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THEORETICAL REVIEW

Trauma-induced insomnia: A novel model for trauma and sleep research

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SUMMARY

Traumatic events have been increasingly recognized as important precipitants of clinically significant insomnia. Trauma is an extreme form of stressful life event that generates a sustained neurobiological response triggering the onset and maintenance of insomnia. Trauma may disrupt the normal sleep-wake regulatory mechanism by sensitizing the central nervous system's arousal centers, leading to pronounced central and physiological hyperarousal. The central concept of hyperarousal has been linked to both the pathogenesis of insomnia and to the neurobiological changes in the aftermath of traumatic events, and may be a neurobiological commonality underlying trauma and insomnia. This paper presents evidence for trauma-induced insomnia and advances a model of it as an important nosological and neurobiological entity. Trauma-induced insomnia may occur in the absence of full-blown posttraumatic stress disorder (PTSD), and may also be a precursor of subsequent PTSD development. Converging lines of evidence from the neuroscience of insomnia with the neurobiology and psychophysiology of stress, fear, trauma and PTSD will be integrated to advance understanding of the condition. Preclinical and clinical stress and fear paradigms have informed the neurobiological pathways mediating the production of insomnia by trauma. Elucidating the underlying neurobiological substrates can establish novel biological markers to identify persons at risk for the condition, and help optimize treatment of the trauma–insomnia interface. Early identification and treatment of trauma-induced insomnia may prevent the development of PTSD, as well as other important sequelae such as depression, substance dependence, and other medical conditions.

Introduction

Insomnia is a prominent feature of the human neurobiological and physiological response to trauma. Following a traumatic event, exposed individuals report marked and enduring patterns of sleep disruption. That clinically significant insomnia (defined by symptoms of difficulty initiating sleep, difficulty maintaining sleep with sleep that is non-restorative, as well as awakening with difficulty returning to sleep) emerges in the aftermath of traumatic events has been increasingly established in sleep, trauma and stress research, and is a growing area of empirical focus [1].

This paper presents evidence for the phenomenon of trauma-induced insomnia and advances a model of it as an important nosological and neurobiological entity. The development of insomnia following a traumatic event may occur without the presence of full-blown posttraumatic stress disorder (PTSD) as a specific, circumscribed consequence of trauma exposure. It is also an important predictor of PTSD development that may serve as an informative marker for subsequent PTSD severity. Moreover, trauma-induced insomnia may be a precursor of other disabling posttraumatic complications such as depression, substance dependence, and other impairing medical conditions [1,2]. The potential importance of targeted, early treatment of trauma-induced insomnia to the prevention of significant posttraumatic sequelae will be discussed.

The central concept of hyperarousal is hypothesized to be a pivotal mechanism linking the posttraumatic response to clinically significant insomnia [3]. Drawing from clinical, physiological, and neurobiological studies, the relevance of hyperarousal to the production of insomnia in the wake of trauma will be evaluated. Although an optimal animal model of the human response to trauma has not yet been developed, knowledge gleaned from animal models utilizing stress and fear paradigms that model important aspects of human traumatic responses has been fruitful [4,5].

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Thus, preclinical stress and fear paradigms have the potential to inform the underlying neurobiological substrates of hyperarousal and insomnia posttrauma in humans, and the relevant findings will be discussed. The importance of establishing novel biological markers and optimizing treatments, and the need for prospective evaluation of trauma-induced insomnia with systematic studies, will be emphasized.

Insomnia and traumatic events

Chronic insomnia is a prominent and debilitating consequence of exposure to traumatic events. Trauma lies at the extreme end of a continuum model of stressful life events [6,7]. Traumatic events are defined formally in the DSM-IV as an event involving actual or potential death or serious injury that an exposed person experiences (or witnesses), and responds with intense fear, helplessness or horror [8].

The experience of a traumatic event can significantly disrupt sleep integrity and continuity in exposed individuals. Following the attacks of September 11, 2001, approximately 25% of New Yorkers suffered from clinically significant insomnia [9]. Survivors of traumatic events, including natural disasters, motor vehicle accidents (MVAs) and industrial accidents, routinely exhibit marked sleep disruption, specifically longer latency to sleep onset, markedly increased nocturnal awakenings, and decreased global ratings of sleep quality compared to before the traumatic event [10]. Greater degree of exposure to the traumatic event has been shown to be related to greater distress and symptomatological impact posttrauma [11].

Varela et al. [12] investigated the psychological impact of the September 7, 1999 earthquake in Athens one year after the event in 305 exposed individuals. The main consequence was sleep disturbance, with 54% of the subjects experiencing significant sleep problems. Within this group, 90% experienced clinically significant insomnia, and 25% reported nightmares. The degree of stress and perception of lack of control after the earthquake was a significant predictor of the resultant insomnia.

Askenasy and Lewin [13] surveyed individuals during missile attacks in the Gulf war and followed them after the war was over. 38% of the sample developed acute insomnia that persisted for several months after the attacks. Lavie et al. [14] found that missile attacks during the Gulf War resulted in significantly increased nighttime awakenings in a random sample of 200 Israeli adults and children, compared to the incidence of sleep disturbance in a sample of Israeli industrial workers in 1981. Taken together, these studies demonstrate that traumatic events are important precipitants of clinically significant insomnia.

Moreover, insomnia often emerges after a severe traumatic event independent of the development of PTSD. McMillen et al. [15] interviewed 130 survivors of the 1993 Northridge California earthquake and found that, at three months, 13% of the sample met full criteria for PTSD. However, 48% of the otherwise psychiatrically healthy sample met full criteria for the hyperarousal and reexperiencing symptom clusters, and the most prevalent hyperarousal symptoms were sleep disturbance and exaggerated startle. Further, insomnia may differentially co-occur with symptoms of the hyperarousal cluster, and not the other symptoms clusters (i.e., emotional numbing, behavioral avoidance) that are manifested in full blown PTSD. Importantly, comorbidity with PTSD or any other psychiatric conditions that existed prior to the earthquake was associated only with those subjects reporting avoidance and numbing (cluster C) and poor concentration (cluster D) following the earthquake, and not hyperarousal.

The significant insomnia and startle that emerged following earthquake exposure were those symptoms most closely linked to fear and stress responses that may be acutely activated following exposure to a traumatic event [16]. Interestingly, in this study nightmares were not present in the acute posttraumatic period. It is possible that nightmares may emerge after a longer interval of time where more complex emotional processing of the traumatic event has had the opportunity to occur [17], whereas insomnia may be proximally linked to, and a more immediate consequence of, acute trauma exposure [15]. However, this is not supported by other studies showing that nightmares also manifest in the immediate aftermath of trauma, and that peritraumatic nightmares often resolve [18,19].

Lewis et al. [20] found similar reports of sleep disturbance in war veterans without PTSD compared to those with PTSD. 100% of PTSD veterans and 90% of non-PTSD veterans had significant disturbances in sleep. Although the majority of sleep measures were more severe in the PTSD group, there were no differences in sleep efficiency between the non-PTSD and PTSD groups. That is, sleep efficiency was not *more* impaired in the PTSD group compared to the non-PTSD group. Importantly, 67% of the non-PTSD veteran group had sleep quality scores in the severely impaired range. Exposure to combat related traumatic events in both groups, independent of the presence of formal PTSD, may have contributed to their comparable frequency of sleep impairment. Similarly, North et al. [21] found that after the Oklahoma City bombing, approximately 70% of 182 adults exposed reported clinically significant insomnia, and hyperarousal symptoms were the most common overall.

In summary, traumatic events have been shown to be important precipitants of insomnia in exposed individuals. The acute emergence of insomnia in the aftermath of trauma can lead to sustained disruptions in sleep continuity and restorative efficacy. Trauma-induced insomnia may occur in the absence of full-blown PTSD. Finally, insomnia induced by a traumatic event often differentially co-occurs with other symptoms of hyperarousal.

Insomnia and PTSD

The development of insomnia following traumatic events has also been demonstrated to be a constitutive element of PTSD. Sleep disturbance is a fundamental and enduring complaint of PTSD patients and has been considered the hallmark of the disorder [17,22]. Studies on PTSD have documented significant subjective sleep complaints [23,24]. Further, a three month prospective study found that insomnia was the most frequently reported symptom and predicted the other symptom clusters of PTSD in a group of war veterans [25]. Disruptions in total sleep time and sleep efficiency are the subjective parameters most specifically associated with PTSD [26].

Objective polysomnographic studies of sleep disturbance in PTSD have produced mixed results [27–30]. This has been attributed to a large number of secondary factors confounding sleep study, such as considerable heterogeneity in samples across studies with respect to age, sex, time elapsed since trauma, and presence of psychiatric comorbidity [31,32]. The majority of studies have focused on the chronic phase of the disorder, and not its acute phase [33]. Numerous studies suggest PTSD patients may overestimate their actual sleep disruption, though a recent meta-analytic study found that PTSD patients actually underestimated sleep problems, and that the discrepancies between subjective and objective sleep reports in PTSD have been overstated [34].

Despite the mixed findings, a regularly observed PSG pattern in PTSD is evidence for increased arousals, awakenings and sleep fragmentation patterns. A large epidemiological study of sleep in PTSD identified significantly increased arousals from REM sleep as objectively measured by PSG [35]. Similarly, Germain and Nielsen

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