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# The effects of an evening structured problem-solving procedure in undergraduate college students with insomnia

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THE EFFECTS OF AN EVENING STRUCTURED PROBLEM-SOLVING  
PROCEDURE IN UNDERGRADUATE COLLEGE STUDENTS WITH INSOMNIA

A Dissertation

Submitted to the Graduate Faculty of the  
Louisiana State University and  
Agricultural and Mechanical College  
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by

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## ABSTRACT

Worry is often reported as interfering with sleep onset and sleep maintenance, and pre-sleep cognitive arousal can persist after successful behavioral treatment of insomnia. The present investigation will examine the effects of a “constructive worry” procedure in an undergraduate population with impaired sleep. Thirty-three undergraduate students who reported three or more nights per week in the last month of sleep onset and/or sleep maintenance problems, either recorded worries and possible solutions (experimental CW group) or recorded worries and completed worry questionnaires (control Worry group) for five nights. As hypothesized, the CW group had decreased pre-sleep cognitive and overall arousal relative to the Worry group and relative to baseline scores. Although the CW group reported decreased cognitive arousal, there were no significant effects on somatic arousal, anxiety, Sleep Diary or actigraphy sleep variables. Suggestions for future investigations, and potential implications for the treatment of insomnia are discussed.

## INTRODUCTION

Subclinical levels of worry are common in everyday life and can contribute to cognitive arousal before bed, and subsequent sleep difficulties. In those with insomnia, there is a tendency to ruminate before bed, which often interferes with sleep onset and sleep quality/continuity (Gross & Borkovec, 1982). There is also evidence that stress-induced ruminative worry decreases delta wave intensity in the first sleep cycle of normal subjects (Hall et al., 1996), which would tend to make sleep lighter and more readily disrupted. Moreover, the pre-sleep cognitive activity of those with insomnia is more focused on worries, environmental stimuli like noise, and the consequences of not sleeping. Good sleepers, in contrast, are characterized by focusing on “nothing in particular” (Harvey, 2000). Results from correlational and regression analyses suggest worry/problem-solving and thinking about sleep and the consequences of sleep loss, are the strongest cognitive predictors of delayed sleep onset latency (Wicklow & Espie, 2000).

This paper will discuss the nature of sleep quality, and the role of arousal in subjective and objective sleep quality. In addition, the mixed evidence for the roles of cognitive and somatic arousal in interfering with sleep quality, and the relation between worry/problem-solving and sleep, will be reviewed. Also, this paper will report and discuss the findings from the present investigation where those with insomnia were taught to problem solve in the early evening or simply monitor their worries. The purpose of the study was to reduce the subjective complaint of pre-sleep cognitive arousal via a problem-solving procedure. There is a largely subjective component to an insomnia complaint, as some insomnia complaints are not validated by objective means (e.g., polysomnography - PSG), and some people with insomnia continue to complain of pre-sleep cognitive arousal after treatment is successful (e.g., increased sleep

efficiency, decreased sleep onset latency, or decreased time awake after sleep onset); thus, there is good reason to try to treat this complaint for those who suffer from insomnia. If the problem-solving procedure is effective in reducing pre-sleep cognitive arousal, future investigations could test whether combining a problem-solving procedure with current behavioral treatment for insomnia adds to successful outcome.

### Sleep Processes

To understand a complex process like sleep quality, one must begin with the very basics of what makes sleep restorative. The mechanisms behind the restorative properties of sleep remain unknown, although sleep depth, determined by increased proportions of delta wave sleep (slow wave sleep) relative to stages 1NREM and 2NREM, and sleep continuity, as determined by a low occurrence of awakenings, arousals, and sleep stage changes have all been implicated. Stages 3NREM and 4NREM constitute slow wave or high voltage delta sleep, which is suspected to play a significant role in whether or not a sleep episode is sufficiently “deep”. Though there is no compelling direct evidence that slow wave sleep is the most restorative of the sleep stages, it is a widely accepted supposition due to consistent indirect evidence such as its proximity to sleep onset, its immediate rebound and reliably demonstrated priority after sleep loss, its high sensory thresholds (i.e. depth) and its close association with anabolic growth hormone secretion (Oswald, 1970; 1980; Borbély, 1990). The increase in, and priority of, delta EEG after sleep loss is well-established, as is its depth and its priority in normal sleep (Berger & Oswald, 1962; Williams et al., 1964; Tobler & Borbely, 1990; Franken et al., 1991b); also, the increased production of human growth hormone in slow wave sleep is anabolic and contributes to tissue growth and repair (Takahashi et al., 1968; Honda et al., 1969; Adam, 1980).

A homeostatic mechanism determines slow wave sleep quantity according to the prior amounts of sleep and waking, increasing slow wave sleep propensity to the extent that prior wake time increases and prior sleep time decreases, and decreasing sleep propensity in response to excess sleep (Borbély, 1994). As an individual becomes sleep deprived throughout the day, the drive for slow wave sleep during the next sleep period is intensified, and as such, the production and priority of slow wave sleep during the night is increased.

Sleep quality is a complex construct, which is determined by number of arousals, proportions of sleep stage changes, and particular amounts of sleep stages (Bonnet, 1986a; 1986b). Impairment on daytime performance measures occurs whether subjects are awakened to full consciousness, physically move during sleep, or experience an EEG shift (Bonnet, 1987). However, subjective perception of sleep loss on mood measures requires full awakenings to a conscious state, for the sleep episode to be perceived as nonrestorative. What makes an episode restorative or not, is not quantity, rather quality, which is defined by continuity (e.g., sleep stage changes and sufficiently low amounts of arousals) and depth (e.g., sufficiently low amounts of stage 1NREM sleep, and sufficiently high amounts of SWS). Disruptions in continuity or depth lead to a rebound of increased SWS on recovery nights, thus suggesting a primary role for SWS in the restorative properties of sleep (Berger & Oswald, 1962; Williams et al., 1964; Tobler & Borbely, 1990; Franken et al., 1991b).

In addition to basic homeostatic processes, sleep researchers are concerned with behaviors that interfere with restorative nocturnal sleep. Sleep hygiene refers to those behaviors, which promote or interfere with normally restorative sleep. Sleep hygiene rules are based upon factors that can delay sleep onset or interfere with sleep maintenance, such as eating a large meal, drinking large amounts of liquid, exercising, smoking, drinking alcohol or consuming

products containing caffeine close to the sleep period (Zarcone, 1994); these are typically physiologically and cognitively arousing stimuli. Large meals before bed can cause indigestion and discomfort that can interfere with sleep. Consumption of liquids before bed, particularly large quantities, can necessitate getting out of bed to urinate, which interferes with sleep maintenance and the continuity of sleep. Engaging in vigorous exercise before bed can be physiologically arousing and may interfere with sleep. Smoking before bed can interfere with sleep onset and maintenance because it can lead to arousal of the cholinergic activating system when blood concentration levels are high (Soldatos et al., 1980). Nicotine is physiologically activating and thus inhibitory of sleep processes (Landolt et al., 1994).

If someone has been drinking alcohol prior to bedtime, the initial state may be one of sedation; however, once the alcohol is metabolized, a compensatory rebound of stage 1NREM sleep and inhibition of slow wave sleep can cause unduly light, disrupted and unrefreshing sleep thereafter. Awakenings are sometimes associated with intense dreaming (REM sleep rebound subsequent to alcohol-induced REM sleep suppression), sweating, and headaches (sympathetic rebound due to alcohol-related parasympathetic activation). This sympathetic arousal usually recurs in the second half of the sleep period, and can persist for 2-3 hours after the blood alcohol levels return to zero (Zarcone, 1994). Finally, caffeine produces an increase in awakenings and a decrease in total sleep time due to its interference with the sleep-facilitating inhibitory substance adenosine (Landolt et al., 1994).

Sleep hygiene training alone is limited in its effectiveness, since poor sleep hygiene is rarely sufficient to produce Primary Insomnia (Reynolds, Kupfer, Buysse, Coble, & Yeager, 1991), and those with insomnia and good sleepers often have the same poor sleep habits (Haynes et al., 1982). Though poor sleep hygiene can be the primary cause of the insomnia, more often it

complicates existing insomnia and treatment, and is generally used in combination with other strategies (Edinger, Hoelscher, Marsh, Lipper, & Ionescu-Pioggia, 1992; Morin, Kowatch, Barry, & Walton, 1993).

Other sleep hygiene rules are based upon circadian and homeostatic principles. The tendency for sleep is bi-phasic, with a peak at night and another mild peak around mid-afternoon (Zarcone, 1994). Taking a sufficiently long nap (e.g., more than 90 minutes in duration) during the mid-afternoon sleep-propensity period interferes with the subsequent night's sleep (Feinberg et al., 1985). This is because sufficient sleep-deprivation (approximately 16-18 hours) is needed to produce adequate amounts of sleep, especially slow wave sleep, in the next sleep period. Further, the timing of sleep is essential for restorativeness, independent of the duration of sleep (Taub & Berger, 1976). For example, sleep during periods of daylight and during rising core body temperature is lighter, and thus, less continuous and restorative. There exists a delicate balance between core body temperature and sleep (circadian mechanism) and prior amounts of wakefulness (homeostatic mechanism), that determines restoration. It is for these reasons that two sleep hygiene rules have been established as essential to restorative sleep: a regular set bedtime and wake-time, and avoiding naps, particularly late in the wake period. These are part of the basic rules in stimulus control (Bootzin, Epstein, & Wood, 1991) treatment of insomnia, which are: 1) go to bed only when sleepy; 2) use the bedroom for sleep and sex only; 3) if you are unable to fall asleep within 20 minutes, leave the bedroom and do not return until sleepy; 4) keep a set wake-time regardless of the amount of sleep obtained the night before; and 5) no daytime naps. Stimulus Control assumes that cues that are normally associated with sleeping (e.g., bedtime, bed, or bedroom) have become associated with nonsleep-conducive activities (e.g., problem-solving, reading, doing work, eating etc.); thus the rules that the bedroom should

be used only for sleep and sex. Stimulus control trains the patient to associate the bedroom with sleep and sex only. Following the rules increases the likelihood that only sleep will occur during the time allotted for sleeping. For example, refraining from naps and keeping a set wake-time will produce a greater homeostatic drive for deep, slow wave sleep on the subsequent night, thereby increasing the probability of a consolidated and continuous nocturnal sleep episode. There are 12 studies evaluating stimulus control as a single treatment, and all twelve demonstrated superiority to other single treatment modalities (progressive relaxation, imagery training, and paradoxical intention) and to wait-list controls (see Morin et al., 1999).

### The Arousal Construct

Even when there is sufficient homeostatic drive for slow wave sleep, arousal is an important factor that can delay the onset of sleep or interfere with its maintenance. Arousal is best understood as a state of excitation or activation, and anxiety can be considered physiological, behavioral and/or cognitive arousal in response to threatening stimuli. Anxiety appears to serve a functional purpose, namely to alert and mobilize the organism away from threat via activation of the sympathetic nervous system (Cannon, 1928). Sleep requires a passive disengagement from the environment (e.g., light, sound, extreme temperature, internal discomfort), and threatening stimuli (e.g., worry content) all of which are activating (e.g., SNS, endocrine and CNS arousal), and thus inhibitory of sleep. Throughout this paper, cognitive and somatic arousal are differentiated, but this does not imply that one can have cognitive arousal without bodily arousal, or somatic arousal without some possible cognitive arousal too. Cognitive and somatic arousal in this paper refers to the perception or subjective self-report of cognitive or somatic arousal in those with insomnia.

Nicassio, Mendlowitz, Fussell, & Petras (1985) suggest there is good reason to assume that what causes arousal is highly individualized, and that arousal is divided into sub-components, namely cognitive, emotional and physiological (somatic) arousal (Borkovec, 1976; Davidson & Schwartz, 1976; Schwartz, 1975). Somatic and cognitive arousal/anxiety have been psychometrically differentiated in scales like the Cognitive-Somatic Anxiety Questionnaire (CSAQ, Schwartz, Davidson, & Goleman, 1978) or the Pre-Sleep Arousal Scale (PSAS, Nicassio, Mendlowitz, Fussell, & Petras, 1985). Factor analytic studies have supported the difference between cognitive and somatic arousal, as distinct although related, dimensions of pre-sleep arousal (Nicassio, Mendlowitz, Fussell, & Petras, 1985). Additionally, cognitive arousal appears to have sub-components as well, as arousal appears to relate to either generalized worry, and/or concern about loss of sleep (Wicklow & Espie, 2000). There are also the related issues of physiological activation caused by specific problem-related stimuli and frustration/anger due to the inability to fall asleep or sleep well.

In a survey study, Lichstein and Rosenthal (1979) reported that those with insomnia perceived cognitive arousal as more prevalent than somatic arousal in the maintenance of their sleep difficulty. Their pre-sleep cognitions were reported to be more negative and worry-related than those of good sleepers (Borkovec, Lane, & Vanoot, 1981). Despite the reported importance of pre-sleep cognitive arousal in insomnia, most interventions have been behavioral, and have not specifically targeted problem cognitions (Levey, Aldaz, Watts & Coyne, 1991). Some attribute the effectiveness of behavioral strategies to cognitive factors, for example, the focusing of attention on muscle groups in Progressive Muscle Relaxation, which diverts attention from sleep-interfering cognitions (Bohlin, 1973). To date, specific cognitive strategies like cognitive restructuring or paradoxical intention have met with mixed results, perhaps because these

interventions have focused on changing sleep-related attitudes when other cognitive approaches might have been more efficacious (Levey, Aldaz, Watts, & Coyle, 1991). One of the most common pre-sleep cognitions reported to interfere with sleep are worry-related cognitions (Wicklow & Espie, 2000), and there are no worry-specific treatments in insomnia. Espie and Lindsay (1987) have suggested that those with insomnia should schedule a time in the evening to address, and then set aside concerns which may interfere with subsequent nocturnal sleep. This has implications for treatment, as tailored interventions could specifically target individual physiological and cognitive arousal profiles (e.g., progressive muscle relaxation for those who are primarily experiencing physiological tension, and cognitive interventions to address the experience of excessive mentation).

### Arousal and Sleep

Certainly anxiety and pre-sleep arousal are common in those with insomnia, as anxiety prevalence estimates for those with insomnia range from 25 to 42 percent (Soldatos, 1994). Minnesota Multiphasic Personality Inventory (MMPI) profiles of those with insomnia are characterized by multiple scale elevations, but especially those corresponding to anxiety and worry (Coursey, Buchsbaum, & Frankel, 1975), which may be either a cause or consequence of insomnia. Prevailing etiological theories of insomnia focus on physiological (Bonnet & Arand, 1995), emotional or cognitive hyperarousal (Morin, 1993) as predisposing, precipitating, or perpetuating factors (Spielman, Caruso, & Glovinsky, 1987).

The effect of arousal on sleep has most often been studied in the context of sleep onset insomnia. In multiple regression analyses, negative emotion, stress, and attention variables have accounted for an average of 41% of the variance in sleep variables, thus supporting the link between emotional arousal and sleep (Waters, Adams, Binks, & Varnado, 1993). Early theories

focused solely on physiological arousal (Bootzin & Nicassio, 1978), and still today, most treatments of insomnia focus on reducing physiological arousal (see Morin et al., 1999). The physiological hyperarousal hypothesis (Monroe, 1967) originates from physiological differences between good and poor sleepers, namely greater autonomic activity pre-sleep and during sleep, and higher mean heart and pulse rates, rectal body temperature, and phasic vasoconstriction pre-sleep in poor sleepers relative to good sleepers. However, attempts to empirically separate good sleepers from poor sleepers on the basis of physiological measures, have not convincingly supported heightened physiological arousal as a primary cause of insomnia (Haynes, Adams & Franzen, 1981); subsequent investigations have not found reliable physiological differences (Frankel, Buchbinder, Coursey, & Syder, 1973; Johns, Gay, Marston, & Bruce, 1971). Some have not found a relationship between physiological arousal and sleep-onset latency (Brownman & Tepas, 1976; Freedman & Papsdorf, 1976; Borkovec, Grayson, O'Brien, & Weerts, 1979). In addition, physiological measures of depth of relaxation do not correlate with objective sleep improvement (Hauri, 1981; Nicassio, Boylan, & McCabe, 1982).

Cognitive hyperarousal has been understated until recent years, despite studies which found that most of those with insomnia report cognitive arousal as a far more salient problem than somatic arousal (Lichstein, Fanning & Cernosek, 1979; Lichstein & Rosenthal, 1980). Cognitive hyperarousal theory suggests that those with insomnia have more worrisome mental content that activates central nervous system activity (cognitive arousal), as well as peripheral sympathetic nervous system and endocrine activity (physiological arousal). Cognitive activity during sleep is often experienced much like cognitive activity during wakefulness (Borkovec, 1982). Gross and Borvocec (1982) found that even in good sleepers, inducing cognitive arousal

by telling subjects they would have to give a speech when they woke, significantly increased sleep onset latency.

Although current studies emphasize the importance of cognitive arousal and sleep, others have failed to find such a link. There are several possible relationships for arousal and sleep. One suggestion is that cognitive arousal is an effect, rather than a cause, of insomnia (Freedman & Sattler, 1982; Morin, 1993). For example, if one experiences difficulty initiating sleep due to a learned association between bedtime and poor sleep quality, the quiet dark bedroom with no stimulation or distraction, may be more conducive to thinking. Once this occurs, active mental activity may then exacerbate the existing insomnia. This may occur in some with insomnia, but studies have found that the excessive mentation occurs well before getting into bed and continues once sleep is attempted (Gross & Borkovec, 1982; Hall et al., 1996; Wicklow & Espie, 2000).

In some studies, pre-sleep cognitive arousal has been linked to sleep variables like sleep onset latency (Nicassio, Mendlowitz, Fussell & Petras, 1985; Sanavio, 1988; Wicklow & Espie, 2000), while other studies have not found a link (Haynes, Adams, & Franzen, 1981). In the validation studies of the Pre-Sleep Arousal Scale (PSAS; Nicassio, Mendlowitz, Fussell & Petras, 1985), both somatic and cognitive subscales correlated highly with sleep onset latency and total sleep time on subjective sleep diaries. Pre-sleep cognitions were also associated with subjective sleep diary data in one study, but were not associated with objective (PSG) sleep variables in another study (van Egeren, Haynes, Franzen, & Hamilton, 1983). Other studies have found similar associations with both objective and subjective variables (Kuisk, Bertelson, & Walsh, 1989). Another study suggests that there is an association between sleep and pre-sleep arousal in those with Psychophysiological Insomnia but not those who are suffering from insomnia and have simultaneous depression and/or anxiety symptoms (Broman & Hetta, 1994).

In general, this literature has little consistency, and studies use different measures that limit comparisons across investigations, and they frequently have low sample size.

### Worry

Worry is commonly reported as the most sleep-interfering cognition (43% report it) in a sample of undergraduate students with insomnia (Wicklow & Espie, 2000). Worry, anxiety and arousal are inter-related processes. Worry is a cognitive attempt to reduce anxiety in response to a future threatening stimulus. Borkovec (1994) has conceptualized worry as a cognitive avoidance strategy, and cites the benefits those with pathological worry report: superstitious avoidance of catastrophe, actual avoidance of catastrophe, avoidance of deeper emotional topics, and coping preparation. Effective problem-solving usually does not occur in worry, because cognitive representation of the anxiety-provoking stimulus is maintained, the individual remains aroused, and effective problem-solving is thus impaired.

The view of worry as ineffective problem-solving is not a new concept. Those who worry are seen as “expert problem-identifiers”, but ineffective problem-solvers. Borkovec (1985) suggests that their inefficacy lies in their difficulty executing solutions to their problems. Marx, Williams, and Claridge (1992) demonstrated that anxious subjects were better able to generate effective solutions than depressed subjects, but those with anxiety often failed in the implementation of their solutions. Others have not found problem-solving deficits (Dugas, Letarte, Rheume, Freeston, & Ladouceur 1995; Dugas, Freeston, & Ladouceur, 1997), and suggest it is poor problem orientation, as defined by poor problem-solving confidence and/or poor perceived control over the problem (Davey, 1994). It is thus argued that a particular response set is engaged when faced with a problem, irrespective of skills, which interferes with effective problem-solving.

It is suggested that worry contains two possible features: a pathological, trait anxiety process and an adaptive task-oriented process (Davey, Hampton, Farrell, & Davidson, 1992). Worry is correlated with features of poor psychological functioning like trait anxiety, avoidance, poor problem-solving confidence, responsibility for negative but not positive outcomes, and the tendency to more readily perceive threats (Davey, 1994). However, once trait anxiety is removed statistically in a partial correlation analysis, worrying is most related to adaptive problem-solving and information seeking. It is suggested that pathological worry occurs when effective, adaptive problem-solving is thwarted (Davey, Hampton, Farrell, & Davidson, 1992) by trait anxiety and resultant catastrophizing or other processes (Vasey & Borkovec, 1983).

Worry is a behavior that occurs in those without clinical disorders, like sub-clinical worriers (e.g., those not meeting full diagnostic criteria for generalized anxiety disorder - GAD) or those with depression, most notably dysthymic disorder (American Psychological Association, 1994). It also occurs in those with disorders characterized by anxiety, as in GAD, obsessive compulsive disorder, agoraphobia, panic disorder, social phobia, and simple phobia (Barlow, 1988). Although worry is a defining feature in GAD, sleep complaints are reported in 41 to 70 percent of those with GAD (Hoehn-Saric & McCleod, 1990; Marten et al., 1993; Noyes et al., 1992); thus disturbed sleep is an important factor as well. Those with GAD often report difficulty inhibiting worry before bed (Uhde, 1994), and that worry exerts negative effects on their sleep.

In GAD, a disorder characterized by excessive worry, there are longer sleep onset latencies and poorer quality of sleep (e.g., decreased sleep efficiency, lower percentage of slow wave sleep, decreased total sleep time, and more arousals) relative to normals (Arriaga & Paiva, 1990; Papadimitriou, Kerkhofs, Kempeaers, & Menlewicz, 1988; Reynolds, Shaw, Newton,

Coble, & Kupfer, 1983). An analysis of polysomnographic variables revealed a discriminant function that accounted for 79% of the variance between low worry and high sub-clinical levels of worry: the high worry group had increased sleep onset latencies, decreased percentage of slow-wave sleep, and more frequent transitions into lighter, stage 1 NREM sleep than the low worry group (Fuller, Waters, Binks, & Anderson, 1997).

In addition to anxiety, other negative mood states such as depression have characteristic sleep changes. Most depressed patients (90%) present with some form of sleep disturbance (Reynolds & Kupfer, 1987), most often increased sleep latency, decreased total sleep, frequent and early awakenings, and decreased slow wave sleep. They also show increased REM sleep or REM density, and most reliably, decreased REM sleep latency (Benca, 1998). In addition to decreased stages 3NREM and 4NREM sleep, there is increased stage 1NREM and 2NREM sleep, which reduce the likelihood of quality sleep because sleep continuity is compromised by the frequent arousals and stage transitions permitted in stage 1 NREM sleep. The same subjective states (i.e. fatigue and dysphoria) that are produced by reduced sleep time and poor sleep quality in normal subjects, are also seen in insomniacs, anxiety disorder patients and depressed patients (Fuller, Waters & Scott, 1994). Such patients usually suffer from delayed sleep onset, frequent awakenings and arousals, decreased delta sleep, increased stage 1 NREM sleep and reduced sleep time, and often report feelings of being unrested/unrestored and mild depressed mood (Ware & Morewitz, 1991).

#### Problem-Solving and the Treatment of Emotional Disturbance

Problem-solving skills training is a component of cognitive-behavior therapy for GAD (Craske, Barlow & O’Leary, 1992), and depression (McCullough, 1991; Nezu, 1986). The rationale underlying treatment of both disorders is that clients are unable to solve stressful

problems effectively. Problem-solving training was popularized by D’Zurilla and Nezu (1982), and teaches clients to engage in four major steps: evaluate the problem, generate possible solutions, select and implement a solution, evaluate the solution and return to the beginning if unsuccessful. Problem-solving coping skills are hypothesized to act as a buffering factor against negative life stress (Billings & Moos, 1981). Social problem-solving therapy (Nezu, 1987) is cited as a probably efficacious treatment for depression by the American Psychological Association Division 12 Task Force on Psychological Interventions (Chambless et al., 1998).

Problem-solving training is also a component of cognitive-behavior therapy (CBT) for GAD (Craske, Barlow & O’Leary, 1992), and CBT is a well-established treatment (Chambless et al., 1998). In Craske, Barlow and O’Leary’s GAD treatment protocol, problem-solving is first introduced to the client via a psycho-educational format. Clients are told that many people with GAD tend to view problems in vague and catastrophic terms, and many fail to generate solutions to problems (Meichenbaum & Jaremko, 1983). Instructions follow as to how to break problems into more manageable segments, and multiple “brainstorming” sessions teach clients to generate multiple solutions.

Problem-solving training is not an element in insomnia treatment packages; in fact there are no “worry-specific” treatment targets in the treatment of insomnia. Given that we know many of those with insomnia are prone to pre-sleep worry, and cognitive arousal interferes with sleep, a problem-solving training component should improve sleep, or at least decrease the amount of reported cognitive arousal. Interventions too close to actual bedtime may be activating, and would therefore be too arousing to be effective. As Espie and Lyndsay (1987) have suggested, a procedure that targets pre-sleep worry at an earlier time in the evening may be more effective than procedures conducted at bedtime. The present study investigated whether an

early evening structured problem-solving procedure (constructive worry) would improve subjective sleep quality, relative to a no-treatment control group, in those with sleep onset and maintenance insomnia. An experimental group (constructive worry; CW group) and a control group (worry only; Worry group) were compared on subjective sleep (i.e., Sleep Diary estimated sleep onset latency, time awake after sleep onset, and Likert ratings of estimated restfulness, sleep quality and sleep initiation difficulty), objective sleep (e.g., actigraphic sleep onset latency, sleep efficiency, and time awake after sleep onset) and pre-sleep arousal (i.e., Pre-Sleep Arousal Scale and State Trait Anxiety Inventory - State Anxiety sub-scale).

### Hypotheses

It was hypothesized that the CW group would experience decreased post-intervention pre-sleep cognitive, somatic and overall arousal on the Pre-Sleep Arousal Scale (PSAS), with cognitive arousal as the greatest change from baseline, and they should also experience decreased State Trait Anxiety Inventory - State Anxiety (STAI-S) relative to baseline scores. The CW group should report improved post-intervention sleep estimates of sleep on the sleep diary; most especially decreased sleep onset latency (SD-SOL), and wakefulness after sleep onset (SD-WASO), but also decreased Likert ratings of difficulty in falling asleep (SD-SID), and increased Likert ratings of restfulness (SD-REST) and sleep quality (SD-SQ) relative to baseline. The CW group should also show improved post-intervention actigraphic estimates of sleep, namely, decreased sleep onset latency (A-SOL) and increased sleep efficiency (A-SE) and total sleep time (A-TST), relative to baseline. The Worry group is not hypothesized to differ significantly from baseline to post-intervention nights on the above variables.

The CW group should also have decreased post-intervention pre-sleep cognitive, somatic and overall arousal on the PSAS (especially cognitive arousal scores), and STAI-S, relative to

the Worry group. The CW group was hypothesized to have better post-treatment sleep on the sleep diary, most especially, decreased sleep onset latency, and wakefulness after sleep onset, but also decreased ratings of difficulty in falling asleep, and increased ratings of restfulness and sleep quality, relative to the Worry group. The CW group was expected to have decreased sleep onset latency and increased sleep efficiency and total sleep time, according to actigraphic estimates of sleep, relative to the post-intervention sleep of the Worry group.

The key variable of interest in this study is the Pre-Sleep Arousal Scale, since the Constructive Worry intervention is designed to address the complaint of pre-sleep arousal in those with insomnia. The second most important variables are the hypothesized reductions in Sleep Diary sleep onset latency and time awake after sleep onset. The third most important variables are the hypothesized improvements in actigraphic sleep onset latency, sleep efficiency and total sleep time, although, this intervention may not be powerful enough to exert effects on anything other than pre-sleep cognitive arousal, since cognitive interventions have not been as effective as behavioral treatments in treating insomnia per se (Morin et al., 1994). It is also hypothesized that problem-solving appraisal will be an important predictor in whether someone reports increased cognitive arousal. More specifically, negative appraisals of problem-solving ability (e.g., on all four indices of the PSI) should predict increased levels of pre-sleep arousal.

## METHODS

### Participants

Participants were undergraduate college students at Louisiana State University, between the ages of 18 and 35 years old ( $M = 20.97$  years old,  $SD = 3.0$ ). There were 33 participants who were randomly assigned to either the CW group ( $N = 16$ ) or the Worry group ( $N = 17$ ). They were given 15 extra course credit points for their participation. Inclusion criteria required the presence of at least one month of three or more nights per week of either sleep onset difficulty (30 minutes or greater) and/or sleep maintenance difficulty (one or more nocturnal awakenings of at least 30 minutes in duration or 3 or more nocturnal awakenings each of at least ten minutes). Participants were screened for emotional disorders with the Primary Care Evaluation of Mental Disorders diagnostic questionnaire and interview (Prime-MD). Those who met Diagnostic and Statistical Manual, fourth edition (DSM-IV) criterion for Alcohol Abuse, or any Anxiety, or Mood Disorders were excluded, as these disorders have sleep-related disturbances associated with them. Those reporting current use of antidepressant or other psychoactive medications on the Demographic Form were excluded (Appendix B), because those medications can exert effects upon sleep. Participants were screened for sleep disorders with the Sleep Disorders Inventory. To control for sleep disturbance related to other sleep disorders, those who met non-PSG International Classification of Sleep Disorders Diagnostic and Coding Manual criteria for sleep disorders were excluded, with the exception of those with primary insomnia symptoms (i.e., insomnia as defined above, and not determined to be secondary to another disorder). Those who scored as an extreme morning person on the Morningness/Eveningness Questionnaire – MEQ (scores in the range of 70-86) or an extreme evening person (MEQ scores in the range of 16-30) were excluded, to screen out extremes in circadian tendency that could

cause or exacerbate insomnia. To control for the effect of school-related stress, participants with an examination during the week of the study were not permitted to participate during the week of the test.

### Procedures

After receiving approval from the Louisiana State University Institutional Review Board, participants were recruited from an online psychology experiment bulletin board. On the first day of participation, the study was explained and informed consent was obtained (Appendix A). Prospective participants were scheduled a 60-minute screening appointment during which time they were given a brief clinical interview (conducted by advanced doctoral candidates), and completed self-report measures. Participants were screened for psychiatric or sleep disorders, or extremes in circadian tendency using the Prime MD, the Sleep Disorders Inventory (SDI), and the Horne Morningness/Eveningness Questionnaire (MEQ). Participants also completed a Problem-Solving Inventory (PSI) to assess appraisal of problem-solving abilities. When participants met insomnia inclusion criteria on the SDI (e.g., presence of sleep maintenance or onset insomnia), they completed a demographic questionnaire (Appendix B), and received an ACTITRAC motion detector, actigraphy instructions (Appendix C), Sleep Hygiene Monitoring Forms (Appendix D), Sleep Diaries (Appendix E), PSAS, and the STAI-S.

Sleep hygiene was regulated to equate the two groups on the degree to which poor sleep habits could account for any experimental changes or differences. Thus, participants were required to follow basic sleep hygiene rules throughout the study (please see Appendix C), and those who had significantly irregular sleep habits (e.g., bedtime or wake times that vary by 4 hours or more on 3 or more times per week; less than 6 hours, or more than 10 hours of sleep, 3 or more times per week; or bedtimes after 1 AM or before 9 PM) were excluded from the study.

Adherence to the sleep rules was assessed using self-report (Sleep Hygiene Monitoring Form) and activity monitoring (actigraphy). To maximize compliance with the sleep hygiene rules, participants were informed that compliance would be verified using the actigraphy data. Carney, Lajos and Waters (2002) found that providing an instruction that adherence would be verified using the participant's actigraphy data, increased the accuracy of reporting adherence to sleep hygiene rules in an instruction group relative to a no-instruction group. Those not complying with the rules were excluded from the study. Sleep hygiene as the sole treatment for insomnia is generally not efficacious (Reynolds, Kupfer, Buysse, Coble, & Yeager, 1991); thus, it is unlikely that significant improvements in sleep would be attributable to the following of sleep hygiene rules.

The first two nights were baseline nights, where participants did not engage in their respective procedures, but completed a PSAS and STAI-S on both nights before bed, and a Sleep Diary and Sleep Hygiene Monitoring form the next two mornings. After the two baseline nights, participants returned to the sleep laboratory to be trained individually for approximately 15 minutes on their retrospective procedures (e.g., constructive worry in the experimental group and worry reporting in the control group). Participants did not receive this training until after the baseline nights to prevent them from engaging in their respective procedures during baseline measurement. The experimental group received the Constructive Worry Instructions and Constructive Worry Worksheet (Appendix G), while control group subjects received the Worry Self-Monitoring form (Appendix H), a Penn State Worry Questionnaire (PSWQ) and a Worry Domains Questionnaire (WDQ). On the five subsequent experimental nights, the groups completed their worry procedures for 15 minutes sometime between 6-8 PM.

During the early evening, the Constructive Worry group recorded a minimum of three problems/worries that they thought had the greatest likelihood of keeping them awake at bedtime, on one half of the Constructive Worry page. On the other half of the page, participants recorded the next step that could contribute to the resolution of the problem. They repeated this procedure for each of the problems they listed. They were instructed to fold the Constructive Worry sheet in half and place it on the nightstand next to their bed. They were also instructed that if they experienced worry before bed, they were to tell themselves that they had dealt with their problems when they were at their problem-solving best, and there was nothing more they could do while they were so tired (for complete procedure, see Appendix F). In contrast, the control group completed a PSWQ and WDQ, and then recorded a minimum of three worries. Thus, each group spent the same amount of time focused on worries (approximately 15 minutes), but the experimental group was given an opportunity to “solve” some of these problems, and the control group was not. Each experimental night, participants in both groups completed a PSAS and STAI S-Anxiety scale just prior to bedtime, and each morning, they completed a Sleep Diary and Sleep Hygiene Compliance form (to verify compliance with sleep hygiene rules).

At the beginning of the study, all participants were informed that though they might experience an improvement in their sleep through participation, they would be receiving a treatment for worry that is not specifically designed for insomnia per se. They were told there was a small, but unestablished, possibility that they might experience a worsening of their sleep during the study. This was to counteract some of the most obvious possible demand characteristics, as improvement due to experimental demand characteristics is a well-established phenomenon in insomnia treatment research (Carr-Kaffashan & Woolfolk, 1979; Borkovec, Kaloupek & Slama, 1975). All participants were told they could participate in a free 4-week

group insomnia treatment at the Louisiana State University Psychological Services Center if they experienced a worsening of sleep symptoms that lasted for more than one week after participation in the study. Upon completion of the study, participants were thanked and given extra course credit.

### Measures

The Prime-MD, originally designed for primary care physicians, is a quick procedure for diagnosing mental disorders. The Prime-MD contains a 26-item patient questionnaire and a 12-page structured interview to follow-up on patient questionnaire responses. The interview responses determine the presence or absence of 18 categories of mental disorders. There is reported good agreement between Prime-MD diagnoses and those of independent physicians ( $k = 0.71$ ; overall accuracy rate was 88%) in a validation study (Spitzer et al., 1994). Patients with Prime-MD diagnoses had lower functioning, more disability days, and higher rates of health care use, than patients without Prime-MD diagnoses. Also, prevalence rates for Prime-MD threshold disorders (26%), in the Spitzer et al. (1994) validation study, were comparable to previous studies of lengthy, structured diagnostic interviews (Schulberg et al., 1988; Barrett et al., 1988; Von Korf et al., 1987). The Prime-MD was used as a screening tool to exclude those participants with serious psychiatric disorders. Because psychiatric disorders vary in the sleep abnormalities associated with them, participants had to be excluded if they met threshold criterion for Axis I disorders.

The SDI is a 60-item self-report questionnaire, designed to assess the self-reported signs of sleep disorders and dysfunctions. It is based upon International Classification of Sleep Disorders (ICSD) criteria. There are no validation studies on the SDI, and there are no other sleep inventories that have been adequately validated, because to validate them would require

literally hundreds of over-night polysomnograms and clinical interviews to document such important, but rare disorders like narcolepsy, idiopathic hypersomnia, and REM sleep behavior disorder. However, it is accepted practice to screen for sleep disorders using ICSD criteria (Speilman, Yang, & Glovinski, 2001), and the SDI covers all of the major ICSD classifications (sleep apnea, psychophysiological insomnia, narcolepsy, idiopathic hypersomnia, periodic limb movement disorder, restless leg syndrome, shift work sleep disorder, nightmares, insufficient sleep syndrome and REM sleep behavior disorder), as well as descriptions of daytime hypersomnolence, insomnia, sleep time allocation and sleep loss. Any responses that indicated a possible sleep abnormality were queried with a brief interview based on ICSD diagnostic criteria to assess whether the participant met ICSD criteria for a sleep disorder other than insomnia (see Appendix J).

The MEQ is a self-report measure consisting of 19 items to assess extreme morningness or extreme eveningness. Those who are extreme morning persons tend to go to bed earlier and rise earlier if work, family or social constraints do not restrict them. They feel “at their best” earlier in the day, relative to intermediate or extreme evening types. In contrast, extreme evening people tend to go to bed later and rise later, if other constraints do not restrict them, and feel “at their best” later in the day. In validation studies, scores on the questionnaire were compared with hourly core body temperature measurements and sleep logs (Horne & Ostberg, 1976; Posey & Ford, 1981). They found that bedtime and wake times were significantly correlated with morningness-eveningness. According to the Horne and Ostberg criteria, less than 20% of the population can be classified as extreme morning or extreme evening types, with the remaining 80% falling somewhere between moderate evening or morning types, about 20% of which are classified as intermediate types. This scale has been validated by Posey and Ford (1981) on a

North American college student population (n=259). The mean score in this study was 50.38 and the standard deviation was 11.36. Horne and Ostberg's criterion for extreme morningness is a score between 16 and 30 (more than 2.5 standard deviations below Posey and Ford's mean) and a score between 70 and 86 (about 2.5 standard deviations above Posey and Ford's mean) is considered an extreme evening type. These criteria were used to exclude extreme morning or evening types; thus, those scoring between 31 and 69 were the only participants included in the study. The Morningness-Eveningness Questionnaire is used regularly in circadian rhythm research as a screening tool and has been found to be a reliable (KR20,  $r = 0.89$ ) and valid instrument, as it correlates significantly with peaks in core body temperature ( $r = -0.51$ ), self-reported bedtime ( $r = 0.79$ ), and self-reported wake time ( $r = -0.67$ ) (Posey & Ford, 1981).

The PSAS (Nicassio, Mendelowitz, Fussell, & Petras, 1985) is a 16-item self-report measure of the state of arousal just before sleep. It measures both somatic and cognitive states of pre-sleep arousal, although the cognitive subscale is more strongly associated with nightly sleep onset latency than the somatic subscale. The PSAS has good reported reliability: Cronbach alpha for the cognitive sub-scale has been reported between 0.76 and 0.88, with the somatic sub-scale between 0.79 to 0.81; test re-test reliabilities for an undergraduate population with a three week interval was 0.72 for the cognitive sub-scale and 0.76 for the somatic sub-scale. Validity studies have also supported use of the PSAS, as somatic and cognitive sub-scales have correlated significantly with the Taylor Manifest Anxiety Scale ( $r = 0.50$  and  $r = 0.58$ , respectively), the Center for Epidemiological Studies Depression Scale ( $r = 0.40$  and  $r = 0.41$ , respectively) and sleep difficulty indices (e.g., "Would you describe yourself as an insomniac?"; sleep onset latency, total sleep time, awakenings from sleep, and ratings of "listfulness" during the day.) The PSAS also distinguishes between good sleepers and those with insomnia, particularly on the

basis of the cognitive sub-scale score; those with insomnia report much higher cognitive arousal than somatic arousal and more cognitive arousal than good sleepers (Nicassio, Mendelowitz, Fussell, & Petras, 1985).

The STAI Anxiety scale (Spielberger, 1983) consists of 20 statements that evaluate the experience of anxiety “right now, at this moment”. The feelings evaluated in the S-Anxiety scale are those of apprehension, tension, nervousness and worry. The STAI was normed on college students, nonpatients, psychiatric and neuropsychology patients, and is considered a valid and reliable instrument (Spielberger, 1983). Internal consistency for State anxiety is high ( $r = 0.92$ ), and correlates significantly with the Minnesota Multiphasic Personality Inventory Psychasthenia scale ( $r = 0.81$ ; Spielberger, 1983).

The PSWQ (Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item self-report measure of propensity to worry. In the Meyer et al. (1990) reliability studies, internal consistency yielded high coefficient alpha ( $r = 0.94$ ), and test-retest reliability was also high ( $r = 0.92$ ). With respect to validity, it has correlated significantly with the STAI-S ( $r = 0.49$ ) and the Cognitive Somatic Anxiety Questionnaire ( $r = 0.69$ ; Meyer et al., 1990). It also has significantly differentiated between those with Generalized Anxiety Disorder and those with other anxiety disorders, and those without anxiety disorders (Brown, Antony & Barlow, 1992); it also is sensitive to successful treatment of Generalized Anxiety Disorder (Stober & Bittencourt, 1998). There is support for the use of the PSWQ as a state rather than trait measure of worry by changing the time frame in the instructions from “how typical or characteristic each statement is of you” to “how typical or characteristic each statement was of you during the past week” (Stober & Bittencourt, 1998). The purpose of this measure in the present study was not to diagnose GAD,

but to ensure that the control group was focused upon worry for the same amount of time as the experimental group, while the latter engaged in constructive worry.

The WDQ (Tallis, Eysenk, & Mathews, 1992) is a 30-item self-report measure designed to assess levels of worry across five domains: relationships, lack of confidence, aimless future, work incompetence, and financial worries. It is suitable for the measurement of worry in sub-clinical worriers, and has been evaluated in an undergraduate population (Tallis, Eysenk, & Mathews, 1992). It is a compliment to the PSWQ, as the PSWQ reflects how much a subject worries, but does not survey worry content. The WDQ has good reliability (Cronbach's alpha,  $r = 0.92$ ) and validity; it significantly correlates with the STAI Trait Anxiety ( $r = 0.785$ ), and the PSWQ ( $r = 0.67$ ) (Tallis, 1989). As with the PSWQ, the WDQ was used to ensure that the control group was focused upon worry for the same amount of time as the experimental group while they engaged in constructive worry.

The PSI (Heppner, 1988) is a 35-item instrument to assess the perception of problem-solving skills; it does not assess actual problem-solving skills. The PSI has three subscales derived from factor analysis: Problem-Solving Confidence, Approach-Avoidance Style, and Personal Control. Low scores on the Confidence subscale indicates trust and self-assurance in one's problem-solving ability. Low scores on the Approach-Avoidance scale reflects a tendency to approach, rather than avoid, problem-solving situations. Low scores on the Personal Control suggests a belief the person is in control of their emotions and behaviors while solving problems. The PSI was originally developed as a research tool to investigate the relationship between problem-solving appraisal and cognitive, affective, and/or behavioral variables. Alpha coefficients for each scale range from adequate (PC subscale,  $r = 0.72$ ) to very good (PSI overall,  $r = 0.91$ ). Test-retest reliability has been reported for two weeks ( $r = 0.89$ ) to 2 years ( $r = .60$ ).

The three subscales and the overall PSI score correlated significantly ( $r = 0.79$ ,  $p < 0.001$ ) with students' ratings of their level of problem-solving skills and their perceived level of satisfaction with their skills (Heppner & Petersen, 1982). The PSI was used in the present investigation to determine if the participants' appraisal of their problem-solving predicts whether the problem-solving procedure affected pre-sleep arousal.

A wrist actigraph is a portable instrument, worn like a wristwatch, which measures body movement to provide an indirect but objective measure of sleep/wake activity. There is also support for the use of actigraphy in increasing the honesty of reporting adherence to sleep hygiene instructions (Carney, Lajos, & Waters, 2001). Actigraphy has been validated with PSG in a variety of settings and populations. Mullaney et al. (1980) compared PSG and wrist actigraphy in 63 psychiatric patients and 39 normal volunteers. There was a high coefficient of agreement for total sleep time ( $r = 0.89$ ) and duration of time awake after sleep onset ( $r = 0.70$ ) with PSG for both normal and psychiatric groups. The actigraph and the PSG were in agreement for 96.3% of normals and 91.6% of psychiatric patients; the actigraph had a tendency to overestimate total sleep time by about 15 minutes per night in those with insomnia. There has been continuing controversy concerning the possible overestimation of sleep duration in insomniacs by actigraphy, because insomniacs can sometimes lay awake, but relatively motionless, for long periods of time (Chambers, 1992). Thus, wrist actigraphy is considered a valid, objective instrument for the measurement of sleep/wake activity, with a possible decrement in accuracy when significant insomnia is present. This means that actigraphy sleep is assessed as being better than it actually is. Actigraphs are scored with a computerized scoring program, which also was validated with PSG (Cole et al., 1992). In the present study, sleep onset latency, sleep efficiency, and total sleep time will be examined for post-intervention changes.

The sleep onset latency is an important index of how long it takes from the attempt to fall asleep until the participant actually falls asleep. ICSD criteria suggest that sleep onset latencies of 20 minutes or more, are pathological. Sleep efficiency refers to the percentage of time spent asleep divided by the percentage of time spent in bed. ICSD criteria suggest that sleep efficiencies less than 85% are pathological, and suggest difficulty difficulties with initiating or maintaining sleep. The total sleep time estimate indicates how long participants actually slept, and should be close to 8 hours (as per the sleep rules) in the absence of insomnia.

The use of sleep diaries to track sleep behavior patterns and subjective ratings of sleep quality, as well as pre-sleep experiences, is a common tool in sleep assessment (Buysse et al., 1997). The Sleep Diary (Lacks, 1987) test-retest reliability is reported to be high for sleep onset latency ( $r = 0.98$ ), number of arousals ( $r = 0.88$ ), and time awake after sleep onset ( $r = 0.84$ ). The Sleep Diary significantly correlates with polysomnographic data for sleep onset latency (ranges from 0.62 to 0.99 depending on the criteria used for PSG sleep onset scoring) (Lacks, 1987), despite the fact that some patients with insomnia view their sleep in a distorted way, which can result in inaccurate estimates of sleep (for Sleep State Misperception, see Spielman, Yang, & Glovinsky, 2001). Regarding sleep quality (normally reflected in PSG measures of SWS, stage 1NREM sleep, awakenings, and transient arousals), this study will be primarily interested in “perceived” sleep quality changes, as some patients with insomnia report pathological subjective estimates of sleep even after sleep is improved according to PSG (Rechtschaffen & Monroe, 1969). Some clinicians have suggested that since insomnia rarely has complications other than those related to the patient’s discomfort, the goal of treatment should be to improve subjective sleep quality, rather than targeting EEG-verified improvements only (see Stepanski, 2001). Regardless, since those with insomnia have a tendency to distort self-reported

sleep ratings, this study will compare subjective Sleep Diaries with the objective sleep estimates of actigraphy. The Sleep Diary used in the present study reports: bedtime, wake time, how much time taken to fall asleep (SOL), how much time spent awake after sleep onset (WASO), and 7-point Likert ratings of sleep quality (SQ), restfulness (REST), sleep initiation difficulty (SID), somatic tension, and mental tension. Items 1 (bedtime, B) and 3 (wake time, W) were used to specify the scoring period for the sleep/wake cycle on the actigraph scoring program. Item 4 (sleep onset latency, SOL) was compared with the actigraph estimate of SOL. SID, SQ, and REST were included as measured dependent variables in the statistical analyses. Items 9 (somatic tension) and 10 (mental tension) were included in the correlational analyses only due to their overlap with PSAS Cognitive and PSAS Somatic subscales, and because the PSAS scales are validated, but the sleep diary somatic and mental tension scores are not.

## RESULTS

### Demographic Analyses

Chi square analyses were conducted to test for differences between the two groups in the distribution of gender and race. The results of both chi-square tests were not statistically significant, chi-square (1, N = 33)= 1.87,  $p = 0.17$ , and chi-square (3, N = 33)= 3.27,  $p = 0.35$ , respectively. Chi square analyses tested whether groups differed on categorical Sleep Disorders Inventory items. All chi square analyses were not significant, thus groups did not differ on SDI presence of sleep onset insomnia, chi-square (1, N = 33)= 0.002,  $p = 0.965$ ; SDI presence of sleep maintenance insomnia, chi-square (1, N = 33)= 0.259,  $p = 0.611$ ; SDI report of problem-solving keeping them awake at night, chi-square (1, N = 33)= 0.267,  $p = 0.606$ ; SDI report of sleeping better at home, chi-square (1, N = 33)= 0.170,  $p = 0.680$ ; SDI report of worry about next-day functioning, chi-square (1, N = 33)= 2.431,  $p = 0.119$ ; or SDI report of anger when having difficulty falling asleep, chi-square (1, N = 33)= 1.006,  $p = 0.316$ . A multivariate analysis of variance (MANOVA) tested whether there were statistically significant differences between the groups on other demographic variables (age, SDI average sleep onset latency, SDI average time awake after sleep onset, circadian tendency score on the MEQ, PSI score, PSI Confidence subscale score, PSI Approach/Avoidance subscale score, and PSI Personal Control subscale score). The omnibus test of demographic variables was not statistically significant, Wilks' Lambda = 0.820,  $F(7,20) = 0.627$ ,  $p = 0.728$ ; thus groups did not significantly differ on these variables. See Table 1 for between-group means and standard deviations on age, presence of sleep onset insomnia, average sleep onset latency, presence of sleep maintenance insomnia, average time awake after sleep onset, report of problem-solving keeping them awake at night, report of sleeping better at home, report of worry about next-day functioning, report of anger

when having difficulty falling asleep. See Table 2 for between-group means and standard deviations on the PSI and MEQ scores.

Table 1: Means and Standard Deviations of Age and Sleep Disorders Inventory Insomnia Variables

Group		Age	SDI Sleep Onset Insomnia	SDI Sleep Onset Latency	SDI Sleep Maintenance Insomnia	SDI Time Awake After Sleep Onset
CW	Mean	19.25	2.00	58.75	1.43	109.75
	S.D.	0.96	0	18.87	0.51	43.85
W	Mean	21.83	2.00	51.33	1.44	85.50
	S.D.	4.83	0	15.32	0.51	25.30

Table 1b: Means and Standard Deviations of Sleep Disorders Inventory Psychophysiological and Cognitive Insomnia Variables

Group		SDI Sleeps Better at Home	SDI Problem-Solving Keeps Them Awake	SDI Worry About Next-Day Functioning	SDI Anger About Not Falling Asleep
CW	Mean	1.00	1.75	1.00	1.50
	S.D.	0	0.50	0	0.58
W	Mean	1.00	2.00	1.83	1.83
	S.D.	0	0	0.41	0.41

Table 2: Means and Standard Deviations of Problem-Solving Inventory and Morningness-Eveningness Questionnaire scores

Group		PSI	PSI Conf.	PSI A-A	PSI PC	MEQ
CW	Mean	85.20	23.93	45.07	16.20	47.73
	Std. Dev.	19.70	6.65	14.16	3.49	8.40
W	Mean	91.59	28.65	45.18	17.76	49.19
	Std. Dev.	19.20	6.04	11.83	5.15	10.44

According to responses on the Sleep Disorders Inventory, 15 participants in the CW group (94%), and 16 participants in the Worry group (84%) reported the presence of Sleep Onset Insomnia (e.g., taking 30 minutes or more to fall asleep on 3 or more nights per week).

According to responses on the Sleep Disorders Inventory, 2 participants in the CW group (14%), and 1 participant in the Worry group (7%) reported the presence of Sleep Maintenance Insomnia (e.g., being awake after initial sleep onset for 30 minutes or more to fall asleep on 3 or more nights per week).

According to responses on the Sleep Disorders Inventory, relatively few participants reported sleeping better when away from home (15.2%). When participants indicate “yes” to this item, it suggests the presence of an association between their bedroom setting and insomnia. Over half of participants reported concern and worry about the effect of poor sleep quality on next-day functioning (58%). Many reported pre-sleep emotional arousal, in the form of anger or frustration when they are not able to sleep (69%), and over three quarters reported pre-sleep cognitive arousal, in the form of problem-solving, keeps them awake at night (79%).

A multivariate analysis of variance (MANOVA) was conducted to determine whether the two groups were different on baseline measures of arousal/anxiety (PSAS, PSAS Cognitive subscale, PSAS Somatic subscale, and STAI-S subscale). There were no statistically significant between-group differences on baseline arousal or anxiety measures, Wilks' Lambda = 0.948,  $F(4, 28) = 0.383$ ,  $p = 0.819$ . The baseline and post-intervention means and standard deviations of the STAI-S, PSAS, PSAS Cognitive and PSAS Somatic are given in Table 3.

Table 3: Means and Standard Deviations of Arousal and Anxiety Variables

			PSAS	PSAS Cognitive	PSAS Somatic	State-Trait Anxiety
Constructive Worry Group	Baseline	Mean	42.38	30.97	11.41	44.38
		Std.Dev.	5.93	5.58	2.74	10.19
	Post- Experiment	Mean	31.69	20.91	10.78	38.69
		Std.Dev.	5.93	5.71	2.30	11.85
Worry Group	Baseline	Mean	42.63	31.72	10.88	44.81
		Std.Dev.	6.84	5.46	2.33	11.39
	Post- Experiment	Mean	38.72	28.69	10.31	44.91
		Std.Dev.	4.32	4.28	2.59	10.67

A multivariate analysis of variance (MANOVA) was conducted to determine whether the two groups were different on baseline measures of subjective and objective sleep (SD-SOL, SD-TST, SD-WASO, SD-REST, A-SOL, A-TST, and A-SE); there were no statistically significant between group baseline differences, Wilks' Lambda = 0.888,  $F(7, 19) = 0.344$ ,  $p = 0.923$ . The baseline and post-intervention means and standard deviations of the Sleep Diary variables are

given in Table 4, Likert Sleep Diary variables are given in Table 5, and actigraphy variables are given in Table 6.

Table 4: Means and Standard Deviations of Sleep Diary Variables

Group		SD SOL (min.)	SD WASO (min.)	Awakenings (#)
CW	BL Mean (SD)	29.61 (21.84)	7.38 (6.50)	1.31 (1.31)
	Post Mean (SD)	42.63 (26.80)	15.41 (13.57)	1.90 (0.99)
W	BL Mean (SD)	32.63 (14.0)	9.18 (8.87)	1.11 (1.03)
	Post Mean (SD)	31.41 (14.6)	7.52 (9.53)	1.14 (1.20)

Table 5: Means and Standard Deviations of Sleep Diary Likert Rating Variables

		Sleep Quality	Rated Restfulness	Cognitive Tension	Somatic Tension	SID
Constructive Worry Group	BL Mean	3.13	3.34	3.09	2.34	2.94
	BL S.D.	0.66	0.85	1.0	0.51	1.03
	X Mean	2.63	2.59	20.91	10.78	2.38
	X S.D.	0.85	0.80	5.71	2.30	0.92
Worry Group	BL Mean	3.06	42.63	31.72	10.88	2.56
	BL S.D.	0.75	6.84	5.46	2.33	0.95
	X Mean	2.75	38.72	28.69	10.31	2.66
	X S.D.	0.75	4.32	4.28	2.59	1.08

Table 6: Means and Standard Deviations of Actigraphy Sleep Variables

Group		Sleep Efficiency %	Total Sleep Time (min.)	Sleep Onset Latency (min.)
CW	BL Mean (SD)	0.79 (0.08)	398 (58.15)	12.75 (6.05)
	Post Mean (SD)	0.82 (0.09)	385 (62.45)	12.79 (9.60)
W	BL Mean (SD)	0.80 (0.10)	394 (50.04)	14.22 (6.36)
	Post Mean (SD)	0.75 (0.13)	376 (44.49)	18.60 (8.32)

An examination of the averaged baseline Sleep Diary sleep onset latency revealed that 11 participants in the CW group (69%), and 11 participants in the Worry group (65%), reported sleep onset latencies that exceeded 30 minutes. The average baseline Sleep Diary sleep onset latency for the CW group was 29.61 minutes (SD = 21.84), and the average baseline Sleep Diary sleep onset latency for the Worry group was 32.63 minutes (SD = 14.0). In contrast, there were no actigraphy sleep onset latencies in either group that exceeded 30 minutes (CW group: M = 12.75 minutes, SD = 6.05; Worry group: M = 14.22 minutes, SD = 6.36).

An examination of the averaged baseline Sleep Diary time awake after sleep onset revealed that 8 participants in the CW group (50%) reported time awake after sleep onset over 30 minutes, and 7 participants in the Worry group (41%) reported time awake after sleep onset over 30 minutes. The average baseline Sleep Diary time awake after sleep onset for the CW group was 7.38 minutes (SD = 6.50), and the average baseline Sleep Diary time awake after sleep onset for the Worry group was 9.18 minutes (SD = 8.87). The average baseline Sleep Diary number of awakenings for the CW group was 1.31 (SD = 1.31), and the average baseline Sleep Diary number of awakenings for the Worry group was 1.11 (SD = 1.03). Baseline actigraph sleep

efficiency was low for each group (CW group:  $M = 79\%$ ,  $SD = 0.08$ ; Worry group:  $M = 80\%$ ,  $SD = 0.10$ ).

#### Between and Within-Group Analysis of Anxiety and Arousal Variables

A repeated measures MANOVA was conducted to determine whether the two groups were different on measures of arousal/anxiety (PSAS, PSAS Cognitive subscale, PSAS Somatic subscale, and STAI-S subscale), and whether there were within-subject differences. There was a significant main effect of time, Wilks' Lambda = 0.269,  $F(4, 27) = 18.307$ ,  $p = 0.001$ , and a statistically significant TIME X GROUP interaction, Wilks' Lambda = 0.592,  $F(4, 27) = 4.659$ ,  $p = 0.005$ . Analyses of variance (ANOVA) on each dependent variable were conducted as follow-up tests to the significant MANOVA interaction. The ANOVA for PSAS was significant,  $F(1,30) = 10.285$ ,  $p = 0.003$ , and so was the ANOVA for PSAS Cognitive subscale,  $F(1,30) = 18.249$ ,  $p = 0.001$ . The previous MANOVA on baseline arousal and anxiety found the two groups did not differ on baseline PSAS and PSAS Cognitive subscale scores, and this MANOVA found that the CW group had significantly lower post-treatment PSAS and PSAS Cognitive scores, than the Worry group. Follow-up within group t-tests on CW scores found that post-intervention PSAS was significantly lower than baseline PSAS,  $t(15) = 6.97$ ,  $p < 0.001$ , and the CW scores on the post-intervention PSAS Cognitive subscale were significantly lower than baseline PSAS Cognitive subscale,  $t(15) = 7.22$ ,  $p < 0.001$ . In contrast, the Worry group's scores did not significantly change from baseline to post-intervention, on either the PSAS,  $t(16) = 2.62$ ,  $p = 0.07$ , or the PSAS Cognitive,  $t(16) = 2.55$ ,  $p = 0.07$ . Using a more conservative Bonferroni approach to control for Type I error across the seven tests, a p value of less than .007 ( $.05/7 = .007$ ) was required for significance, and the results remained the same. The baseline and post-

intervention means and standard deviations of the STAI-S, PSAS, PSAS Cognitive and PSAS Somatic are given in Table 3.

#### Between and Within-Group Analysis of Sleep Variables

A repeated measures MANOVA was conducted to determine whether the two groups were statistically different on measures of subjective sleep (SD-SOL, SD-TST, SD-WASO, SD-Quality, SD-SID, SD-REST). There were no statistically significant between group differences, Wilks' Lambda = 0.900,  $F(6, 20) = 0.37$ ,  $p = 0.89$ , there was no effect of time, Wilks' Lambda = 0.577,  $F(6, 20) = 2.45$ ,  $p = 0.61$ , and there was no significant interaction, Wilks' Lambda = 0.793,  $F(6, 20) = 0.87$ ,  $p = 0.53$ . A repeated measures MANOVA was conducted to determine whether the two groups were different on actigraphic measures of objective sleep (A-SOL, A-TST, A-SE). There were no statistically significant differences for time, Wilks' Lambda = 0.852,  $F(3, 25) = 1.44$ ,  $p = 0.254$ ; group, Wilks' Lambda = 0.868,  $F(3, 25) = 1.27$ ,  $p = 0.307$ ; or an interaction, Wilks' Lambda = 0.831,  $F(3, 25) = 1.70$ ,  $p = 0.193$ . Refer to Table 4 for baseline and post-intervention means and standard deviations of the Sleep Diary SOL and WASO, and Table 5 for Likert sleep variables. Refer to Table 6 for means and standard deviations of actigraphic variables.

#### Correlations Between Anxiety/Arousal and Sleep Variables

Correlation coefficients were computed among the anxiety/arousal scales and the Likert scale ratings of sleep on the Sleep Diary. Using the Bonferroni approach to control for Type I error across the nine correlations, a  $p$  value of less than .006 ( $.05/9 = .006$ ) was required for significance. The results of the correlational analyses presented in Table 7 show that six of the nine correlations were statistically significant and were greater than or equal to 0.56. PSAS scores correlated highly with PSAS Cognitive and PSAS Somatic scores ( $r = .932$  and  $r = .566$

respectively), but they did not correlate with any other measure. The STAI-S was positively related to reports of Somatic Tension ( $r = 0.725$ ) and Mental Tension ( $r = 0.599$ ) on the Sleep Diary. Somatic Tension was positively related to Mental Tension on the Sleep Diary ( $r = 0.614$ ). Thus, although mental and somatic tension were related to each other on the Sleep Diary, and pre-sleep cognitive arousal and pre-sleep somatic arousal were related to each other on the PSAS, Sleep Diary and PSAS variables were not significantly related to each other. Ratings of Sleep Quality were positively correlated with ratings of Restfulness on the Sleep Diary ( $r = 0.694$ ).

Table 7: Correlations between Subjective Sleep Ratings and Anxiety/Arousal Measures

	PSAS Cog.	PSAS Som.	PSAS	STAI	Diary SID	Diary Quality	Diary Somatic	Diary Mental	Diary Rest.
PSAS Somatic	.229 (.199)								
PSAS	.932* (.001)	.566* (.001)							
STAI	.439* (.011)	.048 (.792)	.389* (.025)						
Diary SID	.147 (.416)	.093 (.608)	.156 (.385)	.156 (.385)					
Diary Quality	-.001 (.995)	.033 (.857)	.011 (.952)	.338* (.054)	.206 (.249)				
Diary Somatic	.149 (.407)	.098 (.586)	.163 (.364)	.725* (.001)	.405* (.019)	.309 (.080)			
Diary Mental	.403* (.020)	.030 (.870)	.354* (.043)	.599* (.001)	.248 (.164)	.192 (.285)	.614* (.001)		
Diary Rest.	.239 (.180)	.008 (.964)	.205 (.253)	.329 (.062)	.314 (.075)	.694* (.001)	.275 (.121)	.169 (.346)	

\* Correlation significant at the 0.05 level (two-tailed)

Correlation Between Appraisal of Problem-Solving, Anxiety/Arousal and Sleep

Correlation coefficients were computed to determine whether appraisal of one’s problem solving abilities was related to anxiety/arousal or Likert scale ratings of sleep on the Sleep Diary. The results of the correlational analyses presented in Table 8 show that Sleep Diary ratings of Restfulness was significantly related to overall PSI ( $r = .361$ ) and PSI Approach-Avoidance ( $r = .023$ ).

Table 8: Correlations between PSI, PSAS and Sleep Diary Likert Ratings

	PSI	PSI Confidence	PSI Approach Avoidance	PSI Personal Control
PSAS Cognitive	.044 (.810)	.047 (.797)	-.002 (.992)	.131 (.475)
PSAS Somatic	.055 (.765)	-.128 (.487)	.164 (.368)	-.035 (.847)
PSAS	.058 (.754)	-.008 (.964)	.060 (.746)	.097 (.596)
STAI	-.035 (.850)	.159 (.386)	-.158 (.389)	.060 (.745)
Diary SID	.172 (.347)	.136 (.458)	.151 (.410)	.127 (.487)
Diary Quality	.141 (.441)	.105 (.568)	.145 (.429)	.056 (.761)
Diary Somatic Tension	-.077 (.676)	-.019 (.919)	-.156 (.393)	.135 (.462)
Diary Mental Tension	-.057 (.758)	-.019 (.918)	-.041 (.826)	-.107 (.558)
Diary Restfulness	.361* (.042)	.225 (.217)	.400* (.023)	.122 (.507)

\* Correlation is significant at the 0.05 level (2-tailed)

### Problem-Solving Appraisal as a Predictor of Pre-Sleep Arousal

A multiple regression analysis was conducted to evaluate whether problem-solving ability appraisal predicted pre-sleep arousal. The predictors were the four PSI indices (overall PSI, PSI Confidence, PSI Personal Control, and PSI Approach-Avoidance), while the criterion variable was post-intervention PSAS. The overall PSI score was excluded from the model. The linear combination of PSI Confidence, PSI Personal Control, and PSI Approach-Avoidance was significantly related to PSAS,  $F(3, 27) = 4.498, p = .011$ . The sample multiple correlation coefficient was 0.58, indicating that approximately 29% of the variance of PSAS can be accounted for by the linear combination of the PSI subscales. In Table 9, indices are presented to indicate the relative strength of the individual predictors. PSI Personal Control was the only one of the bivariate correlations that was significantly related to PSAS ( $r = 0.428, p < 0.05$ ). Since the relationship between PSI Personal Control and PSAS is positive, it suggests that high scores on PSI Personal Control (e.g., a negative appraisal of one's personal control over their emotions and behaviors when encountering problems) is associated with a high degree of pre-sleep arousal. On the basis of these correlational analyses, it is tempting to conclude that the only useful predictor is PSI Personal Control. PSI Personal Control alone accounted for 18% of the variance, while PSI Confidence and PSI Approach Avoidance accounted for an additional 11% of the variance ( $29\% - 18\% = 11\%$ ). However, judgements about the relative importance of these predictors are difficult because they are correlated.

### Dropouts and Adherence to Sleep Rules

Adherence to sleep rules was assessed using self-report on the Sleep Hygiene Monitoring Form, self-report on the Sleep Diary of bedtimes and waketimes, and by inspection of actigraphy bedtimes and waketimes. There were two drop-out/removals in the Worry group only. The one

Table 9: The Bivariate and Partial Correlations of the PSI Predictors with Pre-Sleep Arousal

Predictors	Correlation between each predictor and PSAS	Correlation between each predictor and PSAS controlling for all other predictors
PSI Confidence	+.217	+.245
PSI Approach-Avoidance	-.132	-.428*
PSI Personal Control	+.428*	+.466**

\* p<.05

\*\*p<.01

participant did not comply with the sleep rules; she reported noncompliance on the Sleep Diary, and actigraphy confirmed bedtimes and waketimes much later than the required 11 PM to 7 AM. The other participant did not report noncompliance on the Sleep Diary, but actigraphy showed bedtimes and waketimes 2.5 hours earlier than the required 11 PM to 7 AM rule. In addition, this participant was caught filling out five days worth of questionnaires on the day the questionnaires were due. There were four participants who were accepted into the study, but did not show up for their scheduled appointments; these participants were subsequently excluded. The Sleep Hygiene Monitoring Form is comprised of 12 items that measure sleep-promoting habits the day/evening before sleep. A score of 0 reflects ideal sleep hygiene, and a score of 12 reflects extremely poor sleep hygiene. Reported compliance on the Sleep Hygiene Monitoring Form was very high for both groups on both baseline (CW group: M = 0.72, SD = 0.60; Worry group: M = 0.84, SD = 0.90), and post-intervention scores (CW group: M = 0.69, SD = 0.83; Worry group: M = 0.56, SD = 0.68). A repeated measures ANOVA investigated whether groups

differed in their adherence to sleep hygiene rules on either baseline or post-intervention Sleep Hygiene Monitoring Form scores. There were no statistically significant main effects of TIME,  $F(1,30) = 1.36, p = 0.253$ , or GROUP,  $F(1,30) = 0.97, p = 0.278$ , and no statistically significant interaction effect,  $F(1,30) = 0.87, p = 0.358$ ; thus both groups did not statistically differ in their adherence to sleep rules.

### Manipulation Verification

Research assistants who were blind to the study's hypotheses, verified whether the Constructive Worry procedure was completed correctly, by rating participants' problems and solutions as to whether they were reasonable short or long term solutions to the reported worry. This was loosely based on the Means-End Problem-Solving Procedure (MEPS; Platt & Spivack, 1975) criteria for rating solutions to problems (Appendix K). Points were also given for the participants providing at least three solutions per listed problem. This provided a crude measure of problem-solving ability, or more accurately, the problem identification and solution generation steps of formal problem-solving (D'Zurilla, 1986). Points were taken off for failing to mention three concerns/worries, and at training, anyone who had not recorded at least three worries, was instructed to be sure to include three worries for the post-intervention. The maximum possible score for problem-solving ability was 60, which indicates at least three feasible solutions per problem on each of the five post-intervention days. The average score for the CW group was high ( $M = 56.38, SD = 4.34$ ). Research assistants also recorded whether participants could generate reasonable solutions when prompted at the training session. Assistants rated solution generation ability for practice problems at the training session as either Satisfactory or Unsatisfactory. Any participants who were given an initial rating of

Unsatisfactory were re-trained, until the assistant considered their ability Satisfactory. Raters were in 100% agreement for 14 of 16 participants (88%).

Participants were also instructed to take a minimum of 10 minutes to complete the exercise and record the start and finish of their respective procedures. The CW group completed their Constructive Worry task in 14.6 minutes (SD = 1.79), and the Worry group completed their Worry Monitoring, WDQ, and PSWQ in 14.0 minutes (SD = 1.6). The means were examined using an ANOVA, to determine whether one group spent significantly less time than the other performing their worry task. The ANOVA was not statistically significant,  $F(1,31) = 1.83$ ,  $p = 0.19$ ; thus both groups were engaged in their tasks for approximately the same amount of time.

## DISCUSSION

Tests of between-group differences for demographic and baseline variables were not significant; thus, the groups did not statistically differ. The hypotheses concerning pre-sleep arousal were supported. The intervention resulted in a significantly greater decrease in overall arousal (PSAS) and cognitive arousal (PSAS COG) in the CW group, than the Worry group. The decrease in overall arousal and cognitive arousal in the CW group was statistically significant from baseline; but there was no statistically significant decrease from baseline to post-intervention in the Worry group. The constructive worry intervention was designed to address the report of increased pre-sleep cognitive arousal before bed in those with insomnia (Lichstein & Rosenthal, 1979), and the results indicate that structured problem-solving in the early evening reduces pre-sleep cognitive arousal.

The reduction in overall and cognitive arousal did not result in decreased somatic arousal or a decrease in anxiety. The intervention was meant to address pre-sleep worry and excessive mentation specifically, and state anxiety and somatic tension are separate, but related constructs (Nicassio, Mendlowitz, Fussell, & Petras, 1985). However, power estimates for anxiety and somatic arousal were low, which limits the conclusions that can be drawn from the anxiety and somatic arousal data. Unfortunately, by restricting inclusion and exclusion criteria to achieve high experimental control, it was not possible to recruit the number of participants that an a priori power analysis suggested was necessary. Future investigations would require a larger sample size to investigate this preliminary finding of no effect upon somatic tension or pre-sleep anxiety.

There was a near-perfect correlation between PSAS and PSAS COG, and a moderate correlation between PSAS and PSAS SOM, but there was no significant correlation between

PSAS SOM and PSAS COG. The extremely high correlation between PSAS and PSAS COG appears to reflect mostly the Cognitive subscale; thus it is probably more appropriate to refer to the present findings as reflecting cognitive arousal, rather than cognitive and overall arousal. This is not to say that there was not a somatic contribution to the overall PSAS score, but the PSAS scale elevation was being driven primarily by the high PSAS COG scores. Previous investigations have shown that although Cognitive and Somatic subscales are related to each other, they are also empirically distinguishable dimensions of arousal (Nicassio, Mendlowitz, Fussell & Petras, 1985). As the COG and SOM subscales are substantially independent, and most patients do not report high amounts of somatic arousal (Lichstein & Rosenthal, 1980), it is perhaps not that surprising that cognitive arousal, and not somatic arousal, was significantly affected by the intervention.

The significant reduction in pre-sleep cognitive arousal was not associated with any improvement on subjective (e.g., sleep diary), or objective (e.g., actigraphy) sleep variables. This was surprising, given the significant correlations between PSAS COG and sleep reported in other studies (Nicassio, Mendlowitz, Fussell & Petras, 1985). In the same studies of Nicassio and colleagues (1985), PSAS COG was more strongly associated with sleep indices than PSAS SOM. Unfortunately, as with anxiety and somatic arousal, power estimates for sleep variables were also low, and as such, small sample size may have prevented the detection of a significant result. There are other viable possibilities as to why a statistically significant effect was not found. Sleep has much inter-subject and inter-night variability (Speilman, Yang, & Glovinsky, 2001; see also Agnew, Webb, & William, 1966; Hauri & Olmstead, 1989), so it is possible that a 2-day baseline period and a 5-day post-intervention period was simply not enough to establish a stable picture of the individual's sleep, thereby making it difficult to detect any effects. Large-

scale insomnia outcome studies typically observe patients for two baseline weeks, two to four post-intervention weeks, and a later follow-up period of two weeks. Unfortunately, working with an undergraduate population where the allocation of large amounts of extra course credits are restricted, compliance with sleep hygiene rules over long periods of time is typically low, and long periods during which no examinations occur are rare, necessitated an observation period of only one week. Future studies should investigate the effect of this procedure on sleep variables with much longer observation periods.

The finding that changes occurred in pre-sleep cognitive arousal, but not sleep indices, implies that pre-sleep arousal may have little or no effect on sleep disruption. Pre-sleep cognitive arousal has often been strongly linked to sleep variables like sleep onset latency (Nicassio, Mendlowitz, Fussell & Petras, 1985; Sanavio, 1988; Wicklow & Espie, 2000), while other studies have not (Haynes, Adams, & Franzen, 1981). The present study would not support a significant relationship between cognitive arousal and rated sleep, although this conclusion is tempered by the fact that the sleep analyses were under-powered.

Some have suggested that cognitive arousal is associated with the subjective experience of insomnia, because pre-sleep cognitions are significantly related to the discrepancy between lower objective (PSG) sleep onset latencies and higher subjective (Sleep Diary) sleep onset latencies (van Elgeren, Haynes, Franzen, & Hamilton, 1983). However, at baseline both groups reported elevated pre-sleep cognitive arousal, but only the baseline mean sleep diary indices indicated the presence of insomnia, whereas the objective actigraph variables did not. This provides some support for the idea that pre-sleep cognitive arousal is related to the subjective experience of insomnia, although not enough to produce to perceived sleep improvement with pre-sleep cognitive arousal decreases.

One possible explanation for the inconsistent relationship between pre-sleep cognitive arousal and sleep is that cognitive and/or somatic arousal in patients may be experienced differently across individuals, and this may have differential effects on their sleep (Spielman, 1986). The nature of the cognitive arousal may also be important, and different for many patients. For example, in a study where patients wore actigraphs and their pre-sleep thoughts were audiotaped, many different types of sleep-interfering cognitions were reported, but only some of them correlated with Sleep Diary or actigraph SOL (Wicklow & Espie, 2000). Certain types of interfering cognitions may be important for disturbing sleep in some individuals, but not in others. The thresholds for when a particular type of stimulus interferes with sleep (whether environmental, somatic, worry-related, or non-anxiety provoking mentation) may differ from person to person. The present study targeted worry cognitions only, which may not have been key in improving sleep for this particular sample. This would match current insomnia assessment approaches that assume multiple individual causes in a patient's insomnia (Spielman, 2001).

One consideration in interpreting these findings is the generalizability of the results to other people with insomnia. The present sample was highly selected, a young, college undergraduate sample, mainly female and Caucasian. The participants were without extreme circadian tendency, without additional sleep disorders, and without mood, anxiety, or alcohol abuse disorders. They reported that they were not taking any psychoactive medications during the experiment. It is nearly certain that the sample does not fully represent the large majority of those with insomnia, though they did indeed suffer a relatively pure form of the disorder. Thus, this restricted sample, recruited to enhance experimental control, may limit the generalizability of conclusions drawn from the present investigation. Future investigations will be needed to

determine whether these results extend to a broader, more representative sample of persons with clinically significant insomnia.

One could argue that if there is no effect on sleep, why bother treating pre-sleep cognitive arousal? Insomnia itself is a complex construct with many etiologies and cognitive-behavioral treatment has several components to address these multiple factors. Current efficacy estimates suggest that between 69-80% report subjective or EEG-verified improvements in sleep (Morin et al., 1999) using combinations of Sleep Restriction, Stimulus Control, PMR, Sleep Hygiene, and psychoeducation. One rationale for treating pre-sleep arousal is the complaint that excessive mentation and worry before bed is troubling to those with insomnia (Broman & Hetta, 1994; Lichstein & Rosenthal, 1980), and in this study, adding a cognitive treatment for these patients helped with this specific complaint. Some authors argue that insomnia rarely has complications other than discomfort, and since it is such a subjective condition, the goal of treatment should be to improve the subjective discomfort associated with it (Stepanski, 2001). In addition, some patients report not liking behavioral treatments like Sleep Restriction, and may opt for pharmacological treatments that present greater risk, are potentially habit-forming, and can actually perpetuate insomnia (Kripke, 2000; Kripke et al., 1998). Thus, consumer satisfaction with CBT treatments for insomnia may improve by adding an element like Constructive Worry. Satisfaction with treatment is not a trivial matter, as it affects adherence and dropout rates (Vincent & Lionberg, 2001). The present investigation supports the idea that the pre-sleep cognitive arousal complaint of those with insomnia can be treated, thereby providing a preliminary step for future investigations to test a CBT plus Constructive Worry treatment package, against CBT only, and a control group, to see whether CBT + CW produces (1) superior improvements and, (2) superior consumer satisfaction. Lastly, although not formally

tested yet, some have suggested that cognitive arousal be investigated as a potential predictor of future insomnia episodes, just as insomnia is a powerful predictor of future depressive episodes (Breslau, et al., 1996; Chang et al., 1997; Ford & Kamerow, 1989; Mellinger, Balter, & Uhlenhuth, 1985). If this were true, including a cognitive arousal component would be important.

The multiple regression analysis results suggest that low appraisal of one's personal control, low confidence in one's ability to solve problems, and a tendency to avoid problem situations, predict an increased degree of pre-sleep arousal. Similarly, low levels of pre-sleep arousal are predicted by a positive appraisal of one's personal control in problem situations, high confidence in one's ability to solve problems, and a tendency to approach problem activities. Underestimation of problem-solving abilities has been associated with feelings of being overwhelmed by problems (Heppner, & Peterson, 1982; Nezu, 1986) and decreased ratings of benefit from problem-solving training (Heppner, Baumgardner, Larson, & Petty, 1983). This is the first investigation of pre-sleep arousal and self-appraisal of problem-solving abilities with a sample of those with insomnia, and it has possible treatment implications for those complaining of pre-sleep worries, that should be further investigated in those with insomnia. For example, improving problem-solving appraisal with something like problem-solving feedback, may maximize the benefit from the Constructive Worry procedure.

## CONCLUSION

Like other studies, the participants reported that pre-sleep cognitive arousal was a factor in their insomnia, or more specifically, worrying and problem-solving kept them awake at night, and they worried that poor sleep may affect their functioning the next day. The use of a Constructive Worry procedure in the early evening produced a decrease in pre-sleep cognitive arousal in a group of nonpsychiatric undergraduate students with insomnia. In the insomnia literature, pre-sleep cognitive arousal has received equivocal support for its role in insomnia. Its association with sleep appears to vary depending on sample size and statistical power of the study, what type of cognitive activity is reported (e.g., worry versus concern about sleep loss versus general mental activity), and whether PSG, actigraph, or Sleep Diaries are used as dependent variables. The one consistent finding is the report from those with insomnia that pre-sleep cognitive arousal is a problem for them, and the appraisal that it primarily causes their insomnia. The findings from this study, in combination with the current literature suggest there may be individual differences in what type of arousal affects sleep. Since the present Constructive Worry procedure decreased reported levels of the complaint of cognitive arousal in those with insomnia, this suggests its utility can now be tested as a component in current insomnia treatments.

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APPENDIX A  
CONSENT FORM

**1. Study Title:**

A Structured Problem-Solving Procedure and Subjective Sleep  
Quality in Undergraduates with Poor Sleep

**2. Performance Site:**

201 Audubon Hall

**3. Contacts:**

William F. Waters, Ph.D.     761-5852 anytime

Colleen E. Carney, M.A.     578-4229 Monday, Wednesday, Friday

**4. Purpose of the Study:**

To investigate worrying before bedtime and its effect on subsequent  
Sleep patterns.

**5. Subjects:**

**A. Inclusion Criteria**

In order to participate in the study, subjects must be undergraduates at Louisiana State University and between the ages of 18 and 35 years old. In addition, subjects must meet International Classification of Sleep Disorders criteria for insomnia.

**B. Exclusion Criteria**

Those meeting Diagnostic and Statistical Manual, fourth edition (DSM-IV) criterion Psychiatric disorders according to the PRIME MD screening tool; those meeting International Classification of Sleep Disorders criteria for a sleep

disorder (except for insomnia); those currently taking psychoactive medications and those scoring as an extreme morning or extreme evening person according to the Horne and Ostberg Morningness-Eveningness Questionnaire will be excluded. It takes approximately 30-45 minutes to complete these measures.

**C. Maximum number of subjects: 60**

**6. Study Procedures:**

This study requires that you follow sleep hygiene rules for 7 days: refrain from naps, maintain an 8-hour sleep schedule with a bedtime of 11 PM plus/minus one hour and a wake time of 7 AM plus/minus one hour; avoid strenuous exercise within two hours before bedtime; and avoid large meals or caffeinated beverages within three hours of bedtime. Each night you will complete a Pre-Sleep Arousal Questionnaire and a State Trait Anxiety Inventory, and each morning you will complete a sleep diary, which asks you to reflect upon the quality of the previous night's sleep and a Sleep Hygiene Monitoring form to make sure you are following the rules. Each morning you are to drop off the completed questionnaires at the sleep lab, except for Saturday and Sunday morning; you must call the experimenter if there is some reason (e.g., medical excuse) that you cannot drop-off your measures. The two morning questionnaires and the two evening questionnaires require 5 minutes each to complete them. In addition, you will be required to wear an ACTITRAC motion detector. The ACTITRAC is a small motion detection device that is worn like a wristwatch and verifies whether you are active or at rest. It is a sturdy instrument and each unit is under warranty, so there should not be any problem with damage, however, you

will be accountable for any damage due to your negligence. The study requires that attend a worry instruction training session on the third day of the study, this will take a maximum of 30 minutes to complete. From that night onward, you will have to focus upon worries between the hours of 6-8 PM, and complete a worry-related exercise. You will also have to complete a Pre-Sleep Arousal Questionnaire and a State Trait Anxiety Inventory within minutes before your bedtime, and within minutes of waking each morning, you will complete a sleep diary, which asks you to reflect upon the quality of the previous night's sleep and a Sleep Hygiene Monitoring form to make sure you are following the rules. On the following Monday morning, bring the completed questionnaires and ACTITRAC to the sleep lab in room 201 Audubon before 10 am to can receive your extra credit.

**7. Benefits:**

You will be compensated for your time with 15 extra course credits and you will be contributing to our scientific understanding of sleep and worry.

**8. Risks/Discomforts:**

You will be asked to complete a number of initial questionnaires that ask searching questions concerning your personality, mood and habits. Some persons could find these questions disturbing. Some people may find that focusing on worry may make their worry worse. The focus upon worry may affect your sleep adversely. The accidental release of personal information could be damaging to a person's reputation. Those who do not like to wear items on their wrists, like

wristwatches, may find the ACTITRAC unpleasant. If the ACTITRAC is lost or damaged beyond repair, due to an act of negligence, you will assume the cost of the repair/replacement of the ACTITRAC unit. It costs \$500 to replace an ACTITRAC unit. If you are accustomed to sleeping for periods longer than 9 hours per night, the sleep hygiene rules may produce fatigue or irritability.

**9. Measures taken to reduce risk**

Should your sleep become more disturbed and remain disturbed for one week beyond the study, you will be offered a free time-limited insomnia treatment at the Louisiana State University Psychological Services Center. The primary investigators are master's level clinical psychology graduate students and are trained to handle potentially sensitive material with confidentiality and sensitivity.

**10. Right to Refuse:**

You may withdraw from the study at any time, however, you will only receive full course credit upon completion of all aforementioned study requirements (7 sleep diaries, 7 State Trait Anxiety Inventories, 7 Pre-Sleep Arousal Scale, 7 Sleep Hygiene Monitoring forms, 5 Worry Monitoring packets compliance with sleep hygiene rules and all measures and the ACTITRAC are returned to the lab the following Monday before 10 am).

**11. Privacy:**

This study is not anonymous (i.e., there is a code linking data to identity). Results of this study may be published, but no names or identifying information will be included in the publication. All personal information obtained in this study will be kept confidential unless release is legally compelled. Both included and

excluded participants will be assigned a random number and this number will be the only link between your name and your responses. Your forms will be kept in locked filing cabinet.

**12. Financial Information:**

You will be given 15 extra credit points upon submission of completed measures and the ACTITRAC. Extra credit will expire in one year from the date of issue.

**13. Withdrawal:**

You may withdraw from the study at any time without penalty; please contact the experimenter at the phone number above and inform them you will not complete the study, so that arrangements can be made to retrieve unused measures and the ACTITRAC. All participants will receive 2 extra credit points for completing the screen portion of the study, however, you will only receive full course credit (the remaining 13 credits) upon timely submission of all completed measures and the ACTITRAC.

**14. Removal:**

The investigator may remove subjects from the study without their consent if they violate any of the study requirements (e.g., failure to keep appointment, failure to return measures at the specified times, or failure to complete measures).

**Part 5: Signatures:**

‘The study has been discussed with me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigators. If I have questions about subjects' rights or other concerns, I can contact Robert C.

Mathews, Chairman, LSU Institutional Review Board, (225) 578-8692. I agree to participate in the study described above and acknowledge the researchers' obligation to provide me with a copy of this consent form if signed by me.'

Subject Signature      Date

APPENDIX B  
DEMOGRAPHIC FORM

Subject # \_\_\_\_\_

Sex: F \_\_\_\_\_ M \_\_\_\_\_

Age: \_\_\_\_\_

Marital Status: Single \_\_\_\_\_ Married \_\_\_\_\_ Co-Habiting \_\_\_\_\_

Race: Caucasian \_\_\_\_\_ African American \_\_\_\_\_ Asian \_\_\_\_\_

Occupation: \_\_\_\_\_

Level Of Education:

Below Highschool \_\_\_\_\_ Highschool Graduate \_\_\_\_\_ Some College \_\_\_\_\_

College Graduate \_\_\_\_\_

Are you currently taking any medication prescribed for emotional problems (antidepressants, anti-anxiety medication etc...)? \_\_\_\_\_

## APPENDIX C

### SLEEP HYGIENE AND ACTIGRAPHY INSTRUCTIONS

During the course of this three-day study, you will be required to follow the following sleep rules. If you deviate at all from these rules, please contact the experimenter and make a note of which items you did not complete, so that your eligibility can be assessed. You are free to withdraw from this experiment at any time without penalty, however you will only receive course credit upon completion of all the requirements. You will also be required to wear an ActiTrac to verify your daily activities. The ActiTrac looks like a wristwatch and simply provides the experimenter with a reading of whether you are complying with sleep hygiene rules. The following is a list of behaviors that encompass your sleep rules:

1. Absolutely NO daytime napping
2. Bedtime is 11 PM (there is a 1 hour grace time, but you must adjust your wake time accordingly. For example, if you go to bed at 12 AM, wake up at 8 AM, instead of 7 AM. You should be in bed trying to sleep for 8 hours.) It is very important that you are going to bed and rising at the same time each day.
3. Wake time is 7 AM (your wake time and bedtime should be 8 hours apart). It is crucial that you get out of bed within the specified amount of time, so set an alarm.
4. No strenuous exercise within 2 hours of bedtime
5. No caffeinated beverages within 2 hours of bedtime or large meals within 3 hours of bedtime. Please be advised that you may not be aware of some product's caffeine-content. For example, Mountain Dew, Mountain Dew, Coca-Cola, cold remedies, diet pills like Dexatrim, diuretics like Aqua Ban, iced tea/hot tea, chocolate, Excedrin and Midol Menstrual relief all contain caffeine. Please avoid them.

APPENDIX D

SLEEP HYGIENE MONITORING

Subject #: \_\_\_\_\_

Date: \_\_\_\_\_

For each of the following, mark Y for yes if you engaged in that activity or had that experience yesterday. If you did not, mark N for no.

- \_\_\_\_\_ 1. Took a nap
- \_\_\_\_\_ 2. Went to bed between 10 PM and 12 AM
- \_\_\_\_\_ 3. Woke up between 6 AM and 8 AM
- \_\_\_\_\_ 4. Slept more than 7 or 8 hours
- \_\_\_\_\_ 5. Smoked within 2 hours of bedtime
- \_\_\_\_\_ 6. Exercised strenuously within 2 hours of bedtime
- \_\_\_\_\_ 7. Used sleep medications (prescriptions or over-the-counter)
- \_\_\_\_\_ 8. Consumed caffeinated products within 3 hours of bedtime
- \_\_\_\_\_ 9. Had a large snack or a meal within 2 hours of going to bed
- \_\_\_\_\_ 10. Drank more than 2 ounces of alcohol within 2 hours of bedtime
- \_\_\_\_\_ 11. Sleep was significantly disturbed by light, noise or bed partner
- \_\_\_\_\_ 12. Drank 8 ounces or more of fluid at bedtime

APPENDIX E

DAILY SLEEP DIARY

Subject #: \_\_\_\_\_ Date: \_\_\_\_\_

Please respond to these questions soon after you wake up for the day.

1. At what time did you first try to fall asleep? \_\_\_\_\_
2. Approximately how many minutes did it take you to fall asleep? \_\_\_\_\_
3. What time did you wake-up to start the day? \_\_\_\_\_
4. What was the total number of hours and minutes you slept last night? \_\_\_\_\_
5. How many times did you wake in the middle of the night for 5 minutes or more? \_\_\_\_\_
6. About how much time total were you awake after you first fell asleep? \_\_\_\_\_
7. How difficult was it for you to fall asleep last night:  
Not Difficult 1    2    3    4    5 Extremely Difficult
8. Rate the quality of last night's sleep:  
Excellent 1    2    3    4    5 Very Poor
9. What was your level of physical tension when you went to bed last night?  
Extremely Relaxed 1    2    3    4    5 Extremely Tense
10. Rate your level of mental activity when you went to bed last night.  
Very Quiet    1    2    3    4    5 Very Active
11. How rested do you feel this morning:  
Very Rested 1    2    3    4    5 Poorly Rested

## APPENDIX F

### CONSTRUCTIVE WORRY INSTRUCTIONS

Worry is the excessive and poorly timed use of problem solving skills. When we have problems, we tend to use our problem solving skills to make our lives better and relieve ourselves of anxiety. We are strongly rewarded throughout our lives for dealing with problems by finding solutions for them; some of the reward is a reduction in anxiety, and some is the approval we get for dealing with our problems effectively. It is therefore not surprising that some of us may use our problem solving skills to excess and at the wrong times and places, namely bedtime.

The most inappropriate place for worry is in bed, either at bedtime or while lying awake in bed after having awakened during the night. We may think about a problem, trying to solve it adaptively, but unfortunately, the anxiety evoked by the problem will keep us awake.

Constructive worry is a method for reducing the tendency to worry during that quiet time when sleep is supposed to be taking over. During the early evening some time between two and four hours before bed devote fifteen minutes to do this exercise. It is done as follows:

1. Think of the three to five problems facing you that have the greatest likelihood of keeping you awake at bedtime, and list them in the “Problems” column.
2. For the first problem, think of the next step you might take to contribute to its resolution.

Write it down in the “Solutions” column. This need not be the ultimate solution to the problem, since most problems have to be solved in a step-wise fashion anyhow, and you will be doing this again tomorrow night and the night after until you finally get to the best solution. If you know how to resolve the problem completely, then write that down. If you

decide that this is not really a significant problem, and you will just deal with it when the time comes, then write that down. If you decide that you simply do not know what to do about it, and need to ask someone to help you, write that down. If you decide that it is a problem, but there seems to be no practical solution at all, and that you will just have to live with it, write that down, with a note to yourself that maybe sometime soon you or someone you speak with will give you a clue that will lead you to a solution.

3. Do this for each of the problems you have listed (you should have a minimum of three problems).
4. Fold the Constructive Worry sheet in half and place it on the nightstand next to your bed and forget about it until bedtime.
5. At bedtime, if you begin to worry actually tell yourself that you have dealt with your problems already in the best way you know how, and when you were at your problem solving best. Remind yourself that you will be addressing them again tomorrow evening and that nothing you can do while you are so tired can help you any more than you have already done; more effort will only make matters worse. Do this procedure early evening, do not do it any less than two hours of bedtime, as the closer to bedtime you engage in problem solving the more likely you are to be anxious and worried at bedtime.
6. An additional benefit of Constructive Worry may be less anxiety during the daytime.

APPENDIX G

CONSTRUCTIVE WORRY WORKSHEET

Subject # \_\_\_\_\_

Date: \_\_\_\_\_

Time: \_\_\_\_\_

Problems	Solutions
Record a min. of 3	Use other side if necessary
1.	1.  2.  3.
2.	1.  2.  3.
3.	1.  2.  3.

## APPENDIX H

### WORRY SELF-MONITORING FORM

Please complete this form between 6 and 8 PM and record the time you started and stopped the exercise below. We all have worries ranging from mundane to serious matters every day. Please record any specific worries you may have had today below (minimum of three worries).

Worries:

1.

2.

3.

4.

## APPENDIX I

### FORMAL EXCLUSION CRITERIA

#### Sleep criteria:

- \_\_\_ TST of less than 6 or more than 9 hours on more than 3 nights per week
- \_\_\_ bedtime before 9 PM or after 1 AM more than 3 nights per week
- \_\_\_ daily naps of 30 minutes or more in length on more than 5 days per week
- \_\_\_ endorsement of critical items for sleep apnea, restless leg syndrome/periodic leg movement, shift work, sleep restriction or narcolepsy

#### Drug criteria:

- \_\_\_ Antidepressants (TCA, SSRI, MAO-I)
- \_\_\_ Anxiolytic or Barbituates (valium, librium, ativan, xanax)
- \_\_\_ Hypnotic (ambien, halcion, prosom, doral)
- \_\_\_ Antipsychotic (thorazine, haldol, risperdal)
- \_\_\_ Antihistamines
- \_\_\_ Antidyskinetic/Anticholinergic (cogentin, artane, symmetrel)
- \_\_\_ Anti-Obsessional (clomipramine)
- \_\_\_ Stimulants (ritalin, dexedrine, diet pills, non-drowsy cold remedies, excedrin, diuretics)
- \_\_\_ Mood stabilizers (lithium, depakote, tegretol)
- \_\_\_ Narcotics

## APPENDIX J

### SLEEP DISORDER EXCLUSION CRITERIA

1. Sleep apnea: endorsement of “stopping breathing”, or “gaspingsnorting” with “heavy snoring” on fifteen or more nights per month (item 12), plus endorsement of “falling asleep involuntarily while unstimulated or stimulated” or “trouble functioning during the day” (item 10).
2. Narcolepsy: endorsement item 15 (naps must be under half an hour, and restorative) and item 16 or 17, plus endorsement of “falling asleep involuntarily while unstimulated or stimulated” or “trouble functioning during the day” (item 10).
3. Idiopathic hypersomnia: endorsement of item 11 and endorsement of “falling asleep involuntarily while unstimulated or stimulated” or “trouble functioning during the day” (item 10).
4. Periodic limb movement disorder: endorsement of item 14a and 14b.
5. Restless leg syndrome: endorsement of item 13a and 13b.
6. REM sleep behavior disorder: endorsement of item 19 about 10X/month.
7. Shift Work: endorsement of shift work (item 31) on 3 or more nights per week.
8. Sleep disorder secondary to pain: endorsement of pain (item 30) “often” or “most” of the time.
9. Gastroesophageal reflux: endorsement of item 23 on 3 or more nights per week.

## APPENDIX K

### PROBLEM SOLVING RATING SYSTEM

Maximum score is 60 (Maximum of 12 points per day for 5 post-test days)

#### Problem Identification

Give 1 point for each identified problem (maximum of 3). Assign 0 if the problem is vague. For example, if someone writes “cat” as a problem, cat is vague; they have to specifically identify a concern, e.g., “I’m worried the cat may die because she’s sick”.

#### Solution Generation

Assign 1 point if the solution represents one conceivable step in the means to reach a stated goal (whether it is an intermediate step, an initial/first step, or the ultimate/last step). Maximum of 3 points per problem, and 9 points per day.

Assign 1 point per solution for a:

- (1) feasible final step
- (2) feasible “next” or intermediate step
- (3) acknowledgement that there is nothing they can do about it at this moment and/or they will try to solve the problem at a later date

Assign 0 points if the solution is vague, irrelevant, or there is not a solution given.

## VITA

Colleen Elizabeth Carney was born on April 14, 1971, in Toronto, Ontario. She graduated from the University of Toronto in 1997 with a Bachelor of Science degree in psychology, and graduated from Louisiana State University in 2001 with a Master of Arts degree in psychology. Ms. Carney is currently completing a predoctoral clinical internship in psychology at the Grand River Hospital, Kitchener, Ontario. Her area of research is the cognitive mediation of mood and sleep. Her doctoral supervisor was William F. Waters, Ph.D., a board-certified specialist in the area of sleep disorders and psychophysiology. She will earn the degree of Doctor of Philosophy in psychology in the fall of 2003.