Sleep and posttraumatic stress disorder: a review

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Received 30 September 2002; received in revised form 19 December 2002; accepted 10 January 2003

Abstract

Research seeking to establish the relationship between sleep and posttraumatic stress disorder (PTSD) is in its infancy. An empirically supported theory of the relationship is yet to emerge. The aims of the present paper are threefold: to summarise the literature on the prevalence and treatment of sleep disturbance characteristic of acute stress disorder (ASD) and PTSD, to critically review this literature, and to draw together the disparate theoretical perspectives that have been proposed to account for the empirical findings. After a brief overview of normal human sleep, the literature specifying the relation between sleep disturbance and PTSD is summarized. This includes studies of the prevalence of sleep disturbance and nightmares, content of nightmares, abnormalities in rapid eye movement (REM) sleep, arousal threshold during sleep, body movement during sleep, and breathing-related sleep disorders. In addition, studies of the treatment of sleep disturbance in individuals with PTSD are reviewed. We conclude that the role of sleep in PTSD is complex, but that it is an important area for further elucidating the nature and treatment of PTSD. Areas for future research are specified. In particular, a priority is to improve the methodology of the research conducted.

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Keywords: Sleep; Posttraumatic stress disorder; Insomnia; Trauma; Treatment

1. Introduction

There is no doubt that sleep is severely disrupted following exposure to a traumatic event. To illustrate, Kato, Asukai, Miyake, Minakawa, and Nishiyama (1996) reported a survey of 143
people who were interviewed 3 and 8 weeks after the 1995 Hanshin earthquake in Japan. Sleep disturbance was the most common symptom reported (63% reported sleep disturbance at 3 weeks posttrauma, 46% at 8 weeks). Similarly, Goldstein, van Kammen, Shelly, Miller, and van Kammen (1987), in their study of Japanese prisoners of war during World War II, and Kuch and Cox (1992), in their study of survivors of the Holocaust, found sleep disturbance and nightmares to be the most common symptoms reported. Sleep disturbance was reported by 97% of prisoners-of-war camp survivors and 95% of Holocaust survivors. Nightmares were reported by 94% of prisoners-of-war camp survivors and 83% of Holocaust survivors. Moving to more recent times, North et al. (1999) found that 70% of the survivors of the Oklahoma City bombing suffered sleep disturbance 6 months after the event, and more than 50% had nightmares. Using random digit dialling in the United States within 1 week of the September 11 terrorist attack, Schuster et al. (2001) questioned 560 adults about their own reaction and the reaction of their children (n = 170). A total of 11% of adults and 10% of children indicated that they had experienced significant difficulty falling asleep or staying asleep since the attack. Taking these results together, the message is clear; sleep disturbance and nightmares are part of a normal and typical response to trauma (Pillar, Malhotra, & Lavie, 2000). However, while for most people, the sleep disturbance following trauma is transient (Lavie, 2001), for others, the sleep disturbance becomes an enduring problem, typically occurring as part of the acute stress disorder (ASD) and posttraumatic stress disorder (PTSD) diagnoses. Indeed, the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) lists nightmares within the symptom cluster describing reexperiencing symptoms (Cluster B in PTSD, Cluster C in ASD) and difficulty getting to sleep and staying asleep within the symptom cluster describing arousal symptoms (Cluster D in PTSD, Cluster E in ASD).

The aims of the present paper are threefold: to summarise the literature on the prevalence and treatment of sleep disturbance characteristic of ASD and PTSD, to critically review this literature, and to draw together the disparate theoretical perspectives that have been proposed to account for the empirical findings. As an understanding of the nature, measurement, and function of normal sleep is important for realising the implications of disordered sleep, we begin with a brief overview of human sleep. We then move on to characterise the sleep of patients with PTSD. In addition, abnormalities relating to rapid eye movement (REM) sleep, arousal threshold during sleep, body movement during sleep, and breathing-related sleep disorders will be reviewed. The methodology of the research reviewed is then summarised (in Tables 1 and 2) and critically evaluated. Finally, before highlighting future directions for research, we review advances in the treatment of sleep disturbance following trauma. It should be noted at the outset that in some past research, implications for PTSD have been drawn from ‘likely’ traumatised populations. In this paper, only those studies that are specifically based on patients with PTSD will be discussed.

2. Overview of human sleep

Human sleep can be divided into non-rapid eye movement (or ‘NREM’) and REM sleep. Normal sleep follows an organised pattern of NREM–REM cycles, each full cycle taking
between 70 and 120 min. NREM sleep can be subdivided into four stages (Stages 1, 2, 3, and 4) through which sleep progressively deepens (Hirshkowitz, Moore, & Minhoto, 1997; Shneerson, 2000). Polysomnography (PSG) is used to classify sleep into these stages. PSG involves placing surface electrodes on the scalp and face to measure electrical brain activity (electroencephalogram, EEG), eye movement (electro-oculogram, EOG), and muscle tone (electromyogram, EMG). In addition, electrodes can be attached to the legs to monitor for disorders such as periodic limb movement disorder and measures of airflow or breathing can be used to monitor for sleep-disordered breathing. The reason for highlighting these sleep disorders is that several studies, as will be reviewed below, have noted these diagnoses to be more common in individuals with PTSD.

Stages 3 and 4 are the deepest stages of sleep. Hence, when in Stage 3 or 4 sleep, it is most difficult to wake and be woken. REM sleep will be referred to frequently throughout this paper. It is characterised by rapid eye movements, as measured by two electrodes placed beside each eye (EOG). REM sleep has been referred to as ‘paradoxical sleep,’ as it is the stage when the brain is most active, but the body paralysed. It is relatively easy to wake and be woken from REM sleep. While some dreaming does occur in other stages of sleep, most dreams occur during REM sleep. Dreams are typically unavailable for conscious-recall unless they occur on waking (Hirshkowitz et al., 1997; Shneerson, 2000).

In a typical night’s sleep of a normal young adult, Stages 3 and 4 sleep are more prevalent in the first third of the night, with REM episodes increasing in length throughout the night. Accordingly, the proportion of REM sleep is much greater in the last third of the night. Normal sleep patterns change enormously across the life cycle. Average total sleep time declines from about 16–18 h per 24-h cycle in infancy to 10–11 h in childhood to 7–9 h in the mid-20s to 6–8 h in the middle years. Nocturnal sleep is further reduced in older adults, but there is an increase in daytime napping. Not surprisingly, the prevalence of insomnia in older adults is particularly high (Lichstein & Morin, 2000).

Sleep research and sleep medicine are still in their infancy. In fact, sleep researchers cannot agree on the most basic questions of ‘How much sleep do humans require?’ and ‘What is the function of sleep?’. Most agree that Stages 1 and 2 sleep are simply transitional stages between sleep and wake, so the controversy concerns the function of Stages 3 and 4 and REM sleep. Taking Stages 3 and 4 sleep first, Horne (1988) has argued that these stages constitute the ‘core’ sleep and that the other sleep obtained (Stages 1 and 2 and REM) are ‘optional.’ Another perspective is that Stages 3 and 4 sleep have a restorative function in that they promote tissue restoration (Adam & Oswald, 1977). Hypotheses about the function of REM include that it is involved in learning (Karni, Tanne, Rubenstein, Askenasy, & Sagi, 1994; Smith, 1995), memory consolidation (Dewan, 1968; Karni et al., 1994; Smith & Lapp, 1991), and may also be a mechanism for unlearning irrelevant information (Crick & Mitchison, 1983). Another possible function of REM is emotional processing. This hypothesis is derived from the observation that intense REM activity appears to accompany stressful life events. For example, in a study of recently divorced individuals, increased REM activity was associated with lower rates of depression 1 year later (Cartwright & Lloyd, 1994). Brain imaging studies showing amygdala activity during REM sleep (e.g., Maquet et al., 1996) are also consistent with the possibility that
REM sleep is important in the formation of emotional memories (e.g., Wagner, Gais, & Born, 2001).

The gold standard measure of sleep is PSG. The disadvantages of PSG are that it is expensive, requires skilled technical staff, and is associated with a ‘first-night effect’\(^1\) (Agnew, Webb, & Williams, 1966; Browman & Cartwright, 1980). Actigraphy is an alternative means of measuring sleep, based on the amount of movement present. The actigraph is a small, wrist-worn device that senses and stores information and gives an objective estimate of the sleep–wake cycle. Sleep estimates, based on actigraphy, are correlated highly with PSG (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992; Sadeh, Hauri, Kripke, & Lavie, 1995). The advantages of actigraphy over PSG is cost/time-efficiency and that it is not intrusive. The disadvantage of actigraphy is that it does not provide information about sleep stages or the presence of sleep disorders such as sleep-disordered breathing.

Subjective reports of sleep quantity and quality are important measures. Sleep diaries, filled out immediately on waking, have been shown to be highly correlated with objective estimates of sleep (Espie, Lindsay, & Espie, 1989; Wilson, Watson, & Currie, 1998). In addition, several questionnaire measures have been validated to index the presence of sleep disturbance (e.g., Pittsburgh Sleep Quality Index; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), insomnia (e.g., Insomnia Severity Index; Bastien, Vallieres, & Morin, 2001), and daytime sleepiness (e.g., Sleep–Wake Activity Inventory; Rosenthal, Roehrs, & Roth, 1993). However, it should be noted that none of these questionnaires have been specifically developed for or validated with PTSD samples.

Hence, a distinction can be drawn between sleep measured subjectively (via sleep diary, questionnaires) and sleep measured objectively (via PSG, actigraphy). The following section will review studies that have assessed individuals with PTSD for the presence of sleep disturbance and nightmares via subjective means. The discussion will then move on to review the data on sleep in patients with PTSD, as measured objectively.

### 3. Characterising sleep in PTSD: subjective reports

#### 3.1. Sleep-onset insomnia

The prevalence of difficulty getting to sleep, at the beginning of the night, has been reported in two studies. In Neylan et al.’s (1998) reanalysis of the National Vietnam Veterans Readjustment Study, 44% of veterans with PTSD rated themselves as having problems with sleep onset either ‘sometimes’ or ‘very frequently,’ compared to 6% of veterans without PTSD, 9% of era veterans\(^2\), and 5% of civilians. In Ohayon and Shapiro’s (2000) study, based a large sample recruited from the community, participants with PTSD had more difficulties initiating sleep (41%) relative to those without PTSD (13%).

\(^1\) Patients’ sleep is adversely affected while they become accustomed to the wires and electrodes and the alien conditions of a sleep laboratory.

\(^2\) Those who served in the U.S. Armed Forces during the Vietnam era, but not in Vietnam.
3.2. Sleep maintenance insomnia

In Neylan et al.’s (1998) study, 91% of veterans with PTSD rated that they had difficulty maintaining sleep either ‘sometimes’ or ‘very frequently,’ compared to 63% of veterans without PTSD, 63% of era veterans, and 53% of civilians. In Ohayon and Shapiro’s (2000) study, compared to the non-PTSD participants, those with PTSD had more disrupted sleep (PTSD = 47%, non-PTSD = 18%) and would wake too early in the morning (PTSD = 43%, non-PTSD = 13%). This pattern of findings is expected given that the DSM-IV requires the presence of hyperarousal (Cluster D) for a diagnosis of PTSD such that two or more of the following symptoms must be present; insomnia, irritability, difficulty concentrating, hypervigilance, or increased startle. It is therefore intriguing to note that the opposite has been observed in experimental studies, as will be discussed below.

3.3. Nightmares

3.3.1. Prevalence

In Neylan et al.’s (1998) study, 52% of veterans with PTSD reported that they experienced nightmares either ‘sometimes’ or ‘very frequently,’ compared to 5% of veterans without PTSD, 6% of era veterans, and 3% of civilians. In Ohayon and Shapiro’s (2000) study, those with PTSD reported more nightmares (19%) relative to the non-PTSD (4%) sample. In a sample of adult female sexual or physical assault survivors with PTSD, nightmares were reported to occur approximately five times per week (Krakow, Schrader, et al., 2002).

3.3.2. Content

Several studies have systematically investigated the content of the nightmares experienced by people with PTSD. Four studies have been conducted by Mellman’s group. In a comparison of Vietnam veterans who were and were not diagnosed with PTSD, only those with PTSD reported dreams of wartime experiences, but both groups reported that disturbing dreams, along themes other than war, were common (Mellman, Kulick-Bell, Ashlock, & Nolan, 1995). In a later study, with individuals exposed to Hurricane Andrew, David and Mellman (1997) reported that 54% of the sample were able to describe their dreams, only five of which included content that was threatening or disturbing. Each of these five were reported by an individual diagnosed with PTSD. In the third study, dream reports were obtained via diaries completed on waking over two to three mornings (Mellman, David, Bustamante, Torres, & Fins, 2001). The participants in this study were consecutive trauma admissions following a motor vehicle accident, industrial accident, or assault. Sixty five percent of the sample remembered their dreams, and from these, 21 dream descriptions were obtained. Forty six percent of the dreams were about the trauma, with four including deviations from the original trauma and six being exact replications. It is notable that nightmares that were exact replications of the trauma were also reported by Schreuder, Kleijn, and Rooijmans (2000) and van der Kolk, Blitz, Burr, Sherry, and Hartmann (1984), although the latter study reported that while the nightmares reported by individuals with PTSD initially replicated the trauma, they changed over time to simply include threatening themes. Returning to the Mellman et al.
In the (2001) study, the patients who reported distressing dreams were also more likely to have more severe PTSD symptoms. Finally, Esposito, Benitez, Barza, and Mellman (1999) completed a study based on a sample of Vietnam veterans diagnosed with PTSD. Half of the dreams comprised combat themes, 85% were moderate to highly threatening, 53% were set in the present, and 79% had distorted elements.

Two other studies elucidating the content of posttraumatic dreams via subjective report have been published. First, Schreuder, van Egmond, Kleijn, and Visser (1998) assessed veterans with PTSD following their military service in the Dutch East Indies, civilians with PTSD from the Japanese occupation, and individuals with PTSD from the German occupation. Participants were asked to fill in a questionnaire about their sleep and dreams for 28 consecutive days. The results indicated that posttraumatic nightmares (20% of total nights) and anxiety dreams (5%) were very common. Nightmares often appeared to be exact replications of the trauma, and replication and repetition were highly correlated. Second, in a study by Dagan, Lavie, and Bleich (1991), the dreams of PTSD patients were found to be more hostile and aggressive, and in six patients, they were clearly related to war.

3.4. Associated features

Combat exposure has been reported to be strongly correlated with the frequency of nightmares, although only moderately correlated with problems falling asleep and weakly correlated with problems staying asleep (Neylan et al., 1998). In another study, having been a victim of aggression was more likely to be associated with sleep disturbance than other types of trauma (Ohayon & Shapiro, 2000). Woodward, Arsenault, Murray, and Bliwise (2000) assessed Vietnam veterans undergoing treatment for PTSD with over three or four nights. In the morning, dreams were recorded. Nightmares about Vietnam were found to be associated with increased awakenings during the night. It was not possible from the design of this study to determine if these results indicate that nightmares cause more awakenings or that a tendency to arouse more from sleep leads to more nightmares being reported. Recall from the discussion of human sleep presented earlier, dreams are only remembered if they are accompanied by an awakening.

Inman, Silver, and Doghramji (1990) assessed 35 Vietnam veterans with PTSD and 37 patients with insomnia but not PTSD. No differences between the two groups was observed in the severity of the insomnia; the PTSD group averaged 2.7 h of sleep per night, and the non-PTSD insomnia group averaged 2.8 h of sleep per night. However, the PTSD group reported more sleep-related anxiety symptoms, including (1) fear of going to sleep, (2) waking up with the covers torn apart, (3) fear of the dark, (4) having thoughts of the trauma whilst lying in bed, (5) having disturbing thoughts while lying in bed, (6) talking during sleep, (7) yelling/shouting during sleep, (8) waking up confused and disorientated, (9) fear of the dark, and (10) waking up from a frightening dream and then finding it hard to return to sleep.

3.5. Prognostic significance

A method that provides insight into the prognostic significance of sleep disturbance are reports of the predictive power of individual symptoms. For example, Harvey and Bryant
(1998) conducted a study that examined the power of posttrauma symptoms, as assessed within 1 month of the trauma, to predict a diagnosis of PTSD at 6 months posttrauma. Positive predictive power (PPP) was defined as the probability of the presence of ASD when the symptom is present, and negative predictive power (NPP) was defined as the probability of the absence of ASD when the symptom is absent. Of those who had nightmares within 1 month of the trauma, 33% went on to develop PTSD, whereas 9% did not. The PPP and NPP of nightmares was 0.55 and 0.80, respectively. Of those who had sleep disturbance within 1 month of the trauma, 72% went on to develop PTSD, whereas 36% did not. The PPP and NPP of nightmares was 0.41 and 0.87, respectively. That is, the probability of the presence of PTSD when sleep symptoms were present soon after the trauma was moderate, but the probability of the absence of PTSD when the sleep symptoms were absent was high.

A recent study by Koren, Arnon, Lavie, and Klein (2002) assessed sleep more thoroughly using the Mini Sleep Questionnaire (Zomer, Peled, Rubin, & Lavie, 1987) and found sleep disturbance to have strong prognostic significance. One hundred and two injured road traffic accident survivors and 19 matched participants admitted to the same orthopaedic unit for elective surgery were assessed at 1 week, and 1, 3, 6, and 12 months posttrauma. With the exception of the 1-week assessment, at all other assessments individuals with PTSD following a road traffic accident had more insomnia and daytime sleepiness than those without PTSD. Using logistic regression, the authors found that while insomnia at 1 week posttrauma was not predictive of PTSD, sleep complaints at 1 month posttrauma were a significant predictor of PTSD at 12 months posttrauma. That is, a ‘clear divergence’ (p. 856) on sleep symptoms was observed from 1 month posttrauma between those who had been in a road traffic accident but did and did not develop PTSD.

3.6. Summary

It appears that sleep onset and maintenance difficulties and frequent nightmares are commonly reported by PTSD patients. These findings endorse the inclusion of insomnia and nightmares in the DSM-IV criteria for PTSD. Unfortunately few studies have included a control group who was exposed to the trauma but did not develop PTSD. The results of those studies that have included a non-PTSD trauma-exposed group suggest that these features of sleep are fairly specific to PTSD. However, a caveat on this conclusion is that few studies have included a control group of patients diagnosed with other psychological disorders. It is well known that sleep disturbance is characteristic of a wide range of psychological disorders (see Harvey, 2001; McCrae & Lichstein, 2001 for review). Hence, on the basis of the data presented, it is not possible to know whether the level of sleep disturbance observed is specific to PTSD or a characteristic of psychological disorders in general. Nonetheless, giving an insight into the severity of the sleep disturbance experienced by PTSD patients, one study found that it was indistinguishable from the sleep disturbance exhibited by patients with insomnia (Inman et al., 1990). The importance of sleep disturbance in PTSD is clearly indicated by the findings that sleep disturbance has prognostic significance.
4. Characterising sleep in PTSD: objective reports

Interestingly, in contrast to the consistent findings of difficulty getting to sleep, difficulty maintaining sleep and nightmares among PTSD patients based on studies that have indexed sleep subjectively, the findings based on objective indices of sleep are mixed.

Replicating results from an earlier study of individuals with combat neurosis following the Yom Kippur War (Lavie, Hefez, Halperin, & Enoch, 1979), Mellman et al.’s studies with Vietnam veterans (Mellman, Kulick-Bell, et al., 1995; Mellman, Nolan, Hebding, Kulick-Bell, & Dominguez, 1997) and Hurricane Andrew survivors (Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995) have all detected significant differences between the sleep of PTSD patients relative to controls, as assessed with PSG. In Mellman, Kulick-Bell, et al. (1995), the PTSD group were found to have reduced sleep efficiency and more awakenings after sleep onset compared to the control group. In Mellman, David, et al., 1995, while there was no decrease in sleep efficiency, there were more awakenings and entries into Stage 1 sleep for the PTSD group relative to the control group. In a comparison of individuals with combat-related PTSD, depression, and controls over two nights of PSG, sleep efficiency was reduced in the PTSD group compared to the other groups (Mellman et al., 1997). Together, Mellman et al. have concluded that their findings support the proposal that sleep maintenance is impaired in chronic PTSD, and they theorised that the sleep disturbance observed in PTSD is the result of heightened arousal.

Providing evidence consistent with this conclusion, Fuller, Waters, and Scott (1994) reported that although there were no differences detected in the total number of awakenings experienced during the night between those with and without PTSD, there was a different distribution of awakenings across the sleep period with the controls exhibiting a normal pattern (reduced arousal in first half of night). In contrast, PTSD patients had more awakenings during the first half of the night. As most of Stages 3 and 4 sleep occur in the first part of the night, the increased awakenings during the first half of the night in the PTSD group may have caused them to obtain reduced slow-wave sleep (Stages 3 and 4 sleep).

However, other studies have not reported differences between individuals with PTSD and controls in sleep efficiency and awakenings (Dagan, Zinger, & Lavie, 1997; Engdahl, Eberly, Hurwitz, Mahowald, & Blake, 2000; Hurwitz, Mahowald, Kuskowski, & Engdahl, 1998). Further, Woodward, Friedman, and Bliwise (1996) compared individuals diagnosed with PTSD and major depressive disorder and individuals diagnosed with PTSD without major depressive disorder. Controlling for the effect of depression is helpful because depression is characterised by sleep disturbance (American Psychiatric Association, 1994) and is highly comorbid with PTSD (Keane & Wolfe, 1990; Shore, Vollmer, & Tatum, 1989). Few differences were detected between the groups on sleep architecture, suggesting that comorbidity with depression does not provide an explanation of the null findings quoted above.

4.1. The discrepancy between objective and subjective sleep

One potential account of the discrepancy between the subjective and (at least some) of the objective findings is that PTSD may be associated with ‘subjective insomnia,’ a state where
people wake in the morning with the honest and distinct impression that they have slept poorly, but relative to objective measures of sleep, have overestimated how long it took them to fall asleep and underestimated how much sleep they obtained overall. Consistent with this proposal are the studies that have directly compared subjective and objective sleep estimates in individuals with PTSD (Dagan et al., 1997; Engdahl et al., 2000; Hurwitz et al., 1998). Using both actigraphy (Dagan et al., 1997) and PSG (Engdahl et al., 2000; Hurwitz et al., 1998), PTSD patients have been found to underestimate their total sleep time and overestimate their sleep onset latency. This tendency is not only unique to PTSD patients, it is also ubiquitous among patients with insomnia (e.g., Bixler, Kales, Leo, & Slye, 1973; Bonnet, 1990; Borkovec & Weerts, 1976; Carskadon et al., 1976; Coates et al., 1982).

Theoretical accounts of distorted perception of sleep in the insomnia literature are, firstly, that traditional scoring of PSG provides a gross overview of sleep architecture and that more subtle abnormalities, such as alpha-delta sleep (Hauri & Hawkins, 1973), beta EEG during sleep (Perlis, Merica, Smith, & Giles, 2001), or cyclical alternating patterns in the sleep EEG (Terzano & Parrino, 2000) may be present. A second possibility, not necessarily inconsistent with the first, is that excessive pre-sleep cognitive activity and anxiety may cause distorted perception (Borkovec, 1982; Harvey, 2002). Drawing on Cantor and Thomas’ work indicating that time estimation increases with an increase in the number of information units processed (Cantor & Thomas, 1977; Thomas & Cantor, 1975, 1976), Borkovec (1982) proposed that distortions in time estimation during the pre-sleep period may be a direct result of excessive pre-sleep cognitive activity. Note that both worry/rumination are features of insomnia (Harvey, 2000; Wicklow & Espie, 2000) and PTSD (Mayou, Ehlers, & Bryant, 2002; Murray, Ehlers, & Mayou, 2002). A third possibility is based on the finding that PTSD patients have more awakenings from sleep (Mellman, David, et al., 1995). It is possible that in the context of regular brief awakenings, patients with PTSD perceive continuous wakefulness. Misperceptions of this type would not be surprising given that the moment of falling asleep is elusive and defined by the absence of memories and that sleep onset typically occurs in darkness with few or no time cues (Bonnet, 1990; Ogilvie & Harsh, 1994).

4.2. Summary

The findings based on objective measures are mixed with some studies indicating that individuals with PTSD have significantly more disturbed sleep and lower sleep efficiency compared to controls and other studies showing that PTSD patients fall into the normal range, as indexed by objective sleep parameters. Importantly, the studies reviewed highlight the possibility that perceived sleep may be more disturbed than actual sleep. This is an important finding that we will revisit when we discuss directions for future research below.

Those studies that did report objective differences between the sleep of people with and without PTSD pointed to heightened arousal as the explanation. Future testing of this hypothesis should include comparisons between the sleep of patients with PTSD and other

3 Standards for classifying human sleep are described by Rechtschaffen and Kales (1973).
psychological disorders known to be characterised by heightened arousal, such as the anxiety disorders.

5. Arousal/awaking threshold

In the section describing subjective sleep, it was evident that patients with PTSD report that they have difficulty with sleep maintenance. That is, they wake up during the night and have difficulty falling asleep again. This pattern of findings is consistent with the emphasis on hyperarousal in the DSM-IV criteria for PTSD. Hence, it is surprising that several experimental studies indicate that PTSD is not characterised by a decreased arousal threshold during sleep. In contrast, from the two studies described below, it appears that an increased threshold is present. That is, people with PTSD appear to be harder to wake when sleeping.

Dagan et al. (1991) assessed a PTSD and control group over three nights of PSG. On the third night, awakening thresholds were measured. Prior to bedtime, baseline response to the tones used in the experiment was indexed. Then, 15 min after the onset of Stage 3/4 sleep, 1-s 1500-Hz tones were delivered through small earphones starting at 0 dB and incremented in 5-dB steps. The intertone interval was 10 s. As already highlighted, Stage 3/4 sleep is the deepest and the most difficult to wake from. When the patient woke, they were allowed to go back to sleep and the procedure started again 15 min after the reappearance of Stage 3/4. The results indicated that PTSD patients had an increased threshold of awakening. That is, they were harder to wake from slow-wave sleep. As this was greater in patients with more disrupted sleep, Dagan et al. suggested that these findings indicate the presence of a compensatory mechanism designed to stop external or internal stimuli causing frightening awakenings.

In a test of this hypothesis, Lavie, Katz, Pillar, and Zinger (1998) recruited 12 individuals with PTSD and 12 controls from the Israeli Defence Force Veterans. PSG was conducted over four nights. Awaking thresholds to clicks were determined on one of the nights. Starting from the second entry into REM, 1-s 1000-Hz tones that gradually increased by 5 dB were played until the participant woke. In terms of thresholds, the PTSD patients had higher awakening thresholds, and these were positively correlated to levels of depression and anxiety. The authors suggest that the results reflect an ‘active blocking mechanism invoked to suppress trauma-related anxiety-provoking materials during sleep’ (p. 1063) and/or that there is an extreme inwardly directed attention because of intense preoccupation and inability to disengage from traumatic memories.

5.1. Summary

Thus, while many studies have reported increased awakenings to be characteristic of PTSD, the experimental studies have found reduced thresholds for awakening. However, it should be noted that the experimental studies have only indexed arousal thresholds from Stages 3 and 4 sleep. It will be necessary for future research to index arousal thresholds during Stages 1 and 2 or REM sleep. In addition, the arousal stimuli were tones of neutral
valence. It is possible that individuals with PTSD would have lower arousal thresholds for stimuli with threatening meaning.

6. REM sleep

As most dreams and nightmares are thought to occur during REM sleep, it is appropriate to consider findings relating to abnormalities in REM sleep. It is possible that explorations relating to REM sleep may illuminate mechanisms involved in PTSD. Differences between PTSD patients and controls in (1) amount of REM, (2) latency to REM, (3) density of REM, and (4) awakenings from REM have all been noted. When reading this section, it may be helpful to recall that possible functions of REM sleep include learning and emotional processing.

Taking the amount of REM first, studies have produced mixed results with some studies reporting that PTSD is associated with more REM sleep (Engdahl et al., 2000; Ross et al., 1994a, 1994b) and others reporting that PTSD is not associated with more REM (Dow, Kelsoe, & Gillin, 1996; Lavie et al., 1979; Mellman et al., 1997). Given the high comorbidity between PTSD and depression (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), one might expect PTSD, like depression, to be characterised by reduced latency to REM. However, mixed results have been noted with shorter latencies being reported in some studies (Glaubman, Mikulincer, Porat, Wasserman, & Birger, 1990; Schlosberg & Benjamin, 1978) and longer latencies in others (Greenberg, Pearlman, & Gampel, 1972; Kauffmann, Reist, Djenderedjian, Nelson, & Haier, 1987; Reist, Kauffmann, Chicz-Demet, Chen, & Demet, 1995). Less controversial is the finding of increased REM density in PTSD patients. Four studies have noted increased frequency of eye movements within REM periods (known as REM density; Mellman, Kulick-Bell, et al., 1995; Mellman et al., 1997; Ross et al., 1994a, 1994b, 1999), and severity of PTSD is correlated with REM density (Mellman, David, et al., 1995). However, the extent to which this finding is a function of comorbid disorders has yet to be determined. Major depression is well known to be associated with increased REM density (Pillar et al., 2000), and Mellman et al. (1997) did not find a difference between individuals with PTSD and individuals with depression for REM density. Finally, Mellman, Kulick-Bell, et al. (1995) noted more awakenings from REM in PTSD patients. This finding may reflect increased arousal during REM in PTSD (Mellman, 2000), but needs to be replicated.

6.1. Summary

Several authors have reported abnormalities in REM sleep to characterise PTSD. Such findings have led some to conclude that PTSD is an REM disorder (e.g., Ross et al., 1994b). However, as the support for such a view is mixed, more research is required to conclusively determine the role of REM in PTSD. Further, as will be discussed below, the studies conducted have often been based on small samples with the effects of comorbidity and medications being largely overlooked.
7. Body/limb movement during sleep

Prior to the introduction of PTSD into DSM-III, Lavie and Hertz (1979) reported that individuals with ‘combat neurosis’ following the Yom Yippur War showed higher rates of body movement during Stage 2 sleep compared to controls. Interestingly, this early finding has been replicated on several occasions in modern PTSD studies. Inman et al. (1990) assessed 35 Vietnam veterans with PTSD and 37 patients with insomnia but not PTSD. While no differences between the two groups were observed in the severity of the insomnia, the PTSD group were more likely than the insomnia group to report waking up with the covers torn apart, restless legs in bed, and excessive body movement during sleep. Extending this finding by the inclusion of an objective index of limb movements during sleep, Mellman, David, et al. (1995), Mellman, Kulick-Bell, et al. (1995), Ross et al. (1994b), and Brown and Boudewyns (1996) all reported more body movement during sleep for individuals with PTSD relative to control patients.

7.1. Summary

Body movements during sleep are typically conceived to have a medical origin, not a psychological origin, the main relevant medical disorder being periodic limb movement disorder. Periodic limb movement disorder is characterised by sudden and repetitive episodes of leg movement during sleep, often associated with an awakening. Limb movements are most frequent in Stages 1 and 2 of NREM sleep, less frequent in Stages 3 and 4, and mostly absent from REM sleep (American Sleep Disorders Association, 1997). Although a number of studies show increased evidence of limb movement during sleep in individuals with PTSD, it is difficult to know whether the origins are medical or psychiatric. It will be necessary to assess body movement during sleep across a broader range of trauma survivors and compare individuals with PTSD to those with a similar level of trauma exposure and to a control group of the same age. The latter is particularly important as the risk of limb movement disorder during sleep increases with age (see Morgan, 2000 for review).

8. Breathing-related sleep disorder

Breathing-related sleep disorders are characterised by repetitive episodes during sleep of upper airway closure or obstruction. This results in the cessation or decrease of airflow and the person waking from sleep so they can reestablish their breathing (American Sleep Disorders Association, 1997). There are several recent reports of more frequent breathing-related problems during sleep among patients with PTSD.

Krakow, Germain, et al. (2000) assessed 156 female participants who had had unwanted sexual experiences and had weekly nightmares and insomnia. Ninety-five percent of the participants met diagnostic criteria for PTSD. Based on self-report, 52% of the sample reported the presence of sleep-disordered breathing, and this was associated with more severe PTSD. While this study suggests that there may be an association with sleep-disordered breathing and PTSD, it has a number of limitations. First, diagnosing sleep-related breathing problems via
questionnaire is highly problematic. PSG accompanied by various respiratory devices⁴ is essential because the patients are not necessarily aware of their breathing abnormality. Second, a representative sample of individuals with PTSD was not included; the participants included were those who already had sleep problems. Krakow, Melendrez, et al. (2001) also interviewed 45 consecutive crime victims with nightmares and insomnia, 39 of whom had PTSD. In this study, obstructive sleep apnea was assessed with PSG, along with the required respiratory equipment. Ninety percent of the participants were found to have clinically significant levels of sleep-disordered breathing. However, this study was limited by the lack of an age-matched control group. In a further study, Krakow, Melendrez, et al. (2002) assessed 187 sexual assault survivors with posttraumatic stress symptoms. Compared to the 19 women without sleep-disordered breathing, the 168 who were thought to suffer from sleep-disordered breathing also reported more nightmares, poorer sleep quality and worse anxiety, depression, and posttraumatic stress. It should be noted, however, that the diagnosis of sleep-disordered breathing was only confirmed with PSG in 21 of the 168 patients with a suspected problem.

Youakim, Doghramji, and Schutte (1998) reported a case study of a 42-year-old Vietnam veteran with PTSD and obstructive sleep apnea (a severe breathing-related sleep disorder). The key finding was that when the obstructive sleep apnea was treated, the PTSD reduced. Consistently, Krakow, Lowry, et al. (2000) contacted 23 individuals who previously suffered from chronic nightmares (15 with PTSD) and had also been diagnosed with and offered treatment for sleep-disordered breathing. Fourteen accepted treatment and nine discontinued treatment. In those with PTSD, nine in the treatment group reported 75% improvement in their PTSD symptoms and six in the no-treatment group reported 43% worsening in PTSD. Although these treatment studies point to an association between sleep-disordered breathing and PTSD, it should be noted that the Krakow et al. (2000) study has several significant limitations; the presence of PTSD was determined by clinician interview rather than a psychometrically validated interview for the diagnosis of PTSD, random allocation to treatment versus no treatment was not undertaken, and the diagnoses of sleep-disordered breathing were determined retrospectively via inspection of the medical files.

8.1. Summary

The studies reviewed suffer from major methodological limitations. Nonetheless, the results suggest that future explorations of the association between sleep-disordered breathing and PTSD are warranted. Consideration should be given to the development of a stronger theoretical base as a guide to future work.

9. Methodological limitations

One aspect of this literature that is of concern relates to the methodology of the studies. As such, we have summarised the key aspects of the method employed by the studies

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⁴ Including nasal/oral airflow, oximetry, and respiratory effort sensors.
<table>
<thead>
<tr>
<th>Author</th>
<th>Trauma type</th>
<th>Trauma-study interval *</th>
<th>PTSD diagnostic tool</th>
<th>Subjective index of sleep disturbance</th>
<th>Sample recruitment</th>
<th>N PTSD patients</th>
<th>Comparison group (i.e. no PTSD)</th>
<th>Comorbid condition</th>
<th>Medication excluded?</th>
<th>First night excluded?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greenberg et al., 1972</td>
<td>Vietnam War</td>
<td>during or shortly after combat experience</td>
<td>war neurosis ** none</td>
<td>?</td>
<td>7</td>
<td>none</td>
<td>?</td>
<td>depression (clinician interview)</td>
<td>no</td>
<td>no</td>
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<tr>
<td>Schlosberg &amp; Benjamin, 1978</td>
<td>October, 1973 war, Israel</td>
<td>5 years</td>
<td>combat fatigue ** none</td>
<td>hospitalised with acute combat fatigue</td>
<td>3</td>
<td>none</td>
<td>?</td>
<td>depression (clinician interview)</td>
<td>yes</td>
<td>?</td>
</tr>
<tr>
<td>Lavie et al., 1979</td>
<td>Yom Kippur War</td>
<td>2–2.5 years</td>
<td>combat neuroses ** none</td>
<td>hospitalised with combat neurosis</td>
<td>11</td>
<td>non-traumatised, healthy men (N=9)</td>
<td>depression (clinician interview)</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>Lavie &amp; Hertz, 1979</td>
<td>Yom Kippur War</td>
<td>2–2.5 years</td>
<td>combat neuroses ** none</td>
<td>hospitalised with combat neurosis</td>
<td>11</td>
<td>non-traumatised, healthy men (N=9)</td>
<td>depression (clinician interview)</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>van der Kolk et al., 1984</td>
<td>Vietnam War</td>
<td>15 years</td>
<td>clinician interview (DSM-III)</td>
<td>interview about nightmares and sleep disturbance</td>
<td>15</td>
<td>lifelong nightmare sufferers without combat experience (N=10)</td>
<td>bipolar disorder, major depressive disorder, alcohol and drug abuse (clinician interview)</td>
<td>no</td>
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<tr>
<td>Glaubman et al., 1990</td>
<td>mixed</td>
<td>?</td>
<td>clinician interview (DSM-III)</td>
<td>sleep questionnaire, assessing dreams and nightmares</td>
<td>none</td>
<td>7</td>
<td>non-traumatised, healthy men (N=7)</td>
<td>?</td>
<td>yes</td>
<td>?</td>
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<tr>
<td>Dagan et al., 1991</td>
<td>Lebanon War</td>
<td>4–6 years</td>
<td>clinician interview (DSM-III)</td>
<td>psychiatric outpatient’s clinic</td>
<td>?</td>
<td>24</td>
<td>non-traumatised, healthy men (N=6)</td>
<td>none (clinician interview)</td>
<td>yes</td>
<td>no</td>
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<td>Fuller et al., 1994</td>
<td>Vietnam War, World War II</td>
<td>?</td>
<td>Mississippi PTSD Scale (Keane, Caddell, &amp; Taylor, 1988)</td>
<td>none</td>
<td>?</td>
<td>10</td>
<td>non-traumatised, healthy adult males (N=10)</td>
<td>depression, history of alcohol abuse (clinician interview)</td>
<td>no</td>
<td>no</td>
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<tr>
<td>Ross et al., 1994a, 1994b</td>
<td>Vietnam War</td>
<td>25 years</td>
<td>SCID (DSM-III-R) (Spitzer et al., 1987)</td>
<td>veterans’ centre</td>
<td>12</td>
<td>healthy military veterans (N=10)</td>
<td>depression, panic disorder, agoraphobia, GAD, hypomania (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
<td></td>
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<tr>
<td>Author</td>
<td>Trauma type</td>
<td>Trauma-</td>
<td>PTSD diagnostic</td>
<td>Subjective index of sleep disturbance</td>
<td>Sample recruitment</td>
<td>$N$ PTSD patients</td>
<td>Comparison group (i.e. no PTSD)</td>
<td>Comorbid condition</td>
<td>Medication excluded?</td>
<td>First night excluded?</td>
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<tr>
<td>Mellman, Kulick-Bell, et al., 1995</td>
<td>Vietnam War</td>
<td>25 years</td>
<td>Mississippi PTSD Scale</td>
<td>sleep diary</td>
<td>Vietnam medical centres</td>
<td>37</td>
<td>combat-exposed veterans ($N=21$), non-traumatised, healthy males ($N=8$)</td>
<td>major depression, panic disorder, substance use disorder (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>Mellman, David, et al., 1995</td>
<td>Hurricane</td>
<td>6 – 12 months</td>
<td>SCID (DSM-III-R)</td>
<td>PSQI (Buysse et al., 1989), questions about dreams</td>
<td>volunteer sample of community hit by hurricane</td>
<td>19</td>
<td>hurricane survivors ($N=35$), non-traumatised, healthy subjects ($N=9$)</td>
<td>depression, anxiety disorders (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>Mellman, Kumar, et al., 1995</td>
<td>Vietnam War</td>
<td>25 years</td>
<td>Mississippi PTSD Scale</td>
<td>none</td>
<td>Vietnam medical centres</td>
<td>20</td>
<td>Non-combat healthy veterans ($N=8$)</td>
<td>depression, substance abuse (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>Reist et al., 1995</td>
<td>?</td>
<td>?</td>
<td>SCID (DSM-III-R), IES</td>
<td>none</td>
<td>volunteer sample of patients hospitalised with diagnosis of PTSD</td>
<td>21</td>
<td>none</td>
<td>major depression, history of alcohol abuse (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>Brown &amp; Boudewyns, 1996</td>
<td>Vietnam War</td>
<td>25 years</td>
<td>SCID (DSM-III-R), CAPS (Blake et al., 1995)</td>
<td>none</td>
<td>consecutive in-patients from veterans’ centre advertisements</td>
<td>25</td>
<td>none</td>
<td>Past history of substance abuse (SCID [DSM-III-R])</td>
<td>yes</td>
<td>no</td>
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<td>Dow et al., 1996</td>
<td>Vietnam War</td>
<td>25 years</td>
<td>PTSD-I (Watson, Juba, Manifold, Kacala, &amp; Anderson, 1991), Mississippi PTSD Scale</td>
<td>questions about dreams</td>
<td>inpatient veterans’ centre</td>
<td>14</td>
<td>combat-exposed veterans with depression ($N=15$), without depression ($N=12$)</td>
<td>depression (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>Woodward et al., 1996</td>
<td>Vietnam War</td>
<td>25 years</td>
<td>SCID (DSM-III-R), CAPS</td>
<td>none</td>
<td>inpatient veterans’ centre</td>
<td>27a</td>
<td>none</td>
<td>history of alcohol abuse, substance abuse (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
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<td>Dagan et al., 1997</td>
<td>Lebanon War</td>
<td>8 years</td>
<td>clinician interview (DSM-III)</td>
<td>sleep questionnaire (six items concerning sleep behaviour from IES, SCL-90 and DSM-III-R)</td>
<td>random selection from PTSD outpatients</td>
<td>161</td>
<td>combat-exposed, healthy men ($N=11$)</td>
<td>depression, panic disorder, substance use disorder (SCID [DSM-III-R])</td>
<td>?</td>
<td>no</td>
</tr>
<tr>
<td>Mellman et al., 1997</td>
<td>Vietnam War</td>
<td>25 years</td>
<td>Mississippi Scale, SCID (DSM-III-R)</td>
<td>none</td>
<td>Vietnam medical centres, in- and outpatients</td>
<td>25</td>
<td>Men with principal diagnosis of major depression ($N=16$) (SCID [DSM-III-R])</td>
<td>depression, panic disorder, substance use disorder (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Author</td>
<td>Trauma type</td>
<td>Trauma-study interval</td>
<td>PTSD diagnostic tool</td>
<td>Subjective index of sleep disturbance</td>
<td>Sample recruitment</td>
<td>N PTSD patients</td>
<td>Comparison group (i.e. no PTSD)</td>
<td>Comorbid condition</td>
<td>Medication excluded?</td>
<td>First night excluded?</td>
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<tr>
<td>Hurwitz et al., 1998</td>
<td>Vietnam War</td>
<td>30 years</td>
<td>SCID (DSM-III-R), IES</td>
<td>sleep disorders questionnaire</td>
<td>veterans’ centre</td>
<td>18</td>
<td>non-traumatised era veterans</td>
<td>dysthymia, depression, panic disorder, OCD, social phobia, bipolar disorder (SCID [DSM-III-R])</td>
<td>yes</td>
<td>no</td>
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<tr>
<td>Lavie et al., 1998</td>
<td>Lebanon War</td>
<td>15 years</td>
<td>Clinician interview (DSM-III), IES</td>
<td>?</td>
<td>?</td>
<td>12</td>
<td>healthy combat-exposed veterans</td>
<td>depression, GAD, agoraphobia, hypomania, panic disorder (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Youakim et al., 1998</td>
<td>Vietnam War</td>
<td>30 years</td>
<td>?</td>
<td>?</td>
<td>patient at sleep disorders centre</td>
<td>1</td>
<td>none</td>
<td>yes</td>
<td>no</td>
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<tr>
<td>Ross et al., 1999</td>
<td>Vietnam War</td>
<td>30 years</td>
<td>SCID-NP-V (Spitzer et al., 1987)</td>
<td>questions about dreams</td>
<td>veterans’ psychiatry clinic and residential treatment unit</td>
<td>17</td>
<td>healthy men</td>
<td>yes</td>
<td>no</td>
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<tr>
<td>Engdahl et al., 2000</td>
<td>prisoner of war</td>
<td>55 years</td>
<td>SCID (DSM-III-R)</td>
<td>patient rating of quality and quantity of sleep sleep diary</td>
<td>random selection from POWs in the community</td>
<td>30†</td>
<td>prisoners of war</td>
<td>depression, history of alcohol abuse, illicit substance abuse (SCID [DSM-III-R])</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Woodward et al., 2000</td>
<td>Vietnam War</td>
<td>30 years</td>
<td>SCID (DSM-III-R), CAPS</td>
<td>consecutive referrals by private therapists or rape crisis centres</td>
<td>inpatient veterans’ centre</td>
<td>63</td>
<td>none</td>
<td>yes</td>
<td>yes</td>
<td></td>
</tr>
<tr>
<td>Krakow, Johnston, et al., 2001</td>
<td>crime</td>
<td>6 months</td>
<td>PDS (Foa, Riggs, Dancu, &amp; Rothbaum, 1993)</td>
<td>PSQI</td>
<td>consecutive referrals by private therapists or rape crisis centres</td>
<td>39</td>
<td>crime victims</td>
<td>yes</td>
<td>no</td>
<td></td>
</tr>
</tbody>
</table>


This study compared PTSD patients with major depression (N = 17) to PTSD patients without major depression (N = 10).

None of the studies with participants from Vietnam or World War II specified the time elapsed between trauma and assessment. Hence, 1970 and 1945, respectively, have been used as the trauma dates. Then trauma-study interval is estimated to the nearest 5 years.

‘Combat fatigue’, ‘combat neurosis’, ‘war neurosis’ are terms used before the inception of PTSD as a formal diagnosis.

This study used actigraphy rather than PSG.

These patients were also assessed with actigraphy.
reviewed in two tables. Table 1 presents those studies that included objective measures of sleep. Table 2 presents those studies that included only subjective measures of sleep. The tables are divided in this way because the methodological features required in studies that include PSG are more sophisticated.

As evident from Tables 1 and 2 (in the column headed ‘Trauma type’), most of the studies have been conducted on combat veterans. These findings cannot be directly generalised to other trauma groups as the trauma often occurred many years ago and the samples mainly include men older than 50 years of age. The latter point is particularly relevant to sleep because the risk of breathing related and limb movement problems during sleep increases with age (Morgan, 2000). A more general, but related point, is that as the traumas that have been studied differ substantially in terms of the severity, extent of violation, perceived threat, and meaning of the trauma, it is not possible to directly generalise findings from one trauma group to another. As such, it will be important for future research to build an evidence based on sleep in PTSD across a range of trauma types.

The gap between the occurrence of the trauma and participation in the study is also relevant (see the column headed ‘Trauma-study interval’). The longer the time elapsed, the more likely that factors other than PTSD may contribute to the results. Comorbidity, compensation, and the socio-political environment are examples of such factors. It should be noted that few studies have focused on the acute trauma phase. It is possible that acute and chronic PTSD are characterised by different sleep patterns. Mellman (2000 p. 301) argues that the sleep findings in ‘chronic PTSD likely reflect progression over time of an unknown number of secondary factors.’

Most of the studies completed did employ psychometrically validated instruments for the diagnosis of PTSD. Relying on informal clinician interview can lead to inaccuracy, as there is no way to ensure that the questions asked reliably and validly index each of the diagnostic criteria. In terms of the tools to index sleep disturbance, as there can be a discrepancy between subjectively and objectively measured sleep, it is important to index the subjective perception of sleep. Although several studies have done this, few have used assessment tools that have been developed and validated to specifically index sleep disturbance (see the column headed ‘Subjective index of sleep disturbance’).

In terms of the sample recruitment, basing recruitment on consecutive referrals is ideal as then it is possible to estimate how representative the sample is of the population it is supposed to represent. As evident in Tables 1 and 2 (see the column headed ‘Sample recruitment’), few of the studies conducted were based on consecutive samples. A further limitation is that the size of the samples tends to be small; this is particularly true for the studies summarised in Table 1. On the one hand, this is not surprising as Table 1 summarises studies that have used overnight PSG, which is expensive and time consuming and often not prioritised by funding bodies. On the other, small samples raise the concern that the results will not be generalisable.

Unfortunately, several of the studies reviewed did not include a control group (see the column headed ‘Comparison group’ in Tables 1 and 2). As a minimum, there should be a control group who endured an identical trauma but did not develop PTSD, as well as a no-trauma control group. All groups should be matched on age, sex, and socioeconomic status. A crucial question, yet to be fully tackled, is whether patients with PTSD exhibit sleep
<table>
<thead>
<tr>
<th>Author</th>
<th>Trauma type</th>
<th>Trauma-study interval *</th>
<th>PTSD diagnostic tool</th>
<th>Subjective index of sleep disturbance</th>
<th>Sample recruitment</th>
<th>N PTSD patients</th>
<th>Comparison group (i.e., no PTSD)</th>
<th>Comorbid condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goldstein et al., 1987</td>
<td>prisoner of war (Japan)</td>
<td>40 years</td>
<td>clinician interview (DSM-III)</td>
<td>DSM-III (sleep items of PTSD criteria)</td>
<td>participants in a comprehensive evaluation service at a Veterans Administration centre</td>
<td>20</td>
<td>none history of alcohol abuse (clinician interview)</td>
<td>depression (clinician interview)</td>
</tr>
<tr>
<td>Inman et al., 1990</td>
<td>Vietnam War</td>
<td>20 years</td>
<td>clinician interview (DSM-III)</td>
<td>sleep disturbance inventory indexing general sleep, insomnia, and nightmares</td>
<td>random selection of in- and outpatients from PTSD programme at veterans medical centre</td>
<td>35</td>
<td>non-traumatised group seeking treatment for insomnia (N=37)</td>
<td>depression (clinician interview)</td>
</tr>
<tr>
<td>Kuch &amp; Cox, 1992</td>
<td>Holocaust</td>
<td>45 years</td>
<td>clinician interview (DSM-III-R)</td>
<td>DSM-III-R (sleep items of PTSD criteria)</td>
<td>Holocaust compensation applicants</td>
<td>58</td>
<td>Holocaust survivors (N=65)</td>
<td>depression (clinician interview)</td>
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<tr>
<td>David &amp; Mellman, 1997</td>
<td>hurricane</td>
<td>6–12 months</td>
<td>SCID (DSM-III-R) (Spitzer et al., 1987)</td>
<td>PSQI (Buysse et al., 1989), questions about dreams</td>
<td>volunteer sample of community hit by hurricane consecutive admissions</td>
<td>20</td>
<td>hurricane survivors (N=29)</td>
<td>none (clinician interview)</td>
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<td>Harvey &amp; Bryant, 1998</td>
<td>MVA</td>
<td>1 and 6 months</td>
<td>ASDI (Bryant, Harvey, Dang, &amp; Sackville, 1998)</td>
<td>ASDI sleep items</td>
<td>all patients being treated with psychotropic medications</td>
<td>12</td>
<td>none all patients being treated with psychotropic medications</td>
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<tr>
<td>Neylan et al., 1998</td>
<td>Vietnam War</td>
<td>30 years</td>
<td>Mississippi PTSD scale (Keane et al., 1988)</td>
<td>Mississippi PTSD scale sleep items</td>
<td>population-based sample selected to represent entire Vietnam-serving population</td>
<td>180</td>
<td>theatre veterans, era veterans, and civilians (N=1020)</td>
<td>major depression, mania, panic disorder, substance abuse (DIS: Robins, Helzer, Croughan, &amp; Ratcliff, 1981)</td>
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<tr>
<td>Schreuder et al., 1998</td>
<td>World War II</td>
<td>55 years</td>
<td>CAPS (Blake et al., 1995), IES (Horowitz et al., 1979)</td>
<td>NITE</td>
<td>outpatients applying for treatment at centre for victims of violence consecutive appointments at outpatients clinic</td>
<td>33</td>
<td>none</td>
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<tr>
<td>Esposito et al., 1999</td>
<td>Vietnam War</td>
<td>30 years</td>
<td>CAPS</td>
<td>dream rating scale, dream diaries</td>
<td>all patients being treated with psychotropic medications</td>
<td>18</td>
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</tbody>
</table>
Table 2 (continued)

<table>
<thead>
<tr>
<th>Author</th>
<th>Trauma type</th>
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<th>PTSD diagnostic tool</th>
<th>Subjective index of sleep disturbance</th>
<th>Sample recruitment</th>
<th>N PTSD patients</th>
<th>Comparison group (i.e., no PTSD)</th>
<th>Comorbid condition</th>
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<tr>
<td>Krakow, Germain, et al., 2000</td>
<td>sexual assault</td>
<td>?</td>
<td>PSS-I (Foa et al., 1993)</td>
<td>PSQI</td>
<td>participants in a nightmare treatment programme patients attending a health centre with a chronic nightmare disorder</td>
<td>148</td>
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<td>Krakow, Lowry, et al., 2000</td>
<td>mixed</td>
<td>?</td>
<td>clinician interview (DSM-IV)</td>
<td>questions about use of treatment for sleep-disordered breathing and nightmares</td>
<td>sleep-EVAL (sleep items according to DSM-IV sleep disorders, and ICSD classification)</td>
<td>15</td>
<td>none</td>
<td>?</td>
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<tr>
<td>Ohayon &amp; Shapiro, 2000</td>
<td>mixed</td>
<td>?</td>
<td>clinician interview (DSM-IV)</td>
<td>sleep-EVAL (sleep items according to DSM-IV sleep disorders, and ICSD classification)</td>
<td>representative sample of general population</td>
<td>33</td>
<td>trauma-exposed and non-traumatised group (N = 1799)</td>
<td>bipolar disorder, major depressive disorder, dysthymia, panic disorder (clinician interview)</td>
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<tr>
<td>Schreuder et al., 2000</td>
<td>World War II</td>
<td>55 years</td>
<td>CAPS, IES</td>
<td>NITE-I questionnaire indexing general sleep information.</td>
<td>patients from war centre with complaints attributed (by self or a referring professional) to World War II consecutive admissions to trauma centre hospital inpatients</td>
<td>124</td>
<td>World War II survivors (N = 99)</td>
<td>?</td>
</tr>
<tr>
<td>Mellman et al., 2001</td>
<td>mixed</td>
<td>12 days and 6 weeks</td>
<td>SCID (DSM-III-R), CAPS</td>
<td>dream diaries</td>
<td>trauma-exposed group (N = 50) patients being treated with psychotropic medications</td>
<td>10</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Koren et al., 2002</td>
<td>MVA</td>
<td>1 week, 1, 3, 6, and 12 months</td>
<td>IES, SCID (DSM-III-R)</td>
<td>Mini Sleep Questionnaire (Zomer et al., 1987), indexing insomnia, and excessive daytime sleepiness</td>
<td>trauma-exposed group (N = 76) non-traumatised patients with orthopaedic injuries (N = 19)</td>
<td>26</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Krakow, Schrader, et al., 2002</td>
<td>sexual or physical assault</td>
<td>?</td>
<td>CAPS, PSS-I</td>
<td>health centres, and self-referral in response to advertisement</td>
<td>?</td>
<td>101</td>
<td>none</td>
<td>Depression (Hamilton Depression Rating Scale)</td>
</tr>
</tbody>
</table>


* Of the 15 participants, 9 were given a treatment for their sleep-disordered breathing, 6 were not.

* None of the studies with participants from Vietnam or World War II specified the time elapsed between trauma and assessment. Hence, 1970 and 1945, respectively, have been used as the trauma dates. Then, trauma-study interval is estimated to the nearest 5 years.
disturbance that is unique compared to the sleep disturbance exhibited by patients with other psychological disorders.

The columns in Tables 1 and 2 headed ‘Comorbid condition’ describes the comorbid conditions that were present at the time of the assessment. As PTSD is highly comorbid with other disorders, particularly depression and substance abuse (Keane & Wolfe, 1990; Shore et al., 1989), it is critical that studies assess for comorbidity with a psychometrically validated structured clinical interview such as the Structured Clinical Interview of the DSM-IV (SCID; Spitzer, Williams, Gibbon, & First, 1996). Pillar et al. (2000) suggested that the variability in sleep observed across the PTSD/sleep literature may be accounted for by the presence of comorbid conditions. In particular, substance abuse is important to assess for and document, as alcohol and drugs substantially alter sleep architecture (Roehrs & Roth, 2001). It is exciting to note that some recent studies have begun the complicated process of indexing the effects of comorbidity. For example, Woodward et al. (1996) compared a pure PTSD group with a PTSD plus depression group.

Some factors that mainly apply to the studies that employed PSG (see Table 1) are summarised under the columns titled ‘Medication excluded?’ and ‘First night excluded?’. As sleep architecture is significantly affected by a range of medications (Sharpley & Cowen, 1995), it is important that studies utilising PSG assess for, report, and sometimes exclude individuals on medication. In addition, as noted early in this paper, PSG is associated with a first night effect. Accordingly, it is standard practice for the data from the first night of PSG to be excluded. As evident in Table 1, it is encouraging to see that most studies reviewed have not analysed data from the first night of PSG.

10. Psychological treatment

10.1. Treating nightmares

As sleep disturbance is typically regarded as a secondary symptom in PTSD, it has rarely received specific therapeutic attention. The exception is a series of recent RCTs reported by Krakow et al. The aim of the first study was to determine whether imagery rehearsal therapy for nightmares would reduce reported nightmares, improve sleep quality, and decrease distress in patients with PTSD compared with a waiting list control group (Krakow, Hollifield, et al., 2001). One hundred and sixty-eight female sexual assault survivors with self-reported nightmares, insomnia, and posttraumatic stress symptoms were randomized into a treatment group ($n = 88$) or a waiting list control group ($n = 80$). Ninety-five percent of the sample reported moderate to severe posttraumatic stress, and the remainder experienced subsyndromal posttraumatic stress. The treatment was administered over three sessions in groups of four to eight and was specifically focused on the treatment of nightmares. In the first session, participants were engaged in a discussion of two contrasting accounts of nightmares: that nightmares are a function of traumatic exposure versus that nightmares are a function of both traumatic exposure and learned behaviours.
The latter gives rise to the possibility that nightmares may be induced by trauma but sustained by habit. Participants were also trained to engage in pleasant imagery as a distraction to unpleasant imagery. In the second session, participants were taught how to use imagery rehearsal therapy on a single self-selected nightmare. Imagery rehearsal therapy involves writing the dream down, changing it in any way the patient wishes, writing down the changed dream, and rehearsing it using imagery for 10–15 min per day. The patients were instructed never to work on more than two new dreams in 1 week. Requests to describe the traumatic experience and traumatic content of nightmares were discouraged throughout the program in an attempt to minimise direct exposure. The third session was devoted to assessing progress and clarifying the treatment approach. Imagery rehearsal therapy resulted in a significant reduction in the number of nightmares per week and in improved sleep. Interestingly, imagery rehearsal therapy was also associated with decreased mean PTSD severity from moderately severe to moderate levels. These effects were maintained over follow-ups carried out at 3 and 6 months. Although only 114 participants completed both 3- and 6-month follow-ups, intention to treat analysis confirmed significant differences between the control and treatment groups for reduced nightmares, better sleep, and improved PTSD. That is, the improvement in PTSD symptoms with a nightmare focused intervention was both substantial and sustained at 6-month follow-up. Of course, without a control group to ensure nonspecific factors were not responsible, these findings remain tentative.

10.2. Treating insomnia and nightmares

Krakow, Johnston, et al. (2001) further investigated the perspective that targeted sleep therapies may lead to better sleep quality and decreased distress in individuals with PTSD. Extending Krakow, Hollifield, et al. (2001), this study not only employed imagery rehearsal therapy as an intervention for nightmares, but also administered sleep hygiene, stimulus control, sleep restriction, and cognitive restructuring as an intervention for insomnia. Sixty-two crime victims with PTSD reporting weekly episodes of insomnia and nightmares were offered 10 h of group treatment. At the 3-month follow-up, reductions in nightmare frequency were roughly equivalent to a change in clinical severity from severe to moderate nightmare frequency; sleep quality scores changed from severe to moderate sleep disturbance; PTSD scores changed from the upper end to the lower end of moderately severe PTSD; anxiety and depression changed from extremely severe to moderately severe. While these findings are impressive in that a sleep-focused intervention resulted in an improvement in PTSD, all distress measures remained in the abnormal range after treatment. Further, limitations to these results arise from the fact that 66% of participants were in concurrent therapy, so it is difficult to be sure that the improvements were due to the sleep-targeted therapy. Furthermore, there was no control group of any type and no indication of dropout rate. Finally, Krakow, Melendrez, Johnston, Clark, et al. (2002) administered the same treatment in six 2-h weekly sessions to a large group ($n = 66$) of individuals who suffered posttraumatic insomnia and nightmares following the Cerro Grande Fire. Again, no control group was
included, but 53 of the participants benefited from the intervention. It is a major concern, however, that for 10 participants, sleep worsened.

10.3. Summary

Developments in research relating to the treatment of sleep symptoms in individuals with PTSD are relatively recent. Thus far, it appears that the common element for the successful treatment of nightmares is writing down the nightmare and rehearsing it in the imagination and in the waking state. That is, exposure-based treatments, known to be effective in the treatment of the daytime symptoms in PTSD (see Harvey, Bryant, & Tarrier, 2003), can be applied to the treatment of nightmares. It is particularly interesting to note that a sleep-targeted intervention not only reduced sleep disturbance, but also reduced the severity of PTSD symptoms (Krakow, Hollifield, et al., 2001; Krakow, Johnston, et al., 2001). However, the theoretical base for the interventions needs clarification. Is the aim to normalise sleep, reduce the frequency of nightmares, or promote emotional processing? Finally, any finding that indicates an adverse effect of treatment needs to be taken seriously. The finding that 10 of 66 of Krakow, Melendrez, Johnston, Clark, et al.’s (2002) patients worsened suggests that interventions delivered as a large group (n = 66) may be contraindicated for traumatised patients.

11. Perspectives on sleep disturbance in PTSD

The experimental work relating to sleep and PTSD is in its infancy, with many issues remaining to be explored. As such, an empirically supported theory of the relationship between sleep and PTSD has not been proposed. However, several interesting theoretical perspectives on the relation between sleep and PTSD have started to emerge as summarised below.

Krakow, Hollifield, et al. (2001) have proposed that nightmares and insomnia are intertwined in PTSD. According to this view, nightmares trigger a difficulty falling or staying asleep due to fear of having a nightmare. If this account is true, then the successful treatment of nightmares will improve sleep. Krakow et al. also suggested that nightmares are conditioned stimuli that trigger a conditioned response, waking the patient up from the bad dream to avoid the associated unpleasant emotion. Arousal from the dream reinforces the response that the only way to diminish the stimuli is not to sleep, a response that is maintained by the view that nightmares are a fixed and necessary reminder of the traumatic experience. Imagery rehearsal therapy, described above, is thought to act as a ‘reciprocal inhibitor to the original nightmare, providing a cognitive shift which empirically refutes the alleged purpose of the nightmare’ (Krakow, Hollifield, et al., 2001, p. 543).

Several experts have suggested that the sleep disturbance, characteristic of PTSD, is a manifestation of hyperarousal, the group of symptoms described in Cluster D of the PTSD diagnostic criteria and Cluster C of the ASD diagnostic criteria. In a recent review, Mellman (1997, p. 143) concluded that ‘sleep in chronic PTSD can be disrupted by intrusions of more
highly aroused states and behaviours (e.g., awakening and body movement).’ Others have argued that adaptations made to cope with a state of current threat, characteristic of PTSD, are not compatible with sleep (Woodward, 1995). Similarly, Pillar et al. (2000) noted the increased sensitivity and sensitization of the noradrenergic system that may leave the individual hyperaroused and sleep deprived.

The sleep disturbance increases a sense of loss of control. Sleep is controlled by the body’s homeostatic system. As sleep onset is associated with loss of awareness, reduction of thoughts and reduction of emotions, trying too hard to control sleep can make it more difficult to fall asleep and can worsen the feeling of losing control (Espie, 2002).

Sleep deprivation contributes to poor daytime coping. Sleep deprivation is known to increase fatigue, confusion, and tension/anxiety. Rothbaum and Mellman (2001, p. 485) noted that ‘If deprived of adequate sleep, one would expect the trauma survivor to be more sensitized by, reactive to, and therefore, more avoidant of exposures to reminders of the trauma, whereas a well-rested state would enhance an individual’s capacity for coping.’ Others have concluded that if sleep quality is disrupted, daytime functioning will be impaired, and both may function to (1) impair the processing and resolution of trauma and (2) increase anxiety (Krakow, Hollifield, et al., 2001).

Nightmares have been conceptualised as a sign of failed emotional processing (Rachman, 1980). As already alluded to one of the dominant theories of REM sleep is that it promotes learning and emotional processing. Although comparisons between individuals with PTSD and controls have yielded mixed findings regarding several aspects of REM sleep (amount of REM, reduced latency to REM, and awakenings from REM), increased REM density in PTSD patients has been noted more consistently. Such findings have led Ross et al. (1994a, 1994b) to suggest that an abnormal REM mechanism is involved in the pathogenesis of PTSD. Mellman (1997) also suggests that the REM findings are crucial to a full understanding of PTSD, although he concludes that at this stage, the role of REM sleep remains unresolved. Mellman suggests that the abnormalities noted relating to REM may be the result of an adaptive relationship between REM activity and emotional distress. This argument is supported by the empirical findings indicating that increased REM density among individuals suffering normal reactions to bereavement (Reynolds et al., 1993) and short REM latency in individuals affected by a recent negative life event (Williamson et al., 1995), including divorce (Cartwright, Kravitz, Eastman, & Wood, 1991).

Some authors have suggested that PTSD is characterised by avoidance during sleep, an ‘active blocking mechanism invoked to suppress trauma-related anxiety-provoking materials during sleep’ (Pillar et al., 2000, p. 192). This account potentially encompasses the findings of elevated arousal threshold (Dagan et al., 1991; Lavie et al., 1998) and increased limb movement and breathing problems during sleep (Lavie et al., 1998).

Why don’t nightmares promote habituation and recovery? Rothbaum and Mellman (2001) suggest that (1) dreams are not long enough in duration for habituation to occur, (2) following a nightmare, the person will often wake feeling highly anxious, this may sensitise them to the trauma memory, (3) negative reinforcement, a requirement for habituation, is not prevented in dreams, (4) the perception and meaning of the trauma is unlikely to be altered in dreams, and (5) a sense of control is not promoted.
12. Future directions

Inman et al. (1990) found that individuals with PTSD suffer significant levels of sleep-related anxiety symptoms such as fear of going to sleep, fear of the dark, and waking up from a frightening dream and finding it hard to go back to sleep. These symptoms have rarely been assessed in subsequent studies. Another issue yet to be addressed is the relation between daytime PTSD symptoms and nighttime PTSD symptoms. For example, is there a correlation between flashbacks during the day and nightmares at night?

While sleep disturbance in PTSD has sometimes been referred to as the ‘hallmark’ symptom (Ross, Ball, Sullivan, & Caroff, 1989), it has been more common for sleep symptoms to be viewed as secondary symptoms that remit with the successful treatment of PTSD. As such, the role of sleep in PTSD is understudied. Unfortunately, as randomised controlled trials of treatments for PTSD do not typically report whether sleep disturbance and nightmares fully resolve following cognitive behaviour therapy (CBT), there is a lack of empirical data to substantiate or refute this assumption. Although there are positive effects on PTSD symptoms of a treatment targeting sleep (Krakow, Hollifield, et al., 2001; Krakow, Johnston, et al., 2001), such findings cannot be taken as evidence that sleep is a primary mechanism in PTSD (Salkovskis, 2002). They do, however, suggest that the assumption that the sleep symptoms are secondary to PTSD should be revisited.

We also do not know whether the severity of the sleep disturbance varies throughout a course of CBT. It is possible that the early stages of therapy may be associated with poorer sleep, which then resolves as the trauma memories are processed and integrated (A. Hackmann, October 2002, personal communication). Resolving such issues will make clear the extent to which interventions specifically targeting sleep, such as those developed by Krakow et al., should be integrated with CBT for PTSD.

In this review, we have discussed evidence that individuals with PTSD suffer from ‘subjective insomnia.’ That is, they overestimate how long it takes them to go to sleep and underestimate how long they are asleep for. The source of the distorted perception needs investigation. A start would be to score the more subtle aspects of EEG, such as alpha–delta sleep (Hauri & Hawkins, 1973), beta EEG during sleep (Perlis et al., 2001), or cyclical alternating patterns in the EEG (Terzano & Parrino, 2000). If the finding holds, it may be necessary to provide patients with feedback of their objective sleep. In the context of insomnia, such an intervention was associated with a reduction in the perceived sleep distortion and sleep-related worry (Tang & Harvey, 2002).

Questions regarding what people with PTSD think about as they are trying to fall asleep, and during awakenings throughout the night, are unanswered. Such research might be guided by theoretical models of PTSD. Horowitz (1986) highlighted that trauma survivors have a tendency to oscillate between experiencing intrusive images and nightmares about the trauma and periods of avoidance and denial. It is possible that for PTSD patients, the pre-sleep period constitutes a specific inactive time when it is more difficult to adopt avoidance strategies, resulting in free-flowing intrusions. Intrusions are often accompanied by a sense of current threat as well as symptoms of arousal, anxiety, and other emotional responses (Ehlers & Clark, 2000), all of which are likely to prolong sleep-onset latency and promote fear of sleep.
On the basis that sleep disturbance is known to be characteristic of a wide range of psychological disorders (see Harvey, 2001; McCrae & Lichstein, 2001 for review), it has been emphasised that comparisons between the sleep of patients with PTSD and patients diagnosed with other psychological disorders are crucial for determining the extent to which the level of sleep disturbance observed is specific to PTSD or a characteristic of psychological disorders in general. In this paper, two similarities have emerged between the sleep of patients with PTSD and the sleep of patients with insomnia. First, Inman et al. (1990) showed that the two groups did not differ on the amount of sleep obtained. Second, it appears that patients with both disorders appear to overestimate how long it takes them to get to sleep and underestimate how long they sleep overall. Further comparisons between these two diagnostic groups are likely to be helpful in clarifying the mechanisms involved in PTSD. One likely candidate is that both PTSD and insomnia are characterised by worry and rumination, which are antithetical to getting to sleep and have been implicated in distorted perception of sleep (Harvey, 2002).

13. Conclusions

This paper has summarized and critically evaluated the literature on the prevalence and treatment of sleep disturbance characteristic of patients with PTSD. There is a clear association between PTSD and sleep disturbance. However, as most of the studies conducted to date are cross-sectional, the extent to which sleep disturbance is a key mechanism in PTSD is not yet clear. However, future research is warranted given the strength of the association and the range of interesting theoretical perspectives presented that specify testable hypotheses for the role that sleep may have in the maintenance of PTSD.

The relevance of the topic to clinical psychologists is indicated by findings such as Krakow, Johnston, et al.’s (2001) report that a sleep-focused intervention is associated with improvement in PTSD symptoms. Further, two findings raise the possibility that sleep may be an early indicator of those who will go on to develop PTSD. Koren et al. (2002) found that sleep complaints at 1 month posttrauma were a significant predictor of PTSD at 12 months posttrauma. In Mellman, David, et al.’s (1995) study of survivors of Hurricane Andrew, those who scored higher on measures of PTSD also had more sleep disturbance before the hurricane. The search for early indicators marking those who will go on to develop PTSD is important as it highlights those who may benefit from treatment aimed at preventing the development of PTSD (Bryant, Harvey, Dang, Sackville, & Basten, 1998; Harvey & Bryant, 2002).

References


