Published in final edited form as:
J Anxiety Disord. 2010 January ; 24(1): 1–15. doi:10.1016/j.janxdis.2009.08.002.

Temporal Relations between Sleep Problems and both Traumatic Event Exposure and PTSD: A Critical Review of the Empirical Literature

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Abstract

There has been growing interest in the interrelations among traumatic event exposure, posttraumatic stress disorder (PTSD), and sleep problems. A wealth of research has examined the associations among these factors and there is an emerging literature focused on how sleep problems relate to both traumatic event exposure and PTSD across time. The current review provides a detailed analysis of studies pertaining to the temporal patterning of sleep problems and traumatic event-related factors (e.g., traumatic event exposure, PTSD) and draws conclusions regarding the current state of this literature. Research coalesces to suggest (1) exposure to a traumatic event can interfere with sleep, (2) PTSD is related to the development of self-reported sleep problems, but evidence is less clear regarding objective indices of sleep, and (3) limited evidence suggests sleep problems may interfere with recovery from elevated posttraumatic stress levels. Future research now needs to focus on understanding mechanisms involved in these patterns to inform the prevention and treatment of comorbid sleep problems and PTSD.

Keywords

sleep; posttraumatic stress disorder; traumatic event; PTSD; sleep deprivation

The current paper aims to provide a review of the research that speaks to temporal patterning in the well-established associations between sleep problems and both traumatic event exposure and posttraumatic stress disorder (PTSD). A background is first provided that highlights the importance of advancing the currently limited understanding of this area. Individual studies pertaining to how sleep problems and both traumatic event exposure and PTSD relate across time are then reviewed in detail. Finally, an integrative summary is provided that includes conclusions, limitations, and future directions for this area of research.

Historically, sleep problems have been conceptualized as a secondary symptom of PTSD (Hefez et al., 1987). However, recent accounts have suggested that sleep problems may be central to the PTSD syndrome (Spoormaker & Montgomery, 2008). A wealth of data suggests sleep problems are correlated with traumatic event exposure and PTSD (Goldstein, van Kammen, Shelly, Miller, & van Kammen, 1987; North et al., 1999; Pillar, Malhotra, & Lavie,

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2000; Schuster et al., 2001). For example, sleep problems were the most prevalent symptom among individuals who survived the 1995 Hanshin earthquake in Japan (Kato, Asukai, Miyake, Minakawa, & Nishiyama, 1996) and among survivors of the Holocaust (Kuch & Cox, 1992). The relation between traumatic event exposure and sleep problems is particularly pronounced among persons who do not recover from initial symptomatic reactions to such exposure, including persons with PTSD (Mellman & Hipolito, 2006; Neylan et al., 1998). Indeed, approximately 70% of people with PTSD also have co-occurring sleep problems and individuals with, compared to without, PTSD report greater trouble initiating (41% and 13%, respectively) and maintaining (47% and 18% respectively; Ohayon & Shapiro, 2000) sleep. These data highlight that sleep problems commonly occur among people exposed to a traumatic event, particularly those who develop PTSD. However, given these data are cross-sectional, they provide limited information regarding the secondary versus central nature of sleep problems in PTSD (and vice versa). In order to better understand why sleep problems so frequently co-occur with traumatic event exposure and PTSD recent research has focused on understanding the temporal patterning among these factors.

The current review aims to uniquely contribute to this literature by summarizing and critically evaluating the literature regarding temporal relations among sleep problems, traumatic event exposure, and PTSD. A focus on relations across time was adopted because although reviews have outlined the associations between PTSD and sleep problems (Caldwell & Redeker, 2005; Germain, Buysse, & Zofzinger, 2008; Harvey, Jones, & Schmidt, 2003; Kobayashi, Boatres, & Delahanty, 2007; Lamarche & DeKoninck, 2007; Spoormaker & Montgomery, 2008), no review has specifically focused on studies of how these factors relate across time. Focused reviews of this sort are needed as scholars in the area have recently noted that, despite a relatively well-developed literature, the overlap between sleep and mental health problems requires further research as significant gaps in our understanding of this common co-occurrence remain (Mellman, 2008). Further understanding the temporal ordering of these problems is critical both for developing and modifying clinical interventions aimed at impacting the high public health costs associated with these commonly comorbid conditions. A refined understanding in this area will yield testable hypotheses for treatment outcome work. For instance, if posttraumatic stress reactions maintain sleep problems, PTSD treatment may help to alleviate sleep problems (see David, DeFaria, & Mellman, 2006). Understanding the development of these commonly comorbid problems also can inform the development of PTSD and sleep problem prevention protocols targeting this hard to treat population (Bryant, Harvey, Dang, Sackville, & Batsen, 1998; Harvey & Bryant, 2002). For example, if sleep problems potentiate the maintenance of posttraumatic stress reactions, psychosocial programs targeting improvements in this type of health behavior could help prevent PTSD by promoting healthy recovery from traumatic event exposure (Feldner, Monson, & Friedman, 2007). Also, a detailed analysis of all studies pertaining to cross-sectional and longitudinal relations among traumatic event exposure, posttraumatic stress, and sleep problems is beyond the scope of a single review due to the large size of the literature (i.e., over 700 published studies). In contrast, a detailed focus on studies pertaining to temporal patterning will uniquely draw together and evaluate an important and growing subset of this work.

Given this backdrop, the current review aimed to provide a detailed review of research conducted to date on how sleep problems and both traumatic event exposure and PTSD relate across time. Prior to the detailed review, operational definitions of key terms and the method employed herein will be delineated.

Sleep Problems

The terminology utilized to describe sleep problems varies within the literature. For example, sleep deprivation, sleep loss, insomnia, and sleep disturbance have all been used to describe

insomnia. Within the overarching construct of insomnia, two subcomponents (sleep onset and sleep maintenance) have been identified (American Sleep Disorders Association, 1990). Specifically, sleep-onset insomnia refers to difficulty falling asleep; sleep maintenance insomnia refers to frequent awakenings during the night or early morning awakenings. Untreated, insomnia typically lasts about 4 years, with many people (44% of those with untreated insomnia) experiencing insomnia for up to 10 years (Drake et al., 2003), and it results in high levels of impairment and health care costs (U.S. Surgeon General, 2004). Indeed, negative effects of insomnia include daytime fatigue, problems with concentration and memory, irritability, and an increase in errors and mistakes in work-related tasks (Hoelscher et al., 1993; Roth & Ancoli-Israel, 1999; Simon & VonKorff, 1997).

Polysomnography refers to the objective assessment of the architecture of sleep. Polysomnography utilizes measures of heart rate, eye movement, muscle tension (electromyogram; EMG), respiration, and electroencephalogram (EEG) monitoring to index movement as well as track transitions through sleep stages. Polysomnography allows for the assessment of critical components of sleep architecture including rapid eye movement (REM), REM density, and sleep efficiency. REM sleep is the stage of sleep associated with dreaming and consolidation of memories (Maquet, 2001). REM density is a measure of the frequency of rapid eye movements during REM sleep, which typically increases over the course of sleep. Sleep efficiency refers to the ratio between total time asleep and the amount of time spent in bed. For example, an individual with low sleep efficiency may spend a lot of time in bed; however, much of that time is spent awake (Papadimitriou & Linkowski, 2005).

Traumatic Event Exposure and Posttraumatic Stress Disorder

A traumatic event is an event during which an individual experiences perceived threat and helplessness, terror, or horror [American Psychiatric Association (APA), 2000]. Exposure to a traumatic event includes individuals either experiencing or witnessing an event during which they experience intense fear, helplessness, or horror. Potentially traumatic events (PTE) are defined as situations which have been shown to increase the chances of developing PTSD as a result of exposure to the event (e.g., sexual assault, combat, torture), but do not consider the individuals subjective responses currently employed in the definition of a traumatic event (APA, 2000). Exposure to traumatic events is common, with an estimated 68% of children experiencing such an event (Copeland, Keeler, Angold, & Costello, 2007) and 55% (Kessler et al., 1995) to 69% (Norris, 1992; Resnick et al., 1993) of adults in the United States experiencing traumatic events. These rates are even higher among certain at-risk groups, such as military personnel (79%; Schnurr et al., 2002). The majority of individuals exposed to a traumatic event develop acute symptomatic responses (e.g., avoiding the context where the traumatic event occurred; Kessler et al., 1995; Rothbaum et al., 1992). Although traumatic event exposure is most often linked to PTSD (Kessler et al., 1995), it also increases risk for depression, generalized anxiety disorder, specific phobias, and substance use problems (Acierno et al., 2000; Blumenthal et al., 2008; Cohen et al., 2004; Feldner, Babson, & Zvolensky, 2007; Fletcher, 1996, Kaplan et al., 1998; Kassam-Adams & Winston, 2004; Kilpatrick et al., 2000). While posttraumatic stress symptoms remit without intervention among most traumatic event-exposed individuals, symptoms do not decrease among a substantial minority of these individuals (Gilboa-Schechtman & Foa, 2001; Kessler et al., 1995; Kilpatrick & Resnick, 1993).

In contrast to other anxiety disorders, such as panic disorder and social phobia, PTSD is considered a disorder of recovery, characterized by a failure to recover from a stress reaction following exposure to a traumatic event (Kessler et al., 1995). Posttraumatic stress disorder was introduced to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) in version III released in 1980. Between 1980 and 2009 the DSM has undergone three revisions (DSM

III-R, DSM IV, DSM IV TR). As a result of these revisions, the diagnostic criteria for PTSD have also undergone changes throughout this time.[1] Currently, PTSD is defined as the non-remittance of symptoms (i.e., at least one reexperiencing symptom, three or more of avoidance/numbing, two of hyperarousal; APA, 2000) by one month post-traumatic event exposure. Exemplar symptoms include the following: flashbacks (reexperiencing); inability to experience emotion, avoiding people and places associated with the event (avoidance/numbing); and increased startle response and hypervigilance (hyperarousal). Posttraumatic stress disorder is common (Kessler et al., 2005; Resnick et al., 1993), frequently does not remit without intervention (Kessler et al., 1995), and results in high levels of functional impairment and health care costs (Amaya-Jackson et al., 1999; Zatzick et al., 1997).

Selection of Studies

A literature search was conducted using the following electronic search engines: PsycINFO, Medline, Pilots, and PsycArticles. Within each search, all combinations of the following key terms were used: sleep, insomnia, and trauma, traumatic event, or posttraumatic stress disorder (or PTSD) and prospective or longitudinal. Possibly relevant references within these articles were also obtained. Two general areas emerged from this search that warrant brief mention: (1) the effects of anxiolytic and/or sleep medications on one or both conditions and (2) the relation between sleep and general anxiety. The current review does not focus on an overview of these areas as each has been systematically and independently reviewed elsewhere (Papadimintriou & Linkowski, 2005; van Liempt, Vermetten, & Geuze, 2006) and a detailed analysis of this work is beyond the scope of a single review. Overall, the above-described literature search yielded 51 articles. Articles were then only included if there was a specific focus on traumatic event exposure or PTSD and sleep problems. Based upon these criteria, a total of 14 articles were included in the final review and are discussed in detail below.

Retrospective Studies

First, studies that employed retrospective designs aimed at understanding temporal patterns among traumatic event exposure, PTSD, and sleep problems will be reviewed. In the first section, studies that focus on linkages between traumatic event exposure and sleep problems will be reviewed, followed by studies that examine PTSD and sleep problems. Separating studies of traumatic event exposure and PTSD will facilitate conclusions regarding the (possibly) unique contributions of each. To allow for a focus within the text on integration of the studies and drawing conclusions, specific details of the method and results of each study are displayed in Table 1, where studies are listed in alphabetical order by author name.

Traumatic Event Exposure

Four studies have been published in this domain. Three of these focused on aspects of the traumatic event and the associated effect(s) on sleep. The last study examined the relation between childhood traumatic event exposure and adult sleep problems. Hefez, Metz, and Lavie

[1] An overview of definitions of PTSD based on the different versions of DSM.
DSM III: Criteria A: "The individual has experienced an event that is outside the range of usual experience and that would be markedly distressing to almost anyone." DSM III also requires symptoms of (B) reexperiencing, (C) avoidance, (D) hyperarousal, and (E) duration.
DSM III-R: Criteria A: "The person has experienced an event that is outside the range of usual human experience and that would be markedly distressing to almost anyone (e.g., serious threat to one's life or physical integrity, serious threat or harm to one's children, spouse, or other close relatives and friends, sudden destruction of one's home or community, or seeing another person who has been or is being, seriously injured or killed as the result of an accident or physical violence)." DSM III-R also requires symptoms of (B) reexperiencing, (C) avoidance, (D) hyperarousal, and (E) duration.
DSM IV and DSM-IV TR: Criteria A: "The person has been exposed to a traumatic event in which both of the following have been present: (1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others; (2) the person's response involved intense fear, helplessness, or horror." DSM IV also requires symptoms of (B) reexperiencing, (C) avoidance, (D) hyperarousal, (E) duration, and (F) subjective distress.

(1987) examined the role of different types of traumatic event exposure on sleep problems. A total of 11 traumatic event survivors (5 Holocaust survivors, 3 combat veterans, and 3 sea disaster survivors) and 9 age- and gender-matched controls without a history of traumatic event exposure participated. Holocaust survivors were assessed 45 years post-traumatic event, combat veterans were assessed either 6 or 14 years post-traumatic event, and survivors of the sea disaster were assessed 6 and 12 months post-traumatic event. Polysomnography was conducted over two to five consecutive nights. All traumatic event-exposed participants had lower sleep efficiency, increased sleep latency, shorter REM time, and longer REM latency in comparison to control participants. Interestingly, time since traumatic event exposure was related to sleep problems. Within 18 months post-traumatic event, participants displayed increased REM motor activity, fragmented REM, and increased awakenings. In comparison, those assessed multiple years after traumatic event exposure did not display these characteristics; however they did demonstrate shorter REM time and increased latency to REM. These results suggest the effects of traumatic event exposure on sleep architecture may depend, at least to a certain degree, on time since traumatic event exposure. A particular contribution of this study was the utilization of polysomnography techniques to assess sleep architecture of individuals exposed to traumatic events at varying lengths of time since the traumatic event. However, groups differing in time-since-traumatic event also differed in terms of traumatic event type, so conclusions regarding the relation between time-since-traumatic event and variability in sleep architecture must be made cautiously. Finally, it is important to consider the possible role of additional traumatic event exposures that may have occurred between the initial traumatic event exposure (i.e., sea disaster, war) and the assessments.

Kaminer and Lavie (1991) also examined the role of surviving the Holocaust on sleep. Participants were excluded for psychiatric diagnoses (including DSM III-defined PTSD). However, differentiation between groups was made based upon post-Holocaust adjustment, including problems with work, social life, somatic complaints, and general satisfaction with life. Three groups were created as follows: (1) well-adjusted survivors of the Holocaust (n = 12), (2) poorly adjusted survivors of the Holocaust (n = 11), and (3) a control group present during the Holocaust but not involved in concentration camps (n= 10). All participants underwent four nights of polysomnography. Results indicated poorly adjusted survivors experienced longer latency to sleep onset and lower sleep efficiency in comparison to well-adjusted survivors and controls. In addition, dream recall differed between the well-adjusted and poorly-adjusted survivors. Well-adjusted survivors reported recalling fewer dreams compared to the poorly-adjusted survivors and controls. The internal validity of this study, relative to those that preceded it, is noteworthy. Specifically, the design allowed for suggesting poorly-adjusted survivors awoke from sleep more frequently and had a greater difficulty falling back asleep compared to well-adjusted survivors and control participants. Despite such strengths, the study is limited as well. It did not directly test the relation of sleep and PTSD; comparable designs that focus on specific PTSD sequelae would be beneficial. In addition, while emphasizing internal validity, the study results cannot be generalized to other samples or traumatic event types. Replicating these findings across different traumatic event types would be a beneficial next step.

Rosen, Reynolds, Yeager, Houck, and Hurwitz (1991) compared the sleep of Holocaust survivors (n = 42) to a control group of healthy elderly individuals (n = 54), and a group of depressed elderly individuals (n = 37). Participants completed questionnaires and a structured clinical interview pertaining to sleep. Holocaust survivors also completed specific information about their Holocaust experience (e.g., time spent in concentration camps). Results indicated Holocaust survivors reported significantly greater sleep problems than healthy control participants. Furthermore, there was a significant positive correlation between the number of years spent in the concentration camp and the total number of sleep problems. Results suggest survivors of the Holocaust experienced significantly more sleep problems compared to healthy

controls and depressed controls even 45 years after the traumatic event(s) occurred. Furthermore, those who spent more time in the concentration camps experienced greater levels of sleep problems defined as lesser self-reported quality of sleep. While this study addressed the long term effects of severe traumatic event exposure on sleep problems, conclusions drawn from this paper do not generalize to other types of traumatic event experiences. In addition, it is important to assess for additional traumatic event exposures occurring between the index event and the assessment. Future research would benefit from replicating these findings among other traumatic event types and controlling for subsequent traumatic event exposure.

To date, limited research has addressed the role of childhood traumatic event exposure on sleep problems in adulthood. In a recent study Noll, Trickett, Susman, and Putnam (2006) tested the role of childhood traumatic event exposure predicting subsequent sleep problems. Seventy-eight sexually abused females were compared on measures of sleep, PTSD, depression, and revictimization, to 69 age-matched females without a history of childhood sexual abuse. Sexually abused females were identified, and information regarding the abuse and psychological symptom levels (i.e., sleep problems, PTSD) were obtained through protective service agencies of Washington D.C. All females were contacted 10 years post-disclosure of the abuse, and completed an interview including a PTSD diagnostic interview, and measures of sleep and revictimization. Participants in the comparison group also completed the interviews and measures. Results indicated sexually abused females (who reported elevated reexperiencing, arousal, and avoidance symptoms of PTSD, and greater levels of revictimization relative to control participants) experienced significantly greater sleep problems compared to non-traumatic event-exposed females. Results indicated childhood sexual abuse was a unique predictor of sleep problems 10 years post-traumatic event, even after controlling for symptoms of depression and PTSD. While this is one of the only studies examining the effects of childhood traumatic event exposure on adulthood sleep problems, future research may benefit from investigating other types of traumatic events and key components of the traumatic event (i.e., number of incidences, age of first traumatic event exposure, social support) as they relate to sleep in adulthood.

PTSD

Three studies are presented in this section, one that speaks to the potential role of pre-traumatic event exposure sleep problems in enhancing the likelihood of PTSD (presented first), and two that address the role of PTSD in exacerbating sleep problems. Mellman, David, Kulick-Bell, Hebding, and Nolan (1995) tested the role of sleep in recovery from recent traumatic event exposure. A total of 54 adults (12 men, 42 women) who were directly affected by Hurricane Andrew participated. Sleep assessments measured current sleep problems as well as retrospectively reported sleep problems before being exposed to a hurricane. Objective measures of sleep (polysomnography) were administered to (1) a subgroup of hurricane victims with psychiatric diagnoses and (2) a healthy comparison group not affected by the hurricane. All participants were free of medications as well as drug abuse and none had psychiatric conditions prior to the hurricane. Assessments were conducted 6 to 12 months after the hurricane to measure sleep problems, PTSD, major depressive disorder (MDD), and anxiety disorders. Post-hurricane, 52% of participants met criteria for a new-onset disorder (within this group, 35% met criteria for PTSD, 30% for MDD, and 20% for another anxiety disorder; an additional 26% met subsyndromal PTSD or MDD). At the time of the 12 month assessment 57% continued to meet criteria for a disorder (within this group, 84% met criteria for PTSD, 48% for MDD, and 32% for another anxiety disorder). A subset of participants with active psychopathology participated in a laboratory assessment of sleep (n = 10; 6 women). Six met criteria for PTSD and four met criteria for the reexperiencing and heightened arousal symptom clusters. Two nights were spent in the sleep laboratory. Measures included electrooculogram, EEG, EMG, and REM density. No significant group differences were observed in relation to

these measures. In terms of pre-hurricane sleep problems, results indicated those with the highest levels of bad dreams, global severity of sleep problems, sleep disturbances, and awakenings had greater levels of active psychiatric morbidity (including PTSD; see Table 1). Specifically, PTSD was related to greater sleep problems, including global sleep problems, sleep quality, sleep disturbance, and greater awakenings compared to those without PTSD. These data coalesce to suggest sleep problems prior to a traumatic event may increase the likelihood of developing PTSD.

Lavie, Hefez, Halperin, and Enoch (1979) compared sleep problems among a sample of patients diagnosed with "combat neurosis" as a result of the Yom Kippur war of 1973 to a control group of active service veterans during the Yom Kippur war without "combat neurosis." [2] Combat neurosis was used to describe the following symptoms: depression, irritability, uncontrolled fits of rage, headaches, pain in various parts of the body, sleep disturbances, and nightmares. All participants were referred for a laboratory assessment of sleep 2 to 2.5 years after traumatic event exposure. Based upon retrospective account, none of the participants had sleep problems prior to the traumatic event exposure. All participants completed three to four consecutive nights of polysomnography. Physiological measures included ECG, EMG, EEG, and respiration. Sleep measures included time asleep, sleep latency, REM latency and depth, and REM percentage. A mean was created from the data over the three nights excluding night one. Results indicated individuals with "combat neurosis" compared to both control groups experienced less REM percentage, increased REM latency, shorter total sleep time, and decreased sleep efficiency. Overall, these findings suggest individuals exposed to a traumatic event that go on to develop symptoms broadly comparable to PTSD, experience significant sleep problems 2 to 2.5 years after the traumatic event. Conclusions drawn from this study should be made cautiously in light of significant limitations. This study was conducted prior to the inclusion of PTSD as a diagnostic category in the DSM. For this reason, while some symptoms are comparable to current conceptualizations of PTSD (e.g., sleep problems, irritability, nightmares), other symptoms are inconsistent with current conceptualizations (i.e., pain, headaches). This difference limits confidence regarding the generalizability of these findings to models regarding the relation between PTSD and sleep problems across time.

Dagan, Lavie, and Bleich (1991) conducted a study on awakening thresholds (i.e., decibel level of a tone presented during stage 3/4 of sleep required to awaken the individual) of individuals with ($n = 19$) and without ($n = 6$) PTSD (defined via DSM-III criteria) as a result of participation in the Lebanon War. Four to six years post-traumatic event all participants' sleep problems were evaluated via polysomnography. Participants were free from all medications and substance use for at least 10 days prior to the study. Findings suggested sleep within the PTSD group was comparable to controls. However, those with PTSD had increased awakening thresholds in comparison to controls. This finding suggests those with PTSD may experience greater depth of sleep and therefore more difficulty in waking. Generalizability of these findings must consider the retrospective nature of reports of the traumatic event as well as the amount of time between the traumatic event and the sleep assessment.

Summary

Several retrospective studies have focused on the role of traumatic event exposure in the development of sleep problems. Broadly, this work has suggested that traumatic event exposure is related to reports of trouble falling asleep, increased awakenings during the night, and trouble regaining sleep after awakening (Hefez et al., 1987; Kaminer & Lavie, 1991; Rosen et al.,

[2]While this study did not utilize a specific diagnosis of PTSD because it was conducted prior to publication of the diagnostic criteria for PTSD in the DSM III (1980), it was included herein because the symptoms addressed were consistent with current PTSD diagnostic criteria. Furthermore, prior reviews focused on sleep and PTSD have included this study as an initial study in this area that has informed more recent research (Harvey et al., 2003).

1991). Additionally, specific characteristics of traumatic event exposure may alter an individual's sleep architecture (Hefez et al., 1987) and subjective sleep (Rosen et al., 1991) post-event exposure. For example, Rosen and colleagues concluded the amount of time in contact with the traumatic event, which may index severity of the traumatic event, was positively correlated to subjective sleep problems (Rosen et al., 1991). This conclusion was based on the finding that survivors of the Holocaust, in comparison to elderly healthy controls, and elderly depressed individuals self-reported significantly more sleep problems. While such conclusions are consistent with data emerging from this literature, additional third factors that may affect (or account for) these relations, such as multiple traumatic event exposures and use of substances and/or medications, need to be considered. In addition, time since traumatic event exposure correlated to changes in sleep architecture, such that the more proximal the event the greater the impact on sleep, including shorter REM time and longer REM latency among traumatic event survivors (Hefez et al., 1987). Importantly, all traumatic event survivors in this study had a diagnosis of PTSD, thereby methodologically controlling for the effects of PTSD diagnoses. Taken together, the aforementioned research has demonstrated that traumatic event exposure (and related characteristics) is related to sleep problems independent of PTSD (Mellman et al., 1995; Noll et al., 2006). Thus, exposure to traumatic events alone can affect sleep problems regardless of recovery from the event. Importantly, the retrospective nature of this work necessitates caution in drawing causal conclusions; the next section reviewing prospective work will further elucidate this relation.

To date, no research has examined sleep problems leading to traumatic event exposure. For example, the effects of sleep loss may impact an individual's ability to drive a vehicle (Philip et al., 2005), thereby increasing the chances of a motor vehicle accident. Future work in this area may prove beneficial in more fully understanding how traumatic event exposure and sleep problems relate across time.

In terms of PTSD (cf., traumatic event exposure), work summarized here suggests individuals exposed to a traumatic event who go on to develop PTSD (and comparable syndromes) experience significant sleep problems 2 - 2.5 years (Lavie et al., 1979) and 45 years post-traumatic event exposure (Rosen et al., 1991). Interestingly, in comparison to findings suggesting PTSD predicts sleep problems, some studies using polysomnography have demonstrated those with PTSD actually have elevated awakening thresholds in comparison to normal controls (Dagan et al., 1991). A possible explanation of this finding is that those with PTSD may experience a blocking process (i.e., deeper sleep and diminished senses) during sleep in order to limit the amount of external and internal stimuli present among this hyperaroused group (Dagan et al., 1991). Furthermore, it is possible individuals experiencing chronic sleep problems will move to stage 4 sleep (deep sleep) more quickly and stay there longer when they do fall asleep. This research demonstrates PTSD is related to the development of sleep problems. However, limited retrospective work has examined the relation between sleep problems and the maintenance of posttraumatic stress symptoms. One study in this domain has suggested that experiencing bad dreams and poor sleep quality prior to a traumatic event increased vulnerability for the development of psychiatric morbidity, including PTSD and MDD after the traumatic event (Mellman et al., 1995). Further research is needed to assess the relation between sleep problems and the development of PTSD post-traumatic event exposure.

Common limitations of the studies reviewed above, in addition to the specific limitations noted within the review of each individual study, warrant consideration. First, relatively small sample sizes in these studies highlights that results must be considered cautiously as this may lead to unstable data patterns. Second, the majority of studies sampled survivors of the Holocaust and combat veterans. Due to the focus on these specific groups, generalizability of these findings may be limited. Future research would benefit from the inclusion of different types of traumatic

events that may affect sleep in different ways (e.g., natural disasters versus sexual assaults occurring in a bedroom). Third, the use of retrospective accounts of traumatic event exposure and sleep problems may be subject to memory biases, particularly among persons suffering from PTSD. Fourth, methodological inconsistencies in participants' substance use prior to polysomnographic examinations (e.g., screening for versus inclusion of) may affect results. Finally, studies defined PTSD based on different iterations of the DSM. For this reason, variability in diagnostic criteria are present across studies. Based upon these limitations, it is important to examine relations between sleep and both traumatic event exposure and PTSD using prospective longitudinal tests. We now turn our attention to a review of these studies.

Prospective Studies

In contrast to the section above, no prospective studies of the effects of traumatic event exposure alone on sleep problems were identified. Thus, studies of the effects of PTSD, relative to traumatic event exposure alone, will now be reviewed. Please see Table 1 for details regarding methods and results of these studies. Presented below are five studies. The first reviews a study on the effects of traumatic event exposure and sleep problems predicting the development of PTSD. The following two present data on the development of sleep problems after recent traumatic event exposure. This is followed by two studies that focus on factors related to sleep that may impact PTSD.

Klein, Koren, Arnon, and Lavie (2002) explored the development of sleep problems after relatively recent traumatic event exposure. Sleep problems were monitored among traffic accident victims with PTSD for up to one year after the traumatic event via polysomnography. Participants included individuals with PTSD (n = 8) as a result of a car accident and a comparison group of individuals without PTSD (n = 6), but who experienced injury due to a car accident. An initial interview was conducted within the first week of hospitalization. Individuals completed follow-up interviews 1, 3, 6, and 12 months after the accident. During the 12-month follow-up assessment, a structured clinical interview was administered to diagnose current PTSD. Self-report and actigraphy measures of sleep were obtained during every follow-up interview. Three consecutive nights of polysomnography were conducted during the 12-month follow-up session. Results demonstrated no significant differences in self-reported or polysomnographic assessments of sleep between those with and without PTSD. Overall, results suggested only marginal differences in self-reported sleep problems between those with and without PTSD. Importantly, the particularly small sample size may have interfered with detection of group differences in sleep problems.

In another analysis from this research team, with a larger sample size, Koren, Arnon, Lavie, and Klein (2002) prospectively examined self-reported insomnia symptoms among 102 motor vehicle accident victims across 1-year. A group of 19 demographically matched individuals who had experienced elective surgeries was included as a comparison condition. All participants were free of psychiatric and sleep disorders at the time of the accident. Repeated assessment for PTSD and sleep problems was conducted 1 week and 1, 3, 6, and 12 months after the accident. Results indicated of the original 102 accident victims, 26 developed PTSD by the 12 month follow-up. In comparison none of the control participants met diagnostic criteria for PTSD throughout the study. Furthermore, results indicated the presence of insomnia symptoms from the 1 month assessment and subsequent assessments significantly predicted the development of PTSD at the 12 month follow-up. These data suggest that sleep problems occurring shortly after a traumatic event are related to an increased likelihood of developing PTSD. Importantly, the fact that sleep problems were measured post-traumatic event precludes conclusions regarding causality. It is possible that more severe symptomatic reactions to the traumatic event resulted in both sleep problems and an increased likelihood of developing PTSD. Furthermore, the study did not take into consideration physical pain as a result of the

accident. Future research may benefit from assessing physical pain, as this could be a confound affecting sleep among survivors of motor vehicle accidents.

Mellman, Pigeon, Nowell, and Nolan (2007) conducted a study investigating the role of REM sleep and PTSD symptoms after recent traumatic event exposure. Participants included 38 individuals recruited from level I trauma centers. Individuals were excluded if they had been diagnosed with an axis I disorder (including PTSD), were taking psychotropic medications within 1 month prior to the traumatic event exposure, or had physical pain that interfered with sleep. Participants completed a polysomnographic assessment in the weeks immediately following the traumatic event exposure. Severity of PTSD symptoms and diagnostic status were determined via the CAPS administered two months post-traumatic event. Results indicated at the 2-month follow-up: 10 individuals met criteria for PTSD, 6 met criteria for subthreshold PTSD (i.e., met criteria for 2 of the 3 diagnostic clusters), and 22 did not meet criteria for PTSD. Results indicated subjective insomnia ratings and nightmares were greatest among the PTSD group (compared to the sub-threshold and control groups). Furthermore, REM duration was negatively correlated with severity of PTSD symptoms and subjective insomnia ratings. This is to say, as REM density decreased, PTSD symptom severity and subjective insomnia increased.

Mellman, Bustamante, Fins, Pigeon, and Nolan (2002) conducted a study on factors that may affect the early development of PTSD in relation to sleep problems. Participants in this study included 21 individuals that were admitted to a hospital due to life-threatening injuries and either had, or had not, developed PTSD. Overall, 10 of the 21 individuals developed symptoms of PTSD, while 11 were asymptomatic. A comparison group of 10 healthy non-injured people also participated. All participants were free from psychiatric diagnoses for at least six months prior to entering the hospital and reported physical pain was not interfering with their sleep. Participants completed assessments of PTSD symptoms and sleep one month after the traumatic event and again six months post-traumatic event. Comparisons of sleep measures were made among the three groups (i.e., PTSD, traumatic event exposure without PTSD, and no-traumatic event history). The injured group without PTSD at one-month post-traumatic event demonstrated more disruption during sleep, including awakenings and increased general activity during sleep, compared to those with PTSD and non-injured controls. However, those with PTSD evidenced greater number of REM cycles even though each cycle was significantly shorter in time compared to all other groups. During the follow-up session, sleep duration and maintenance were similar among the injured group and those with PTSD compared to controls. Interestingly, a subset of injured individuals developed symptoms of PTSD by the six month follow-up. This group was marked by significantly more fragmented REM sleep in comparison to all other groups. These findings, although potentially limited by the small sample, suggest traumatic event exposure predicts poor sleep quality, even as compared to a group with PTSD. Furthermore, elevated sleep fragmentation during REM sleep among traumatic event exposed participants was related to the development of PTSD. Importantly, these findings were determined after methodologically controlling for the effects of physical pain on sleep. The methodological rigor evidenced in this study increases confidence in the overall results and conclusions drawn.

Breslau and colleagues (2004) examined the relation between sleep and PTSD within a subset (n = 292) of a larger (n = 913) longitudinal project that sampled young adult community residents in Michigan who were followed for 10 years. All participants were free of substance abuse and medications, had been exposed to a traumatic event between 1994 and 1999, and did not meet criteria for PTSD at the initial assessment. Within this larger group, approximately 25% developed PTSD during the follow-up interval; the remaining participants did not develop PTSD, allowing for a comparison of these two subsamples in terms of sleep problems. Two consecutive nights of polysomnography and multiple sleep latency tests were conducted.

Results showed 87% of individuals with PTSD reported sleep problems. However, polysomnigraphic assessment of sleep showed no differences in sleep initiation or maintenance between those with and without PTSD. Furthermore, no differences in objective sleep measures were found when comparing those with past PTSD to those with current PTSD. Comparisons indicated those exposed to a traumatic event without PTSD had significantly lower rates of arousal during REM compared to those with PTSD. These conclusions should be considered given several strengths and potential limitations of the current study. First, this study was the first longitudinal, epidemiological study which implemented polysomnography assessment of sleep. Due to the longitudinal nature of the design, traumatic event exposure was measured throughout the study on all groups, which allowed for examination of multiple traumatic event exposures prior to, and during, the study. Potential limitations include participant attrition throughout the study, as well as potential for false positive diagnoses of PTSD. However, the strengths of this study increase the confidence in the conclusions drawn.

Summary

Relatively few prospective tests of linkages among sleep problems, traumatic event exposure, and PTSD have been conducted. The limited research allows for multiple tentative conclusions. First, traumatic event exposure alone, relative to the presence of PTSD and no-traumatic event exposure, predicts increased sleep problems, including greater frequency of awakenings and lower rates of arousal during REM sleep (Mellman, Bustamante, Fins et al., 2002). Second, REM fragmentation one month post-traumatic event appears positively correlated with the development of PTSD symptoms by six months post-traumatic event (Mellman, Bustamante, Fins et al., 2002), and decreased REM duration is negatively correlated with PTSD symptom severity (Mellman et al., 2007). Together, it appears that PTSD may result in self-reported sleep problems, while evidence resulting from objective observations of sleep problems is mixed (Breslau et al., 2004; Klein et al., 2002).

There are several noteworthy gaps in this area. To the best of our knowledge, no study has directly tested the role of sleep problems prospectively predicting subsequent traumatic event exposure. Research in this domain is necessary in order to better conceptualize the interplay between sleep problems and traumatic event exposure. Also, as noted above, the majority of retrospective work has been conducted with combat and holocaust-related samples, whereas the prospective research has been conducted with people reporting injury, motor vehicle accidents, and a range of traumatic events within the community. Thus, discrepancies between these methodologically distinct studies (e.g., differences in objective measures of sleep problems) could be due to factors inherent to the methods (e.g., reliance on retrospective report) or due to characteristics of the traumatic event type. Future work examining the generalizability of findings across traumatic event types would help to address this issue.

Integrative Synthesis and Future Directions

Results of the reviewed studies coalesce to suggest complex bi-directional relations among sleep problems, traumatic event exposure, and PTSD. In relation to traumatic event exposure, prospective evidence suggests traumatic event exposure is related to the development of sleep problems (Hefez et al., 1987; Kaminer et al., 1991; Mellman et al., 1995; Mellman, Bustamante, Fins et al., 2002; Neylan et al., 1998; Rosen et al., 1991), although no research has examined the likelihood of traumatic event exposure secondary to sleep problems. Therefore, research is needed that examines the relation between sleep problems and subsequent traumatic event exposure. For instance, evidence suggests 24 hours of sleep deprivation increases risky and impulsive behaviors among women (Acheson, Richards, & deWit, 2007). Therefore, a prospective test of the role of sleep problems in potentiating exposure to potentially traumatic events would be informative in understanding temporal patterning between these factors. Importantly evidence combines to suggest the relation between sleep problems and traumatic

event exposure is not fully mediated by psychopathology, including PTSD (Mellman et al., 1995; Noll et al., 2006). Thus, there appears to be something specific about the experience of a traumatic event that affects sleep. However, future research may investigate other potential third variables that may affect this relation. For example, isolated sleep paralysis (temporary immobility that may occur during sleep onset or waking) has been shown to be related to sleep problems and PTSD (Ohayon & Shapiro, 2000; Ramsawh, Raffa, White, & Barlow, 2008). In addition, demographic characteristics including ethnicity, socioeconomic status, and gender may be considered in future research to better understand the role of these factors on the relation between sleep problems and traumatic event exposure/PTSD. While undoubtedly important to consider in future work, it is noteworthy that sleep problems appear associated with PTSD above and beyond the variability accounted for by these factors (Babson et al., 2008).

Two general areas of research are needed to advance understanding of why traumatic event exposure increases the likelihood of developing sleep problems. First, it is important to consider the possible role of characteristics of traumatic event exposure. For example, time since the traumatic event (Dagan et al., 1991; Hefez et al., 1987; Lavie et al., 1979), type of traumatic event exposure (Klein et al., 2002; Mellman et al., 1995; Mellman, Bustamante, Fins et al., 2002), and number of traumatic event exposures (Rosen et al., 1991) may differentially affect sleep. However, direct prospective tests of the relation between specific traumatic event types and the development of sleep problems is needed. For instance, in light of research linking chronic pain to sleep problems (Drewes, Nielsen, Arendt-Nielsen, Birket-Smith, & Hansen, 1997; Lavigne et al., 2000), traumatic event types that are perhaps more commonly linked to chronic pain than others [e.g., combat exposure and motor vehicle accidents; Beckham et al. (1997) and Bryant, Marosszeky, Crooks, Baguley, and Gurka (1999), respectively] may be more likely to result in subsequent sleep problems and changes in sleep architecture. Second, prospective investigation of the relations among sleep problems (particularly objectively measured sleep disturbance), traumatic event exposure, and PTSD among children is needed. The importance of extending this literature to youth is evidenced by the high rates of traumatic event exposure (Copeland et al., 2007) and sleep problems (Carskadon, 1990) within this relatively early developmental period. The bulk of research conducted to date in this area has examined adulthood traumatic event exposure. Relatively few studies have examined the effects of childhood traumatic event exposure on subsequent objectively-measured sleep problems. Indeed, the one study reviewed that addressed the effects of childhood traumatic event exposure on adults (Noll et al., 2006) did not include objective measures of sleep problems. Additional research in this area would allow for important insights into developmental factors (e.g., puberty, which has been linked to alterations in sleep; Carskadon et al., 2002) that affect the degree to which traumatic event exposure impacts sleep. For example, future prospective research could conduct repeated laboratory assessments of sleep from childhood through adulthood among youth exposed to a traumatic event during childhood.

The literature regarding PTSD (cf., traumatic event exposure) and sleep yields two main conclusions: (1) people with PTSD self-report subsequent elevations in sleep problems up to 45 years after the traumatic event and (2) direct monitoring of sleep among these samples currently does not clearly substantiate the self-reports. Laboratory assessments of sleep conducted 1 to 10 years after traumatic event exposure have not evidenced objective differences in sleep compared to controls (Breslau et al., 2004; Klein et al., 2002). One important methodological confound apparent in the current review that may account for this apparent inconsistency is substance use. Substance use is common among people with PTSD (Blumenthal et al., 2008; Feldner et al., 2007; Kilpatrick et al., 2000; Sareen, Chartier, & Paulus, 2006) and sleep problems (Bootzin & Stevens, 2005). In fact, recent recommendations highlight the importance of considering substance use as a potential confound in research comparing people with versus without PTSD (Rasmusson, Picciotto, & Krishnan-Sarin, 2006). Numerous studies have excluded participants on the basis of current substance use

(Breslau et al., 2004; Dagan et al., 1991; Klein et al., 2002; Lavie et al., 1979; Mellman et al., 1995), while other designs do not include this exclusionary criterion (Hefez et al., 1987; Kaminer et al., 1991; Mellman, Bustamante, Fins et al., 2002). Studies that did not exclude for substance use have typically observed sleep problems using objective measures, whereas those excluding on the basis of substance use typically have not documented sleep problems using objective measures. Given evidence suggesting substance use interferes with sleep (Johanson, Roehrs, Schuh, & Warbasse, 1999; Roehrs, Papineau, Rosenthal, & Roth, 1999; Wetter & Young, 1994), it is possible that substance use (potentially secondary to the development of PTSD) may be related to objectively measured sleep problems. For example, preliminary cross-sectional evidence suggests nicotine dependence partially mediates the relation between sleep problems and PTSD (Babson et al., 2008). A recent meta-analytic review (Kobayashi et al., 2007) comparing polysomnography studies of individuals with and without PTSD has begun to elucidate these inconsistent findings. Results indicated PTSD patients did in fact demonstrate objective sleep disturbances specifically related to stage 1 sleep, slow wave sleep, and REM density compared to individuals without PTSD. Inconsistencies in these findings were attributed to moderating third variables (sex, age, comorbid disorders, and substance use disorders). For example, studies which controlled for comorbid depression or had a majority male sample, evidenced greater objective differences in sleep of individuals with compared to without PTSD. Combined with the current findings, this suggests inconsistencies in objective indicators of sleep problems may be due to methodological variability in the related literature. In addition to the two main conclusions noted above, there also is limited evidence suggesting that sleep problems may interfere with recovery from posttraumatic stress reactions (Mellman et al., 1995). Given the paucity of work in this domain, independent replication is now needed.

Additional research also is needed that examines underlying processes that may explain the patterns of results summarized herein. The current review indicates there has not been a direct prospective test of such processes. Although there currently is not a generally accepted explanation for how posttraumatic stress interferes with sleep, scholars have suggested multiple (non-mutually exclusive) avenues by which posttraumatic stress may maintain sleep problems. Suggestions include the following: (1) waking during the night may be negatively reinforced via avoidance of feared traumatic event-related cues in nightmares (Krakow et al., 2001); (2) sleep may be interrupted by post-traumatic event elevated arousal and restlessness during sleep (Mellman, 1997); (3) coping strategies for managing posttraumatic stress cannot be implemented during sleep (Woodward, 1995); (4) hypersensitivity to mid-sleep arousal among individuals with PTSD may result in more frequent awakenings (Dagan et al., 1991; Lavie et al., 1998); and (5) post-traumatic event exposure elevations in sensitivity of the noradrenergic system interfere with sleep (Pillar et al., 2000). Also, a recent review in this area hypothesized an important role of the amygdala and prefrontal cortex as neuroanatomical structures implicated in sleep disturbance that may partially mediate the relation between traumatic event exposure and the development of PTSD (Germain et al., 2008). Research that prospectively examines these hypothesized mechanisms of action is now needed.

In addition to consideration of why posttraumatic stress may interfere with sleep, scholars have considered why sleep may interfere with recovery from traumatic event exposure. Here, there are at least five considerations that warrant prospective examination. First, it has been hypothesized that disruptions in rapid eye movement (REM) sleep may interfere with the integration of new associations into traumatic event-related memory (e.g., safety-related learning; Krakow, Hollifield et al., 2001; Mellman, Bustamante, Fins et al., 2002; Pillar et al., 2000), thereby preventing emotional processing critical to recovery from a traumatic event (Brewin, Dalgleish, & Joseph, 1996). Second, it is possible that sleep problems impair daytime coping, resulting in increased avoidance of traumatic event-related cues, thereby preventing the extinction of learned fear of traumatic event-related cues (Rothbaum & Mellman, 2001). Third, pairing of fear experienced during a nightmare with cognitive cues of a traumatic event

present during the nightmare may further sensitize persons to traumatic event cues (Rothbaum & Mellman, 2001). Fourth, experimental studies have suggested sleep deprivation increases anxiety generally (Babson, Feldner, Trainor, & Smith, in press; Sagaspe, Sanchez-Ortuno, & Charles, 2006), which may maintain elevated posttraumatic stress symptoms by virtue of maintaining relatively elevated basal levels of anxiety. Furthermore, as discussed above, it also is possible that substance use developing subsequent to sleep problems after a traumatic event may interfere with healthy recovery. Indeed, studies suggest people with PTSD may use substances to self-medicate sleep problems (Nishith, Resick, & Mueser, 2001) and negative affect (Feldner, Babson, Zvolensky, Vujanovic et al., 2007; Kaysen et al., 2007) and prospective research is emerging suggesting substance use in the wake of traumatic event exposure may potentiate the development of PTSD (e.g., van der Velden, Kleber, & Koenen, 2008. Finally, the role of gender must be considered in the relation between sleep and PTSD. Evidence indicates women are more likely to have sleep problems (Soares, 2005) and develop PTSD (Breslau & Anthony, 2007). Therefore, gender may be an important moderator of this relation which needs further testing. Collectively, there are multiple possible (interactive) pathways that may explain, at least in part, how sleep problems may interfere with normal healthy recovery from exposure to a traumatic event. Prospective work that examines the role of each of these factors in isolation and relative to each other would significantly advance our understanding of the relations between PTSD and sleep problems across time.

Finally, the role that nightmares may play in the development of sleep problems among people with PTSD needs additional exploration. Nightmares are more frequent and prevalent in individuals with PTSD compared to traumatic event-exposed people without PTSD (Neylan et al., 1998). Furthermore, the presence of nightmares is correlated with sleep problems, such as sleep maintenance insomnia (Mellman et al., 1995). Therefore individuals may experience nightmares as the primary sleep complaint, but have structurally normative sleep patterns after sleep initiation occurs because movement during sleep is not objectively observed during nightmares due to REM-related paralysis (Neylan et al., 1998). It is possible that people with PTSD begin to fear (and perhaps even avoid) sleep due to nightmares. This may delay sleep onset resulting in sleep onset insomnia, thereby supporting individuals self-report of trouble sleeping. However, once sleep is initiated, maintenance may be relatively unaffected (Neylan et al., 1998). Thus, prospective research is needed to understand the role of nightmares in affecting sleep problems. For example, future studies may benefit from comparing the structure of sleep between individuals with PTSD and nightmares and those with PTSD without nightmares. This may help tease apart the role that nightmares play in the structural versus self-reported indices of sleep problems.

In addition to testing theoretically-derived mechanisms of action postulated in the above work via correlational analyses (e.g., prospective mediational tests), there is a need for additional experimental work in this area. For instance, analog designs that aim to experimentally test the effects of sleep deprivation on reactivity to traumatic event cues could shed light on how sleep problems may maintain traumatic event-related affective reactivity. While experimentally manipulating traumatic event exposure is unethical and unreasonable, experimental psychopathology methods could be used to examine if experimentally manipulated acute (e.g., 24 hour) sleep deprivation affects psychobiological reactivity to traumatic event cues presented in the safe and controlled environment of the laboratory. These types of designs would allow for experimentally manipulating multiple factors that may prove critical to advancing this area of research. For instance, degree of sleep deprivation (e.g., complete versus partial, REM disruption) and type of emotional reactivity (e.g., response to traumatic event cues, reactivity to bodily arousal, depressive mood inductions) could be studied in isolation or concurrently. These types of tests would advance research regarding why sleep deprivation may maintain posttraumatic stress symptoms. Moreover, combining this type of experimental psychopathology work with longitudinal designs may be particularly informative. For

example, examining the association between a laboratory assessment of emotional reactivity to traumatic event cues shortly after event exposure and responding to such a test 12 months later in terms of varying degrees of sleep problems may allow for novel and important insights into processes involved in the relation between posttraumatic stress symptoms and sleep problems.

Conclusion

As noted at the outset of this review, studies that speak to temporal patterning of sleep problems, traumatic event exposure, and PTSD were reviewed in detail to uniquely contribute to recent reviews focused on other aspects of this literature. In doing so, the current review specifically delineated extant findings and highlighted gaps in this work. Contemporary research in this area has begun to shift from conceptualizing sleep problems as secondary to PTSD toward thinking of sleep problems as central to PTSD. As noted by Spoormaker and Montgomery (2008) and evidenced in the current review, three main lines of evidence support the latter. First, the presence of sleep problems subsequent to traumatic event exposure has been linked to an increased likelihood of developing PTSD (Koren et al., 2002). This suggests sleep problems may function, in part, to maintain posttraumatic stress as opposed to only being secondary to the development of PTSD. Second, sleep disturbances are the most common residual symptom after successful treatment of PTSD (Zayfert & DeViva, 2004). Third, insomnia treatment reduces PTSD symptoms (Jacobs-Rebhun et al., 2000; Zayfert & DeViva, 2004). Treatment of a secondary symptom would not be expected to affect the more global disorder, thereby suggesting sleep may be a mechanism involved in the maintenance of PTSD symptoms. A methodological challenge to further advancing this area of inquiry given the apparent centrality of sleep problems to PTSD is isolating the effects of sleep problems relative to other aspects of PTSD. Given sleep problems are a symptom of PTSD (APA, 2000), researchers have often defined PTSD, in part, by the presence of sleep problems. This methodological approach places strict constraints on understanding causal relations between sleep problems and PTSD. While sleep problems are not necessary for a diagnosis of PTSD, disturbed sleep and traumatic event-related nightmares are symptoms of the disorder (hyperarousal and reexperiening, respectively) commonly endorsed by individuals with PTSD (Breslau, Roth, et al., 2007). In fact, some research has concluded that up to 88% of individuals with PTSD endorse clinically significant insomnia (score of 3 on frequency and 2 on intensity) on the CAPS (Zayfert & DeViva, 2004). Thus, many efforts to understand how sleep problems and PTSD relate are confounded by the overlap in these constructs. Nonetheless, increasingly sophisticated methods for decreasing such overlap are being developed. The Pittsburg Sleep Quality Index-Addendum for PTSD (PSQI-A; Germain, Hall, Krakow, Shear, & Buysse, 2005) is a self-report measure that measures the frequency of seven disruptive nocturnal behaviors common among individuals with PTSD. These seven behaviors include: hot flashes, nervousness, nightmares of the traumatic event, anxiety/panic not related to the traumatic event, bad dreams not related to the traumatic event, episodes of terror, and acting out dreams. Further methodological advances in this area are now needed. For instance, psychophysiological procedures have been developed to identify people with PTSD (Orr, Metzger, Miller, & Kaloupek, 2004) that would allow researchers to examine the relation between PTSD and sleep problems without relying on diagnostic definitions of PTSD that include sleep problems.

Considering how the work reviewed herein relates to other recently reviewed bodies of work in this general domain also yields several potentially fruitful directions for future work. For instance, treatment outcome work focused on this comorbidity [see Maher, Rego, and Asnis, (2006) for a review] may be particularly informative for further narrowing in on possible mechanisms involved in the maintenance of comorbid PTSD and sleep problems. Here, some psychopharmacological interventions (e.g., Selective Serotonin Reuptake Inhibitors) that

target neuroanatomical structures implicated in both PTSD and sleep problems (e.g., Hypothalamic-Pituitary-Adrenal Axis; Drake et al., 2003) have resulted in reductions in both PTSD symptoms and sleep problems (e.g., Neylan et al., 2001). In contrast, other pharmacological interventions focused on PTSD symptom reduction (e.g., Monoamine Oxidase Inhibitors, Benzodiazapines, Tricyclic antidepressants) have been shown to increase sleep problems (Maher et al., 2006) and efficacious psychosocial treatments for PTSD do not appear to affect sleep problems (Zayfert & DeViva, 2004). This interesting pattern points to potentially specific neuroanatomical functioning (e.g., Hypothalamic-Pituitary-Adrenal Axis) that, in addition to postulations regarding the role of such functioning in the etiology of PTSD (Germain et al., 2008), may be involved in the maintenance of this comorbidity. However, the current review demonstrates that prospective work in this area is needed to more confidently draw such conclusions. Moreover, these data suggest that PTSD may not be necessary for the maintenance of sleep problems once sleep problem – PTSD comorbidity develops, highlighting that continued development in the area of psychosocial treatments for PTSD that concurrently target sleep problems may prove beneficial. The need for this type of treatment development is further emphasized by iatrogenic effects observed in previous work in the area. Targeting improvements in sleep quality among recently traumatic event exposed individuals via benzodiazepine administration suggested such an approach may increase the likelihood of developing PTSD (Mellman, Bustamante, David et al., 2002; see also Gelpin, Bonne, Peri, & Brandes, 1996). These findings highlight that approaches aiming to prevent either sleep problems or PTSD via early intervention require great caution and advancement of prospective research in the area testing hypothesized mechanisms of action would aid in this process.

In conclusion, evidence pertaining to the patterning of sleep problems and traumatic event-related factors suggests (1) exposure to a traumatic event can interfere with sleep, (2) PTSD is related to the development of self-reported sleep problems, but evidence is less clear regarding objective indices of sleep, and (3) limited evidence suggests sleep problems may interfere with recovery from elevated posttraumatic stress levels. However, this review has highlighted a clear need for additional work in the area prior to drawing firm conclusions regarding temporal patterning among these factors.

Acknowledgments

This project was supported, in part, by a National Research Service Award (F31 MH081402) awarded to Kimberly Babson and a Center for Disease Control and Prevention contract with (U49 CE001248), and a Department of Defense grant (PT073945) award to, Dr. Feldner.

The authors would also like to thank Dr. Ellen W. Leen-Feldner for her helpful comments on drafts of this paper.

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Table 1

Overview of methods and results from studies (listed alphabetically) of the relations between traumatic event exposure, posttraumatic stress disorder, and sleep problems that speak to temporal patterning

AUTHOR(S) / DESIGN	SAMPLE	Primary Variables and Measures				Results
		Sleep		Trauma/PTSD		
		Variable	Measure	Variable	Measure	
Breslau et al., 2004 Prospective (10 years)	Subset of a longitudinal HMO study in Michigan. Participants age 21-30 years (M_{age} = 37.6, SD = 1.70) with (n = 71) and without (n =212) PTSD. Total sample = 283 (Females = 66).	• Objective sleep measures • Daytime sleepiness	• 2 consecutive nights of polysomnography • Multiple sleep latency test	• PTSD	• Diagnostic Interview Schedule for DSM-III-R • Clinician Administered PTSD Scale	One way ANOVAS indicated: • No objective differences between those with and without PTSD (p > .05). All d's < .5 • Those with PTSD had greater arousals from REM compared to those with PTSD (F = 5.92; p <01; d = .33
Dagan et al., 1991 Retrospective	24 Survivors of the Lebanon War (M_{age} = 31.90, SD = 4.55) with PTSD and 6 healthy controls (M_{age} = 32.17, SD = 6.60). All participants were men.	• Objective sleep measures	• 3 consecutive nights of polysomnography	• PTSD	• DSM-III defined. Specific measure not stated	• One way ANOVA showed Increases in awakening thresholds in those with PTSD compared to those without PTSD (d = 1.09).
Engdahl et al., 2000 Retrospective	59 male combat veterans with (n = 30; M_{age} = 71.5, SD = 4.2) and without (n = 29; M_{age} = 71.0, SD = 4.2) PTSD. All participants were free from sleep affecting medications and comorbid	• Objective sleep measures • Daytime sleepiness	• 3 consecutive nights of polysomnograhy. • Actigraphy monitor for 3 nights and 2 days while in the lab and for 12 nights and days at home prior to the study.	• PTSD • Combat exposure	• Structured Clinical Interview for DSM-III-R (SCID) PTSD module. • Structured Clinical	Correlations indicated: • REM percentage was associated with PTSD severity (r = .28, p<.05). MANOVAs indicated:

AUTHOR(S) / DESIGN	SAMPLE	Primary Variables and Measures				Results
		Sleep		Trauma/PTSD		
		Variable	Measure	Variable	Measure	
	disorders. The study was conducted 28-50 years post trauma.		• Multiple sleep latency test (20 minute intervals for 2 hours).		Interview for DSM-III-R, Non-Patient Edition. • Combat Exposure Scale	• No significant differences in objective measures of sleep between those with and without PTSD [f (17,28) = 1.36, p >.05, d = .27]. • Those with PTSD reported more awakenings [f (1,58) = 6.6, p < .05, d = .59], and more restless sleep [f (1,58) = 8.1, d = .65, p < .01] compared to those without PTSD.
Hefez et al., 1987 Retrospective	11 survivors of trauma including Holocaust (n = 5; age = 45-68 years), combat veterans (n = 4; age = 33, 35, 44 years), and sea disaster victims (n = 2; age = 20 and 25 years). 9 age matched controls were also included.	• Objective sleep measures	• 2-5 consecutive night of polysomnography	• PTSD	• Not Defined	• Compared to controls all participants had prolonged sleep latency, greater movements, and more frequent awakenings[a].
Kaminer et al., 1991 Retrospective	• 33 total participants including 23 Holocaust survivors including 12 well adjusted (M_{age} = 62.7, SD = 4.4), and 11 poorly adjusted	• Objective sleep measures	• 4 consecutive nights of polysomnography	• Trauma • PTSD	• Impact event scale • Posttraumatic stress disorder questionnaire	A series of MANOVAs yielded: • Less well adjusted survivors had increased sleep latency (M = 33, SD = 25.4) compared to well adjusted (M = 20, SD = 7.6) survivors and controls

AUTHOR(S) / DESIGN	SAMPLE	Primary Variables and Measures				Results
		Sleep		Trauma/PTSD		
		Variable	Measure	Variable	Measure	
	(M_{age} = 57.5, *SD* = 5.7) survivors, and 10 control participants (M_{age} = 61.1, *SD* = 5.4).					(M = 12.3, *SD* = 6.8)[a].
Klein et al., 2002 Prospective (1 year)	14 traffic accident victims, with PTSD (n = 8; M_{age} = 22.1, *SD* = 2.0) and without PTSD (n = 6; M_{age} = 22.0, *SD* = 2.5). All participants were hospitalized for injuries. Participants were assessed 1 week post trauma then again 1,3,6, and 12 months post trauma.	• Subjective sleep measures • Objective sleep measure	• Mini-sleep questionnaire • 48 hours of Actigraphy during each follow up • 3 consecutive nights of polysomnography done at 12 months post trauma	• PTSD	• Structured clinical interview for DSM-III-R (SCID) administered at 12 months post trauma.	T-tests and chi-squared analyses were conducted. • There were no differences in subjective sleep problems between those with (M = 15, *SD* = 5.3) and without (M = 13.7, *SD* = 7.2) PTSD (t = .4, p > .05; d = .23). • Objective measures of sleep showed a trend for lower awakening thresholds in those with PTSD (M = 73.5, *SD* = 11.2) compared to those without (M = 80.5, *SD* = 12.7; d = .64).
Koren et al., 2002 Prospective (1 year)	102 motor vehicle accident victims (age range 18-65 years) and 19 demographically matched control participants (elective surgery patients). All participants were free from psychiatric and	• Subjective sleep measures	• Mini-sleep questionnaire.	• PTSD	• Structured clinical interview for DSM-III-R (SCID) administered at 12 months post trauma.	Logistic Regressions • Sleep disturbances starting from 1-month (X^2= 15.73, p < .001), 3-months (X^2= 20.44, p < .001), and 6-

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AUTHOR(S) / DESIGN	SAMPLE	Primary Variables and Measures				Results
		Sleep		Trauma/PTSD		
		Variable	Measure	Variable	Measure	
	sleep disorders upon initiation of the study.					months (X^2= 25.10, p < .001) post-trauma predicted the development of PTSD 12 months post-trauma.
Lavie et al., 1979 Retrospective (2-2.5 years)	11 combat veterans from the Yom Kippur War age 24-33 years (M_{age} = 26.54, SD = 3.35) referred to a sleep laboratory 2-2.5 years post war for emotional and/or sleep problems. Male control participants (n = 9) aged 24 to 27 years who actively participated in the Yom Kippur War.	• Objective sleep measures • Subjective sleep measures	• 3-4 consecutive nights of polysomnography. • All participants self reported no sleep problems prior to combat exposure.	• PTSD	• Psychiatric interviews. Specific interview was not provided.	Two tailed t-tests were conducted • Those with PTSD had significantly longer REM latency (M = 99.9 SD = 34.5 versus M = 68.6, SD = 15.2; p <.05; d = 1.17); shorter total sleep time (M = 345.4, SD = 50.8 versus M = 377.4, SD = 23.6; p <.05; d = .80), and shorter sleep efficiency (M = 84.9, SD = 9.5 versus M = 91.1, SD = 3.2; p <.05; d = .87).
Mellman, Bustamante, Fins et al., 2002 Prospective (6 weeks)	21 Victims of traumatic injuries presenting to the hospital (M_{age} = 35.4, SD = 9.5) and 10 healthy non-injured participants M_{age} = 35.4, SD = 12.0).	• Objective sleep measures	• 2 consecutive nights of polysomnography	• PTSD	• Structured Clinical Interview for DSM-IV (SCID). • Clinician Administered PTSD Scale (CAPS).	ANOVAs were conducted with post hoc significant least-square tests. • Those with PTSD had more wake time during sleep compared to controls [f (2,27) = 4.32, p < .05; d = .83]. • Those with PTSD had

AUTHOR(S) / DESIGN	SAMPLE	Primary Variables and Measures				Results
		Sleep		Trauma/PTSD		
		Variable	Measure	Variable	Measure	
						greater number of REM periods [$f(2,27) = 3.89$, $p < .05$; $d = .78$] and greater REM duration [$f(2,27) = 4.22$, $p < .05$; $d = .82$] in comparison to those trauma exposed without PTSD.
Mellman et al., 1995 Prospective (1 year)	54 adults involved in Hurricane Andrew (42 women; $M_{age} = 40.4$, $SD = 12.3$) without psychiatric conditions prior to the hurricane. Assessments were conducted 6 and 12 months post trauma.	• Objective sleep measures • Subjective sleep measures	• 10 participants with PTSD completed 2 consecutive nights of polysomnography • The Pittsburg Sleep Quality Index	• Trauma severity • PTSD	• Self report measure developed for this study • SCL-90 • Structured Clinical Interview for DSM-III-R Non-Patient Version	A series of ANOVAs were conducted • PTSD predicted sleep problems including global sleep problems, sleep quality, sleep disturbance, and greater awakenings compared to those without PTSD. • Those with PTSD also had greater sleep disturbance prior to the hurricane compared to those without PTSD in relation to overall sleep disturbance ($t = 2.3$, $p < .05$; $d = 1.66$), bad dreams ($t = 3.6$, $p < .05$; $d = 2.6$), and awakenings ($t =$

AUTHOR(S) / DESIGN	SAMPLE	Primary Variables and Measures				Results
		Sleep		Trauma/PTSD		
		Variable	Measure	Variable	Measure	
						2.3, p <.05; d = 1.66)
Mellman, Pigeon, et al., 2007 Prospective (1 Year)	160 adults recruited from Level 1 trauma centers participated in descriptive portion. 35 of these participants completed a sleep assessment; 16 with PTSD and 7 without PTSD.	• Objective sleep measures • Subjective sleep measures	• Single night of polysomnography • Self-report	• PTSD	• CAPS; SCID	Correlations demonstrated: REM duration was negatively correlated with subjective insomnia and early symptoms of PTSD (r = -.*38*, r = -.36, respectively).
Noll et al., 2006 Prospective (10 Years)	78 females who were sexually abused as children were compared with 69 non abused healthy females (M_{age} = 20.41, SD = 3.38).	• Subjective sleep measures	• 6 self-report questions assessing typical sleep patterns over the past 2 years. 4 items were drawn from the Child Behavior Checklist (CBCL). 2 additional items were drawn from the Brief Symptoms Inventory	• Revicti mization • PTSD • Sexual abuse info	• Comprehensive Trauma Interview • Modified PTSD symptom scale • Caseworker abuse history questionnaire	Hierarchical Linear Regressions demonstrated: • Experiencing abuse accounted for 5% of unique variance predicting sleep disturbance above an beyond that of depression and PTSD. • Sleep disturbances (f = 6.06, p <.05; d = .41) and arousal symptoms (f = 6.65, p <.05; d = .43) predicted revictimization. MANOVAs demonstrated: • Abused women had greater sleep disturbances (M = .22, SD = 1.06) compared to healthy control women

AUTHOR(S) / DESIGN	SAMPLE	Primary Variables and Measures				Results
		Sleep		Trauma/PTSD		
		Variable	Measure	Variable	Measure	
						(*M* = -.24, *SD* = .90; *d* = .46).
Rosen et al., 1991 Retrospective	Sleep measures were mailed to 166 Holocaust survivors affiliated with the Holocaust Center in Pittsburg. 42 people responded (M_{age} = 67.3, *SD* = 6.3; 14 females). In addition, elderly depressed individuals (*n* = 37; M_{age} = 70.4, *SD* = 6.2; 30 females) and healthy comparisons (*n* = 54; M_{age} = 71.2, *SD* = 4.7; 25 females) were recruited.	• Subjective sleep measures	• Pittsburg Sleep Quality Index (PSQI)	• PTSD	• Schedule for Affective Disorders and Schizophrenia-Lifetime Version	Correlations demonstrated: • A significant correlation between the amount of time spent in concentration camps and global scores of the PSQI (*r* = .39, *p* <.05). ANOVAs demonstrated: • Survivors experienced greater global sleep problems in comparison to healthy controls [*f* (2,117) = 61.69, *p* <.05; *d* = 1.63).

[a] effect size could not be calculated due to a lack of information;

[d] Cohen's d measure of effect size;

PTSD = Posttraumatic Stress Disorder, DSM = Diagnostic and Statistical Manual (APA, 1980, 1987, 1994). **PTSD and Trauma Measures:** SADS = Schedule for Affective Disorders and Schizophrenia (SADS; Endicott et al., 1978), DIS = Diagnostic Interview Schedule (DIS; Robins et al., 1981), SCID = Structured Clinical Interview (Spitzer & Williams, 1989), CAPS = Clinician Administered PTSD scale (Blake et al., 1995), CES = Combat Exposure Scale (Keane et al., 1989), PCL-C = PTSD Checklist Civilian Version (Weathers & Ford, 1996), Mississippi Scale = The Mississippi Scale for Combat-Related PTSD (Keane et al., 1988), Impact Event Scale, Comprehensive Trauma Interview (CTI; Noll et al., 2003), PTSD Symptom Scale (Davidson et al., 1989), Caseworker Abuse History Questionnaire (CAHQ; Trickett et al., 2001), SCL-90 (Derogatis 1977). **Sleep Measures:** Mini-Sleep Questionnaire (Zomer et al., 1985), Child Behavior Checklist (CBCL; Achenbach, 1991), Brief Symptom Interview (Derogatis & Spencer, 1982), Pittsburg Sleep Quality Index (PSQI; Buysse et al., 1989).