

An Exploration of Fear of Sleep and Experiential Avoidance in the Context of PTSD and Insomnia

Symptoms

by

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DISSERTATION ABSTRACT

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Fear of sleep (FoS) has been posited to develop following trauma exposure and significantly contribute to the maintenance of insomnia symptoms. While FoS has been operationalized within the Fear of Sleep Inventory- Short Form (FoSI-SF), preliminary examinations of the measure have yielded diverging factor structures. Experience avoidance (EA), a trait-based measure of avoidance implicated in PTSD and insomnia symptomatology, is thought to be conceptually akin to FoS and may be an important foil to clarify the unique contributions of the construct in trauma-induced insomnia. In the present study, the psychometric properties of the FoSI-SF were evaluated in a population of college students ($N = 197$), including the underlying factor structure, convergent validity with EA as well as discriminant validity with sleep hygiene, another sleep-related process implicated in insomnia. A conceptual model of FoS was investigated within a subsetting sample ($n = 50$) that had clinically-significant PTSD and sub-threshold insomnia symptoms. An exploratory factor analysis revealed the following three-factor structure: (1) fear of loss of control and/or vulnerability (FoSI-V); (2) fear of darkness (FoSI-D); and (3) fear of re-experiencing traumatic nightmares (FoSI-N). The FoSI-SF was found to have convergent validity with EA, but did not display discriminant validity with sleep hygiene. The FoSI-V and FoSI-N were significantly predicted by trauma-related hypervigilance and nightmares, respectively. Analyses indicated that FoS was a more robust predictor of PTSD and insomnia symptom severity than EA. Theoretical implications of the findings were discussed to guide future research into the role of FoS in trauma-induced insomnia.

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DEDICATION

To those who saw me, believed in me, and gave me the confidence to use my voice.

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Chapter 1: The Impact of Experiential Avoidance in Comorbid PTSD and Insomnia

Experiential avoidance

EA is the exacerbation of psychological distress due to avoidance of internal discomfort (e.g., thoughts, emotions, physical sensations). The construct has been historically represented in various theoretical orientations, including psychodynamic therapy, client-centered therapy, and Gestalt therapy (Hayes et al., 1996). More recently, cognitive-behavioral therapies (CBTs) have sought to reduce psychological distress through the modification of aversive internal experiences and their antecedents (e.g., cognitive restructuring; Beck, 1979; Deacon et al., 2011). Additionally, the treatment of maladaptive forms of avoidance is at the heart of third-wave therapies, such as dialectical behavior therapy (DBT; Linehan, 2018; Linehan et al., 1991) and acceptance and commitment therapy (ACT; Hayes et al., 1996; Hayes et al., 1999). In contrast with CBTs, ACT and DBT promote acceptance of unwanted internal experiences, rather than explicitly altering or avoiding them as a primary mechanism of alleviating distress. Similarly, exposure-based techniques seek to increase the willingness to come into contact with unwanted internal experiences (e.g., anxiety, chronic pain; Abramowitz et al., 2019; Vlaeyen et al., 2012). Given the consistent representation across therapeutic systems, maladaptive avoidance of internal distress may be a transtheoretical construct with significant intervention utility. However, modern EBTs for common psychiatric disorders remain only 44-66% effective in achieving remission, so there is still room for improvement (Morina et al. 2014; de Matt et al., 2007; Jonas et al., 2013). The current efficacy gap could be closed by more clearly conceptualizing, and explicitly addressing maladaptive avoidance in the context of common psychiatric disorders.

First conceptualized by Hayes et al. (1996) within the framework of ACT (Hayes et al., 1999; Hayes et al., 2006), EA is the unwillingness to remain in contact with private (i.e., internal) experiences, such as traumatic memories, painful thoughts, overwhelming emotions, and uncomfortable physiological

sensations, as well as attempts to avoid or modify the frequency/form of the private experiences or the contexts in which they occur. The operationalization of “avoidance” in EA extends to explicit cognitive, emotional, and behavioral attempts to avoid or alter the unwanted private experience, which may include dissociation, distraction, suppression, and/or control of the environment. Since the short-term consequences of enduring an unwanted private experience are salient (Hayes et al., 1996), the immediate benefits of EA are reinforced despite the paradoxically amplified long-term consequences. For example, an individual may actively suppress a painful thought in the moment to reduce short-term discomfort, only to find that the same thought increases in frequency and associated distress over time (Magee et al., 2012). While EA can temporarily provide relief from the source of distress, an individual is inadvertently conditioned to use the same strategy over larger time scales despite the cumulative negative impact on various domains of their life (Chawla & Ostafin, 2007). EA consequently constricts the range of possible behaviors that will not produce the aversive private experience, such that an individual may see their world shrink (e.g., social withdrawal). As a result of the reinforced behavioral pattern, EA appears to be effective in reducing distress, even though it is not only ineffective long term, but also deleterious (Hayes et al., 2011). By avoiding unwanted internal experiences, EA also serves to maintain belief in the feared outcome as reality (e.g., “If I interact with others, I will be hurt”) rather than seeing it as just a thought. In summary, individuals engaging in EA tend to overestimate both the cost of sitting with unwanted internal experiences (e.g., “I am overwhelmed by my thoughts”) and the effectiveness of various avoidance strategies (e.g., thought suppression; Sheppes, Suri, & Gross, 2015).

EA encompasses a broad topography of behaviors due to the emphasis on context and function rather than content and form. Distress that leads to use of EA strategies is a consequence of how an individual relates to their private experience, rather than the inherent nature of the private experience itself. For instance, EA has been shown to predict subjective distress associated with an unpleasant physiological

experience (i.e., pain) independent of the severity of the experience (Zettle et al., 2005). As a result, current conceptualizations of EA include the following six domains: behavioral avoidance, distress aversion, procrastination, distraction/suppression, repression/denial, and distress endurance (Gámez et al., 2011). Behavioral avoidance and distress aversion were determined to be the two most representative subscales of EA as a construct among a population with depression and anxiety disorders (Gámez et al., 2011), though the remaining four subscales remained significant during factor analysis. Behavioral avoidance encompasses both overt and passive avoidance of situations that provoke distress related to private experiences (e.g., “I’m quick to leave any situation that makes me feel uneasy”). Situational avoidance is a common maladaptive strategy seen in anxiety disorders (White et al., 2006), depression (Trew, 2011), and post-traumatic stress disorder (PTSD; Asmundson et al., 2004). Distress aversion captures a non-accepting attitude and/or evaluative stance toward discomfort related to private experiences (e.g., “Pain always leads to suffering”; Gámez et al., 2011). Negative judgment towards private experience may increase the associated distress and unacceptability (e.g., anxiety about your anxiety; Hayes et al., 1996), and consequently reinforces the unwillingness to engage with the private experience. The remaining four domains constitute other specific methods of engaging in EA, as well as measuring the degree to which present distress prevents values-congruent behavior. By adopting a functional contextualism lens, the utility of EA can be expanded considerably to various forms of psychopathology.

EA has also been identified as a transdiagnostic process that underlies a number of behavioral disorders, such as PTSD (Walser & Hayes, 2006) and insomnia (Zetterqvist et al., 2011). Therefore, EA may be a prime target for cross-cutting treatments, which could alleviate distress among individuals with varied and comorbid presentations. Existing CBTs for PTSD, such as cognitive processing therapy (CPT) and prolonged exposure therapy (PE), seek to reduce behavioral avoidance of trauma-related stimuli by

modifying maladaptive/inaccurate cognitions (CPT; Resick & Schnicke, 1992) and encouraging emotional processing (PE; Foa & Kozak, 1986; McLean & Foa, 2011). Distress aversion and other aspects of EA may not be satisfactorily captured within the therapeutic focus of CBTs for PTSD, and require additional attention. For example, distress aversion has been operationalized to assess metacognitive attitudes toward psychological distress (Gámez et al., 2011), which are not explicitly targeted in CBTs for PTSD. There is a growing body of research in support of ACT, which has been specifically designed to ameliorate EA, as an effective treatment for PTSD (Orsillo & Batten, 2005). Furthermore, insomnia in PTSD may be uniquely driven by contextually-based distress aversion to sensations of loss of control and safety (Werner et al., 2021). However, research focused on the links between various presentations of insomnia (i.e., primary, comorbid) and experiential avoidance are currently scarce (Dalrymple et al., 2010). Given the current state of the literature, there is a real opportunity for better understanding and characterizing the role of EA in comorbid PTSD and insomnia with the intention of informing treatment approaches. In the present review, we will first outline the manner in which EA is independently implicated in both the etiology and maintenance of PTSD and primary insomnia. Next, we will integrate findings from the aforementioned sections to elucidate the unique presentation of EA in comorbid PTSD and insomnia, as well as residual insomnia. Then, we will examine current empirically-supported treatments for PTSD and insomnia, and identify gaps in which EA-focused interventions may provide added benefit in reducing global distress. Finally, we will integrate empirical findings from the review to highlight the clinical implications and discuss possible future directions for the field.

Experiential avoidance in PTSD

Guiding behavior based on an implicit or explicit belief that particular internal experiences must be avoided may leave trauma survivors in an ever-deepening stuckness in their lives. Since EA may be at the heart of this suffering, there is much to gain from understanding its role in the development and

maintenance of PTSD. The disorder develops after exposure to significant trauma, and can be characterized by the presence of several symptom clusters, including re-experiencing, avoidance, changes to cognition and mood, and hyperarousal (Sareen, 2014). Given the intense, often overwhelming nature of unwanted internal experiences in PTSD, EA is an expected contextually-relevant response that often becomes pathological over time. EA may also contribute to the development (Kumpula et al., 2011; Gil, 2005; Thompson et al., 2018), maintenance (Orcutt et al., 2014), as well as symptom severity of PTSD (Thompson & Waltz, 2010; Meyer et al., 2013; Tull et al., 2004; Plumb et al., 2004; Bardeen, 2015). For example, in a cross-lagged study design, Kumpula and colleagues (2011) examined the impact of the Northern Illinois University shooting on pre-trauma EA and peritraumatic dissociation in a large sample of university students. Baseline EA prospectively predicted the development of post-traumatic symptoms one month after the traumatic event. EA at one-month post-traumatic event further predicted intrusion (e.g., distressing memories) and dysphoric (e.g., feelings of detachment from others) symptoms at the eight-month follow-up. Incredibly, baseline EA significantly predicted the maintenance of chronic post-traumatic symptoms 30 months following the shooting (Orcutt et al., 2014). In a separate study of university students, Gil (2005) identified trait avoidant coping as a significant predictor for the development of PTSD following a terrorist explosion. Thompson and Waltz (2010) further characterized EA as an important process in predicting post-traumatic symptom severity. Therefore, EA may not only lead to the development of PTSD following a traumatic event, but also significantly contribute toward the intensity and long-term entrenchment of symptoms.

Following a traumatic experience, individuals often seek to avoid painful reminders of the event or nullify associated emotions (Badour et al., 2012). Chronic EA serves to reinforce and maintain the perceived unacceptability of certain private experiences, and limit new opportunities to receive corrective feedback (Thompson & Waltz, 2010). For example, a combat Veteran who socially isolates themselves due to

concerns of safety is unable to have new, safe experiences in a social context. Therefore, EA serves to not only perpetuate trauma symptoms (Pineles et al., 2011), but may also worsen distress and impact across multiple domains in a person's life (Brockman et al., 2016). Due to the over-corrective nature of EA, fear habituation (i.e., learning the probability that fear predicts danger) and subsequent trauma recovery are severely disrupted, if not prevented altogether. Coping strategies that are in service of EA often lead to ineffectual, long-term suffering, and immediate negative reinforcement in the short-term (e.g., avoiding discomfort associated with feelings of shame; Brem et al., 2018). Since EA strategies have also been directly implicated across all symptom clusters within PTSD (Walser & Westrup, 2007; Seligowski et al., 2016), there is utility in more clearly characterizing the nature of the construct.

While acute psychological distress is inevitable in the wake of trauma, prolonged suffering may not be as predetermined. Chronic distress sustained within the context of PTSD may be due in part to an adversarial relationship with internal experiences as traumatic symptoms are re-experienced in the present. According to the DSM-5, Criterion B encompasses various ways in which trauma is re-experienced in the present, including: memories, nightmares, dissociative reactions, psychological distress, and physiological reactivity in response to trauma-related stimuli (American Psychiatric Association, 2013). Traumatic stress places immense strain on the ability to interface with private experiences, such that an individual may become overwhelmed (i.e., experiencing too much) or numb (i.e., experiencing too little; Center for Substance Abuse Treatment, 2014). Since internal responses to traumatic symptoms may be innate (e.g., feelings of fear; Lanius et al., 2017), the decision to engage in EA as a means to reduce short-term distress is often reflexive. Due to the perceived unacceptability of intrusion symptoms, EA becomes a default solution to a complex problem. As a result, an individual becomes cued to and compelled by intrusion symptoms to engage in ineffectual coping strategies over time, which further entrenches the symptoms. For example, EA has been found to moderate the

relationship between PTSD severity and substance dependence (Bordieri et al., 2014), which highlights the insidiousness of avoidance strategies. Substance dependence may be conceptualized as a functional sequelae of EA, where uncomfortable internal experiences (e.g., shame) are temporarily avoided through numbing, distraction, and/or dissociation (Levin et al., 2012). EA strategies are explicitly represented in the operationalization of Criterion C, which involves the avoidance of internal (e.g., thoughts, memories) and/or external reminders (e.g., people, places) of trauma-related stimuli (American Psychiatric Association, 2013). Distilling the essence of this symptom cluster, there are marked efforts to escape unwanted private experiences whether born of the mind or from the environment. For example, a rape survivor may persistently avoid the location in which they experienced their trauma, even if doing so is a significant inconvenience. Consequently, functional impairment becomes a necessary cost to escape re-experiencing traumatic thoughts, memories, and emotions. Since both the internal and external environment are viewed as potentially dangerous, avoidance of psychological distress becomes even more salient. EA coping strategies, such as distraction, are significantly predictive of increased PTSD severity (Scarpa et al.; Gil, 2005), and may also mediate the relationship between past trauma and current psychological distress (Orcutt et al., 2005). Furthermore, the avoidance cluster is predictive of not only meeting full diagnostic criteria for PTSD (Nemeroff et al., 2006), but also overall PTSD symptom severity (Boeschen et al., 2001; Marshall et al., 2006; Marx & Sloan, 2005).

Criterion D symptoms are defined by the onset or worsening of distressing thoughts and emotional affect following a traumatic event (American Psychiatric Association, 2013). The symptom cluster, including anhedonia, estrangement from others, emotional numbness, and memory loss, has been found to be significantly associated with PTSD onset (Nemeroff et al., 2006) and symptom severity (O'Bryan et al., 2015). For instance, an individual may begin to hold the belief "I'm a bad person," which then becomes inextricably connected to painful emotions (e.g., shame, anger) and physical sensations (e.g., tension,

heart palpitations). As a result, essential beliefs about the self can quickly become a wellspring for suffering and subsequent avoidant behavior. EA can become ingrained as a means to inefficiently disregard painful thoughts about the self, others, and the world that arose due to the trauma. Over time, negative cognitions in PTSD generalize to non-trauma related contexts (Ehlers & Clark, 2000), which may lead to more profound impairment and distress across multiple domains. Fear generalization is common when the trauma survivor believes the experience to have broad implications in their life. Additionally, cognitive overgeneralization in PTSD has been found to predict worse treatment response (Ready et al., 2015). The incorporation of trauma-related beliefs into irrelevant contexts becomes problematic when individuals begin to seek respite via EA. Unfortunately, avoidant coping reinforces the connection between trauma-related cognitions and psychological distress, which may then maintain the chronicity of PTSD (Badour et al., 2012). Consequently, internal experiences related to the traumatic event become viewed as uncontrollable and unacceptable due to perceived psychological distress (e.g., “I shouldn’t feel this way”). Moreover, emotional components of PTSD, such as feelings of social estrangement and inability to experience positive emotions, may also be etiologically based in EA strategies. For example, a Veteran with the thought “I cannot trust other people/other people will hurt me” will engage in social avoidance due to the associated psychological distress (e.g., anxiety) and lack of pleasure (i.e., anhedonia), despite the importance of social connection in their life. Pervasive avoidance of close relationships has devastating consequences in that an individual with PTSD may not only lose access to social support as a protective factor, but also unintentionally strengthen the perceived threat (Gerhart et al., 2014). Furthermore, EA has been shown to mediate the relationship between PTSD symptoms and post-deployment social support among Veterans (Kelly et al., 2019), which further highlights the cyclic stuckness of distress avoidance.

Criterion E symptoms are characterized by marked reactivity across physiological systems, including hypervigilance, exaggerated startle response, and sleep disturbances, and may also be closely tied to EA. There is evidence to suggest self-regulation via emotional non-acceptance is significantly associated with increased hyperarousal symptoms (O'Bryan et al., 2015). Since hyperarousal symptoms cue use of avoidance strategies, fear related to trauma is not extinguished and may actually increase vulnerability in the development of PTSD (Foa et al., 1992). Furthermore, hyperarousal symptoms significantly predict emotional numbing, a common form of dissociative avoidance, above and beyond other PTSD symptom clusters (Tull & Roemer, 2003). Reffi and colleagues (2019) recently established a significant negative association between PTSD hyperarousal symptoms and present-focused attention, an anti-correlate of EA. In other words, efforts to modify the locus of attention in EA appear to underlie increased hyperreactivity. A Veteran may engage in hypervigilance following an unexpected sound in their environment as a means to control their psychological distress related to safety. Scanning the environment for potential threats redirects attention to external stimuli, and reinforces implicit beliefs that internal experiences (i.e., fear) must be removed through safety behaviors and always indicate actual danger. In PTSD, hypervigilant individuals have an immediate attentional bias to trauma-related stimuli, though they also appear to paradoxically engage in behavioral avoidance within a few seconds of presentation (Thomas et al., 2013). Neuroimaging research has indicated that intentional labeling of internal experiences diminishes amygdala reactivity in response to negatively-valenced imagery (Torre & Liberman, 2018). Therefore, EA is diametrically opposed to effective emotion regulation, especially in the context of trauma-related hyperarousal.

Since EA appears to be closely implicated in both the etiology and chronicity of PTSD, clinical interventions may increase efficacy by explicitly addressing this detrimental process. However, many current therapeutic frameworks primarily seek to alleviate PTSD symptoms through the modification

and/or desensitization of unhelpful trauma-related internal experiences. In other words, CBTs for PTSD address the content of internal experiences, rather than the context in which the internal experiences occur. While there is a wealth of research supporting the efficacy of CBTs for PTSD (Bradley et al., 2005), the gold-standard interventions typically encounter high dropouts among participants, particularly among non-responders (Scottenbauer et al., 2008). Subsequently, there remains a sizable proportion of individuals who continue to experience chronic PTSD despite receiving empirically-supported interventions. Since CBT interventions may achieve a reduction in syndrome symptomatology through changing the form of internal experiences, there is an implicit and evaluative stance of unacceptability being reinforced. Unfortunately, individuals who could most benefit from reassessing the relationship with their internal experiences are instead further conditioned to believe that they should be removed. For example, an individual who successfully learns to restructure trauma-related cognitions cannot prevent the unhelpful version of the cognitions from being experienced altogether. Since they have maintained a non-accepting stance towards their trauma-related internal experiences, the thought may be experienced with the same intensity of psychological distress in the future after therapy has terminated. In comparison, a similar individual who does not engage in evaluation of their internal experiences may experience reduced subjective distress, despite the similar symptom profile (Bullis et al., 2014). Therefore, treatment modalities that incorporate an explicit focus on alleviating EA may have significant utility in reducing both current and long-term psychological well-being.

Experiential avoidance in insomnia

Insomnia is operationalized as an inability to initiate or reinitiate sleep onset, and/or experience restorative, restful sleep leading to subsequent daytime impairment when afforded sufficient sleep opportunity (Roth, 2007; Seow et al., 2018). A profoundly common and impairing disorder (Buysse et al., 2006), the consequences of insomnia are far reaching and emblematic of a public health crisis (Schutte-

Rodin et al., 2008). The disorder has been strongly linked with functional impairments in numerous areas of quality of life, including mental well-being, occupational, and interpersonal relationships (Ancoli-Israel & Roth, 1999; Bastien et al., 2004; Katz & McHorney, 2002), as well as an increased risk for all-cause mortality (Parthasarathy et al., 2014). Insomnia is not only one of the most frequently diagnosed sleep disturbances (Mai & Buysse, 2008), but also inextricably implicated in the presentation of psychiatric disorders (Ancoli-Israel, 2006). While insomnia symptoms are self-reported in 50-80% of adults diagnosed with a psychiatric disorder in a given year (Sivertsen et al., 2021; Smith et al., 2002), the holistic impact of the sleep disturbance is much more detrimental than being written off as a comorbid collateral. Pre-existing insomnia symptoms have been identified as both a significant risk (Hertenstein et al., 2019; Wright et al. 2011) and causal factor (Gao et al., 2019) in the development of comorbid psychopathology. Therefore, insomnia has both an etiologically- and temporally-complex relationship with the onset of psychopathology (Harvey, 2001; Morin et al., 2015). Given that the development of insomnia symptoms and psychopathology is not uniform, the etiology of the sleep disturbance may be founded in stress dysregulation.

While the etiology of insomnia remains largely multifactorial, research has highlighted the deleterious interplay of stressors and subsequent sleep reactivity (Drake et al., 2006; Kalmbach et al., 2018). Sleep reactivity has been operationalized as the propensity to experience sleep disturbances (e.g., difficulty initiating, maintaining, or reinitiating sleep) in response to on-going stressors (Drake et al., 2014). In a diathesis-stress model of insomnia, individuals with increased sleep reactivity are more vulnerable to later meeting diagnostic criteria for insomnia disorder, as compared to individuals who have more sleep-related resilience (Griffiths & Pearson, 2005). Recent research has also indicated an underlying genetic and epigenetic vulnerability in the onset of insomnia disorder (Palagini et al., 2014; Dauvilliers et al., 2005). Furthermore, individuals diagnosed with primary insomnia disorder are prone to heightened

cortical activity and cognitive-emotional reactivity (Baglioni et al., 2010) as well as suppressed parasympathetic activity (Bonnet & Arand, 2003) in response to stress. Pre-sleep hyperarousal has also been identified as a mediating factor in the relationship between daytime distress and perceived sleep quality (Morin et al., 2003; Winzeler et al., 2014). Therefore, the progression of transient sleep disturbances to chronic insomnia disorder may be significantly influenced by a premorbid vulnerability to sleep disruptions rooted in physiological systems and exposed by acute stressors. As a moderating factor, EA may then destabilize less resilient phenotypes during stress dysregulation and serve to entrench insomnia via maladaptive responses to hyperarousal.

EA may underlie a series of converging cognitive, affective, and physiological factors in the maintenance and aggravation of insomnia, providing insight into the persistence of symptoms even after initial acute stressors have dissipated. Insomnia disorder can be characterized by exaggerated pre-sleep arousal (Harvey et al., 2014; Ellis et al., 2012) and high sleep reactivity (Palagini et al., 2018), which can be more finely delineated into primary and secondary levels of arousal. Primary arousal encompasses problematic cognitive activity that is explicitly implicated in difficulty initiating or reinitiating sleep onset, and includes safety behaviors, dysfunctional sleep-related beliefs, as well as uncontrollable worry and rumination (Riemann et al., 2010; Perlis et al., 1997). Secondary arousal involves the metacognitive attitudes (i.e., “thinking about thinking”), emotional valence, and closeness with which an individual holds onto their primary arousal (Ong et al., 2012). In other words, primary arousal is due to the content of cognitive activity, whereas secondary arousal is invoked by the way in which an individual relates to their cognitive activity in the context of sleep. Primary arousal frequently activates secondary arousal, especially among individuals with chronic insomnia. Consequently, a significant number of individuals diagnosed with insomnia disorder maintain their symptomatology for years after initial onset (Jansson & Linton, 2006; Mendelson, 1995).

Conceptualizations of primary arousal have been ubiquitous in insomnia research for the last several decades (Bonnet & Arand, 1997; Lundh & Broman, 2000), with a focus on the role of cognitive arousal in perpetuating insomnia. For example, Harvey (2002) conceptualized “excessive negatively-toned” cognitions as a central component in the maintenance of insomnia disorder, which are posited to initiate a cascade of hyperarousal and distress, narrow attentional focus on perceived sleep-related threats, and cause a discrepancy in perceived impairment due to sleep loss. Individuals with insomnia disorder have a propensity to ruminate excessively about their difficulty sleeping (e.g., “I’ll never be able to get to sleep feeling so anxious”; Carney et al., 2010; Lancee et al., 2017) and catastrophize the subsequent daytime impairment (e.g., “I won’t be able to get any work done tomorrow, since I’ll be too tired”; Carney et al., 2013). Dysfunctional sleep-related beliefs (e.g., “I must get eight hours of sleep or I won’t be able to function”) quickly develop in the wake of insomnia, which inadvertently increases cognitive and emotional reactivity (Kalmbach et al., 2018) and leads to overt safety behaviors (i.e., “I avoid talking about my sleep”; Ree & Harvey, 2004; Woodley & Smith, 2006). Safety behaviors can be characterized as intentional or unintentional behaviors intended to avoid a feared outcome (Salkovskis, 1991). While there are numerous negatively-valenced cognitive styles associated with disrupted sleep onset (Hiller et al., 2015), efforts made to escape, modify, or avoid the associated psychological distress may be the common, underlying process. Dysfunctional sleep-related beliefs quickly become paired with psychological discomfort at the primary arousal level due to the inherent unacceptability of sleep difficulty itself. For example, an individual may have the belief that they *should not* have difficulty falling asleep or avoiding talking about sleep *should* be helpful. The discrepancy between reality and their expectations then proliferates distress via emotional, cognitive, and somatic reactivity. EA is therefore embedded within the initial arousal experienced in insomnia, and may facilitate the entrenchment of acute symptoms.

With the relatively recent rise of third-wave therapies, processes underlying secondary arousal, such as metacognition, have become more of a focus in the literature (Hayes, 2009). Secondary arousal may intensify psychological distress by acting upon multiple contextual aspects of a dysfunctional sleep-related belief, such as the inflexibility/rigidity of the thought, emotional valence attached to the thought, the manner in which the thought is interpreted, and subsequent attempts to control the thought (Ong et al., 2012). In case of the thought “I must get eight hours of sleep or I won’t be able to function,” primary arousal may be first prompted due to an established association between expected daytime impairment and psychological distress. The rigidity with which an individual adheres to the thought may also govern the associated emotional valence as well as occlude the ability to recognize other, more flexible interpretations of their insomnia symptoms. EA is functionally important in efforts to fall asleep due to the power with which dysfunctional sleep-related beliefs are held among insomniacs. However, behaviors intended to initiate sleep onset often have harmful effects on overall sleep health. For example, increased sleep effort has been identified as a significant predictor of subjective insomnia severity (Hertenstein et al., 2015). Secondary arousal commonly amplifies the use of safety behaviors and includes actions such as drinking alcohol to fall asleep or reducing energy expenditure during the day due to perceived sleep-related deficit. Similar to other forms of EA, use of safety behaviors reinforces their own perceived utility in the short-term and prevents integration of new information that would otherwise disprove their actual utility in the long-term. An individual who engages in thought suppression of sleep-related worries may attribute their ability to eventually fall asleep to the use of the coping strategy, which reinforces the perceived need of the behavior in the context of sleep. Thus, the frequency of safety behaviors increases, which in turn paradoxically increases occurrence of the feared sleep outcome (Neitzert Semler & Harvey 2007). More severe presentations of insomnia disorder are also associated with more rigid dysfunctional sleep-related cognitions and consequently greater perceived utility of safety behaviors (Hood et al.,

2011). Interestingly, safety behaviors may more directly explain the severity of sleep disturbance, rather than more distal factors, such as prompting dysfunctional sleep-related beliefs (Fairholme & Manber, 2014). Therefore, chronic insomnia may not only be maintained, but also worsened by the manner in which an individual relates to their perceived sleep difficulty, rather than the specific nature of the dysfunctional thoughts. Altogether, the insidious cycle of engaging in avoidance-minded behavior to alter sleep-related distress serves to maintain symptoms of insomnia.

Experiential avoidance in comorbid PTSD and insomnia

Insomnia and PTSD individually cause significant psychological distress and functional impairment, and the intersection of the clinical disorders is even more debilitating and all too common. Insomnia symptoms are present in as many as 70-90% of individuals with PTSD (Richards et al., 2020; Talbot et al., 2014; Maher et al., 2006), and 35-61% meet DSM-5 diagnostic criteria for comorbid insomnia even after covarying for nightmares (Colvonen et al., 2018). When adjusting for PTSD, comorbid insomnia further explains a significant amount of variance for several adverse health outcomes, such as gastrointestinal distress and headaches (Clum et al., 2001). Premorbid insomnia may also predict PTSD onset (Gehrman et al., 2013; Wright et al., 2011; Bryant et al., 2010), persistence (Gilbert et al., 2015), symptom severity (Koffel et al., 2013; McKay et al., 2010), and hamper the natural process of recovery (Babson & Felder, 2010). For example, Gerhman and colleagues (2013) used a longitudinal study design to characterize the role of insomnia in the onset of post-deployment combat-related PTSD among Veterans. In the large study sample, pre-deployment sleep duration and insomnia symptoms independently predicted an increased likelihood of post-deployment PTSD development. Furthermore, military personnel who endorsed multiple forms of sleep disturbance were most likely to develop PTSD post-deployment. Additional longitudinal studies have replicated the results among Veterans, suggesting that pre-deployment insomnia significantly increases the likelihood of meeting diagnostic criteria for PTSD

following deployment (Wang et al., 2019; van Liempt et al., 2013). Conversely, insomnia may also be a sequelae of trauma exposure prior to and independent of the pathogenesis of PTSD (Sinha, 2016; McMillen et al., 2000), which emphasizes a complex, bidirectional relationship between the two clinical disorders.

The co-occurrence of insomnia and PTSD may indicate a shared underlying set of disrupted internal systems that significantly contribute to chronic impairment. Subjectively disrupted and non-restorative sleep is a core and seemingly indelible element of PTSD that has been included in the diagnostic criteria within the hyperarousal symptom cluster. Objective measures of comorbid sleep disturbances have been characterized as involving less slow wave sleep (SWS), more frequent nocturnal awakenings, and increased rapid eye movement (REM) sleep fragmentation throughout the night (Baglioni et al., 2014; Germain & Nielsen, 2003; Kobayashi et al., 2007). REM instability in the peritraumatic period is a predictor of both PTSD and insomnia (Mellman et al., 2002), and may also reflect an inability to attenuate hyperarousal during sleep (Riemann et al., 2012). As previously noted, hyperarousal is a well-established neurological substrate of insomnia that is experienced as elevated EEG activity, heart rate, and activation of the sympathetic nervous system, which altogether disrupt the continuity of restorative sleep (Bonnet & Arand, 2010). Hyperarousal symptoms appear to be a robust predictor of both general symptom severity (Schell et al., 2004) and the degree of functional impairment among individuals diagnosed with PTSD (Heir et al., 2010). Notably, hyperarousal symptoms are endemic throughout the peritraumatic period and may be a primary pathogenic determinant in the etiology of PTSD (Bryant et al., 2003; Felmingham et al., 2012). In co-occurring PTSD and insomnia, hyperarousal-induced sleep disruptions, especially leading to a reduction in REM sleep (Spoormaker et al., 2010), may further prevent extinction of fear responses following trauma exposure (Pace-Schott et al., 2009; Pace-Schott et al., 2012). Since consolidation of emotionally-salient memories occurs during both SWS and REM sleep (Van Der Helm et

al., 2011; Payne et al., 2012), fear responses emblematic of PTSD may become generalized such that the context of sleep begins to generate psychological distress in of itself (e.g., triggering traumatic symptoms; Werner et al., 2021). As a result, FoS may become a primary mechanism through which chronic insomnia is uniquely perpetuated among individuals with comorbid PTSD.

FoS encompasses dysfunctional sleep-related beliefs regarding safety and loss of control (e.g., “I’m not safe when I’m asleep”), emotional experience of fear, and subsequent avoidance strategies to mitigate the psychological distress (e.g., sleeping with television/lights on; Werner et al., 2021; Pruiksma et al., 2014; Zayfert et al., 2006). From the perspective of functional contextualism, FoS is also a multidimensional manifestation of EA due to the perceived unacceptability and/or intolerability of loss of control and safety in the context of sleep. Survivors of trauma often hold maladaptive appraisals of their traumatic experience (e.g., “It’s my fault that I was assaulted”) and the lived consequences (e.g., “I can’t return to work feeling like this”), which may lead to an attentional bias toward threat perception accompanied by an elevated fear response (Fani et al., 2012). As a result, traumatic cognitions (e.g., “I’m not safe at night”) become more closely associated with psychological distress (e.g., fear), which thereby promote safety- and control-seeking behavior. Since sleep is a context in which individuals must relinquish control of their environment, both internal experiences and efforts to reduce the associated discomfort become further exaggerated. For instance, a trauma survivor who engages in extensive hypervigilant behaviors at bedtime (e.g., scanning their bedroom) to mitigate intense feelings of fear may inadvertently begin to believe that their EA strategies are effective in upholding their own sense of safety/control. However, the sleep-interfering behaviors become incentivized in the long-term and perpetuate the belief that the trauma survivor is not safe or in control unless they utilize the EA-minded behavior again. Trauma survivors may then begin to fear the context of sleep itself due to the perceived loss of control and vulnerability, which becomes perpetually reinforced by maladaptive nighttime

behaviors (Werner et al., 2021). Therefore, the sleeping environment, rather than traumatic symptoms, will induce or exacerbate uncomfortable internal experiences, which further instigates use of ineffective EA strategies. Ultimately, EA may be a process through which trauma-induced insomnia chronically persists even after reduction of traumatic symptoms (e.g., trauma-related cognitions).

Current gaps in pre-existing therapies for comorbid PTSD and insomnia

Residual insomnia is the maintenance of clinically-significant sleep disturbances following empirically-supported treatment for a comorbid disorder (e.g., PTSD). The lingering sleep disturbances are particularly insidious in that trauma survivors may be at a heightened risk for developing future mental health complications, such as a relapse in traumatic symptoms. For example, Kartal and colleagues (2021) used a cross-lagged analysis to investigate the longitudinal association between comorbid insomnia and PTSD before, during, and following trauma-focused treatment. Residual insomnia was determined to significantly aggravate post-treatment traumatic symptoms at both the three- and nine-month follow-up. While PTSD severity can be significantly alleviated by trauma-focused treatment (Powers et al., 2010), over half of individuals continue to experience residual insomnia (Schnurr & Lunney, 2019; Zayfert & DeViva, 2004; Pruiksma et al., 2016). Following PTSD treatment, residual insomnia often persists at a clinically significant severity that is experienced as markedly distressing and impairing (Belleville et al., 2011; Galovski et al., 2009; Gutner et al., 2013). Furthermore, residual insomnia symptoms have also been found to predict an overall decreased response to PTSD treatment (López et al., 2017; Milanak et al., 2019), and are often accompanied by other hyperarousal symptoms, such as hypervigilance and exaggerated startle response (Larsen et al., 2019; Tanev et al., 2022). This body of research highlights the entrenched nature of trauma-induced insomnia, which may be a hallmark of PTSD (Germain, 2013), rather than a secondary characteristic residing within the disorder. Given the non-responsiveness of

comorbid insomnia toward most common PTSD treatments, there is clinical significance in examining current clinical approaches to identify and address gaps.

CBTs are widely considered to be the gold standard for PTSD treatment with meta-analyses demonstrating a remission of clinically significant symptoms in 56-70% of treatment completers (Bradley et al., 2005). However, common trauma interventions, such as CPT and PE, are far less effective in reducing insomnia symptoms (Gutner et al., 2013). Therefore, the direction of scientific inquiry may be most effectively guided in service of delineating putative mechanisms of change among trauma interventions to understand why insomnia symptoms do not commonly remit. Rooted in emotional processing theory (Foa & Kozak, 1986), PE aims to address the pathological manner in which (1) traumatic fear is generalized among non-traumatic stimuli; and (2) cognitive architecture of oneself, others, and the world become erroneously shifted due to trauma (Cooper et al., 2017). PE intentionally activates trauma-related fear in an effort to provide corrective information to the trauma survivor via emotional processing (Zalta & Foa, 2012). Through in vivo and imaginal exposures, a trauma survivor systematically engages with distressing traumatic stimuli to modify the content of the associated beliefs and habituate traumatic fear (Zalta, 2015). CPT seeks to directly change maladaptive trauma-related cognitions that govern subsequent emotions and behavior (Resick & Schnicke, 1992). Based on social cognitive theory, CPT posits that maladaptive cognitions may take the form of assimilation (e.g., "It's my fault that I was assaulted") and over-accommodation (e.g., "Everyone is dangerous") of trauma-related information into pre-existing cognitive schema (Gallagher & Resick, 2012). Cognitive restructuring is used to directly challenge trauma-related cognitions and guide trauma survivors toward a more balanced and adaptive set of beliefs (e.g., "Some people can be dangerous, and I can keep myself safe") to promote recovery (Price et al., 2016). While the two interventions may differ in the specific therapeutic mechanisms, PE and CPT appear to both modify the distressing and functionally impairing content related to trauma. In the

context of comorbid insomnia and PTSD, trauma-related content is implicated closely to primary arousal. For instance, the maladaptive cognition “I’m not safe at night” may prompt hypervigilance, heart palpitations, and persistent fear. While the form of the traumatic distress may be modified by CPT or PE, secondary arousal and metacognitive elements may remain intact. In other words, a trauma survivor may be able to achieve a more balanced trauma-related cognition while trying to fall asleep (e.g., “I can feel safe even after I turn my lights off”), however, the underlying relation to the belief is unchanged (e.g., “I must get rid of my anxious thoughts”). While a trauma-related belief may be somewhat more flexible, the uncontrollability or dangerousness attached to experiencing the beliefs maintains rigidity (Simons & Kursawe, 2019). Therefore, residual insomnia could be perpetuated by a trauma survivor’s metacognitive relationship to their psychological distress, even after receiving adequate treatment for PTSD (Fergus & Bardeen, 2017).

Cognitive-behavioral therapy for insomnia (CBT-I) is one of the most commonly implemented and efficacious treatments for chronic insomnia (Trauer et al., 2015). Since psychiatric comorbidity is significant among insomniacs, the first-line treatment has also been shown to be effective in reducing insomnia symptoms in the presence of PTSD in a randomized controlled trial (RCT; Talbot et al., 2014). CBT-I is devised into several core components, which include stimulus control, sleep restriction, and cognitive restructuring (Taylor & Pruiksma, 2014). Sleep restriction is a behavioral strategy in which time in bed is curtailed to increase homeostatic sleep drive, whereas stimulus control aims to reinforce the association between the sleeping environment and sleep (e.g., not watching TV in bed; Koffel et al., 2015). Cognitive restructuring in CBT-I serves to challenge and modify dysfunctional sleep-related beliefs (e.g., “I must sleep for eight hours or I won’t be able to function the next day”) that may otherwise generate psychological distress (Mitchell et al., 2012). Since the therapeutic mechanisms involve direct behavioral manipulation or change of content, CBT-I may only intervene at the level of primary arousal

among individuals with comorbid insomnia and PTSD. While CBT-I often reduces the prevalence of sleep-related dysfunctional beliefs among individuals with chronic insomnia, the beliefs themselves have not been shown to mediate improvements in insomnia severity (Okajima et al., 2014). In other words, the form of insomnia symptoms may be less relevant than the function of the underlying processes. Consequently, the way in which an individual relates to their psychological distress in the context of sleep may be an equivalently valuable source of intervention rather than the specific content or form of the symptom.

Despite the significant role of EA in the maintenance of both PTSD and insomnia, the deleterious process is not explicitly integrated into many empirically-supported PTSD treatments. While CBTs for PTSD may aim to behaviorally modify avoidance, the scope of the protocols may be too broad to adequately address the negative reinforcement between internal experiences (e.g., cognitive, emotional, and somatic hyperarousal) and the sleeping environment. As a result, there may be clinical utility in specifically targeting EA as a central process in the perpetuation of chronic insomnia. ACT is a third-wave therapy that is rooted in a cognitive-behavioral foundation, although it aims to directly alleviate EA through a shift in the relationship to internal experiences. More specifically, ACT utilizes acceptance-minded interventions to encourage individuals to drop the struggle with internal experiences and learn to coexist in service of pursuing a values-oriented life (Hayes, 2004). A recent systematic review has also provided support for the transdiagnostic effectiveness of ACT in the treatment of primary and comorbid insomnia (Salari, 2020). In this approach, the antidote to EA is not removing distressing symptoms altogether, but increasing acceptance and willingness to experience what has been habitually avoided. Therefore, a primary goal of an EA-focused intervention would be to uncouple the ineffective association between unwanted internal experiences and subsequent avoidance strategies that interfere with sleep. One such intervention to target FoS could involve the following core elements: (1) learn the costs and

ineffectiveness of engaging in EA (e.g., “No matter how hard or what I try, I cannot get rid of my fear of sleep”); (2) practice building non-judgmental awareness of distressing internal experiences (e.g., “I’m noticing feelings of fear around being vulnerable while I sleep”); and (3) allow the internal experience to be present without engaging in avoidance behavior (e.g., “My feelings of fear are just a natural experience of emotion that I do not need to get rid of”; Harris, 2019). Acceptance-focused interventions may also be able to enhance the efficacy of pre-existing insomnia treatments, such as CBT-I. For example, acceptance strategies may be utilized to mitigate experiential avoidance that is inherent in the short-term behavioral modification of stimulus control (Dalrymple et al., 2010). Therefore, alleviating EA may have clinical utility in tailoring and sequencing interventions to individuals with comorbid PTSD and insomnia, as well as residual insomnia.

Summary and future directions

The present review primarily aimed to (1) characterize the role of EA in the etiology, maintenance, and exacerbation of comorbid insomnia and PTSD; and (2) elucidate the clinical utility of EA-focused interventions for residual insomnia. By consolidating findings from the literature, EA appears to be an important transdiagnostic process independently underlying both insomnia and PTSD. Following a traumatic event, acute stress may become entrenched through implicit or explicit attempts to avoid or modify uncomfortable internal experiences (Orcutt et al., 2014). Findings further indicate that EA is heavily implicated across all PTSD symptom clusters, and serves to worsen clinical distress and impairment in the long-term (Walser & Westrup, 2007; Seligowski et al., 2016). Similarly, EA drives much of the ineffectual responses that arise during the onset of insomnia, such as dysfunctional sleep-related beliefs and safety behaviors (Hood et al., 2011; Fairholme & Manber, 2014). Furthermore, EA may be acting on both primary (e.g., distress related to the content) and secondary (e.g., metacognitive attitudes toward the content) levels of arousal in insomnia, which could explain the chronicity of the disorder (Ong

et al., 2012). In a comorbid presentation, insomnia and PTSD has been shown to have a bidirectional, causal relationship, which may indicate a shared set of disrupted internal processes (Wang et al., 2019; Sinha, 2016). Hyperarousal-induced sleep disruptions may be the catalyst through which PTSD-related fear responses are generalized to the sleeping environment (Pruiksma et al., 2014). Thus, the unwillingness of a trauma survivor to accept their distressing internal experiences may lead to a FoS itself, which serves to perpetuate chronic insomnia even after PTSD-focused treatment (Werner et al., 2021). While there are efficacious interventions for PTSD (Bradley et al., 2005) and primary insomnia (Trauer et al., 2015), residual insomnia continues to be significantly impairing and distressing in a majority of trauma survivors (Schnurr & Lunney, 2019; Zayfert & DeViva, 2004; Pruiksma et al., 2016). Since the aforementioned CBTs do not explicitly target the reduction of EA, there may be an opportunity to assess the clinical effectiveness of treating this transdiagnostic, deleterious process. EA may be clinically alleviated through acceptance-focused interventions that seek to reduce ineffective metacognitions (e.g., evaluative judgment of emotions) and encourage mindful presence with the unwanted internal experience (Hayes, 2004).

Findings from the review may be extended by exploring the nature in which acceptance-focused interventions may be implemented to enhance the effectiveness of PTSD treatment in reducing residual insomnia. Given the nascent state of the research, there is a healthy amount of space for future work to explore not only the integration of acceptance intervention delivery, but also to identify a palatable, efficacious sequencing of treatment options for trauma survivors. For example, acceptance-based strategies could be implemented as a frontline approach to reduce distress related to insomnia, which has been shown to undermine treatment response to many gold standard PTSD treatments (López et al., 2017; Milanak et al., 2019). The role of EA in comorbid insomnia and PTSD could also be investigated at a foundational level by more concretely discriminating the degree of construct overlap. For instance, future

studies could clarify how much variance in measures of psychological distress due to PTSD and/or insomnia is accounted for by EA as compared to other competing explanations. Since FoS may instigate and maintain residual insomnia via avoidance strategies, other research could explore the degree to which secondary as compared to primary arousal is implicated. The metacognitive questionnaire for insomnia (MCQ-I; Waite et al., 2009) is one such methodological option that may be able to reliably quantify secondary arousal, though additional, longitudinal research in clinical populations is necessary. Altogether, there is considerable opportunity and direction for future work to continue the investigation of EA in comorbid insomnia and PTSD.

Chapter 2: Investigation of a Conceptual Model of Fear of Sleep

Brief conceptual overview for a model of fear of sleep

Pursuant to a traumatic event, acute insomnia symptoms are thought to initially arise from a confluence of heightened physiological, emotional, and cognitive sources of arousal (Germain, 2013; Sinha, 2016). While traumatic symptoms and the associated sleep disturbances often resolve in a matter of weeks without therapeutic intervention, a significant minority of trauma-exposed individuals may develop a chronic presentation of PTSD (Breslau, 2009; Hidalgo & Davidson, 2000). As many as 40% of those diagnosed with PTSD further meet criteria for clinically-severe insomnia, which may develop and persist uniquely in the context of traumatic symptoms (Harvey et al., 2003; Spoormaker & Montgomery, 2008). Differentiated from other common sleep disturbances in PTSD, trauma-induced insomnia includes all components of insomnia that follow in the wake of a traumatic event, namely difficulty initiating/reinitiating sleep onset and achieving restorative sleep (Sinha, 2016). Albeit a common precipitant of PTSD, trauma-induced insomnia may also be a critical precursor to the development of the disorder (Koren et al., 2002) due to REM fragmentation (Kobayashi & Mellman, 2012; Mellman et al., 2002) and/or overgeneralization of fear responses to intrusion symptoms after trauma exposure (Wright et al., 2011).

Despite the vast evidence base for the effectiveness of gold-standard, evidence-based treatments (EBTs) for PTSD in the remission of traumatic symptoms, including nightmares (Lee et al., 2016; Schnurr, 2017), chronic insomnia persists in a significant minority of trauma survivors (Belleville et al., 2017; DeViva et al., 2005; Galovski et al., 2009; Gutner et al., 2013; Lommen et al., 2016; Pruiksma et al., 2016; Schnurr & Lunney, 2019; Woodward et al., 2017; Zayfert & DeViva, 2004). Although there is evidence to suggest that sleep-focused interventions, such as cognitive-behavioral therapy for insomnia (CBT-I; Ho et al., 2016; Taylor & Pruiksma, 2014) and imagery rehearsal for nightmares (Casement & Swanson, 2012), may

alleviate the severity of trauma-induced insomnia and traumatic symptoms, clinically-significant, residual sleep disturbances are still observed, especially in more severe presentations of PTSD (Swanson et al., 2009; Ulmer et al., 2011). Residual insomnia may be particularly detrimental to trauma survivors given the positive association with PTSD symptoms following treatment (López et al., 2019; McHugh et al., 2014), significantly increased risk of PTSD relapse, and onset of new mental health disorders (Hertenstein et al., 2019). Consequently, the perseverance of insomnia symptoms may indicate underlying factors that have yet to be explicitly targeted in EBTs for PTSD and/or sleep disturbances.

Werner and colleagues (2021) have recently proposed a conceptual model in which fear of sleep (FoS) may not only exacerbate insomnia symptoms following a traumatic event, but also maintain chronic trauma-induced insomnia even after PTSD symptoms have remitted with treatment. Within the model, FoS has been operationalized to include three interconnected domains: (1) dysfunctional cognitions regarding loss of safety and control; (2) persistent fear related to initiating/reinitiating sleep and/or experiencing nightmares; and (3) behavioral avoidance of maladaptive cognitions and/or fear in the sleeping environment. Following the onset of acute traumatic symptoms, FoS is posited to develop due to a marked sense of vulnerability at bedtime and fear of re-experiencing the traumatic event via nightmares (Werner et al., 2020). Accompanied by heightened arousal in the sleeping environment (i.e., cognitive, emotional, and physiological), avoidance via hypervigilant behaviors entrench and maintain insomnia symptoms even after evidence-based PTSD treatment. Importantly, FoS is thought to be orthogonal to other insomnia-related processes that also contribute to the onset of trauma-induced insomnia, which are thought to instead emerge from general hyperarousal symptoms associated with trauma (Werner et al., 2021). The explanation for this separate onset pathway is rooted in the cognitive model of insomnia, where dysfunctional beliefs about the consequences of truncated or non-restorative sleep may amplify psychological distress and lead to maladaptive, avoidance-driven behaviors in the

context of sleep (e.g., “I won’t be able to function in my job if I don’t get eight uninterrupted hours of sleep”; Harvey, 2002). Altogether, FoS appears to develop as a proximal consequence of negative beliefs about vulnerability in the sleeping environment and fear of re-experiencing nightmares, which then uniquely instigates and maintains chronic trauma-induced insomnia over time through avoidance behaviors.

The onset of fear of sleep following a traumatic event

There are two key processes from which FoS is hypothesized to develop: (1) fear of loss of control and safety; and (2) fear of re-experiencing nightmares (Werner et al., 2021). In the first pathway, nocturnal hyperarousal, endemic in PTSD, may precipitate FoS through the exacerbation of trauma-related cognitions and subsequent behavioral avoidance. (Sinha et al., 2016). Trauma survivors who demonstrate higher sympathetic activation in the immediate post-traumatic period are significantly more likely to develop clinically-significant PTSD (Bryant et al., 2000; Shalev et al., 1998). Consequently, trauma exposure induces an intense stress or fear response that subsequently activates central and peripheral arousal systems (Charney, 2003; Dong & Li, 2014). Through a series of signal cascades, interconnected neural structures, including the amygdala and prefrontal cortex (PFC; Bonnet & Arand, 2010; Buysse et al., 2011; Spoormaker & Montgomery, 2008), generate and sustain an exaggerated state of hyperarousal (Williams et al., 2006). Notably, overactivation of the amygdala, a critical component of the limbic system implicated in the regulation of stress responses, may lead to the heightened emotional and cognitive hyperarousal (Paré et al., 2004). Due to the manner in which trauma responses are encoded as a memory within fear networks, physiological and behavioral responses as well as the appraisal of these response elements become closely linked with the feared stimulus situation (Foa & Kozak, 1986). Thus, a “hyperfunctional” amygdala comes with a tremendous cost to the trauma survivor (Diamond & Zoladz, 2016)—hyperarousal begets trauma-related cognitions, which may further invoke re-experiencing

symptoms, intensify feelings of helplessness, increase worries around loss of control and safety, and promote safety-seeking behaviors (Beck et al., 2015; Ehlers et al., Foa, 1993; Foa & Rothbaum, 2001; 2004; Kimble et al., 2013).

Above and beyond the contribution of acute, nocturnal hyperarousal symptoms, trauma-related cognitions and subsequent behavioral avoidance play a significant role in the development of FoS. Elaborated further in a cognitive model of PTSD (Ehlers & Clark, 2000), trauma survivors who develop PTSD can be characterized by holding negative appraisals of the traumatic event (e.g., “The world is a dangerous place”) and a persistent sense of current threat despite the traumatic event existing in the past. The overestimation and overgeneralization of present danger may subsequently lead to a perceived loss of control and/or safety in their environment (Werner et al., 2021). Motivated by concerns of vulnerability, hypervigilance is then perpetually employed to monitor potential environmental threats via scanning and high cognitive alertness (Richards et al., 2014). However, the strategy is marked by inaccurate threat detection toward neutral and ambiguous stimuli (Kimble et al., 2014), which is further exacerbated by difficulty disengaging from trauma-relevant information (Pineles et al., 2009). Accordingly, hypervigilance is not only associated with the aggravation of traumatic symptoms (Ehlers & Clark, 2000; Salcioglu et al., 2017), but also profound functional impairment (Norman et al., 2007). Psychological distress and the functional consequences of trauma are also experienced in the sleeping environment, where control and vigilance are inherently incompatible (Germain, 2013). In the case of FoS, hypervigilance is embodied through a multitude of safety behaviors, which are intended to prevent and/or reduce anticipated danger through attempts of controlling the environment (Ehlers & Clark, 2000). As such, the inability to discontinue hypervigilance while initiating sleep onset may paradoxically reinforce intense fear and feelings of vulnerability leading to FoS onset (Werner et al., 2020).

In addition to trauma-related thoughts about loss of control and safety in the sleeping environment, FoS may develop due to intense fear of re-experiencing the traumatic event during distressing nightmares (Werner et al., 2021). Nightmares, a hallmark of PTSD (Campbell & Germain, 2016), are characterized as dysphoric dreams that contain content directly related to a traumatic event and/or trauma-related emotions, often including threats to well-being (Giesermann et al., 2018; Sateia, 2014). Trauma-related nightmares have been further identified as a robust predictor of both PTSD (Pigeon et al., 2013; Short et al., 2014; van Liempt, 2012) and insomnia symptom severity (Gellis et al., 2010), and may even lead to frequent nocturnal awakenings and pronounced feelings of helplessness (Wittmann & de Dassel, 2015). Levin and Nielsen (2007) posited an affect network dysfunction model in which inadequate fear extinction underlies the formation of trauma-related nightmares, particularly among trauma survivors with a high load of psychological distress (Foa & Kozak, 1986; Lang, 1979). More specifically, amygdalar hyperactivity in conjunction with impaired frontal pathway functioning, particularly the medial PFC, disrupt the affect network such that trauma-related fear memory fragments cannot be successfully integrated (Germain et al., 2008; Nielsen, 2017). The model further posits that nightmares persist due to an intersection of the traumatic content, multidimensional responses to fear (e.g., physiological, cognitive, behavioral), and associated interpretations to the responses (Levin & Nielsen, 2007). Thus, hyperarousal and emotional distress lead to fearful appraisals of trauma-related nightmares (Giesermann et al., 2018; Krakow et al., 1995; Neylan et al., 1998), which may contribute to the etiology of FoS. Accordingly, trauma-related nightmares have been shown to hold a bidirectional positive relationship with FoS (Short et al., 2018; Krakow et al., 1995), especially among trauma survivors with nightmare content replicating that of their traumatic experience (Davis et al., 2007). Additional research has provided emerging evidence for cross-sectional associations between nightmare-related distress and FoS severity among individuals with PTSD (Kanady et al., 2018a; Neylan et al., 1998; Pruiksma et al., 2011).

Therefore, the fear of re-experiencing nightmares is a likely process by which FoS develops following trauma.

Fear of sleep processes that maintain trauma-induced insomnia

Acute insomnia symptoms that spontaneously arise following a traumatic event are significantly worsened and gradually entrenched by sleep-interfering, maladaptive FoS behaviors (Werner et al., 2021). Driven by concerns of vulnerability and/or fear of re-experiencing nightmares, trauma survivors repeatedly engage in exaggerated behavioral strategies to enhance perceived safety at the cost of impairing the duration, efficiency, and restorative quality of sleep. For instance, individuals may interminably scan their bedroom for anticipated environmental threats, leave all bedroom lights on, use weighted blankets, repeatedly check that their door/windows are locked (Pruiksma et al., 2014), as well as use stimulating substances (Werner et al., 2021). In an RCT investigating the application of CBT-I for individuals with PTSD, FoS was found to maintain a significant, positive relationship with hypervigilance intensity, overall PTSD severity, and decreased total sleep time at pre-treatment (Kanady et al., 2018a). Interestingly, FoS was alleviated in the active treatment group, which coincided with decreased hypervigilance and PTSD severity. Endorsement of the fear of the loss of vigilance, which underlies many maladaptive FoS strategies, has also robustly predicted insomnia symptoms in a separate population of treatment-seeking Veterans (Pietrzak et al., 2010; Hull et al., 2016). Hypervigilance is not only more severe among individuals who have experienced trauma in a sleep-related context (Huntley et al., 2014), but has also displayed a positive association with greater risk of residual insomnia (Zayfert & DeViva, 2004). Since hypervigilant behaviors are effective in mitigating sleep-related fears, avoiding nightmares, and increasing perceived control in the short-term, trauma survivors quickly begin to overestimate their utility such that the maladaptive FoS behaviors maintain trauma-induced insomnia symptoms in the long-term (Werner et al., 2021). Thus, the avoidance strategies become reinforced and automatically

appraised as the only effective solution to reduce FoS-related psychological distress, which further solidifies their perceived necessity in spite of the deleterious impact on sleep.

Trauma-induced insomnia may also be perpetuated by physiological, cognitive, and affective hyperarousal in the sleeping environment that co-occurs with or becomes exacerbated by FoS (Werner et al., 2021; Sinha, 2016). For instance, hyperarousal symptoms during waking hours have been found to predict FoS above and beyond all other traumatic symptom clusters among individuals with PTSD (Gupta & Sheridan, 2018a). In the same civilian sample of trauma survivors, FoS was determined to be significantly associated with increased sleep onset latency, reduced sleep efficiency, and heightened sympathetic arousal (i.e., heart rate, respiratory disturbance, oxygen desaturation) during sleep. Furthermore, pre-sleep arousal, an index of both cognitive and physiological hyperarousal, has been found to predict much more severe FoS among trauma-exposed individuals (Werner et al., 2020). The resulting fragmentation of consolidated, restorative sleep actively worsens traumatic symptoms (e.g., concentration, irritability; Ehlers & Clark, 2000), undermines psychological recovery (Seelig et al., 2016; Gehrman et al., 2013), and serves to reinforce the various components of FoS. Therefore, arousal driven by FoS may be an integral and pathological mechanism that underlies the maintenance of truncated and/or fragmented sleep via sympathetic overaction (Gupta & Sheridan, 2018b).

Altogether, maladaptive, sleep-interfering behaviors and heightened arousal in the sleeping environment may become conditioned to sleep-related stimuli and uncoupled from traumatic symptoms over time, such that residual insomnia symptoms persist despite PTSD remission (Werner et al., 2021). In other words, trauma survivors with residual insomnia may begin to fear the context of the sleeping environment itself, rather than traumatic symptoms, due to the manner in which perceived vulnerability and the subsequent learned behavioral responses that accompany FoS become rooted (Kanady et al.,

2018b). For instance, individuals who report a history of trauma exposure in darkness and/or the sleeping environment display greater FoS intensity and associated insomnia symptoms (Zayfert & DeViva, 2004). Additionally, sleep disturbances, including nightmares, are more likely to persist following EBTs for PTSD among trauma survivors who reported greater anxiety and depressive symptoms at post-treatment (Belleville et al., 2011). Therefore, residual insomnia may be perpetuated by pathological pathways beyond those forged by trauma exposure. As such, FoS may be a critically-important process in which to intervene to prevent the crystallization of trauma-induced insomnia. While FoS, alongside hyperarousal symptoms, has been attenuated by CBT-I (Kanaday et al., 2018a), there are no specific interventions that target the mechanism at present. Consequently, the unique nature of FoS in perpetuating residual insomnia requires further clarification and characterization to inform the development of evidence-based care.

Conceptual overlap and differentiation between fear of sleep and experiential avoidance

The inconsistent manner in which FoS has been defined within the literature serves to undermine a coherent approach to measurement and subsequent development of targeted treatments. While FoS does not have an established diagnostic criteria, the multidimensional conceptualization initially proposed by Werner and colleagues (2021) may be an important foundation from which to examine and refine the construct. The model emphasizes the problematic manner in which a lack of control in the sleeping environment begins to acutely disrupt sleep onset, lead to an entrenchment of avoidance behaviors, and perpetuate residual trauma-induced insomnia symptoms. More specifically, FoS is thought to be driven by counterproductive control strategies, such as sleep-interfering behaviors and hyperarousal, that seek to reduce trauma-related distress, but instead serve to worsen insomnia symptoms. As a result, control efforts are the problem itself, rather than the unwanted internal experiences. The ensuing residual insomnia symptoms may increase the likelihood for a relapse in PTSD

symptoms (Kartal et al., 2021), and/or lead to the onset of other mental health disorders (Biddle et al., 2018). While there is some preliminary support for the benefit of pre-existing insomnia treatments in the reduction of FoS (i.e., CBT-I; Kanady et al., 2018a), there are no current interventions that explicitly target the deleterious process. Therefore, in order to disrupt the chronic control strategies of FoS and prevent long-term consequences, alternative treatment approaches must be explored.

An acceptance-based approach, as embodied in acceptance and commitment therapy (ACT; Hayes et al., 1996), may be more effective in coping with the absence of control that is inherent in the sleep state. While CBT interventions often target change in the content of internal experiences, such as cognitive restructuring of dysfunctional sleep-interfering beliefs in CBT-I, ACT instead seeks to address the relationship with the content itself (Forsyth et al., 2006; Hayes et al., 1996). More specifically, ACT utilizes acceptance to lessen the desire to avoid unwanted internal experiences in service of engaging in a values-congruent life. With respect to FoS, the control agenda may be functionally described through the lens of experiential avoidance (EA), a trait-based form of avoidance within the ACT framework. Moreover, the clarity and utility of FoS may be improved through comparing and contrasting the process with conceptually-similar constructs that also contribute to the pathophysiology of PTSD and insomnia symptoms, such as EA. The construct, which has been implicated in the symptomatology of both insomnia (Ong et al., 2012) and PTSD (Thompson & Waltz, 2010), may serve as a valuable foil from which to elucidate the unique nature and contribution of FoS in the maintenance of trauma-induced insomnia symptoms.

While a broadly-defined transdiagnostic construct, EA shares considerable conceptual overlap with FoS with respect to the function of avoidance. In ACT, the two constituent elements of EA, non-acceptance of internal experiences and subsequent efforts to avoid and/or modify internal experiences (Hayes et al.,

1996; Hayes et al., 1999), are both represented in the multidimensional definition of FoS (Werner et al., 2021). Acceptance is defined as a willingness to have internal experiences, whether construed as pleasurable or painful, exactly as they are in service of a values-congruent life (i.e., “Sadness is an intrinsic part of my emotional experience”; Harris, 2019). In contrast, a non-accepting attitude towards internal experiences is marked by evaluative judgment and willfulness to engage in meaningful aspects of life (i.e., “My negative emotions shouldn’t be so uncomfortable”). Hence, acceptance is characterized by a flexibility, curiosity, and openness toward internal experiences, whereas non-acceptance involves more rigidity, reluctance to learn, and narrowness. Once a decision has been made to no longer tolerate discomfort in a moment, non-acceptance prompts a multitude of strategies designed to alter the nature of or contact with the internal experience (i.e., “I have to distract myself from this pain”). In the case of FoS, internal experiences associated with the sleeping environment precipitate a diminished sense of safety and control as well as fear of re-experiencing distressing nightmares. Consistent with a non-accepting stance, the ensuing psychological and physiological distress is interpreted as unwanted, unbearable, and disruptive, which elicits further arousal (Nagy et al., 2022; Deliens et al., 2014; Yoo et al., 2007). Residual trauma-bound concerns regarding the loss of safety and control (i.e., “I’m not safe if I close my eyes in bed”) are further intensified by the inherent need to reduce environmental monitoring prior to sleep (Werner et al., 2021). Altogether, the desire to preserve control amidst perceived vulnerability in the sleeping environment leads to maladaptive, sleep-interfering behaviors. Akin to EA from a perspective of functional contextualism, FoS-driven avoidance may reduce short-term discomfort at the cost of long-term well-being through an overestimation of the utility of the strategies. Despite the conceptual overlap of the constructs, FoS and EA can be differentiated by characterizing the specificity of the nature of the avoidance.

As reflected in FoS and EA, avoidance can be conceptualized and measured in two distinct ways.

Contextual avoidance is specific to certain internal and external environments, which is exemplified by use of safety behaviors in FoS (e.g., delaying sleep onset, sleeping with lights on; Werner et al., 2021).

Driven by post-traumatic hypervigilance, FoS safety behaviors seek to reduce psychological distress experienced only within the sleeping environment (Gupta & Sheridan, 2018b; Hull et al., 2016; Kanady et al., 2018b). Safety behaviors become reinforced through the reduction in contact with the threatening situation and prevent new learning from occurring (e.g., felt fear does not equate to actual danger).

Conversely, trait-based avoidance, as in the case of EA, is an affinity or orientation towards non-acceptance of uncomfortable internal experiences that is generalized across most contexts and leads to a diversity of avoidance strategies (Gámez et al., 2011; Hayes et al., 1996). When applied inflexibly and with urgency, EA becomes a psychological vulnerability that pathologically underlies numerous mental disorders (Kashdan et al., 2006; Thompson & Waltz, 2010; Webb et al., 2012), as well as reductions in valued living (Gámez et al., 2009; Wilson, 2009) and physical well-being (Berghoff et al., 2017). As a result, a tendency toward the overutilization of avoidance strategies may also be a predisposing factor in the development of FoS and subsequent reliance on safety behaviors (Kirk et al., 2019). Therefore, clarifying the manner in which trait-based and contextual avoidance are interlinked in trauma-induced insomnia is necessary to better understand the maintenance of residual insomnia symptoms, predict clinical outcomes, and apply beneficial treatment approaches.

Chapter 3: Examination of the Psychometric Properties of Fear of Sleep

Literature review of fear of sleep measures

Despite the complex, multidimensional nature of FoS, the construct has been historically captured by a single, Likert-scale question (Krakow et al., 1995, Davis & Wright, 2007; Davis et al., 2007; Davis et al., 2011). For example, studies have often assessed FoS via the Trauma-Related Nightmare Survey (TRNS) item, “In general, how fearful are you to go to sleep?” (Davis, Wright, & Borntrager, 2001). In an effort to broadly characterize chronic nightmares following trauma exposure, the TRNS provided preliminary evidence linking FoS with nightmares and PTSD (Davis, 2008). However, the specific emotional, cognitive, and behavioral sequelae of FoS remained nebulous due to the constraints of the construct measurement. Consequently, Zayfert and colleagues (2006) sought to expand the manner in which FoS was assessed by developing the 23-item FoSI with an emphasis on measuring sleep-interfering trauma-related behaviors, such as evening hypervigilance, and nightmare-related fear. While the study is unpublished, results indicated that the FoSI, as well as the two subscales (i.e., nightmare avoidance and nighttime vigilance) had satisfactory reliability and convergent validity with measures of insomnia and sleep quality (Zayfert et al., 2006).

In response to the limitations of the Zayfert et al. (2006) study, Huntley and colleagues (2014) aimed to characterize the psychometric properties of the FoSI through implementation of EFA (exploratory factor analysis) and CFA (confirmatory factor analysis). Notably, the study also expanded upon racial diversity of the primarily white female participants that composed the unpublished FoSI study with a sample of Black American young adults. The sample may have particular merit in the investigation of FoS due to well-established racial disparities in sleep disturbances and sleep quality among Black Americans compared to their white counterparts (Cheng et al., 2020). Additionally, the Black American sample also had a more balanced distribution of gender (48.4% women) and higher incidence of trauma exposure (88.3%; Huntley

et al., 2014). The resulting EFA extracted five factors with high internal consistency, which were operationalized as the following domains: (1) *fear of sleep*; (2) *fear of the loss of vigilance*; (3) *fear of re-experiencing trauma*; (4) *vigilance behavior*; and (5) *fear of the dark*. Results of the ensuing CFA indicated an acceptable model fit in contrast to the one- and two-factor structure that was initially proposed by Zayfert et al. (2006). Importantly, one of the factors was composed of items closely associated with a core criterion of PTSD (i.e., re-experiencing symptoms), which may compromise the integrity of the FoSI as a measure of FoS specifically. FoS was further defined as a distinct factor from vigilant behaviors and fear of loss of control in the sleeping environment, despite the latter two domains being conceptually essential to FoS. Despite theoretical limitations of the factor structure, the FoSI demonstrated high convergent validity with measures of PTSD and insomnia symptom severity across all five factors (Huntley et al., 2014). As research continues to investigate the psychometric properties of the FoSI, the domains must be thoroughly distinguished from trauma-related symptoms.

During an additional examination of the psychometric properties of the FoSI, researchers refined the measurement of the construct and achieved satisfactory convergent validity with measures of PTSD and insomnia symptoms (Pruiksma et al., 2014). The predominantly white (46.9%) and female (68.8%) undergraduate student sample reported high rates of trauma exposure (71%) and subthreshold PTSD symptom severity. Initially, an EFA of the FoSI displayed an unclear factor structure, which was notably contaminated by a factor composed of PTSD-specific symptoms. The remaining 13 items of the abbreviated measure (i.e., FoSI - Short Form [FoSI-SF]) explained a significant amount of the total variance (i.e., 60.21%) and clearly loaded onto the following two-factor structure: (1) *fear of loss of control*; and (2) *fear of darkness* (Pruiksma et al., 2014). In contrast to the FoSI, items maintained within the FoSI-SF appeared to be theoretically consistent with FoS while maintaining divergence from related psychopathology. A follow-up validation study sought to extend findings of the undergraduate student

sample with a clinical, community sample of trauma-exposed adults with chronic nightmares. The clinical sample was predominantly white (82.1%) female (74.6%), and middle-aged (Mean age = 42.18) with a much higher incidence of trauma exposure. Among the community population, the FoSI-SF was found to have good internal consistency among the two factors ($r = .88-.91$) as well as robust convergent validity with PTSD ($r = .39$) and insomnia ($r = .48$) symptoms (Pruiksma et al., 2014). The FoSI-SF was further found to be significantly correlated with the TRNS FoS item. While the FoSI-SF does not have an established clinical threshold of severity, the clinical community sample displayed a significantly higher score on the measure as compared to the undergraduate student sample from the first study ($d = 1.26$; Pruiksma et al., 2014). Altogether, the FoSI-SF is a foundational and multidimensional measure of FoS that is sensitive to trauma-exposed populations.

More recently, Brown et al. (2018) sought to extend previous examinations of the psychometric properties of the FoSI and FoSI-SF in a novel, adolescent population. Adolescence is a developmental period marked by increasing social demands and shifting biological cues. Consequently, insufficient sleep is a common occurrence throughout adolescence due to the incongruence of (1) a delayed circadian rhythm; and (2) non-delayed social rhythms (e.g., school; Niu et al., 2021). In other words, adolescents typically initiate sleep at a later time than childhood, though maintain the same early morning awakening due to school and/or extracurricular commitments (Crowley et al., 2007). Therefore, FoS resulting from trauma exposure may significantly exacerbate the normative sleep disruptions typical of adolescence. The FoSI may be a particularly impactful measure among adolescents to capture trauma-related FoS. Participants in the adolescent study (Mean age = 16.00), who were primarily male (66%) and Black American (94%), reported a lower incidence of trauma exposure (57%) than previous studies involving FoS (Brown et al., 2018). An EFA of the 23-item FoSI was not able to determine a clear factor structure, such that 22 of the items loaded onto multiple factors. In an effort to replicate the Pruiksma et al. (2014)

study in an adolescent population, an additional EFA of the 13-item FoSI-SF resulted in a more clear two-factor structure. However, two items cross-loaded onto both factors and could not be adequately interpreted (i.e., items 3, 12). Accounting for 50% of the variance, the remaining 11 items were categorized into the following two factors: (1) *fear of sleep*; and (2) *vigilant behaviors*. The newly-formed FoSI-11 was found to have satisfactory internal consistency for the overall measure ($\alpha = .79$) as well as the two subscales ($\alpha = .79; .73$). In-line with previous studies involving the FoSI and FoSI-SF, the FoSI-11 maintained high convergent validity with insomnia symptom severity. Only the vigilant behaviors subscale was positively correlated with PTSD symptom severity, which highlights the particular relevance of hypervigilance in the context of FoS.

Researchers sought to adapt the FoSI-SF to German-speaking populations with the intention of addressing limitations of the initial Pruiska et al. (2014) validation study (Drexler et al., 2019). More specifically, the German study aimed to confirm the two-factor structure of the FoSI-SF and further examine the clinical utility of the measure in identifying group differences among a clinical and non-clinical sample. The overall sample was characterized by predominantly middle-aged (Mean age = 48.69) and female (61.9%) participants, although racial/ethnic demographic information was not reported. Participants who screened positive for PTSD (i.e., PTSD Checklist for DSM-V ≥ 33) were assigned to a clinical subsample for comparison during analyses. Following translation of the FoSI-SF, the sample was randomly bisected to conduct an EFA and CFA within the subsamples. The German version of the FoSI-SF yielded acceptable internal consistency for the overall measure ($\alpha = .87$), as well as the fear of loss of control ($\alpha = .88$) and fear of darkness subscales ($\alpha = .76$). Furthermore, a parallel analysis was conducted within the EFA sample to first identify the optimal number of factors and then examine the subsequent factor loadings. Results of the parallel analysis suggested the extraction of four factors, and the subsequent EFA identified a Heywood case (i.e., factor loadings greater than one and negative item

variance) for item 12 of the fear of darkness subscale. The CFA also encountered a Heywood case for item 12, which compromised the interpretability of the model fit to the two-factor structure derived by the English version of the FoSI-SF due to a potential model misspecification (Brown, 2014). In spite of the limited interpretability of the factor structure, analyses of group differences revealed an interaction effect for insomnia on FoS across levels of trauma exposure, but not among participants with probable PTSD. Additionally, FoSI-SF scores were significantly higher for participants with probable PTSD as compared to the trauma-exposed and unexposed groups. The FoSI-SF also maintained high convergent validity with measures of insomnia and PTSD severity, as well as sleep quality, which is consistent with the earlier validation study (Drexler et al., 2019; Pruiksma et al., 2014).

The FoSI-SF was further adapted to Turkish-speaking populations with the intent of evaluating the psychometric properties, replicating the two-factor structure first identified by Pruiksma et al. (2014), and expanding the utility of the measure (Altan-Atalay et al., 2022). Similar to the German-speaking adaptation (Drexler et al., 2019), participants for the Turkish study were skewed toward middle adulthood (Mean age = 41.03) and disproportionately women (57.9%). The resulting EFA produced a three-factor solution that was operationalized as (1) *fear and threat perception*; (2) *vulnerability and hypervigilance*; and (3) *fear of darkness*. Interestingly, items within the factor fear of loss of control as operationalized by Pruiksma et al. (2014) diverged into separate domains in the Turkish-speaking population. For example, hypervigilant behaviors (e.g., “I was fearful of letting my guard down while sleeping”) were distinguished from nightmare-related fear, which is consistent with the multiple paths maintaining FoS proposed by Werner et al. (2021). The internal consistency for the overall FoSI-SF was deemed acceptable ($\alpha = .81$), though was marginally lower within the fear and threat perception subscale ($\alpha = .70$; Altan-Atalay et al., 2022). Examinations of convergent validity for the FoSI-SF produced significantly positive associations with PTSD and insomnia symptoms as well as perseverative, negative cognitions. When controlling for

ruminative behavior and demographic-related characteristics, the fear of darkness subscale was no longer a significant predictor of PTSD symptom severity. Furthermore, a follow-up hierarchical regression analysis, controlling for the effect of perseverative cognitions, revealed that only the vulnerability and hypervigilance subscale remained a significant predictor of insomnia severity for participants with high PTSD symptoms. The analyses suggested that PTSD-specific processes underlying FoS, such as hypervigilance, may be independently contributing to insomnia symptoms, regardless of general trait rumination. The measurement and characterization of the FoSI-SF, despite current limitations, has provided a promising window into a potentially-critical mechanism underlying the maintenance of trauma-induced insomnia.

As iterations of the FoSI continue to be investigated across developmentally- and culturally-distinct populations, there have been several notable findings in support of the measure's efficacy among comorbid insomnia and PTSD symptoms. Due to the construct validity concerns identified in the 23-item FoSI (Brown et al., 2018; Pruiksma et al., 2014), the short-form version has emerged as a more theoretically-adherent and concise measure of FoS. The remaining items on the FoSI-SF can be primarily characterized as a measure of nighttime vigilance and nightmare-related distress, which are consistent with the two primary pathways in which FoS is posited to perpetuate trauma-induced insomnia (Werner et al., 2021). Additionally, the internal reliability of the FoSI-SF has been determined to be robust across all recent studies ($\alpha = .79-.93$; Altan-Atalay et al., 2022; Brown et al., 2018; Drexler et al., 2019; Pruiksma et al., 2014), suggesting that the current inventory items are adhering to the conceptualization of FoS. Overall, the FoSI-SF has consistently demonstrated high convergent validity with measures of both PTSD and insomnia symptomatology (Altan-Atalay et al., 2022; Brown et al., 2018; Drexler et al., 2019; Pruiksma et al., 2014), while maintaining discriminant validity with distinct constructs, such as sleep hygiene (Pruiksma et al., 2014) and perseverative cognitions (Altan-Atalay et al., 2022). Furthermore, participants

who endorsed clinically-relevant PTSD symptom severity tended to score significantly higher on the FoSI-SF as compared to those trauma exposure alone (Altan-Atalay et al., 2022; Drexler et al., 2019; Pruiksma et al., 2014). The FoSI-SF has also been shown to predict insomnia symptom severity above and beyond the variance explained by comorbid PTSD (Altan-Atalay et al., 2022). Importantly, the results highlight the clinical sensitivity of the FoSI-SF in capturing unique variance among comorbid PTSD and insomnia symptoms. Despite emerging research in support of the FoSI-SF, there remain several critical limitations that must be addressed in future research.

While the FoSI-SF has shown promise as a measure of FoS, emerging literature has identified aspects of the abbreviated inventory that require further revision. Examinations of the psychometric properties have revealed factor structures inconsistent with the initial model proposed by Pruiksma et al. (2014), which may be a consequence of the current operationalization of FoS within the FoSI-SF. Across the limited pool of studies that have applied EFA to the FoSI-SF, a concerning number of items within the fear of loss of control factor have failed to consistently load together (Altan-Atalay et al., 2022; Brown et al., 2018; Drexler et al., 2019). More specifically, six of the eleven items loaded onto a distinct factor that was interpreted as either vigilant behaviors (Brown et al., 2018) or vulnerability and hypervigilance (Altan-Atalay et al., 2022). The vigilance-minded items, as exemplified by “I tried to stay alert as I could while lying in bed,” reflect behavioral countermeasures motivated by distressing thoughts, emotions, and physiological sensations. Whereas, the remaining five items that maintained stable loading were characterized as fear of sleep (Brown et al., 2018) or fear and threat perception (Altan-Atalay et al., 2022). The items, such as “I felt that it was dangerous to fall asleep,” most readily indicate an emotional experience of fear due to a perceived loss of safety in the sleeping environment. From an ACT-based framework, incongruity across the eleven items may be explained by the internal experience (i.e., behavior, emotion, cognition) at the core of the question. As a result, FoS may be more effectively

measured by items with a clear delineation and intent in the description of each internal experience. For example, a future iteration of the FoSI-SF may seek to balance items pertaining to emotions, behaviors, and thoughts specific to FoS to capture the inherently multidimensional nature of internal experiences. Additionally, the fear of darkness factor has presented a significant hindrance in the replication of the two-factor structure model. The factor, which is composed of only two items, may be particularly susceptible to an improper solution when conducting an EFA (Chen et al., 2001). Altogether, the manner in which FoS is conceptualized within the FoSI-SF appears to adequately measure aspects of the construct, but remains incomplete in scope.

Inconsistencies in the factor structure of the FoSI-SF may also be a corollary of significant cross-cultural and/or development age group differences in sleep, insomnia, and trauma. Despite the successful translation of the FoSI-SF into multiple languages, the measure has yielded distinct model outcomes within each sample (Drexler et al., 2019; Altan-Atalay et al., 2022). More specifically, the German adaptation of the FoSI-SF did not produce a satisfactory solution during both the EFA and subsequent CFA (Drexler et al., 2019). Recently, a systematic review on cross-cultural sleep-related factors in children and adolescents identified robust discrepancies in sleep duration, nighttime disturbances, and sleep hygiene-related behaviors/practices (Jeon et al., 2021). A separate comparative study involving Canadian and Japanese undergraduate students further identified significant differences in cognitions involving sleep-related health outcomes (Cheung et al., 2021). Although relevant literature in this field is sparse, available research has generally indicated cultural dissimilitude with regard to sleep. While epidemiological research has demonstrated that cross-cultural differences in the prevalence of insomnia may be negligible (Chung et al., 2015), the phenomenology behind and relationship with trauma-related nightmares may vary between societies (Hinton, 2009; Hinton et al., 2013). At the level of measuring FoS, the FoSI includes the item, “I kept a weapon near my bed at night,” which may not be as relevant in non-

American societies or younger populations (Drexel et al., 2019). Despite being removed from the abbreviated inventory, the item may indicate the presence of other culturally-bound biases in the operationalization of FoS. Beyond cultural considerations, the German and Turkish samples were characterized by broad age ranges (18-92, 18-68 years old) and a skew toward middle adulthood (Mean age = 41.03, 48.69; Drexel et al., 2019; Altan-Atalay et al., 2022), whereas the adolescent study had a constricted age range (14-18 years old; mean age = 16.00; Brown et al., 2018) in comparison. Given the preliminary stage in which the FoSI-SF has been investigated across different populations, the impact of demographic (e.g., age, race/ethnicity), environmental (e.g., rural/urban area), and trauma-based (e.g., cumulative trauma, type of trauma) factors on the development of FoS is currently unclear. Therefore, future research examining the FoSI-SF should prioritize the replication of findings within similar samples to clarify the utility of the measure for the intended population.

Noted limitations in the current measurement of FoS may be addressed by (1) a nuanced investigation and clarification of the construct; and (2) replication of the FoSI-SF factor structure with a similar population. Given that FoS is a multidimensional manifestation of psychological distress, the pathological process could be more clearly contextualized within comorbid PTSD and insomnia by exploring the relation with specific traumatic symptoms, particularly hypervigilance and fear of nightmares. Since the symptoms are posited to be key etiological pathways in which trauma-induced insomnia becomes entrenched, the present study aims to elucidate the association with FoS more specifically. Additionally, non-acceptance and avoidance in response to discomfort associated with FoS-driven internal experiences can also be classified within the EA umbrella. Considering the scarcity of literature exploring the theoretical overlap between FoS and EA, both constructs could be further illuminated and distinguished for clinical application. Additionally, discrepancies in FoSI-SF factor structure within the literature may be remedied by using a demographically-similar sample to that of the initial validation study, both in age and

predominant culture (Pruiksma et al., 2014). Consequently, utilization of an American undergraduate sample and subsetting by clinical severity may present the best-suited opportunity for replicating the elusive two-factor structure.

Justification for exploratory factor analysis as the primary methodology

Given the recency with which FoS was identified as an important pathological process in comorbid PTSD and insomnia, the existing literature has primarily cast the construct in a one-dimensional manner. Building upon initial research, the FoSI and FoSI-SF were created to represent FoS with a more robust and multifaceted operationalization. Studies involving the FoSI and/or FoSI-SF have heavily relied upon exploratory factor analysis (EFA) and confirmatory factor analysis (CFA) to contribute to the current theoretical conceptualization of the construct. However, the resulting factor structures have been marred by inconsistency across populations. Consequently, there remains ample opportunity for the continued analysis of the measurement of FoS with the intention of providing a stable factor solution. Given the nascent stage of FoS research, EFA may be the more appropriate statistical technique to concretize the factor structure of the construct.

EFA is one of the most commonly utilized tools in the interpretation of items within psychological questionnaires (Williams et al., 2010). More specifically, the multivariate statistical methodology seeks to identify the fewest number of latent factors that can parsimoniously explain covariation between the measured/observed variables (i.e., items on questionnaires; Watkins, 2018). In other words, latent factors are considered to be the underlying cause for the intercorrelation among a set of observed variables. Therefore, the accurate measurement of a construct is reliant on a foundation of theoretically-sound conceptualization of both unobserved factors and the observed variables. Once the latent factors are

adequately identified within a measure, clinical interventions can be directed toward alleviating the underlying pathological process.

Several alternative approaches could be considered for evaluation of FoS factors, including confirmatory factor analysis, structural equation modeling (SEM), and principal component analysis (PCA). However, these methods may be premature due to the divergent factor structure of the FoSI-SF across the current literature. While EFA allows for the generation of theory, CFA is intended to verify theory with a preordained factor structure (Williams et al., 2010). CFA is built upon *a priori* hypotheses that predict the number of expected factors and best model fit. As a result, a rigid set of model predictions would not be suited for the provisional factor structure of the FoSI/FoSI-SF and understanding of FoS more broadly (Brown & Moore, 2012). CFA is a specialized type of structural equation modeling (SEM), which further includes path analysis to represent the relationship between latent factors and observed variables (Ullman, 2006). Since SEM is a confirmatory approach predicated on a well-established theoretical foundation, the analysis may be a poor fit for the current status of the FoSI/FoSI-SF (Ockey, 2013). Another multivariate tool, Principal components analysis (PCA), seeks to simplify a set of data into the most essential parts while largely preserving the variance explained (Abdi & Williams, 2010). By optimizing dimensionality, variable relationships are then coherently represented by a small number of components, which can then be plotted for visual illustration (Ringnér, 2008). Although PCA is a useful tool in capturing variance among a set of observed variables, the approach is not suited to theoretically interpret latent factors that require the measurement of covariance. Considering the concerns regarding the fit of confirmatory approaches for understanding FoS as conceptualized within the FoSI-SF, the measure could first benefit from further examination through exploratory methodology.

Chapter 4: Present Study

Introduction

Post-traumatic stress disorder (PTSD) is a mental health condition that develops after direct or indirect exposure to a traumatic event, such as assault, sexual violence, and/or natural disasters (American Psychiatric Association, 2013). PTSD has a lifetime prevalence rate of 6.1-9.2% among the general population (Koenen et al., 2017; Goldstein et al., 2016; Kessler et al., 2005; Van et al., 2008), as well as 12.1-30.9% among Veterans (Kulka et al., 1990; Kang et al., 2003; Tanielian et al., 2008). Symptoms of the disorder fall into four discrete clusters: re-experiencing, internal/external avoidance, changes to thoughts/emotions, and hyperarousal (Sareen, 2014). For example, trauma survivors may experience distressing thoughts about themselves following a traumatic event (e.g., “I’m a broken person”), which can be accompanied by an elevated heart rate, intrusive memories related to the traumatic event, and attempts to avoid reminders of trauma-related stimuli. Since the symptoms are experienced across multiple domains, PTSD may follow a chronic and debilitating trajectory without adequate treatment (deRoos-Cassini et al., 2010; Bonanno et al., 2005). The impact of the disorder is substantial due to the increased likelihood of developing secondary comorbid psychiatric disorders, experiencing occupational impairment, and educational attainment failure (Kessler, 2000). Insomnia, a core feature of PTSD (Germain, 2013), is thought to significantly contribute to the overall psychological distress and functional impairment due to trauma-related symptoms (Koffel et al., 2016).

Insomnia is characterized by the disrupted initiation and reinitiation of sleep resulting in fragmented, truncated, and non-restorative rest when given sufficient sleep opportunity (Harvey, 2002). Notably, insomnia symptoms are ubiquitously reported among trauma survivors (i.e., 70-90%; Richards et al., 2020; Talbot et al., 2014; Maher et al., 2006) and have been implicated in the onset (Gehrman et al., 2013; Wright et al., 2011; Bryant et al., 2010), maintenance (Gilbert et al., 2015), and exacerbation of

PTSD (Koffel et al., 2013; McKay et al., 2010). Since the onset of insomnia may also occur following trauma exposure (Sinha, 2016), a mutual set of disrupted internal systems may underlie the bidirectional relationship. More specifically, trauma-related hyperarousal may inimically delay sleep onset, fragment sleep (Spoormaker et al., 2010), and subsequently inhibit fear extinction that typically occurs during slow wave and rapid-eye movement sleep (Van Der Helm et al., 2011; Payne et al., 2012). However, despite the effectiveness of PTSD treatment in reducing trauma-related symptoms, insomnia symptoms often linger and continue to undermine psychological health (Zayfert & DeViva, 2004; Pruiksma et al., 2016) via a heightened risk for PTSD relapse and/or the onset of other mental health problems due to sleep disruptions (Hertenstein et al., 2019).

Residual insomnia may be maintained by processes that are not alleviated by evidence-based treatments for PTSD and subsequently require further examination to achieve a similar degree of remission. Recently conceptualized, the trauma-induced model of insomnia posits that fear of sleep (FoS) may be a unique construct implicated in the instigation and exacerbation of insomnia symptoms following exposure to a traumatic event, such that residual insomnia is perpetuated even after PTSD symptoms are diminished (Werner et al., 2020; Werner et al., 2021). FoS has been characterized by the following components in the context of sleep: (1) persistent fear; (2) unhelpful cognitions about vulnerability and/or loss of control; and (3) maladaptive behavioral strategies to enhance perceived safety and control (Werner et al., 2021). The onset of FoS is thought to be rooted in trauma-related cognitions about safety (e.g., “The world is always a dangerous place”; Kanady et al., 2018a) and fear of re-experiencing nightmares (Davis et al., 2007; Krakow et al., 1995; Neylan et al., 1998; Pruiksma et al., 2011), which are further accompanied by distressing hyperarousal symptoms (e.g., hypervigilance; Gupta & Sheridan, 2018a). FoS then becomes entrenched through maladaptive avoidance strategies, such as safety behaviors (e.g., turning on all lights, being on guard and alert in bed), which reinforce the perceived danger in the sleeping environment and

inhibit new learning (Werner et al., 2021). Consequently, components of FoS may then become uncoupled from PTSD and generalized to the sleeping environment over time, such that the context of sleep engenders significant psychological distress even after the remission of PTSD symptoms (Werner et al., 2020; Sinha, 2016; Kleim et al., 2013). Although there is preliminary evidence in support of cognitive-behavioral therapy for insomnia in the treatment of FoS (Kanady et al., 2018a), there are no present interventions that explicitly seek to disrupt the deleterious process. Therefore, alternative treatment approaches that alleviate FoS must be considered to reduce long-term, problematic health outcomes due to residual insomnia.

Acceptance and commitment therapy (ACT; Hayes et al., 1996) may provide a fruitful foundation from which to effectively cope with the loss of control that is inevitable when initiating sleep. More precisely, the acceptance-based approach aims to shift the relationship with unwanted internal experiences from animosity to willingness, such that the avoidance strategies that maintain psychological distress and functional impairment are gradually reduced (Forsyth et al., 2006; Hayes et al., 1996). The control agenda that pervades FoS may be described within an ACT framework as experiential avoidance (EA), a trait-like stance of non-acceptance towards internal experiences that is accompanied by a heterogeneous repertoire of avoidance strategies (Gámez et al., 2011; Hayes et al., 1996). When EA becomes generalized across multiple contexts, the psychological vulnerability has been identified in the pathogenesis of various mental disorders (Kashdan et al., 2006; Thompson & Waltz, 2010; Webb et al., 2012) and implicated in the loss of valued living (Gámez et al., 2009; Wilson, 2009) and physical functioning (Berghoff et al., 2017). Furthermore, since EA has also been closely linked with PTSD (Thompson & Waltz, 2010) and insomnia (Ong et al., 2012) symptomatology, the construct may be an essential comparator from which to clarify the unique and pernicious contributions of FoS in the perpetuation of trauma-induced insomnia.

While FoS is gaining recognition as a deleterious process in comorbid PTSD and insomnia (Kanady et al., 2018a; Pruiksma et al., 2014; Huntley et al., 2014), there remains numerous avenues to explore in further characterizing the construct. For example, a notable measure of FoS, the Fear of Sleep Inventory - Short Form (FoSI-SF), has been found to have a diverging factor structure across different populations (Altan-Atalay et al., 2022; Brown et al., 2018; Drexler et al., 2019; Huntley et al., 2014; Pruiksma et al., 2014). Considering the nascent stage in which FoSI-SF has been investigated within various populations of clinical severity, developmental stage, and environment, further examination of FoS could benefit from replicating findings and providing a more nuanced examination of the construct in relation to prevailing models and related constructs.

In Aim 1 of the present study, we hoped to address current gaps in the literature by examining the psychometric properties of the FoSI-SF, including the factor structure, convergent validity with EA, and discriminant validity with sleep hygiene in a general population of college students. We hypothesized that the FoSI-SF will demonstrate robust internal consistency and reproduce the two-factor structure of the initial FoSI-SF validation study (i.e., Factor 1 = Fear of loss of control/safety; Factor 2 = Fear of darkness; Pruiksma et al., 2014). Given the divergence of FoSI-SF factor structure across different populations (Altan-Atalay et al., 2022; Brown et al., 2018; Drexler et al., 2019), we sought to replicate the findings of the study with a college student sample akin to that of the validation study (Pruiksma et al., 2014). To elucidate the degree to which EA may have convergent validity with FoS, we plan to examine the two most representative aspects of the construct, Distress Aversion and Behavioral Avoidance, as operationalized by the multidimensional experiential avoidance questionnaire (MEAQ; Gámez et al., 2011). More specifically, Distress Aversion is defined as a non-accepting attitude toward internal experiences (i.e., “Happiness means never feeling any pain or disappointment”). Also central to EA as a construct, Behavioral Avoidance is constituted by attempts to avoid situations that are predicted to elicit

various forms of distress (i.e., “I go out of my way to avoid uncomfortable situations”). Therefore, the resulting factor scores from the EFA will demonstrate significant positive correlations with the MEAQ total score as well as Distress Aversion and Behavioral Avoidance subscales. Similar to the FoSI-SF validation study (Pruiksma et al., 2014), we hoped to clarify FoS as a distinct construct from other measures of sleep-related behaviors that are associated with insomnia symptoms, such as sleep hygiene (Stepanski & Wyatt, 2003). As a result, we hypothesized that the FoSI-SF will be found to display discriminant validity with the sleep hygiene index (SHI; Mastin et al., 2006), such that FoS will be evidenced as a distinct sleep-related construct ($p < .30$).

To evaluate the trauma-bound conceptualization of FoS and purported relationship with insomnia (Werner et al., 2021), we plan to subset the overall sample in Aim 2 to include only individuals with clinically-significant PTSD and clinically-subthreshold insomnia symptoms. Since FoS has been posited to develop due to distress associated with negative cognitions around fear of loss of control/safety in the sleeping environment and concerns over re-experiencing traumatic nightmares, we hypothesize that FoS factor scores (computed from the resulting EFA in the first aim) will display differential greater association with the hypervigilance and nightmare symptoms of PTSD as compared to the association with the remaining PTSD symptoms. In particular, hypervigilance has been regarded as a sequela of loss of control and has been linked with FoS (Gupta & Sheridan, 2018a; Huntley et al., 2014). Finally, we predict that the FoS factor scores will be more robustly associated with PTSD and insomnia symptom severity as compared to the association with EA. While FoS and EA may have construct overlap with respect to function, FoS is thought to be a unique, deleterious process that maintains insomnia through the downregulation of specific trauma-related symptoms (Werner et al., 2021), as compared to the more general trait-based measure of non-acceptance and avoidance.

Method

Participants

The sample in the present study consisted of 197 college-aged students recruited through the Department of Psychology's Human Subjects Pool at the University of Oregon. The sample for the present study included 132 women (66.50%), 62 men (31.47%), and 4 other gender students (2.03%), who had a mean age of 19.54 years old (Range = 18-27; SD = 1.54). With respect to race and ethnicity, 61.93% percent of the sample self-identified as white ($n = 122$), 19.80% multiracial ($n = 39$), 7.11% Asian ($n = 14$), 6.60% Hispanic or Latinx ($n = 13$), 2.03% Black or African American ($n = 4$), and 1.02% American Indian or Alaska Native ($n = 2$). Comprehensive demographics information for the sample is reported in Table 1.

Procedure

Potential participants registered for the study online via the Sona Systems portal as part of their research requirement for an introductory psychology course. After consenting to take part in the present study, participants were asked to complete a series of self-report measures that queried current alcohol and cannabis use, trauma history, insomnia and PTSD symptoms, sleep hygiene, fear of sleep, as well as experiential avoidance. All participants were fairly compensated for their time with 0.75 hours of research credit. The study was approved by the University of Oregon Institutional Review Board as well as the Department of Psychology's Human Subjects Pool.

Measures

Alcohol Use Disorder Identification Test (AUDIT). The AUDIT is a 10-item screening tool used to identify problematic alcohol consumption in the past 12 months (e.g., consequences of use; Saunders et al., 1993) as well as alcohol dependence and abuse (Babor et al., 1992). The measure uses a five-point Likert scale (0 = *Never* to 4 = *4 or more times a week*; 0 = *No* to 4 = *Yes, during the last year*) and is scored

by summing all 10 items (range 0-40). For the AUDIT total score, 8-15 may indicate hazardous alcohol consumption, whereas scores ≥ 16 suggest harmful drinking (Babor et al., 2001). The AUDIT has robust internal consistency ($\alpha = .94$) and acceptable test-retest reliability (de Meneses-Gaya et al., 2009).

Cannabis Use Disorder Identification Test - Revised (CUDIT-R). The CUDIT-R is an eight-item screening tool used to identify problematic cannabis consumption in the past six months, including abuse, dependence, and psychological features (Adamson et al., 2010). The self-report questionnaire employs a five-point Likert scale (0 = *Never* to 4 = *4 or more times a week*; 0 = *Never* to 4 = *Yes, during the past 6 months*) and is scored by totaling all eight items (range 0-32). Total scores of ≥ 8 may suggest problematic cannabis use, whereas scores ≥ 12 indicate a possible cannabis use disorder. The CUDIT-R has been found to have high internal consistency ($\alpha = .91$) and test-retest reliability ($r = .87$).

Demographics. Participants were asked to provide the following demographic information: age, race and ethnicity, gender identity, highest educational attainment and income level among parents (i.e., to calculate socioeconomic status), as well as history of sleep disorders and sleep disorder treatment.

Fear of Sleep Inventory - Short Form (FoSI-SF). The FoSI-SF is a 13-item measure used to evaluate the frequency of sleep disturbances related to FoS among individuals with PTSD. The questionnaire uses a four-point Likert scale (0 = *Not at all* to 4 = *Nearly every night*) and is scored by tallying all items (range 0-52), where higher scores indicate increased FoS (Zayfert et al., 2006). The FoSI-SF has been found to possess high internal consistency ($\alpha = .88-.91$) as well as discriminant validity from a measure of sleep hygiene (Pruiksma et al., 2014).

Insomnia Severity Index (ISI). The ISI is a brief self-report questionnaire used to determine the subjective severity, psychological distress, and impairment associated with insomnia in the past two weeks. The questionnaire uses a five-point Likert scale (0 = *None* to 4 = *Very severe*; 0 = *Very satisfied* to 4 = *Very dissatisfied*) across seven questions with a total score range of 0-28. While a total score ≥ 15 was initially determined to be a clinically-significant threshold on the ISI, other research has found that a total score may provide an adequate diagnostic cutoff for insomnia (Morin et al., 1999). The ISI has been determined to have stable and high internal consistency (Bastien et al., 2001).

Life Events Checklist for DSM-5 (LEC-5). The LEC-5 is a 17-item questionnaire used to assess lifetime history of Criterion A traumatic event exposure. While the LEC-5 has no formal scoring procedure, items include six categorical responses (*Happened to me*, *Witnessed it*, *Learned about it*, *Part of my job*, *Not sure*, *Doesn't apply*) to differentiate level of exposure (Weathers et al., 2013). The LEC-5 has been found to have stable test-retest reliability, as well as adequate discriminant/convergent validity (Gray et al., 2004).

Multidimensional Experiential Avoidance Questionnaire (MEAQ). The MEAQ is a 62-item questionnaire intended to measure domains of experiential avoidance, including metacognitive beliefs and various forms of avoidance. The measure uses a six-point Likert scale (1 = *Strongly disagree* to 6 = *Strongly agree*), and contains six subscales: (1) behavioral avoidance; (2) distress aversion; (3) procrastination; (4) distraction/suppression; (5) repression/denial; and (6) distress endurance. The MEAQ total score is calculated by summing all items within each of the subscales (total score range 62-372) with higher scores indicating greater EA. While a clinically-significant threshold for the MEAQ has not yet been determined, a sample of psychiatric outpatients had a mean total score of 224.61 in the initial validation

of the measure. The MEAQ was determined to possess good internal consistency with a Cronbach alpha of .83 (Gámez et al., 2011).

PTSD Checklist for DSM-5 (PCL-5). The PCL-5 is a 20-item instrument designed to evaluate the severity of PTSD symptomatology in the past month and uses a five-point Likert scale (0 = *Not at all* to 4 = *Extremely*). The total score is calculated by summing all items and has a range of 0-80. Several studies have indicated that a total score ≥ 31 is able to accurately predict clinically-significant levels of PTSD (Blevins et al., 2015; Wortmann et al., 2016), though a slightly lower threshold (i.e., ≥ 28) may also be representative of meeting diagnostic criteria (Bovin et al., 2016). The measure has been found to have robust internal consistency ($\alpha = .94$) and test-retest reliability ($r = .82$; Blevins et al., 2015).

Sleep Hygiene Index (SHI). The SHI is a 13-item instrument used to assess the frequency of sleep hygiene-related behaviors. Items are rated on a five-point Likert scale (0 = *Never* to 4 = *Always*) and the total score is calculated by summing all item responses (range 0-52). The SHI has been found to have adequate internal consistency ($\alpha = .66$) and temporal stability ($r = .71$; Mastin et al., 2006).

Statistical Analyses

Aim 1

FoSI-SF Exploratory Factor Analysis. The EFA was conducted in accordance with a recent best practice guide (Watkins, 2018), including satisfying recommendations for an adequate sample size (DeVellis & Thorpe, 2021; Mundfrom et al., 2005). The data were determined to be appropriate for EFA following subjective examination of the correlation matrix (i.e., significant number of correlations $> .30$; Hair et al., 2010). Within the REdaS R software package (Maier, 2015), Bartlett's test of sphericity provided further support for the factorability of the correlation matrix (Bartlett, 1954), significant $\chi^2(78) =$

1661.69, $p < .001$, and the Kaiser-Meyer-Olkin test ($KMO = .89$; Kaiser, 1974) revealed a “meritorious” sampling adequacy (Hoelzle & Meyer, 2013; Lloret et al., 2017). Furthermore, the internal consistency of the FoSI-SF items were found to be acceptable and in line with the original short-form validation study ($\alpha = .92$; Pruiksma et al., 2014).

Data were assessed for linearity through evaluation of scatterplots between FoSI-SF items (Goodwin & Leech, 2006). While the data displayed skew (1.94) and kurtosis (3.35) within an acceptable range (Curran et al., 1996), the Shapiro-Wilk test ($W = .73$, $p < .001$; Shapiro & Wilk, 1965) revealed a non-normal distribution. Due to the likely influence of non-normality on EFA results, polychoric correlations were utilized instead of Pearson correlation coefficients (Goodwin & Leech, 2006). Polychoric correlations, as a more robust method of estimating correlations, are also appropriate for determining the true factor for a set of ordinal variables (Fabrigar & Wegener, 2012), especially for those with fewer than seven categories (i.e., FoSI-SF has a five-point Likert scale; Bandalos & Gerstner, 2016; Fabrigar et al., 1999). Since there were no missing data for the FoSI-SF, imputation methods were not employed (Baraldi & Enders, 2010).

Since the purpose of the study was to evaluate the underlying latent factors of the FoSI-SF while preserving all of the measured variables, we selected common factor analysis for our model as opposed to PCA (Fabrigar et al., 1999). The optimal number of factors to extract were determined through use of parallel analysis (Horn, 1965) and a scree plot (Cattell, 1966). Following the specification of common factor analysis, we selected an iterated principal axis (PA) estimation method due the non-normality of the data (i.e., PA does not make distributional assumptions; Brown, 2015; Cudeck, 2000) and relatively smaller sample size (i.e., ≤ 300 ; Curran et al., 1996; MacCallum et al., 2001). The common factor analysis was replicated through use of maximum likelihood, which is an estimation method that is more appropriate for large sample sizes, data with multivariate normality, and the correct number of factors

have been identified (Fabrigar et al., 1999; Gaskin & Happell, 2014; Norman & Streiner, 2014). Salient factor loadings ($\geq .30$ -.40; Brown, 2015) with moderate or higher communalities ($\geq .20$; Child, 2006) and low cross-loadings ($\leq .32$; Tabachnick & Fidell, 2001) were interpreted as acceptable. Additionally, only overdetermined factors were retained for analyses (≥ 3 variables; Watkins, 2018), since the factor may otherwise be characterized as unstable (Costello & Osborne, 2019). Given that FoSI-SF factors were expected to intercorrelate (Altan-Atalay et al., 2022; Brown et al., 2018; Pruiksma et al., 2014), we selected a promax rotation for the EFA (i.e., promax; Fabrigar & Wegener, 2012). We retained all salient indicators, which demonstrated factor loadings that exceeded .30 (Brown, 2015; Gorsuch, 1983).

In an effort to ensure that the most parsimonious and best fitted model was utilized for analysis, we compared other possible model solutions with similar methodology (i.e., polychoric correlations, PA estimation). Comparisons between the models were made utilizing the following indicators within the lavaan and psych R packages (Revelle & Revelle, 2015; Rosseel, 2012): Akaike information criterion (AIC; Akaike, 1978), Bayesian information criterion (BIC; Schwarz, 1978) and/or sample-size adjusted BIC (BIC_{adjust} ; Sclove, 1987), comparable fit index (CFI; Bentler, 1990), root mean square error of approximation (RMSEA; Steiger, 1990), standardized root mean square residual (SPMR; Jöreskog, 1984), and Tucker-Lewis Index (TLC; Tucker & Lewis, 1973). Chi-squared tests were also examined to compare model fit between possible factor solutions.

FoSI-SF and Sleep Hygiene. A bivariate correlation between the FoSI-SF total score and SHI total score was conducted to measure discriminant validity between the constructs. Since the Shapiro-Wilk test revealed that the FoSI-SF scores were not normally distributed ($W = .73$, $p < .001$; Shapiro & Wilk, we used Spearman's rank correlation coefficient for the analysis. A comparison between correlations with Fisher z-transformation did not determine a significant difference in results (Myers & Sirois, 2004), so

FoSI-SF outliers were not removed from analyses. Discriminant validity between FoS and SHI was interpreted by a weaker correlation (i.e., $< .30$).

FoSI-SF and EA. After the EFA was conducted, refined factor scores were calculated based on the ensuing factor solution within the EFA.dimensions R package (O'Connor, 2020). Since an oblique rotation was applied during the EFA, pattern coefficients were selected for the 'loadings' input to control for the influence of other common factors (Cudeck, 2000; Fabrigar & Wegener, 2012; Hair et al., 2010; Price, 2017). Additionally, pattern coefficients were reviewed for statistical usefulness such that all values exceeded $|.30-.40|$ (Bandalos & Gerstner, 2016; Hair et al., 2010). Following use of pattern coefficients, Bartlett's method for computing factor scores was included (Bartlett, 1937). Polychoric correlations were utilized within the analysis to maintain consistency with robust correlation methodology selected to determine the factor loadings (O'Connor, 2020). To measure the degree of factor score indeterminacy, validity coefficients (i.e., recommended to be $\geq .80$; Gorsuch, 1983), univocality (i.e., correlations between refined factor scores and non-corresponding factors similar to interfactor correlations), and correlational accuracy (i.e., correlations among refined factor scores similar to interfactor correlations) were examined (Brown, 2015; Grice, 2001).

Bivariate correlations between the computed refined factor scores and the MEAQ total score and six subscales were conducted. Spearman's rank correlation coefficient was utilized for analyses after Shapiro-Wilk tests determined that the refined factor scores were not normally distributed ($W = .65-.71$, $p < .001$; Shapiro & Wilk, 1965) and outliers were observed (Chok, 2010). To identify the amount of construct overlap between FoS and EA, results of the correlations will be interpreted in the following manner: $r \geq 0.70$ = likely same construct/robust association, $0.30 \leq r < 0.70$ = distinct constructs/moderate association, and $r < 0.30$ = distinct constructs/weak association.

Aim 2

FoSI-SF PTSD/Insomnia. Participants who reported at least moderate symptom severity for insomnia (ISI total score ≥ 10) and clinically-significant PTSD symptom severity (Endorsement of a lifetime Criterion A traumatic event and PCL-5 total score ≥ 31) were subsetting for second aim analyses. Although 101 participants endorsed at least moderate insomnia symptoms and 65 participants reported clinically-significant PTSD symptoms, only 50 participants total met the comorbid threshold for the second aim. The ISI and PCL-5 symptom severity thresholds were informed by the trauma-induced model of insomnia (Werner et al., 2021), and set to adequately capture a sufficient participant population experiencing distress related to insomnia/PTSD symptoms while balancing recruitment feasibility (Morin, Colecchi, Stone, Sood, & Brink, 1999; Bastien, Vallières, & Morin, 2001; Marx et al., 2021; Bovin et al., 2016).

After subsetting data, refined factor scores were correlated with PCL item 2 (Nightmares) and PCL item 17 (Hypervigilance) using Spearman's rank correlation coefficient. Similar to correlation interpretations in the first aim, we anticipated moderate or greater associations (i.e., $r \geq 0.3$) between the factor total scores and the PCL-5 items 2 and 17. The remaining PCL items were summed into a single PCL score for multiple regression analyses. The summed PCL score alongside PCL item 2 and PCL 17 were inputted as predictors for each of the refined factor scores.

Bivariate correlations with Spearman's rank correlation coefficient were conducted among the refined factors scores with PCL total score and ISI total score. Correlations were repeated with MEAQ total score and the PCL and ISI total scores. We built separate multiple regressions to predict PCL and ISI total scores with MEAQ and refined factor scores, while controlling for the AUDIT and CUDIT-R total scores. Alcohol and cannabis use symptomatology were not found to have high multicollinearity with the FoS factor

scores or MEAQ total score, so the measures were included as covariates (i.e., VIF < 2.5; Senaviratna & Cooray, 2019).

Results

Aim 1

FoSI-SF Exploratory Factor Analysis. The scree plot and parallel analysis jointly yielded a three-factor solution for the data, which was further supported by sufficient eigenvalues prior to rotation (Factor 1: 6.86; Factor 2: 1.51; Factor 3: 1.03). The three-factor solution was evaluated for conceptual coherence (see Figure 1), such that Factor 1 corresponded to fear of loss of control and/or vulnerability (FoSI-V; items 1-3, 8-11), Factor 2 replicated the fear of darkness component from similar studies (FoSI-D; items 12-13; Pruiksma et al., 2014; Altan-Atalay et al., 2022), and Factor 3 was characterized as fear of re-experiencing traumatic nightmares (FoSI-N; items 4-7). Internal consistency was also determined to be high for all three factors ($\alpha = .80-.91$). Collectively, the three factors explained 64.59% of the total variance and displayed moderate correlations ($\rho = .40-.57$). FoSI-V accounted for a majority of the total variance (50.15%) and common variance (44.61%), as compared to the total (FoSI N: 9.03%; FoSI-D: 5.42%) and common variance of the other factors (FoSI N: 30.76%; FoSI-D: 29.20%). The three-factor model was replicated using maximum likelihood as an estimation method. All indicators were found to have salient factors loadings, acceptable communalities, and did not cross-load substantially (see Table 2).

As compared to the unidimensional ($\chi^2(62) = 229.29, p < .001$) and two-factor ($\chi^2(62) = 70.39, p < .001$) models, model comparisons confirmed that the three-factor model was the best-fitted and most parsimonious solution (CFI = .91, RMSEA = .11, SRMR = .06, TLI = .88), with the lowest AIC, BIC, and BIC_{adjust} values. Therefore, the EFA revealed a three-factor model in this study that is inconsistent with the

two-factor solution generated from the initial FoSI-SF validation study (Pruiksma et al., 2014). Of note, while FoSI-D was found to be possibly underdetermined (i.e., two indicators), this factor was retained given the support for a three-factor model as well as for subsequent Aim 2 analyses to assess its clinical utility among trauma-exposed individuals with insomnia symptoms.

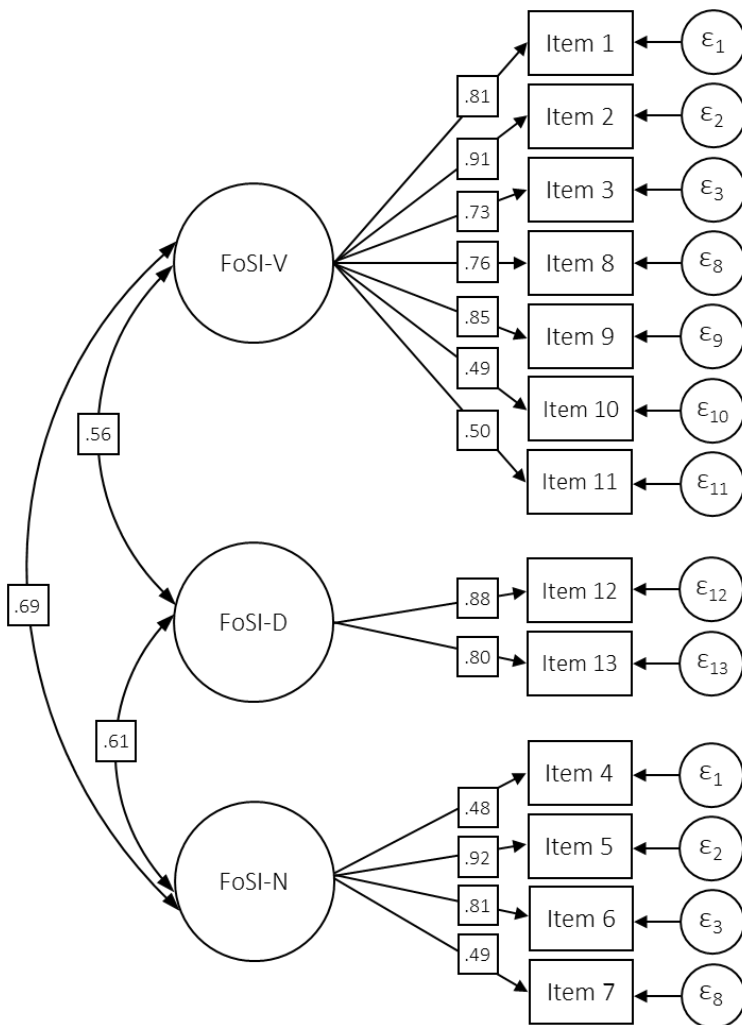


Figure 1. Factor structure of the Fear of Sleep Inventory - Short Form (FoSI-SF) with factor loadings and inter-factor correlations. FoSI-V: Fear of Loss of Control and/or Vulnerability; FoSI-D: Fear of Darkness; FoSI-N: Fear of Re-experiencing Traumatic Nightmares

FoSI-SF and Sleep Hygiene. The bivariate correlation revealed that the FoSI-SF was significantly positively associated with SHI ($p < .001$) and did not display strong evidence for discriminant validity ($\rho = .30$; see Table 3).

FoSI-SF and EA. Overall, the refined factor scores were characterized by low indeterminacy (Grice, 2001). The calculated validity coefficients all exceeded minimum recommendations (Validity = .91-.96) suggesting that the computed factor scores are adequate substitutes for the three factors in subsequent analyses. Univocality was also found to be satisfactory (maximum absolute difference in $r = .089$), which provides evidence that refined factor scores are not heavily influenced by variance from other factors in the analysis. Additionally, the results indicated that correlational accuracy was high (maximum absolute difference in $r = .086$), such that there were robust correlations between the refined factor scores and factors themselves.

Bivariate correlations determined that FoSI-V ($p = .00188$, $\rho = .22$) was significantly positively associated with the MEAQ total score, but not FoSI-D ($p = .253$, $\rho = .083$) and FoSI-N ($p = .634$, $\rho = .034$). However, the refined factor scores were not found to be significantly associated with the Behavioral Avoidance (FoSI-V: $p = .0511$, $\rho = .14$; FoSI-D: $p = .773$, $\rho = .021$; FoSI-N: $p = .243$, $\rho = .084$) or Distress Aversion subscales (FoSI-V: $p = .0589$, $\rho = .14$; FoSI-D: $p = .0623$, $\rho = .13$; FoSI-D: $p = .960$, $\rho = .0036$) of the MEAQ. For the remaining MEAQ subscales, only the Repression/Denial subscale displayed a significant positive association with a refined factor score (FoSI-V: $p < .001$, $\rho = .25$). Collectively, the results revealed that the FoSI-SF refined factor scores held weak associations with the MEAQ, suggesting that the underlying constructs are distinct.

Aim 2

FoSI-SF and PTSD/Insomnia. Comparisons between the full and subsetted samples revealed significantly higher FoSI-SF severity ($t = 4.62$, $d = .73$; see Table 4). With respect to the refined factor scores, FoSI-V was found to be significantly associated with the nightmares item ($p = .0270$, $\rho = .31$) and hypervigilance ($p = .0371$, $\rho = .30$). Conversely, FoSI-D was not found to be significantly associated with either PCL item (Nightmares: $p = .196$, $\rho = .19$; Hypervigilance: $p = .387$, $\rho = .13$). FoSI-N was determined to be significantly associated with the nightmares item ($p < .001$, $\rho = .46$), but not hypervigilance ($p = .945$, $\rho = .0101$). The overall multiple regression model for FoSI-V was significant ($F(3, 46) = 5.10$, $p = .00394$, $R^2 = .25$) and further indicated that hypervigilance ($t = 2.41$, $p = .0198$) was a significant predictor, but not nightmares ($t = 1.93$, $p = .0603$) nor the PCL summed item ($t = .20$, $p = .840$). The FoSI-D model was not significant ($F(3, 46) = 1.36$, $p = .266$, $R^2 = .082$), nor were any of the individual predictors (PCL 2: $t = 1.84$, $p = .0716$; PCL 17: $t = .062$, $p = .536$; PCL summed item score: $t = -.36$, $p = .723$). Finally, the model for FoSI-N was significant ($F(3, 46) = 7.66$, $p < .001$, $R^2 = .33$) as well as nightmares ($t = 4.75$, $p < .001$), whereas hypervigilance ($t = 1.50$, $p = .140$) and the PCL summed item score were not ($t = 1.99$, $p = .0523$). The results are consistent with our hypothesis that nightmares and hypervigilance would display a significant association with the corresponding refined factor scores, above and beyond the other PTSD symptoms.

The bivariate correlations revealed that FoSI-V ($p = .00786$, $\rho = .37$), rather than FoSI-D ($p = .287$, $\rho = .15$) and FoSI-N ($p = .711$, $\rho = .054$), was significantly associated with the PCL total score. Additionally, the ISI total score was found to be significantly related to FoSI-V ($p = .00652$, $\rho = .38$) and FoSI-N ($p < .001$, $\rho = .45$), but not FoSI-D ($p = .943$, $\rho = -.010$). However, the MEAQ was not significantly associated with either the PCL ($p = .0867$, $\rho = .25$) or ISI ($p = .739$, $\rho = .048$) total scores. Prior to adding covariates, FoSI-V was determined to be a significant predictor of the PCL total score ($t = 2.11$, $p = .0409$), but not FoSI-D ($t = .48$, $p = .637$), FoSI-N ($t = -.026$, $p = .980$), or the MEAQ ($t = 1.10$, $p = .277$), $F(4, 44) = 2.36$, $p = .0675$, $R^2 = .10$

(see Table 6). When controlling for alcohol and cannabis use, none of the individual predictors were found to be significant predictors of the PCL total score (FoSI-V: $t = 1.86, p = .0693$; FoSI-D: $t = .32, p = .750$; FoSI-N: $t = .55, p = .583$; MEAQ: $t = 1.62, p = .113$), $F(6, 42) = 2.61, p = .0306, R^2 = .17$ (see Table 6). Similarly, ISI total score was significantly predicted by FoSI-V ($t = 2.32, p = .0251$), FoSI-N ($t = 3.60, p < .001$), while the FoSI-D ($t = -1.96, p = .0569$) and MEAQ ($t = .57, p = .575$) did not contribute added predictive value, $F(4, 44) = 8.42, p < .001, R^2 = .37$ (see Table 5). Once the substance use covariates were added to the model, FoSI-D ($t = -2.29, p = .0274$), FoSI-N ($t = 3.81, p < .001$), and FoSI-V ($t = 2.18, p = .0351$) were revealed as significant predictors, but not the MEAQ ($t = 1.09, p = .284$), $F(6, 42) = 6.83, p < .001, R^2 = .42$ (see Table 5). Consistent with the trauma-induced model of insomnia (Werner et al., 2021), the refined factor scores appear to be uniquely-robust predictors of insomnia symptom severity above and beyond several other common forms of maladaptive avoidance. Additionally, One multivariate outlier was removed from analyses in the second aim due to the significant influence on the data (see Figure 2). In investigations relationships between the covariates, the AUDIT was found to have a positive association with the CUDIT ($p = .0186, \rho = .33$), which remained after the outlier was removed ($p = .0437, \rho = .29$). However, the MEAQ was not determined to have a significant association with either the AUDIT ($p = .303, \rho = -.15$) or CUDIT ($p = .537, \rho = -.090$).

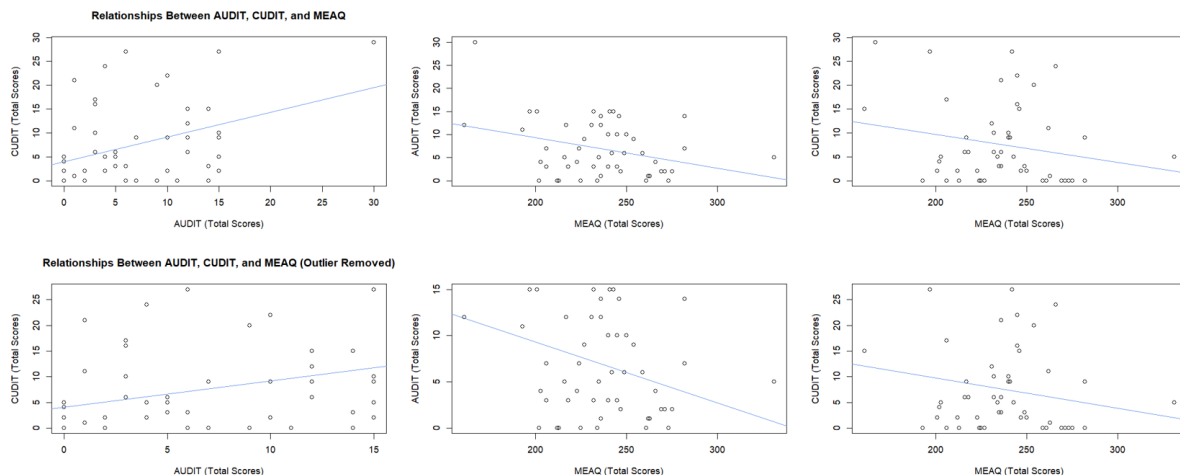


Figure 2. Row 1: Bivariate correlations between the AUDIT, CUDIT, and MEAQ for Aim 2 (n = 50). Row 2: Bivariate correlations between the AUDIT, CUDIT, and MEAQ for Aim 2 (n = 49) with single multivariate outlier removed. Notes: Linear regression line fitted for each of the plots. AUDIT: Alcohol Use Disorder Identification Test; CUDIT-R: Cannabis Use Disorder Identification Test- Revised; Multidimensional Experiential Avoidance Questionnaire.

Discussion

FoSI-SF Exploratory Factor Analysis

The present study utilized EFA to evaluate the psychometric properties of the FoSI-SF and subsequently investigate the relationships between the resulting factor scores and EA as well as PTSD and insomnia symptom severity in a sample of young adults. In contrast with the initial FoSI-SF validation study that yielded a two-factor solution (Pruiksma et al., 2014), our results specified the following three-factor model: (1) *fear of loss of control and/or vulnerability* (FoSI-V); (2) *fear of darkness* (FoSI-D); and (3) *fear of re-experiencing traumatic nightmares* (FoSI-N). Items captured by the FoSI-V component reflect internal responses and safety behaviors in response to perceived external threats in the sleeping environment. Akin to similar research investigating the factor structure of the FoSI-SF (Altan-Atalay et al, 2022, Pruiksma et al., 2014), FoSI-D is characterized by nocturnal fears of being in darkness and an associated avoidance strategy to mitigate discomfort. Whereas the third factor, FoSI-N, concerns the fear of re-experiencing traumatic reminders, such as nightmares and/or intrusive imagery, and subsequent delay of sleep initiation/reinitiation. Interestingly, the FoSI-V and FoSI-N appeared to be conceptually congruent with the two distinct pathways in which PTSD leads to the onset of FoS as proposed in the trauma-induced model of insomnia (Werner et al., 2021). More specifically, the deleterious process has been posited to develop due to unhelpful cognitions related to fear of loss of control and safety as well as the fear of re-experiencing the traumatic event through nightmares.

While the FoSI and FoSI-SF have been examined among a number of heterogeneous populations that include variable demographic background (e.g., age, race/ethnicity), clinical severity of traumatic

symptoms, and environment (e.g., country of origin, rural/urban area), the manner in which the items have loaded across factors has been inconsistent (Altan-Atalay et al., 2022; Brown et al., 2018; Drexler et al., 2019; Huntley et al., 2014; Pruiksma et al., 2014). Despite the present study being characterized by a population of college students akin to that of the initial validation study of the FoSI-SF (Pruiksma et al., 2014), the resulting factor structure significantly diverged from the pre-existing literature. While the FoSI-D was replicated in the present study (Altan-Atalay et al., 2022; Pruiksma et al., 2014), the factor remained underdetermined with only two indicators. As a result, the factor may continue to be characterized by limited stability and clinical utility in the application of the measure in future studies. In examining the language of the FoSI-SF items to investigate factor structure inconsistency, we identified a pattern of ambiguity in the underlying cause of fear and subsequent avoidance of sleep. More specifically, FoSI-SF items 4 (i.e., “I woke up in the night and I was terrified of returning to sleep”), 7 (i.e., “I was afraid to close my eyes”), 10 (i.e., “I stayed up late to avoid sleeping”), and 12 (i.e., “Being in the dark scared me”) all do not address the specific reason in which fear is experienced and/or why sleep is avoided. Importantly, FoSI-SF item 4 nearly cross-loaded onto FoSI-V, suggesting that respondents may have interpreted the question as capturing a sense of vulnerability in the sleeping environment, rather than a fear of re-experiencing traumatic nightmares. FoSI item 10 also displayed suboptimal communality with the remainder of the item pool, which is likely due to the vague, open-ended wording of the question (e.g., avoidance may be driven by non-specific avoidance as opposed to FoS). Consequently, different populations may continue to differentially interpret the FoSI-SF items, such that factor structure remains divergent. Altogether, the underlying factors implicated in FoS appear to be captured by the short-form version of the questionnaire, though specific items may require marked retooling to enhance item clarity and replicability of the factor structure.

FoSI-SF and EA

Following investigation of the construct overlap between FoS and EA, the FoSI-V displayed a significant positive relationship with the MEAQ total score. Interestingly, comparisons conducted with the remaining subscales indicated that the FoSI-V was positively associated with Repression/Denial, but not significantly associated with the Behavioral Avoidance ($p = .0511$) and Distress Aversion ($p = .0589$) subscales. The results suggest that individuals who report more pervasive concerns of vulnerability in the sleeping environment tend to be characterized by greater trait avoidance (e.g., stable across many settings), decreased emotional awareness (i.e., alexithymia), and increased efforts to dissociate from internal experiences. Concerningly, elevated alexithymia may lead to significantly increased PTSD symptom severity (Frewen et al., 2008). Dissociation from traumatic symptoms, particularly during the onset of PTSD, has been paradoxically linked with the perseveration of hyperarousal and re-experiencing symptoms as well (Monson et al., 2004). The relationship between hyperarousal symptoms and emotional numbing or other forms of dissociation may also develop into a positive feedback loop, further entrenching the symptoms (Tull & Roemer, 2003). Therefore, individuals with lower emotional clarity and/or high dissociative avoidance are likely to engage in chronic hypervigilance in response to distress, particularly in environments that prompt fear. Hyperarousal-induced insomnia may then be the impetus for which trauma-related fear responses become generalized to the sleeping environment (Pruiksma et al., 2014). Conversely, the FoSI-D and FoSI-N factors did not exhibit significant relationships with the MEAQ or any of the six subscales, which may indicate that the specificity of the factors is not captured by a measure of broad avoidance strategies. For example, the FoSI-D and FoSI-N factors are each uniquely described by context-dependent fear and sleep-specific avoidance.

FoSI-SF and PTSD/Insomnia

Consistent with the literature (Altan-Atalay et al., 2022; Drexler et al., 2019; Brown et al., 2018; Huntley et al., 2014; Pruiksma et al., 2014), the overall FoSI-SF measure was characterized by convergent validity

with insomnia and PTSD symptom severity while maintaining conceptual distinction from sleep hygiene, consistent with previous research (Pruiksma et al., 2014). Additionally, insomnia symptom severity and sleep hygiene were found to have higher construct overlap than FoS, which indicates that the FoSI-SF is a distinct measure (Drexler et al., 2019). Furthermore, FoSI-SF total scores among individuals with probable PTSD were significantly higher than individuals without notable traumatic symptom severity, which replicated the results of similar studies (Altan-Atalay et al., 2022; Drexler et al., 2019; Huntley et al., 2014; Pruiksma et al., 2014). Consistent with theoretical predictions (Werner et al., 2021), individuals with comorbid presentations of clinically-significant PTSD and insomnia displayed the most severe intensity of FoS. Collectively, the findings support the FoSI-SF as a conceptually-sensitive measure of trauma-induced insomnia that is most robust in presentations of comorbid PTSD and insomnia.

Further examination of the relationship between the FoSI-SF factor scores and PCL items provided support for specific pathways in which select PTSD symptoms may contribute to the onset of FoS among individuals with clinically-significant PTSD. Consistent with our hypotheses, hypervigilance significantly predicted the FoSI-V factor score after controlling for the collective influence of all other PTSD symptoms. Hypervigilance, a sequela of perceived loss of control, has been previously identified as a predictor of FoS intensity, which decreased in lockstep following CBT-I (Kanady et al., 2018b). Moreover, sensitivity to threat perception and subsequent hypervigilance may be further heightened among individuals with a history of traumatization in a sleep-related context (Huntley et al., 2014) and insidiously lead to prolonged insomnia symptom severity (Gupta & Sheridan, 2018a; Zayfert & DeViva, 2004). Additionally, nightmare symptom severity was a significant determinant of the FoSI-N factor score above and beyond the influence of the remaining PTSD symptoms. The result is consistent with emerging evidence, which has repeatedly found cross-sectional links between levels of FoS and nightmare distress/frequency (Kanady et al., 2018b; Krakow et al., 1995; Neylan et al., 1998; Pruiksma et al., 2011). FoS is even more

pronounced in individuals who report trauma-related nightmares as opposed to trauma-unrelated nightmares (Davis et al., 2007). Since the subsetting sample was characterized by clinically-significant PTSD, elevated FoSI-N factor scores likely indicate the presence, distress, and avoidance related to traumatic nightmares. Together, the two findings provide preliminary support for salient connections between hypervigilance and nightmares and components of FoS in line with both onset pathways proposed by the trauma-induced model of insomnia (Werner et al., 2021). For instance, hypervigilance is functionally conceptualized as a control strategy to reduce discomfort associated with unhelpful cognitions related to loss of control and vulnerability that become endemic in the sleeping environment (Kanady et al., 2018b). Similarly, distress associated with re-experiencing traumatic events while asleep has been posited as a precursor to the development of FoS (Krakow et al., 1995; Neylan et al., 1998). Dissimilar to previous findings (Altan-Atalay et al., 2022), FoSI-D was found to be uncorrelated with nightmares, hypervigilance, and overall PTSD symptom severity. Since the FoSI-D is underdetermined and possibly contains a vaguely-worded indicator, the results may indicate that the factor could benefit from evaluating construct clarity and/or validity.

While comparing the predictive validity of the FoSI-SF refined factor scores and MEAQ, FoS was deemed to be a far more robust predictor than EA in determining PTSD symptom severity. Although the MEAQ had significant associations between the ISI and PCL-5 in the overall sample, the measure was uncorrelated with both measures in the comorbid PTSD and insomnia subset. Previous literature has repeatedly identified a positive association between the MEAQ and PCL-5 in populations with low levels of PTSD (Dvorak et al., 2013) and those with probable PTSD (Lewis & Naugle, 2017; Lewis & Loverich, 2019). However, more recent research has also yielded null findings between the MEAQ and a measure of PTSD symptomatology (McKernan et al., 2019). Since self-report measures of EA necessitate an awareness of both unwanted internal experiences and avoidance behaviors (Thompson & Waltz, 2010),

the mixed findings may be explained by low emotional awareness, as supported by the earlier association with FoS. Moreover, individuals with probable PTSD are likely to report increased severity of dissociative symptoms as well as a diminished connection and understanding of their emotions (Powers et al., 2015). Therefore, measures of EA for trauma-exposed individuals may benefit from clinical interviews that more directly assess awareness of internal experiences, such that the heterogeneous pattern of avoidance strategies can be better captured.

The multiple regression model found that the PCL-5 was significantly predicted by the FoSI-V while covarying for the other factor scores and MEAQ. In addition to the FoSI-V no longer being a significant predictor of PCL-5 total scores ($p = .0693$), neither of the other factor scores nor MEAQ were found to be significant when controlling for the influence of alcohol and cannabis use. The finding first suggests that an increased perception of the sleeping environment as inherently vulnerable may be uniquely predictive of elevated traumatic symptom severity. Negative beliefs about loss of control may be more rigidly held among individuals with clinically-significant PTSD symptoms, such that they are unable to flexibly consider alternative beliefs and tend to see their thoughts as reality (Ben-Zion et al., 2018; Daneshvar et al., 2022). The additional significant variance explained by substance use may indicate the importance of investigating multiple forms of avoidance (Shorey et al., 2017), particularly those that are markedly comorbid with PTSD (Flanagan et al., 2016; Roberts et al., 2015). More precisely, alcohol (Brady et al., 2004) and cannabis (Boden et al., 2013) use have been functionally characterized as problematic forms of avoidance coping in the context of PTSD. Additionally, not only does substance use tend to precede the onset of PTSD (Jacobsen et al., 2001) and therefore FoS, but also is decidedly elevated among college student samples (O'Malley et al., 2002; Welsh et al., 2019).

Similarly, results from the other multiple regression models indicate that FoS is a comparatively better predictor of insomnia symptom severity than EA. More specifically, analyses revealed that both the FoSI-V and FoSI-N were significantly predictive of ISI total scores, while FoSI-D was not a significant explanatory variable ($p = .0569$). After the substance use covariates were added to the model, all of the factor scores retained their significance as predictors of insomnia symptom severity. The results indicate that distress related to fear of nightmares and fear of loss of control is likely to predict more severe insomnia symptoms among individuals with clinically-significant PTSD independent of substance use, which is consistent with previous findings (Drexler et al., 2019) as well as the trauma-induced model of insomnia (Werner et al., 2021). More specifically, trauma-exposed individuals are likely to report heightened cognitive and physiological pre-sleep arousal (Sinha, 2016), which may be exacerbated by feelings of helplessness due to intrusively re-experiencing traumatic nightmares (Davis et al., 2009; Werner et al., 2020). Mid-sleep awakenings evoked by nightmares may further fragment sleep and inimically interfere with extinction learning that typically occurs during REM (Pace-Schott et al., 2015; Riemann et al., 2012; Schierenbeck et al., 2008). As a result, sleep becomes significantly disturbed, delayed, and truncated by FoS. Additionally, trauma-related cognitions that often result in the overappraisal of environmental threats may become particularly exacerbated in context of sleep, where the ability to monitor surroundings is inherently in conflict with falling asleep (Werner et al., 2021). Consequently, individuals with clinically-significant PTSD likely struggle to simultaneously remain vigilant for threats at night time while seeking temporary respite from their cognitive and affective arousal in the sleep state. Due to the perpetual conflict between vigilance and rest, insomnia symptoms become significantly worsened and maintained over time. Additionally, the significant negative association between the FoSI-D and insomnia symptom severity was inconsistent with previous findings (Altan-Atalay et al., 2022) and contrary to our hypothesis. The finding suggests that the tendency to characterize darkness in the sleeping environment as fear-inducing predicted fewer disruptions in sleep. Perhaps, individuals with a fear of darkness are

better able to directly remedy their anxiety through overt avoidance strategies, such as sleeping with a light on, as compared to the prolonged impact of uncontrollable internal experiences and/or nightmares. Given the unexpected significant negative relationship with insomnia symptom severity and absence of a significant relationship with PTSD symptom severity, the FoSI-D may capture a more general intolerance of unwanted internal experiences that is not rooted in trauma. Considering that the FoSI-D is also likely underdetermined, further clarification of the factor is needed to discern if the indicators are capturing a truly distinct latent factor related to FoS or rather could be subsumed by the other, more clear components of the FoSI-SF. Altogether, the results provide convincing evidence for the clinical utility of the FoSI-SF as an incisive predictor of comorbid PTSD and insomnia above and beyond the MEAQ.

Strengths and Limitations

There are several merits to the present study, including the robust sample size, rigor in analyses, and novel investigation of EA in the context of comorbid PTSD and insomnia. The present study was characterized by a large sample ($N = 197$), which exceeded recommendations for EFA based on the number of indicators (Brown, 2015; Watkins, 2018) and was similar to other studies exploring the factor structure of the FoSI-SF (Brown et al., 2018; Pruiksma et al., 2014). In comparison with similar research, analyses conducted in this study utilized a more conservative approach, where potential factors that could provide significant explanatory power in PTSD and insomnia symptom severity were included (i.e., alcohol and cannabis use). In line with a seminal review of EFA methodology (Brown, 2015), the present study was the first to compute refined factor scores for the FoSI-SF in an effort to reduce the bias that coarse factor scores typically engender (Grice, 2001). Additionally, the refined factor scores were further evaluated for indeterminacy (i.e., validity coefficients, univocality, and correlational accuracy) to sufficiently characterize the degree of factor score estimation (Brown, 2015). Furthermore, the present study contributed preliminary evidence in support of a significant relationship between FoS and EA as

well as a comparative prediction of comorbid trauma-related and insomnia symptom severity among individuals with clinically-significant PTSD.

An important limitation to consider while interpreting the results of the present study is the cross-sectional design. Causal claims about the nature of the temporal relationship between FoS, insomnia, and PTSD cannot be drawn as a result. Therefore, longitudinal studies are necessary to disentangle the influence of PTSD symptoms in the onset of FoS and subsequent maintenance of hypervigilance and safety behaviors in the sleeping environment consistent with the trauma-induced model of insomnia (Werner et al., 2021). Since the study sought to replicate a college student population akin to the initial validation study (Pruksma et al., 2014), the resulting sample is similarly characterized by a narrow age range and predominantly female gender composition, and therefore has limited generalizability to more representative populations. While the sample was also characterized by a marked number of individuals with clinically-significant PTSD symptoms, confidence in the accuracy of a PTSD diagnosis could have been enhanced through use of an in-depth diagnostic assessment rather than a self-report questionnaire. Furthermore, pre-sleep hyperarousal and insomnia symptoms were wholly interpreted through the PCL-5, ISI, and FoSI-SF scores, and did not include physiological measures that assessed autonomic arousal (i.e., electrocardiogram, polysomnography) or objective measures (e.g., actigraphy).

Future Directions and Conclusions

Based on the current state of the literature, FoS could most benefit from examining the temporal assumptions of the trauma-induced model of insomnia (Werner et al., 2021) with particular focus on elucidating the onset and maintenance of the process in individuals with acute, chronic, and remitted PTSD. An important foundational step in achieving this goal is refining the operationalization and measurement of FoS, such that unhelpful cognitions about loss of control in the sleeping environment

(i.e., perception of heightened vulnerability), affective experiences related to sleep (i.e., fear), and safety behaviors implemented to maintain vigilance and reduce distress (e.g., delaying sleep onset) are coherently and distinctly captured. The present study provided additional evidence for the manner in which the FoSI-SF is characterized by a diverging factor structure across samples, ambiguously-constructed indicators, and inclusion of an underdetermined factor (i.e., FoSI-D). Therefore, the measurement could be significantly improved by more closely adhering to current conceptualizations of FoS (i.e., trauma-induced model of insomnia; Werner et al., 2021) and work towards adapting the items to address specific forms of trauma (e.g., increased FoS for traumas occurring in sleep context; Huntley et al., 2014) and/or cross-cultural differences (Altan-Atalay et al., 2022; Drexler et al., 2019). Additionally, the FoSI-SF could benefit from retooling the current items to more explicitly measure the hallmark components of FoS (i.e., fear of loss of control/vulnerability, behavioral avoidance in sleeping environment) and alleviate the current concerns around the broad interpretability of some items. The item pool could be further expanded in the refinement process so that factors would be more robust and no longer underdetermined (i.e., FoSI-D). Once the operationalization of FoS has been further honed, the resulting measure could be adequately utilized in service of the development, examination, and integration of interventions into pre-existing treatment regimens for comorbid PTSD and insomnia, such that maintenance of the process is disrupted (e.g., reduction in nocturnal hypervigilance, more flexible cognitions about loss of control). Finally, the methodological rigor of study design could be enhanced by including an assessment of autonomic arousal with ambulatory equipment to determine potential mediators between FoS and residual insomnia symptoms.

In summary, the present study extended the previous literature by providing support for the cross-sectional, positive relationship between FoS, PTSD, and insomnia, as well as distinguishing the process from other sleep-related measures (i.e., sleep hygiene). Additionally, we identified preliminary evidence

for the positive relationship between FoS and EA broadly as well as dissociative avoidance and/or low emotional awareness subtype (i.e., Repression/Denial subscale). The finding is important in contextualizing hyperarousal symptoms and use of safety behaviors in the sleeping environment, which are heightened among trauma-exposed individuals who engage in dissociative avoidance (e.g., emotional numbing; Flack et al., 2000; Pietrzak et al., 2014). Moreover, the present study provided compelling evidence for the unique associations of hypervigilance and trauma-related nightmares in FoS severity, which could benefit from further examination such that temporal claims can be made regarding the onset and perpetuation of the deleterious process. The factors underlying FoS were also found to have differential associations with PTSD and insomnia symptom severity, which underscores the importance of including multidimensional measurements of pre-sleep physiological, cognitive, and affective hyperarousal as well as the resulting unhelpful avoidance behaviors. Overall, the current study provided additional insight into the manner in which FoS is uniquely implicated in comorbid PTSD and insomnia symptom severity and further identified novel avenues for clarifying the process in support of treatment considerations. Moreover, the resulting three-factor structure provided further support for the critical need to rework a number of FoSI-SF items prior to the application of the measure in treatment development for FoS.

Chapter 5: Summary and Clinical Implications

Summary of Dissertation

Initially, experiential avoidance (EA) was investigated as a transdiagnostic process implicated in the etiology, worsening, and maintenance of residual insomnia following remission of PTSD symptoms. The literature review evaluated the contributions of EA in the onset and maintenance of insomnia, PTSD, and comorbid presentations. There is evidence to suggest that EA is involved in the pathogenesis (Gil, 2005; Kumpula et al., 2011; Thompson et al., 2018), perpetuation (Orcutt et al., 2014), and exacerbation of PTSD symptom severity (Bardeen, 2015; Meyer et al., 2013; Plumb et al., 2004; Thompson & Waltz, 2010; Tull et al., 2004). Furthermore, research indicated that EA was at the heart of cognitive and physiological pre-sleep arousal that precedes insomnia (Perlis et al., 1997; Palagini et al., 2018; Riemann et al., 2010). In particular, pre-sleep hyperarousal becomes aggravated by the valence and rigidity with which dysfunctional sleep-related beliefs are experienced (Kalmbach et al., 2018; Ong et al., 2012) and leads to overt behavioral avoidance that perpetuates insomnia symptoms. Within the context of co-occurring PTSD and insomnia symptoms, the manner in which trauma-related hyperarousal disrupted sufficient, consolidated, and restorative sleep was found to be notably distinct from that in primary insomnia disorder. More precisely, fear of sleep (FoS) was determined to be an important pathogenic process involved in the maintenance of insomnia symptoms following trauma exposure (Werner et al., 2021). While there is preliminary evidence that suggests CBT-I may reduce FoS (Kanady et al., 2018a), there are no current interventions that explicitly target the process, despite the significant and distressing functional consequences of residual insomnia (Hertenstein et al., 2019; López et al., 2019; McHugh et al., 2014). Ultimately, the review provided a comprehensive assessment of the role of EA in comorbid PTSD and insomnia as well as an evaluation of empirically-supported insomnia treatments as potential pathways to reduce FoS severity. Nonetheless, the precursory examination of FoS conducted during the first chapter necessitated a more nuanced review in support of future, tailored interventions.

An extensive exploration of the trauma-induced model of insomnia revealed that the deleterious process develops following trauma exposure and co-occurs with acute insomnia symptoms (Sinha, 2016; Werner et al., 2020; Werner et al., 2021). More specifically, FoS is posited to arise from (1) distressing thoughts regarding fear of loss of control in the sleeping environment; and (2) fear of re-experiencing traumatic nightmares. Trauma-related internal experiences then become entrenched by hypervigilance-driven safety behaviors (e.g., sleeping with lights on), which seek to maintain control in the sleeping environment and prevent or delay traumatic nightmares (Pruiksma et al., 2014). As a result, FoS is conceptualized to mechanistically detach from trauma-related symptoms and become embedded in sleep-specific pathways over time, such that the process is unfettered in the perpetuation of insomnia even after PTSD remission (Werner et al., 2021). However, FoS has been operationalized inconsistently across the literature, such that the multidimensional conceptualization has not been adequately examined. Consequently, the construct was compared and contrasted with EA to determine the manner in which the two processes may overlap and thus benefit from similar treatment approaches. FoS was identified as a form of contextual avoidance (i.e., safety behaviors in the sleeping environment; Gupta & Sheridan, 2018b; Hull et al., 2016; Kanady et al., 2018b), whereas EA was characterized as trait-based avoidance that involves numerous strategies (i.e., generalized across many contexts; Gámez et al., 2011; Hayes et al., 1996). Since the processes appear to be closely linked as avoidance-driven constructs, further exploration is needed to disentangle their respective contributions to trauma-induced insomnia symptoms.

A literature review of FoS measures was conducted to characterize the current state of research, identify limitations, and provide impetus for gaps to be further addressed. Conclusions from the review indicated that the Fear of Sleep Inventory Short Form (FoSI-SF; Pruiksma et al., 2014) significantly improved upon

concerns identified in the larger inventory (i.e., FoSI; Zayfert et al., 2006), including the contamination of the factor structure by PTSD-specific symptoms. An exploratory factor analysis (EFA) of the FoSI-SF yielded a two-factor structure composed of (1) *fear of loss of control*; and (2) *fear of darkness* (Pruiksma et al., 2014). Subsequent studies identified diverging factor structures for the FoSI-SF, including an underdetermined factor (i.e., fear of darkness; Altan-Atalay et al., 2022; Brown et al., 2018; Drexel et al., 2019; Huntley et al., 2014), which calls for additional research to replicate the results of the initial validation study (Pruiksma et al., 2014). All of the projects reproduced significant positive associations between FoS and insomnia and/or trauma-related symptoms (Altan-Atalay et al., 2022; Brown et al., 2018; Drexel et al., 2019; Pruiksma et al., 2014) and determined the FoSI-SF to possess robust internal validity. The FoSI-SF was found to have discriminant validity from measures of sleep disruption (i.e., sleep hygiene; Pruiksma et al., 2014) and rumination (Altan-Atalay et al., 2022) and shown to be a sensitive measure of comorbid insomnia among individuals with clinically-significant trauma symptoms (Altan-Atalay et al., 2022; Drexel et al., 2019; Pruiksma et al., 2014). Altogether, the literature review found promising support for the FoSI-SF as a robust predictor of trauma-induced insomnia symptoms, though more nuanced investigations of the psychometric properties uncovered concerning limitations. Given the scarcity of research involving the FoSI-SF and potential of clinical utility, there is a critical need to replicate the two-factor structure of the initial validation study among a similar sample.

The present study sought to address current gaps in the literature by (1) evaluating the psychometric properties of the FoSI-SF in a population of college students, including a confirmation of convergent validity with EA and discriminant validity with the sleep hygiene; and (2) validate components of the trauma-induced model of insomnia and compare the predictive validity of EA and FoS in the symptom severity of PTSD and insomnia. In the first aim, the study utilized EFA to identify the factor structure of the FoSI-SF and compute subsequent refined factor scores for correlation tests. In the second aim, the

sample was subsetting to individuals with clinically-significant PTSD symptoms (i.e., PCL-5 $\geq$ 31) and at least moderate insomnia symptoms (i.e., ISI $\geq$ 10). The refined factor scores from the first aim were applied in multiple regression analyses to clarify the differential association of trauma-related symptoms implicated in the onset of FoS as well as overall symptom severity. Results of the first aim revealed a three-factor structure described as (1) *fear of loss of control and/or vulnerability* (FoSI-V); (2) *fear of darkness* (FoSI-D); and (3) *fear of re-experiencing traumatic nightmares* (FoSI-N), mixed support for convergent validity with EA (i.e., only the FoSI-V factor), and no strong evidence for discriminant validity with sleep hygiene. The second aim found compelling support for the significant contributions of hypervigilance and nightmares into predicting FoS severity, particularly among the FoSI-V and FoSI-N factors, respectively. Additionally, results further indicated that the FoSI-SF factor scores were far superior than EA in predicting PTSD and insomnia symptoms severity, even when controlling for current alcohol and cannabis use.

The three-factor structure, while conceptually coherent, suggests that the FoSI-SF may need to be retooled to improve consistency, clarity, and utility in measurement. In addition to the FoSI-D being underdetermined, several indicators were interpreted as vague and incongruent with the current conceptualization of FoS (e.g. fear of darkness may be orthogonal from trauma-related fears). However, the present study replicated a similarly weak correlation between the FoSI-SF and sleep hygiene index (Mastin et al., 2006) as first identified in the initial validation study (Pruiksma et al., 2014). The FoSI-SF displayed limited convergent validity with the multidimensional experiential avoidance questionnaire (MEAQ; Gámez et al., 2011), such that only the total score and Repression/Denial subscale were significantly correlated with the FoSI-V. The result highlights the possible role of low emotional awareness and dissociative avoidance in exacerbating the frequency of trauma-related hyperarousal/re-experiencing symptoms (Monson et al., 2004). In support of the trauma-induced model of insomnia (Werner et al.,

2021), the second aim found preliminary support for trauma-related symptoms implicated in FoS. While nightmares have been frequently identified in relation to FoS (Kanady et al., 2018b; Krakow et al., 1995; Neylan et al., 1998; Pruiksma et al., 2011), the present study was among the first to specifically evaluate the role of hypervigilance in uniquely predicting FoS severity (Kanady et al., 2018b). Furthermore, the superiority of FoS over EA in predicting comorbid PTSD and insomnia symptom severity is consistent with previous studies investigating the predictive utility of contextual measures of avoidance (Kirk et al., 2019). Given the marked sensitivity of the FoSI-SF in identifying comorbid PTSD and insomnia presentations, the measure has high potential, despite current limitations, for use in clinical settings.

Clinical Implications

Future research can continue to build off findings from the present study with particular attention toward further validating aspects of the FoSI-SF, establishing a clinical threshold for assessment, and identifying effective treatment approaches. The construct validity of the FoSI-SF could be refined by applying the multidimensional operationalization of FoS as diagnostic criteria, such that fear in the context of sleep, unhelpful cognitions about loss of control and safety, and subsequent maladaptive safety behaviors are explicitly and distinctly measured (Werner et al., 2021). Once the items have been clarified through a retooling process, a clinical threshold could be determined to increase the clinical utility for screening purposes (Pigeon & DeViva, 2021). Consistent with the findings of the present study and past research (Drexler et al., 2019; Werner et al., 2020), FoS appears to be most pronounced among individuals with clinically-significant PTSD and comorbid insomnia symptoms, and lowest among individuals without trauma-related symptoms. For example, in the present study, we found mean FoSI-SF scores for the subsetting sample in the second aim were 15.32 as compared to 2.29 in among individuals without trauma-related or insomnia symptoms. Since FoS appears to be a valid clinical indicator of co-occurring PTSD and insomnia symptoms, cognitive-behavioral therapy for insomnia (CBT-I) could be adapted to

address concerns around safety and nightmares in the context of sleep (e.g., restructuring to achieve more balanced thoughts; Lancee et al., 2008; Riemann et al., 2017). Given the persistence of hyperarousal symptoms following trauma treatment (Larsen et al., 2019; Schnurr & Lunney, 2019; Tanev et al., 2022; Tripp et al., 2020; Zayfert & Deviva, 2004), interventions that target the downregulation of autonomic activity, such as those based in acceptance and mindfulness principles (Ong et al., 2012; Ong et al., 2014), may also be beneficial in the reduction of hypervigilance that maintains residual insomnia. Nonetheless, future interventions could most benefit from taking a multifaceted approach toward reducing components of FoS in an effort to reduce sleep onset delay and fragmentation due to hyperarousal, thereby increasing sleep duration and quality for trauma survivors.

APPENDIX: LIST OF TABLES

Table 1

Demographic Characteristics for Study Participants (N = 197)

Variable	M or n	SD or %
Age	19.54	1.54
Gender		
Man	62	31.47
Woman	132	66.50
Other	4	2.03
Race/Ethnicity		
American Indian or Alaska Native	2	1.02
Asian	14	7.11
Black or African American	4	2.03
Hispanic or Latinx	13	6.60
Multiracial	39	19.80
Other	3	1.52
White	122	61.93

Table 2

FoSI-SF Items and Factor Loadings for Aim 1 Sample (N = 197)

Item	Factor 1	Factor 2	Factor 3
1. I was fearful of letting my guard down while sleeping.	.81	-.20	.098
2. I tried to stay as alert as I could while lying in bed.	.91	-.13	-.037
3. I was fearful of the loss of control that I experience during sleep.	.73	-.051	.17
4. I woke up in the night and I was terrified of returning to sleep.	.32	.17	.48
5. I avoided going to sleep because I thought I would have really bad dreams.	.015	-.033	.92
6. I awoke in the middle of the night from a nightmare and avoided returning to sleep because I might go back into the nightmare.	-.0094	-.029	.81
7. I was afraid to close my eyes.	.14	.29	.49
8. I felt that it was dangerous to fall asleep.	.76	.096	.036
9. I was aware of being especially vulnerable when I'm asleep.	.85	.083	-.088
10. I stayed up late to avoid sleeping.	.49	.24	-.025
11. I tried to stay alert to any strange noises while going to sleep.	.50	.25	.033
12. Being in the dark scared me.	-.080	.88	-.041
13. I slept with a light on to feel safer.	-.065	.80	.033

Note. Factor 1: Fear of Loss of Control and/or Vulnerability (FoSI-V); Factor 2: Fear of Darkness (FoSI-D); Factor 3: Fear of Re-experiencing Traumatic Nightmares (FoSI-N).

Table 3*Convergent and Discriminant Validity Coefficients in Aim 1 Sample (N = 197)*

Measure	1	2	3	4	5
1. FoSI-SF	-				
2. ISI	.43**	-			
3. MEAQ	.20*	.25**	-		
4. PCL-5	.49**	.44**	.33**	-	
5. SHI	.30**	.51**	.28**	.35**	-

Note. * $p < .01$ (two-tailed); ** $p < .001$ (two-tailed). FoSI-SF: Fear of Sleep Inventory - Short Form; ISI: Insomnia Severity Index; MEAQ: Multidimensional Experiential Avoidance Questionnaire; PCL-5: PTSD Checklist for DSM-5; SHI: Sleep Hygiene Index.

Table 4

Means, Standard Deviations, and Comparisons of Variables for Aim 1 (N = 197) and Aim 2 Samples (n = 50)

Measure	Aim 1		Aim 2		<i>t</i>	Cohen's <i>d</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
AUDIT	5.98	5.25	6.92	6.09	.099	.17
CUDIT-R	5.76	7.14	7.60	8.33	1.43	.25
FoSI-SF	5.93	8.17	12.32	10.6	4.62**	.73
ISI	10.55	5.75	16.40	4.42	6.69**	1.06
MEAQ	226.41	29.17	236.38	30.28	2.09*	.34
PCL-5	26.18	17.81	46.88	11.85	7.78**	1.24
SHI	24.99	6.61	29.04	6.09	4.12**	.62

Note. * $p < .05$ (two-tailed); ** $p < .001$ (two-tailed). AUDIT: Alcohol Use Disorder Identification Test; CUDIT-R: Cannabis Use Disorder Identification Test- Revised; FoSI-SF: Fear of Sleep Inventory - Short Form; ISI: Insomnia Severity Index; MEAQ: Multidimensional Experiential Avoidance Questionnaire; PCL-5: PTSD Checklist for DSM-5; SHI: Sleep Hygiene Index. The Aim 1 sample was subsetting for Aim 2 analyses based on moderate symptom severity for insomnia (ISI total score ≥ 10) and clinically-significant PTSD symptom severity (PCL-5 total score ≥ 31).

Table 5*Regression Coefficients for Predicting Insomnia Symptom Severity in Aim 2 Sample (n = 50)*

Variable	R ²	ΔR ²	B	SE	t	p
Step 1	.37**	-				
FoSI-D			-.83	.43	-1.96	.0569
FoSI-N			1.49	.41	3.60	.00**
FoSI-V			.92	.40	2.32	.0251*
MEAQ			.011	.019	.57	.575
Step 2	.42**	.056				
AUDIT			.15	.10	1.46	.151
CUDIT-R			.10	.070	1.49	.144
FoSI-D			-.94	.41	-2.29	.0274*
FoSI-N			1.64	.43	3.81	.00**
FoSI-V			.84	.39	2.18	.0351*
MEAQ			.020	.018	1.09	.284

Note. * $p < .05$ (two-tailed); ** $p < .001$ (two-tailed). AUDIT: Alcohol Use Disorder Identification Test; CUDIT-R: Cannabis Use Disorder Identification Test- Revised; FoSI-D: Fear of Darkness; FoSI-N: Fear of Re-experiencing Traumatic Nightmares; FoSI-V: Fear of Loss of Control and/or Vulnerability; Multidimensional Experiential Avoidance Questionnaire. Insomnia symptom severity was represented by the total score for the Insomnia Severity Index.

Table 6*Regression Coefficients for Predicting PTSD Symptom Severity in Aim 2 Sample (n = 50)*

Variable	R ²	ΔR ²	B	SE	t	p
Step 1	.10	-				
FoSI-D			.64	1.35	.48	.637
FoSI-N			-.034	1.32	-.026	.978
FoSI-V			2.66	1.26	2.11	.0409*
MEAQ			.066	.060	1.10	.277
Step 2	.17*	.066				
AUDIT			.29	.33	.86	.397
CUDIT-R			.41	.22	1.82	.0766
FoSI-D			.42	1.32	.32	.750
FoSI-N			.76	1.38	.55	.583
FoSI-V			2.31	1.24	1.86	.0693
MEAQ			.096	.059	1.62	.113

Note. * $p < .05$ (two-tailed). AUDIT: Alcohol Use Disorder Identification Test; CUDIT-R: Cannabis Use Disorder Identification Test- Revised; FoSI-D: Fear of Darkness; FoSI-N: Fear of Re-experiencing Traumatic Nightmares; FoSI-V: Fear of Loss of Control and/or Vulnerability; Multidimensional Experiential Avoidance Questionnaire. PTSD symptom severity was represented by the total score for the PTSD Checklist for DSM-5.

Table 7

Hazardous Consumption and Substance Use Disorder Counts for Alcohol and Cannabis for Aim 1 (N = 197) and Aim 2 Samples (n = 50)

Measure	Aim 1		Aim 2	
	<i>Hazardous Consumption Count (%)</i>	<i>Substance Use Disorder Count (%)</i>	<i>Hazardous Consumption Count (%)</i>	<i>Substance Use Disorder Count (%)</i>
AUDIT	47 (23.86)	14 (7.11)	13 (26.00)	6 (12.00)
CUDIT-R	24 (12.18)	38 (19.29)	7 (14.00)	12 (24.00)

Note. AUDIT: Alcohol Use Disorder Identification Test; CUDIT-R: Cannabis Use Disorder Identification Test – Revised. AUDIT: Hazardous consumption = 8-14 total score; probable moderate-severe substance use disorder = 15 or greater total score. CUDIT: Hazardous consumption = 8 or greater total score; probable substance use disorder = 12 or greater total score.

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