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The Effect of Psychosocial Stress on Sleep: A Review of Polysomnographic Evidence

Eui-Joong Kim and
Department of Psychiatry Eulji University School of Medicine, Daejeon, Korea

Joel E. Dimsdale
Department of Psychiatry University of California, San Diego

Abstract

This systematic review examines the effect of diverse psychosocial stressors on polysomnographic measures of sleep. Sixty-three articles were located and categorized in terms of the types of stressors imposed. Experimental stress resulted in fairly consistent changes: decreases in slow wave sleep, REM sleep, and sleep efficiency (SE), as well as increases in awakenings. Data were limited in terms of response to non-experimental stressors, except for the case of post-traumatic stress disorder (PTSD) on sleep, where a number of reports suggest that PTSD patients have increased awakenings and decreased SE. Future research needs to define stress more precisely in terms of duration and severity and to measure its impacts on sleep in populations that differ in terms of age, comorbid illness, gender, and so forth. Without such fine-grained analyses, it is difficult to draw definitive conclusions about this important area.

Sleep researchers traditionally have focused on primary sleep disorders such as sleep apnea, narcolepsy, or periodic limb movement disorder; but common knowledge has it that the stresses of daily life can perturb even healthy sleep. As Mason (1968) described, psychological stressors are potent stimuli for physiological stress responses. Most studies on psychosocial stress and its physiological effects on sleep are based solely on subjective reports. However, these subjective reports of sleep are not sufficient to evaluate physiological sleep changes as measured by polysomnography (PSG; Akerstedt, Hume, Minors, & Waterhouse, 1994; Schneider-Helmert & Kumar, 1995). What really happens in sleep physiology after exposure to stressors? Although the gold standard of measurement of sleep is PSG, there are relatively little data concerning the effects of ordinary or severe stress on polysomnographically examined sleep. This article systematically reviews the diverse literature in this area.

Stress is notorious for being difficult to define. It encompasses all kinds of stimuli of varying amounts of aversiveness and duration (Dimsdale, Irwin, Keefe, & Stein, 2005). Kecklund and Akerstedt (2004b) grouped stressors conceptionally into physical versus psychological or social, acute versus chronic, and high intensity versus low intensity.

Correspondence should be addressed to Joel E. Dimsdale, University of California, San Diego, 9500 Gilman D., La Jolla, CA 92093-0804. jdimsdale@ucsd.edu.

Although there are numerous animal models of social stress, it is still challenging to generalize psychosocial stress responses in animals to those in humans. This review focuses on psychosocial stressors; other types of stressors such as environmental manipulations (e.g., temperature), exercise, or intentionally imposed sleep disruption are not covered in this article.

It is possible that different types of stressors may bring with them different impacts on sleep. Issues such as the chronicity (Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002) and virulence (Brantley, Waggoner, Jones, & Rappaport, 1987) of the stress exposure thus need to be considered in defining stressors' effects on sleep. Thus, we attempted to group articles around a common theme as to the nature of the stress. Unfortunately, the total number of retrieved articles was so small (surprisingly) that, aside from the articles on post-traumatic stress disorder (PTSD), the "grouping" of articles was difficult. We wound up contrasting articles that dealt with (a) some aspect of daily stress (such as job stress) and life events (such as divorce, bereavement), (b) experimentally imposed stressors (laboratory-based studies), and (c) traumatic stressors (such as disasters or PTSD). The first and last categories typically involve a longer duration of the stress exposure with varying levels of intensity; the experimental stressors, on the other hand, typically involve a short-term exposure to mild stress. We have grouped the occasional article involving brief exposure to a severe stressor with the traumatic stressor category.

Examination of the relationship between stress and objectively measured sleep will allow a deeper understanding of sleep physiology, possible modes of influence of stress on sleep, and an appreciation of methodological limitations in other sleep research when stress is not considered as a confounder.

METHOD

We explored references in several ways. As a first step we used MeSH terms (Medical Subject Headings of the National Library of Medicine) in PubMed that related to stress and its effects on sleep characteristics that are measured polysomnographically. The MeSH terms used were "stress" and "sleep." To refine the concept of stress, we used subheadings, or related MeSH terms, such as "stress, psychological," "life change events," "crowding," "disasters," "stress disorders, post traumatic," and "combat disorders." To define objectively measured sleep we used "sleep, physiology," "polysomnography," and "sleep stages." Some additional articles were found through the "related articles, links" function of the PubMed site. All searching in PubMed or Korean Med was limited to human studies written in English or Korean. We included studies that measured sleep with PSG and provided sufficient information so that the type of stressor could be defined. Articles were excluded if they were solely reviews or if they involved medication trials.

We also searched the database PsycINFO with the thesaurus "environmental stress," "occupational stress," "physiological stress," "psychological stress," "social stress," "disaster," "distress," and "adjustment disorder" versus "REM sleep," "NREM sleep" (non-REM sleep), "sleep wake cycle," and "polysomnography" using similar selection criteria as explained earlier. We also examined the textbook, *Principles and Practice of Sleep*

Medicine (Kryger, Roth, & Dement, 2000), to look at the descriptions of stress on sleep; we searched the keyword stress in the Web site of the journal *Sleep* from Volumes 1 through 27. Finally, we contacted researchers in the field in an effort to locate additional references about the effect of stress on sleep.

PSG findings in the searched articles were arranged in tables according to the sleep variables. Total sleep time (TST) was chosen as a representative of sleep duration and defined as the sum of sleeping time spent from sleep onset to wake. Sleep latency (SL), REM sleep latency (REML), total REM sleep time (REMT), and slow wave sleep time (SWST) were chosen as measures of sleep architecture. REMT and SWST were presented in tables first as a percentage of TST and then as the actual time in minutes, if the article did not present the percentage of total sleep. To better examine sleep continuity, we looked at a number of measures. These included sleep efficiency (SE) defined as percentage of TST over time in bed, total wake time, number of awakenings, number of arousals, and wake time after sleep onset (WASO). In addition, we evaluated the REM density (number of rapid eye movements per hour of REM sleep) and body movement (number of body movements while sleep) data when it was provided.

RESULTS

Search Results Using Differing Search Criteria

Searching PubMed by MeSH terms “stress” and “sleep,” only 17 articles measured sleep objectively out of 190 articles that looked at the relation between sleep and stress. Alternate searches of “stress, psychological” and “polysomnography” resulted in 8 articles; “life change event” and “polysomnography” resulted in 12 articles; and “stress, psychological” and “sleep stages” found 114 articles. There were predictably many overlapping articles among these citations, and regrettably only a small portion of those articles actually described objective sleep measures. After checking each article, we found 28 articles that met our study criteria—that is, included polysomnographic findings in the results in response to a definable stressor. The excluded articles reported only subjective sleep findings. Using the phrase “stress disorders, post traumatic” with several subheadings of “sleep,” we found an additional 18 articles. Nineteen articles dealt with environmental stressors such as temperature or noise and were excluded from the review. This left 46 articles from PubMed that were retained for this review. Another search through PsycINFO using the thesaurus entries listed in the Method section located 4 additional articles.

In the textbook by Kryger et al. (2000), there was no separate chapter describing stress effects; rather, the text discussed environmental and “stress-related” factors such as unfamiliar sleep environment, noise, temperature, sleep surface, sleep position, altitude, life events, and expectancies that might contribute to transient insomnia. These stressor factors were generally discussed in the text in terms of their tendency to be associated with increased SL, REML and wakefulness, and decreased REM sleep and slow wave sleep (SWS).

In the journal *Sleep*, using the term *stress* in a keyword search on the journal's Web site, we found 10 additional articles dealing with the effects of stress on sleep. Correspondence with scholars in the sleep medicine field yielded 1 additional article.

The total number of articles we analyzed was 63. These 63 articles were classified by type of stressor, and tables summarizing studies in each of these categories are cited throughout this article.

DAILY LIFE STRESS AND STRESSFUL LIFE EVENTS

Investigators have examined a wide variety of daily life stressors. The interpretation of some of these studies is difficult because they include factors such as shift work with its effects on chronobiology, superimposed on sleep. Goncharenko (1979; see Table 1) reported that shift workers manifested long SL, decrease in SWS, increase in REM sleep, and shorter REML, as compared to students. Kecklund and Akerstedt (2004a) reported that worry about going to work the next morning resulted in a reduction of SWS. Both of these studies considered emotional stress as the main contributing factor that resulted in sleep architecture change. Söderström, Ekstedt, Akerstedt, Nilsson, and Axelsson (2004) recently reported that the sleep of workers before a workday showed shorter TST and shorter REM sleep time when it was compared with sleep before a day off. Furthermore, workers with high burnout scores had more arousals before both workdays and days off when compared with workers with low burnout scores in this study.

Kobayashi, Ishikawa, and Arakawa (1998) studied the effects of daytime mental and physical task activity on REM sleep. Mental task activity associated with high tension (driving a car for 600 km) was associated with highly variable REM onset time in the later part of the night, whereas physical task activity delayed REML and increased SWS in the first sleep cycle.

In summary, diverse stressors that are commonly encountered in the daily environment resulted in several changes in sleep architecture, but it is difficult to see any consistent patterns because of the differences among the study populations and the variant nature of daily life stresses. However, everyday emotional stress might lead to less SWS.

In studying the effects of stressful life events on sleep, many researchers tried to control for the confounding effect of depression. Williamson et al. (1995) measured sleep and stressful life events in normal adolescents and adolescents with major depressive disorder. They found that decreased REML and increased REM sleep time were found only in the normal controls having a high life events score. On the other hand, increased stressful life events correlated with decreased SWS in both groups. SWS was the PSG variable most tightly related to stressful life events in this study. Reynolds et al. (1993) measured sleep in 27 non-depressed bereaved elderly and non-bereaved controls. They found no significant difference in sleep measures except that REM density increased after 23 months of bereavement. Cartwright and Wood (1991) found that ongoing marital separation in non-depressed persons resulted in less delta sleep, a higher REM sleep percentage, and shorter REML.

Hall et al. (1997) reported that more stressful event-related intrusive thoughts and avoidance behaviors were associated with longer SL and lower delta sleep ratio. Hall et al. (1998) also reported that greater frequency of intrusion and avoidance was significantly correlated with longer SL and greater awake time during the first NREM period. In quantitative EEG analysis, stress-related intrusive thoughts were associated with higher beta power, and increased subjective stress burden was associated with decreased delta power (Hall et al., 2000).

In summary, major stressful life events affect sleep in normal persons. These changes included decreased REML, an increased REM sleep percentage, and reduced SWS. The unique observation of increased REM density almost 2 years after bereavement is intriguing and needs to be replicated.

EXPERIMENTAL STRESS ON SLEEP

In the laboratory, researchers can readily impose different kinds of physical or psychological stressors and measure their effect on sleep. Several studies recorded sleep while using an indwelling venous catheter as a mild stressor (see Table 2). Adam (1982) and Vitiello et al. (1996) reported that blood sampling through an indwelling venous catheter decreased TST and SE, increased awakenings, and reduced REM sleep when compared to baseline sleep. Using the same study paradigm, Prinz, Bailey, and Woods (2000) and Prinz, Bailey, Moe, Wilkinson, and Scanlan (2001) reported reduced SE, SWS, REM sleep, and increased awakenings when participants slept with an indwelling catheter. Vitiello et al. also reported SWS reduction, and Jarrett et al. (1984) reported decreased SE on the night of cannulation. Dimsdale, Coy, Ancoli-Israel, Clausen, and Berry (1993) reported that blood pressure cuff inflation was associated with increased arousals and wakefulness especially in the first night of recording. In summary, there were inconsistent findings regarding SL, but these diverse studies generally found that wearing medical equipment tended to decrease SE, SWS, and REM sleep, and to increase awakening. It is uncertain if these changes persist with multiple nights of sleep monitoring. One form of experimental stress may be the sleep laboratory itself. The so-called “first-night effect,” which has been seen in individuals of all ages (Aber, Block, Hellard, & Webb, 1989; Carvalhaes-Neto et al., 2003; Scholle et al., 2003), might represent an adaptation burden to the sleep laboratory. Since Agnew, Webb, and Williams (1966) reported and defined the first-night effect, several researchers have described its association with reduced TST, poor SE, less REM sleep, increased SL, and increased WASO (Aber et al., 1989; Curcio, Ferrara, Piergianni, Fratello, & De Gennaro, 2004; Le Bon et al., 2000; Li et al., 2004; Scholle et al., 2003; Toussaint et al., 1997). Carvalhaes-Neto et al. reported reduced SWS in the first hospitalization night, and Le Bon et al. also reported fewer SWS minutes on the first night of sleep study. On the other hand, Coble, McPartland, Silva, and Kupfer (1974) and Kim and Jeong (1998) reported no first-night effect in a sleep laboratory that resembled a hotel-like comfortable setting. Individual participant characteristics may affect the extent of the first-night effect on sleep. Drake, Richardson, Roehrs, Scofield, and Roth (2004) divided participants into a group highly vulnerable to insomnia and a group with low vulnerability to insomnia using the Ford insomnia response to stress test. The high vulnerability group showed decreased TST and increased SL during the first-night PSG when compared to the low vulnerability group.

Acute experimental psychological stress in healthy participants had a consistent effect on REM sleep in early studies. In the 1960s and 1970s, many researchers reported that pre-sleep stimulation influenced REM activity and awakenings (Baekeland, Koulack, & Lasky, 1968; Cluydts & Visser, 1980; Cohen, 1975; Goodenough, Witkin, Koulack, & Cohen, 1975). Pre-sleep stress, such as an aversive film (Baekeland et al., 1968; Cluydts & Visser, 1980; Goodenough et al., 1975; Lauer, Riemann, Lund, & Berger, 1987) or impersonal treatment (Cohen, 1975), was associated with increased frequency of rapid eye movements (REM density; Goodenough et al., 1975; Lauer et al., 1987), number of awakenings (Baekeland et al., 1968; Cluydts & Visser, 1980), and longer SL (Goodenough et al., 1975).

More recently, Koulack, Prevost, and De Koninck (1985) gave an intellectually challenging test to 16 male university students; the suggestion of inferiority led to longer SL and lower REM density than at baseline. Germain, Buysse, Ombao, Kupfer, and Hall (2003) reported that acute experimental psychological stress reduced rapid eye movement counts during the last REM sleep period in healthy participants when compared to non-stressed control participants. They analyzed this change through a model in which a situational factor (stress) influenced REM counts while a dispositional factor modified that change. Vein et al. (2002) reported that negative emotions induced by provocative questions lengthened SL and redistributed SWS, such that it increased SWS in the second sleep cycle.

Levin, Strygin, & Korabel'nikova (2002) also reported that emotional stress decreased SWS in the first sleep cycle. They then divided participants into adaptive and non-adaptive groups according to the coping strategies employed, and observed that the non-adaptive group had more changes in sleep structure after emotional stress; these changes included increased SL, decreased number of awakenings and movements, and reduced SWS in the first sleep cycle. This sort of design whereby sleep is considered in the context of both stress and adaptation is a promising research direction and one that is relatively lightly explored in the literature.

There are, of course, many variants of experimental stress including observations made outside of the laboratory itself. For instance, Beaumaster, Knowles, and MacLean (1978) measured the pre- and post-stress effect of a parachute jump on sleep, and found that experienced parachutists had less pre-jump SL when compared with the novice group.

In summary, mild manipulation such as an indwelling catheter or adaptation to the sleep laboratory environment resulted in lower SE, frequent awakenings, decreased REM sleep, and less SWS. Experimental psychological stressors were also associated with increased SL and decreased SWS in the first cycle. There are conflicting observations regarding the effect of laboratory stressors on REM-related variables.

TRAUMATIC STRESS

Sorrow and horror come in all-too-many "flavors." Given the fact that disrupted sleep is commonly reported in PTSD, there have been many studies of sleep in PTSD patients (see Table 3). Characteristically, the traumatic stress was severe and usually chronic or reverberating, reflecting stressors such as combat, natural disaster, holocaust, or traffic accident. Some studies suggested that a history of exposure to severe stress was associated with sleep architecture changes, regardless of PTSD; others specifically investigated the

differences of sleep architecture between those exposed to severe trauma versus those diagnosed with PTSD.

Many articles reported only one or two sleep variables in PTSD patients that were significantly different from those in controls. However, those variables differed from study to study, and some authors (Breslau et al., 2004; Hurwitz, Mahowald, Kuskowski, & Engdahl, 1998; Klein, Koren, Arnon, & Lavie, 2002; Lavie, Katz, Pillar, & Zinger, 1998; Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995) found no differences in sleep architecture between PTSD patients and controls.

Increased awakenings were commonly reported in PTSD patients (Astrom, 1989; Germain & Nielsen, 2003; Hefez, Metz, & Lavie, 1987; Kramer & Kinney, 1988; Lavie, Hefez, Halperin, & Enoch, 1979; Mellman, Kulick-Bell, Ashlock, & Nolan, 1995; Mellman, Nolan, Hebding, Kulick-Bell, & Dominguez, 1997; Mikulincer, Glaubman, Wasserman, Porat, & Birger, 1989; Woodward, Arsenault, Murray, & Bliwise, 2000). Decreased SE (Astrom, 1989; Germain & Nielsen, 2003; Hefez et al., 1987; Kramer & Kinney, 1988; Lavie et al., 1979; Mellman, Kulick-Bell, et al., 1995; Mellman et al., 1997; Mikulincer et al., 1989), decreased TST (Astrom, 1989; Dow, Kelsoe, & Gillin, 1996; Hefez et al., 1987; Lavie et al., 1979; Mellman et al., 1997), and increased REM density (Hefez et al., 1987; Mellman et al., 1997; Ross et al., 1994a; Ross et al., 1999) were also commonly reported. Mellman, David, et al. (1995) reported that the “re-experiencing symptom” item from the PTSD severity scale was significantly correlated with REM density in PTSD patients. However, the reported change of REM sleep across studies has been variable; Engdahl, Eberly, Hurwitz, Mahowald, and Blake (2000), Ross et al. (1994b), Ross et al. (1999), and Woodward, Murburg, and Bilwise (2000) reported an increased REM sleep percentage; whereas Hefez et al., Kramer and Kinney (1988), Lavie et al. (1979), and Mikulincer et al. (1989) reported a decrease in the REM sleep percentage. Four studies reported increased REML (Hefez et al., 1987; Kramer & Kinney, 1988; Lavie et al., 1979; Mikulincer et al., 1989), and one study reported decreased REML (Greenberg, Pearlman, & Gampel, 1972). Unfortunately, the analysis of the PTSD participants in the laboratory did not control for depression and did not contain a control group for comparison.

Woodward, Friedman, and Bliwise (1996) reported reduced SWS in individuals with both PTSD and depression when compared with individuals with PTSD but without depression. Astrom (1989) found decreased SWS in the victims of torture when compared to controls. Kramer and Kinney (1988) reported that Vietnam veterans showed less SWS, but there was no control group in their study. Mikulincer et al. (1989) observed reduced SWS in PTSD patients when compared to controls.

Arousals may be abnormal in patients with PTSD. Mellman, David, et al. (1995) reported increased arousals during sleep, and Breslau et al. (2004) reported increased arousals during REM sleep. Woodward, Murburg, and Bil-wise (2000) found that PTSD patients have more beta-band EEG power in REM sleep and observed that this phenomenon was correlated with subjective hyper-arousal symptoms. On the other hand, Hurwitz et al. (1998) and Engdahl et al. (2000) reported decreased arousals during non-REM sleep. Klein et al. (2002) found that PTSD patients have an increased threshold to awakening, so this observation was

compatible with the observation of no increase of wake time in PTSD patients. Lavie et al. (1998) found that more time was needed to awaken the combat PTSD patients from their REM sleep.

Abnormalities of body movements during sleep have been reported in PTSD patients as compared to controls. Woodward, Leskin, and Sheikh (2002) and Woodward, Leskin, and Sheikh (2003) reported reduced body movements during sleep in PTSD patients and described this as “freezing body.” However, several authors reported increased body movement and periodic limb movement during sleep (Germain & Nielsen, 2003; Hefez et al., 1987; Lavie & Hertz, 1979; Ross et al., 1994a). Mellman, Kulick-Bell, et al. (1995) also observed a trend of increased body and limb movements during sleep in combat survivors.

Nightmares are felt to be hallmarks in defining symptoms of PTSD. Interestingly, some authors found that nightmares were not associated with increased REM sleep in PTSD patients (Woodward et al., 2002).

Schlosberg and Benjamin (1978) reported three acute combat fatigue cases whose sleep showed reduced TST, delayed SL and REML, reduced REM sleep and SWS, increased awakenings, and poor SE. These findings are similar to those observed during acute laboratory adaptation stress. Mellman et al. (2002) recorded sleep within 1 month of injury, and found that injured patients who did not develop PTSD showed reduced TST, increased WASO, and increased REM density when compared to healthy controls. In contrast, patients who went on to develop PTSD showed more periods of REM sleep and shorter average duration of REM sleep as compared to patients without PTSD.

Many studies reported that PTSD patients present with increased awakenings, reduced TST, and decreased SE. There was less consistency regarding effects of PTSD on REM activity. Inspection of the study characteristics summarized in Table 3 reveals that the sample size for many of these studies was quite small. In addition, the influence of comorbidity factors such as depression or substance abuse was frequently not addressed. To the extent that PTSD is confounded by other factors such as chronic pain, understanding the PSG consequence of PTSD is further complicated. Dow et al. (1996) designed a study to compare PTSD patients with major depression to those with just major depressive patients and to healthy controls. The results were difficult to interpret because the sleep change observed in PTSD with comorbid depression was only decreased TST, and that observed in major depression without PTSD was also a tendency to reduced TST.

DISCUSSION

In looking at the effect of stress on sleep polysomnographically, we were surprised at the paucity of studies during the last 4 decades. With the possible exception of PTSD, there have been few studies with replicated designs on particular groups of participants exposed to specific types of stressors. This limitation is an obvious one in trying to draw overall conclusions. We felt that it was sensible as a first step to group stressors with similar characteristics of time and intensity rather than attempt to analyze possible “global effects”

of stress on polysomnographic findings. We also felt that, perhaps with the exception of PTSD, there were too few studies with common methodologies to warrant a meta-analysis.

By way of aside, it is important to acknowledge that in comparison to the paucity of human studies on this topic, there are numerous animal studies of stress and its effects on PSG. In animal models of experimental stress, researchers commonly studied social defeat stress and immobilization stress (Meerlo, Easton, Bergmann, & Turek, 2001). Although there were individual differences in the effects of acute stress in the rat (Bouyer, Deminiere, Mayo, & Le Moal, 1997), social defeat stress enhanced slow wave activity in NREM sleep (Meerlo, Pragt, & Daan, 1997), whereas 2-hr immobilization stress increased paradoxical sleep (Rampin, Cespuglio, Chastrette, & Jouvet, 1991). Unlike the human studies, SWS and REM sleep changes were fairly consistent according to the types of stress. Social stress increased SWS activity, and immobilization increased REM sleep (Palma, Suchecki, & Tufik, 2000). When the stress duration was lengthened, SWS or REM sleep effects were changed. For example, immobilization stress more than 4 hr reduced the paradoxical sleep increase (Marinesco, Bonnet, & Cespuglio, 1999); chronic stress decreased SWS, REM sleep, and even altered the circadian pattern (Kant et al., 1995).

The main focus for the studies that we categorized as “daily life stress” was the effects of occupation-related stress and psychological distress on sleep. We considered this group as reflecting exposure to chronic, non-severe stressors. There were few studies, and no consistent patterns were noted concerning their effect on sleep (see Tables 1 & 4). However, the number of studies was so small and the diversity of life troubles so extensive (ranging from job stress to marital disruption) that the lack of consistency might be expected. Recently, some researchers have begun examining participants who are caregivers of chronic patients (von Kanel et al., 2006); this design would potentially be a promising one in that the stress is chronic and frequently encountered. Similarly, there certainly are enough individuals exposed to job stress or to marital disruption to allow replicated studies. In this regard, it would be important to recognize the continuing discussion in the stress literature that events are not stressful unless they are perceived as such by the participant (Shaw, Dimsdale, & Patterson, 2000). As Shakespeare noted, “there is nothing either good or bad but thinking makes it so.” Thus, considerations of continuing daily stressful events might need to consider the individual's construal of how distressing an event was (for some, marital separation is a blessing not a tribulation). For the most part, the articles reviewed here do not allow an exploration of the emotional “texture” of the life event exposure, the individual's appraisal of the intensity of the stress as related to its effects on sleep.

There were only five studies that examined stressful life events such as bereavement and divorce with PSG (Table 5). Here again, it is difficult to summarize across these few articles, as timing of the life event vis à vis the sleep recording differed. Similarly, in some instances depression is superimposed on the life events exposure; thus, it is difficult to tease apart the effects of stress exposure from depression. Two studies in normal participants confronted with stressful life events showed a decrease of REML, increase of REM sleep, and a decrease of SWS. These changes in sleep architecture resemble the sleep changes in depression. Monroe, Thase, and Simons (1992) reported that life stress before depression onset did not change the REML, whereas depression without stress reduced REML. Thus,

further studies need to be performed that explicitly examine exposure to life events with and without depression comorbidity. The measurement of stressful life event-related intrusive thought and avoidance behavior in several studies (Hall et al., 1998; Hall et al., 1998; Hall et al., 2000) suggests a promising model for examining how the response to a stressor is related to effects on SL and delta sleep.

There were a large number of studies that examined exposure to diverse experimental stressors. Having an indwelling intravenous catheter or being in the sleep laboratory itself resulted in consistent polysomnographical findings: delayed SL, lower SE, frequent awakenings, decreased REM sleep, and less SWS. Experimental psychological stress was associated with increased SL and awakening. It seemed to have more diverse sleep effect, especially on REM sleep and SWS. In understanding the sleep changes associated with indwelling catheter or with the first-night effect in the sleep laboratory, it is difficult to disentangle psychological burden from novelty with the data at hand. The lack of consistency in REM sleep and SWS changes after experimental psychological stressors might reflect varying sleep changes corresponding to the psychological meaning or coping entailed by the task. REM sleep and SWS seemed to be reactive to both nonspecific simple stress and complex diverse stress. We need more sophisticated examination in the future to understand these subtle changes of REM sleep or SWS. Of course, stressors elicit multiple physiological effects in terms of autonomic functioning, and some investigators (Hall et al. 2004) have begun examining stressors in the context of both their peripheral autonomic activity and their PSG effects. This is a profitable direction for future research.

Short-term experimental stressors, as a whole, were associated with increased SL and awakening, as well as with decreased SE (see Table 6). We infer that these changes of sleep architecture are fairly typical stress responses because polysomnographic findings were relatively consistent across numerous types of acute stressors.

In traumatic stress, the most frequent findings were no significant changes in any specific sleep variable (see Table 7). This observation might be due to the timing of studies. For example, many researchers performed their sleep studies 10 to 20 years after combat exposure, and there could be many factors influencing sleep including psychological, physiological, and the compensation process itself. It is still unclear whether the traumatic exposure itself influenced the sleep or whether the subsequently developing PTSD adversely influenced sleep. In the case of acute traumatic stress studies, the results were similar to the experimental stress studies. This may imply that the time course of stressor exposure (i.e., immediate effects of stress on sleep) may be similar, regardless of the intensity of the stressor.

Several issues emerged from our research on stress and sleep. Many authors defined stressors in an idiosyncratic fashion, and the range of stressors was enormous. There is no agreement as to how such diverse stressors should be classified. We adopted an approach that facilitated rapid grouping of articles with a common methodology. Other classification schemes may come up with different overall observations. It is also difficult to quantitate stress in term of dosage. Finally, the duration of stressor exposure may be protracted or brief; a catastrophic stress, for example, might reverberate for decades.

Psychosocial stressors appear to be related increased SL, decreased SE, decreased REM sleep and SWS, and increased awakening (see Table 8). Increased awakening and decreased SE seemed to be the most sensitive variable to stress, and increased SL seemed to be very sensitive to acute experimental stressors.

Although PSG is the best way of objectively measuring sleep, sleep is an actively changing complex process. Many studies found no effect of stress on sleep variables (Table 8); such observations may simply reflect the enormous diversity of human response to stressors or a need for new ways of examining polysomnographic records. Other sophisticated objective tools for measuring sleep, such as neuroimaging techniques, may be useful in future research; and incorporating new paradigms for analysis of physiological measures such as heart rate, respiration, and vascular change will certainly add more information when examining human sleep.

Readers will note that this review sidesteps the question of the physiological mechanism whereby stress influences sleep. Whether these effects are being driven by factors such as elaboration of cytokines, hypothalamic–pituitary– adrenal axis disruption, or autonomic nervous system alteration will be difficult to tease apart until the literature makes more progress in specifying the necessary study design for future studies.

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REFERENCES

Aber WR, Block AJ, Hellard DW, Webb WB. Consistency of respiratory measurements from night to night during the sleep of elderly men. Chest. 1989; 96:747–751. [PubMed: 2791668]

Adam K. Sleep is changed by blood sampling through an indwelling venous catheter. Sleep. 1982; 5:154–158. [PubMed: 7100745]

Agnew HW Jr. Webb WB, Williams RL. The first night effect: An EEG study of sleep. Psychophysiology. 1966; 2:263–266. [PubMed: 5903579]

Akerstedt T, Hume K, Minors D, Waterhouse J. The meaning of good sleep: A longitudinal study of polysomnography and subjective sleep quality. Journal of Sleep Research. 1994; 3:152–158. [PubMed: 10607120]

Astrom C. Sleep disturbances in torture survivors. Acta Neurologica Scandinavica. 1989; 79:150–154. [PubMed: 2711821]

Baekeland F, Koulack D, Lasky R. Effects of a stressful pre-sleep experience on electroencephalograph-recorded sleep. Psychophysiology. 1968; 4:436–443. [PubMed: 5662813]

Beaumaster EJ, Knowles JB, MacLean AW. The sleep of skydivers: A study of stress. Psychophysiology. 1978; 15:209–213. [PubMed: 208094]

Bouyer JJ, Deminiere JM, Mayo W, Le Moal M. Inter-individual differences in the effects of acute stress on the sleep-wakefulness cycle in the rat. Neuroscience Letters. 1997; 225:193–196. [PubMed: 9147403]

Brantley PJ, Waggoner CD, Jones GN, Rappaport NB. A daily stress inventory: Development, reliability, and validity. Journal of Behavioral Medicine. 1987; 10:61–74. [PubMed: 3586002]

Breslau N, Roth T, Burduvali E, Kapke A, Schultz L, Roehrs T. Sleep in lifetime posttraumatic stress disorder: A community-based polysomnographic study. Archives of General Psychiatry. 2004; 61:508–516. [PubMed: 15123496]

Cartwright RD, Wood E. Adjustment disorders of sleep: The sleep effects of a major stressful event and its resolution. Psychiatry Research. 1991; 39:199–209. [PubMed: 1798820]

Carvalhaes-Neto N, Ramos LR, Suchecki D, Tufik S, Huayllas MK, Kater CE. The effect of hospitalization on the sleep pattern and on cortisol secretion of healthy elderly. Experimental Aging Research. 2003; 29:425–436. [PubMed: 12959876]

Cluydts R, Visser P. Mood and sleep. II. Effects of aversive pre-sleep stimulation. Waking Sleeping. 1980; 4:199–203. [PubMed: 7281631]

Coble P, McPartland RJ, Silva WJ, Kupfer DJ. Is there a first night effect? (A revisit). Biological Psychiatry. 1974; 9:215–219. [PubMed: 4371884]

Cohen DB. Eye movement density during REM sleep: The influence of personality and pre-sleep condition. Journal of Personality and Social Psychology. 1975; 32:1090–1093. [PubMed: 175146]

Curcio G, Ferrara M, Piergianni A, Fratello F, De Gennaro L. Paradoxes of the first-night effect: A quantitative analysis of antero-posterior EEG topography. Clinical Neurophysiology. 2004; 115:1178–1188. [PubMed: 15066543]

Dimsdale JE, Coy TV, Ancoli-Israel S, Clausen J, Berry CC. The effect of blood pressure cuff inflation on sleep. A polysomnographic examination. American Journal of Hypertension. 1993; 6:888–891. [PubMed: 8267947]

Dimsdale, JE.; Irwin, MR.; Keefe, FJ.; Stein, MB. Stress and psychiatry.. In: Sadock, BJ.; Sadock, VA., editors. Kaplan and Sadock's comprehensive textbook of psychiatry. 8th ed.. Lippincott; Philadelphia: 2005. p. 2180-2195.

Dow BM, Kelsoe JR Jr. Gillin JC. Sleep and dreams in Vietnam PTSD and depression. Biological Psychiatry. 1996; 39:42–50. [PubMed: 8719125]

Drake C, Richardson G, Roehrs T, Scofield H, Roth T. Vulnerability to stress-related sleep disturbance and hyperarousal. Sleep. 2004; 27:285–291. [PubMed: 15124724]

Engdahl BE, Eberly RE, Hurwitz TD, Mahowald MW, Blake J. Sleep in a community sample of elderly war veterans with and without posttraumatic stress disorder. Biological Psychiatry. 2000; 47:520–525. [PubMed: 10715358]

Germain A, Buysse DJ, Ombao H, Kupfer DJ, Hall M. Psychophysiological reactivity and coping styles influence the effects of acute stress exposure on rapid eye movement sleep. Psychosomatic Medicine. 2003; 65:857–864. [PubMed: 14508032]

Germain A, Nielsen TA. Sleep pathophysiology in posttraumatic stress disorder and idiopathic nightmare sufferers. Biological Psychiatry. 2003; 54:1092–1098. [PubMed: 14625152]

Goncharenko AM. Electrophysiological investigation of sleep in shift workers exposed to emotional stress due to work. Human Physiology. 1979; 5:468–474. [PubMed: 233000]

Goodenough DR, Witkin HA, Koulack D, Cohen H. The effects of stress films on dream affect and on respiration and eye-movement activity during rapid-eye-movement sleep. Psychophysiology. 1975; 2:313–320. [PubMed: 168607]

Greenberg R, Pearlman CA, Gampel D. War neuroses and the adaptive function of REM sleep. The British Journal of Medical Psychology. 1972; 45:27–33. [PubMed: 4335845]

Hall M, Baum A, Buysse DJ, Prigerson HG, Kupfer DJ, Reynolds CF III. Sleep as a mediator of the stress-immune relationship. Psychosomatic Medicine. 1998; 60:48–51. [PubMed: 9492239]

Hall M, Buysse DJ, Dew MA, Prigerson HG, Kupfer DJ, Reynolds CF III. Intrusive thoughts and avoidance behaviors are associated with sleep disturbances in bereavement-related depression. Depression and Anxiety. 1997; 6:106–112. [PubMed: 9442984]

Hall M, Buysse DJ, Nowell PD, Nofzinger EA, Houck P, Reynolds CF III, et al. Symptoms of stress and depression as correlates of sleep in primary insomnia. Psychosomatic Medicine. 2000; 62:227–230. [PubMed: 10772402]

Hall M, Vasko R, Buysse D, Ombao H, Chen Q, Cashmere JD, et al. Acute stress affects heart rate variability during sleep. Psychosomatic Medicine. 2004; 66:56–62. [PubMed: 14747638]

Hefez A, Metz L, Lavie P. Long-term effects of extreme situational stress on sleep and dreaming. American Journal of Psychiatry. 1987; 144:344–347. [PubMed: 3826435]

Hurwitz TD, Mahowald MW, Kuskowski M, Engdahl BE. Polysomnographic sleep is not clinically impaired in Vietnam combat veterans with chronic posttraumatic stress disorder. Biological Psychiatry. 1998; 44:1066–1073. [PubMed: 9821572]

Jarrett DB, Greenhouse JB, Thompson SB, McEachran A, Coble P, Kupfer DJ. Effect of nocturnal intravenous cannulation upon sleep-EEG measures. Biological Psychiatry. 1984; 19:1537–1550. [PubMed: 6542805]

Kant GJ, Pastel RH, Bauman RA, Meininger GR, Maughan KR, Robinson TN III, et al. Effects of chronic stress on sleep in rats. Physiology & Behavior. 1995; 57:359–365. [PubMed: 7716216]

Kecklund G, Akerstedt T. Apprehension of the subsequent working day is associated with a low amount of slow wave sleep. Biological Psychiatry. 2004a; 66:169–176.

Kecklund, G.; Akerstedt, T. Report on methods and classification of stress, inattention and emotional states. Sensation (Contract No. IST-507231). Karolinska Institute; Stockholm: 2004b.

Kim EJ, Jeong DU. Investigation of “first-night effect” in normal young adult male subjects on polysomnography. Sleep Medicine and Psychophysiology. 1998; 5:111–117.

Klein E, Koren D, Arnon I, Lavie P. No evidence of sleep disturbance in post-traumatic stress disorder: A polysomnographic study in injured victims of traffic accidents. The Israel Journal of Psychiatry and Related Sciences. 2002; 39:3–10. [PubMed: 12013708]

Kobayashi T, Ishikawa T, Arakawa K. Effects of daytime activity upon the timing of REM sleep periods during a night. Psychiatry and Clinical Neurosciences. 1998; 52:130–131. [PubMed: 9628110]

Koulack D, Prevost F, De Koninck J. Sleep, dreaming, and adaptation to stressful intellectual activity. Sleep. 1985; 8:244–253. [PubMed: 4048740]

Kramer M, Kinney L. Sleep patterns in trauma victims with disturbed dreaming. Psychiatric Journal of the University of Ottawa. 1988; 13:12–16. [PubMed: 3283795]

Kryger, M.; Roth, T.; Dement, WC. Principles and practice of sleep medicine. Saunders; Philadelphia: 2000.

Lauer C, Riemann D, Lund R, Berger M. Shortened REM latency: A consequence of psychological strain? Psychophysiology. 1987; 24:263–271. [PubMed: 3602281]

Lavie P, Hefez A, Halperin G, Enoch D. Long-term effects of traumatic war-related events on sleep. American Journal of Psychiatry. 1979; 136:175–178. [PubMed: 216274]

Lavie P, Hertz G. Increased sleep motility and respiration rates in combat neurotic patients. Biological Psychiatry. 1979; 14:983–987. [PubMed: 508888]

Lavie P, Katz N, Pillar G, Zinger Y. Elevated awaking thresholds during sleep: Characteristics of chronic war-related posttraumatic stress disorder patients. Biological Psychiatry. 1998; 44:1060–1065. [PubMed: 9821571]

Le Bon O, Hoffmann G, Tecco J, Staner L, Noseda A, Pelc I, et al. Mild to moderate sleep respiratory events: One negative night may not be enough. Chest. 2000; 118:353–359. [PubMed: 10936124]

Levin YI, Strygin KN, Korabel'nikova EA. Effect of personality on changes of sleep structure caused by emotional stress. Human Physiology. 2002; 28(3):37–42.

Li AM, Wing YK, Cheung A, Chan D, Ho C, Hui S, et al. Is a 2-night polysomno-graphic study necessary in childhood sleep-related disordered breathing? Chest. 2004; 126:1467–1472. [PubMed: 15539714]

Marinesco S, Bonnet C, Cespuglio R. Influence of stress duration on the sleep rebound induced by immobilization in the rat: A possible role for corticosterone. Neuroscience. 1999; 92:921–933. [PubMed: 10426533]

Mason JW. A review of psychoendocrine research on the pituitary–adrenal cortical system. Psychosomatic Medicine. 1968; 30:576–607. [PubMed: 4303377]

Meerlo P, Easton A, Bergmann BM, Turek FW. Restraint increases prolactin and REM sleep in C57BL/6J mice but not in BALB/cJ mice. American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology. 2001; 281:R846–R854.

Meerlo P, Pragt BJ, Daan S. Social stress induces high intensity sleep in rats. Neuroscience Letters. 1997; 225:41–44. [PubMed: 9143013]

Mellman TA, Bustamante V, Fins AI, Pigeon WR, Nolan B. REM sleep and the early development of posttraumatic stress disorder. American Journal of Psychiatry. 2002; 159:1696–1701. [PubMed: 12359675]

Mellman TA, David D, Kulick-Bell R, Hebding J, Nolan B. Sleep disturbance and its relationship to psychiatric morbidity after Hurricane Andrew. American Journal of Psychiatry. 1995; 152:1659–1663. [PubMed: 7485631]

Mellman TA, Kulick-Bell R, Ashlock LE, Nolan B. Sleep events among veterans with combat-related posttraumatic stress disorder. American Journal of Psychiatry. 1995; 152:110–115. [PubMed: 7802100]

Mellman TA, Nolan B, Hebding J, Kulick-Bell R, Dominguez R. A polysomno-graphic comparison of veterans with combat-related PTSD, depressed men, and non-ill controls. Sleep. 1997; 20:46–51. [PubMed: 9130334]

Mikulincer M, Glaubman H, Wasserman O, Porat A, Birger M. Control-related beliefs and sleep characteristics of posttraumatic stress disorder patients. Psychological Reports. 1989; 65:567–576. [PubMed: 2798672]

Monroe SM, Thase ME, Simons AD. Social factors and the psychobiology of depression: Relations between life stress and rapid eye movement sleep latency. Journal of Abnormal Psychology. 1992; 101:528–537. [PubMed: 1500610]

Palma BD, Suchecki D, Tufik S. Differential effects of acute cold and footshock on the sleep of rats. Brain Research. 2000; 861:97–104. [PubMed: 10751569]

Prinz P, Bailey S, Moe K, Wilkinson C, Scanlan J. Urinary free cortisol and sleep under baseline and stressed conditions in healthy senior women: Effects of estrogen replacement therapy. Journal of Sleep Research. 2001; 10:19–26. [PubMed: 11285051]

Prinz P, Bailey SL, Woods DL. Sleep impairments in healthy seniors: Roles of stress, cortisol, and interleukin-1 beta. Chronobiology International. 2000; 17:391–404. [PubMed: 10841212]

Rampin C, Cespuglio R, Chastrette N, Jouvet M. Immobilization stress induces a paradoxical sleep rebound in rat. Neuroscience Letters. 1991; 126:113–118. [PubMed: 1922920]

Reynolds CF III, Hoch CC, Buysse DJ, Houck PR, Schlernitzauer M, Pasternak RE, et al. Sleep after spousal bereavement: A study of recovery from stress. Biological Psychiatry. 1993; 34:791–797. [PubMed: 8292683]

Ross RJ, Ball WA, Dinges DF, Kribbs NB, Morrison AR, Silver SM, et al. Motor dysfunction during sleep in posttraumatic stress disorder. Sleep. 1994a; 17:723–732. [PubMed: 7701184]

Ross RJ, Ball WA, Dinges DF, Kribbs NB, Morrison AR, Silver SM, et al. Rapid eye movement sleep disturbance in posttraumatic stress disorder. Biological Psychiatry. 1994b; 35:195–202. [PubMed: 8173020]

Ross RJ, Ball WA, Sanford LD, Morrison AR, Dinges DF, Silver SM, et al. Rapid eye movement sleep changes during the adaptation night in combat veterans with posttraumatic stress disorder. Biological Psychiatry. 1999; 45:938–941. [PubMed: 10202585]

Schlosberg A, Benjamin M. Sleep patterns in three acute combat fatigue cases. Journal of Clinical Psychiatry. 1978; 39:546–549. [PubMed: 207680]

Schneider-Helmert D, Kumar A. Sleep, its subjective perception, and daytime performance in insomniacs with a pattern of alpha sleep. Biological Psychiatry. 1995; 37:99–105. [PubMed: 7718685]

Scholle S, Scholle HC, Kemper A, Glaser S, Rieger B, Kemper G, et al. First night effect in children and adolescents undergoing polysomnography for sleep-disordered breathing. Clinical Neurophysiology. 2003; 114:2138–2145. [PubMed: 14580612]

Shaw, WS.; Dimsdale, JE.; Patterson, TL. Stress and life events measures.. In: Rush, AJ., Jr.; Pincus, HA.; First, MB.; Blacker, D.; Endicott, J.; Keith, SJ., et al., editors. Handbook of psychiatric measures. American Psychiatric Association; Washington, DC: 2000. p. 221-237.

Söderström M, Ekstedt M, Akerstedt T, Nilsson J, Axelsson J. Sleep and sleepiness in young individuals with high burnout scores. Sleep. 2004; 27:1369–1377. [PubMed: 15586790]

Toussaint M, Luthringer R, Schaltenbrand N, Nicolas A, Jacqmin A, Carelli G, et al. Changes in EEG power density during sleep laboratory adaptation. Sleep. 1997; 20:1201–1207. [PubMed: 9493933]

Vein AM, Sudakov KV, Levin YI, Yumatov EA, Strygin KN, Kovrov GV. Stages of sleep after psychoemotional tension: The individual character of changes. Neuroscience and Behavioral Physiology. 2002; 32:513–518. [PubMed: 12403003]

Vitiello MV, Larsen LH, Moe KE, Borson S, Schwartz RS, Prinz PN. Objective sleep quality of healthy older men and women is differentially disrupted by nighttime periodic blood sampling via indwelling catheter. Sleep. 1996; 19:304–311. [PubMed: 8776788]

von Kanel R, Dimsdale JE, Ancoli-Israel S, Mills PJ, Patterson TL, McKibbin CL, et al. Poor sleep is associated with higher plasma proinflammatory cytokine interleukin-6 and procoagulant marker fibrin D-dimer in older caregivers of people with Alzheimer's disease. Journal of the American Geriatric Society. 2006; 54:431–437.

Williamson DE, Dahl RE, Birmaher B, Goetz RR, Nelson B, Ryan ND. Stressful life events and EEG sleep in depressed and normal control adolescents. Biological Psychiatry. 1995; 37:859–865. [PubMed: 7548460]

Woodward SH, Arsenault NJ, Murray C, Bliwise DL. Laboratory sleep correlates of nightmare complaint in PTSD inpatients. Biological Psychiatry. 2000; 48:1081–1087. [PubMed: 11094141]

Woodward SH, Friedman MJ, Bliwise DL. Sleep and depression in combat-related PTSD inpatients. Biological Psychiatry. 1996; 39:182–192. [PubMed: 8837979]

Woodward SH, Leskin GA, Sheikh JI. Movement during sleep: Associations with posttraumatic stress disorder, nightmares, and comorbid panic disorder. Sleep. 2002; 25:669–676. [PubMed: 12224845]

Woodward SH, Leskin GA, Sheikh JI. Sleep respiratory concomitants of comorbid panic and nightmare complaint in post-traumatic stress disorder. Depression and Anxiety. 2003; 18:198–204. [PubMed: 14661189]

Woodward SH, Murburg MM, Bliwise DL. PTSD-related hyperarousal assessed during sleep. Physiology & Behavior. 2000; 70:197–203. [PubMed: 10978496]

TABLE 1

Studies of Daily Life Stress and Stressful Life Events

Author	Stressor and Participants	PSG Findings									Comment on Findings
		TST	SL	REML	REMT	REM Density	SWST	Awakening	SE	Body Movement	
Goncharenko (1979)	• Emotional stress due to shift work • 22 male, 8 female shift workers (aged 21–55) versus 23 male, 6 female students (aged 20–30)	—	↓	↓	↑	—	↓	—	—	—	Age differences in groups
Kecklund and Akerstedt (2004a)	• Occupational apprehension • 14 men, 23 women	—	—	—	—	—	↓	—	—	—	
Kobayashi et al. (1998)	• Daytime activity versus relaxation • 5 men (aged 19–25)	—	—	↑	—	—	↑ (1st sleep cycle after physical task)	—	—	—	No presented PSG data; description only
Söderström et al. (2004)	• Burnout high vs. low • Work day vs. day off • 10 men, 14 women (aged 24–43)	*ns* ↓	*ns* *ns*	*ns* *ns*	*ns* ↓	— —	*ns* *ns*	*ns* (arousal ↑) *ns*	↓ *ns*	— —	
Cartwright and Wood (1991)	• Ongoing marital separation 19 versus 42 complete divorce	—	*ns*	↓	↑	—	↓	—	*ns*	—	Incomplete divorce; non-depressive only
Hall et al. (1997)	• Stressful events (intrusion, avoidance) • 10 men, 30 women (aged 50–80)	↓	↑	*ns*	—	—	*ns*	—	*ns*	—	
Hall et al. (1998)	• Bereavement (intrusion, avoidance) • 29 major depressive disorder (aged 40–78)	—	↑	*ns*	—	—	—	↑ (1st NREM)	*ns*	—	
Reynolds et al. (1993)	• Bereavement (no depression) • 8 men, 19 women (aged 68.4 ± 6.1 years) versus 27 control (aged 72.7 ± 6.1 years)	—	*ns*	*ns*	*ns*	↑ (1st and 3rd REM period)	*ns*	—	*ns*		
Williamson et al. (1995)	• Stressful life events • 35 depressed and 33 average adolescents	— —	*ns* *ns*	*ns*	*ns* (↓) ↑	— —	*ns* (↓) ↓	— —	— —	— —	

Note. PSG = polysomnography; TST = total sleep time; SL = sleep latency; REML = REM sleep latency; REMT = total REM sleep time; REM density = frequency of rapid eye movement per hour; SWST = slow wave sleep time (% or minute if specified); SE = sleep efficiency; NREM = non-REM sleep; ↑ = increased; ↓ = decreased.

TABLE 2

Studies of Experimental Stress

Author	Stressor and Participants	PSG Findings									Comment on Findings
		TST	SL	REML	REMT	REM Density	SWST	Awakening	SE	Body Movement	
Adam (1982)	• Indwelling venous catheter • 4 men, 3 women (aged 53–63)	↓	*ns*	*ns*	↓	—	*ns*	↑	↓	—	
Prinz et al. (2001)	• Indwelling catheter • 42 women (aged 69.6 ± 6.2 years)	—	↓	—	↓ (minute)	—	↓ (minute)	↑	↓	—	
Prinz et al. (2000)	• Indwelling catheter • 60 women, 28 men (aged 55–82)	—	—	—	—	—	↓	—	↓	—	
Vitiello et al. (1996)	• Blood sampling via indwelling catheter • 68 women, 45 men (aged 69.1 ± 0.6 years)	↓	↑	*ns*	↓	—	↓ (women only)	↑ (total wake time)	↓	—	
Dimsdale et al. (1993)	• Blood pressure cuff inflation • 12 (aged 47.6 ± 8.0 years)	—	—	—	—	—	—	↑	—	—	No sleep architecture data
Aber et al. (1989)	• First-night difference • 14 men (aged 61–83)	↓	↑	*ns*	↓	—	*ns*	↑	—	—	Apnea hypopnea index; *ns*
Agnew et al. (1966)	• First-night effect • 27 men, 16 women (aged 16–31)	—	*ns*	↑	↓	—	*ns*	↑	—	—	
Carvalhaes-Neto et al. (2003)	• Hospitalization (1st night) versus 2nd night • 8 men, 6 women (aged 70–85)	↓	*ns*	*ns*	*ns*	—	↓ (women only)	*ns*	*ns*	—	Apnea index ↑
Coble et al. (1974)	• First-night effect • 9 (aged 20–32)	—	—	↑	*ns*	—	*ns*	↑	*ns*	*ns*	
Curcio et al. (2004)	• First-night effect • 8 men (aged 20–28)	*ns*	↑	*ns*	*ns*	—	*ns*	↑	↓	—	
Kim and Jeong (1998)	• First-night effect • 8 men (aged 23.5 ± 0.9 years)	*ns*	*ns*	*ns*	*ns*	—	*ns*	*ns*	*ns*	—	
Drake et al. (2004)	• First-night effect • High vulnerable versus low • 48 men, 56 women (aged 40.4 ± 12.9 years)	↓	↑	—	*ns*	—	*ns*	—	—	—	
Le Bon et al. (2000)	• First-night adaptation • 243 suspected apnea	↓	↑	↑	↓ (minute)	—	↓ (minute)	↑	↓	—	
Li et al. (2004)	• First-night adaptation	↓	↑	↑	↓	—	*ns*	—	↓	—	

Author	Stressor and Participants	PSG Findings									Comment on Findings
		TST	SL	REML	REMT	REM Density	SWST	Awakening	SE	Body Movement	
	• 46 obese and 44 average (aged 11.2 ± 2.2 years) • 46 obese and 44 average (aged 11.2 ± 2.2 years)										
Scholle et al. (2003)	• First-night effect • A 37 (aged 2–6); B 60 (aged 7–12); C 34 (aged 13–17)	*ns* *ns*	— —	*ns* *ns*	↓ ↓	— —	*ns* *ns*	↑ ↑	*ns* ↓	— —	
Toussaint et al. (1997)	• Lab adaptation • 13 men, 5 women (aged 25.8 ± 5.2 years)	*ns*	*ns*	*ns*	*ns*	—	*ns*	↑	↓	—	
Baekeland et al. (1968)	• Stress film versus neutral film • 12 men	—	*ns*	*ns*	*ns*	↑	—	↑ from REMP	—	*ns*	
Cluydts and Visser (1980)	• Surgical color film • 6 men, 4 women (aged 22–25)	—	—	—	*ns*	—	↓	↑ number of awakening	*ns*	—	
Cohen (1975)	• Negative versus positive pre-sleep condition • 17 high neuroticism, 14 low neuroticism men	—	—	—	—	↑ in low neuroticism group	—	—	—	—	
Goodenough et al. (1975)	• Stress film versus neutral film • 28 male night workers	*ns*	↑	—	—	*ns*	—	—	—	—	
Germain et al. (2003)	• Expectation of speech • 32 men, 31 women (aged 18–24)	—	*ns*	*ns*	*ns*	*ns* (↓ REM counts in last REMP)	*ns*	—	*ns*	—	Influenced by situational factor more than dispositional factor
Hall et al. (2004)	• Expectation of speech • 30 men, 29 women (aged 19.6 ± 1.8 years)	*ns*	—	—	*ns*	—	*ns*	*ns*	*ns*	—	Heart rate variability difference in NREM
Koulack et al. (1985)	• Intelligence test before sleep • 16 men (aged 18–24)	*ns*	↑	*ns*	*ns*	↓	—	—	—	—	
Lauer et al. (1987)	• Stressful movie versus neutral movie • 11 men (aged 19–27)	—	*ns*	*ns* (↑)	—	*ns*	*ns*	*ns*	*ns*	—	
Levin et al. (2002)	• Emotional stress • 5 men, 5 women (aged 21–33) • Non-adaptive group versus adaptive group	— —	*ns* ↑	— —	↑ (1st cycle) *ns*	— —	↓ (1st cycle) ↓ (1st cycle)	↑ (number) ↓ (number)	— —	*ns* ↓	First-night effect in adaptive group (REM ↓, movement time ↑, awakening ↑)
Beaumaster et al. (1978)	• Parachute jump • 22 men, 5 women (aged 18–29) • 9 novice, 9 experienced jumpers versus 9 control	*ns* *ns*	*ns* ↑	*ns* *ns*	*ns* *ns*	— —	*ns* *ns*	*ns* *ns*	— —	— —	
Vein et al. (2002)	• Psychoemotional tension	—	↑	*ns*	*ns* (1st cycle ↑)	—	*ns* (1st cycle ↓, 2nd cycle ↑)	*ns* (↑ waking time)	—	—	

Author	Stressor and Participants	PSG Findings									Comment on Findings
		TST	SL	REML	REMT	REM Density	SWST	Awakening	SE	Body Movement	
	• 5 men, 5 women (aged 21–33) • 5 men, 5 women (aged 21–33)										

Note. PSG = polysomnography; TST = total sleep time; SL = sleep latency; REML = REM sleep latency; REMT = total REM sleep time; REM density = frequency of rapid eye movement per hour; SWST = slow wave sleep time (% or minute if specified); SE = sleep efficiency; NREM = non-REM sleep; REMP = REM sleep period; ↑ = increased; ↓ = decreased.

TABLE 3

Studies of Traumatic Stress

Author	Stressor and Participants	PSG Findings									Comment on Findings
		TST	SL	REML	REMT	REM Density	SWST	Awakening	SE	Body Movement	
Astrom (1989)	• Torture in prison (6 months–7 years) • 7 men (aged 23–27), 7 controls	↓	*ns*	*ns*	*ns*	—	↓	↑	↓	—	Different ethnicity in groups
Breslau et al. (2004)	• Trauma? • 71 PTSD versus 113 exposed, no PTSD versus 99 not exposed	*ns*	*ns*	*ns*	*ns*	—	*ns*	*ns* (↑ arousal from REM)	*ns*	—	10-year cohort Fragmentation ↑
Dow et al. (1996)	• Vietnam combat • PTSD + MDD 14, MDD 15, 12 controls	↓ *ns* (↓)	*ns* ↑	*ns* (↓) *ns*	*ns* *ns*	*ns* *ns*	*ns* *ns*	— —	*ns* *ns* (↓)	— —	Including summary of other articles
Engdahl et al. (2000)	• War trauma • 30 PTSD among 59 elderly veterans	*ns*	*ns*	*ns*	↑	*ns*	*ns*	*ns* (↓ arousal from NREM)	*ns*	—	
Germain & Nielsen (2003)	• Trauma • 9 PTSD nightmare versus 11 idiopathic nightmare • 7 men, 6 women controls	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	↑	↓	↑ PLMS	
Greenberg et al. (1972)	• Vietnam War • 7 war neuroses	—	—	↓	*ns*	—	—	—	—	—	No control/mixed with depression
Hefez et al. (1987)	• Traumatic events survivors • 11 men (Holocaust survivors 5, combat, sea disaster 6)	↓ ↓	*ns* ↑	*ns* (↑) ↑	↓ ↓	↑ (overall)	↑ *ns*	↑ ↑	↓ ↓	↑ ↑	
Hurwitz et al. (1998)	• Vietnam combat • 18 PTSD versus 10 controls	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	SL to stage 2 ↑ Arousal from SWS ↓
Klein et al. (2002)	• Traffic accidents (after 1 year) • 8 PTSD versus 6 victims without PTSD	*ns*	*ns*	*ns*	*ns*	—	*ns*	*ns*	*ns*	—	All are victims
Lavie et al. (1979)	• Combat • 11 combat neurosis (aged 24–33) versus 9 controls (aged 24–27)	↓	*ns*	↑	↓	—	*ns*	↑	↓	—	
Lavie et al. (1998)	• Combat • 12 PTSD (aged 31 ± 4.4 years) versus 12 controls (aged 32.8 ± 5.3 years)	*ns*	*ns*	*ns*	*ns*	—	*ns*	*ns*	*ns*	*ns*	Awakening latency from REM ↑
Lavie & Hertz (1979)	• Combat	—	—	—	—	—	—	—	—	↑ in Stage 2	Higher respiratory rate in NREM

		PSG Findings									
Author	**Stressor and Participants**	**TST**	**SL**	**REML**	**REMT**	**REM Density**	**SWST**	**Awakening**	**SE**	**Body Movement**	**Comment on Findings**
	• 11 combat neurosis versus 9 controls • 11 combat neurosis versus 9 controls										
Mellman, Kulick-Bell, et al. (1995)	• Combat • 21 veterans PTSD versus 8 control	*ns*	*ns*	*ns*	*ns*	—	*ns*	↑	↓	—	
Mellman, David, et al. (1995)	• Hurricane Andrew • 10 victims, 9 unaffected	*ns*	*ns*	*ns*	*ns*	*ns*	*ns*	*ns* (↑ micro-awakening)	*ns*	—	Significant correlation between PTSD symptom and REM density
Mellman et al. (1997)	• Combat • 24 PTSD versus 15 depressed versus 9 controls	↓	*ns*	*ns*	*ns*	↑ (P = D > control)	*ns*	↑	↓	—	
Mellman et al. (2002)	• Various injury, <1 month • 16 male, 5 female victims (10 PTSD & 11 non-PTSD) versus 10 controls	*ns* ↓	*ns* *ns*	*ns* *ns*	*ns* *ns*	*ns* ↑	*ns* *ns*	*ns* ↑	— —	— —	Patients who developed PTSD: shorter duration of REMP, more number of REMP
Ross et al. (1994a)	• Combat • 12 male PTSD versus 10 male controls	—	—	—	—	*ns* (↑ with REM sleep phasic leg activity)	—	—	—	↑ REM sleep phasic leg activity, ↑ PLMS in NREM	
Ross et al. (1994b)	• Combat • 12 PTSD versus 10 controls	*ns*	*ns*	*ns*	↑	↑	*ns*	*ns*	*ns*	—	
Ross et al. (1999)	• Adaptation • 17 PTSD versus 11 controls	*ns*	*ns*	*ns*	↑	↑ (1st REMP)	*ns*	—	*ns*	—	No interaction between night and group
Schlosberg & Benjamin (1978)	• Acute combat fatigue • 3 men	↓	↑	↑	↓	—	↓	↑	↓	—	Case report
Woodward et al. (1996)	• Combat • 27 PTSD inpatients (aged 39–8): +MDD 17 versus –MDD 10	*ns*	*ns*	*ns*	*ns*	—	↓	*ns*	*ns*	—	
Woodward, Arsenault, et al. (2000)	• Combat • 63 PTSD inpatients (with nightmares)	*ns*	*ns*	*ns*	*ns*	—	*ns*	↑ (WASO)	*ns*	*ns*	No control, no stress effect
Woodward, Murburg, & Bliwise (2000)	• Combat • 56 PTSD inpatients versus 15 controls	*ns*	—	—	↑	—	*ns*	*ns*	*ns*	—	
Woodward et al. (2002)	• Combat • 84 male, 4 female PTSD inpatients • 16 male, 7 female controls	*ns*	*ns*	*ns*	*ns*	—	*ns*	*ns*	*ns*	↓ (MT)	
Woodward et al. (2003)	• Combat	*ns*	*ns*	*ns*	*ns*	—	*ns*	*ns*	*ns*	↓ (MT)	PTSD with panic: MT ↓ PTSD with nightmare: MT ↓, Stage 4 ↓

		PSG Findings									
Author	**Stressor and Participants**	**TST**	**SL**	**REML**	**REMT**	**REM Density**	**SWST**	**Awakening**	**SE**	**Body Movement**	**Comment on Findings**
	• 11 PTSD + panic and 38 PTSD inpatient versus 15 controls • 11 PTSD + panic and 38 PTSD inpatient versus 15 controls • 11 PTSD + panic and 38 PTSD inpatient versus 15 controls										
Kramer & Kinney (1988)	• Vietnam combat • 8 PTSD versus 8 veterans	*ns* *ns*	*ns* ↑	↑ *ns*	↓ ↓	— —	↓ ↓	↓ ↓	↓ ↓	—	No control (compared to norm)
Mikulincer et al. (1989)	• 7 PTSD (aged 31–60) versus 7 male controls	—	↑	↑	↓	—	↓	↑	↓	—	

Note. PSG = polysomnography; TST = total sleep time; SL = sleep latency; REML = REM sleep latency; REMT = total REM sleep time; REM density = frequency of rapid eye movement per hour; SWST = slow wave sleep time (% or minute if specified); SE = sleep efficiency; NREM = non-REM sleep; PLMS = periodic limb movements during sleep; PTSD = post-traumatic stress disorder; REMP = REM sleep period; MDD = major depressive disorder; WASO = wake after sleep onset; MT = movement time; ↑ = increased; ↓ = decreased.

TABLE 4

Summary of Changes of Major Sleep Variables in Response to Daily Stress–Four Studies

Variable	No Data	Reported Data	↓ (% Among Reported)	↑ (% Among Reported)	No Change (% Among Reported)
Slow wave sleep	0	4	50.0	25.0	25.0
REM sleep	2	2	50.0	50.0	0.0
Sleep efficiency	3	1	0.0	0.0	100.0
Sleep latency	2	2	50.0	0.0	50.0
REM sleep latency	1	3	33.3	33.3	33.3
Awakening	3	1	0.0	100.0	0.0

TABLE 5

Summary of Changes of Major Sleep Variables in Response to a Stressful Life Event–Five Studies

Variable	No Data	Reported Data	↓ (% Among Reported)	↑ (% Among Reported)	No Change (% Among Reported)
Slow wave sleep	1	4	50	0.0	50
REM sleep	2	3	0.0	66.7	33.3
Sleep efficiency	1	4	0.0	0.0	100.0
Sleep latency	0	5	0.0	40	60
REM sleep latency	0	5	40	0.0	60
Awakening	4	1	0.0	100.0	0.0

TABLE 6

Summary of Changes of Major Sleep Variables in Response to Experimental Stress–27 Studies

Variable	No Data	Reported Data	↓ (% Among Reported)	↑ (% Among Reported)	No Change (% Among Reported)
Slow wave sleep	5	22	31.8	0.0	68.2
REM sleep	5	22	36.4	4.5	59.1
Sleep efficiency	11	16	56.3	0.0	43.8
Sleep latency	7	20	5.0	45.0	50.0
REM sleep latency	9	18	0.0	22.2	77.8
Awakening	7	20	0.0	75.0	25.0

TABLE 7

Summary of Changes of Major Sleep Variables in Response to Traumatic Stress–27 Studies

Variable	No Data	Reported Data	↓ (% Among Reported)	↑ (% Among Reported)	No Change (% Among Reported)
Slow wave sleep	3	24	20.8	4.2	75.0
REM sleep	2	25	20.0	16.0	64.0
Sleep efficiency	4	23	39.1	0.0	60.9
Sleep latency	4	23	0.0	8.7	91.3
REM sleep latency	3	24	4.2	16.7	79.2
Awakening	5	22	0.0	45.5	54.5

TABLE 8

Meta-Summary of Stress Effects on Changes of Major Sleep Variables–63 Studies

Variable	No Data	Reported Data	↓ (% Among Reported)	↑ (% Among Reported)	No Change (% Among Reported)
Slow wave sleep	9	54	29.6	3.7	66.7
REM sleep	11	52	26.9	15.4	57.7
Sleep efficiency	19	44	40.9	0.0	59.1
Sleep latency	13	50	4.0	26.0	70.0
REM sleep latency	13	50	8.0	18.0	74.0
Awakening	19	44	0 0	61.4	38.6