The Relations Between Stress, Mood and Insomnia.

Susan Rubman

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The relations between stress, mood and insomnia

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THE RELATIONS BETWEEN STRESS,
MOOD AND INSOMNIA

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree
of Doctor of Philosophy

in

The Department of Psychology

by
Susan Rubman
B.S., Union College, 1984
M.A., Louisiana State University, 1986
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Abstract

Stress is hypothesized to cause increased cognitive and physiological arousal which is incompatible with sleep. Major life events have been implicated as a significant factor in the onset of chronic insomnia (Healey et al., 1981), and there is some suggestion that minor life events influence symptoms as well (Haynes et al., 1981). The present study was designed to evaluate and further clarify the role of daily minor stressors and mood variables on sleep in both normal sleepers and insomniacs.

Subjects with insomnia secondary to medical or physical disorders were excluded from this study. Fifty four adult subjects (20 mixed-complaint insomniacs, 17 sleep onset insomniacs, and 17 normal sleepers) volunteered from the community. Each subject completed the Daily Stress Inventory, the State-Trait Anxiety Inventory (State Form) and the Zung Self-rating Scale for Depression each night between supper and retiring. They completed a Daily Sleep Diary in the morning upon awakening, and mailed back completed forms daily. In this manner, 21 consecutive days of monitoring were collected for each subject.

Stress was divided into two conditions; the seven days with the highest stress scores for each individual became the high stress condition, and the seven days with the lowest stress scores became the low stress condition for each individual. Results comparing sleep disturbances on those days indicated that under relatively high stress conditions, subjects slept fewer minutes, achieved a lower quality of sleep and lower sleep efficiency than they did in the low stress
condition. There was a significant group by stress interaction such that all insomniacs slept fewer minutes under the high stress condition than under the low stress condition, but normal subjects showed no change across stress conditions. Regression analyses indicated that frequency of daily minor stressors was predictive of latency to sleep onset and difficulty falling asleep. Depression scores were predictive of latency to sleep onset, difficulty falling asleep, restedness upon awakening and sleep efficiency. Depression scores were substantially more predictive of dependent sleep measures than anxiety scores were. Results lend modest support to the hypothesis that stress contributes to symptoms of insomnia. Additional theoretical and clinical implications are discussed.
Although waking, not sleeping, is the predominant state for humans, sleep occupies approximately one third of our lives. Further, all known animals are thought to have regular cycles of activity and rest (Wheatley, 1981). The absolute function of sleep has not yet been ascertained; there are many theories, but none are definitive. What has been demonstrated, however is that our "waking performance, vigilance and vigor, health and happiness, the whole quality of our waking lives are profoundly affected by the way we sleep" (Parkes, 1985; p. 5). Consequently, a problem with sleep is frequently associated with other problems. Investigation into the nature of dysfunctional sleep, then certainly appears warranted.

Current trends in the research examining etiological hypotheses of sleep disturbances have suggested that stress may play a causal role in insomnia. Given that stress has recently been implicated in a variety of psychological and physiological disorders, this is a reasonable hypothesis, particularly if insomnia is conceptualized as a specific case of physical symptoms. Furthermore, the application of a stress-illness relation theory enables the incorporation of mood variables as possible intermediaries in the stress-sleep disturbance hypothesis. To date, however, relatively little research has been conducted to assess these relations.

The purpose of this paper is to present an examination of some of the current thought regarding the roles of stressful events and mood in the etiology of sleep disturbances. First, the clinical description and incidence rates of insomnia will be presented. This
will provide the background necessary for a thorough exploration of the major assessment and treatment techniques utilized with insomnia. This will be followed by a review of the major psychological hypotheses considered to be etiologically related to complaints of insomnia. This will also provide a rationale for the investigation of the use of major and minor life events and mood in the assessment of sleep disturbances. The second major section of this paper will examine the concept of stress and the methodological issues regarding its measurement. Finally, recent literature will be presented regarding the relation of stress to psychological and physical symptoms which serves as part of the rationale for this study.

Clinical Description and Diagnosis of Insomnia

Since very little is known about the function of sleep, the precise implications of lack of sleep are difficult to determine. Typically, individuals who have been either experimentally or unintentionally sleep deprived show deficits in several realms of functioning following these episodes. The initial effect of total sleep deprivation appears to be an increase in sleepiness, rather than any decline in mood, performance or behavior (Horne, 1978). However, following prolonged sleep deprivation much more serious consequences, such as impairment in stereoscopic vision, may result for the individual (Parkes, 1985).

The nature of sleep disorders themselves is varied, and they may take one (or several) of many forms. A sleep disorder usually refers to one or more of the following:

(a) a response or pattern of behavior during sleep
or associated with sleep that differs from the response and pattern observed in the majority of the population; (b) a complaint attributed to inadequate or excessive sleep; (c) a dysfunction in the process governing sleep; or (d) a pathology tied directly to sleep (Coates & Thoresen, 1983, p. 240). Many classification schemes have been attempted categorizing disorders on the basis of a variety of phenomena including theoretical etiologies, symptom clusters and physiological data. Categorization of sleep disorders thus has been quite controversial.

Early classification issues involved the absolute length of time asleep (i.e., long vs. short sleepers). It was gradually acknowledged that sleep duration was not the sole criterion by which a diagnosis of a sleep disorder could be made. Although adults, on the average, require 7 1/2 hours of sleep per night, individuals vary greatly in the actual amount of sleep that they require (Parkes, 1985; van Oot, Lane & Borkovec, 1984). Consequently, diagnosing a sleep disorder by this criterion alone was invalid and was discarded.

A somewhat more salient and useful set of criteria was subsequently proposed to classify sleep as "good" vs. "poor". The differentiating features of these categories involved length of time to sleep onset, "soundness" or depth of sleep, and feelings of being rested upon awakening (Monroe, 1967). Good sleepers had shorter sleep onset latencies, slept more soundly and reported feeling more rested upon awakening than poor sleepers. Current classification of disorders involving the initiation and maintenance of sleep is essentially based on these factors.
Most recently, the Association for Sleep Disorders Centers (ASDC, 1979) has devised a comprehensive medical-model classification scheme for all sleep disorders (Hartmann, 1985). This paradigm separates disorders into four major diagnostic categories. The first and most frequently used category is Disorders of Initiating and Maintaining Sleep (DIMS), or insomnia, sleep apnea, myoclonus and restless legs syndrome. The second category is Disorders of Excessive Somnolence (DOES), or the hypersomnias (e.g., narcolepsy). The third category is Disorders of the Sleep-Wake Schedule (e.g., "Jet lag", shift-work sleep problems), and the fourth category is Dysfunctions Associated with Sleep, Sleep Stages or Partial Arousals; the parasomnias (e.g., sleep walking, sleep-related bruxism). The remainder of this paper will focus almost exclusively on DIMS, especially insomnia.

There are two different types of complaints associated with insomnia; difficulty falling asleep, or sleep onset insomnia; and difficulty remaining asleep, or sleep maintenance insomnia. Sleep maintenance insomnia may be further defined as awakening multiple times during the night and having difficulty falling back to sleep, or early morning awakening, or both. These two complaints are combined under the broader heading of sleep maintenance because they generally occur together (Hartmann, 1985). Sleep onset insomnia is generally regarded as the most common sleep complaint. Individuals may complain of both sleep onset and sleep maintenance symptoms.

The ASDC has made provision for classifying the insomnias as transient or chronic; associated with psychiatric disorders; and secondary to the use of drugs and alcohol. The symptom of insomnia may also be associated with a variety of physical, metabolic or medical
conditions such as ulcer pain, angina, chronic renal insufficiency, and Parkinsonism. In these cases insomnia is considered secondary to the physical disorder. Although there is great controversy surrounding the actual criteria for a diagnosis of insomnia, most researchers and clinicians agree with the following guidelines; at least the subjective complaint of insomnia, sleep onset latency of 30 minutes or more, 30 minutes or more spent awake during the night after sleep onset, and/or less than 6 1/2 total hours of sleep in a night. Furthermore, these symptoms occur at least three nights per week (Lacks, 1987). Some researchers (e.g., Haynes, Adams & Franzen, 1981) then further distinguish severity (mild vs. severe) of the disorder based primarily on latency to sleep onset and/or total time awake during the night.

A final consideration in the description and diagnosis of insomnia is the issue of subjective versus psychophysiological insomnia. The ASDC provides for these conditions as subcategories of DIMS. Psychophysiological insomniacs show distinct polysomnographic (EEG) abnormalities either at sleep onset or during sleep maintenance. Psychophysiological insomnia is thought to account for up to 75% of insomniacs (van Oot et al., 1984) and may be multiply determined. The ASDC suggests that this phenomenon frequently is associated with chronic somatized tension and anxiety. Subjective insomniacs show no objective abnormalities in sleep. It has been suggested that either no abnormalities exist, or technology has not developed to the point that we can measure the extant abnormalities (Bootzin & Engle-Friedman, 1981; de la Pena, 1978; Lacks, 1987). All types of insomnia and its subtypes are notable for their heterogeneity.
Prevalence of Insomnia

Complaints of insomnia may be second only to headaches as a health problem in the United States (Coates & Thoresen, 1977). In a nationally representative probability sample of noninstitutionalized adults aged 18-79 years (N = 3161), 35% indicated that insomnia (sleep onset insomnia, sleep maintenance insomnia, or both) had been a problem during the past year (Mellinger, Balter and Uhlenhuth, 1985). Further, 17% of those individuals reported that the problem was serious. Approximately half of the national sample reported that insomnia had never been a problem. Twelve percent of individuals surveyed indicated that insomnia had been a problem at one time, but not in the past 12 months. Of those individuals who complained of serious insomnia, approximately one third appeared to be sleep onset insomniacs, one third appeared to be sleep maintenance insomniacs, and one third appeared to have mixed complaints.

Similar results were obtained in a large scale survey of the Los Angeles metropolitan area. Subjects were selected to match the 1970 census of the area (N = 1006). It was determined that insomnia was the most common sleep complaint; 42.5% of the entire sample reported that they had either experienced some type of insomnia in the past or were currently experiencing the problem (Bixler, Kales, Soldatos, Kales, & Healey, 1979). Furthermore 32.2% of the total sample was experiencing insomnia at the time of the survey, and 43.5% of those individuals with current complaints of insomnia had had the complaint for 1-5 years.

Incidence estimates (e.g., Hauri, 1982) suggest that each year approximately 10 million Americans will consult with a physician about
sleep problems. In general, women are more likely than men to report complaints of insomnia. Further, age is positively related to insomnia. All surveys report that sleep complaints increase as individuals grow older. However, the type of complaint tends to change with age, younger adults are more likely to report sleep onset problems, while older adults are more likely to report sleep maintenance problems. In addition, complaints of insomnia frequently are also reported by individuals who work late or swing shifts (Bixler et al., 1979; Parkes, 1985). Possibly related to, or confounded with this is the finding that socioeconomic status and level of education have been found to be inversely related to complaints of insomnia. Finally, insomnia is quite frequently associated with a variety of physical disorders (Bixler et al., 1979; Johns, Gay, Masterson, & Bruce, 1971; Parkes, 1985).

**Stages of Sleep**

Human sleep is more than simply the absence of wakefulness; it is a complex process characterized by changes in both behavioral and physiological activity. The normal adult typically experiences a regular, predictable sequence of changes throughout the night. Electrophysiological measurement is usually employed to define these changes more objectively. Common assessment tools include electroencephalograms (EEG), electrooculograms (EOG) and electromyograms (EMG). The term "sleep architecture" refers to the pattern of changes in these variables. Broadly speaking, these fluctuations are separated into two categories; Rapid Eye Movement (REM) sleep and Non Rapid Eye Movement (NREM) sleep.

In the transition from wakefulness to sleep,
electroencephalographic (EEG) measurements typically show signs of
deepening relaxation; wave frequency slows and amplitude increases.
The first signs of sleep are the appearance of theta waves (waves
with a frequency between 4-7 Hz) and a decrease in the presence of
alpha waves (which are associated with a state of relaxed
wakefulness). Stage 1 typically shows mixed frequencies, low voltage
and little rhythmicity. Technically, stage 1 sleep occurs when greater
then 50% of a 30 second EEG epoch is theta and alpha waves are
absent (van Oot, et al., 1984). Slow rolling and horizontal eye
movements can be observed at this time. Responsiveness to external
stimuli diminishes and many individuals aroused at this point report
being half awake rather than asleep (Lacks, 1987; Parkes, 1985).
Stage 1 sleep lasts from 30 seconds to 7 minutes.

Stage 2 sleep is generally thought to represent the first level of
"true," unequivocal sleep (Lacks, 1987; Parkes, 1985; Rechtshaffen &
Kales, 1968). It is characterized by stage 1 frequencies plus the
occurrence of either sleep spindles or K complexes. Sleep spindles are
bursts of rhythmic 12-15 Hz waves which last 0.5 to 3 seconds. Sleep
spindles are so named because the waves in the middle of each spindle
are larger than the waves at either end (resembling an actual
spindle). Spindles occur approximately 5 times each minute in normal
stage 2 sleep and may occur throughout stages 2-4.

K complexes are sudden high amplitude spikes which are
characterized by an initial negative wave followed quickly by a
positive wave. They occur 2-3 times per minute and may appear
singly or in small groups. They are frequently associated with sleep
spindles. K complexes can occur both spontaneously and in response
to some external stimulus. Meaningful auditory stimuli are said to produce K complexes more readily than nonmeaningful stimuli (Oswald, 1962). Some researchers contend that K complexes are sleep-preserving mechanisms which denote a wave of inhibition, preventing arousal in response to environmental sounds (Lacks, 1987; van Oot, et al., 1984). The first K complex or sleep spindle is generally thought to be the first objective marker of sleep. Individuals awakened after this point will usually report having been asleep (Lacks, 1987; Parhes, 1985; van Oot, et al., 1984).

Stage 3 sleep is distinguished by further increases in wave amplitude and decreases in wave frequency. Technically, stage 3 is defined when delta waveforms (0.5-3 Hz) constitute 20-50% of the recording epoch. Stage 4 sleep is defined when delta waves constitute greater than 50% of the recording epoch. The presence of delta waves is associated with very deep sleep, consequently, stages 3 and 4 together are often referred to as deep or slow wave sleep. Stages 3-4 occur initially within 30-45 minutes of falling asleep and may last for up to an hour.

Stages 1-4 collectively comprise NREM sleep. NREM sleep is behaviorally characterized by the presence of large muscle movements (e.g., tossing and turning). NREM sleep is also shown to become progressively deeper in that there is a progressive increase in the stimulus threshold; requiring more intense stimulation to provoke arousal in successive stages.

REM sleep is both quantitatively and qualitatively different from NREM sleep. The defining feature of REM, or paradoxical sleep is the presence of quick, jerky, binocularly symmetrical horizontal, vertical
or oblique eye movements. Skeletal tone is diminished, although muscle twitches, especially in the face, hands and legs may be seen. These movements may occur simultaneously with or independent of eye movements, and all may occur singly or in groups. They are typically discrete rather than continuous movements. The EEG during REM may resemble that of an individual who is awake and active. The pattern is generally low voltage, mixed frequency with a sawtoothed appearance. This sawtoothed activity is usually associated with rapid eye movements. Eighty percent of subjects awakened during REM will report dreaming (Reynolds & Kupfer, 1987).

The first sequence of NREM (stages 1, 2, 3 and 4) in a sleep cycle may take up to 90 minutes. This is typically followed by a brief return to stage 2 sleep and then the initiation of REM. The first REM period of the night usually lasts 5-10 minutes. There are between 4-6 cycles of NREM-REM sleep per night. Over the course of the night, the duration of stages 3 and 4 decrease, while the duration of stage 2 and REM sleep increases. Toward the second half of the night, REM and stage 2 sleep are dominant, and episodes of REM may lengthen to an average of 20-45 minutes.

It is important to recognize here that the markers which define the changes in sleep are arbitrarily delineated. The structure by which we can identify and refer to the changes in sleep patterns has been artificially imposed by researchers to facilitate communication, and does not necessarily imply scientific "truth" about sleep. The stages of sleep as we know them are a convenience, or scientific shorthand for what actually appears to be a continuous process. This is particularly relevant to recall when this communication system is
inadequate, or inconsistent with other scientific data. Such has been the case with sleep onset insomnia: when stage 1 sleep is defined as the onset of sleep, insomniacs are much less accurate than normals in estimating their latency to sleep. When stage 2 is the criterion for sleep onset however, insomniacs' accuracy ratings are increased significantly. These findings may indicate that sleep, for some individuals, begins in stage 1 and for others, begins later in stage 2. In any case, sleep onset is not necessarily the same for all individuals. In addition, EEG recordings may signal the presence of alpha for very brief periods of time. Technically speaking, the individual is awake. However, the individual may never recognize nor subjectively experience this wakefulness, rendering the EEG potentially very misleading. These findings are discussed in greater detail below.

Physiological Causes of Insomnia

The pathophysiologies of insomnia have not yet been fully determined. Experiments conducted in the neurophysiology of sleep tend to be performed chiefly by one of two methods; either stimulation, or ablation or lesion. Consequently, much of our knowledge about sleep and brain structure comes from the animal literature. Little actual experimentation has been performed on human subjects. However, medical disorders and CNS trauma known to disrupt sleep have provided some relevant information regarding the roles of the physiological variables discussed below. Psychological factors in insomnia have also been hypothesized. These will be discussed in detail in a later section of this paper.
ARAS It is known that wakefulness "depends on the activity of the brain stem diencephalic ascending reticular activating system, (ARAS)," (Parkes, 1985, p. 73). Sleep onset, on the other hand, is achieved by the reduction of normal waking stimuli, reduction in activity of the ARAS, and the activity of a presumed, and as yet unlocated, hypnogogic center or system (Parkes, 1985). One postulated integrative theory holds that any type of discomfort (e.g., pain, anxiety, stimulant medications) increases activity in the ARAS, which leads to difficulty falling asleep, while in addition, it increases dopamine (and possibly norepinephrine) activity, which leads to difficulty in remaining asleep (Hartmann, 1982).

Hypothalamus The hypothalamus is considered a continuation of the ARAS, and has been implicated as an important structure in sleep and waking. Hypothalamic damage in general impairs normal arousal mechanisms (Plum & Posner, 1986). More specific effects have been found to occur with more systematic procedures. For example, stimulation of the posterior hypothalamus has been found to produce insomnia in laboratory animals (Ranson, 1939), while damage to this area results in somnolence (van Oot, et al., 1984).

Raphe Nuclei Jouvet and Renault (1966, as cited in Parkes, 1985), in attempts to locate the "sleep center" reported that the destruction of the Raphe nuclei was followed by 3-4 days of insomnia in cats. The extent of insomnia was directly related to the extent of the lesion; total destruction caused total wakefulness, whereas partial lesions selectively destroyed stage 3-4 NREM sleep. Raphe nuclei lesions in man, induced by injury or disease, have been shown to produce partial or complete insomnia as well.
Neurochemical Influences Specific pharmacological interventions have been found to produce effects similar to lesioning the Raphe nuclei. The Raphe nuclei have been found to contain cell bodies of the majority of the serotonergic neurons in the brain. Insomnia has been produced by chemically inhibiting serotonin production. This effect can be reversed by stimulating serotonin production with its' precursor, 5-HTP. (Parkes, 1985). When the Raphe system is altered, production of 5-HTP is altered as well. This reduces the amount of available serotonin, making sleep unlikely, and thus producing the symptoms of insomnia, (Hartmann, 1982). These results may be most reliable however, in the short run. When adult cats were administered long term amounts of serotonin-depleting drugs, reducing the brain's supply of serotonin to unmeasurably low levels, sleep was absent only at the outset of the study. Although the returning sleep was altered from normal, it did return, suggesting that the brain can produce sleep without measurable levels of serotonin (Dement, Milster, & Henrikson, 1979).

Assessment of Insomnia

A variety of techniques have been utilized in the assessment of insomnia. Each of these methods is aimed at quantifying the specific complaints which characterize the disorder. Wholly objective procedures such as psychophysiological measurement, and more behavioral techniques such as self-monitoring are used with the greatest frequency by current investigators. Each method has both advantages and drawbacks.
Psychophysiological recording  The most commonly employed laboratory assessment technique is polysomnographic recording. Generally, this consists of all night EEG, EOG, and EMG recordings (Bootzin & Engle-Friedman, 1981). Other measures, such as heart rate and skin conductance are also frequently included to assess overall levels of physiological arousal (e.g., Haynes, et al., 1981; Haynes, Follingstad & McGowan, 1974; Monroe, 1967).

Objective psychophysiological recordings allow the discrimination of sleep from wakefulness and the observation of more specific sleep stages. EEG recordings have been used to measure latency to sleep onset, number of awakenings during the night and total length of time asleep in both the initial assessment of sleep disturbances (e.g., Haynes et al., 1981; Haynes et al., 1974) and in the assessment of response to treatment (e.g., Coursey, Frankel, Gaarder & Mott, 1980). Other types of physiological recording have been used in sleep assessment. Several studies have examined movement as an index of sleep or wakefulness (Hyuppa et al., 1987; Kupfer, Detre, Foster, Tucker & Delgado, 1972; Kripke, Mullane, Messin & Wybourney, 1978). In general, individuals who complain about sleep show more movement during sleep than do normal sleepers. However, when more specific comparisons are made these studies have yielded mixed results. When comparing wrist movement to EEG stages, Kupfer et al. (1972) found no relation between the two for either latency to sleep or total time asleep. Kripke et al., (1978) found completely contradictory results; wrist movements produced more accurate estimates of total time asleep and latency to sleep onset than EEG data. In their review of the literature, Bootzin & Engle-Friedman (1981) conclude that motility is
most useful as an adjunct to other measures of sleep problems.

To date, polysomnographic recordings provide some of the most precise data about sleep, but this methodology is not without its limitations. Frequently, polysomnographic recording cannot distinguish between DIMS and DOES complaints, especially when the mechanism that causes the disorder is the same (e.g., in sleep apnea; Thorpy, 1988). Experts in this area suggest that EEG measurements provide only one aspect of the definition of sleep, and are not necessarily the "true" measure of sleep (Bootzin & Engle-Friedman, 1981; Carskadon, Dement, Mitler, Guilleminault, Zarconi & Spiegel, 1976; Hartmann, 1988; Lacks, 1987). This is particularly relevant when it is recognized that EEG sleep stage definitions are arbitrarily delineated, and although they are discriminable from one another, it is not clear at which stage subjects are actually asleep (Bootzin & Engle-Friedman, 1981). This idea is additionally substantiated by the discrepancies between subject reports and EEG data when subjects are awakened at various points in the sleep cycle. Although EEG measures are associated with behavioral indices of sleep (e.g., types of body movements), good and poor sleepers alike may report that they were not asleep when EEG measurements indicate stage 2, 3, or 4 NREM sleep (e.g., Borkovec, 1979; Foulkes & Vogel, 1965; Goodenough, Lewis, Shapiro, Jaret, & Sleser, 1965). Furthermore, EEG data can be extremely misleading in the case of severely brain injured individuals who report being asleep when data suggest wakefulness (Murray, 1965).

Another criticism directed at psychophysiological recording as an assessment tool for insomnia is related to stimulus control theory (Bootzin, 1972; Bootzin & Nicassio, 1978; Haynes, Adams, West, Kamens &
Safranek, 1982). Subjects whose sleep difficulties are related to specific factors in their habitual sleep environment may not necessarily show sleep disturbances in the lab (de la Pena, 1978). Additional disadvantages of this method reflect its inconvenience. In research projects, data collection becomes quite drawn out, as subjects must generally be assessed individually or in small groups. Furthermore, the equipment is quite costly and requires trained technicians, data reduction is time consuming, and individuals must "leave the comfort of home" to sleep in the laboratory (Lichstein & Kelly, 1979).

In an attempt to remediate some of the weaknesses of all night recording strategies, Haynes and his colleagues (Haynes, Fitzgerald, Shute & O'Meary, 1985) began examining the utility and validity of assessing sleep in daytime nap periods rather than nighttime sleep periods. Their results indicate that daytime naps show sleep onset latencies similar to nighttime sleep in groups of both normals and insomniacs. However, the study employed a between groups design, utilizing two separate data sets involving different subjects to compare naps and all night sleep. Within subject comparisons would enhance the credibility of this assessment technique. In addition, while this methodology may prove quite useful in the assessment of sleep onset insomnia, it may have somewhat less utility for subjects who complain of mixed or sleep maintenance insomnia.

Kelley and Lichstein (1980) developed a different type of objective procedure to assess sleep and wakefulness. Subjects respond verbally to an automatic tone that sounds at 10 minute intervals. Subjects' responses are recorded, yielding a measure of
sleep status every 10 minutes. From this, latency to sleep and total
time asleep can be calculated. The unit containing this device is
portable and can be used in the subject's home, and data are
relatively easily scored, thus avoiding some of the pitfalls of the other
types of objective data collection. The results of this technique are
promising, however to date, the device has been used only with a
very small (n = 3) sample of normal sleepers and without further
testing, reliability and validity are somewhat suspect.

Behavioral observations Behavioral observations have also been
employed in the assessment of sleep disturbances. When data are
collected at home, subjects' roommates or spouses have been enlisted
to assess or substantiate subjects' self report of sleep onset latency
or response to treatment (Nicassio & Bootzin, 1974). When subjects
sleep in institutions, nurses or aides have been the observers (Weiss,
McPartland & Kupfer, 1973). Observers typically evaluate respiration
rates, movements and subjects' responses to having their name called,
to determine wakefulness or sleep at any given time (Baekeland & Hoy,
1971). Specifically problematic to this type of data collection is the
fact that roommates or spouses may be normal sleepers and may
become inattentive or fall asleep long before the insomniac,
jeopardizing the accuracy of their recording. In addition, Bootzin and
Engle-Friedman (1981) point out that observers are rarely trained in
recording techniques and may be particularly vulnerable to unreliable
recording habits (e.g., rater drift).

Daily Monitoring. Perhaps one of the most commonly used sleep
assessment techniques is the daily sleep log (Bootzin & Engle-
Friedman, 1981; Evans, 1977; Lacks, 1987; Thorpy, 1988). In the early
1970's researchers made a shift from retrospective sleep questionnaires to continuous daily monitoring of sleep problems. Sleep logs or diaries are completed in the morning upon final awakening, and provide an inexpensive, convenient, nonintrusive (as compared with physiological monitoring) measure of an individual's sleep in his or her own bed at home (Bootzin & Engle-Friedman, 1981; Lacks, 1987).

Typically, researchers develop their own sleep diaries, but their content and format are relatively consistent across studies. The variables which are generally examined can be traced to Monroe's (1967) study. Sleep logs usually ask subjects to record the number of minutes it took to fall asleep, number of nocturnal awakenings, duration of each nocturnal awakening, total number of minutes slept, and questions related to sleep quality (e.g., restedness or sleepiness upon awakening, subjective rating of the difficulty falling asleep that night, etc.).

Investigations into the psychometric properties of sleep diaries appear acceptable. Reliability measurements for sleep diaries have been found to be equivalent to those of EEG measurements especially for insomniacs (Bootzin & Engle-Friedman, 1981; Coates, Rosekind, Strossen, Thoresen, & Kirmil-Gray, 1979). Test-retest reliability of sleep onset, as measured by sleep diaries, was .92 for poor sleepers and .58 for good sleepers; EEG reliabilities were .70 and .58 for good and poor sleepers respectively. Measures of reliability for number of awakenings during the night were somewhat lower but equally consistent with measures of reliability using EEG data. Test-retest of number of awakenings for poor sleepers on both EEG and self report
measures were .66 and .69 respectively (Bootzin & Engle-Friedman, 1981).

Sleep diaries have construct validity with other measures of the same behavior. Sleep diary estimates of latency to sleep onset correlate well with observer estimates of the same behavior (r = .84, Turner & Acher, 1979). In addition, several investigators have reported substantial correlations between EEG measures of sleep latency and sleep diary estimates (Baekeland & Hoy, 1971; Carskadon et al., 1976, Freedman & Papsdorf, 1976; Lacks, 1987). Correlations range from .62 - .99; higher correlations are found when more stringent EEG definitions of sleep onset are employed (Lacks, 1987).

Hauri and Olmstead (1983) noted that insomniacs tend to show frequent alternations between wakefulness and stage 2 NREM sleep in the process of falling asleep. Consequently, by employing a conservative EEG estimate of latency to sleep, i.e., elapsed time from lights-out to the start of 15 minutes of stage 2 sleep, without reversals back to stage 1, insomniacs' sleep diary estimates of latency to sleep become quite accurate (within approximately 3 minutes of EEG estimates by the second night of recording). Coates et al. (1982) obtained similar results; significant differences were found between insomniacs estimates of sleep onset latency and EEG stage 1 NREM sleep, but not between estimates of sleep onset latency and stage 2 NREM sleep.

Despite the strength of these results, there is some controversy over the validity of sleep diaries. Some investigators report discrepancies between sleep diary estimates and EEG data, and suggest that subjects tend to overestimate latency to sleep and underestimate
number of awakenings during the night and total time asleep (Borkovec & Weerts, 1976; Carskadon et al., 1976; Monroe, 1967). Additionally, the issue of the individuals’ ability to estimate the passage of time has been raised in general (Ornstein, 1969), and with insomniacs in particular (Borkovec, 1982).

Findings across studies, however, yield a consistent size of the error in estimations. Bootzin & Engle-Friedman (1981), in their comprehensive review of the literature, suggest that the findings of consistent differences may not be of great concern, given the substantial correlations between EEG and sleep diary measures. They go on to suggest that because of the consistency of errors, the same underlying variable is being assessed by two different measures, and that altering the EEG criteria which defines sleep (changing from Stage 1 to the first sleep spindle or K complex) would reduce the differences between the two. Lacks (1987) concurs, and suggests that self-report estimates are actually closer to experienced onset than the EEG markers of sleep onset.

Finally, most investigators conclude that the "experiential" component of insomnia as assessed via sleep diaries is crucial to the diagnosis of the disorders and are essential to the assessment of the problem (Borkovec, 1982; Hartmann, 1988; Lacks, 1987). Borkovec (1982) states that a subjective complaint of insomnia is a necessary aspect of the sleep disturbance, and in some cases, may be the sole identifiable problem in the diagnosis. Consequently, the assessment of insomnia via self monitoring appears warranted.
Treatment of Insomnia

Professional treatment of insomnia is generally dictated by the cause of the disorder. When complaints of excessive sleep onset latencies or sleep maintenance problems are clearly secondary to physical, metabolic or severe psychiatric disorders, the intervention is aimed at ameliorating those underlying disorders. When insomnia is the primary complaint or, in the judgment of the clinician, it was initially caused by some other condition but has become an independent pathological condition, treatment is geared directly toward relieving the sleep complaint. Treatment in this case typically takes the form of either a pharmacological or behavioral intervention.

The insomnia treatment outcome literature as a whole is remarkable for two features. First, most outcome studies involve either sleep onset insomniacs or insomniacs with mixed onset and maintenance complaints. Very little research has been conducted on groups of pure sleep maintenance insomniacs, which may limit the generalizability of these findings. Second, most of the research in this area uses self report outcome data to assess changes in sleep. Consequently, the ability to generalize outcome changes to changes in EEG is limited (Borkovec, 1982). However, as discussed above, the individual's own assessment of his sleep problem is a highly important component of the disorder. Thus, self report of change following treatment may be a satisfactory criterion on which to evaluate treatment efficacy.

Pharmacological Intervention  The most common treatment for insomnia is some form of pharmacotherapy (Morin & Kwentus, 1988). Sleep medications are defined as "any medication that promotes sleep,
or that the insomniac believes will promote sleep," (Lacks, 1987, p. 9).
Sedative hypnotic medications have effects on both sleep and
wakefulness, and these effects cannot be separated from one another.
All sedative hypnotics are general central nervous system depressants,
and not specifically sleep promoting agents. All hypnotics shorten
latency to sleep, decrease the number of middle of the night
awakenings, increase total time asleep, decrease body movements
during sleep, and cause difficulty in arousal (Parkes, 1985). Estimates
suggest that between 15-50% of insomniacs regularly take hypnotics
(Dement, 1972; Kales & Kales, 1974; Morin & Kwentus, 1988; Parkes,
1985). The length of time on medication can vary greatly, ranging
from two weeks to continuous use for several years (Kales, & Kales,

In general, sedative hypnotic medications are most effective
when taken 1 - 2 times per week to avoid habituation, which is a
danger, particularly with the barbiturates. For chronic insomniacs,
medications taken infrequently for a short period of time may help
break a self perpetuating cycle of sleepless nights and worrying about
sleepless nights, and can allow an individual 1 or 2 nights of adequate
sleep per week (Borkovec, 1982). Most sleep experts conclude that
hypnotics are best used with healthy individuals who are experiencing
short term, situational or transient sleep problems (Borkovec, 1982,
Lacks, 1987; Morin & Kwentus, 1988).

Behavioral Interventions  The past two decades of research on
the treatment of insomnia has emphasized behavioral techniques. This
is consistent with the majority of etiological hypotheses of
psychophysiological insomnia. It also reflects the notion that sleep
disturbances which were originally the result of medical illness or environmental disturbances may be maintained or aggravated by behavioral factors (Borkovec, 1982; van Oot et al., 1984). There are several behavioral treatment paradigms including relaxation and biofeedback, stimulus control and paradoxical intentions. Overall conclusions from studies utilizing behavioral techniques suggest that the heterogeneity of subjects with insomnia is reflected in responses to treatment; individuals respond differently to different types of treatment strategies.

**Relaxation Techniques** Relaxation and biofeedback have received the most attention in the literature. A variety of procedures have been experimentally investigated, including autogenic training (Nicassio & Bootzin, 1974), progressive muscle relaxation, (Borkovec, Grayson, O'Brien & Weets, 1979), hypnotic relaxation (Borkovec & Fowles, 1973), and meditation (Woolfolk, Kaffashan & McNulty, 1976). The theoretical basis of these treatments is derived from Monroe's (1967) findings of greater psychophysiological arousal in poor sleepers than in good sleepers. Although results consistently indicate that techniques designed to reduce arousal are superior to placebo and no-treatment control groups in reducing the identified sleep complaints, no concomitant reduction in physiological arousal has been demonstrated (Borkovec, 1982; Borkovec et al., 1979; Coursey et al., 1980; Freedman & Papsdorf, 1976; Haynes, Sides & Lockwood, 1977; Morin & Kwentus, 1988).

The exact mechanism of action in relaxation techniques is still, as yet, undetermined. Borkovec and his colleagues (Borkovec, et al., 1979; Borkovec, Kaloupek, & Slama, 1975; Borkovec & Fowles, 1973;
Borkovec & Hennings, 1978) conducted a series of dismantling studies. They determined that the most important component of the successful techniques is the muscle tension-release strategy. Borkovec (1982; van Oot et al., 1984; van Oot & Borkovec, 1988) speculates that these findings may be the result of a cognitive distraction which occurs during the tension-release tasks. Subjects focus on the monotonous muscle tension and release task, thus freeing them from sleep-incompatible cognitive activity (e.g., worry), and allowing them to fall asleep. However, in a more recent study, Woolfolk and McNulty (1983) determined that techniques which required subjects to focus their attention using imagery without muscle tension-release exercises were more effective than somatic-focusing conditions.

Overall, studies which employ relaxation techniques yield treatment outcomes ranging from 40-60% improvement (Borkovec, 1982; Morin & Kwentus, 1988). Studies which have employed biofeedback techniques have yielded similar results. Morin & Kwentus (1988) suggest that given the expense and equipment associated with biofeedback, relaxation procedures alone in most cases are the more cost-effective technique.

Stimulus Control. Treatments designed within the stimulus control paradigm are geared toward eliminating the associations of sleep-incompatible behaviors and thoughts with sleep related stimuli, such as the bed and bedroom (cf. Bootzin, 1972). The actual components of treatment include retiring to bed only when sleepy, eliminating naps, maintaining a consistent awakening time in the morning, avoiding non-sleep behaviors (e.g., eating and talking on the telephone) in the bedroom, and getting out of bed if sleep has not
occurred after approximately 10 minutes. This promotes the operant pairings of rapid sleep onset and sleep related cues. Research support for the effectiveness of this type of intervention is perhaps the strongest of all. Results consistently show reductions in sleep onset latency (Lichstein & Fischer, 1985) and improvements in sleep maintenance (Lacks, Bertleson, Sagerman & Kunkel, 1983). Further, in direct comparisons between treatment modalities, stimulus control has been found to be more effective than imagery training (Morin & Azrin, 1987) and relaxation techniques (Turner & Ascher, 1979). Overall reports of improvement in sleep complaints utilizing stimulus control techniques range from 58-70% (Borkovec, 1982; Lichstein & Fischer, 1985; Morin & Kventus, 1988).

**Paradoxical Intentions.** Paradoxical intentions in the treatment of insomnia have received less empirical attention than either relaxation procedures or stimulus control. Within this perspective, subjects are given a behavioral prescription to stay awake as long as possible. The theory suggests that adherence to these instructions reduces the sleep-incompatible anxiety (and arousal) some insomniacs associate with unsuccessful attempts at falling asleep. Results of investigations with this technique have been variable. Initial studies yielded promising outcomes (e.g., Ascher & Turner, 1979), however, more recent data are less supportive of the benefits of this technique (e.g., Lacks et al., 1983).

**Psychological Factors in Insomnia**

A variety of etiological hypotheses of insomnia have been proposed. This section will attend specifically to psychological
theories of insomnia. Several factors have been implicated including depression, anxiety, inappropriate stimulus control and stress.

Depression Psychological theories have tended to focus on mood disorders as causative agents in sleep problems. Theories proposed to explain the relation between mood and sleep generally hold that the disruptions of "biological rhythms" (circadian rhythms for endocrine hormone release, body temperature, etc.) associated with mood disorders are causative agents in sleep disturbances (Noll, Davis & DeLeon-Jones, 1985). However results of research in this area are inconsistent (Parkes, 1985).

Research attempts to confirm empirically the clinical association of insomnia and depression, have generally been correlational or descriptive in nature, involving populations of self-reported insomniacs completing the Minnesota Multiphasic Personality Inventory (MMPI), or other psychological measures. An overview of the results indicates that there is indeed a relation between complaints of depression and complaints of insomnia (Beutler, Thornby & Karacan, 1978; Coursey, Buschbaum & Frankel, 1975; Hartmann, 1982; Kales, Caldwell, Preston, Healy, & Kales, 1976; Kupfer, Foster, Reich, Thompson & Delgado, 1976; Lacks, 1987; Monroe, 1967; Parkes, 1985). However, as a whole, this body of literature is flawed. Few studies employ adequate controls to permit conclusions about the disorders involved to be drawn with confidence (e.g., Johns et al., 1971; examined only sleep disordered subjects for a variety of variables including depression, without any control group). Inadequate statistical analysis often complicate the matter further. In addition, the quality of inclusion criteria varies
greatly across studies, ranging from mild self-reported sleep onset insomnia only to no discrimination among many types of DIMS. This causes difficulty in making comparisons and generalizations about the insomnias.

Two of the most frequently cited studies in this area illustrate these flaws. A study by Kales et al. (1976) was designed to assess personality patterns in insomnia. Subjects were recruited via advertisements or were referred from a university sleep clinic. They were given the MMPI and a sleep questionnaire. Data analysis consisted of descriptive statistics about elevations on a given MMPI scale or group of scales.

The results of this study showed that 85% of subjects had one or more pathological MMPI scale elevations (T-score of 70 or greater), and 61% of the total sample had pathological elevations on scale 2 (Depression). Other data suggest that having multiple sleep complaints was associated with gender (females were more likely than males to complain), age (a positive relation), elevations on MMPI scales 1, 3 and 8 (Hypocondriasis, Hysteria and Schizophrenia), and the use of sleep medications.

On the surface, the results of this study seem to indicate that there are strong connections between psychopathology, particularly depression, and DIMS. However, the methodological flaws of this study make the results difficult to interpret. There is no evidence of any type of subject screening for possible confounds (e.g., current use of medications, physical illness, type of insomnia). Thus there may be any number of 'third variables' influencing trends in the data. In addition, this study does not employ a control group of any kind.
The data from the sleep disordered subjects should be compared to data from matched normal controls, and/or a group of patients with some other type of chronic disorder (e.g., patients with a chronic dermatological disorder) in order to determine whether these results are, in fact, unique to insomnia, and not an artifact of other phenomena the subjects may have shared.

In the second study Coursey et al. (1975) confirmed the self-report of 18 insomniacs (8 onset, 4 maintenance and 6 mixed complaint) with all-night electroencephalogram (EEG) measurement. These subjects were then matched with normal control subjects on the variables of age, sex and education. All subjects slept in the lab for five nights. The first night was considered an adaptation night, and EEG measures were averaged over the remaining nights. Subjects completed the MMPI, Weschler Adult Intelligence Scale (WAIS), and several other paper and pencil measures. It was hypothesized that there were two personality constellations which would characterize the insomniacs. The first suggested constellation depicted insomniacs as mildly depressed, anxious, obsessively worrisome, with mild hypochondriacal concerns. The second personality group consisted of subjects who could be considered either inadequate sensory reducers (those who reduce the perceived intensity of sensory input) or excessive sensory augmentors (increasing the intensity of sensory input). This dimension was determined by both physiological and paper and pencil measures.

Data analysis revealed that sleep disordered subjects were significantly more depressed and anxious than normals were. In addition, they appeared to be sensory reducers, according to the
definition employed by this study. At first glance, this study is well conceived and controlled. The major flaw of this study lies in the data analysis. The authors used 25 dependent measures (each scale of the MMPI was considered a separate dependent measure), yet had only 36 subjects. Furthermore, in an attempt to reduce the number of dependent measures, the authors performed a factor analysis. Again, the limited number of subjects makes data interpretation difficult; this procedure generally requires 5-10 subjects per item included in the factor analysis (Nunnally, 1978). The trends in this data are interesting, but require replication with larger numbers of subjects to rule out the possibility that they are artifacts of inadequate sample size.

The overall consistency in results suggests that there is in fact a strong relation between depression and insomnia, but limitations of the data argue against generalization of the results. One other significant aspect of this research which must be addressed is that much of what we know clinically about the relation between sleep and depression comes from the depression literature. This automatically introduces a bias; subjects included in these studies typically have already received a diagnosis of depression, and thus the studies examine only depressed subjects for evidence of sleep disturbances. When the problem is approached from the sleep disturbance side, subjects may or may not be depressed. Consequently in this case, results may be more representative of the population of insomniacs.

Further, most of the research in this body of literature uses a single assessment of depression, suggesting it is a stable condition. It can be argued that mood is a very variable condition, and that it’s
relation to sleep may be more complex than the data indicate. Repeated assessments of mood and sleep would clarify this relation.

Anxiety/Arousal Anxiety is also frequently associated with the phenomena of insomnia (Baekeland & Hoy, 1971; Beutler, Thornby & Karacan, 1978; Friedman & Sattler, 1982; Hartmann, 1982; Haynes et al., 1985; Haynes et al., 1974; Monroe, 1967; Parkes, 1985;). Complaints of muscle tension while individuals are falling asleep, or apprehensive feelings or ruminative thoughts are often cited by insomniacs as the stumbling blocks to sleep (Hartmann, 1982).

Physiological arousal is the proposed mechanism responsible for initiating and maintaining insomnia symptoms in these cases. Haynes et al. (1974) described the hypothesis succinctly,

...Because sleep is associated with low levels of autonomic arousal, individuals who are highly aroused may experience difficulty in falling asleep or may awaken frequently at night. If an elevated level of autonomic arousal is associated with sleeping difficulties, it would be expected that insomniacs would score higher than non-insomniacs on self-report measures of autonomic arousal and anxiety (p. 69).

More technically, high autonomic nervous system arousal elicits increased ARAS activation, which, in turn elicits increased central nervous system activity. This response is incompatible with the central nervous system functions (e.g., decreased ARAS activity and increased serotonin production) which are associated with nonproblematic sleep onset and maintenance. Thus, high levels of
autonomic arousal is hypothesized to be associated with sleep disturbances.

Several studies have indeed found higher arousal (either by self-report or psychophysiological subjects who complain of sleeplessness as compared with their normal counterparts (Freedman & Sattler, 1982; Haynes et al., 1974; Monroe, 1967; Rechtschaffen & Monroe, 1969;). Hartmann (1982) reports that, in general, polysomnographic recordings of both sleep onset and sleep maintenance insomniacs tend to yield high rates of muscle tension just preceding sleep onset and often into sleep, and rapid heart rate (often at waking levels) before and during sleep.

Important to the concept of arousal in sleep disorders is the consideration of arousal thresholds. In all sleepers there is a progressive increase in the stimulus intensity necessary to produce arousal in stages 1, 2, 3 and 4 NREM sleep (Hartmann, 1985). The strength of a stimulus required to produce arousal in REM sleep is similar to that necessary in stage 2 sleep (Hartmann, 1982). It is hypothesized that insomniacs, as compared to noninsomniacs, "may be more sensitive and physiologically reactive to environmental (particularly auditory) stimuli," (Haynes et al., 1985). If follows then, in theory, that this enhanced sensitivity may be the cause of the reported more frequent awakenings and longer back-to-sleep latencies (Haynes et al., 1985).

Bonnet and Johnson (1978) performed a study to test for differences in arousal thresholds between insomniacs and noninsomniacs, and found no significant differences. These results must be interpreted with caution however, because of the poor
methodological design. In one aspect of the study, the authors employ seven subjects and conduct 12 t-tests. These inappropriate statistical procedures limit the confidence with which conclusions about this study may be drawn. In addition, the lax inclusion criteria used for this study further calls into question the validity and generalizability of these results.

Haynes et al. (1985) conducted research to assess and quantify some more specific aspects of arousal thresholds in three separate groups of sleep subjects. Self-declared insomniacs were characterized by differences in self-report and physiological measurement of sleep onset latencies, with the former exceeding the latter. Psychophysiologicaly defined sleep-onset insomniacs achieved congruence between self-report and objective measurement. Normal sleepers served as controls. The relation of stress as a mediating variable in auditory awakening threshold was also examined in this study.

Consistent with the findings of Bonnet and Johnson (1978) no differences were found between insomniacs and normals on auditory awakening thresholds. Psychophysiological insomniacs, however, took longer to fall back to sleep than normals did after awakening. Stress as defined in this study (Life Events Schedule, state anxiety, and heart rate at sleep onset) was significantly and positively related to return-to-sleep-latency, but was not related to awakening thresholds. It seems possible then, that a centrally mediated arousal mechanism is related to some, but not all aspects of sleep disorders (Haynes et al., 1985).
**Stimulus Control**  A variant of the anxiety/arousal paradigm is the operant stimulus control theory of insomnia (Bootzin, 1974). In this model, previously neutral stimuli, such as the bed, furnishings in the bedroom, etc., become associated with behaviors other than sleep. For example, studying on the bed, watching television, listening to music, illness, or even trauma in the bedroom are events commonly considered incompatible with sleep. Stimuli in the bedroom are associated with these arousing activities and acquire the capacity to elicit a similar arousal, decreasing the likelihood of the reduction in arousal necessary for sleep.

The stimulus control theory was first endorsed then rejected by Haynes in a series of studies (Haynes et al., 1974; Haynes, Price & Simmons, 1975; Haynes et al., 1982). In his refutation of this hypothesis, Haynes reported data showing no significant differences between insomniacs and normal controls on measures of frequency of sleep-incompatible behaviors or variability of sleep habits. However, Haynes did not assess for the possibility of a conditioned link between the behaviors and insomnia. Sleep-incompatible behaviors may have occurred with great frequency or intensity at one time in an insomniac’s past (e.g., a trauma), building a strong enough association between the event(s) and sleeplessness that the actual stimuli or behaviors need not be present to produce the response of insomnia. Cognitive events (memories, worry) could account for some maintenance of the symptoms and should be assessed before the theory is rejected.

**Stress**  Stress has been found to impact a wide variety of both psychological and physiological disorders (cf. DeLongis, Folkman & Lazarus, 1988; Maes, Vingerhoets & Van Heck, 1988). On the data-
based premise that physical illness and psychological complaints are more likely to occur when there are relatively high numbers of life-changing events it is reasonable to hypothesize that greater numbers of stressors occur in insomniacs' lives than in normals'. Current research in the area of sleep disturbances has begun to consider the etiological significance of stress in insomnia. The findings of this research will be discussed below, but before the empirical investigation of the role of stress in sleep disturbances is reviewed, an examination of the nature of stress itself is warranted.

**Definition and Measurement of Stress**

Models or theories of stress can be generally conceptualized as belonging to one of two schools of thought. In one paradigm, stress is viewed as the response of an individual to an event; in the second, stress is defined as the event or stimulus itself. Researchers who adhere to the first definition are called response oriented theorists, those who adhere to the second have been referred to as stimulus oriented theorists (Derogatis, 1982).

Response theories stem primarily from the work of Hans Selye. In his General Adaptation Syndrome, Selye considered stress to be the body's nonspecific response to any demand made upon it (Selye, 1956). He posited three stages of response to stress; they are 1) the alarm phase, 2) the resistance phase and 3) exhaustion. According to this model, stress is defined by the presence of physiological changes. More current theorists have expanded this model to consider psychological changes as a defining characteristic of the stress
response as well (Derogatis, 1982). Typically, measurements of this type of stress utilize questionnaires aimed at assessing these changes. This may involve the use of psychological symptom inventories such as mood and personality measures (Derogatis, 1982).

Stimulus theorists attend more to the attributes of the event (or stressor), and the environment's potential to be demanding or disorganizing (i.e., stressful) for the individual. When an individual encounters more stressors than he can withstand, a decline in his ability to function results. The focus of interest for the stimulus theorist then becomes the association between the magnitude of stressors and the onset or exacerbation of physiological and psychological symptoms in an individual. The empirical thrust of this argument has been the measurement and quantification of life events, and their subsequent relation to disorder. Stimulus theorists further define stress as any event which requires change or adaptation. Proponents of this paradigm hold that response theories are potentially confounded, and run the risk of circularity, using a measure of distress to predict distress (Dohrenwend, Dohrenwend, Dodson & Shrout, 1984).

Waters (personal communication, 1989) suggests that "from the response perspective, however, it can be stated that the measurement of "distress" (emotional distress) can be used properly to predict "distress" (physical symptomatic distress) without any circularity. In essence, a negative emotional response may be considered as one part of a complex of responses to a given stressor, this occurring in concert with (and perhaps adding to) the physiological stress response that has etiological and/or exacerbating effects. Further, when the
physical symptomatic "distress" occurs or is made worse, additional emotional "distress" may occur in response to it, and this may cause still greater exacerbation. Obviously, what matters here is how one defines "distress", the definition used determining whether or not there is circularity.

"There is also a question as to whether it is even appropriate to talk of opposing stimulus and response theories when it is clear that both approaches require a stimulus that evokes an adaptational response complex that, most likely is behavioral, cognitive, and physiological in nature. It seems rather that there are valid and interactive stimulus and response perspectives or emphases to what is actually a stimulus-organism-response theory of stress" (Waters, personal communication, 1989).

Current measurement of life changing events began with the construction of the Schedule of Recent Events (SRE; Hawkins, Davies & Holmes, 1957). In its original form, the SRE consisted of 42 items designed to assess the incidence of life-changing events. Individuals indicate the occurrence of events during a given period of time. Stress was quantified by the number of events which had occurred. Items on the SRE cover a variety of different types of events, from annual occurrences (e.g., vacations) to presumably less frequent happenings (e.g., marriage, birth of a child, illness). Rahe (1978) reports that although minor alterations have occurred over time, revisions of the scale have retained the original 42 items.

In an attempt to evaluate the varying degrees of life change and adaptation (i.e., readjustment) that followed from the experience of these events, life event scaling research was conducted. Normal
subjects served as judges and were asked to rate the 42 SRE items in terms of the estimated amount of change or adaptation that was required after the occurrence of the event. Ratio scaling methodology was employed to determine mean life change scores, or Life Change Units (LCU), which represented weights assessing amounts of stress associated with each event. The items from the SRE were then ranked in order of their mean LCU scores, yielding the current Social Readjustment Rating Scale (SRRS; Holmes & Rahe, 1967). Stress could then be quantified by summing LCUs.

As theory and investigations in this area became more sophisticated, the SRE and SRRS were criticized on a variety of issues, including ambiguity of items (Mechanic, 1975) and novelty of the event (Rabkin & Streuning, 1976). However, the issue that has created the most controversy concerns the degree of desirability of events. The basis of this issue is the idea that some events are more positive or desirable than others and are less likely to have an adverse effect upon individuals. Likewise, more negative or undesirable events are more stressful and have a more adverse effect on individuals. (Brown, 1974; Mechanic, 1975; Sarason, de Monchaux & Hunt, 1975). Subsequent empirical evaluation of this debate remains open ended, but the majority of the data support the argument that negative events are more stressful than positive events (Brown, 1974) and that they may be more important than positive events in accounting for the relation between life events and disorder (Dohrenwend & Dohrenwend, 1981; Mueller, Edwards & Yarvis, 1977).

A further issue associated with the measurement of life events concerns weighting schemes, or methods for determining the negativity
and the relative impact of events. More specifically, the debate has pitted individualized assessment of the negativity and impact of events (cf. Lazarus & Launier, 1978) against a normalized approach to the evaluation of these variables (cf. Holmes & Rahe, 1967). This conflict becomes less important, however, when outcome data regarding the predictive ability of different types of weighting schemes are examined. The preponderance of data indicate that weighting schemes, regardless of type, are often no more valuable in predicting disorder than simple frequency counts of events (e.g., Jones, 1987; Lei & Skinner, 1980; Ross & Mirowsky, 1978).

Attempts to address these and other controversial issues in the measurement of life events have led to the development of other scales. Most notable among these is the Life Experiences Survey (LES; Sarason, Johnson & Siegel, 1978). The LES employs many items from the SRE, but has expanded and clarified them. Respondents may also endorse events within two time frames; 0-6 months and 7-12 months prior to the completion of the inventory. Further, the LES provides both positive and negative life change ratings (a Likert-type scale of +3 to -3, respectively). Thus, this measure allows individuals to indicate both the objective occurrence of the event and assess the desirability and impact of the event. The LES is discussed in detail in a later section of this paper.

**Minor Stressors.** With further evaluation of the association between stress and disorder, researchers began to recognize the importance of minor daily events. Kanner and colleagues (Kanner, Coyne, Schaefer, & Lazarus, 1981) proposed that stress is not limited to major life events which occur infrequently, and may have very
large effects on an individual. Rather, stress may also involve lower impact events which occur on a much more frequent basis. Minor stressful events are viewed as the day to day irritating or frustrating hassles of common existence. They include such occurrences as inclement weather, losing things, and having to wait (Kanner et al., 1981). It was further suggested that because of the temporal proximity of minor stressors to outcome measures of disorder, there would be a strong association between the two (DeLongis, Coyne, Dakof, Folkman & Lazarus, 1982).

The Hassles Scale (Kanner et al., 1981) is a 117 item self-report inventory of minor stressful events. It was designed to be administered once a month, respondents indicate which events they experienced during that time. The impact of each item is rated on a Likert scale which ranges from 1 (somewhat severe) to 3 (extremely severe). The greatest difficulty with this scale is that it does not allow events to be experienced without being labeled a "hassle." Critics of this scale cite the argument of confounding and circularity discussed above; marking the occurrence of an event on this scale implies that it was a negative event and thus caused distress (cf. Dohrenwend & Shroult, 1985).

Brantley (1980) developed a similar measure of minor stressors composed of events generated from daily monitoring by community adults. A later version of this measure, the Daily Stress Inventory (DSI; Brantley, Waggoner, Jones & Rappaport, 1987), contains 58 items designed to assess daily fluctuations in stress, and has been shown to have concurrent validity with the Hassles scale, and also with daily subjective ratings of stress (Brantley et al., 1987). An advantage of
the DSI, particularly in comparison with the Hassles scale, is that subjects rate separately the occurrence and impact of an item, thus subjects may indicate that an event occurred, but that it was not stressful. More discussion of the DSI and its psychometric properties can be found below.

**Major and Minor Stressors**

Several relations between major life events and minor stressors have been proposed. Hinkle (1974) has suggested that major life events affect patterns of daily life, and in that manner, influence daily minor stressors. An alternative possibility is that major life events actually cause or create minor hassles. For example, having a baby would undoubtedly lead to a variety of minor stressors including such things as interruptions during tasks and performing unpleasant tasks. Kanner et al. (1981) suggest that minor stressors may serve as mediators of major life events. Minor stressors may disrupt an individual's ability to cope with a major life event, or even by their frequency and intensity, serve as indicators of the degree to which an individual's routine has been disrupted by a major life event.

In general, findings suggest that the relation between major life events and minor stressors is complex and not wholly understood. What is clear, however, is that both major life events and minor stressors contribute uniquely and independently to the prediction of various psychological and physical symptoms (Caspi, Bolger & Eckenrode, 1987; Gilchrist, 1987; Jones, 1987; Monroe, 1983). Further, minor life events have been shown to account for greater amount of variance than major life events in predicting physical and
psychological symptoms (Gilchrist, 1987; Jones, 1987; Kanner et al., 1981). Logically then, investigations into the nature of stress and its relations to disorder should include both major life events and minor stressors to gain a more thorough understanding than would be obtained by using either approach alone.

Stress and the Relation to Physical Symptoms

Initial investigations into the relation between stress and physical disorder centered on major life events and the onset of serious illness (e.g., coronary disorders). Major life events have consistently been found to account for a significant but modest proportion of the variance in the stress-illness relation (approximately 12%; Paykel, 1974; Rabkin & Streuning, 1976). More recently, investigators have begun to include the assessment of daily minor stressors in this research.

The first studies of this sort utilized single stressors, examining the effects on individuals of such minor stressors as noise (Glass & Singer, 1972) and workload (Frankenhauser & Gardell, 1976). Brantley et al. (1987) have suggested that laboratory or experimental stressors (e.g., mental arithmetic, exposure to aversive photographs, noise) may be viewed as minor stressors and considered in this body of literature as well. Overall findings indicate that individual minor stressors can have a variety of physiological effects, including increases in heart rate, skin temperature, blood pressure, and changes in serum cholesterol, (Mustacchi, 1977; van Doornen & Orlebeke, 1982; Rubman, Waters & Dobbins, 1988). Results of these studies suggest that further
evaluation of a broader range of daily minor stressors is warranted.

In addition to examining a wider spectrum of stressors, a more comprehensive approach to physical symptoms has also been incorporated. Numerous recent studies have yielded similar results; the measurement of hassles or daily minor stressors enhance our ability to predict general somatic health. More specifically, the data indicate that higher levels of daily stress are positively related to both the onset and exacerbation of general physical symptoms (DeLongis et al., 1982; DeLongis et al., 1988; Jones, 1987; Stone, Jandorf & Neale 1986). In addition, higher levels of daily minor stress have been associated with specific physical disorders. For example, Waggoner (1986) found that minor stress was significantly related to a variety of headache parameters, and Goreczny, Brantley, Buss and Waters (1988) found that high levels of daily stress led asthmatics and other pulmonary patients to experience more breathing problems than on low stress days.

The findings that minor stressors have a greater influence than major life events on physical health lend empirical strength to the position that the temporal proximity of events to symptoms is important. Further, given that both health and daily stress may fluctuate rapidly, our ability to assess minor stressors frequently is valuable. Finally, it may be helpful to conceptualize both stress and health as state rather than trait variables, implying that variability is characteristic of both.

**Stress and Insomnia**

Stress is hypothesized to cause increased cognitive and
physiological arousal which is incompatible with sleep. In addition to Haynes et al., (1985), discussed above, other researchers have begun to examine this relation (Cernovsky, 1984; Healey et al., 1981; Rubman, Brantley & Jones, 1987).

Healey et al. (1981) conducted perhaps the most comprehensive study in the area to test this hypothesis. Two groups of sleepers (poor and good) were identified by subjective self-report measures. Subjects were matched for age, gender and level of education. Poor sleepers were later further broken down into three groups; those with difficulty falling asleep, difficulty remaining asleep, and early morning awakening. Both groups of sleepers were given the Life Events Schedule and the Marlowe-Crowne Social Desirability Scale (SDS). There were no significant demographic differences between groups, nor were there differences on scores on the SDS.

Results of this study indicate that all types of insomnia had a gradual course of onset, as assessed via self-report, although most of the poor sleep subjects considered the onset of their insomnias to be temporally related to some major life event. In addition, good sleepers matched with poor sleepers reported significantly fewer stressful events than the poor sleepers for the same period (year of the onset of sleep problems). These results suggest that life stress is an important variable in the onset of insomnia. From this study, however, it cannot be determined if the role of these stressors is actually etiological, for the data are correlational and it could be that other variables account for the observed relation.

The role of minor stressors in sleep disturbances is also being considered. Haynes et al. (1981), examined the effects of pre-sleep
stress on sleep onset insomnia. Ten sleep onset insomniacs and 11 normal sleepers spent five nights in a sleep lab. On the fourth and fifth nights, after the lights had been turned off, subjects were exposed to a minor cognitive stressor (a mental arithmetic task). Sleep latency was defined as time between the end of the cognitive task and stage 2 sleep. Polygraph protocols were scored by researchers blind to the subjects’ experimental condition.

The results of the study indicate that noninsomniac subjects showed longer objective and self-report sleep onset latencies following exposure to the stressor. Insomniac subjects, however showed reduced latencies to sleep. Haynes et al. speculate that the nature of the stressor was such that if the insomniac subjects were cognitively aroused, or worrying, before the onset of the stressor, then the task may have served as a distraction from their intrusive thoughts (cf. Borkovec, 1982). Thus, in this case, the laboratory stressor may have facilitated sleep in the insomniac by reducing worry and cognitive arousal and allowing the insomniac to refocus his attention toward something else. The results of this study are provocative. The authors suggest (and logically so) that perhaps the reason for these unexpected findings is the relatively unconventional and artificial nature of the stressor. They further suggest that a more realistic stressor might have yielded the anticipated results.

In a study designed to test this hypothesis, Rubman et al. (1988) assessed the effects of daily minor stressors on nightly sleep. Forty undergraduates monitored minor stress using the Daily Stress Inventory, and nightly sleep activity with a sleep diary, for 35 consecutive days. Results compared the seven highest-stress days
with the seven lowest-stress days to determine if minor stress impacted sleep onset latency, maintenance and quality of sleep.

The results of the study suggest that when individuals experience a high level of daily stress, they are less satisfied with their night’s sleep. Conversely, subjects report a higher or better quality of sleep following low stress days. Quality of sleep has been previously related to the number of awakenings during the night (Baekeland & Hoy, 1971) although these results were not replicated in the study by Rubman et al., (1988). It may be the case that when subjects reported poorer quality sleep on high stress days, they may have also experienced a lighter depth of sleep, and were partially aroused, though not fully awakened at various times during the night.

Daily stress was not, in this study, found to be related to sleep onset. Two explanations for this finding are offered. First, it is possible that the subject sample is too narrow. As sleep disturbances are positively related to age, normal college students may be too young and resilient to the effects of minor stressors to have had them affect their sleep. However, as disturbances are seen on the more subtle, evaluative measures (e.g., quality of sleep, restedness upon awakening), the possibility exists that the resiliency of the subjects’ sleep obscured the effects on more objective, but less sensitive sleep measures (e.g., latency to sleep). It is possible that the expected effects would be evidenced on a more representative sample.

The second possible explanation involves a possible flaw in the sleep monitoring diary; it may not have been sensitive enough to measure variations in sleep latencies, thus masking any possible effects which may have occurred (Rubman et al., 1988). Replication
and extension of this study with a more appropriate population and a more adequate and comprehensive dependent variable would significantly increase our understanding of the relation between daily stress and insomnia.

The Present Study

The present study was designed to further investigate the relations between stress, mood and insomnia. Community adults were studied in an attempt to obtain a representative sample of individuals with sleep disturbances.

Insomnia may be conceptualized as a specific type of physical symptom. Given the data which indicate that major and minor stressors affect physical complaints, it is logical to hypothesize that stress influences nightly sleep. In contrast to previous studies examining stress and sleep, this investigation assessed the effects of both major and minor life events. Stress was inferred from the assessment of the frequency of major life events and of daily minor stressors. Daily levels of self-reported depressive symptoms and anxiety, which are also hypothesized to influence sleep, were the mood variables in this investigation. Both daily stress and mood were treated as state variables.

Subjective report of difficulty falling asleep or remaining asleep was the primary criteria for insomnia in this study. Because one of the hallmarks of insomnia is the variability of symptoms, sleep was assessed on a daily basis. Similarly, because of the rapid fluctuations in minor stress, and to gain a close temporal relation between the
variables of interest, minor stress were measured on a daily basis as well. Further, because daily stress has been found to affect mood, and mood has been found to influence sleep, mood was also assessed daily. In this manner, a more thorough exploration of the relations between these variables could be accomplished.

Finally, in order to be able to make stronger statements about the relation of stress, mood and sleep in insomniacs, a control group comprised of normal sleepers was included. By examining a group of individuals who do not complain of sleep problems, it can be determined if the effects of stress and mood on sleep are unique to insomniacs, or if generalization to normal sleepers is appropriate.

Hypotheses

I. Insomniacs vs. Control Subjects

It was hypothesized that there would be a significant difference between groups on all sleep disturbance measures, and that insomniacs would show more disturbed and variable sleep than control subjects (longer onset latencies, more frequent awakenings, etc.), and they would show less total time asleep than controls.

II. Stressors and Sleep Disturbances

IIA. It was hypothesized that the number of major life events would be significantly related to symptoms of both sleep onset and maintenance insomnia. Measures of major life events have been previously shown to be related to the onset of insomnia (Healy et al., 1981), and the available data also suggest that major life events may be related to the frequency of insomnia symptoms within individuals (Cernovsky, 1984). The relation of major life events to physical
symptoms has been previously demonstrated, a relation of similar magnitude was expected.

IIB. It was hypothesized that daily minor stressors would show a stronger relation to sleep variables than major stressors, primarily because of the closer temporal relation between the recorded events and sleep, and the DSI’s sensitivity to daily fluctuations in stress. This hypothesis is consistent with the literature in the area of stress and physical symptoms in general, (e.g., Brantley & Jones, 1989; DeLongis et al., 1981) and stress and insomnia in particular (e.g., Haynes et al., 1981).

IIC. It was further hypothesized that there would be an association between minor stress and sleep disturbances in insomniacs such that sleep would be significantly more disturbed on high stress days than on low stress days. Normal control subjects were expected to show more stable patterns of sleep regardless of stress conditions.

III. Mood and Sleep Disturbances

It was hypothesized that fluctuations in anxiety and depression would be related to fluctuations in both sleep onset and maintenance. More specifically, this relation was expected to be in a positive direction (e.g., higher scores on anxiety and depression measures would be associated with longer latencies to sleep, more frequent and longer awakenings, etc).

IV. Stress, Mood and Sleep Disturbances

It was hypothesized that major life events, daily minor stressors, depression and anxiety would all contribute significantly to fluctuations in sleep onset and sleep maintenance insomnia. Specifically, there would be an effect of stressors on mood and sleep
disturbances such that the greater the frequency of minor stressors, the greater the mood disturbances, and the greater the magnitude of sleep disturbances.
Methods

Subjects

Subjects were 54 adult volunteers. There were 20 mixed complaint subjects (subjects who complained primarily of sleep maintenance problems but who may have had onset difficulties as well), 17 sleep onset complaint subjects, and 17 controls, obtained primarily from the community of Baton Rouge, Louisiana. Subjects were recruited from advertisements in the "Health" section of a local newspaper, and from community service bulletins on television and radio. In exchange for their participation in this study, subjects were invited to participate in a free treatment program to help them manage their sleep disturbance symptoms.

Demographic information including age, gender, martial status, and sleep history are presented in Table 1. (See Appendix A for demographic questionnaire.) There were no differences between groups in gender ($X^2 = 2.74, p < .25$), marital status ($X = 7.85, p < .45$), age, ($F(2,51) = 2.77, p < .07$) or duration of sleep problem ($F(1,34) = 1.50, p < .43$). Subjects were screened in a telephone interview to determine their appropriateness for inclusion in this study. Subjects were included in this study in one of the sleep disordered groups if they complained of insomnia symptoms at a frequency of at least three times per week and were free of exclusion criteria. Specific criteria for groups was as follows: for onset subjects, sleep onset latency $\geq 30$ minutes, with less than 15 minutes awake during the night, and less than 6 1/2 hours of sleep per night, on nights when sleep is disturbed; for mixed complaint subjects, $\geq 30$
### Table 1

**Demographic Characteristics of the Sample**

<table>
<thead>
<tr>
<th></th>
<th>Sleep onset</th>
<th>Mixed complaint</th>
<th>Controls</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>15</td>
<td>13</td>
<td>12</td>
<td>40</td>
</tr>
<tr>
<td>Male</td>
<td>2</td>
<td>7</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>40.4</td>
<td>48.1</td>
<td>39.4</td>
<td>42.9</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>11.8</td>
<td>11.9</td>
<td>13.4</td>
<td>12.8</td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>17.6%</td>
<td>15.0%</td>
<td>29.4%</td>
<td>20.4%</td>
</tr>
<tr>
<td>Married</td>
<td>64.7%</td>
<td>75.0%</td>
<td>58.8%</td>
<td>66.7%</td>
</tr>
<tr>
<td>Divorced/Widowed</td>
<td>17.7%</td>
<td>10.0%</td>
<td>11.8%</td>
<td>13.1%</td>
</tr>
<tr>
<td><strong>Duration of Sleep</strong></td>
<td>10.94</td>
<td>12.65</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td><strong>Problem (years)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family Hx of</td>
<td>43.8%</td>
<td>20.0%</td>
<td>43.8%</td>
<td></td>
</tr>
<tr>
<td>Onset Problems</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family Hx of</td>
<td>31.3%</td>
<td>25.0%</td>
<td>37.5%</td>
<td></td>
</tr>
<tr>
<td>Maintenance Problems</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
52

minutes of time spent awake during the night after initial sleep onset (including early morning awakenings) and less than 6 1/2 hours of sleep on a night when sleep is disturbed. Sleep onset latencies for mixed complaint subjects were also assessed, and subject to the same criteria as onset subjects. Control subjects were included in the study if they slept a minimum of 7 hours per night, did not complain of a sleep disturbance, did not meet criteria for either of the other two groups and did not meet any of the exclusion criteria.

Subjects were excluded from this study if they were currently under psychological or psychiatric care; if they had been diagnosed with a sleep disorder other than insomnia by a physician or other professional; if they had a diagnosed physical or metabolic disorder known to influence sleep patterns (e.g., arthritis, Parkinson's disease, hyperthyroidism); if they were taking medications known to influence sleep; if they endorsed a pathological pattern of alcohol or substance abuse; if they were older than 69 years of age; if they took naps more than once per week and any naps were longer than 20 minutes, or if their employment caused them to do shiftwork. In addition, subjects taking medications solely to aid sleep who expressed interest in participating in this study were required to consult with their physician prior to initial interview. These subjects were required to sign a form listing their physician's name, indicating that their physician had consented both to the subject's inclusion in this program and discontinuation of their sleep medications (as per instructions). The medication release form may be found in Appendix B. These subjects did not complete an initial interview until they had been medication-free for at least two weeks.
Measures

Life Experiences Survey (LES). The LES (Sarason, Johnson & Siegel, 1978) is a 57 item self-report measure. (This study will employ the 47 item version, which excludes the 10 school-related items that are designed primarily for use with students.) This measure allows individuals to indicate the number of events that they have experienced over the course of the past year, and in which part of the year they occurred (0-6 months, or 7 months to a year prior to completion of the inventory). In addition, subjects rate the desirability and the impact of these events on a 7-point Likert-type scale ranging from -3 ("extremely negative") to +3 ("extremely positive"). Given the data that indicate that weighting scores are no more predictive of symptoms than frequency counts (cf. Lei & Skinner, 1980), and for the sake of statistical parsimony, this study will employ only a frequency score.

The items listed in the LES were chosen to be representative of events frequently experienced by the general population. Items were drawn from several sources, including the SRE. Thirty four items in the LES are similar in content to the SRE. In the construction of the LES, however, the items were reworded and designed to be as specific as possible and simplify responding.

The reliability and validity of the LES appear adequate. In the initial studies, no gender differences were found on any of the life change measures. In addition, positive and negative life change scores were found to be uncorrelated. Test-retest reliability for the negative impact score following a five- to six-week interval is
satisfactory; \( r = .56 \), and \( r = .88 \), \((p < .001)\). It should be noted that correlations of this type may be underestimated secondary to events which occur during the intervening time period. The LES was found to correlate significantly with state and trait anxiety, depression and school problems (Sarason et al., 1978).

The Daily Stress Inventory (DSI). The DSI (Brantley, Waggoner, Jones, & Rappaport, 1987) is a 58-item standardized inventory of common minor stressors such as arguments, job strains, and social pressures. The scale is designed for daily administration, and respondents indicate the occurrence or non-occurrence of each event over a 24-hour period. Respondents then rate the perceived stress of that event on a 7-point Likert-type scale ranging from 1 ("occurred but was not stressful") to 7 ("caused me to panic"). The DSI yields both a frequency of events score (Event) and a sum of the subjective stress ratings for each of the events that occurred (Impact). Again, this study utilized only the Event score because of the finding that weighting schemes have not increased the predictive ability related to physical symptoms (cf., Jones, 1987). For this study, item 23, which pertains to sleep disturbance, was deleted from scoring to avoid the possibility of artificially inflating the relation between stress and sleep. In addition, as the first day of monitoring has been shown to be inflated by reactivity (Brantley et al., 1988), it was not included in the data reduction and analyses.

The DSI has a number of advantageous features. First, the items were generated by community adults, and the scale was normed and standardized on large samples of community adults. Further, the scale shows acceptable reliability. Chronbach alpha coefficients for the
Frequency and Sum scores were .83 and .87, respectively, indicating that the scale is comprised of a relatively homogeneous set of items (Brantley et al., 1987). In addition, test-retest coefficients of generalizability were modest, reflecting the DSI's ability to measure daily fluctuations in stress. There is no basis to assume that the number or content of daily stressors would be consistent over time thus, the use of the DSI as a "state" rather than "trait" measure of daily stress is supported.

Validity of the DSI has also been established. The DSI has concurrent validity with both a monthly measure of stress and a daily measure of subjective stress (Brantley et al., 1987). The DSI is also associated with endocrine changes. Elevated levels of vanillylmandelic acid were significantly associated with increases in both DSI Event and Impact scores (Brantley, Deitz, McKnight, Jones & Tulley, 1988). In addition, the DSI is sensitive to the differences in stressfulness between weekends and workdays (Brantley, Cocke, Jones, & Goreczny, 1988). Finally, the DSI shows both convergent and divergent validity, as the Impact score correlates with daily state anxiety, but was not found to be related to state hostility (Brantley et al., 1987).

**State-Trait Anxiety Inventory-Form Y (STAI-State, Spielberger, Gorsuch & Lushene, 1970).** The STAI is a 40 item questionnaire composed of two 20 item surveys assessing tension, nervousness, worry and apprehension. The questionnaires are of similar formats, and were designed to be administered independently or together. The STAI Trait-A scale asks respondents to indicate how they generally feel, while the STAI State-A scale focuses on current feelings. Respondents encircle one of 4 responses which range from "almost
never," to "almost always." Each scale takes approximately 5-10 minutes to complete.

The STAI is the most frequently used measure of anxiety in the literature (Buros, 1978). The use of these measures to assess anxiety is supported by the psychometric stability of the Trait-A scale and by the sensitivity to change of the State-A scale (Gotlib, 1984). Test-retest reliability has typically been found to be low for the state form of the questionnaire, which is consistent with theoretical conceptualizations of state variables. Test-retest coefficients are adequate (.80) for the trait measure.

This study employed the STAI State-A scale to measure the transient emotional experience of anxiety at night, before going to bed. Scoring the State-A scale is relatively straightforward. Ten of the 20 items are reverse scored, and the numbers assigned to all items are simply added together. Higher total scores indicate stronger feelings of apprehension, tension and worry at the time of administration.

Zung Self-Rating Depression Scale (Zung, 1965). The SDS is a 20 item self-report inventory of depressive symptoms. This scale was developed in an attempt to operationally define and quantify depressive symptomatology. From his review of the contemporary clinical and multivariate literature on depression, Zung concluded that disturbances in affective, psychological, physiological and psychomotor functioning were the four most commonly observed clinical characteristics of depression. Items on the scale were developed to be representative of these four areas.

Each item contains a 4-point ordinal scale ranging from "none or a little of the time," to most all of the time." Respondents indicate
which is the most applicable to them for each item. For the purposes of this study, item 4, which pertains to sleep disturbance, was excluded from the scoring to avoid artificially inflating the relation between mood and insomnia.

The Zung has respectable psychometric properties. A split-half reliability coefficient reported in a recent study was .94 (Gabrys & Peters, 1985). The Zung has further been shown to be sensitive to various degrees of depressive symptomatology, and can discriminate levels of severity from individuals with no symptoms or mild symptoms to individuals who are severely depressed (Biggs, Wylie, Ziegler, 1978). The Zung has also been found to correlate significantly with other measures of depression including the MMPI-Depression scale, MAACL-D and the BDI (Dobson, 1985; Bosscher, Koning & Van Meurs, 1986).

Derogatis (1982) concludes that although the Zung has a number of valuable attributes to recommend its use in stress research, it has been, until recently, overlooked.

Daily Sleep Diary. The Daily Sleep Diary (Lacks, 1987) is an 11 item sleep-monitoring log. It is designed for daily administration in the morning upon final awakening, to assess self-report complaints of insomnia and the previous night’s sleep. Items are designed to directly assess latency to sleep onset, frequency and duration of nocturnal awakenings and total number of minutes slept during the night. Also, on a 1-7 Likert-type scale, subjects evaluate difficulty falling asleep, restedness upon awakening, and quality of the previous night’s sleep. In addition, sleep efficiency, a ratio of time asleep to time spent in bed can be calculated from the Diary. Finally, the Diary assesses whether or not respondents consumed alcohol within 4 hours.
of retiring. Items are derived from Monroe's original 1967 investigation, and are similar in both form and content to other sleep diaries currently in use (e.g., Lacks, 1988).

To accurately assess the frequency and duration of awakenings during the night, subjects were asked to record the event that awakened them, and to leave a blank space if there was no such event, or if they could not recall an event. At this time, there is no documented methodology in the literature for determining which events should contribute to scores of frequency and duration of awakenings. Because some middle of the night awakenings are not pathological (e.g., awakenings secondary to telephone calls, or in response to children who are ill), it was necessary to devise an inclusion scheme which accounted for these awakenings. Based on consultation with a noted sleep researcher in another laboratory (Morin, personal communication, August 10, 1990) it was decided that awakenings secondary to unusual external events (telephone calls, pets, children) were discounted, and thus not included as an awakening in measures of either frequency or duration of awakening. Events secondary to internal phenomena (e.g., needing to void, nightmares, etc.,) were included in both measures of sleep maintenance for all subjects.

Psychometric properties of this type of instrument are reported to be adequate for use in scientific investigation of sleep disturbances (Lacks, 1988; Nino-Murcia & Keenan, 1988). Typical evaluations of this type of instrument focus on the assessment of individual items. As discussed above, test-retest reliabilities are comparable for sleep diaries and EEG recordings (e.g., Coates et al., 1982). Furthermore, insomniacs estimates of sleep onset latency correlate highly with EEG
recordings of the same parameter (e.g., r = .84; Frankel, et al., 1976). The Daily Sleep Diary can be found in Appendix C.

**Procedures**

In an initial telephone contact, subjects were informed as to the nature of the project. If they still wished to participate, screening for exclusion criteria (mentioned above) was conducted (the telephone screening form can be found in Appendix D). If subjects were considered appropriate for inclusion, they were scheduled for an individual interview at their convenience. Subjects who were asked to participate were encouraged to recruit a cohort (control subject) at this time.

At the first appointment, further screening was conducted. To rule out the possibility of subjects having an untreated mood disorder or thought disorder, a clinical interview based on the Structured Clinical Interview for DSM-III-R (Spitzer, Williams & Gibbon, 1986) emphasizing these disorders was conducted by the experimenter. Seven subjects who met the diagnostic criteria for these disorders were excluded from the study. In addition, subjects were screened in the interview for the presence of any type of Disorders Of Excessive Somnolence or Parasomnias. Five subjects were excluded for this reason.

Subjects who met all eligibility requirements then received an informed consent sheet to read and sign (See Appendix E). At this time the monitoring procedures were explained. Subjects began by completing an LES. Following this, subjects were given a packet of blank forms and self addressed, stamped envelopes for the first week
of monitoring. Each packet contained seven DSIs, seven Zungs, seven STAI State-A forms, seven daily sleep diaries and seven self addressed, stamped envelopes. Subjects were instructed in how to complete each instrument. Subjects completed the DSI, Zung and STAI State-A at approximately the same time each evening: between dinner and retiring. The sleep diary was completed upon final awakening. To ensure that the monitoring was completed in a timely fashion, subjects mailed the completed forms back daily (cf. Lick & Heffler, 1977). Following procedures used successfully by Lacks et al. (1983), if two days passed without receiving data from a subject, the subject received a telephone call to answer any questions which may have arisen and gently remind the subject to continue monitoring.

At the end of the first interview, subjects scheduled an appointment with the experimenter for the following week (six or seven days from the time of the interview). Subjects were encouraged to bring their cohorts as well. At this time subjects returned to the clinic for a fresh set of forms, and the interviewer reviewed the subjects folder with them to assure appropriateness of completion of forms and answered subjects’ questions. This same procedure was followed for the following week as well. In this manner, twenty one days of continuous self-monitoring were completed by each subject.

One hundred seventy one subjects initially expressed interest in participating in the study. Nineteen subjects did not wish to participate after receiving more information about the study. Fourteen respondents were excluded because they performed shiftwork, sixteen subjects were excluded due to age, twenty one reported medical conditions that interfered with sleep (primarily arthritis or headaches),
fifteen either did not wish to consult their physician about discontinuing their medications or were currently receiving psychological care elsewhere, seven were excluded because their sleep problem was not severe enough to meet inclusion criteria, seven subjects reported symptoms suggestive of sleep apnea, four subjects reported symptoms of restless legs syndrome, and two subjects were excluded because of alcohol use. Of the seven subjects who began but did not complete the monitoring process, three dropped out secondary to complaints about the monitoring process, two dropped out secondary to time complaints, one dropped out because of illness, one dropped out because she moved during the monitoring period. These seven were equally distributed across groups.
Results

Mean values calculated for all subjects in this study indicate that across groups subjects obtained 403.8 minutes or 6.73 hours ($SD = 116.87$) of sleep each night. Subjects took an average of 47.66 minutes ($SD = 42.95$) to fall asleep, woke up 1.3 times ($SD = .90$), and were awake for a total of 29.1 minutes ($SD = 32.52$) during the night. They indicated some difficulty falling asleep [$M = 3.2$, $SD = 1.46$, on a 1 (not very difficult) to 7 (extremely difficult) likert type scale]. Similarly, they reported moderate restedness upon awakening ($M = 3.7$, $SD = 1.31$) and moderate overall quality of sleep ($M = 3.6$, $SD = 1.34$). Finally, on the ratio of time asleep to time spent in bed (sleep efficiency) the average score was .83 ($SD = .12$).

The mean State-A score was 37.09 ($SD = 8.17$), which is well within the normal range for working adults (Speilberger, Gorsuch, Lushene, Bagg and Jacobs, 1983). Zung scores although slightly higher than those obtained by a normal control sample, were also within the normal range, with a mean of .42 (Zung, 1965). Subjects experienced an average of 10.2 minor stressors each day ($SD = 4.19$), which is within the normal range for adults, and reported the occurrence of 4.3 major life events ($SD = 3.25$) in the year prior to the beginning of the study.

To evaluate the possibility that the data were influenced by subjects' alcohol use, the sleep on nights that subjects consumed alcohol within four hours of retiring was compared to nights of no alcohol consumption. Subjects in the study consumed alcohol relatively infrequently (8% of the nights in the study). Consequently,
to minimize the disparity in observations per cell, only subjects who consumed alcohol on 6 or more nights during the study were included in this analysis. There were 8 subjects who met this criteria. Total minutes asleep and number of awakenings during the night on alcohol versus no alcohol nights were felt to be the most sensitive to the effects of alcohol, and were contrasted using paired t-tests. No significant differences were revealed.

The relation of major life events to frequency of daily stressors and mood variables was explored using correlations. Frequency of major life events during the year prior to the onset of participation in the study was significantly correlated with State-A scores, \( r = .325, p < .02 \). It was not significantly correlated with Event scores or Zung scores. Neither State-A nor Zung scores were significantly correlated with DSI Event scores. State-A and Zung scores were highly correlated, \( (r = .678, p < .001) \). This correlation indicates that State-A and Zung scores share 46% of their variance (see Table 2).

The relations of frequency of major life events (LES), minor stressors (Event), state anxiety (State-A) and depressive symptoms (Zung) to sleep variables were also explored using correlations (see Table 3). For each subject, mean scores for each variable were calculated from the entire 20 day monitoring period, and correlations were based on these mean scores.

Event scores were significantly correlated with latency to sleep onset (in minutes) and ratings of difficulty falling asleep, and accounted for 14% and 9% of the variance in these scores, respectively. State-A was also significantly correlated with two sleep measures; ratings of restedness upon awakening and quality of sleep.
Table 2
Correlation Matrix

<table>
<thead>
<tr>
<th></th>
<th>MLE</th>
<th>Event</th>
<th>State-A</th>
<th>Zung</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Life</td>
<td>----</td>
<td>-----</td>
<td>-------</td>
<td></td>
</tr>
<tr>
<td>Events</td>
<td></td>
<td></td>
<td>-------</td>
<td></td>
</tr>
<tr>
<td>Event</td>
<td>.158</td>
<td>----</td>
<td>-------</td>
<td></td>
</tr>
<tr>
<td>State-A</td>
<td>.325*</td>
<td>-.053</td>
<td>-------</td>
<td></td>
</tr>
<tr>
<td>Zung</td>
<td>.212</td>
<td>.018</td>
<td>.678**</td>
<td>----</td>
</tr>
</tbody>
</table>

Note: N = 54;

* = p < .02

** = p < .001
Table 3
Correlation Matrix With Dependent Measures

<table>
<thead>
<tr>
<th></th>
<th>MLE</th>
<th>Event</th>
<th>State-A</th>
<th>Zung</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Time</td>
<td>-.015</td>
<td>.001</td>
<td>-.005</td>
<td>-.089</td>
</tr>
<tr>
<td>Asleep</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of</td>
<td>.095</td>
<td>.037</td>
<td>.048</td>
<td>.142</td>
</tr>
<tr>
<td>Awakenings</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of</td>
<td>.059</td>
<td>.007</td>
<td>.200</td>
<td>.288*</td>
</tr>
<tr>
<td>Awakenings</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset</td>
<td>-.002</td>
<td>.374*</td>
<td>.178</td>
<td>.332*</td>
</tr>
<tr>
<td>Difficulty</td>
<td>.150</td>
<td>.297*</td>
<td>.160</td>
<td>.329*</td>
</tr>
<tr>
<td>Rested</td>
<td>.205</td>
<td>.190</td>
<td>.364*</td>
<td>.435**</td>
</tr>
<tr>
<td>Quality</td>
<td>.136</td>
<td>.225</td>
<td>.308*</td>
<td>.457**</td>
</tr>
<tr>
<td>Efficiency</td>
<td>-.021</td>
<td>-.256</td>
<td>-.214</td>
<td>-.379*</td>
</tr>
</tbody>
</table>

Note: \(N = 54;\)

* \(p < .05\)

** \(p < .001\)
Duration of awakenings during the night, latency to sleep onset, subjects' ratings of difficulty falling asleep, restedness upon awakening, quality of sleep, and sleep efficiency were all significantly correlated with Zung scores. Major life events were not associated with any sleep scores.

High Versus Low Stress Conditions

To more closely examine the differential effects of stress on sleep, separate repeated measures analysis of variance (ANOVA) tests were conducted for each of the dependent measures. The repeated measures factor was high versus low stress conditions. For each subject, the 7 days with the highest Event scores and the 7 days with the lowest Event scores made up the high and low stress conditions, respectively. Sleep variables for the days associated with those scores were the dependent measures. Because several comparisons were performed, the significance level for all analyses was increased to $p < .01$, to reduce the likelihood of Type I error.

A preliminary repeated measures ANOVA evaluating high and low Event scores was performed to ensure that subjects in fact experienced a higher frequency of stressful events during the high stress condition than they did during the low stress condition. The mean for Event scores defining the high stress condition was 13.79 ($SD = 4.97$), and the mean for Event scores defining the low stress condition was 6.79 ($SD = 3.65$). These scores differed significantly, $F(1, 51) = 313.27, p < .001$. Onset, mixed and control subjects did not differ significantly on Event scores. Means and standard deviations are presented in Table 4.
Table 4
Preliminary Repeated Measures ANOVA With Event Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>15.29 (5.8)</td>
<td>7.54 (4.1)</td>
<td>11.4</td>
</tr>
<tr>
<td>Onset</td>
<td>14.47 (3.9)</td>
<td>6.99 (3.6)</td>
<td>10.7</td>
</tr>
<tr>
<td>Control</td>
<td>11.40 (4.2)</td>
<td>5.70 (2.9)</td>
<td>8.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>157.53</td>
<td>2</td>
<td>78.76</td>
<td>2.47</td>
<td>.094</td>
</tr>
<tr>
<td>Error</td>
<td>1623.05</td>
<td>51</td>
<td>31.82</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>1299.10</td>
<td>1</td>
<td>1299.10</td>
<td>313.27</td>
<td>.001</td>
</tr>
<tr>
<td>Interaction</td>
<td>20.89</td>
<td>2</td>
<td>10.44</td>
<td>2.52</td>
<td>.091</td>
</tr>
<tr>
<td>Error</td>
<td>211.49</td>
<td>51</td>
<td>4.15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Repeated measures ANOVAs also were used to evaluate anxiety and depressive symptoms in high and low stress conditions. Subjects in all groups reported significantly higher state anxiety on high stress days than they did on low stress days, $F(1, 51) = 24.27, p < .001$. There were no differences between groups in State-A scores (see Table 5). Subjects also endorsed more depressive symptoms on high stress than they did on low stress days, $F(1, 51) = 7.04, p < .01$, and control subjects reported fewer symptoms than either of the sleep disordered groups, $F(2, 51) = 4.75, p < .01$ (see Table 6).

Differences between groups on the sleep variables were evaluated by repeated measures ANOVAs. The analysis of total time asleep indicated a main effect for stress such that subjects slept significantly fewer minutes in the high stress condition than they did in the low stress condition, $F(1, 51) = 18.26, p < .001$. Furthermore, the ANOVA yielded a significant group effect, $F(2, 51) = 16.57, p < .001$. Post-hoc analysis utilizing Newman-Keuls multiple range test indicated that control subjects acquired significantly more total time asleep than the two sleep disordered groups, whose scores did not differ significantly from each other ($p < .01$, see Table 7). In addition, there was a significant groups by stress level interaction indicating that the stress conditions affected the groups differently. Paired t-tests indicated that control subjects' sleep was more resistant to the effects of stress than either mixed complaint or onset subjects, as their total length of time asleep did not differ significantly between stress conditions. On the other hand, both onset and mixed complaint subjects slept significantly fewer minutes in the high stress condition.
Table 5
Repeated Measures ANOVA With State-A as the Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>38.97 (9.7)</td>
<td>36.54 (7.2)</td>
<td>37.8</td>
</tr>
<tr>
<td>Onset</td>
<td>41.01 (7.7)</td>
<td>35.92 (8.3)</td>
<td>38.5</td>
</tr>
<tr>
<td>Control</td>
<td>35.95 (8.8)</td>
<td>32.99 (10.0)</td>
<td>34.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>312.94</td>
<td>2</td>
<td>156.47</td>
<td>1.14</td>
<td>.327</td>
</tr>
<tr>
<td>Error</td>
<td>6978.26</td>
<td>51</td>
<td>136.83</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>326.81</td>
<td>1</td>
<td>326.81</td>
<td>24.27</td>
<td>.000</td>
</tr>
<tr>
<td>Interaction</td>
<td>35.23</td>
<td>2</td>
<td>17.61</td>
<td>1.31</td>
<td>.279</td>
</tr>
<tr>
<td>Error</td>
<td>686.83</td>
<td>51</td>
<td>13.47</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 6
Repeated Measures ANOVA With Zung Scores as the Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>34.13 (6.5)</td>
<td>33.54 (5.3)</td>
<td>33.8\textsuperscript{a}</td>
</tr>
<tr>
<td>Onset</td>
<td>34.79 (7.1)</td>
<td>33.31 (7.1)</td>
<td>34.1\textsuperscript{a}</td>
</tr>
<tr>
<td>Control</td>
<td>28.62 (5.2)</td>
<td>28.23 (5.5)</td>
<td>28.4\textsuperscript{b}</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>699.04</td>
<td>2</td>
<td>349.52</td>
<td>4.75</td>
<td>.013</td>
</tr>
<tr>
<td>Error</td>
<td>3748.86</td>
<td>51</td>
<td>73.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>16.48</td>
<td>1</td>
<td>16.48</td>
<td>7.04</td>
<td>.011</td>
</tr>
<tr>
<td>Interaction</td>
<td>5.47</td>
<td>2</td>
<td>2.74</td>
<td>1.17</td>
<td>.319</td>
</tr>
<tr>
<td>Error</td>
<td>119.29</td>
<td>51</td>
<td>2.34</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.05.
Table 7
Repeated Measures ANOVA With Total Minutes Asleep as the
Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>319.24 (88.0)</td>
<td>344.76 (85.7)</td>
<td>332.0(^a)</td>
</tr>
<tr>
<td>Onset</td>
<td>343.46 (48.9)</td>
<td>386.37 (54.0)</td>
<td>364.9(^a)</td>
</tr>
<tr>
<td>Control</td>
<td>445.56 (33.4)</td>
<td>442.95 (30.9)</td>
<td>444.5(^b)</td>
</tr>
</tbody>
</table>

**Mean**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>366.63</td>
</tr>
<tr>
<td></td>
<td>388.77</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>239697.23</td>
<td>2</td>
<td>119848.62</td>
<td>16.57</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>368854.16</td>
<td>51</td>
<td>7232.43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>12927.48</td>
<td>1</td>
<td>12927.48</td>
<td>18.26</td>
<td>.001</td>
</tr>
<tr>
<td>Interaction</td>
<td>8984.32</td>
<td>2</td>
<td>4492.16</td>
<td>6.35</td>
<td>.003</td>
</tr>
<tr>
<td>Error</td>
<td>36101.16</td>
<td>51</td>
<td>707.87</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, \(p<.01\)
than they did in the low stress condition, \( t(1, 16) = 3.79, p < .002 \), and \( t(1, 19) = 3.23, p < .004 \), respectively.

A similar repeated measures ANOVA employing number of awakenings during the night as the dependent measure produced only a significant group effect, \( F(2, 51) = 24.19, p < .001 \). Post-hoc analysis revealed that the mixed complaint group awoke significantly more frequently than either the onset or control groups (\( p < .01 \)). There were no significant effects for stress, and no interactions. Means and standard deviations for the number of awakenings are presented in Table 8.

When latency to sleep onset was the dependent measure, only a main effect for group was found, \( F(2, 51) = 19.51, p < .001 \). Subjects in the control group reported the shortest sleep onset latency, followed by the subjects in the mixed complaint group, while subjects in the onset group reported the longest time to sleep onset. All groups were significantly different from each other, (\( p < .01 \), see Table 9).

The ANOVA for time awake during the night resulted in main effects for groups, \( F(2, 51) = 42.41, p < .001 \). Newman-Keuls post-hoc analyses revealed that subjects in the mixed complaint group spent more minutes awake during the night than either of the other groups (\( p < .01 \)), which did not differ significantly from each other. Although the main effects for stress approach significance, by the criterion set for this study, column means cannot reliably be considered significantly different. There was also no significant interaction (see Table 10 for means and standard deviations).
Table 8
Repeated Measures ANOVA With Number of Awakenings as the Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>2.17 (.74)</td>
<td>2.06 (.79)</td>
<td>2.12a</td>
</tr>
<tr>
<td>Onset</td>
<td>.82 (.85)</td>
<td>.71 (.70)</td>
<td>.77b</td>
</tr>
<tr>
<td>Control</td>
<td>.83 (.71)</td>
<td>.78 (.56)</td>
<td>.80b</td>
</tr>
</tbody>
</table>

Mean

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>44.90</td>
<td>2</td>
<td>22.45</td>
<td>24.19</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>47.33</td>
<td>51</td>
<td>.93</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>.24</td>
<td>1</td>
<td>.24</td>
<td>1.66</td>
<td>.204</td>
</tr>
<tr>
<td>Interaction</td>
<td>.02</td>
<td>2</td>
<td>.01</td>
<td>.08</td>
<td>.922</td>
</tr>
<tr>
<td>Error</td>
<td>7.44</td>
<td>51</td>
<td>.15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.01
Table 9
Repeated Measures ANOVA With Latency to Sleep Onset (in Minutes) as the Dependent Measure

<table>
<thead>
<tr>
<th></th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>53.74 (41.9)</td>
<td>48.52 (42.1)</td>
<td>51.1^a</td>
</tr>
<tr>
<td>Onset</td>
<td>87.80 (40.8)</td>
<td>80.95 (57.2)</td>
<td>84.4^b</td>
</tr>
<tr>
<td>Control</td>
<td>9.84 (6.9)</td>
<td>11.20 (7.4)</td>
<td>10.5^c</td>
</tr>
</tbody>
</table>

|                  |            |            |
| Mean             | 51.06      | 46.55      |

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>E</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>93070.10</td>
<td>2</td>
<td>46535.05</td>
<td>19.51</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>121657.75</td>
<td>51</td>
<td>2385.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>537.64</td>
<td>1</td>
<td>537.64</td>
<td>1.06</td>
<td>.309</td>
</tr>
<tr>
<td>Interaction</td>
<td>135.55</td>
<td>2</td>
<td>67.77</td>
<td>.13</td>
<td>.876</td>
</tr>
<tr>
<td>Error</td>
<td>25946.22</td>
<td>51</td>
<td>508.75</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.01
Table 10
Repeated Measures ANOVA With Duration of Awakenings (in Minutes) as the Dependent Measure

<table>
<thead>
<tr>
<th></th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>71.02 (45.2)</td>
<td>58.00 (26.2)</td>
<td>64.5a</td>
</tr>
<tr>
<td>Onset</td>
<td>13.87 (13.5)</td>
<td>8.35 (7.5)</td>
<td>11.2b</td>
</tr>
<tr>
<td>Control</td>
<td>7.12 (6.2)</td>
<td>7.90 (7.1)</td>
<td>7.5b</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>32.91</td>
<td>26.60</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
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<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>76957.06</td>
<td>2</td>
<td>38478.53</td>
<td>42.41</td>
<td>0.001</td>
</tr>
<tr>
<td>Error</td>
<td>46270.40</td>
<td>51</td>
<td>907.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>941.55</td>
<td>1</td>
<td>941.55</td>
<td>4.46</td>
<td>0.040</td>
</tr>
<tr>
<td>Interaction</td>
<td>882.05</td>
<td>2</td>
<td>441.02</td>
<td>2.09</td>
<td>0.134</td>
</tr>
<tr>
<td>Error</td>
<td>10769.55</td>
<td>51</td>
<td>211.17</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.01
Table 11
Repeated Measures ANOVA With Difficulty Falling Asleep as the Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>4.09 (1.56)</td>
<td>3.92 (1.64)</td>
<td>4.01(^a)</td>
</tr>
<tr>
<td>Onset</td>
<td>4.04 (.86)</td>
<td>3.55 (1.28)</td>
<td>3.80(^a)</td>
</tr>
<tr>
<td>Control</td>
<td>1.74 (.57)</td>
<td>1.66 (.68)</td>
<td>1.70(^b)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>114.79</td>
<td>2</td>
<td>57.39</td>
<td>24.09</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>121.51</td>
<td>51</td>
<td>2.38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>1.67</td>
<td>1</td>
<td>1.67</td>
<td>3.26</td>
<td>.077</td>
</tr>
<tr>
<td>Interaction</td>
<td>.80</td>
<td>2</td>
<td>.40</td>
<td>.78</td>
<td>.462</td>
</tr>
<tr>
<td>Error</td>
<td>26.16</td>
<td>51</td>
<td>.51</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.01
When ratings of difficulty falling asleep were evaluated by repeated measures ANOVA, a significant groups effect was revealed, $F(2, 51) = 24.09, p < .001$. Subjects in the control group reported significantly less difficulty falling asleep than either of the two sleep disordered groups ($p < .01$), which did not differ significantly from each other. Again, there were no significant effects for stress, and no interactions (see Table 11). The same pattern of results was obtained for subjects' ratings of restedness upon awakening $F(2, 51) = 26.35, p < .001$ (see Table 12 for means and standard deviations).

Ratings of overall quality of sleep produced a slightly different pattern of ANOVA results. All groups differed significantly from each other [$F(2, 51) = 38.75, p < .001$], such that subjects in the mixed complaint group reported the poorest quality of sleep, followed by subjects in the onset group, and control subjects gave the highest overall evaluation of their nights' sleep. There was also a main effect of stress, in which column means indicate that subjects reported higher quality of sleep in the low stress condition than they did in the high stress condition, $F(1, 51) = 6.86, p < .01$. There was no group by stress interaction. Means and standard deviations are presented in Table 13.

Finally, sleep efficiency was evaluated for differences between groups and between stress levels. The repeated measures ANOVA revealed significant main effects for both factors. Control subjects obtained the highest sleep efficiency score, followed by sleep onset subjects and then mixed control subjects $F(2, 51) = 27.61, p < .001$. Further, across groups, subjects obtained lower sleep efficiency scores (indicative of poorer sleep) in the high stress condition than they did.
Table 12
Repeated Measures ANOVA With Restedness Upon Awakening as the Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>4.74 (.99)</td>
<td>4.49 (1.17)</td>
<td>4.67a</td>
</tr>
<tr>
<td>Onset</td>
<td>4.10 (1.07)</td>
<td>4.04 (1.06)</td>
<td>4.07a</td>
</tr>
<tr>
<td>Control</td>
<td>2.35 (.90)</td>
<td>2.42 (.99)</td>
<td>2.38b</td>
</tr>
</tbody>
</table>

Mean .81 .84

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>96.92</td>
<td>2</td>
<td>48.46</td>
<td>26.35</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>93.81</td>
<td>51</td>
<td>1.84</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>.16</td>
<td>1</td>
<td>.29</td>
<td>.55</td>
<td>.461</td>
</tr>
<tr>
<td>Interaction</td>
<td>.47</td>
<td>2</td>
<td>.24</td>
<td>.81</td>
<td>.449</td>
</tr>
<tr>
<td>Error</td>
<td>14.81</td>
<td>51</td>
<td>.29</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.01
Table 13
Repeated Measures ANOVA With Quality of Sleep as the Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>4.89 (0.90)</td>
<td>4.52 (0.95)</td>
<td>4.71&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Onset</td>
<td>3.87 (1.12)</td>
<td>3.58 (1.11)</td>
<td>3.73&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Control</td>
<td>2.17 (0.68)</td>
<td>2.21 (0.61)</td>
<td>2.19&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>117.25</td>
<td>2</td>
<td>58.62</td>
<td>38.75</td>
<td>0.001</td>
</tr>
<tr>
<td>Error</td>
<td>77.15</td>
<td>51</td>
<td>1.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>1.14</td>
<td>1</td>
<td>1.14</td>
<td>6.86</td>
<td>0.012</td>
</tr>
<tr>
<td>Interaction</td>
<td>.85</td>
<td>2</td>
<td>.43</td>
<td>2.57</td>
<td>0.087</td>
</tr>
<tr>
<td>Error</td>
<td>8.46</td>
<td>51</td>
<td>.17</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.01
in the low stress condition, \( F(1, 51) = 8.92, p < .01 \). There was no

group by stress interaction (see Table 14 for means and standard
deviations).

**Predicting Sleep Variables**

Multiple regression analyses were performed to evaluate the
overall relations between stress, mood and sleep disturbances.
Predictors included in this model were LES (frequency of major life
events over the past year), DSI Event scores, age and Zung SDS
scores. Although State-A was hypothesized to be a significant
predictor of sleep disturbances, it was not initially included in the
regression model because of the substantial relation between it and
Zung scores \( (r = .678) \), discussed earlier. The magnitude of that
association suggested that including both predictors in the equation
would add relatively little new information, or increases in \( R^2 \), as both
may have been measuring similar phenomena (Schroeder, Shoquist and
Stephan, 1986). Consequently, as the Zung scores showed higher
correlations with the dependent measures (and would thus, likely be a
better predictor of those variables), it became the variable of choice.
Although there was no difference between groups in the average age
of subjects, age was included in the regression equation because of
it's established relation to sleep patterns (c.f., Parkes, 1985).

To limit the number of regression analyses, only those
dependent measures which produced significant correlations with
predictor variables were utilized. These variables were: duration of
time awake during the night, latency to sleep onset, difficulty falling
asleep, restedness upon awakening, quality of sleep and sleep
Table 14
Repeated Measures ANOVA With Sleep Efficiency as the Dependent Measure

<table>
<thead>
<tr>
<th>Group</th>
<th>High Stress</th>
<th>Low Stress</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed</td>
<td>.712 (.15)</td>
<td>.756 (.14)</td>
<td>.734a</td>
</tr>
<tr>
<td>Onset</td>
<td>.775 (.08)</td>
<td>.822 (.10)</td>
<td>.799b</td>
</tr>
<tr>
<td>Control</td>
<td>.960 (.02)</td>
<td>.961 (.02)</td>
<td>.961c</td>
</tr>
</tbody>
</table>

Mean .81 .84

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>.98</td>
<td>2</td>
<td>.49</td>
<td>27.61</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>.91</td>
<td>51</td>
<td>.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>.02</td>
<td>1</td>
<td>.02</td>
<td>8.92</td>
<td>.004</td>
</tr>
<tr>
<td>Interaction</td>
<td>.01</td>
<td>2</td>
<td>.01</td>
<td>2.03</td>
<td>.142</td>
</tr>
<tr>
<td>Error</td>
<td>.14</td>
<td>51</td>
<td>.0027</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Column means with different superscripts differ significantly, p<.05
efficiency. In addition, as with the ANOVAs, as several regressions were performed, the significance level was increased to \( p < .01 \), to reduce the likelihood of Type I error. The sleep disordered and control subjects were evaluated together, as the results of all of the relevant ANOVAs failed to yield significant interactions. Although there may have been differences in the level of these variables across groups, (i.e., main effects for groups), subjects responded relatively similarly to stress conditions regardless of group membership, and thus groups could be combined. Data were analyzed by stepwise regression techniques.

Age was the sole independent measure which yielded a significant contribution in the regression predicting duration of awakenings in the night (\( R^2 = .13, p < .01 \)). The overall regression for latency to sleep onset was significant (\( R^2 = .25, p < .001 \)). Event and Zung scores contributed significantly to the prediction of latency to sleep onset, and accounted for 14% and 11%, of the variance in this measure, respectively. Similarly, Event and Zung scores accounted for 19% of the variance in ratings of difficulty falling asleep (\( p < .01 \)). Zung score was the only significant predictive variable associated with restedness upon awakening, (\( R^2 = .19, p < .001 \)), quality of sleep, \( R = .21, p < .001 \), and sleep efficiency, \( R = .14, p < .01 \). Details are presented in Tables 15 through 20.

To determine whether or not State-A contributed any unique variance to the prediction of these dependent variables, it was later added to the regression model. Results for all variables were unchanged. When State-A was entered into the regression equation instead of Zung scores, only results for duration of time awake during
Table 15
Regression of Age, Major Life Events, Event and Zung Scores Predicting Duration of Time Awake During the Night (In Minutes)

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
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<th>F</th>
<th>R</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>1</td>
<td>7008.28</td>
<td>7.43</td>
<td>.354</td>
<td>.009</td>
</tr>
<tr>
<td>Error</td>
<td>52</td>
<td>49056.22</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>56064.50</td>
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</table>

Variables in the equation

<table>
<thead>
<tr>
<th>Age</th>
<th>Beta</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.354</td>
<td>.009</td>
</tr>
</tbody>
</table>
Table 16
Regression of Age, Major Life Events, Event and Zung Scores Predicting Latency to Sleep Onset (In Minutes)

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sum of Squares</th>
<th>F</th>
<th>R</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>1</td>
<td>24027.69</td>
<td>8.31</td>
<td>.496</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>52</td>
<td>73731.66</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>97759.35</td>
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</table>

Variables in the equation

<table>
<thead>
<tr>
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<th>Beta</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event</td>
<td>.367</td>
<td>.004</td>
</tr>
<tr>
<td>Zung</td>
<td>.326</td>
<td>.01</td>
</tr>
</tbody>
</table>
Table 17
Regression of Age, Major Life Events, Event and Zung Scores Predicting Difficulty Falling Asleep

<table>
<thead>
<tr>
<th>Source</th>
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<th>Sum of Squares</th>
<th>F</th>
<th>R</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>1</td>
<td>21.83</td>
<td>6.11</td>
<td>.440</td>
<td>.004</td>
</tr>
<tr>
<td>Error</td>
<td>52</td>
<td>91.19</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>113.02</td>
<td></td>
<td></td>
<td></td>
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</table>

Variables in the equation

<table>
<thead>
<tr>
<th></th>
<th>Beta</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zung</td>
<td>.324</td>
<td>.01</td>
</tr>
<tr>
<td>Event</td>
<td>.292</td>
<td>.03</td>
</tr>
</tbody>
</table>
Table 18

Regression of Age, Major Life Events, Event and Zung Scores Predicting Restedness Upon Awakening

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sum of Squares</th>
<th>F</th>
<th>R</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>1</td>
<td>17.29</td>
<td>12.13</td>
<td>.435</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>52</td>
<td>74.17</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>91.46</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Variables in the equation

<table>
<thead>
<tr>
<th>Zung</th>
<th>Beta</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.435</td>
<td>.001</td>
</tr>
</tbody>
</table>
Table 19
Regression of Age, Major Life Events, Event and Zung Scores Predicting Quality of Sleep

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sum of Squares</th>
<th>F</th>
<th>R</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>1</td>
<td>19.74</td>
<td>13.71</td>
<td>.457</td>
<td>.001</td>
</tr>
<tr>
<td>Error</td>
<td>52</td>
<td>74.19</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>94.65</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Variables in the equation

<table>
<thead>
<tr>
<th></th>
<th>Beta</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zung</td>
<td>.457</td>
<td>.001</td>
</tr>
</tbody>
</table>
Table 20

Regression of Age, Major Life Events, Event and Zung Scores Predicting Sleep Efficiency

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sum of Squares</th>
<th>F</th>
<th>R</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>1</td>
<td>.12</td>
<td>8.71</td>
<td>.379</td>
<td>.005</td>
</tr>
<tr>
<td>Error</td>
<td>52</td>
<td>.69</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>.81</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Variables in the equation

<table>
<thead>
<tr>
<th></th>
<th>Beta</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zung</td>
<td>.379</td>
<td>.005</td>
</tr>
</tbody>
</table>
the night remained unchanged. State-A, unlike Zung scores, contributed significantly (by the criterion defined in this study) only to the prediction of restedness upon awakening ($R^2 = .13, p < .01$). In the regression for latency to sleep onset, Event score was the only significant predictor, accounting for 14% of the variance, ($p < .01$). Similarly, only Event scores contributed significantly to the prediction of difficulty falling asleep, ($R^2 = .08, p < .01$). There were no significant predictors of either quality of sleep or sleep efficiency in these regressions.
Discussion

The present study was designed to investigate the relations between daily stress, mood and sleep disturbances. Broadly speaking, the results of this study are supportive of the hypothesis that the frequency of daily minor stressors impacts the reported severity of global indices of sleep disturbances. Further, the frequency of daily minor stressors and mood, particularly depressive symptoms, are predictive of specific sleep disturbances. These data also indicate that, for the most part, the sleep of normal subjects and insomniacs is not differentially affected by stress.

Differences Between Groups

Based on the results of the repeated measures ANOVAs, differences were demonstrated in the expected directions between groups on all dependent sleep measures. When compared to the two insomnia groups, control subjects generally had the least disturbed sleep (e.g., on measures of latency to sleep onset and frequency and duration of awakenings), and their scores always differed significantly from those achieved by subjects in the mixed group. On the two dependent measures related to sleep maintenance however (number and duration of awakenings), control subjects did not achieve significantly different scores from sleep onset insomniac subjects. Control subjects were the only subjects to achieve a sleep efficiency index in the normal range (Williams, Karacan & Hursch, 1974). Onset and mixed insomniacs obtained indices well below that expected for normal
sleepers (Williams et al., 1974). In essence, these findings serve as a "manipulation check" because they are consistent with inclusion criteria for these groups.

Subjects in the onset and mixed groups were different from one another on five of the eight dependent sleep measures (latency to sleep onset, number of awakenings and duration of awakenings, quality of sleep, and sleep efficiency). With the exception of scores on latency to sleep onset, when onset and mixed complaint subjects differed, mixed complaint subjects achieved scores indicative of greater impairment in sleep. Interestingly, although onset subjects reported sleep onset latencies approximately half an hour longer than mixed subjects, there was no difference between the groups in their subjective ratings of difficulty falling asleep. Thus there is some indication that mixed insomniac subjects may evaluate that sleep phenomenon differently from onset insomniacs.

It is noteworthy also that onset and mixed subjects were not found to differ significantly in their total time asleep, yet mixed subjects rated the quality of their night’s sleep significantly more negatively than onset subjects. Although for both groups the same total amount of sleep was achieved, it was obtained in a different fashion for each group. Onset subjects had greater sleep onset latencies than mixed subjects, and mixed subjects were more likely to both awaken during the night, and stay awake for longer periods than onset subjects (again, this was consistent with inclusion criteria). This structural inequality may be the source of the difference in quality of sleep ratings. Simply stated, although onset subjects took more time to fall asleep, they were more likely to remain asleep than
mixed subjects. While ratings of quality of sleep are likely affected by total amount of sleep achieved, fragmentation of sleep also is likely a significant factor in the evaluation the quality of sleep (Williams et al., 1974). These findings are consistent with those of Baekeland and Hoy (1971), who demonstrated a relation between quality of sleep and the number of awakenings that a subject experiences during the night.

Effects of Stress on Sleep

Across subject groups, stress differentially affected three sleep scores: Total minutes asleep, quality of sleep and sleep efficiency, such that all scores indicated greater sleep impairment in the high stress condition than they did in the low stress condition. Because there was a significant group by stress interaction involving total time asleep, these results will be discussed in detail below (cf., McCall, 1990). Across stress conditions, subjects appeared to have a higher quality of sleep, and greater sleep efficiency (less time in bed awake) on nights following low stress days. Conversely, they reported sleeping more poorly and less efficiently on nights following high stress days. Similar findings for quality of sleep were obtained when college students’ sleep was evaluated utilizing comparable methodology (Rubman et al., 1988), though this finding is not universal. When Goreczny, Brantley, Buss and Waters (1988) examined quality of sleep in asthmatics and COPD patients, they found no differences between scores on high and low stress days.

Unexpectedly, other specific symptoms of insomnia (frequency of awakenings, latency to sleep onset) did not differ significantly
between high and low stress conditions. There is a strong trend in the expected direction for scores on duration of awakenings, but by the significance criteria determined for this study, the scores cannot be deemed significantly different. This finding may be related to the size of the current sample. More subjects would have produced greater power, which could potentially have yielded significant effects.

Data from this study would suggest that stress similarly impacts the sleep of onset insomniacs, mixed complaint insomniacs and normal sleepers, with one notable exception; total time asleep. For normal control subjects, the total number of minutes slept did not vary as a function of stress condition; they obtained slightly less than 7 1/2 hours of sleep per night regardless of stress condition. For both sleep disordered groups, however, stress conditions produced a statistically significant change in total time asleep. Onset subjects slept an average of 43 minutes less (12% of their mean total time asleep) in the high stress condition than in the low stress condition, and mixed complaint subjects slept an average of 26 minutes less (or 7.7% of their mean total time asleep) in the high stress condition than in the low stress condition.

There are then two issues of concern here. The first is at what point or points on the high stress nights (e.g., sleep onset or sleep maintenance) are sleep disordered subjects awake more than control subjects? In the absence of another significant interaction, the answer is unclear. The number of minutes slept reflects all of the time spent awake during the night, and is thus a global measure of sleep disturbance. It may be that relatively high levels of stress cause small increases in all types of awakenings, (cause a general
tendency to remain awake), not targeting a specific increase in sleep onset latency or number or duration of awakenings. It may also be the case that for some but not all individuals, relatively high stress increases sleep onset latency, number of awakenings or duration of awakenings, and thus the effect was not strong enough to produce significant results.

Another possible explanation for these findings may be related to subjects' frequency of daily minor stressors (reflected by Event scores). Although, as discussed above, the mean Event score for all subjects was within the normal range, the mean Event score for the high stress condition was 13.79, which is the equivalent of a T-Score of 53 (using normative data for adults; Brantley & Jones, 1989). It may be that it takes a higher or more extreme level of daily stress to effect a significant change in specific symptoms of insomnia. Given a higher level of daily stress, sleep onset latencies, number of awakenings and/or duration of awakenings may have been increased thereby producing significant stress by group interactions. These explanations are not mutually exclusive, and as such it is conceivable that the answer is a combination of these possibilities.

The second issue that warrants discussion is the absence of significant correlations involving total time asleep with stress, anxiety or depression scores and it's consequent exclusion from the regression analyses. The significant main effects and interaction, in the absence of correlations suggests that there is an effect of stress on total time asleep, but that stress may effect some individuals more than others, as mentioned above (cf., McCall, 1990). It does not appear that the presence or absence of insomnia defines these possible subgroups as
there were no significant correlations involving individual subject
groups and total time asleep. There still, however, may be a
moderating or mediating variable not assessed in this study which
impacts subjects' resistance or vulnerability to the effects of stress
on sleep. This line of speculation would be consistent with other
research which suggests that traits or characteristics of individuals
are likely to influence both the reporting of stressors and physical
symptoms (Kobassa, Maddi & Courington, 1981; Watson & Pennebaker,
1989). Personality factors, coping strategies, and social support are
some of the variables that have been examined in relation to stress
and physical disorders (DeLongis, Folkman & Lazarus, 1988; Folkman,
Lazarus, Dunkel-Schetter, DeLongis & Gruen, 1986; Kobassa, et al.,
1981; Waters & Rubman, 1990). It is logical to suspect that these
factors may operate significantly with insomniacs as well.

Prediction of sleep variables

The results of the regression analyses in general suggested that
there are significant relations between the frequency of daily minor
stressors, depressive symptoms and sleep variables. Specifically,
stress and depressive symptoms each contributed substantially to the
prediction of latency to sleep onset and ratings of difficulty falling
asleep. These results are rather noteworthy given that stressors and
depressive symptoms accounted for 25% of the variance in time to
sleep onset and 19% of the variance in difficulty falling asleep. The
largest portion of variance in latency to sleep onset was accounted for
by frequency of daily minor stressors (14%). The largest portion of
the variance in ratings of difficulty falling asleep was accounted for by depressive symptoms (11%).

As these two dependent measures are theoretically related, it is logical that there is consistency in the variables that predict them. These data are consistent with the results obtained by Haynes and his colleagues (1981) who found that estimates of sleep onset latency were strongly affected by a pre-sleep cognitive stressor. In addition, these findings also support those of Reynolds et al., (1989) and Gillen et al., (1981) who found that frequency of depressive symptoms contributed to latency to sleep onset.

It may be observed from these data that depressive symptoms (as measured by Zung scores) are a strong and consistent predictor of scores on sleep variables, and particularly of evaluations of a night's sleep. It must be recalled that subjects included in this sample were not clinically depressed, thus, these data support the hypothesis that fluctuations in depressive symptoms (not clinical depression) are related to fluctuations in sleep variables. These results may not be inconsistent with an arousal theory of insomnia, as inspection of the Zung SDS reveals that many items are indicative of activation or excitement.

Unexpectedly, anxiety (as assessed by the State-Trait Anxiety Inventory, State form; State-A) did not show all of the hypothesized relations to the sleep measures. Despite the substantial amount of shared variance between anxiety and depression measures, it appears that the unique variance associated with anxiety scores is not very predictive of sleep disturbances in this sample. This is somewhat inconsistent with other research, linking anxiety symptoms to latency
to sleep onset and frequency and duration of awakenings (Coursey et al., 1975; Freedman & Sattler, 1982; Haynes et al, 1974; Haynes et al, 1985), and this raises a significant issue. As discussed earlier, items on the Zung reflect four domains of depressive symptoms; affective disturbances, psychological disturbances, physiological disturbances and psychomotor disturbances (Zung, 1965). The State-A items were designed to evaluate "feelings of apprehension, tension, nervousness and worry" (Speilberger et al., 1983, p. 2). It is unknown exactly where the overlap between the two measures lies, consequently it is difficult to pinpoint precisely the component that relates to sleep disturbances. In terms of the implications for this study, it may be the case that the that variance specific to depression, not associated with anxiety is the crucial factor in relation to sleep. It may also be, however, that the Zung assesses physiological arousal more directly than the State-A, thus accounting for the present findings.

The frequency of major life events in the year prior to the onset of subjects' monitoring did not account for a statistically significant portion of the variance in any of the sleep variables in this study. This findings help clarify the role of major life events in insomnia, when taken in conjunction with results obtained by Healey et al. (1981), who suggested that major life events are important in the onset of chronic insomnia. Data from the current study do not contradict Healey et al.'s conclusions about the onset of chronic insomnia, but they do imply that the frequency of major life events over a period of a year does not significantly influence the maintenance of insomnia symptoms during a three week period at the end of that year.
In a related vein, the absence of a significant correlation between frequencies of major and minor stressors is somewhat at odds with the results of other studies (Kanner et al., 1981; DeLongis et al., 1982; Jones, 1987). Major and minor stressors share 3% of their variance in this study, as opposed to 11% in others (e.g., Jones 1987). Although the obtained Event scores were relatively normally distributed (skew = .56), as discussed above, their mean was slightly lower than would be expected. In addition, the range for this sample (3-23) is somewhat smaller than it is for either normal subjects (range 0-58) or medical subjects (range 0-34; Brantley & Jones, 1989). Thus, these scores may be representative of the population of insomniacs and normal sleepers who volunteer for studies of this sort, but not representative of all stress scores. If a broader range of scores had been obtained, perhaps there would have been a significant relation between major and minor life events.

Overall, these findings are still somewhat supportive of those models which include a component of physiological activation as a causal agent in insomnia (e.g., Bootzin, 1974; Haynes et al., 1981). It is plausible that the mechanism by which this is accomplished is a generalized arousal response involving activation of the ascending reticular activating system (ARAS) which is generally accepted as the primary wakefulness system (Parkes, 1985). The stress-arousal theory (and a great deal of clinical lore) holds that stress is responsible for producing the cognitive and physiological arousal that maintains wakefulness via ARAS inhibition of sleep. In a simple model, it would be expected then that insomniacs should experience more stressors than normal sleepers. This hypothesis was not borne out by the
present findings. However, an extrapolation from the stress literature may be helpful. With the inclusion of minor events in stress research, investigators have cited the importance of taking measurements more frequently, suggesting that the temporal proximity of the minor stressors to outcome measures of disorder would increase the associations of stress to the disorder (Kanner et al., 1981). Now, if stressors do, in fact, cause increased ARAS activation, the question is how long do the effects of ARAS activation last? If the effects are short-lived, and they occur early in the day there may be no reason for them to directly influence sleep that night. Perhaps it is the frequency of stressors that occur within a short interval of going to bed that is crucial to the equation. If the insomniacs in this study experienced the majority of their stressors shortly before bedtime, then it is conceivable that their sleep would be more strongly effected than the controls’ whose equal frequency of stressors should theoretically be distributed throughout the day. This issue is relatively easily resolved by having respondents indicate the time of day that they experienced their stressors, and evaluating that data accordingly.

Holding still to the stress-arousal paradigm for a moment, a second, and equally important component of this model should be considered; the possible cognitive sequelae elicited by stressors. It may not be the time or frequency of stressors that is important, it may be cognitions related to those stressors that affect sleep. At the end of the day, individuals may think about the events that occurred during the day, in essence, extending the effects of these events. Indeed clinically, it is quite common for insomniacs to report that they
"can't shut off their minds" when attempting to go to sleep. Arousal may then be produced as a result of these cognitive events, thus disrupting sleep. This is consistent with Borkovec's work (Borkovec et al., 1975; Borkovec et al., 1979; Borkovec & Fowles, 1973; Borkovec, 1982; Van Oot et al., 1985), and Woolfolk & McNulty (1983) in which they concluded that the most important component of relaxation techniques in the treatment of insomnia is the cognitive distraction which occurs during the tasks.

Alternatively, stress may effect sleep via compliance with good sleep hygiene practices. It is conceivable that when subjects experience a greater number of stressors and a greater frequency of depressive symptoms, they exhibit poor sleep hygiene. It is easy to imagine that after a (relatively) stressful day, individuals decide to go to bed earlier than usual even if they are not sleepy, but thinking that they need added rest that night. If subjects cannot fall asleep immediately, this could increase reported latency to sleep onset. If subjects do fall asleep earlier than usual, they may awaken earlier than usual, and thus report this as an increase in their time awake during the night, or both. These effects would reduce sleep efficiency and be consistent with the findings of this study.

Finally, the possibility exists that the reason that more effects for stress on sleep were not found is related to the stimulus control theory. The sleep of the insomniacs in this study may be so strongly conditioned that there is a ceiling effect on sleep disturbances. More simply put, stimuli in the bedroom may have become so aversive (and thus arousing) through repeated associations of wanting to sleep and not being able to, that sleep is unlikely regardless of the level of
stress. The effectiveness of treatments aimed at establishing more appropriate stimulus control and sleep hygiene (Lacks et al., 1983, Lichstein & Fischer, 1985; Morin & Azrin, 1987; Turner & Ascher, 1979) lend support to this contention.

The results of this study have modest clinical implications; they do not suggest dramatically changing the structure of effective treatment programs as they currently exist. They do suggest that although the frequency of daily minor stressors and depression (as indicated by the Zung) are somewhat important, their reduction should not be the sole focus of an intervention to aid insomnia. More appropriately, these factors should be incorporated into a more comprehensive treatment program which includes components of stimulus control and cognitive distraction techniques. Should future research bear out the significance of hypotheses generated from this study, then perhaps a larger role for these variables would be warranted in treatment.

While the findings in this study promote opportunities to speculate and develop additional hypotheses, it is important to recognize that from these data it cannot specifically be determined that frequency of daily stressors or anxiety or depressive symptoms causes sleep disturbances. It is not difficult to imagine an argument for "reciprocal causality," (Cohen & Cohen, 1983) in which the lack of sleep produces it's own stressors (e.g., getting a poor night's sleep leads to increased fatigue or irritability and thus interferes with social relationships). Two points must be made, however about reciprocal causality. First, causality cannot move backward in time; a night of poor sleep cannot be said to have caused an increased
frequency of minor stressors on the day prior to that sleep period. Second, if reciprocal causality were at work in this data, then there should have been differences between groups in the frequency of minor stressors. Either that, or if insomnia was producing its own stressors then there was a phenomenon of equal intensity influencing stress scores for the control subjects.

The current findings must be evaluated within the limitations of this study. First, it must be recognized these data are drawn from a sample of volunteers. It is conceivable that this introduces a bias in the data, as individuals who were experiencing extreme levels of stress or exceptional sleep disturbances at the time of recruitment for the study may have elected not to respond, thus limiting the generalizability of these findings. There may also be a bias with regard to the control subjects, their reinforcement for participating in this project is not as apparent as the insomniacs' (free treatment), and this may make them an unusual group of individuals. In addition, because of the inclusion criteria, the sample is comprised of normal sleepers and relatively severe insomniacs, leaving a population of less severe or "occasional" insomniacs unstudied. Consequently, it is unknown how or if the findings of this study can be applied to this group. Finally, the current sample was predominantly female. Although females are more likely than males to complain of insomnia (and thus comprise a larger portion of the population of insomniacs), the possibility exists that there is a gender-related difference in the effects of stress and mood on sleep. If this is the case, then these findings may be most applicable to female subjects. Further research addressing this issue would be of value.
This research has lent modest support to the hypotheses that stress and mood influence sleep, but has raised other questions and issues as well. The relations between stress, mood and sleep are rather more complex than they initially appeared. The results of this study are multifaceted and are consistent with a theory that includes increased autonomic arousal as a causal agent in sleep disturbances. In addition, there may be homogeneous subgroups of individuals within normal and sleep disordered populations for whom stress and mood impact sleep through different mechanisms. Future research should be directed toward uncovering factors which define these subgroups and moderate or mediate the effects of stressors and mood on sleep.
References


Appendix A

Demographic Questionnaire
Demographic Information

Name______________________ Age__________

1. Sex (circle): M   F

2. Marital Status: single married divorced widowed separated

3. Number of people living in household (including yourself)_____

4. Education completed (circle one):
   1. Graduate or Professional Training
   2. College or University Degree
   3. Partial College Training
   4. High School Graduate
   5. Partial High School
   6. Junior High School
   7. Less than Seven Years of School

5. Occupational Information: Please circle the number next to the best description of your job position and responsibilities.
   1. Executive of Large Company (President, Mayor, Director)
      Large Business Owners
      Professionals (C.P.A., Engineer, Lawyers, M.D.)
   2. Business Managers of Large Concerns (Branch Manager)
      Proprietors of Medium Sized Businesses
      Professionals (Pharmacists, Teachers, Accountants)
   3. Administrative Personnel (Insurance Adjuster, Store Manager)
      Small Business Owners
      Semi-Professionals (Actors, Programmers, Commercial Artist)
   4. Clerical and Sales Workers (Clerk, Stenographer, Salesman)
      Technicians (Dental or Laboratory Technician, Draftsmen)
      Little Business Owner
      Farm Owner
   5. Skilled Manual Employee (Auto Repair, Fitter, Fireman, Stylist)
      Small Farmers
      Semi-Skilled Employees (Guards, Welders, Bartender)
      Farm Workers
   7. Unskilled Employees (Janitor, Laborer)
6. Please circle all that apply to you:

1. Full Time Job
2. Part Time Job
3. Homemaker
4. Student
5. Unemployed
6. Retired
7. Second Part Time Job
8. Second Full Time Job

7. Annual Income Level of Household

8. Race (please circle):

1. White
2. Black
3. Hispanic
4. Oriental
5. Other

9. Have you been diagnosed as having any chronic or serious illnesses in the past 6 months?

10. What illness?

11. Are you currently taking any prescription medications?

12. What medications?

13. Does anyone in your immediate family complain of problems falling asleep? (circle one) YES NO

14. Does anyone in your immediate family complain of problems staying asleep or waking up early? (circle one) YES NO
Appendix B

Medication Release Form
Discontinuation of Medication Consent Form

I, ______________________, have spoken with my physician, ______________________, and it is with his/her informed consent that I will refrain from taking my sleep medication ______________________ for the time that I am participating in the sleep project at the LSU Medical Center Stress and Chronic Illness Clinic.

Signature ______________________ Date __________

Witness ______________________ Date __________
Appendix C

Daily Sleep Diary
Daily Sleep Diary

1. At what time did you begin trying to fall asleep last night? _______

2. How many minutes did it take you to fall asleep last night? _______

3. How many times did you wake up during the night? _______________________

4. Please record how long you were awake (in minutes) for each occurrence listed above in question number 3.
   _______   _______   _______   _______

5. What is the total number of hours and minutes you slept last night?
   Hours _____ Minutes ______

6. How difficult was it for you to fall asleep last night?
   _______-2-_______-3-_______-4-_______-5-_______-6-_______-7
   not very difficult extremely
difficult

7. How rested do you feel this morning?
   _______-2-_______-3-_______-4-_______-5-_______-6-_______-7
   very poorly
   rested

8. Rate the quality of last night's sleep.
   _______-2-_______-3-_______-4-_______-5-_______-6-_______-7
   excellent very poor

9. Were you awakened by any external causes (e.g., baby crying, phone call, etc.)? Please specify. ____________________________

10. Did you drink alcohol within 4 hours of retiring? . . . . Yes No

11. Did any activity cause you to lose sleep? .......... Yes No

*****Please mail this and your other monitoring forms daily*****
Appendix D
Telephone Screening Form
Insomnia Study Telephone Screening Form

Name:_________________________  Sex:  M  F  Age:_____

Phone: (h)____________________  (w)_____________

Address:_____________________________________________________

Occupation:___________________________________________________

Please describe the nature of the symptoms (e.g., sleep onset insomnia, early morning awakening, etc.) __________________________

________________________________________________________________

How frequently does it occur? _____________________________________

When did this problem begin? ____________________________________

What (if any) treatments have you sought for the problem? _______________

________________________________________________________________

Have you ever been diagnosed with a sleep problem other than insomnia?  ____________________________

Are you currently under the care of a psychologist or psychiatrist (or other mental health professional)? ______________________________

Do you have any chronic medical conditions (e.g., Parkinson's, severe arthritis)? ______________________________________________

Does your employment cause you to do shiftwork? _____________?

What is the best time to contact you?

Date __________________________
Appendix E

Informed Consent Form
INFORMED CONSENT

I, ____________________________, freely and willingly consent to be a participant in a research project investigating sleep disturbances.

I agree to participate in a 21 day assessment period during which time I will be asked to monitor my daily stress, mood and sleep. I understand that I will be asked to mail my monitoring forms back daily. In addition, I understand that I must attend one appointment each week during the assessment period. In return for my efforts I will be able to participate in a free treatment program for my sleep disturbance.

I understand that I may withdraw from this project at any time with no adverse consequences. In addition, any information that I provide during this project will be kept in strict confidence, and if the information is presented publicly (e.g., at a conference, in a journal article) no information will be identified with me personally. I realize that I have a right to ask questions at any time and to have these questions answered to my satisfaction. I have read and thoroughly understand this consent form.

________________________________________  _____________
Participant                                    Date

________________________________________  _____________
Witness                                      Date
VITA

SUSAN RUBMAN, Ph.D.

PERSONAL DATA

Address: Department of Medicine
Sleep Disorders Center
Mount Sinai Hospital
500 Blue Hills Avenue
Hartford, Connecticut, 06112

Telephone: Office: (203) 286-4715
Home: (203) 232-6003

CURRENT POSITION

Behavioral Medicine Consultant
Department of Medicine
Sleep Disorders Center
Mount Sinai Hospital
Hartford, Connecticut

EDUCATION

1986-1990
Louisiana State University
Baton Rouge, LA 70808
Major: Clinical Psychology
Specialty Area: Behavioral Medicine
Minor: Behavioral Neurology
Ph.D. 1990

Dissertation: The Relations Between Stress, Mood and Insomnia.

1984-1986
Louisiana State University
Baton Rouge, LA 70808
Major: Clinical Psychology
M.A., August, 1986

Thesis: Sex differences in psychophiology with the sex of the experimenter controlled.

1980-1984
Union College
Schenectady, NY 12308
Major: Psychology
B.S. with Departmental Honors, June, 1984

CLINICAL EXPERIENCE

September 1990 to present

Behavioral Medicine Consultant: Sleep Disorders Center, Mount Sinai Hospital, Hartford, CT. Primary responsibilities include assessment of in- and out-patient sleep related disorders, with an emphasis on disorders of initiating and maintaining sleep. Behavioral treatment is provided on both an individual and group basis. Additional activities include interpreting polysomnographic assessments, assessment and treatment of other psychophysiological disorders, consultation to specific disciplines within the Department of Medicine. Administrative responsibilities include coordinating bi-monthly Sleep Rounds for the Center's treatment team and consultation/ liaison with physicians referring to the Center.

August 1989 to August 1990

Psychology Resident: University of Mississippi Medical Center/Veterans Affairs Medical Center, Clinical Psychology Residency Training Consortium, Jackson, Mississippi. APA approved internship program.

6 months

Behavioral Medicine/Psychophysiology Rotation (UHC). Primary rotation activities include assessment and treatment of in- and out-patient stress-related and psychophysiological disorders, particularly recurrent headache and chronic pain. Specific rotation activities as a Headache Clinic staff member include leading treatment groups and participating in research with headache patients. Additional activities include conducting behavioral medicine consultation-liaison services. Supervisor: Donald B. Penzien, Ph.D.

3 months

Behavioral Medicine/Addictive Behaviors Rotation (VAMC). Responsibilities include assessment and treatment services for a variety of in- and out-patients. Primary referrals are for cardiovascular risk modification, chronic pain and adjustment to chronic disease. As Smoking Clinic team member perform smoking evaluations, lead smoking cessation programs and participate in research with veterans who smoke. Additional duties include evaluation of visually impaired veterans for determination of rehabilitation services. Supervisor: Thomas J. Payne, Ph.D.

3 months

Behavioral Gerontology (VAMC). Rotation responsibilities involve consultation to a variety of medical services within the VAMC including the geriatric outpatient clinic, geriatric consultation service, and nursing home care service. Specific duties include neuropsychological screening, differential evaluation of dementia and depression, assessment of adaptive living skills, facilitating compliance with medical regimens and training nursing home staff. Supervisor: Phillip R. Godding, Ph.D.
February 1988 to July 1989

Clinical Extern: Rehabilitation Unit, Our Lady of the Lake Regional Medical Center, Baton Rouge, LA. Member of multidisciplinary treatment team on 40 bed rehabilitation unit. Patient population included CVA, head injury, spinal cord injury, surgical joint replacements, etc. Responsibilities included evaluations (e.g., neuropsychological screening), cognitive rehabilitation and adjustment to disability groups, family conferences, weekly staffings. Supervisors: Phillip J. Brantley, Ph.D. and W. Drew Gouvier, Ph.D.

Sept 1987 to July 1989

Clinical Assistant: Behavioral Science Consultants, Baton Rouge, LA; an outpatient behavioral medicine center. Conducted behavioral and objective assessments, participated in treatment programs including social skills training, relaxation training, biofeedback and a variety of other behavioral and cognitive-behavioral techniques with a general outpatient population. Supervisors: Phillip J. Brantley, Ph.D. and Stanford W. Granberry, Ph.D.

June 1987 to July 1989

Chief Psychology Extern: Stress and Chronic Illness Clinic, LSU Medical Center. Assisted in program and research design, development of treatment packages, and public relations for the newly formed clinic. Projects included headache and insomnia treatment programs. Supervisor: Phillip J. Brantley, Ph.D.

June 1986 to July 1989


August 1986 to June 1987

Co-leader Parent Training Group: Psychological Services Center, Louisiana State University, Baton Rouge, LA. Conducted assessments and behavioral group treatments of both home and school-based problems. Techniques utilized included time-out and response cost programs, as well as communication and problem solving skills. Supervisor: Mary L. Kelley, Ph.D.

August 1985 to June 1986

Pediatric Psychology Trainee: Earl K. Long Memorial Hospital, Baton Rouge, LA. Conducted intellectual, behavioral and objective psychological evaluations for physician referred in-patient and out-patient low SES children, adolescents and their parents. Treatment experiences consisted mainly of behavioral or cognitive-behavioral programs. Supervisor: Mary L. Kelley, Ph.D.
August 1985 to December, 1985  
**Pediatric Psychology Trainee:** Earl K. Long Memorial Hospital, Baton Rouge, LA, High Risk Clinic. Developmental assessment of high risk infants and young children. Other duties included report writing, and developing suggestions for parents for remediation of skills deficits in high risk infants. **Supervisor:** Mary L. Kelley, Ph.D.

Sept. 1985 to March 1986  
**Clinical Extern:** Baton Rouge Psychological Associates, Baton Rouge LA; private practice. Conducted projective and objective personality assessments, psychoeducational evaluations, and neuropsychological screenings for adult and child out-patient clients. Treatment experiences included parent training. **Supervisor:** Arthur Rosenkrantz, Ph.D.

August 1984 to June 1985  
**Student Therapist:** Psychological Services Center, Louisiana State University, Baton Rouge, LA. Conducted psychological assessment and behavioral and cognitive-behavioral treatment of adults, in an out-patient psychology clinic. **Supervisor:** William F. Waters, Ph.D.

Sept. 1983 to May 1984  
**Psychology Assistant:** CARA Club, Ellis Hospital, Schenectady, NY. Off-premises day program for chronic schizophrenics and halfway house residents. Duties included supervising patient activities and implementing vocational and treatment programs. **Supervisor:** Timothy H. Dowling, Ph.D.

Sept. 1982 to December 1982  
**Psychology Extern:** Oswald D. Heck Center for the Developmentally Disabled, Schenectady, NY. Duties included implementing token economies, and vocational, language and self-maintenance skills programs to autistic children and adults in an inpatient setting. **Supervisor:** Suzanne Benack, Ph.D.

**CLINICAL SUPERVISION EXPERIENCE**

June 1987 to July 1989  
**Chief Psychology Extern,** Consultation-Liaison Service, Earl K. Long Memorial Hospital. Assisted in direct supervision of clinical psychology graduate students, and responsible for coordination of patient referrals and case disposition. The service receives over 300 cases per year, exhibiting the full range of psychopathology. **Supervisor:** Phillip J. Brantley, Ph.D.

**TEACHING EXPERIENCE**

August 1989 to August 1990  
**Instructor of Record,** Psychiatry 612: Clinical Interviewing for Medical Undergraduate Students, University of Mississippi Medical Center, Jackson, MS. Didactic instruction of interviewing techniques including mental status exams, patient history, evaluation of presenting problems. Supervised student/patient interviews.
June 1987 to July 1989

Instructed and supervised Family Practice Residents in psychological interviewing and assessment. Provided training for residents with a general outpatient psychological population. Training culminated in formal case presentations. Specific skills targeted for development included mental status examinations, and effective utilization of psychological referral sources. Supervisor: Phillip J. Brantley, Ph.D.

August 1986 to June 1988

Teaching Assistant: Psychophysiology Laboratory, Psychology Department, Louisiana State University, Baton Rouge, LA. Duties included teaching graduate students and research technicians how to perform physiological hook-ups, assisting in laboratory research design, coordinating data collection and analysis, polygraph maintenance. Director: William F. Waters, Ph.D.

August 1984 to June 1985

Teaching Assistant: Psychological Services Center, Louisiana State University, Baton Rouge, LA. Duties included overseeing case management and disposition for three adult practicum teams, organizing test library, scheduling intakes, and audio-visual equipment overseer. Director: Donald A. Williamson, Ph.D.

January 1984 to June 1984

Teaching Assistant: Introductory Psychology Class, Psychology Department, Union College, Schenectady, NY. Duties included giving lectures, writing test questions, writing study guides for students, holding weekly study groups, and proctoring exams. Instructor: Jack P. Lipton, Ph.D.

WORKSHOPS, SYMPOSIA AND INVITED ADDRESSES

May 1990

Minor Stress and Illness. Symposium to be presented at the annual meeting of the Society of Teachers of Family Medicine, Seattle, WA.

January 1990

Daily Stress and Insomnia. Invited address to be presented at VA Research Rounds, VAMC, Jackson, MS.

August 1989

Professional Burnout--Stress Recognition and Management. Workshop presented at the Annual Pharmacy Clinical Preceptors Seminar, School of Pharmacy, University of Mississippi Medical Center.

May 1989

Stress and Illness. Invited address presented at the Annual Family Practice Update meeting, New Orleans, LA.
June 1987 to July 1989 Behavioral Science Conference series for Family Practice Residents. Recruited lecturers and presented addresses for this weekly conference program. Topics included neuropsychological screening, behavioral interventions with gastro-intestinal disorders, sleep disorders, headache, and agoraphobia, methodology for single case design, and clinical case presentations.

December 1987 to November 1988 Stress Management Seminars. Full day workshops for industrial employees, Baton Rouge, LA.

CONSULTANTSHIPS

May 1988 to May 1989 Quality Care II Nursing Care Facility, Baton Rouge, LA. Consultant to physicians, nurses and staff of this geriatric facility. Performed evaluations, staff training in the care of patients with psychological and behavior problems. Supervisor: Phillip J. Brantley, Ph.D.

June 1986 to July 1989 Earl K. Long Memorial Hospital, Baton Rouge, LA. Consultant to Family Practice residents and physicians, and general hospital medical faculty and staff. Supervisor: Phillip J. Brantley, Ph.D.

August 1985 to June 1986 Earl K. Long Memorial Hospital, Baton Rouge, LA. Consultant to pediatric out-patient clinic medical residents and physicians. Supervisor: Mary L. Kelley, Ph.D.

PROFESSIONAL ORGANIZATIONS

Association for the Advancement of Behavior Therapy
Society of Behavioral Medicine
Southeastern Psychological Association

HONORS, AWARDS & STIPENDS

1986 Elected representative to Clinical Training Committee.
1985 Awarded research presentation funds, Louisiana State University.
1984 John Lewis March Prize, Union College, Schenectady, NY. Awarded to a graduating senior for performance in psychology.
1984 Awarded Psi Chi Research Certificate.
1980 Awarded Regents Scholarship by the State of New York.
**GRANTS**

May 1989  The relations between stress, mood and insomnia.  
Principal Investigator  
Louisiana State University Medical Center, New Orleans, LA  
$1500

Principal Investigator  
Union College Internal Education Fund, Schenectady, NY  
$250

**PUBLICATIONS**

Pseudocyesis and depression: Etiological and treatment considerations.  
Journal of the Louisiana State Medical Society.  
141(11) 39-42.

**MANUSCRIPTS SUBMITTED FOR PUBLICATION**


Rubman, S., Waters, W.F. & Dobbins, G.H.  Gender differences in psychophysiology with the gender of the experimenter controlled.  Manuscript submitted for publication.


**PRESENTATIONS**


RESEARCH IN PROGRESS


Brantley, P.J., Rubman, S. and others. Relative efficacy of stress management vs stimulus control treatment paradigms in insomnia.

Brantley, P.J., Rubman, S. and others. The role of attributional style and personality variables in insomnia.

REFERENCES

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Thomas J. Payne, Ph.D.
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1500 E. Woodrow Wilson Avenue
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(601) 362-4471 ext. 1153
Candidate: Susan Rubman

Major Field: Psychology

Title of Dissertation: The Relations Between Stress, Mood and Insomnia

Approved:

[Signature]
Major Professor and Chairman

[Signature]
Dean of the Graduate School

EXAMINING COMMITTEE:

[Signature]

[Signature]

[Signature]

Date of Examination: November 9, 1990