COMPREHENSIVE REVIEW

An integrated model of chronic trauma-induced insomnia

Erika M. Roberge1,2 | Craig J. Bryan1

1National Center for Veterans Studies, The University of Utah, Salt Lake City, UT, USA
2Mental Health Service, George E. Wahlen Veterans Affairs Medical Center, Salt Lake City, UT, USA

Abstract
Insomnia is the most commonly reported symptom of posttraumatic stress disorder (PTSD), with at least 70% of patients with PTSD reporting disturbed sleep. Although posttraumatic insomnia has traditionally been conceptualized as a consequence of PTSD, it is the most likely symptom to not remit following otherwise successful PTSD treatment. This suggests that the relationship between PTSD and insomnia is more complex, such that they likely share underlying pathological mechanisms and that factors non-specific to PTSD maintain chronic trauma-induced insomnia. Although several theories and hypotheses have been presented to explain the relationship between PTSD and insomnia, neurobiological and psychological models have not been integrated, thereby limiting their comprehensiveness and abilities to inform effective intervention. Further, existing models have not addressed how acute trauma-induced insomnia becomes chronic. The present review examined models of PTSD and insomnia separately, as well as existing theorized mechanisms of their comorbidity. The distinct characteristics of trauma-induced insomnia were also reviewed and presented to describe the unique clinical presentation of trauma-induced insomnia. Review and integration of the literature were used to propose an integrated model of chronic trauma-induced insomnia informed by a neuropsychobiological framework. Clinical implications and future research directions are presented and discussed.

KEYWORDS
insomnia, interdisciplinary, posttraumatic stress disorder, trauma

1 | INTRODUCTION

Posttraumatic stress disorder (PTSD) is a mental illness characterized by a combination of adverse and impairing symptoms following the experience of a traumatic event. In response to this event, affected individuals then experience a variety of clusters of symptoms, including re-experiencing of the event, avoidance of reminders, negative changes to thinking patterns and mood, and hyperarousal and reactivity (American Psychiatric Association [APA], 2013).

Current clinical practice guidelines strongly recommend cognitive behavioural therapy, cognitive processing therapy, cognitive therapy, and prolonged exposure therapy for the treatment of adult patients with PTSD based on the continuously growing number of randomized controlled trials that attest to their feasibility and effectiveness (American Psychological Association, 2017). However, despite their general effectiveness (often on PTSD and depression symptom severity), 10–25% of individuals are left with ongoing clinical concerns and/or functional impairment after treatment completion (Larsen et al., 2019; Larsen, Fleming, & Resick, 2019). Attempts to identify predictors of nonresponse, dropout, and/or variability in treatment response that would elucidate the limitations of our recommended psychotherapies are relatively new areas of scientific inquiry and have yielded inconsistent findings (Roberge, Weinstein, & Bryan, 2019). Yet, a consistent finding is that insomnia-like sleep disturbance often persists and continues to cause significant distress after individuals have otherwise effectively responded to PTSD treatment (Pruiksm...
et al., 2016; Schnurr & Lunney, 2018; Zayfert & DeViva, 2004), even though many no longer report trauma-related nightmares (Pruiksma et al., 2016). Insomnia has been referred to as the “hallmark symptom of PTSD” (Lydiard & Hamner, 2009), with an estimated 70–91% of civilian and military samples reporting co-morbid PTSD and insomnia symptoms (Neylan et al., 1998; Ohayon & Shapiro, 2000). Trauma-induced insomnia may also perpetuate symptoms observed in PTSD (e.g., irritability, difficulty concentrating, and low mood) and impact quality of life. Indeed, several authors have suggested that insomnia in the acute phase following trauma is a significant risk factor for subsequent PTSD development (Kobayashi & Mellman, 2012; Koren, Arnon, Lavie, & Klein, 2002; Wright et al., 2011). Additionally, this co-morbidity has been associated with increased risk for death by suicide (Pigeon, Britton, Ilgen, Chapman, & Conner, 2012). Early intervention of trauma-induced insomnia may reduce patients’ risk of developing fully developed PTSD or other negative outcomes such as chronic insomnia, depression (Sinha, 2016), or suicidal thinking and behaviours (Britton, McKinney, Bishop, Pigeon, & Hirsch, 2019). Therefore, trauma-induced insomnia is an imperative target for clinical attention (Krakow et al., 2007; Pigeon & Gallegos, 2015). The astounding prevalence of this co-morbid presentation begs the question: To what extent do these distinct diagnoses share underlying pathological mechanisms? Although the answer to this question remains unclear (DeViva, Zayfert, & Mellman, 2004), multiple mechanisms and models have been presented. Review of these models will be amongst the primary goals of the current paper.

Several theoretical mechanisms and models have been proposed to explain this clinical presentation, yet none have been integrated or discussed within the context of one another. Therefore, careful examination and integration of existing proposed mechanisms are warranted. Additionally, the current pool of literature on this topic, while plentiful, suffers from several limitations. For example, although several controlled trials have assessed the efficacy of various treatments on reduction of insomnia in PTSD (see Ho, Chan, & Tang, 2016 for meta-analysis), few have investigated the mechanisms by which these treatments work, and none have dismantled components of therapies that could work via multiple mechanisms. Further, although several authors have provided reviews of this literature (e.g., Germain, Buyssse, & Nofzinger, 2008; Pigeon & Gallegos, 2015; Pigeon & Mellman, 2005; Spoormaker & Montgomery, 2008) and proposed models of trauma-induced insomnia (i.e., Germain et al., 2008; Sinha, 2016), the inclusion and integration of psychological constructs and theories have been sparse. This is problematic, as psychological factors such as maladaptive cognitions, avoidance, nightmares, and behavioural conditioning have demonstrated significant impacts on both PTSD and insomnia and play unique roles in the maintenance of trauma-induced insomnia in addition to hyperarousal. Therefore, in order to fully conceptualize the nature of trauma-induced insomnia, contributions of both neurobiological and psychological factors must be integrated.

This review will first review models of PTSD and insomnia separately, as to provide a basis for the theorized mechanisms. Next, the literature that has evaluated the distinct symptoms and presentation of trauma-induced insomnia will be discussed. Then existing proposed mechanisms will be presented and analysed in order to inform an integrated model that will be presented. Clinical implications and future research directions will then be discussed.

2 | MODELS OF PTSD

The two predominating psychological models of PTSD, social cognitive theory and emotional processing theory, each conceptualize the incidence and maintenance of PTSD in distinct but similar ways. Both are rooted in cognitive theory (Beck, 1970), which asserts that thoughts (cognitions), emotions, physiology, and behaviours all dynamically interact with each other; that is, when encountering a benign situation, an individuals’ cognitive appraisal of the situation will determine their emotional, behavioural, and physiological reaction to the situation, and any of these reactions may change as cognitive appraisals change.

2.1 | Psychological models

Social cognitive theory (Resick & Monson, 2006) asserts that following a traumatic experience, PTSD can result if existing negative belief systems are reinforced by the trauma or, more often, if the traumatic experience is discrepant with existing belief systems. Cognitive dissonance may result, and distorted cognitions are created to reconcile discrepant beliefs and experiences (e.g., “I think I am a good person, but I believe that bad things only happen to bad people. Something bad happened to me, therefore I must be bad.”). Additionally, prior negative beliefs/expectations reinforced by trauma (e.g., “My childhood experiences have taught me that adults in authority are not trustworthy. The sexual abuse I suffered at the hands of my drill sergeant prove this belief is true”) may contribute to feelings of
helplessness and further cognitive distortions. According to social cognitive theory, PTSD symptoms are theorized to be maintained by the influence of these distorted cognitions on emotions, such that the distressing emotions experienced by PTSD patients are often in response to their new trauma narrative and associated distorted cognitions and not in response to the actual event. Such cognitions may frequently inform beliefs and behaviours that serve to maintain heightened arousal and defensiveness (e.g., Belief: “The world is not safe. I must always be on guard to protect myself.” Behaviour: frequent checking/monitoring to ensure safety. Consequence: difficulty relaxing enough to sleep well). The intervention associated with social cognitive theory, cognitive processing therapy (CPT), addresses these dysfunctional cognitions with cognitive restructuring (Resick, Monson, & Chard, 2017). Indeed, neuroimaging research has demonstrated improvements in negative affect (Webb, Miles, & Sheeran, 2012) and engagement of brain regions necessary for executive functioning (e.g., attention, inhibition, and working memory; Buhle et al., 2014; Messina, Bianco, Sambin, & Viviani, 2015) in response to cognitive restructuring. Buhle et al. (2014) performed a meta-analysis of neuroimaging studies of cognitive restructuring and concluded that this skill allows individuals to change their evaluations of emotional stimuli, which leads to decreased amygdala activity. Such changes are associated with reduction of dysfunctional behaviours and remission of PTSD symptoms.

Foa and Kozak’s (1985, 1986) emotional processing theory is centralized around Lang’s conceptual framework of a fear image (Lang, 1977, 1984). A fear image is a belief system that includes a feared stimulus, an expected response, and cognitions about the relationship between the stimulus and response. For example, someone with combat-related PTSD may fear tunnels (stimulus) because they experience anxiety in these situations (response), and they believe that tunnels cause them anxiety (cognition). Although fear images can be adaptive, pathological ones include distortions of reality and cause impairment, primarily via avoidance of safe situations of the fear image and via cognitive distortions in processing of the fear image (Foa, Huppert, & Cahill, 2006). Avoidance and cognitive distortions are thought to maintain PTSD symptoms by preventing the acquisition and incorporation of information into the fear image that was created by the traumatic experience, thereby preventing emotional processing. Thus, emotional processing is the treatment target of prolonged exposure (PE) and achieved via imaginal and in vivo exposures, as well as processing the memory (Foa, Hembree, & Rothbaum, 2007). Recent systematic reviews have examined indicators of emotional processing during PE and concluded that belief change, between-session habituation, fear activation/inhibitory learning, and interpersonal engagement are the targets that facilitate reduction of PTSD symptoms in PE (Brown, Zandberg, & Foa, 2019; Cooper, Clifton, & Feeny, 2017). Therefore, restructuring of dysfunctional beliefs and activation of the fear network to enable new learning (i.e., addressing avoidance behaviours) appears to be crucial to effectively reduce symptoms of PTSD. Yet, even after otherwise successful treatment of PTSD symptoms with gold-standard treatments like CPT and PE, hyperarousal symptoms, primarily insomnia, generally persist (Pruksma et al., 2016; Schnurr & Lunney, 2018; Zayfert & DeViva, 2004).

2.2 | Neurobiology of PTSD

Examining neurobiological correlates of PTSD has been of much interest to scientists over the past several decades, and neuroscience research has explored mechanisms such as genetic influences, fear conditioning, memory consolidation deficiencies, and epigenetic considerations. Each of these mechanisms have been reviewed in depth by Ross et al. (2017), but their review is not within the scope of the present manuscript. However, dysregulated brain circuitry implicated in the fight-or-flight response, which was discussed as a primary mechanism underlying PTSD by Ross and colleagues, is relevant to the present discussion of trauma-induced insomnia.

Amongst several changes to the DSM-5 classification and description of PTSD was the re-categorization of PTSD into the new Trauma-and Stressor-Related Disorder category, from its previous categorization as an anxiety disorder. This reconceptualization of PTSD as a stress-related disorder suggests that the stress system is involved in its incidence. Many organisms, including humans, have two coordinating stress response systems, the sympathetic-adrenal medullary (SAM) system and the hypothalamus-pituitary-adrenal (HPA) axis that coordinate the fight-or-flight response. The fight-or-flight response is an adaptive, automatic, biological response that facilitates evasion/avoidance of danger or harm that is activated upon the perception of threat. Although the activation of the fight-or-flight response is adaptive in scenarios involving acute threat, the HPA axis can remain chronically activated or easily mobilized, as long as threat continues to be perceived. Therefore, situations that are not actually threatening (such as sitting in traffic or giving an important talk in front of a large crowd) may initiate or maintain cortisol secretion (Andrews, Ali, & Pruessner, 2013). Emerging research in the field of human social genomics suggests that subjective perceptions of conditions not only affect our thoughts (which influence brain networks), but they also impact gene expression (for review, see Slavich & Cole, 2013), and brain circuits that process stimuli and regulate behavioural responses and cognitions may be altered as a result (Morilak & Sandi, 2017). While this has important physiological and health-related consequences, this point is important in understanding trauma induced-insomnia to understand how these disorders are not only initiated but maintained, which is in part by cognitive factors.

Dysregulated SAM and HPA systems have been popular topics of scientific inquiry in attempts to understand PTSD. Limited evidence supports the hypothesis of a dysregulated SAM system; however, sufficient (although not wholly consistent) evidence suggests that the HPA axis may be dysregulated in PTSD (Ross et al., 2017). The most common finding in this regard is that individuals with PTSD have lower thresholds for cortisol secretion (Ross et al., 2017) and greater response to triggers that cause an exaggerated response and prolonged recovery time (Southwick, Vythilingam, & Charney, 2005). During the fight-or-flight response, medial prefrontal cortex (mPFC)
activity decreases. The mPFC is believed to play a role in decision-making and memory consolidation (Euston, Gruber, & McNaughton, 2012) and has been shown to play a role in anxiety in animals (e.g., Lacroix, Spinelli, Heidbreder, & Feldon, 2000). Upon recognition that the acute threat has ended, the mPFC communicates with the amygdala (an area of the brain that plays a central role in fear response regulation, Kessler, 2010; arousal; and sleep promotion, Dong, Wellman, Yang, & Sanford, 2012) to allow the sympathetic and parasympathetic nervous systems to resume baseline functioning. However, studies of patients with PTSD have shown disruptions in this reciprocal relationship, such that the amygdala remains easily activated (Liberzon & Sripada, 2007), whereas the mPFC remains suppressed following trauma (Shin et al., 2004). This dysregulation then leads to deficits in acknowledgement that threat is no longer present and, therefore, prolonged stress system activation or chronic hyperarousal/low threshold for arousal.

Together, psychological and neurobiological models indicate that traumatic events can elicit psychological (e.g., changes in belief systems, strong emotions, and behaviours; Resick & Monson, 2006) and neurobiological (e.g., initiation of the fight-or-flight response, dysfunction of the mPFC, and amygdala; Shin et al., 2004) reactions. Because only a subset of people who experience trauma develop PTSD, a mediating factor must explain the maintenance of dysfunctional thoughts, feelings, behaviours, and physiology following exposure to a traumatic event. Consolidation of neurobiological and psychological models would suggest that altered decision-making and memory consolidation and increased reactivity (i.e., impaired mPFC-amygdala functioning) promote dysfunctional thoughts (e.g., mislabeling benign situations as dangerous and overestimating likelihood of risk; Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003; Cisler & Koster, 2010), and behavioural avoidance promotes such thoughts by inhibiting learning (Foa & Kozak, 1985, 1986). When paired with dysfunctional beliefs, inhibition of learning impedes restoration of appropriate mPFC and amygdala functioning and, therefore, maintains symptoms of PTSD (Sripada, Garfinkel, & Liberzon, 2013). This suggests that a reciprocal relationship between dysfunctional beliefs and observed neurobiological dysfunction exists. As a result of this reciprocal relationship increased reactivity, fear, worry, and dysfunctional thoughts in turn, cause disturbed sleep.

Although acute reactions to trauma explain the onset of trauma-induced insomnia, such factors do not explain the persistence of disturbed sleep (i.e., chronic trauma-induced insomnia) after successful treatment of PTSD symptoms. In order to explore additional possible mechanisms, the biobehavioural process of sleep and models of insomnia will be reviewed.

3 | BIOBEHAVIOURAL PROCESS OF SLEEP

Sleep is defined as a discrete state characterized by the lack of alertness and consciousness, as well as absence of memory and lack of behavioural responses to stimuli (Ogilvie, 2001). In other words, sleep is a state of low arousal. In normal circumstances, a reciprocal relationship between sleep and wakefulness exists, such that as one is inhibited, and the other is promoted (Saper, Chou, & Scammell, 2001). This understanding of the biobehavioural process of sleep has led to the reconceptualization of insomnia from a disorder of sleep to a disorder of 24-h hyperarousal (Perlis, Merica, Smith, & Giles, 2001; Bonnet & Arand, 1997). When conceptualizing insomnia as a disorder of hyperarousal, the first major clue at a shared underlying mechanism between insomnia and PTSD is illuminated, as hyperarousal is a major component of PTSD.

Typical healthy adults generally enter the sleep state through Stage 1 of non-rapid eye movement (NREM) and progress through two subsequent stages prior to their first REM cycle. The sleep cycle (NREM into REM) is then repeated throughout the night until waking. Stages of NREM sleep (Stages 1-3) roughly correspond to a continuum of depth of sleep or threshold for arousal. Stage 1 is characterized as the shallowest stage of sleep or the stage in which the lowest level stimulus would be required to cause waking. Stage 3, often referred to as “deep sleep,” has the highest arousal threshold (Carskadon & Dement, 2017). Most dreaming is believed to occur during REM sleep, and individuals who wake during REM are more likely to vividly remember their dreams (Siegel, 2017). Although much basic research has been conducted on REM sleep, the purpose of REM sleep remains unclear, as popular theories about its function, such as playing an important role in memory consolidation (Walker & van der Helm, 2009), have limited and mixed evidence (Siegel, 2017).

3.1 | Neurobiology of insomnia

Although a wealth of knowledge has accumulated to classify normal sleep, the neurological foundation of disturbed sleep, or insomnia, is not as well developed (Nardo, Högborg, Jonsson, Jacobsson, & Hällström, & Pagani, M., 2015); however, it is clear that electroencephalogram (EEG) patterns differ between normal sleepers and insomnia patients (Basta, Chrousos, Vela-Bueno, & Vgontzas, 2007; Noftinger et al., 2004). Differences in EEG patterns and observed metabolic differences suggest that insomnia patients experience higher levels of arousal during wake and sleep. Additionally, robust evidence indicates that the amygdala plays a significant role in the regulation of arousal and sleep (Dong et al., 2012).

3.2 | Behavioural models of insomnia

Until recently, conceptualizations of insomnia were limited to two behavioural models: Stimulus control (Bootzin, 1972) and the 3P behavioural model of insomnia (Spielman, Caruso, & Glovinsky, 1987). The stimulus control model was included as a part of the 3P behavioural model, and both remain influential; however, these models have been further built upon with the incorporation of neuroscience research. Spielman’s model proposed the interaction of predisposing, precipitating, and perpetuating factors (i.e., the “three P’s”) to explain how insomnia originates and becomes chronic. Predisposing
factors include risk factors across the biopsychosocial spectrum (e.g., family history of insomnia, anxiety proneness, and shift work) that are generally stable over time (Perlis, Shaw, Cano, & Espie, 2010). Precipitating factors are stressors that initiate sleep disturbance (e.g., divorce), and perpetuating factors are behaviours that are intended to compensate for sleep loss, but paradoxically increase risk for sleep disturbance, primarily wakefulness/arousal in the bedroom, and excessive time in bed. First discussed by Bootzin’s stimulus control model, engaging in nonsleep activities in the bedroom and staying and excessive time in bed. First discussed by Bootzin’s stimulus control model, engaging in nonsleep activities in the bedroom and staying in bed while awake are believed to create a conditioned association between the bedroom and wakefulness. This association then promotes disturbed sleep, which persists until the association is broken.

3.3 | Neurocognitive model of insomnia

The neurocognitive model (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997) is an extension of the 3P model of insomnia. The role of hyperarousal in both the initiation and maintenance of insomnia is central in this model; specifically, cortical hyperarousal is presumed to be fundamental to the pathophysiology of insomnia and is then maintained by classical conditioning. Further, this model asserts that increased sensory and information processing during sleep initiation and NREM sleep causes sleep-state misperception and are responsible for sleep initiation and maintenance problems (Perlis et al., 2010).

4 | CHARACTERIZING INSOMNIA IN PTSD

Insomnia has been referred to as the “hallmark symptom” of PTSD (Ross, Ball, Sullivan, & Caroff, 1989) and is unlikely to resolve even after PTSD symptoms naturally reduce over time (Pigeon, Campbell, Possemato, & Ouimette, 2013). But what is the nature of sleep impairment in individuals with PTSD? Although trauma-induced insomnia shares many similarities with primary insomnia (Pigeon & Gallegos, 2015), there also appear to be unique sleep pattern characteristics in this population.

Characterization of the qualities of trauma-induced insomnia is a goal that has been approached by numerous researchers since the late 1970s. Although findings vary throughout the literature, several findings have been replicated. Specifically, more Stage 1 sleep, less slow wave sleep, and increased REM density (in other words, less restful sleep; Pigeon & Gallegos, 2015; Spoormaker & Montgomery, 2008) have been repeatedly observed and largely uncontested. Additionally, REM abnormalities such as increased arousals from REM sleep, decreased REM time (Breslau et al., 2004; Mellman, Nolan, Hebding, Kulick-Bell, & Dominguez, 1997; Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002; Mellman, Pigeon, Nowell, & Nolan, 2007), and increased REM density (Mellman et al., 1997) have been reported. Although some (i.e., Germain & Nielsen, 2003; van Liept et al., 2013) did not observe differences in REM sleep or other sleep architecture variables, as compared with the selected control groups, the samples in each of these studies were fairly small. A meta-analysis of sleep impairment in PTSD reported small to medium effects for above average REM density and Stage 1 sleep, as well as below average slow-wave sleep in PTSD (Kobayashi, Boarts, & Delahanty, 2007). Together, these findings suggest that increased Stage 1 and less deep sleep and REM disturbances are characteristics of sleep problems in PTSD (Kobayashi et al., 2007; Pigeon & Mellman, 2005; Spoormaker & Montgomery, 2008).

Others have observed increased sleep latency (e.g., Calhoun et al., 2007; Krakow et al., 2007) and wake after sleep onset (e.g., Mellman et al., 1997; van Liept et al., 2013), less sleep time (e.g., Krakow et al., 2007; Mellman et al., 1997; Mellman, Kulick-Bell, Ashlock, & Nolan, 1995), and decreased sleep efficiency amongst PTSD patients, a finding that has been observed using multiple methods (e.g., self-report, actigraphy, and polysomnography) (e.g., Calhoun et al., 2007; Germain & Nielsen, 2003; Krakow et al., 2007; Mellman et al., 1997; van Liept et al., 2013). Others have reported more frequent awakenings, microawakenings, and restlessness (e.g., Breslau et al., 2004; Calhoun et al., 2007; Germain & Nielsen, 2003; Mellman et al., 1995; van Liept et al., 2013; ). Germain and Nielsen (2003) reported that awakenings mediated the relationship between PTSD and decreased sleep efficiency, and van Liept et al. (2013) found a moderate negative association between number of awakenings and perceived sleep depth. Although several of these sleep characteristics have been replicated by numerous studies, these findings have been inconsistent (Sinha, 2016) and not verified by meta-analytic procedures, which suggests that whereas some sleep characteristics are shared between individuals with PTSD, others may vary, with sleep fragmentation likely one of the shared sleep disturbance characteristics of trauma-induced insomnia.

4.1 | Novel intermediate state

Additional insights into the nature of trauma-induced insomnia may be gleaned from animal research. The rodent model of acute stress-induced insomnia (Cano, Mochizuki, & Saper, 2008) outlined the neurobiological factors in insomnia following an acute stressor. Data informing this model originate from rodent research using a cage-exchange stressor paradigm that activates the fight-or-flight response and, as a result, prolonged disruption of sleep. Interestingly, rats acutely exposed to the stressor were observed to experience paradoxical increased activation of both arousal and sleep-promoting systems. Activation of the arousal system during NREM sleep was named the novel intermediate state. This finding has been replicated in human patients with primary and secondary insomnia (e.g., Buysse et al., 2008; Perlis, Smith, Andrews, Orff, & Giles, 2001; Spiegelhalder et al., 2012). Perlis and colleagues (2010) suggested that during trauma-induced insomnia, the sleep-promoting system functions as usual due to unaffected homeostatic and circadian drives, but inhibition of the arousal system during sleep initiation does not occur because of the effect of stress on the cortical and limbic systems, resulting in the simultaneous activation of arousal and sleep-promoting systems. Simultaneous activation of these systems may
explain the ease and frequency of awakenings and associated sleep fragmentation observed in and reported by patients with PTSD.

While the specific nature of sleep complaints associated with PTSD may vary between people (i.e., increased sleep latency, less sleep time, and wake after sleep onset), similarities in sleep architecture appear to be more consistent. That is, REM disturbance (Kobayashi et al., 2007; Pigeon & Gallegos, 2015), impaired deep sleep (Kobayashi et al., 2007), and increased awakenings characterize sleep disturbance in PTSD (Sinha, 2016) and may be explained by the novel intermediate state, which is created by dysregulated arousal systems.

5 | EXISTING MODELS AND PROPOSED MECHANISMS OF TRAUMA-INDUCED INSOMNIA

The topic of co-morbid PTSD and insomnia is a popular one that has been theoretically and empirically addressed across scientific disciplines including psychology, medicine, and neuroscience. From these distinct fields of study, several divergent hypotheses to explain the incidence of trauma-induced insomnia have been proposed. Although each of these hypotheses has several unique properties, they share several common elements. Review of the proposed mechanisms suggests that they are more complimentary than distinct, in that they may influence one another to fully articulate the mechanisms for trauma-induced insomnia. Unfortunately, models that have integrated findings across fields or theoretical mechanisms are lacking. Existing models and theoretical mechanisms will be presented before they are integrated into the proposed model of chronic trauma-induced insomnia.

5.1 | Behavioural model of insomnia extended to PTSD

Spielman's 3P model of insomnia (1987) easily extends to understanding sleep disturbance in PTSD and serves as the general foundation of the present model. Not only does a traumatic experience serve as a precipitating factor of acute insomnia by introducing an increased state of cognitive and/or physiological arousal, but insomnia is also a predisposing factor for development of PTSD (Ho et al., 2016; Roberge, Williams, Heron, & Bryan, 2016; Sinha, 2016; Walker & van der Helm, 2009). Further, like those with insomnia and without co-morbid psychiatric diagnosis, patients with PTSD engage in perpetuating factors of their insomnia (e.g., staying in bed while unable to sleep and attempts to make up for sleep loss by napping). Specific perpetuating factors to consider in this population include fear of sleep that may be introduced by nightmares, fear of the dark, and intrusive thoughts in bed (Inman, Silver, & Doghramji, 1990). Some of these factors may be potentially salient for patients who were traumatized in their bed and/or while sleeping. Specifically, fear of sleep may represent broader anxiety about the potential consequences of loss of vigilance/control/compromised safety while asleep, which may create sleep behaviours and thoughts that are counterproductive to sleep onset and maintenance (Craske & Tsao, 2005).

5.2 | Hyperarousal

Hyperarousal, or a state of heightened physiological and psychological stress that can be experienced cortically, cognitively, and somatically, is one of four symptom clusters of PTSD (APA, 2013). Patients with insomnia experience both physiologic and cognitive arousal that make sleep difficult and are risk factors for the onset and maintenance of insomnia (Perlis, Smith, & Pigeon, 2005). Importantly, various manifestations of hyperarousal have been hypothesized as the mechanism that causes sleep disturbance in PTSD and underlie existing frameworks.

A well-supported vehicle for hyperarousal in traumatized patients is dysfunction in the amygdala and mPFC pathway (Germain et al., 2008). Combined with findings from neurobiological models of fear conditioning, PTSD, and sleep–wake regulation, Germain et al.'s (2008) neurobiological hypothesis of PTSD contends that altered amygdala and mPFC activity are central to increased activity of wake-promoting regions, as well as reduced sleep promotion (consistent with the novel intermediate state), which causes REM disruption that further promotes dysfunction of the amygdala-mPFC pathway. Similarly, Sinha's model of trauma-induced insomnia (2016) also describes the progression from trauma to observed sleep impairment through hyperarousal mechanisms. In this model, trauma activates the acute stress response via activation of the amygdala, reticular activating system, and prefrontal cortex, which contributes to HPA axis and sympathetic nervous system activation along with increased startle that cause sleep fragmentation, disturbed deep sleep, microarousals, and REM sleep disruption (trauma-induced insomnia); that is, Germain and colleagues' and Sinha's models are largely consistent, whereas Sinha's may be viewed as more comprehensive. However, neither of these models explains how acute trauma-induced insomnia becomes chronic.

Additionally, although these models provide an excellent foundation for understanding the neurobiological components of trauma-induced insomnia, neither incorporate the roles of other posttrauma symptoms (e.g., nightmares, avoidance, and dysfunctional cognitions) or important psychological factors (e.g., behavioural conditioning) into their explanations of the origin or maintenance of trauma-induced insomnia. The roles of avoidance (emotional processing theory; Foa & Kozak, 1985, 1986), dysfunctional cognitions (cognitive processing theory; Resick & Monson, 2006), and behavioural conditioning (stimulus control model; Bootzin, 1972) have been reviewed above, and discussion of the roles of nightmares and cognitive arousal (which have not been integrated into larger theoretical models) follows.

5.2.1 | Cognitive arousal

Cognitive arousal, or preoccupation with thoughts (prior to sleep onset in the case of insomnia), has been theorized to play a role in
sleep-state misperception (Perlis, Merica, et al., 2001) that is common in individuals with PTSD, as well as subjectively and objectively measured primary insomnia (Pilai, Steenburg, Ciesla, Roth, & Drake, 2014). Such worries may be sleep-specific, (e.g., “I am not going to be able to sleep again tonight and tomorrow is going to be horrible”) or in the context of PTSD, beliefs about the necessity of vigilance to maintain safety (e.g., “If I do not maintain awareness of my surroundings, someone will break into my house and kill me”; Pietrzak, Morgan, & Southwick, 2010). Cognitive arousal has been cited by patients with insomnia as the primary barrier to sleep initiation (Lichstein & Rosenthal, 1980) and has shown strong correlations with both perceived and observed sleep onset latency in good sleepers (Wicklow & Espie, 2000), suggesting that cognitive arousal may be a cause, rather than effect, of insomnia (Tang & Harvey, 2004).

5.3 | Nightmares

Symptoms of PTSD have significant overlap with a number of other psychological disorders, but one commonly experienced symptom that is unique to PTSD is trauma nightmares (Mellman et al., 1995; Neylan et al., 1998). In addition to experiencing nightmares at significantly higher rates than patients without PTSD (Krakow et al., 2001), PTSD sufferers’ nightmares appear to be unique, in that trauma nightmares generally consist of negative memories of actual experiences—a dream quality that is specific to PTSD (Ross et al., 1989). Although some PTSD nightmares have been observed in Stages 1 and 2 sleep, they most often occur during REM sleep (Pigeon & Mellman, 2005; Stickgold, 2005). Therefore, nightmares may account for the more frequent awakening observed during REM sleep. The impact of nightmares on sleep is intuitive: Disturbing nightmares are often associated with waking in terror and cause hyperarousal, thereby impairing ability to return to sleep until arousal has decreased. Nightmares may also cause the sufferer to choose to stay awake to prevent another nightmare, which may contribute to sleep fragmentation and poorer sleep.

Relationships between nightmares and prolonged sleep latency increased awakenings, early morning awakening, and nonrestorative sleep have been consistently reported (as summarized by DeViva et al., 2004), and some have reported that nightmares explain unique variance in PTSD symptoms, independent of insomnia (DeViva et al., 2004). Although treatment of trauma nightmares may generalize to improvements in sleep quality (Lancee, Spoormker, Krakow, & van den Bout, 2008), this effect is likely explained by the reduction in sleep fragmentation that can be partially attributed to nightmares, which translates to improved sleep efficiency (DeViva et al., 2004; Zayfert & DeViva, 2004). What appears more likely is that a reciprocal relationship between nightmares and insomnia exists. Specifically, sleep deprivation has been shown to cause REM rebound—an increase in REM sleep following REM deprivation, which, in turn, causes more intense dreaming (Pigeon & Gallegos, 2015). Therefore, as PTSD patients accumulate more and more sleep debt, their risk for experiencing nightmares is increased, which promotes sleep fragmentation and reduces sleep efficiency, thereby creating a feedback loop into increasing risk for trauma nightmares. Additionally, the prevalence of trauma nightmares is approximately half of the prevalence of sleep disturbance in PTSD, leaving substantial variance in insomnia and PTSD symptoms unexplained by nightmares and suggesting that nightmares are not the sole mechanism by which sleep is disturbed in patients with PTSD.

5.4 | Summary

Several complementary mechanisms of sleep disturbance in PTSD, as well as existing models, have been presented and reviewed. Significant evidence exists to support the hyperarousal theory, and this theory is central to models proposed by Germain et al. (2008), as well as by Sinha (2016). However, existing models have not addressed the role of nightmares, classical conditioning, cognitions, or behaviours in the maintenance of trauma-induced insomnia. Therefore, integration of such psychological variables and associated research is necessary to obtain a holistic understanding of chronic trauma-induced insomnia. Without a complete conceptualization of the problem, effective intervention is unlikely, and adverse outcomes associated with prolonged insomnia (e.g., suicide) can be expected. This assertion is supported by the fact that treatments that conceptualize hyperarousal as the sole mechanism of sleep disturbance (e.g., sleep medications, prazosin, and ganaxolone [a synthetic derivative of a GABAergic neuromodulator associated with amygdala activity and dorsomedial prefrontal cortex-amygdala connectivity; Rasmusson et al., 2017]) are not sufficiently effective in treating trauma-induced insomnia (Kung, Espinel, & Lapid, 2012), suggesting that additional mechanisms are at play. However, results from a meta-analysis indicate that cognitive behavioural treatments for insomnia (which primarily target deconditioning of associations between bed and arousal) have large effects on trauma-induced insomnia (Ho et al., 2016). Yet, existing cognitive behavioural treatments of nightmares and insomnia may be necessary but not sufficient for treatment of trauma-induced insomnia because they are non-specific to PTSD/sleep disturbance following trauma exposure and fail to integrate neurobiological mechanisms that clearly play a role in PTSD. Together, the research suggests that components from each of these theories must be integrated. Integration of psychological theories and constructs (i.e., conditioning and maladaptive cognitions) may fill the gaps of existing neurobiological hypotheses and models to create a more comprehensive description of the onset and maintenance of trauma-induced insomnia. In light of such an advancement, more effective interventions may be created and applied.

6 | INTEGRATED MODEL OF CHRONIC TRAUMA-INDUCED INSOMNIA

The integrated model of chronic trauma-induced insomnia is illustrated in Figure 1. The 3P model of insomnia informs the underlying structure of the integrated model of trauma-induced insomnia.
Predisposing factors are theorized to interact with most paths in this model, such that those without predisposing factors will be at decreased risk for any of the stages/consequences following trauma and therefore at reduced risk for experiencing trauma-induced insomnia. Risk factors associated with increased risk of trauma-induced insomnia include, but are not limited to, abnormal GABA functioning, genetic vulnerabilities (Sinha, 2016), early and/or chronic stress exposure (Germain et al., 2008), and disturbed sleep preceding the traumatic event (Ho et al., 2016; Roberge et al., 2016; Sinha, 2016; Walker & van der Helm, 2009).

The onset of the acute phase of trauma-induced insomnia is explained by Germain’s neurobiological model of PTSD (2008) and Sinha’s model of trauma-induced insomnia (2016); that is, acute trauma-induced insomnia is precipitated by a traumatic event that elicits the fight-or-flight response and dysfunction of the amygdala and mPFC. This dysfunction is characterized by increased sensitivity of the amygdala to threat and decreased ability of the mPFC to regulate threat perception. As predicted by social cognitive theory (Resick & Monson, 2006) and emotional processing theory (Foа & Kozak, 1985, 1986), in addition to other risk factors, dysfunctional thoughts and avoidance behaviours are proposed to mediate the relationship between the immediate trauma response and dysregulated neurobiological systems. Such thoughts and behaviours are further proposed to have a reciprocal relationship with this impaired pathway, as impaired mPFC and amygdala functioning negatively affects cognitive appraisals (i.e., neutral stimuli and situations are more likely to be interpreted as dangerous) and behaviours (i.e., fear of stimuli is associated with avoidance of such stimuli), aids in the development and maintenance of maladaptive trauma-related cognitions (e.g., “The world is dangerous”), and maintains cognitive arousal. Distorted thoughts and avoidance behaviours also increase risk of trauma-related nightmares, as sleep becomes an opportune time for a dysregulated mind to process a traumatic event. Further, the hyperarousal associated with dysfunctional amygdala and mPFC functioning contributes to heightened 24-h activity of arousal systems and partial inhibition of sleep-promoting systems and instigates acute trauma-induced insomnia.

Acute trauma-induced insomnia is uniquely characterized by REM disturbance (i.e., increased arousals during REM sleep, decreased REM time, and increased REM density), reduced deep sleep (i.e., less restorative sleep), and sleep fragmentation. These sleep characteristics are associated with REM rebound, which increases the frequency and intensity of dreaming (Pigeon & Gallegos, 2015) and impairs sleep. Therefore, REM rebound is a catalyst for the reciprocal relationship between nightmares and acute trauma-induced insomnia.

Following the onset of acute trauma-induced insomnia, sleep- and PTSD-specific perpetuating factors maintain sleep disturbance and lead to chronic trauma-induced insomnia (Short, Allan, Stentz, Portero, & Schmidt, 2018). Sleep-specific factors include those in Spielman’s 3P model of insomnia, including conditioned arousal, paradoxical compensatory strategies (e.g., substance use), poor sleep hygiene, and dysfunctional beliefs about sleep. PTSD-specific perpetuating factors include fear of sleep (secondary to nightmares, loss of vigilance/ability to maintain safety while sleeping), fear of the dark, intrusive thoughts in bed (Inman et al., 1990), safety-related behaviours (e.g., checking behaviours and weapons in the bedroom), and nightmares. Therefore, trauma-induced insomnia will persist and become chronic after amelioration of PTSD symptoms if these perpetuating factors are still present.
The proposed model of chronic trauma-induced insomnia is a result of integration of literature across multiple disciplines. Review and consolidation of several meta-analyses, systematic reviews, and empirical studies allowed for characterization of unique characteristics of trauma-induced insomnia. Additionally, this model is consistent with and builds on Germain et al.'s (2008) neurobiological hypothesis of PTSD and Sinha's (2016) model of trauma-induced insomnia. The present model was further developed via incorporation of several psychological (e.g., social cognitive theory, emotional processing theory, and 3P model of insomnia) and neurocognitive (Perlis et al., 1997) theories and proposed specific sleep- and PTSD-related factors that explain how acute trauma-induced insomnia becomes chronic. Via the integration of neurobiological and psychological findings, a more comprehensive description of the characteristics and maintaining factors of trauma-induced insomnia with significant clinical implications and directions for future research investigations has been proposed.

### 7.1 | Clinical implications

This model lends itself towards clinical application, as a number of targets of intervention are hypothesized. Primarily, assessment of the stage of insomnia the patient is experiencing (i.e., acute or chronic) and the role of nightmares in contributing to sleep disturbance are proposed to be vital to determination of the appropriate treatment strategy. For acute stages of trauma-induced insomnia, hyperarousal, dysfunctional thinking, and avoidance behaviors should be primary targets of treatment. Evidence-based treatments for PTSD (e.g., CPT or PE) may be considered the first-line intervention to address major cognitive and behavioral components that maintain a certain level of hyperarousal that impairs sleep. A specific adaptation that may improve sleep outcomes in these treatments is to directly target sleep-related cognitions and fear images in treatment (e.g., challenge worries about reduced vigilance and safety at night, process the meaning of a traumatic event occurring at night). Empirical evidence for such an approach is provided by observed improvements in brain regions associated with cognitive processing and arousal in patients with PTSD, social anxiety disorder, and major depressive disorder who received cognitive behaviour therapy (Aupperle et al., 2013; Månsson et al., 2013; Ritchey, Dolcos, Eddington, Strauman, & Cabeza, 2011). Additionally, targeted reduction of physiological hyperarousal is also likely indicated and may be treated with pharmaceutical methods, relaxation strategies, mindfulness (Crawford, Talkovsky, Bormann, & Lang, 2019), exercise (Babson et al., 2015), and/or cognitive restructuring. Interestingly, mindfulness-based stress reduction (MBSR) has demonstrated significant reductions in self-reported PTSD symptoms, attention, depression, emotion regulation, and general hyperarousal symptoms in various PTSD patient populations (Boyd, Lanius, & McKinnon, 2018); however, no trials of MBSR in patients with PTSD have reported on the effects on sleep disturbance. Therefore, this is a promising area for future research investigation. Patients whose nightmares do not remit following PTSD treatment may also benefit from treatments specifically designed to address them (e.g., Imagery Rehearsal Therapy, Exposure, Relaxation, and Rescripting Therapy, prazosin). For patients whom have chronic trauma-induced insomnia (i.e., those whose insomnia is, in part, behaviourally conditioned) will likely benefit from cognitive behaviour therapy for insomnia (CBT-I), which directly targets behavioural conditioning of insomnia and works to re-regulate biological systems that influence sleep (e.g., circadian rhythm). Indeed, others have suggested that CPT is ineffective in reducing sleep disturbance despite otherwise moderate to large effects on PTSD symptoms, because behavioural learning mechanisms are not addressed (Haynes et al., in press). Results of a meta-analysis of sleep-specific cognitive behavioural treatments in traumatized populations revealed significant reductions in insomnia severity (Ho et al., 2016), which suggests that targeted intervention of behavioural maintain factors is necessary (that is, in addition to cognitive factors) to address trauma-induced insomnia.

Further, as is standard of practice when treating anyone with insomnia-related complaints, careful assessment of co-morbid conditions may be of increased importance with patients with trauma-induced insomnia. Specifically, screening for sleep-disordered breathing conditions such as obstructive sleep apnoea or upper airway resistance syndrome is particularly important. Krakow et al. (2007) reported that approximately 20% of patients reporting posttraumatic sleep disturbance met criteria for sleep-disordered breathing. The impact of sleep-disordered breathing on sleep patterns is similar to those observed in PTSD, as these conditions both cause fragmented sleep, and nightmares were more commonly reported by those with posttraumatic sleep disturbance and sleep-disordered breathing. Untreated sleep apnoea is associated with grave health conditions such as type 2 diabetes, stroke, heart attacks, and shortened life span (Johns Hopkins, n.d.). Therefore, accurate diagnosis (insomnia vs. sleep-disordered breathing) is necessary for effective intervention and prevention of possible side effects of untreated sleep-disordered breathing.

Further, the present review and Sinha's (2006) review and model both imply there appears to be limited accuracy in discussing the phenomenon of trauma-induced insomnia as one unique to PTSD, and therefore, patients who do not meet criteria for PTSD may experience trauma-induced insomnia and benefit from the associated interventions. Readers are encouraged to remember that the complexities of the stress system and brain networks are not restricted to the same nosology of our clinical disorders, and mechanisms of clinical disorders are not unique to a singular constellation of symptoms. Rather, mechanisms that involve stress system dysfunction will impact a variety of symptoms and disorders that operate via this mechanism. Therefore, when assessing a patient with sleep disturbance, clinicians should carefully evaluate the role of the precipitating event in the presentation and maintenance of insomnia. For patients whose precipitating events do not appear to play a significant role in their chronic insomnia, CBT-I and/or pharmaceutical sleep aids are indicated; however, patients whose trauma plays a role in the presentation and
maintenance of their insomnia (e.g., by fear associated with reduced vigilance, and nightmares) may better be conceptualized through the present model.

7.2 | Future research directions

Despite substantial investment of empirical research into exploring and delineating the mechanisms of chronic trauma-induced insomnia, much further work is needed. First, preliminary evidence from cross-sectional and prospective studies suggest that impaired sleep at the time of a traumatic event increases risk for several consequences of trauma, including PTSD (Sinha, 2016; Sotres-Bayon, Bush, & LeDoux, 2004; Roberge et al., 2016; Walker & van der Helm, 2009). Importantly, fMRI research has observed significant reductions in functional connectivity of the amygdala and mPFC following only one night of sleep deprivation (Sotres-Bayon et al., 2004). Further, REM sleep deprivation following fear-inciting conditions has been shown to impair memory consolidation and next-day memory retention in animals (as summarized by Walker & van der Helm, 2009). Such impairments are central to Foa and Kozak’s (1985, 1986) emotional processing theory of PTSD. Therefore, impaired sleep prior to and/or following stress exposure may represent a modifiable risk factor for populations with more predictable stress/trauma exposure such as military personnel and first responders (Roberge et al., 2016) or a prevention target for traumatized individuals immediately following their traumatic event. However, it is not clear if quality sleep prior to or in the days following traumatic event exposure serve a protective role. Future research should evaluate the longitudinal effects of trauma on individuals with good sleep prior to and following trauma and investigate the effects of purposefully protecting sleep quality in acutely traumatized individuals (e.g., by prescribing sleep aids immediately after trauma exposure).

Another area requisite of further investigation is examination is the full effect and consequences of attenuated mPFC functioning in PTSD, specifically, the impact on cognitive flexibility, influence on interpretation of neutral stimuli, and learning. Two gold-standard PTSD treatments cite dysfunctional thinking and impaired extinction learning as the primary targets of intervention (i.e., CPT, Resick & Monson, 2006; PE, Foa et al., 2007). However, although the mPFC is understood to play a critical role in attention and decision-making, its role in the cognitive processes central to popular conceptualizations of PTSD has not been empirically studied.

Additionally, future research studies should carefully select their comparison groups and consider the limitations of various sleep measurement methods. Although theoretical models of PTSD and the DSM distinguish trauma from stress and PTSD from subthreshold symptomatology, neurobiological models of PTSD would not predict the same distinctions regarding the impact of stress on sleep because stress systems activation is normal (and adaptive), and hyperarousal (not all PTSD symptoms) appears to be the primary mechanism underlying this problem. Comparison of individuals with trauma-induced insomnia with those with primary insomnia, but without significant trauma history, may provide the best group to assess factors and mechanisms that are specific to insomnia caused by trauma.

Further methodological advancements, such as increased use of multiple subjective and objective measures of sleep, as well as consideration about what divergent results between methods might mean, are necessary. Additionally, use of ambulatory PSG or actigraphs to study trauma-induced insomnia would be helpful, as well as comparison of findings with traditional PSG in order to empirically test behavioural models of insomnia (i.e., is the sleep complaint restricted to the patient’s bed?). Such methods are more cost-effective and provide higher ecologically valid results (i.e., patients are not in a new environment). Multiple measures of hyperarousal should also be used to reflect cognitive arousal, functional neurological arousal, neuroendocrine variables, and biological proxies of arousal (e.g., heart rate and perspiration). Use of symptom cluster scores from the hyperarousal and reactivity section of self-report PTSD scales may not be appropriate, as these symptoms are not specific indicators of hyperarousal (e.g., risk-taking behaviours and angry outbursts).

Finally, although the majority of the proposed integrated model of trauma-induced insomnia has a strong empirical basis and is informed by existing theories, this model has yet to be tested and should be rigorously evaluated in patients of all ages, genders, with different trauma histories, and degrees of posttraumatic stress. Perhaps the most crucial components of the model to evaluate are the factors proposed to instigate trauma-induced insomnia, that is, amygdala and mPFC dysfunction in the presence of dysfunctional thoughts and avoidance behaviours, as well as the responsibility to chronic trauma-induced insomnia to sleep-specific treatments (e.g., CBT-I) and mindfulness-based approaches (e.g., MBSR).

8 | SUMMARY

Chronic trauma-induced insomnia is a common experience of individuals whom have suffered traumatic experiences and/or from PTSD. Although several effective treatments of PTSD and insomnia exist, no effective treatment for trauma-induced insomnia has yet to be disseminated. A primary factor limiting the development of such a treatment is likely the lack of integration of multidisciplinary knowledge about trauma-induced insomnia. Without the fields’ experts communicating with one another or considering complementary findings and/or interventions outside of their areas of study, the condition of trauma-induced insomnia is unlikely to be completely understood or effectively treated. An integrated model of trauma-induced insomnia across numerous fields of study has been presented to facilitate communication across disciplines, incite research investigations, and to inspire a multimethod approach to treatment.

ACKNOWLEDGEMENT

We would like to thank Brian Curtis for lending his expertise and providing consultation about the theoretical model of chronic trauma-induced insomnia.
CONFLICT OF INTEREST
The authors have no conflicts of interest to disclose.

ORCID
Erika M. Roberge https://orcid.org/0000-0001-9102-7193

REFERENCES


