close relationship. For example, worry is a common occurrence in those suffering from insomnia (Morin, 1993), and insomnia likely plays a role in exacerbating symptoms of psychopathology (Ford & Kamerow, 1989), including chronic and severe worry. Several forms of psychopathology that involve chronic worry are also associated with sleep difficulties. One reason for this relationship is that sleep disturbance is a symptom or clinical feature of several Axis I disorders in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV; American Psychiatric Association [APA], 1994) and is particularly associated with anxiety disorders. For example, Ohayon, Caulet, and Lemoine (1998) found that 41.6% of individuals with insomnia were diagnosed with an anxiety disorder, and difficulty with sleep onset is a feature of many anxiety disorders (Morin, 1993). Furthermore, generalized anxiety disorder (GAD), which is characterized by chronic worry, is the most prevalent anxiety disorder diagnosis among individuals with sleep disturbance (Ohayon et al., 1998). Among those with severe sleep disturbance, 13% report symptoms of GAD (Mellinger, Balter, & Uhlenhuth, 1985). Additionally, it is estimated that 50-70% of individuals with GAD have insomnia (Anderson, Noyes, & Crowe, 1984). Given that the diagnostic criteria for GAD includes sleep disturbance, it is not surprising that individuals with GAD often complain of difficulty engaging in sleep due to excessive and uncontrollable worry (Monti & Monti, 2000).

Despite strong evidence indicating a general association between worry and sleep disturbance, little research has examined how they affect each other on a daily basis. There are three possible ways in which worry and sleep disturbance are related: (1) it is possible that worry contributes to sleep disturbance; (2) it is possible that sleep disturbance contributes to worry; or (3) there may be a bidirectional relationship between worry and sleep disturbance. An important

step in understanding the relationship between these two phenomena is to examine how they interact over the course of a day. Additionally, understanding the relationship between worry and sleep disturbance has the potential to elucidate the processes involved in chronic worry and insomnia, and may aid in the development and implementation of interventions.

A. Worry Leads to Sleep Disturbance

According to the cognitive model of insomnia (Harvey, 2002), worry contributes to poor sleep quality, thus maintaining and exacerbating insomnia. Generally, individuals with insomnia engage in worrisome thinking about their sleeping patterns that serves to exacerbate insomnia symptoms and create a self-fulfilling prophecy. The model suggests that individuals with insomnia engage in worry both prior to sleep onset and throughout the day such that they become vigilant to cues that support their sleep-related concerns. Specifically, at bedtime individuals with insomnia focus on internal (e.g., feeling restless) and external (e.g., watching the clock) threat cues that signify difficulty with sleep and their perceived poor functioning associated with insufficient sleep (e.g., “If I don’t get enough sleep, I will not be able to function”). During the day, poor performance at work or symptoms of fatigue are attributed to insufficient sleep (instead of a host of other possible contributing factors). In turn, individuals with insomnia develop core beliefs about themselves as poor sleepers and overestimate the negative effects of a poor night’s sleep. They engage in counterproductive safety behaviors (e.g., drinking alcohol before bedtime) that prevent them from modifying their maladaptive beliefs. Additionally, the worrisome thoughts that occur prior to bedtime in combination with hypervigilance toward threatening cues create conditions that actually inhibit successful sleep onset, making it difficult for these individuals to fall asleep. Thus, as individuals become trapped in this cyclical cognitive

process, the escalation of anxiety and emotional distress can lead to an actual deficit in sleep, which subsequently reinforces and strengthens their concerns.

The cognitive model of insomnia is strongly supported by empirical research. Individuals with insomnia often report intrusive thoughts and excessive and uncontrollable worry while trying to fall asleep (Borkovec, 1979, 1982; Morin, 1993). Although there are several other biological and psychological factors that contribute to insomnia (e.g., sleep apnea, psychosis; Bootzin, Mauber, Perlis, Salvio, & Wyatt, 1993), a survey of individuals with insomnia revealed that cognitive intrusions (e.g., worry) were more to blame for sleep disturbance than were somatic complaints (e.g., feeling sweaty; Lichstein & Rosenthal, 1980). Additionally, individuals with insomnia engage in greater catastrophizing worries that result in increases in anxiety and distress (Harvey & Greenall, 2003), which are not conditions conducive to successful sleep onset (Epsie, 2002). It is therefore not surprising that insomniacs exhibit greater autonomic arousal during the night as compared with good sleepers (Freedman & Sattler, 1982; Monroe, 1967). Additionally, during the day these individuals have distorted beliefs about the previous night's sleep and the perceived consequences of a poor night's sleep (Bonnet, 1990; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). These beliefs lead them to engage in counterproductive safety behaviors (Woodley & Smith, 2006), which serve to strengthen those beliefs. Thus, the cognitive model of insomnia suggests that a relationship exists between worry, examination of threat cues, beliefs about sleep, and counterproductive safety behaviors that ultimately result in the maintenance and strengthening of insomnia.

Although the content of worry discussed in the cognitive model of insomnia is specifically related to sleep concerns, it is also possible that increased general worry (not necessarily specific to sleep) leads to the development of insomnia. For example, Gross and

Borkovec (1982) manipulated participants' likelihood of engaging in cognitive mentation prior to sleep onset by informing one group of good sleepers that they would have to give a speech immediately following a nap. Participants who were informed about the speech took significantly longer to fall asleep than did participants who were not asked to give a speech upon awakening. This finding suggests that participants had difficulty falling asleep as a result of the cognitive intrusions (i.e. worry) related to giving a speech. Additionally, Hall, Buysse, Reynolds, Kupfer, and Baum (1996) manipulated pre-sleep stress in female good sleepers and found a positive relationship between subjective stress-related intrusive thoughts and objective sleep onset latency.

Although there is ample evidence suggesting that worry plays a role in sleep disturbance, it is not necessarily the case that a causal relationship exists wherein worry leads to sleep difficulties. Alternatively, it may be that sleep disturbance leads to the experience of worry.

B. Sleep Disturbance Leads to Worry

It is possible that sleep disturbance leads to the emergence and exacerbation of worry. Individuals with insomnia report higher trait worry symptoms during the day as compared with individuals without insomnia (Means, Lichstein, Epperson, & Johnson, 2000). One study demonstrated that individuals who sleep less tend to worry more even after controlling for sleep disturbance attributed to worry, suggesting that reduced sleep length continues to be associated with worry beyond the negative effects of pre-sleep cognitive activity (Kelly, 2002). Research has shown that individuals who sleep for less than six hours per night report more anxiety than do individuals who sleep nine or more hours per night (Kumar & Vaidya, 1984). This relationship was empirically tested by inducing objective (i.e., increased sleep onset latency, decreased total sleep time) and subjective (i.e., perceived sleep quality, number of awakenings)

sleep disturbance in good sleepers over the course of a week (Bonnet & Arand, 1992). Results indicated that participants reported significant increases in anxiety, dysphoria, and tension, demonstrating that insomnia leads to psychological distress.

Although insomnia is often conceptualized as an epiphenomenon of various physical and mental conditions (Spielman & Glovinsky, 1997), a growing body of literature suggests that insomnia may predate the onset of these disorders and contribute to their development (Ford & Kamerow, 1989; Vollrath, Wicki, and Angst, 1989; see Harvey, 2001 for a review). The National Institute of Mental Health Epidemiologic Catchment Area study surveyed 7,954 community residents about their sleep and psychopathology at two time points separated by one year (Ford & Kamerow, 1989). Results indicated that individuals who had insomnia at Time 1 and not at Time 2 were at 1.6 times the risk of developing any psychiatric disorder (and 1.5 times the risk of developing an anxiety disorder) over that year. Individuals who had insomnia at both time points were at 4.0 times the risk of developing any psychiatric disorder (and 6.3 times the risk of developing an anxiety disorder) over that year. Additionally, a longitudinal study by Vollrath et al. (1989) indicated that compared with individuals who did not endorse sleep disturbance (22%), 42%-48% of individuals who experienced occasional or brief periods of insomnia suffered from anxiety or depressive disorders during the subsequent year.

Furthermore, in addition to other psychological constructs, worry may be a mediating factor in the relationship between sleep disturbance and psychopathology. Specifically, it is possible that sleep disturbance exacerbates psychological domains (e.g., worry, neuroticism, negative affect) that increase risk for psychopathology. Indeed, individuals with insomnia demonstrate more anxious worrying behavior and neuroticism (Coursey, Buchsbaum, & Frankel, 1975). Chronic insomnia leads to psychological symptoms such as irritability, tension, and

dysphoria in the absence of a diagnosed psychiatric disorder (Ford & Kamerow, 1989). Finally, longer sleep onset has been associated with negative affect (Berry & Webb, 1983, 1985). Thus, it is likely that insomnia contributes to worry as well as symptoms of negative affect and tension, which create vulnerability for psychopathology. Another possibility is that negative affect, tension, and/or anxiety mediate the relationship between sleep disturbance and worry.

C. Worry and Sleep Disturbance are Bidirectionally Related

Given that there is research to support the notion that worry contributes to sleep disturbance *and* that sleep disturbance contributes to worry, it is also possible that a bidirectional relationship exists wherein worry and sleep disturbance mutually affect and exacerbate one another. Specifically, increases in worry may lead to increases in sleep difficulties, which in turn might exacerbate worry such that a cyclical relationship develops. This point is supported by evidence that the relationship between worry and insomnia likely becomes stronger over time (Jansson & Linton, 2006a). Indeed, some researchers have suggested a bidirectional relationship between insomnia, anxiety, and worry (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010; Jansson & Lindblom, 2008; Jansson & Linton, 2006a,b). However, support for this assertion is limited by the fact that many of the sources that suggest such a bidirectional relationship are theoretical or cross-sectional and thus cannot adequately test theories of bidirectionality. Additionally, those studies employing a longitudinal design suggest that a bidirectional relationship exists based on findings that insomnia is associated with new cases of anxiety disorders, and anxiety disorders are associated with new cases on insomnia (Jansson & Lindblom, 2008; Jansson & Linton, 2006a,b). No studies to our knowledge have empirically examined a bidirectional relationship between sleep disturbance and worry on a daily basis, and few studies have utilized an analytic approach that allows for the examination of a temporal relationship. If a

bidirectional relationship between worry and insomnia exists, this would suggest that the experience of either phenomenon might begin a negative spiral wherein each state leads to the experience of the other state. Additionally, this would suggest that intervening at any point in the cycle could lead to improvements in both phenomena.

D. Examining the Relationship between Worry and Sleep Disturbance

One way to understand the relationship between worry and sleep disturbance is to examine whether treating one phenomenon leads to improvements in the other. For example, if a treatment designed to reduce worry also ameliorates sleep disturbance, it would provide further support for their relationship and would suggest that worry plays a role in the maintenance of insomnia. Recent research has demonstrated that interventions targeted at reducing worry have ameliorating effects on sleep disturbance. For example, Bélanger, Morin, Langlois, and Ladouceur (2004) examined the effects of cognitive behavioral therapy (CBT) that targeted excessive worries, with no specific intervention targeted at sleep disturbance, among individuals diagnosed with GAD. Interestingly, results indicated that insomnia symptoms were significantly reduced at the end of treatment. Furthermore, GAD severity and insomnia severity were weakly correlated, suggesting that the beneficial effects of CBT on insomnia symptoms were not merely due to the reduction of overall symptom severity or a common underlying construct such as negative affect. Additionally, a treatment that is specifically targeted at reducing sleep disturbance – cognitive behavioral therapy for insomnia (CBT-I) – has been shown to have ameliorating effects on comorbid mood and anxiety disorders (Blais, Mimeault, & Morin, 2000; Vallieres, Bastien, Ouellet, & Morin, 2000). For example, Blais et al. (2000) found that providing CBT-I to individuals with both insomnia and GAD produced significant reductions in GAD symptoms as well as insomnia symptoms. Together, these studies provide further support

for the strong relationship between worry and insomnia, and also suggest that treating one phenomenon results in reductions in the other.

Unfortunately, these studies did not examine the direction of the relationship between worry and insomnia and thus can only indicate that the two phenomena are related in some way. However, examining the relationship between worry and sleep disturbance throughout the course of an intervention targeted at one of the phenomena would provide an ideal context in which to investigate a directional relationship. In addition to providing information regarding the nature of the relationship between the two phenomena, such an approach would also allow for the examination of the mechanism of change within the intervention. Specifically, utilizing an intervention that solely targets worry but that also leads to reductions in sleep disturbance could elucidate (1) the nature of the relationship between worry and sleep, and (2) whether or how the relationship between worry and sleep changes throughout the course of the intervention. It is possible that interventions that reduce both worry and sleep disturbance produce changes by affecting the way that worry and sleep influence each other, or it may be that those interventions target some other constructs (e.g., negative affect, anxiety, tension), which in turn produce reduction in worry and sleep disturbance. Additionally, such an approach allows for an investigation of whether different interventions utilize different pathways to influence this daily relationship between worry and sleep.

One behavioral intervention targeted at worry that is often included in CBT packages for GAD and that has produced reductions in sleep disturbance is stimulus control for worry. For chronic worriers, excessive worry becomes associated with numerous aspects of daily life (e.g., time of day, environmental cues). As a result, individuals become conditioned to worry about many different topics in a variety of contexts and are thus described as having poor

discriminative stimulus control. In stimulus control for worry, individuals are taught to break their maladaptive associations in order to gain greater control over the incidence of their worry. Interestingly, the rationale behind stimulus control for worry comes from Bootzin's (1972) stimulus control treatment for insomnia. In the first empirical test of stimulus control for worry, Borkovec, Wilkinson, Folensbee, and Lerman (1983) provided clients with four general rules: (1) identify worrisome and unpleasant thoughts and learn to distinguish those from other more pleasant thoughts; (2) establish a 30-minute "worry period" to occur at the same time and in the same location each day; (3) delay spontaneous worry to the worry period and instead focus on the present moment; and (4) use the 30-minute worry period to worry about concerns and problem solve to reduce or eliminate concerns. McGowan and Behar (2013) recently provided preliminary evidence for the efficacy of stimulus control for worry by comparing it to a credible placebo control condition (called "focused worry"). Although both conditions evidenced significant reductions in worry, anxiety, negative affect, and insomnia symptoms, stimulus control produced greater reductions in these symptoms. Additionally, despite stimulus control being targeted at general worry without the inclusion of any strategies for improving sleep, it produced clinically significant reductions in sleep disturbance.

Several interventions that target worry have demonstrated improvements in sleep disturbance, suggesting that worry – particularly worry related to sleep – plays an important role in sleep disturbance. However, relatively few studies have examined whether changes in sleep disturbance affect worry symptoms for individuals undergoing these interventions. Thus, it is important to not only examine the impact that worry has on sleep disturbance, but also to examine the impact that sleep disturbance has on worry. Additionally, given that these two phenomena likely fluctuate on a daily basis, it is important to examine the process of symptom

change throughout the course of an intervention, rather than relying on the limited information produced by outcome data. Such an examination could provide insight into the mechanisms of change in various interventions.

E. The Present Study

The aims of this investigation were to examine the nature of the relationship between worry and sleep disturbance among high trait worriers, with attention to how these phenomena affect each other on a daily basis. Specifically, the primary aim was to examine whether worry experienced during the day and prior to sleep onset predicted characteristics of sleep that night. The secondary aim was to examine whether characteristics of nighttime sleep predicted worry the following day. Should both predictive relationships exist between worry and sleep, it would suggest a bidirectional relationship. The tertiary aim was to examine whether the relationship between worry and sleep disturbance changed after individuals began an intervention for reducing worry. Relatedly, we sought to examine whether the relationship between worry and sleep disturbance is moderated by type of intervention. McGowan and Behar (2013) indicated that stimulus control was superior to focused worry in reducing both worry and sleep disturbance; however, it is unclear whether the two conditions impacted the relationship between worry and sleep disturbance differentially throughout the intervention period. Such information would add to our understanding of how mechanisms of change may differ between different interventions. Finally, given the relationship between worry, anxiety, and negative affect (Borkovec, Ray, & Stober, 1998), as well as between insomnia, tension, and negative affect (Berry & Webb, 1983, 1985; Ford & Kamerow, 1989), our final aim was to examine whether the relationship between sleep disturbance and worry was mediated by tension, anxiety, or affect.

Given that there are relatively few studies examining the daily relationship between worry and sleep disturbance, this investigation provides an important first step in identifying the potential predictive relationship between these two phenomena. We utilized a daily process approach (Affleck, Zautra, Tennen, & Armeli, 1999) in which we included repeated measurements of variables that are thought to change in meaningful ways each day. Several studies have used a daily process approach to examine the relationship between chronic pain and depression history, coping strategies, and sleep disturbance (Keefe et al., 1997; Rumble et al., 2010; Tennen, Affleck, & Zautra, 2006). This approach (1) allows for the examination of fluctuations in the relationship between variables as they change over time; (2) reduces biases due to retrospective reporting by minimizing the time between an event and emotional responding; and (3) allows for the examination of a temporal relationship between variables (Laurenceau, Hayes, & Feldman, 2007; Tennen & Affleck, 1996). Finally, this approach allows investigators to examine more sophisticated questions in the process of symptom change than can be addressed using outcome data.

Data for the present study came from McGowan and Behar (2013), described earlier. Specifically, high trait worriers were assigned to either the stimulus control intervention or a credible control procedure called *focused worry*. Following the therapeutic model established in Borkovec et al. (1983), the stimulus control intervention included having participants identify a 30-minute worry period to occur at the same time and in the same location each day. In the focused worry intervention, participants were instructed to worry as they normally would and to not avoid naturally occurring worry so that worry and anxiety would not paradoxically increase. Participants first monitored (using personal data assistants; PDAs) worry and sleep symptoms during a baseline week (prior to random assignment to an intervention) and then monitored

worry, sleep, and emotional functioning for two weeks while complying with one of the two interventions aimed at reducing worry symptoms.

F. Hypotheses of the Present Study

Given the limited research in this area, our analyses were exploratory in nature. First, we hypothesized that after controlling for the previous night's sleep characteristics, worry would predict that night's sleep characteristics. This result would provide support for the cognitive model of insomnia and further evidence that worry negatively impacts sleep disturbance. Second, controlling for the previous day's worry, we predicted that sleep characteristics would predict worry the following day. This result would suggest that poor sleep impacts worrisome thinking. If both hypotheses 1 and 2 are supported, our results would suggest a bidirectional relationship between worry and sleep disturbance.

Third, we examined whether the type of intervention would moderate the relationship between worry and sleep. This analysis was exploratory in nature because we did not have any theory or past evidence on which to base a prediction of moderation. Finding a moderating effect of intervention would suggest that the relationship between worry and sleep is influenced by external factors of intervention and might give some insight into the mechanisms of change inherent to each intervention. However, if intervention does not moderate the relationship between worry and sleep disturbance, it would suggest that although the interventions differed in their ability to *reduce* worry and insomnia symptoms, they did not affect the relationship *between* worry and sleep. This finding would suggest that the relationship between worry and sleep is relatively stable. Finally, we expected that emotions of affect, anxiety, and tension would mediate the relationship between sleep and worry. Some research suggests that poor sleep quality negatively impacts emotional responding (Baglioni et al., 2010). Although we did not measure

subjective sleep quality, the variable of sleep efficiency has often been used as an index of sleep quality (e.g., Spielman, Saskin, & Thorpy, 1987). We hypothesized that emotional functioning (e.g., affect, anxiety and tension) would mediate the relationship between sleep efficiency and daily worry. This finding would suggest that worry and sleep do not directly impact one another, but rather do so through the influence of emotion.

II. METHOD

A. Participants

Fifty-three introductory psychology students from a large Midwestern university were included in this investigation. Participants were invited to take part in the study if they scored 67 or higher on the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), which was administered as part of a group screening process. This score has been shown to distinguish GAD individuals from non-anxious individuals (Molina & Borkovec, 1994). Participants were included in the study if they scored 53 or higher on the PSWQ at the baseline assessment (shown to be one standard deviation above the mean for normal individuals; Gillis, Haaga, & Ford, 1995). On average, participants' scores on the PSWQ decreased from the group screening administration to the baseline assessment ($M = -4.11$, $SD = 6.53$); however, participants still evidenced elevated PSWQ scores at the baseline assessment ($M = 68.00$, $SD = 5.84$). It is important to note that although participants in this study were selected for their high trait worry, they were not formally diagnosed with insomnia. Three participants' data were excluded from analyses because the participants dropped out of the study for personal reasons prior to being randomly assigned to an intervention ($n = 2$) or no longer met the inclusionary criteria at the baseline assessment ($n = 1$). Remaining participants were predominantly female (82.0%), with a mean age of 19.72 years ($SD = 3.7$ years). Our sample was ethnically diverse and was comprised of 42% Caucasian, 10% African-American, 20% Asian, 14% Latino, and 14% other participants. Participants included in the analyses did not differ from excluded participants with respect to age ($t[50] = -0.98$, ns), sex ($\chi^2[1] < 0.01$, ns), ethnicity ($\chi^2[1] = 1.62$, ns) or race ($\chi^2[7] = 7.33$, ns). The purpose and hypothesis of the study was masked throughout the experiment (as detailed below). Participants were randomly assigned to one of two interventions

aimed at reducing worry – Stimulus Control ($n = 26$) or Focused Worry ($n = 24$). Participants received class credit as compensation for participation in the experiment.

B. Symptom Measures

1. Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). The PSWQ is a 16-item self-report questionnaire that assesses the frequency and intensity of worry. The PSWQ has demonstrated favorable reliability and validity for both clinical and nonclinical populations (Brown, Antony, & Barlow, 1992). It has good sensitivity (0.75) and specificity (0.86) in distinguishing GAD samples from non-anxious controls and from other anxiety groups (Behar, Alcaine, Zuellig, & Borkovec, 2003; see also Brown et al., 1992). In this study, the PSWQ had acceptable internal consistency (0.72).

2. Insomnia Severity Index (ISI; Bastien, Vallieres, & Morin, 2001). The ISI is a 7-item self-report questionnaire that assesses sleep problems, impairment of functioning due to inadequate sleep, and perceptions of severity of insomnia. The ISI has good concurrent validity at baseline (0.32-0.55) and following CBT, pharmacotherapy, or combination treatment (0.50-0.91; Bastien et al., 2001). Additionally, the ISI has good internal reliability at baseline (0.76) and follow-up (0.78; Bastien et al., 2001). In this study, the ISI had good internal consistency (0.82).

3. Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegan, 1988). The PANAS is a 20-item self-report measure that assess both positive (PA) and negative (NA) affect. Participants were asked to rate their affect in the moment. The PANAS evidences good convergent (0.81-0.92 for positive; 0.76-0.91 for negative) and discriminant (-0.36-0.12 for PA; -0.43-0.11 for NA) validities. Additionally, the PANAS demonstrates good reliability for both PA (0.47-0.68) and NA (0.39-0.71). The PANAS is used widely as a measure of general affect.

C. Daily Diaries

1. Sleep diaries. Sleep diaries are an essential component of studies examining sleep and insomnia symptoms (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006). As recommended by Buysse et al. (2006), sleep was assessed by having participants estimate the time they attempted to fall asleep (e.g., 21:30), the amount of time they spent trying to fall asleep (e.g., 30 minutes), the time they actually fell asleep (e.g., 23:00), an estimate of the number of hours slept (e.g., 6.5 hours), the number of nocturnal awakenings (e.g., 3), and the time of awakening in the morning (e.g., 7:15). Standard sleep continuity measures were extracted from the sleep diaries including sleep efficiency, sleep onset latency, total sleep time, and number of awakenings (Buysse et al., 2006). Sleep efficiency ($[\text{total sleep time}] / [\text{time in bed}] * 100$) was calculated as one index of sleep quality (Spielman et al., 1987) as it is an important measurement to extract from sleep diaries (Buysse et al., 2006). Additionally, participants provided a rating of the extent to which they felt worried prior to falling asleep on a scale of 1 (not at all worried) to 5 (extremely worried).

2. Daily emotion diaries. Measures of emotion included PA, NA, anxiety, and tension. PA and NA were calculated from the PANAS. Anxiety was measured by asking participants to indicate (using a 1 ‘not at all’ to 5 ‘extremely’ scale) how anxious they felt in the moment. Tension was measured by asking participants to indicate (using a 1 ‘not at all’ to 5 ‘extremely’ scale) how tense they felt in the moment. These measures were independently averaged each day to create one daily emotion value for each variable. Specifically, daily averages of PA, NA, tension, and anxiety were calculated for each day during the intervention weeks.

3. End of day diary. Participants were asked to rate the percentage of the day (0-100%) during which they engaged in worry.

D. Procedure

1. Baseline laboratory session. Following participants' consent to take part in the study, they were asked to complete the PSWQ and the ISI. Participants were then given instructions on how to complete the sleep diaries during the baseline week.

2. Baseline week. Participants completed a sleep diary each morning for seven days using the online survey tool Survey Monkey (1999-2013). Participants were instructed to complete daily sleep diaries immediately upon waking.

3. Intervention laboratory session. Participants returned to the laboratory to receive instruction and training in their intervention. The experimenter met with each participant individually to explain the intervention instructions. All instructions were scripted in order to ensure consistency of information across participants and to reduce the risk of experimenter bias. Participants were also provided with written materials that reiterated the rationale for the intervention and all instructions that had been delivered by the experimenter.

a. Stimulus control. The experimenter first provided participants with the rationale for the intervention. Participants were told that when worry occurs throughout the day, it can become associated with many places, times, and situations, such that over time mere exposure to those places, times, and/or situations can come to elicit spontaneous worry. The goal of stimulus control is thus to reduce the frequency of worry by gradually coming to associate worry with more distinct and specific times and locations, so that only those times and locations come to elicit worry and its associated emotional experiences. Participants were given the same four instructions as outlined earlier from Borkovec et al. (1983). Specifically, participants were taught to identify their worrisome thoughts and were asked to identify a specific and consistent 30-minute worry period and location each day during which they would focus on these worries.

In helping participants to identify a 30-minute worry period, the first author ensured that the period was at least three hours prior to participants' bedtimes so that the worry process would not interfere with their ability to fall asleep, given that worry and anxiety that occur before bedtime can lead to increased subjective reporting of insomnia and daytime fatigue (Chambers & Kim, 1993). Participants were instructed to worry as they normally do during their prescribed worry period, to do so as intensely as possible, and to keep the focus of their attention on the worry process. They were instructed to postpone spontaneous worry during the day to the worry period and to instead focus on the present-moment experience when they noticed themselves worrying outside of their prescribed worry periods. Finally, they were instructed to use the worry period to worry about their concerns and problem solve when appropriate.

b. Focused worry. In this condition, we sought to provide participants with a rationale that would encourage them to engage in worry as they normally do, and to expect that this practice would lead to a reduction in worry and its associated emotions. Compared to non-anxious individuals, individuals with GAD are more susceptible to experiencing paradoxical increases in worry while trying to suppress worrisome thoughts (Becker, Rinck, Roth, & Margraf, 1998). Thus, participants in this condition were told that people often try to avoid the occurrence of worry throughout the day, which paradoxically leads to increased levels of worry and its associated emotional experiences. The goal of the intervention was thus to help them *not* avoid spontaneous worry, but rather to allow it to naturally occur in order to decrease the frequency and intensity of paradoxical increases in anxious thoughts and emotions. Participants were instructed to worry as they normally do anytime they noticed such thoughts occurring, to make this worry as intense as possible, and to keep the focus of their attention on the worry process. Although participants in this condition did not have a prescribed amount of time during

which they were to engage in worrying each day, and thus may have engaged in more worry relative to participants in the stimulus control intervention, it is important to note that chronic worriers report worrying for the majority of the day (60% of the day, as compared with 18% for non-anxious individuals; Craske, Rapee, Jackel, & Barlow, 1989). Thus, we anticipated that a credible control intervention would most likely lead to reductions (as opposed to further increases) in overall levels of daily worry. It should be noted that the focused worry condition contained some elements of existing treatment packages. For example, the instructions to not avoid spontaneous worry are similar to the experiential exercises taught in Acceptance and Commitment Therapy (ACT; Hayes, 2004; Hayes, Luoma, Bond, Masuda, & Lillis, 2006). Similarly, the instruction to focus attention on the process of worry is consistent with mindfulness techniques that have been shown to reduce worry among individuals with GAD (Roemer, Orsillo, & Salters-Pedneault, 2008).

Participants were provided with a PDA and instructions on how to use the device. Experience sampling software was programmed onto all PDAs that included all study materials and diaries. Experience sampling is a method of data collection that allows for “in the moment” responses and can assess real-time feelings and reactions to experiences (Barrett & Barrett, 2000). The Experience Sampling Program (ESP; Barrett & Barrett, 2000) is downloaded onto PDAs that the participants carry and that prompts the participants to answer specific questions at specific times. One advantage of using experience sampling over self-report questionnaires is that experience sampling reduces memory biases and it can track participant compliance with study procedures (Barrett & Barrett, 2000). Participants in each intervention were asked to identify three times throughout the day during which they would be prompted by the PDA to complete diaries about their emotions in the moment. Each PDA was individually programmed

to remind participants to complete these diaries at their scheduled times. Additionally, all participants were instructed to complete a sleep diary each morning and an end-of-day diary before going to bed each night.

4. Intervention weeks. Participants engaged in their interventions and completed five assessments each day (sleep diary, emotion diaries, end-of-day diary) over a 14-day period. Participants completed all diaries on the PDAs. Participants were instructed to complete sleep diaries immediately upon awakening (the sleep diary was identical to the one completed during the baseline week). Participants were instructed to complete measures of their emotions in the moment three times throughout each day. These times were pre-arranged by the participant and the experimenter according to the participant's schedule, and were programmed by the experimenter into the PDA to alert participants to complete the questionnaires. Finally, participants were instructed to complete end-of-day diaries as their last activity of the day.

Given that participants in the present study were undergraduate students, special consideration was given to the timing that participants engaged in this procedure. Participants exclusively followed this protocol during the school year and never engaged in the study protocol during school breaks or during finals in order to reduce the effects of environmental changes and stressors.

III. RESULTS

A. Daily Diary Compliance

Participants were instructed to complete one diary entry per day during the baseline week and five diary entries per day during each of the two intervention weeks. Of the 3,850 total diaries requested from the final sample ($N = 50$), 3,413 (88.6%) were completed. Specifically, during the baseline week, participants completed 93.14% of the requested sleep diaries. During the intervention weeks, participants completed 91.29% of the requested sleep diaries and 85.71% of the requested end-of-day diaries used in the main analyses.

B. Descriptive Statistics of Baseline Measures and Sleep Diary Variables

Table 1 presents descriptive statistics for baseline measures (i.e., PSWQ and ISI) and sleep diary variables (i.e., sleep efficiency, sleep onset latency, total sleep time, number of awakenings, and worry before bed).

Table 1

Means (SDs) and Ranges for Baseline Symptom Measures and Sleep Variables

Variable	Mean (SD)	Range
Baseline laboratory session		
PSWQ	68.00 (5.84)	56-80
ISI	13.35 (5.60)	4-27
Average across baseline days		
Sleep Efficiency (%)	84.74 (8.95)	60.13-97.05
Sleep onset latency (mins)	38.58 (33.28)	5.71-203.86
Total sleep time (hours)	6.72 (0.99)	4.17-8.13
Number of Awakenings	2.69 (0.91)	1.29-4.86
Worry before bed	2.63 (0.81)	1-5

Note. SD = Standard Deviation; PSWQ = Penn State Worry Questionnaire; ISI = Insomnia Severity Index.

As a reminder, participants in the present study were recruited on the basis of their high trait worry; thus, as expected, the mean baseline PSWQ score was in the clinically significant range.

Furthermore, the mean baseline ISI score was also in the clinically significant range. More specifically, 14% were within the ‘no clinical insomnia’ range, 46% were within the ‘subthreshold insomnia’ range, 34% were within the ‘clinical insomnia-moderate severity’ range, and 6% were within the ‘clinical insomnia-severe’ range (as per cutoffs established by Morin, 1993).

Descriptive statistics for the sleep diary variables presented in Table 1 were calculated by averaging responses across the entire baseline week. On average, participants took 39 minutes to fall asleep, slept 6.7 hours per night, and slept 85% of the time that they were in bed, consistent with subjective reports of sleep characteristics in other populations of anxious individuals (Rosa, Bonnet, & Kramer, 1983). One participant was an outlier (> 4 SDs above the mean) with respect to his/her reports of sleep onset latency. Results were nearly identical when this participant was excluded from analyses; therefore, we retained this participant in all analyses. Participants also reported moderate levels of worry prior to bed.

C. Approach to Multilevel Analyses

The present study utilized a repeated measures multilevel modeling approach. Data for the main analyses were nested (i.e. observations nested within individuals), and thus multilevel modeling analyses were conducted using Mplus (Muthén & Muthén, 2012). In multilevel modeling, two sources of variance are partitioned in the dataset: differences between persons on the average levels of daily variables and differences within persons on their daily reporting of variables over time. Level 1 variables represent the within-person observations that are being measured over time, which in the present study included sleep efficiency, sleep onset latency, total sleep time, number of awakenings, worry before bed, and percentage of daily worry. Level

2 variables are the between-person groupings, which in the present study was intervention (Stimulus Control vs. Focused Worry).

A multilevel repeated measures analysis was used to examine the lagged within-persons relationship between level 1 variables. More specifically, we examined two directional pathways: (1) whether worry during the day predicted sleep disturbance that night and (2) whether nighttime sleep disturbance predicted worry the following day. Separate analyses were conducted for data collected during the baseline week and intervention weeks, and analyses for data collected during the intervention weeks also examined whether the level 2 variable intervention (Stimulus Control vs. Focused Worry) moderated the association between sleep and worry. One of the advantages of using Mplus is that it allows for the examination of both repeated measures pathways (i.e. worry predicting sleep and sleep predicting worry) in the same model by using a multilevel path modeling approach. As a result, we can examine whether one or both pathways are significant, and, if both pathways are significant, we can compare the relative strength of each pathway to the other.

The Mplus procedure for multilevel analyses utilized a maximum likelihood estimator to allow for the modeling of individually varying times of observations and random slopes for time-varying covariates. For all analyses, dependent variables were left in their original metric. Independent predictor variables were person-centered in order to eliminate any between-persons variation and produce a regression model that is solely based on within-persons variation (Enders & Tofighi, 2007). To ensure that the complex model described above was the optimal fit for the data, a process of testing nested models was followed wherein the simplest model was first estimated. The models grew more complex as one additional consideration was added at each step until the full model was estimated. At each step, we compared the models using a deviance

test to determine which model best fit the data (Snijders & Bosker, 2012). In deviance testing, the difference between the two deviances of each model, D_I and D_0 , results in the χ^2 distribution with degrees of freedom equal to the difference in parameters estimated in each model. A significant χ^2 test is evidence that the null hypothesis is implausible and the two models significantly differ from each other. In this case, the model with a lower deviance estimate (i.e. the more complex model) is determined to be a better fit for the data.

We will illustrate our modeling process using the generic variables *worry* and *sleep*. In the first model examining the worry predicting sleep pathway, the simplest model was the regression of worry_{today} onto sleep_{tonight}. In the second model, we controlled for the previous day's sleep by adding the covariate of sleep_{yesterday}. In the third model, we added the level 2 between-persons variable of intervention.¹ Similarly, in the first model examining the sleep predicting worry relationship, the simplest model was the regression of sleep_{tonight} onto worry_{tomorrow}. In the second model, we controlled for the previous day's worry by adding the covariate of worry_{today}. In the third model, we added the level 2 between-persons variable of intervention. It should be noted here that intervention did not moderate the within-person associations for any worry-sleep relationship and thus was not included in the final models (all $ps > .30$). The final model included two fixed-slopes regression pathways: (1) worry_{today} predicting sleep_{tonight} while controlling for sleep_{yesterday}; and (2) sleep_{tonight} predicting worry_{tomorrow} while controlling for worry_{today} (see Figure 1)². The same procedure was repeated for each worry-sleep relationship. For baselines analyses, this utilized 1 'worry' variable and 4 'sleep'

¹ Because data from the baseline week occurred prior to participants' random assignment to condition, the level 2 variable of intervention was not included in any baseline analyses.

² We estimated these models with random slopes; however, due to convergence issues we were unable to run the complex pathway models with random slopes and thus used fixed slopes for all following models (Geiser, personal communication, 2012).

variables yielding four full models³. For analyses during the treatment weeks, this utilized two ‘worry’ variables and four ‘sleep’ variables, yielding eight full models.

Figure 1. Representation of complex models estimated using multilevel repeated measures analysis.

For the multilevel mediation models (see Figure 2), we utilized a 1-1-1 multilevel model mediation design following the procedures outlined in Preacher, Zyphur, and Zhang (2010) as well as the syntax for Mplus provided in their supplemental material. The confidence intervals for the indirect effects in multilevel mediation were estimated using the Monte Carlo method (MacKinnon, Lockwood, & Williams, 2004) utilizing an online calculator (Preacher & Selig, 2010). Measurements of emotion (NA, PA, anxiety, and tension) only occurred between measurements of sleep and worry; thus, we only examined whether emotion mediated the sleep predicting worry relationship. To be consistent with our hypotheses, we examined whether NA,

³ There was half as much data for the baseline week as there was for the intervention weeks; thus, we could not estimate the complex pathway models. Results from the baseline week reflect each pathway from the full models reported independently.

PA, anxiety, and tension independently mediated the relationship between sleep efficiency and percentage of daily worry.

Figure 2. Representation of the mediation pathways between sleep efficiency, emotion, and daily worry.

D. Baseline Week: Lagged Within-Person Analyses

Results indicated that greater worry before bed predicted decreased sleep efficiency, decreased total sleep time, and greater sleep onset latency (see Table 2).

Table 2

Repeated Measures Multilevel Regression Results during Baseline Week

Predictor:	Outcome:															
	Sleep efficiency ^a tonight				Sleep onset latency ^b tonight				Total sleep time ^c tonight				Number of awakenings ^d tonight			
	<i>B</i>	β	<i>t</i>	<i>p</i>	<i>B</i>	β	<i>t</i>	<i>p</i>	<i>B</i>	β	<i>t</i>	<i>p</i>	<i>B</i>	β	<i>t</i>	<i>p</i>
<i>Worry variables (today)</i>																
Worry before bed	-5.26	-.29	-4.81	< .001	12.52	.25	2.93	< .01	-0.39	-.20	-3.61	< .001	0.14	.08	0.99	.32

Predictor:	Outcome:			
	Worry before bed^e tomorrow			
<i>Sleep variables (tonight)</i>	<i>B</i>	β	<i>t</i>	<i>p</i>
Sleep efficiency	0.01	.07	1.01	.31
Sleep onset latency	< 0.01	.06	1.31	.19
Total sleep time	< 0.01	< .001	< 0.01	.99
Number of awakenings	0.04	.05	0.52	.60

Note. *B* = unstandardized coefficient; β = standardized coefficient; *t* and *p* values refer to the unstandardized coefficients; ^acontrolling for previous day's sleep efficiency, ^bcontrolling for previous day's sleep onset latency, ^ccontrolling for previous day's total sleep time, ^dcontrolling for previous day's number of awakenings, ^econtrolling for previous day's worry before bed.

A comparison of the β s indicates that worry before bed had the strongest association with decreased sleep efficiency, although worry before bed also had a strong association with increased sleep onset latency. Worry before bed had a significant, but weaker, association with total sleep time. However, worry before bed was not associated with number of awakenings. Results indicated that none of the sleep variables (sleep efficiency, sleep onset latency, total sleep time, or number of awakenings) significantly predicted worry before bed the following day. In other words, worry before bed significantly predicted several measures of sleep characteristics; however, none of the measured sleep characteristics predicted worry before bed.

E. Intervention Weeks: Lagged Within-Person Analyses

Similar to the baseline week analyses, greater worry before bed during the intervention weeks predicted decreased sleep efficiency and total sleep time as well as increased sleep onset latency and number of awakenings (see Table 3).

Table 3

Repeated Measures Multilevel Regression Results during Intervention Weeks

Predictor:	Sleep efficiency ^a tonight				Sleep onset latency ^b tonight				Total sleep time ^c tonight				Number of awakenings ^d tonight			
	<i>B</i>	β	<i>t</i>	<i>p</i>	<i>B</i>	β	<i>t</i>	<i>p</i>	<i>B</i>	β	<i>t</i>	<i>p</i>	<i>B</i>	β	<i>t</i>	<i>p</i>
<i>Worry variables (today)</i>																
Percentage of daily worry	-0.02	-.07	-2.62	< .01	0.08	.05	1.11	.27	-0.02	-.16	-3.75	< .001	< 0.01	.04	1.21	.23
Worry before bed	-2.36	-.28	-5.03	< .001	7.71	.28	5.67	< .001	-0.29	-.16	-3.46	.001	0.45	.08	5.67	< .001

Predictor:	Percentage of daily worry ^e tomorrow				Worry before bed ^f tomorrow			
	<i>B</i>	β	<i>t</i>	<i>p</i>	<i>B</i>	β	<i>t</i>	<i>p</i>
<i>Sleep variables (tonight)</i>								
Sleep efficiency	0.17	.08	1.47	.14	> -0.01	-.02	-0.41	.68
Sleep onset latency	-0.06	-.10	0.05	.25	< 0.01	-.01	-0.16	.87
Total sleep time	0.46	.05	1.02	.31	0.03	.05	1.43	.15
Number of awakenings	-0.65	-.04	-1.05	.30	0.02	< .01	0.40	.67

Note. *B* = unstandardized coefficient; β = standardized coefficient; *t* and *p* values refer to the unstandardized coefficients; ^acontrolling for previous day's sleep efficiency, ^bcontrolling for previous day's sleep onset latency, ^ccontrolling for previous day's total sleep time, ^dcontrolling for previous day's number of awakenings, ^econtrolling for previous day's percentage of daily worry, ^fcontrolling for previous day's worry before bed.

A comparison of the β s indicated that worry before bed had the strongest relationship with decreased sleep efficiency, although worry before bed also had a strong association with increased sleep onset latency. Worry before bed had significant, yet weaker, associations with total sleep time and number of awakenings. Consistent with baseline week results, none of the sleep variables (sleep efficiency, sleep onset latency, total sleep time, number of awakenings) significantly predicted worry before bed.

Results indicated that greater percentage of daily worry predicted decreased sleep efficiency and total sleep time. However, percentage of daily worry did not predict sleep onset latency or number of awakenings. A comparison of the β s indicated that percentage of daily worry had a greater association with total sleep time than with sleep efficiency, although both associations were relatively weak. None of the sleep variables (sleep efficiency, sleep onset latency, total sleep time, number of awakenings) significantly predicted percentage of daily worry. In sum, both worry variables predicted several sleep variables; however, sleep consistently did not predict worry.

F. Multilevel Affect Mediation

Results of the multilevel mediation analyses appear in Table 4.

Table 4

The Relationship between Sleep Efficiency and Percentage of Daily Worry Mediated by Affect, Anxiety, and Tension

Mediator	<i>a</i> path	<i>b</i> path	Indirect effect [95% CI]
Negative affect	-0.06 (0.03)	1.51 (0.38)*	-0.07 [-0.45 - 0.24]
Positive affect	0.04 (0.04)	-0.32 (0.30)	0.03 [-0.14 - 0.20]
Anxiety	-0.01 (0.03)	5.94 (8.38)	-0.11 [-17.47 - 15.30]
Tension	< -0.01 (0.01)	6.48 (1.60)*	-0.04 [-1.47 - 1.17]

Note. Standard errors are shown in parentheses. Confidence intervals are based on the Monte Carlo method (Preacher & Selig, 2010, July).

* $p < .001$.

Indirect pathways are considered significant if the confidence interval does not include zero.

Results indicated that emotion did not mediate the relationship between sleep efficiency and worry.

IV. DISCUSSION

The present study utilized a daily process approach to examine the relationship between worry and sleep in a sample of high trait worriers. Participants completed daily reports of sleep and worry during a 7-day baseline period, as well as daily reports of sleep, worry, and emotion during a 14-day intervention period. The aims of the study were to examine whether (1) worry experienced during the day and prior to sleep onset predict characteristics of sleep that night; (2) characteristics of nighttime sleep predicts worry the following day; (3) beginning an intervention and/or the type of intervention moderates the relationship between worry and sleep; and (4) emotion mediates the relationship between sleep and worry.

Results indicated that worry occurring throughout the day and prior to bed had a negative impact on several sleep variables. Specifically, during the baseline and intervention weeks, worry before bed predicted decreased sleep efficiency and total sleep time as well as increased sleep onset latency. During the intervention weeks, worry before bed additionally predicted increased number of awakenings. Furthermore, the percentage of worry that participants experienced during the day predicted decreased sleep efficiency and total sleep time during the intervention weeks. These results suggest that worry has negative effects on sleep.

Our finding that worry occurring before bed negatively affects sleep is consistent with the cognitive model of insomnia (Harvey, 2002). The cognitive model of insomnia posits that worry before bed causes increased autonomic arousal and emotional distress that interferes with sleep onset. Extant literature supports the claim that excessive worry – especially worry occurring before bed – contributes to sleep disturbance (Borkovec 1979, 1982; Harvey, 2002; Morin, 1993). Harvey (2002) posits that pre-sleep worry accounts for problems with sleep onset latency as well as sleep maintenance (i.e. sleep efficiency). Consistent with this model, worry before bed

evidenced the strongest association with decreased sleep efficiency and increased sleep onset latency. Furthermore, the present study supports the cognitive model of insomnia by demonstrating that worry occurring throughout the day also directly contributes to sleep disturbance. Although Harvey (2002) theorized that worrisome thought occurring during the day contributes and maintains insomnia, our study is one of the first to provide empirical support for the claim that worry on a particular day negatively impacts sleep characteristics that night. This finding suggests that sleep disturbance is not only affected by worrying while attempting to fall asleep. Rather, worry has lasting effects such that its occurrence throughout the day continues to have negative effects that contribute to sleep disturbance.

Although daily worry predicted reduced sleep efficiency and total sleep time, it did not predict sleep onset latency, which is often assumed to be the result of cognitive intrusions before bed (Harvey, 2000; 2002; Monti & Monti, 2000). Interestingly, worry before bed was associated with all aspects of sleep disturbance (trouble falling asleep and staying asleep), whereas daily worry seemed to only be associated with characteristics of sleep maintenance (sleep efficiency and total sleep time). One possible reason for this finding is that the accumulation of worry experienced throughout the day does not have a direct impact on the time it takes one to fall asleep, but rather has other lasting effects that interfere with sleep maintenance. Brosschot, Van Dijk, and Thayer (2007) found that prolonged worry was associated with higher heart rate and lower heart rate variability not only during waking, but also during sleep periods, suggesting that worry's maladaptive physiological effects might interfere with the sleep process. Thus, it is likely that worry has both cognitive and physiological consequences that interfere with sleep. Specifically, worry that is experienced immediately prior to bedtime contributes to sleep onset latency (as it is cognitive activity occurring before bed) as well as other indices of sleep

disturbance, whereas worry experienced throughout the day has a negative effect on sleep maintenance. These findings provide further support for the cognitive model of insomnia and suggest that there are multiple pathways through which worry negatively impacts sleep.

It is important to note that although the cognitive model of insomnia is specific to worry about sleep itself, the present study did not assess worry content. In fact, because participants were recruited for their high trait worry (and not for their insomnia status), it is likely that these individuals' worry was not focused on sleep. Anxious individuals tend to worry about a number of different topics, including work, school, finances, and health (Roemer, Molina, & Borkovec, 1997). Watts, Coyle, and East (1994) examined the content of pre-sleep cognitions in self-described insomniacs who demonstrated high or low trait worry. Results indicated that worried insomniacs reported a broad range of topics during their pre-sleep cognitions; however, non-worried insomniacs reported that their pre-sleep cognitions focused specifically on sleep-related concerns (Watts et al., 1994). Thus, although it is well supported that worry contributes to sleep disturbance, the content of worry that interferes with sleep might differ as a function of whether an individual demonstrates high or low trait worry. Such a distinction is important because it has implications for how to target and treat the maladaptive cognitions that occur prior to sleep onset. Additionally, results from the present study suggest that individuals with high trait worry demonstrate a similar pattern of worry negatively affecting sleep as described by the cognitive model of insomnia despite the fact that none of the participants were formally diagnosed with insomnia. This suggests that the model posited by Harvey (2002) may not be unique to individuals with insomnia.

Results from the present study demonstrate the negative effects that worry has on sleep characteristics and suggest that addressing worry is vital to impacting change in sleep

disturbance. Several pharmacological (Monti & Monti, 1995), behavioral (Morin, Hauri, Epsie, Spielman, Buysse, & Bootzin, 1999), cognitive (Harvey, 2005), and cognitive-behavioral (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Morin et al., 1999) interventions exist for the treatment of insomnia. Cognitive behavioral therapy for insomnia (CBT-I; Edinger, et al., 2001; Morin & Epsie, 2003) is a relatively new intervention for insomnia compared with other pharmacological and behavioral interventions that have existed for decades. Cognitive therapy was included in treatment packages given evidence that addressing cognitive maintaining factors of disorders increases the short-term and long-term efficacy of treatments (Harvey, 2005; Salkovskis, 2002). CBT-I uses an integrated approach of psychoeducation about insomnia, behavioral interventions (sleep hygiene, sleep restriction, stimulus control, relaxation training), and cognitive therapy (see Morin & Epsie, 2003), and has been shown to be an effective treatment for individuals with insomnia (Morin et al., 1999; Morin et al., 2006). Although research has demonstrated that behavioral interventions for insomnia are efficacious on their own when compared to waitlist or no-treatment conditions (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Epsie, Lindsay, Brooks, Hood, & Turvey, 1989; Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001), CBT-I additionally addresses the maladaptive cognitions and worries associated with sleep disturbance. Results from the present study suggest that addressing worry and negative cognitive activity may be crucial to creating significant changes in sleep disturbance, especially for individuals who experience high trait worry. Additionally, it is important to address and reduce worry experienced throughout the day in addition to worry prior to bedtime in order to reduce the effects of worry on sleep. In fact, it was previously demonstrated that participants from this study evidenced significant reductions in insomnia symptoms after undergoing an intervention targeted at reducing worry (McGowan & Behar,

2013). Additionally, Bélanger et al. (2004) found that CBT for worry also significantly reduced insomnia symptoms. Thus, there is preliminary evidence to suggest that reducing worry might be efficacious as a stand-alone treatment for improving sleep disturbance. However, a dismantling study of CBT-I is needed to determine which elements of the intervention are necessary and sufficient.

Importantly, our results did not find evidence of a bidirectional relationship between worry and sleep in that sleep characteristics did not predict worry. Although several studies suggest that insomnia and reduced sleep length are associated with and possibly contribute to worry and anxiety (e.g., Kelly, 2002; Kumar & Vaidya, 1984; Means et al., 2000), many of these studies use methodological designs that preclude conclusions about directionality. Additionally, few studies have utilized a daily process approach to examine the direct impact of sleep disturbance on worry. Instead, many investigations have examined the relationship between insomnia and anxiety disorders more globally. Specifically, several studies have demonstrated that the development of insomnia predates the development of anxiety and mood disorders and have suggested that insomnia might contribute to the onset of these disorders (Ford & Kamerow, 1989; Vollrath, Wicki, and Angst, 1989; see Harvey, 2001 for a review). These studies suggest that insomnia represents a risk factor for the later development of psychiatric disorders (Roth, 2007). One interpretation of these findings is that sleep disturbance leads to symptoms of anxiety, irritability, and worry, which later develop into anxiety and mood disorders. Another interpretation of these findings is that symptoms of worry, anxiety, and tension initially contribute to the development of insomnia and later contribute to the development of mood and anxiety disorders. Results of the present study are more consistent with the latter interpretation. In other words, for some individuals, the emergence of insomnia may be a prodrome to the

development of other psychological disorders. Indeed, several investigations have demonstrated that insomnia is a prodromal feature of mood and anxiety disorders (Eaton, Badawi, & Melton, 1995; Neckelmann, Nykletun, & Dahl, 2007; see Gillin, 1998 for review). Although these investigations suggest that insomnia symptoms also contribute to the development of psychological disorders, our results suggest that sleep disturbance does not affect worry on a daily basis.

Also inconsistent with our hypotheses was the finding that daily emotions did not mediate the relationship between sleep and worry. Because sleep did not predict worry the following day, it was unlikely that daily emotional functioning would mediate this non-significant relationship. Nonetheless, these findings contradict evidence suggesting that sleep disturbance leads to increases in tension, negative affect, and psychological distress (Bonnet & Arand, 1992). Acute sleep deprivation has been shown to elicit increases in negative affect, anxiety, and depression the following day (Bartle et al., 1988; Light et al., 1989; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). However, those studies specifically examined acute episodes of sleep deprivation (which are typically characterized as less than four hours of sleep; Samkoff & Jaques, 1991), whereas participants in the present study did not report episodes of sleep deprivation and thus may not have experienced the immediate effects of such deprivation on the next day's emotions. Another possible reason for our null findings regarding sleep's effects on worry and emotion is that perhaps the negative effects of sleep disturbance are only apparent after prolonged periods of sleep disturbance. As individuals continue to experience sleep disturbance, the accumulation of sleep debt may eventually negatively impact mood, tension, and possibly worry (Friedman, Bigger, & Kornfeld, 1971; Friedman, Kornfeld, & Bigger, 1973; see Samkoff & Jacques, 1991 for review). For example, one study by Bonnet and

Arand (1992) found that objective and subjective sleep disturbance in good sleepers led to increases in anxiety, tension, and dysphoria only after a week of inducing sleep disturbance (Bonnet & Arand, 1992). Additionally, medical interns who experience sleep disturbance over long periods of time demonstrate increases in tension, dysphoria, and mood disturbance throughout their internship (Ford & Wentz, 1984; Sharp, Vaughn, Cosby, Sewell, & Kennaway, 1988). One night or a few nights of disturbed sleep might not directly impact worry the following day, but several weeks of poor or insufficient sleep might create conditions under which individuals experience an increase in worrisome thought and mood disturbance. Thus, it might be difficult to capture this relationship between sleep and worry using a daily process approach. Rather, it is possible that these symptoms need to be measured over a prolonged period of time in order to observe the negative effects of sleep disturbance on worry.

The daily relationship between worry and sleep seems to be stable, as evidenced by the fact that environmental changes (i.e. beginning an intervention to reduce worry) did not moderate their relationship. Additionally, the type of intervention that participants underwent to reduce worry did not affect the daily relationship between worry and sleep. Results indicated that although both worry and sleep disturbance decreased significantly at the end of the intervention (McGowan & Behar, 2013), the daily relationship between both phenomena were not affected. In other words, worry continues to have an impact on sleep even while symptoms of worry and sleep disturbance are improving. Additionally, the mechanism by which interventions for worry reduce sleep disturbance does not seem to be through decreasing worry's ability to impact sleep. Results indicated that the strength of worry predicting sleep was equally as strong during the baseline and treatment weeks. One potential criticism of this investigation is that because individuals with high trait worry underwent an intervention that solely targeted their worry (and

not their sleep disturbance), it is more likely that changes in worry would affect sleep than that changes in sleep would affect worry. However, this is unlikely to be the case given that a similar pattern of results was found during the baseline week prior to participants being randomly assigned to a specific intervention. Further research is necessary to determine the mechanisms of change within interventions targeted at worry and sleep disturbance.

The present study had several limitations. First, it is possible that we failed to find an effect of sleep on worry the next day due to a methodological detail of our investigation. Specifically, greater time periods between the measurement of two phenomena decreases their ability to predict each other. For example, Stone, Neale, and Shiffman (1993) demonstrated that lagged or next-day effects of stressors on mood are rarely found in the literature. This is because the ability of an experience to affect subsequent mood decreases with time. Thus, because there was a longer period of time between sleep_{tonight} and worry_{tomorrow} measurements than there was between worry_{today} and sleep_{tonight} measurements, there may have been a decreased likelihood of detecting the lasting effects of sleep. Future studies should implement similar time periods between measurements of worry and sleep in order to reduce this threat. For example, investigations could include a measurement of worry upon awakening in order to examine the immediate effects of sleep on worry. A second limitation is that we did not include a measurement of sleep quality. According to the cognitive model of insomnia, an individuals' perception of the quality of their sleep might have a stronger influence on their propensity to worry the following day. A third limitation is that participants retrospectively reported both worry before bed and sleep variables at the same time point the following morning, thus introducing the risk of a mood-memory effect (i.e. participants who slept poorly might have inaccurately recalled more pre-sleep worry activity). However, it is unlikely that this limitation

can account for the finding that percentage of daily worry (measured at a different time point) predicted sleep characteristics. Future research utilizing sleep diaries should seek to measure worry and sleep characteristics at different time points. For example, these studies could have participants complete measures of worry prior to bedtime and additional measures of sleep characteristics upon awakening.

A fourth limitation is that results from the present study may not be generalizable to a non-anxious population. Sleep characteristics of anxious individuals have been shown to differ from a normal population (Rosa et al., 1983). Additionally, Chambers and Kim (1993) found that state anxiety was significantly correlated with sleep onset latency among insomniacs but not among normal sleepers. The relationship between worry and sleep might differ for individuals who do not demonstrate high trait worry and/or sleep disturbance. A fifth limitation is that although participants reported moderate insomnia symptoms at the baseline assessment and evidenced moderate sleep disturbance during the baseline week, they were not formally diagnosed with insomnia. It is possible that the conclusions drawn about the relationship between worry and sleep disturbance are only valid for individuals who demonstrate high trait worry. Further research is needed to determine whether individuals with insomnia show a similar daily relationship between worry and sleep disturbance. A sixth limitation is the relatively small sample size employed in this study, which limited our ability to examine more complex models. For example, some of the multilevel analyses (e.g., examining the full worry-sleep models during the baseline week) were unable to be estimated due to the small sample size. A final limitation is that we did not ask participants about the use of medications or sleep aids that could have influenced sleep. The use of drugs that alter sleep (either positively or negatively) may have led to null findings regarding the effects of sleep on worry the next day.

In spite of these limitations, our investigation has several strengths. First, this study is among the first to examine the relationship between worry and sleep using a daily process approach and multilevel modeling techniques. Second, ecological momentary assessment was used to assess worry, emotion, and sleep measures in the moment. This is a superior approach to assessing daily phenomena as it reduces the time elapsed between an experience and an account of that experience. Third, the present study included a baseline week in order to compare results before participants had been randomly assigned to an intervention. The baseline week allowed for the examination of the daily relationship between worry and sleep in a high trait worry population without external influences of intervention. Taken together, these strengths are noteworthy refinements over prior research.

In conclusion, the present study demonstrated that worry consistently has a negative impact on sleep characteristics, but sleep does not influence worry symptoms the following day. This relationship was consistent for individuals with high trait worry and moderate reports of sleep disturbance. Additionally, engaging in an intervention to reduce worry did not change the relationship between worry and sleep. Results from this study underscore the importance of identifying and treating worry in individuals with comorbid worry and sleep difficulties.

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EDUCATION

- 2013-2014 **Pre-doctoral Internship**, Boston Consortium in Clinical Psychology
 VA Boston Healthcare System
 Clinical Fellow in Psychology in the Department of Psychiatry, Harvard Medical School
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- 2011-2013 **Ph.D. Candidate, Clinical Psychology**, University of Illinois at Chicago
 Dissertation: *Examining the Relationship between Worry and Sleep: A Daily Process Approach*.
 Committee: Evelyn Behar, Ph.D. (Chair), Robin Mermelstein, Ph.D., Stewart Shankman, Ph.D.,
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- 2008-2011 **M.A., Clinical Psychology**, University of Illinois at Chicago
 Master's Thesis: *Efficacy of Stimulus Control Training for Worry*.
 Committee: Evelyn Behar, Ph.D. (Chair), Robin Mermelstein, Ph.D., and Stewart
 Shankman, Ph.D.
- 2004-2008 **B.A., Psychology** (with Departmental Honors), Northwestern University
 Honors Thesis: *Patterns of Regional Brain Activity During Emotion-Eliciting Tasks in
 Young Children*. Advisor: Emily Durbin, Ph.D.

AWARDS/DISTINCTIONS

- 2011-2012 LAS PhD Student Travel Award (Total Award: \$500/year)
- 2008-2012 UIC Graduate Student Council/Graduate College Travel Award (Total Award: \$500/year)
- 2008-2012 UIC Department of Psychology Travel Award (Total Award: \$200/year)
- 2008 Underwood Travel Grant (Total Award: \$400)
- 2007 Honors Research Thesis, Northwestern University
- 2007 Weinberg College of Arts and Sciences Research Grant, Northwestern University
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CLINICAL EXPERIENCE

- 2013-present **General Mental Health Intern (Boston Consortium in Clinical Psychology)**
 Boston VA Healthcare System, Boston, MA.
General Mental Health Clinic
- Performed psychological services including intake and diagnostic interviews.
 - Provided individual psychotherapy for veterans with severe and complicated presentations of psychopathology including anxiety disorders, depressive disorders, substance abuse, bipolar disorder, and personality disorders.
 - Co-lead cognitive-behavioral manualized depression management group.
- Supervisors: Gabrielle Liverant, Ph.D., Stephen Lancey, Ph.D., and Justin Hill, Ph.D.
- Mood and Anxiety Disorders Clinic*
- Performed psychological services including intake and diagnostic interviews.
 - Provided individual psychotherapy for veterans with severe and complicated presentations anxiety and depression.
 - Co-lead Acceptance and Commitment Therapy Group for depression and anxiety.
- Supervisors: Justin Hill, Ph.D., and Stephen Lancey, Ph.D.
- Primary Care Behavioral Health Clinic*
- Performed psychological services including intake interviews, warm handoffs, and short-term individual psychotherapy for veterans with various presentations of psychopathology within a primary care setting.
- Supervisor: Justin Hill, Ph.D.

National Center for PTSD-Behavioral Sciences Division, PTSD Clinic (Adjective Training Experience)

- Performed individual psychotherapy utilizing Cognitive Processing Therapy for veterans with PTSD.
- Participated in weekly Case Conceptualization meetings.

Supervisors: Yael Nillni, Ph.D., Denise Sloan, Ph.D., and Wendy Bassett, LCSW.

National Center for PTSD-Women's Stress Disorders Treatment Team (4-month rotation)

- Will provide empirically supported treatments for women with PTSD, trauma-related and comorbid disorders.
- Will provide group therapy for veterans.

Supervisors: TBD.

2012-2013

Stress and Anxiety Disorders Clinic (UIC Department of Psychiatry)

University of Illinois-Chicago Medical Center, Chicago, IL

- Performed psychological services including intake interviews, individual psychotherapy for patients with more severe forms of anxiety disorders in an outpatient medical setting.

Supervisor: Cheryl Carmin, Ph.D.

2011-2012

Inpatient Cognitive Behavioral Therapy Group (UIC Department of Psychiatry)

University of Illinois-Chicago Medical Center, Chicago, IL

- Performed group therapy for individuals on the inpatient unit.
- Groups introduced the CBT model and discussed one 'strategy of the day' from various CBT interventions for patients with severe psychopathology.

Supervisor: Stewart Shankman, Ph.D.

2011-2012

Pediatric Mood Disorders Clinic (Institute for Juvenile Research)

University of Illinois Medical Center, Chicago, IL.

- Provided individual and family treatment and conducted intake interviews with patients presenting with severe mood disorders.
- Provided manualized individual and family therapy for children diagnosed with pediatric bipolar disorder.
- Co-lead group therapy for children diagnosed with pediatric bipolar disorder.
- Co-lead group therapy for adolescents diagnosed with major depression.

Supervisors: Amy West, Ph.D. and Sally Weinstein, Ph.D.

2008-2012

Office of Applied Psychological Services (UIC Department of Psychology)

University of Illinois at Chicago, Chicago, IL.

- Provided individual treatment and conducted intake interviews with patients presenting with a variety of anxiety and mood disorders.
- Conducted psychological assessments for clients presenting with learning disabilities, ADHD, and other psychological disorders.
- Co-lead manualized therapy group for patients diagnosed with social anxiety disorder.

Supervisors: Nancy Dasso, Ph.D., Audrey Ruderman, Ph.D., Evelyn Behar, Ph.D., and Elise Massie, Ph.D.

Summers 2003-2005

Semel Institute for Neuroscience and Human Behavior (formally Neuropsychiatric Institute)

University of California, Los Angeles, Los Angeles, CA

- Taught autistic children utilizing an intensive therapeutic model in an outpatient hospital school setting.
- Coded observational data.

Supervisor: Stephanny Freeman, Ph.D.

2000-2004

Teen-Line National Crisis/Suicide Prevention Hotline

Cedar-Sinai Medical Center, Los Angeles, CA

- Completed intensive 13-week training process; weekly commitment for a

- national telephone/online crisis intervention hotline for adolescents.
- Participated in community outreach and in the development of videos on depression and suicide prevention.

Supervisor: Elaine Leader, Ph.D.

SPECIALIZED CLINICAL TRAINING/EXPERIENCE

- 2013-2014 **Cognitive Processing Therapy Case Conceptualization.** Weekly supervision for certification in CPT for PTSD. Conducted by Wendy Bassett, LCSW, VA Connecticut Healthcare System
- 2013 **Cognitive Processing Therapy Workshop.** Two-day training by Patricia Resick, Ph.D., Boston VA Healthcare System
- 2012 **Improving Care for Veterans with PTSD.** Presented by Josef I. Ruzek, Ph.D., National Center for PTSD, VA Palo Alto Healthcare System
- 2012 **Adolescent Trauma and Substance Abuse Training.** Presented by Liza Suárez, Ph.D., Institute for Juvenile Research, University of Illinois Medical Center
- 2012 **Cognitive Behavior Group Therapy for Psychiatric Inpatients** (Protocol therapy training)
- 2011 **Group Therapy for Pediatric Bipolar Disorder** (Protocol therapy training)
- 2011 **Individual/Family Therapy for Pediatric Bipolar Disorder** (RAINBOW; Protocol therapy training)
- 2010 **Group Therapy for Social Anxiety Disorder** (Protocol therapy training)
- 2010 **Beck Institute Cognitive Therapy Workshop.** Presented by Aaron Beck, Ph.D. and Judith Beck, Ph.D., Beck Institute for Cognitive Behavioral Therapy
- 2010 **Performing Clinical Services with Hearing Impaired Clients.** Presented by the UIC Disability Center
- 2008 **Wechsler Adult Intelligence Scale - IV (WAIS-IV) Workshop**

INVITED ADDRESSES

The Assessment and Treatment of Insomnia. University of Illinois, Chicago graduate-level Interviewing class, Chicago, IL (November 2009, 2010, 2011, 2012).

Efficacy of Stimulus Control for Worry. Department of Psychology, University of Illinois, Chicago (February 2011).

The Etiology, Maintenance, and Treatment of Insomnia. Rush University Medical Center monthly meeting of the Cognitive Therapy Conference, Chicago, IL (February 2009).

PROFESSIONAL CONFERENCE SYMPOSIA

McGowan, S.K., & Behar, E. (2012). *A preliminary investigation of stimulus control for worry: Effects on anxiety and insomnia.* Oral paper presented at 46th Annual Association of Behavioral and Cognitive Therapy (ABCT), National Harbor, MD.

Shankman, S. A., Nelson, B. D., Sarapas, C., Robison-Andrews, E. J., Campbell, M. L., Altman, S. E., **McGowan, S. K.**, Katz, A. C., & Gorka, S. M. (September, 2012). *A psychophysiological investigation of reward and threat sensitivity in individuals with depression and/or panic disorder.* Oral paper presented at 26th annual meeting of the Society for Research in Psychopathology, Ann Arbor, MI.

McGowan, S.K., Durbin, C.E., Hespos, S. (2008). *Patterns of regional brain activity during emotion eliciting tasks in young children.* Oral paper presented at 17th Annual UCLA Psychology Undergraduate Research Conference (PURC), Los Angeles, CA.

PROFESSIONAL CONFERENCE POSTER PRESENTATIONS

Black, S.K., **McGowan, S.K.**, Liverant, G.I., Kamholtz, B.W., & Sloan, D.M. (March, 2014). Fear of depressed mood in clinical depression: Association with symptom severity and use of emotion regulation strategies. Poster submitted to the annual meeting of the Anxiety and Depression Association of America, Chicago IL.

Nelson, B. D., Robison-Andrew, E. J., Altman, S. E., Campbell, M. L., Sarapas, C., Katz, A. C., Gorka, S. M., **McGowan, S. K.**, & Shankman, S. A. (September, 2012). Comparing reward sensitivity vs. DSM-defined

major depressive disorder on how they predict family history of psychopathology. Poster presented at the 26th annual meeting of the Society for Research in Psychopathology, Ann Arbor, MI.

Nelson, B. D., Robison-Andrew, E. J., Altman, S. E., Sarapas, C., Campbell, M. L., Katz, A. C., Gorka, S. M., **McGowan, S. K.**, & Shankman, S. A. (September, 2012). Does heightened sensitivity to predictable vs. unpredictable threat index risk for panic disorder? A startle electromyography investigation. Poster presented at 52nd annual meeting of the Society for Psychophysiological Research, New Orleans, LA.

Gorka, S.M., Nelson, B.D., Sarapas, C., Campbell, M.L., Katz, A., **McGowan, S.K.** & Shankman, S.A. (2012, September). Sensitivity to unpredictable threat in those with panic disorder and/or alcohol dependence. Poster presented at 52nd annual meeting of the Society for Psychophysiological Research, New Orleans, LA.

McGowan, S.K. & Behar, E. (2011) *Stimulus Control for Worry: Effects on Sleep Quality*. Poster presented at the 45th Annual Association for Behavioral and Cognitive Therapies, Toronto, Canada.

McGowan, S.K., Gorka, S., Nelson, B.D., & Shankman, S.A. (2011) *Anxiety and Autonomic Flexibility in Conditions of Unpredictable Threat*. Poster presented at the 25th annual meeting of the Society for Research in Psychopathology, Boston, MA.

Sarapas, C., Bishop, J. R., Nelson, B. D., Campbell, M. L., Robison-Andrew, E. J., Altman, S. E., Katz, A. C., Gorka, S. M., **McGowan, S. K.**, & Shankman, S. A. (September, 2011). *Indirect genetic effects on internalizing symptoms through frontal EEG asymmetry*. Poster session presented at 25th annual meeting of the Society for Research in Psychopathology, Boston, MA.

Campbell, M. L., Nelson, B. D., **McGowan, S. K.**, Robison-Andrew, E. J., Sarapas, C., Altman, S. E., Gorka, S., Katz, A. C., Shankman, S. A. (September 2011). *Startle Habituation in Individuals With Depression and/or Panic Disorder*. Poster session presented at 25th annual meeting of the Society for Research in Psychopathology, Boston, MA.

Robison-Andrew, E.J., Shankman, S.A., **McGowan, S.K.** (May, 2011). Impact of cognitive therapy analogue on startle reactivity and subjective response during anticipation of aversive stimuli. Poster presented at the Association for Psychological Science Annual Convention, Washington, D.C.

Nadelson, J., **McGowan, S.K.**, Shankman, S.A., Shroff, A.R., & Vidovich, M.I. (May 2011). Depression Screening in Patients with Cardiovascular Disease in a Veterans Administration Hospital. Poster presented at Quality of Care and Outcomes Research in Cardiovascular Disease and Stroke, Washington, D.C.

McGowan, S.K., Stanfill, S., & Behar, E. (November, 2010). Efficacy of a Stimulus Control Treatment for Worry. Poster presented at the 44th Annual Association for Behavioral and Cognitive Therapies, San Francisco, CA.

Campbell, M.L., Shankman, S.A., Nelson, B. D., Robison-Andrew, E. J., Altman, S.E., Sarapas, C., **McGowan, S.K.** (October, 2010). Personality correlates of startle habituation: An examination in three separate samples. Poster presented at the 24th Annual Society for Research in Psychopathology, Seattle, WA.

McGowan, S.K., Goldwin, M., Bjorkquist, O., Bohacz, B., Behar, E., McLaughlin, K., & Borkovec, T.D. (November, 2009). The effects of positive, negative, and neutral perseverative thinking on abstractness and concreteness of thought content. Poster presented at the 43rd Annual Association for Behavioral and Cognitive Therapies, New York, NY.

Goldwin, M., **McGowan, S.K.**, Sibrava, N.J., Behar, E., & Borkovec, T.D. (November, 2009). Emotional experiences during recall of negative events. Poster presented at the 43rd Annual Association for Behavioral and Cognitive Therapies, New York, NY.

McGowan, S.K., Durbin, C.E., Hespos, S. (2008). Patterns of Regional Brain Activity During Emotion Eliciting Tasks in Young Children. Poster presented at Northwestern University Undergraduate Research Symposium, Chicago, IL.

McGowan, S.K., & Rapp, D.N. (2007). Spoiler alert: Increasing and decreasing psychological experiences of suspense. Poster presented to the annual meeting of the Midwestern Psychological Association, Chicago, IL.

AD HOC REVIEWER FOR SCIENTIFIC JOURNALS

*Journal of Abnormal Psychology**

*Behavior Therapy***

* conducted under the supervision of Stewart Shankman, Ph.D.

**conducted under the supervision of Evelyn Behar, Ph.D.

RESEARCH EXPERIENCE

- 2013-present **Intern Research Assistant**, Liverant Research Laboratory
Boston VA Healthcare Systems
- Aided in preparing manuscripts for publication.
 - Prepared poster presentations for conferences.
- Supervisors: Gabrielle Liverant, Ph.D., Barbara Kamholtz, Ph.D.
- 2008-2013 **Graduate Research Assistant**, Behar Research Laboratory
Department of Psychology, University of Illinois at Chicago
- Designed research protocols.
 - Programmed experience sampling software using PDAs.
 - Trained research assistants on coding system.
 - Ran research participants through experimental protocols.
- Supervisor: Evelyn Behar, Ph.D.
- 2010-2012 **Graduate Research Assistant**, Affective Science and Physiology Laboratory
Department of Psychology, University of Illinois at Chicago
- Conducted Structured Clinical Interviews for the DSM-IV (SCID).
 - Processed EMG startle data (NeuroScan) and HRV data (CardioEdit).
- Supervisor: Stewart Shankman, Ph.D.
- 2010-2011 **Graduate Research Assistant**, Institute of Juvenile Research, Pediatric Mood Disorders Clinic,
Department of Psychiatry, University of Illinois at Chicago
- Administered psychological tests for study examining a child and family-focused cognitive behavioral therapy for the treatment of pediatric bipolar disorder.
- Supervisor: Amy West, Ph.D.
- 2007-2008 **Honors Research Thesis**, Department of Psychology, Northwestern University
- Primary experimenter for a study that examined brain correlates of emotion in children.
 - Developed of experimental design and procedures.
 - Managed recruitment system.
 - Experience using Hitachi optical imagine machine (NIRS).
- Supervisors: C. Emily Durbin, Ph.D. and Susan Hespos, Ph.D.
- 2007-2008 **Lab Coordinator/Research Assistant**, Department of Psychology, Northwestern University
- Primary experimenter; office management (recruitment and obtaining consent).
 - Administered WPPSI and PPVT.
 - Coded and maintained databases.
- Supervisor: C. Emily Durbin, Ph.D.
- 2007 **Independent Directed Research Project**, Department of Psychology, Northwestern University
- Self initiated independent research study examining the effects of prior warning on anomalous suspense while reading fictional stories.
 - Design and implementation experimental material
 - Proficiency in Super Lab software
- Supervisor: David Rapp, Ph.D.

- 2006 **Research Assistant**, Department of Psychology, Northwestern University
- Measured the effects of suppression on delayed pain responses.
 - Ran participants through experimental protocol (proficiency with skin conductance and heart monitoring machine).
- Supervisor: Richard Zinbarg, Ph.D.
- 2005 **Research Assistant**, Department of Psychology, Northwestern University
- Examined heterosexual and homosexual orientation based on childhood behavioral data obtained from family videos.
 - Ran participants through experimental protocol, assisted in recruitment, compiling/maintaining databases, preparing video for coding and advertising research.
- Supervisor: Michael Bailey, Ph.D.

PUBLICATIONS

1. Campbell, M.L., Gorka, S.M., **McGowan, S.K.**, Nelson, B.D., Sarapas, C., Katz, A.C., ... Shankman, S.A. (in press). Does anxiety sensitivity correlate with reductions in startle responding? An examination in three separate samples. *Cognition and Emotion*.
2. Nelson, B. D., **McGowan, S. K.**, Sarapas, C., Robison-Andrew, E. J., Altman, S. E., Campbell, M. L., ... Shankman, S. A. (2013). Are individual differences in threat and reward sensitivity associated with family history of psychopathology? *Journal of Abnormal Psychology*, 122, 662-671.
3. Gorka, S.M., **McGowan, S.K.**, Campbell, M.L., Nelson, B.D., Sarapas, C., Bishop J.R., & Shankman, S.A. (2013). Association between respiratory sinus arrhythmia and reductions in startle responding in three independent samples. *Biological Psychology*, 93, 334-341.
4. Shankman, S. A., Nelson, B. D., Robison-Andrews, E. J., Sarapas, C., Campbell, M. L., Altman, S. E., **McGowan, S. K.**, Katz, A., & Gorka, S. M. (2013). A psychophysiological investigation of threat and reward sensitivity in individuals with panic disorder and/or depression. *Journal of Abnormal Psychology*, 122, 322-338.
5. **McGowan, S.K.** & Behar, E. (2013). A preliminary investigation of stimulus control training for worry: Effects on anxiety and insomnia. *Behavior Modification*, 37, 90-112.
6. Shankman, S.A., Nadelson, J., **McGowan, S.K.**, & Vidovich, M.I. (2012). Validity of depression screening procedures for veterans with coronary artery disease. *Vascular Health and Risk Management*, 8, 233-238.
7. Behar, E., **McGowan, S.K.**, McLaughlin, K.A., Borkovec, T.D., Goldwin, M., & Bjorkquist, O. (2011). Concreteness of positive, negative, and neutral repetitive thinking about the future. *Behavior Therapy*, 43, 300-312.

MANUSCRIPTS IN PREPARATION

1. **McGowan, S.K.**, Behar, E., & Luhmann, M. (in prep). Examining the relationship between worry and sleep: A daily process approach.
2. Black, S.K., Liverant, G.I., Sloan, D.M., Kamholtz, B.W., & **McGowan, S.K.** (in prep). Apprehension of depressed mood: Relationships with psychiatric symptoms and emotion regulation in clinical depression.
3. **McGowan, S.K.** & Behar, E. (in prep). Concreteness of idiographic periods of worry and anticipatory processing.

TEACHING EXPERIENCE

- 2010-2012 **Teaching Assistant-Psychological Assessment**
- Attended lecture and graded papers
- 2010-2011 **Teaching Assistant-Psychological Interviewing**
- Supervised undergraduates in conducting psychological interviews; provided supervision
- 2010 **Teaching Assistant-Abnormal Psychology**
- Attended lecture and graded papers
- 2010 **Teaching Assistant-Field Work in Applied Psychology**
- Supervised undergraduates in completing field research and writing senior research theses
- 2010 **Teaching Assistant-Statistical Methods in Behavioral Sciences**
- Led two weekly discussion sections and graded papers

- 2009 **Teaching Assistant-Theories of Personality**
- Attended lecture and graded papers
- 2008-2009 **Teaching Assistant-Introduction to Psychology**
- Led four weekly discussion sections and graded papers

MEMBERSHIP IN PROFESSIONAL ASSOCIATIONS

2008-2012 **Association of Behavioral and Cognitive Therapies**, Student Member

PROFESSIONAL REFERENCES

Evelyn Behar, Ph.D. (Ph.D. mentor)

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 Department of Psychology (M/C 285)
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 University of Illinois at Chicago
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Gabrielle Liverant, Ph.D.

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 VA Boston Healthcare System - Brockton Campus
Assistant Professor, Department of Psychiatry
 Boston University School of Medicine
 VA Boston Healthcare System – Brockton Campus (116A)
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 Brockton, MA 02301
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 Email: Gabrielle.Liverant@va.gov