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Insomnia and Emotion Dysregulation: Independent and Interactive Associations with Posttraumatic Stress Symptoms among Trauma-Exposed Smokers

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Abstract

Introduction—Traumatic event exposure is common among cigarette smokers, and elevated posttraumatic stress symptoms (PTSS) are associated with increased smoking levels. As such, the current study examined factors that may contribute to elevated PTSS among trauma-exposed smokers. Insomnia and emotion dysregulation may be particularly relevant among smokers, and are each associated with PTSS. However, it remains unclear whether these factors are associated with PTSS after accounting for the effects of dispositional factors and each other, and whether they may interact to predict PTSS. Thus, the current study sought to test whether insomnia and emotion dysregulation are independently associated with PTSS after accounting for negative affectivity and number of traumas experienced, and to investigate the potential interactive influence of these factors on PTSS.

Method—Hypotheses were tested cross-sectionally among a community sample of trauma-exposed individuals who presented for smoking cessation treatment (n=349).

Results—Results demonstrated that insomnia and emotion dysregulation each predicted elevated PTSS after controlling for the other, negative affectivity and number of traumas experienced. In addition, the interaction between insomnia and emotion dysregulation was significant, such that higher levels of insomnia and emotion dysregulation were associated with the most severe PTSS.

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Contributors

The first author designed the idea for the manuscript, analyzed the data, and wrote the introduction, results, and discussion. The second author provided feedback on all drafts and assisted in writing the introduction. The third author provided feedback on drafts and wrote the methods. The fourth author provided feedback on drafts and collected data for the study. The fifth author provided feedback on all drafts and collected data for the study. All authors have contributed to and have approved the final manuscript.

Conflicts of interest

The authors have no conflicts of interest to disclose.

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Limitations—Future research should examine these factors among a clinical sample of individuals with PTSD, as well as utilize prospective designs.

Conclusions—Findings highlight the roles of insomnia and emotion dysregulation in contributing to elevated PTSS among trauma-exposed smokers, and the potential importance of targeting these factors in the context of PTSD treatment.

Keywords

Posttraumatic stress; Insomnia; Emotion regulation; Smoking

Introduction

Exposure to a traumatic event is common among smokers (Ganz, 2000), with smokers reporting traumatic event exposure at greater than twice the rate of non-smokers (Vest et al., 2002). While responses to traumatic events vary, most individuals experience some degree of posttraumatic stress symptoms (PTSS) in the acute aftermath of a trauma (Kilpatrick and Resnick, 1993). These PTSS include re-experiencing the event via nightmares, flashbacks, and intrusive memories; avoidance of reminders of the event; numbing symptoms such as an inability to feel positive emotions and loss of interest in previously enjoyed activities; and hyperarousal symptoms, such as hypervigilance and trouble sleeping (Ballenger et al., 2000). A majority of individuals exposed to a traumatic event recover from these symptoms within a few months (Kilpatrick and Resnick, 1993). However, a significant minority continue to experience PTSS, with some developing associated clinically significant distress or impairment and meeting criteria for posttraumatic stress disorder (PTSD). Both elevated PTSS severity and PTSD diagnosis are associated with a number of negative outcomes, including current smoking status as well as elevated levels of smoking (for a review, see Feldner et al., 2007). Considering research linking elevated PTSS with smoking, it is important to identify factors that may contribute to increased PTSS severity among this population.

One factor shown to contribute PTSS, sleep disturbance, may be particularly relevant among smokers, as disturbed sleep is common in this population (Wetter and Young, 1994). Specifically, insomnia is often reported among smokers and is the most commonly endorsed PTSS among individuals with PTSD (Neylan et al., 1998; Wetter and Young, 1994). While insomnia was previously viewed as a consequence of psychological disorders, as well as posttraumatic stress, more recent research demonstrates that insomnia may play a causal and/or maintaining role in various mental health conditions (Harvey, 2008). Specifically, insomnia prospectively predicts episodes of depression (Perlis et al., 2006), the onset of problematic alcohol and substance use (Wong et al., 2004), and anxiety disorder development (Breslau et al., 1996). In addition, experimental research has indicated that sleep is critical for mood regulation and appropriate emotional responding (Dinges et al., 1997; Yoo et al., 2007), as well as cognitive functioning (e.g., the encoding and consolidation of memories, particularly emotional memories; Walker and Stickgold, 2006), leading some to hypothesize that insomnia affects mental health conditions via impaired emotional and cognitive functioning (e.g., Harvey, 2008).

In terms of PTSS, previous research indicates insomnia is associated with elevated PTSS, with prospective studies finding that insomnia predicts development of PTSD after traumatic event exposure (Koren et al., 2002; Wright et al., 2011). In addition, longitudinal studies show that insomnia often does not remit spontaneously or through PTSD treatment alone, and is associated with ongoing PTSS (Pigeon et al., 2013), whereas treating insomnia through CBT-I may lead to a reduction in PTSS (Smith et al., 2005). There are several pathways through which sleep disturbances such as insomnia are thought to impact PTSS, including cognitive impairment, emotional disturbances, and impaired processing of emotional memories due to sleep loss (Goldstein and Walker, 2014; Walker, 2009). Specifically, studies show that sleep loss is associated with increased negative emotions such as irritability, as well as emotional lability and increased intensity of both subjective negative emotions and neural reactivity to negative emotional cues, in combination with decreased positive emotions during rewarding activities (Dinges et al., 1997; Horne, 1985; Yoo et al., 2007; Zohar et al., 2005). Thus, sleep loss due to insomnia may be associated with emotional disturbance and difficulties coping with the negative emotions associated with posttraumatic stress, leading to increased PTSS (Germain, 2013).

Another factor predictive of elevated PTSS is emotion dysregulation (Tull et al., 2007). Gratz and Roemer conceptualized emotion dysregulation as problems with the ability to understand and differentiate emotions, as well as difficulties modulating emotions (2004). Specifically, emotion dysregulation may include problems with: 1) awareness and understanding of emotions, 2) acceptance of emotions, 3) control of impulsive behaviors and achievement of goals when distressed, and 4) use of appropriate emotion regulation strategies to modulate emotional responses (Gratz and Roemer, 2004). Emotion dysregulation may exacerbate negative emotional states by leading to increased physiological arousal, distress, and avoidance (Cisler et al., 2010). Indeed, recent research suggests that difficulties with emotion regulation may play a role in a variety of psychological disorders (Aldao et al., 2010). Finally, emotion dysregulation may be a factor of particular importance among smokers, as smoking has often been conceptualized as a maladaptive attempt to regulate one's emotions (e.g., Gehricke et al., 2007).

Both cross-sectional and prospective studies show that emotion dysregulation predicts elevated PTSS severity (Bardeen et al., 2013; Ehring and Quack, 2010; Tull et al., 2007). Some have hypothesized that emotion regulation is particularly important in the context of posttraumatic stress because intense negative emotions, such as those triggered by reminders of the traumatic event, are common in the acute aftermath of a trauma. Thus, individuals who have difficulty regulating negative emotions and arousal associated with posttraumatic stress may be at risk for developing elevated PTSS or PTSD (Bardeen et al., 2013; Tull et al., 2007). For example, individuals with emotion regulation difficulties may see their emotions as uncontrollable, and develop a fear of internal and external triggers leading to emotional reactions, such as trauma reminders (Bardeen et al., 2013; Bouton et al., 2001). In addition, limited access to emotion regulation strategies may contribute to avoidance of these reminders, which would prevent emotional processing of the event (Bardeen et al., 2013).

Despite research indicating that both insomnia and emotion dysregulation predict PTSS (Bardeen et al., 2013; Koren et al., 2002; Tull et al., 2007; Wright et al., 2011), and that insomnia is associated with emotional disturbances (Walker, 2009), relatively little research has examined these factors together in terms of PTSS. Furthermore, no known research to date has examined these factors in a smoking sample. One recent cross-sectional study of patients with comorbid PTSD and alcohol use disorder (AUD) found although insomnia and emotion dysregulation are related, both factors are independently associated with PTSS (Fairholme et al., 2013). However, this study did not account for dispositional factors predictive of PTSS, such as negative affectivity (Monson et al., 2004). In addition, it is unclear whether insomnia and emotion dysregulation may interact to predict PTSS. Because sleep loss is associated with emotional disturbances (Yoo et al., 2007; Zohar et al., 2005), it is possible those with elevated levels of emotion dysregulation may be more susceptible to the effects of insomnia on PTSS. For example, sleep loss due to insomnia may be associated with increased negative emotional reactivity and intensity when confronted with negative emotional cues, such as reminders of the trauma (Yoo et al., 2007; Zohar et al., 2005). These negative emotional reactions may be amplified by existing problems with emotion dysregulation, such as a lack of understanding of emotions, limited access to effective emotion regulation strategies, and problems with impulse control when distressed. Thus individuals may experience increased anxiety regarding the event and its reminders due to the intensity and perceived uncontrollability of associated negative emotions (Bardeen et al., 2013; Bouton et al., 2001). In addition, a lack of effective emotion regulation strategies may lead to avoidance of reminders of the event and thus elevated PTSS (Bardeen et al., 2013; Foa and Kozak, 1986).

The current study sought to, 1) replicate and extend previous research by testing whether elevated levels of insomnia and emotion dysregulation each predict more severe PTSS after accounting for the effects of the other, negative affectivity, and number of traumas experienced, and 2) to test the interactive influence of insomnia and emotion dysregulation on PTSS. We chose to investigate these hypotheses among a sample of trauma-exposed daily smokers, as they represent a population with increased risk for PTSS and sleep disturbance (Feldner et al., 2007; Wetter and Young, 1994). We hypothesized that both insomnia and emotion dysregulation would independently predict PTSS. In addition, we predicted a significant interaction between insomnia and emotion dysregulation in predicting PTSS, such that those with greater levels of insomnia and emotion dysregulation would have the most severe PTSS. Finally, we investigated which components of emotion dysregulation would predict PTSS and interact with insomnia to predict PTSS. Consistent with previous research, we expected limited access to emotion regulation strategies, lack of emotional clarity, and impulse control problems would be associated with PTSS, and that these components would interact with insomnia to predict to PTSS (Tull et al., 2007).

Method

Participants

The sample consisted of 349 adult daily smokers selected from a larger smoking cessation treatment study. To be eligible for inclusion, participants had to be 18 years of age or older,

a daily smoker for at least 1 year, currently smoke a minimum of 8 cigarettes per day, and report a motivation to quit smoking within the next month. Individuals were excluded if they were diagnosed with a psychotic disorder, using any smoking cessation pharmacotherapy or other tobacco products, or if they had a history of a severe medical condition that would preclude their participation in the smoking cessation trial.

Participants were selected for the current study if they endorsed experiencing a traumatic event on the Posttraumatic Stress Diagnostic Scale (PDS; Foa et al., 1997). Participants selected the following as their most disturbing traumatic events: an accident (23.3%), sexual assault by someone they knew (9.7%), non-sexual assault by someone they knew (9.4%), non-sexual assault by a stranger (9.4%), life-threatening illness (8.5%), a major disaster (7.4%), sexual assault by a stranger (5.1%), imprisonment (4.8%), sexual contact under age 18 with someone at least 5 years older (4.5%), combat (1.4%), torture (1.4%), and other (15.1%; e.g., victim of a serious crime, first responder for emergencies, witness of another person killed, dead, or badly hurt).

Participants' ages ranged from 18 to 68 ($M = 38.59$, $SD = 13.46$). The sample was evenly distributed in terms of gender (51.4% female). Most of the sample self-identified as Caucasian (81.8%), followed by Black Non-Hispanic (9.7%), Hispanic (4.0%), other (2.5%; e.g., mixed race), Asian (1.4%) and Black Hispanic (0.6%). Approximately one-third of the sample was married or living with someone (38.1%), while 37.5% were never married, 19.3% divorced or annulled, 3.1% separated, and 2.0% widowed. Regarding education history, 36.7% completed some college, 18.4% received a high school diploma, 14.2% graduated from a four year college, 10.8% graduated from a two year college, 9.1% completed graduate or professional school, 6.0% never graduated from high school, and 4.8% completed some graduate or professional school. Approximately half of the sample (49.1%) presented with at least one Axis I diagnosis according to the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996), with 7.7% of the sample diagnosed with PTSD (primary or other current). Other primary diagnoses were as follows: 24.5% anxiety disorders, 8.6% mood disorders, 6.5% substance-related disorders, 3.8% other (e.g., anorexia nervosa), and 1.4% obsessive-compulsive and related disorders.

Procedure

If initial eligibility requirements were met during a brief telephone screen, participants were scheduled for a structured clinical interview to assess for the presence of Axis I psychopathology (SCID; First et al., 1996). Individuals who were deemed eligible after the interview process were then scheduled to complete a baseline appointment. During this baseline appointment, participants completed self-report questionnaires and were randomly assigned to either a standard smoking cessation program or an anxiety management smoking cessation program. Following the treatment phase, participants were followed up two years post treatment. The current investigation utilizes data collected at the baseline appointment only, which took place prior to randomization and treatment onset. The study was approved by the university's IRB, and informed consent was obtained from all participants.

Measures

Structured Clinical Interview for DSM-IV (SCID; First et al., 1996)—All participants were interviewed using the SCID-NP (non-patient version). Trained doctoral level therapists administered the SCID after receiving intensive training in SCID administration and scoring. Training consisted of reviewing SCID training tapes, observing live SCID administrations, and conducting practice interviews with trained therapists. Trainees received feedback until they demonstrated high levels of reliability. All SCIDs were reviewed by a licensed clinical psychologist to ensure accurate diagnoses. Finally, interviews were audiotaped and the reliability of a random selection of 12.5% of interviews were reviewed (MJZ) for accuracy; no cases of diagnostic disagreement were noted.

Posttraumatic Stress Diagnostic Scale (PDS; Foa et al., 1997)—The PDS includes both a 12-item checklist of traumatic event exposure, as well as a 17-item self-report questionnaire corresponding with the DSM-IV-TR symptoms of PTSD. Respondents were first asked to indicate whether they experienced any of 12 traumatic events and then indicate which event was the most disturbing. The PDS provides a count of traumatic events endorsed that was used as a covariate in all analyses in the current study. In addition, the PDS assesses the severity of the 17 PTSS experienced in the past month. Respondents are asked to rate each item on a 4-point Likert scale ranging from 0 (*Not at all/Only one time*) to 3 (*Five or more times a week/Almost always*). The PDS yields a total severity score, which was used to index PTSS in the present study. Prior to calculating the total severity score, sleep items were removed to account for conceptual overlap with our measure of insomnia. The PDS is psychometrically sound (Foa et al., 1997), and demonstrated excellent internal consistency ($\alpha = .93$) in the present sample.

Inventory of Depression and Anxiety Symptoms (IDAS; Watson et al., 2007)—The IDAS is a 64-item self-report questionnaire in which respondents rate the degree to which each statement describes their feelings and experiences during the past two weeks using a 5-point Likert scale ranging from 1 (*Not at all*) to 5 (*Extremely*). In the present investigation, the insomnia subscale was used to index severity of insomnia symptoms (e.g., “I had trouble falling asleep”). Research has shown the IDAS subscales have good internal consistency, short-term stability, and convergent and discriminant validity (Watson et al., 2007). In the current study, the insomnia subscale demonstrated good internal consistency ($\alpha = .88$).

Difficulties in Emotion Regulation Scale (DERS; Gratz and Roemer, 2004)—The DERS was used in the present investigation to assess emotion dysregulation. The DERS is a 36-item scale comprised of six subscales and a total score. Respondents were asked to rate each item on a 5-point Likert-type scale ranging from 1 (*Almost never*) to 5 (*Almost always*). In the current study, the DERS total score was employed as a global index of emotion dysregulation. In addition, we also used the subscales of emotion regulation difficulties including: nonacceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity. Consistent with previous research (Gratz and Roemer, 2004), the DERS total score demonstrated excellent internal

consistency in the present sample ($\alpha = .95$). In addition, the clarity ($\alpha = .83$), awareness ($\alpha = .83$), impulse problems ($\alpha = .88$), nonacceptance ($\alpha = .91$), goals ($\alpha = .88$), and strategies ($\alpha = .92$) subscales demonstrated at least good internal consistency.

Positive and Negative Affect Schedule (PANAS; Watson et al., 1988)—The PANAS is a 20-item questionnaire designed to assess two global dimensions of affectivity: positive and negative. Individuals are asked to read each emotion (e.g., sad) and rate the extent to which they generally feel that way using a 5-point Likert scale ranging from 1 (*Very slightly or not at all*) to 5 (*Extremely*). Previous research has found the PANAS to be psychometrically sound (Watson et al., 1988). In the present investigation, only the PANAS-NA subscale was utilized. Internal consistency in the current sample was excellent ($\alpha = .95$).

Data Analytic Plan

A hierarchical regression was computed to test the hypotheses that 1) insomnia and emotion dysregulation would each independently predict increased PTSS, and 2) there would be a significant insomnia by emotion dysregulation interaction. In step one of the model, negative affectivity (as measured by the PANAS – NA) and number of traumatic events experienced (as measured by the PDS) were entered as covariates. In step two, the centered predictor variables of insomnia (as measured by the IDAS – insomnia) and emotion dysregulation (as measured by the DERS) were entered. Finally, in step three, the centered interaction term of IDAS – insomnia by DERS was entered. Total PTSS served as the dependent variable. Next, we constructed six similar hierarchical regressions to test which DERS subscales predicted PTSS, and interacted with insomnia to predict PTSS. Again, negative affectivity and number of traumas experienced were entered in the first step of the model, followed by the centered predictor variables (IDAS – insomnia and the relevant DERS subscale) in the second step, and the centered interaction term (IDAS - insomnia by DERS subscale) in the third step. PTSS served as the dependent variable in each regression. Significant interactions were probed following recommendations of Aiken and West (1991).

Results

Preliminary Analyses

First, we examined the means, standard deviations, and zero-order correlations of all variables (see Table 1). In terms of negative affectivity and emotion dysregulation, the means of our sample were similar to but slightly lower than levels found in previous research among a trauma-exposed sample (Tull et al., 2007). Regarding trauma exposure and PTSS, participants endorsed a mean of approximately three traumatic events experienced. PDS scores were slightly higher than those found in previous research among a trauma-exposed sample, despite our mean being calculated with the two sleep items removed (Moser et al., 2007). In addition, according to PDS ranges and cut-offs, 37.8% of our sample met criteria for mild PTSS, 20.2% for moderate, 9.7% for moderate to severe, and 1.4% for severe, with 6.0% meeting the cut-off for a probable PTSD diagnosis (Foa et al., 1997). Finally, IDAS – insomnia scores were lower than those found in previous research conducted in a sample of individuals with PTSD, which is expected considering our

sample is composed of trauma-exposed participants with and without PTSD (Watson et al., 2008).

Primary Analyses

Step one accounted for 34.7% of the variance in PTSS severity ($F(2, 345) = 91.55, p < .001$), with both negative affect ($\beta = .50, t = 11.29, p < .001, sr^2 = .24$) and number of traumas ($\beta = .23, t = 5.12, p < .001, sr^2 = .05$) significantly predicting elevated PTSS. Step two accounted for an additional 5.5% of the variance in PTSS ($F \text{ Change} = 15.68, p < .001$). Both elevated insomnia ($\beta = .20, t = 3.87, p < .001, sr^2 = .03$) and emotion dysregulation ($\beta = .23, t = 3.62, p < .001, sr^2 = .02$) significantly predicted more severe PTSS. Finally, the interaction of insomnia and emotion dysregulation accounted for an additional 1.6% of the variance in PTSS and significantly predicted elevated PTSS ($\beta = .14, t = 3.06, p = .002, sr^2 = .02$). To interpret this finding, the interaction was probed following recommendations by Aiken and West (1991; see Figure 1). Specifically, we examined the simple effect of insomnia for those with high and low (± 1 SD from the mean) DERS scores. Insomnia was significantly associated with PTSS for those with high DERS scores ($\beta = .30, t = 4.95, p < .001, sr^2 = .04$). However, for those with low DERS scores, insomnia was not associated with PTSS ($\beta = .20, t = 3.06, p = .022, sr^2 = .00$).

DERS Subscales

Strategies—Strategies significantly predicted PTSS ($\beta = .29, t = 4.47, p < .001, sr^2 = .03$), but the interaction term between insomnia and strategies did not reach statistical significance ($\beta = .05, t = 1.22, p = .222, sr^2 = .00$).

Clarity—Clarity significantly predicted PTSS ($\beta = .17, t = 3.31, p = .001, sr^2 = .02$), as well as interacted with insomnia to predict PTSS ($\beta = .12, t = 2.65, p = .008, sr^2 = .01$). The interaction followed a similar pattern to that found with total emotion dysregulation (see Figure 1), such that insomnia was associated with PTSS for those with high ($\beta = .29, t = 4.75, p < .001, sr^2 = .04$) but not low ($\beta = .09, t = 1.40, p = .162, sr^2 = .00$) scores on clarity.

Impulse problems—Impulse problems significantly predicted PTSS ($\beta = .13, t = 2.34, p = .020, sr^2 = .01$), as well as interacted with insomnia to predict PTSS ($\beta = .18, t = 3.77, p < .001, sr^2 = .02$). In this case, similar to total emotion dysregulation (see Figure 1), insomnia was related to PTSS for those with high scores on impulse problems ($\beta = .33, t = 5.53, p < .001, sr^2 = .05$), but not for those with low scores on impulse problems ($\beta = .06, t = .93, p = .354, sr^2 = .00$).

Awareness—Awareness did not significantly predict PTSS ($\beta = .02, t = .48, p = .633, sr^2 = .00$). However, it did interact with insomnia to predict PTSS ($\beta = .10, t = 2.22, p = .027, sr^2 = .01$). Again, similar to the pattern of findings with total emotion dysregulation (see Figure 1), insomnia was associated with PTSS for those with high ($\beta = .29, t = 4.65, p < .001, sr^2 = .04$) rather than low ($\beta = .12, t = 1.77, p = .078, sr^2 = .01$) scores on awareness.

Nonacceptance—Nonacceptance neither significantly predicted PTSS ($\beta = .06$, $t = 1.07$, $p = .283$, $sr^2 = .00$), nor interacted with insomnia to predict PTSS ($\beta = .06$, $t = 1.07$, $p = .284$, $sr^2 = .00$).

Goals—Goals predicted PTSS ($\beta = .14$, $t = 2.63$, $p = .009$, $sr^2 = .01$), but did not significantly interact with insomnia to predict PTSS ($\beta = .06$, $t = 1.13$, $p = .181$, $sr^2 = .00$).

Discussion

The current study aimed to examine, among a sample of trauma-exposed smokers, the independent and interactive associations of insomnia and emotion dysregulation with PTSS severity, as well as which specific components of emotion dysregulation played a role in these relationships. Findings indicated insomnia and emotion dysregulation may be important factors contributing to elevated PTSS among smokers. Specifically, regarding the first aim, the present study replicated findings that insomnia and emotion dysregulation are predictive of PTSS severity above and beyond the effects of each other (Fairholme et al., 2013), negative affectivity, and number of traumas experienced. This finding is consistent with empirical and theoretical work linking sleep disturbances such as insomnia to increased PTSS (Germain, 2013; Koren et al., 2002; Wright et al., 2011). Sleep loss associated with insomnia may cause impaired cognitive and emotional functioning for the individual, which may lead to increased reactivity to negative emotional triggers (Dinges et al., 1997), such as trauma reminders, and decreased ability to cope with posttraumatic stress (Germain, 2013; Walker, 2009). Additionally, this finding is consistent with recent research demonstrating emotion dysregulation may contribute to PTSS (Bardeen et al., 2013; Tull et al., 2007). Emotion dysregulation may cause difficulties coping with negative emotions related to posttraumatic stress, as well as increased avoidance of internal and external cues related to the traumatic event (Bardeen et al., 2013; Tull et al., 2007), leading to more severe PTSS.

In terms of our second aim, the current study also expands upon literature on insomnia, emotion dysregulation and PTSS by finding that insomnia and emotion dysregulation interact to predict PTSS, such that individuals with elevated levels of insomnia and emotion dysregulation had the most severe PTSS. This is consistent with our hypothesis that the effect of insomnia and associated impairment in cognitive and emotional functioning on PTSS would be greater in those with elevated levels of emotion dysregulation compared to those with lower levels of emotion dysregulation. It is likely sleep loss due to insomnia would be associated with increased negative emotions overall (Zohar et al., 2005), and increased negative emotional reactivity to negative emotional cues (Yoo et al., 2007), such as reminders of the trauma. These problems may be exacerbated by emotion dysregulation, leading to increased anxiety regarding the traumatic event and its reminders due to the intensity and perceived uncontrollability of associated negative emotions (Bardeen et al., 2013; Bouton et al., 2001). This may also lead to avoidance of reminders of the event, preventing emotional processing of the trauma and contributing to increased PTSS (Bardeen et al., 2013; Foa and Kozak, 1986).

Finally, we examined which emotion dysregulation components were predictive of PTSS and interacted with insomnia to predict PTSS. Consistent with hypothesis and previous

research, limited access to effective emotion regulation strategies, problems with emotional clarity, and impulse control problems were most associated with PTSS (Tull et al., 2007). However, we also found that problems pursuing goals while distressed was similarly associated with PTSS, which is consistent with recent findings from a study of comorbid PTSD and AUD patients (Fairholme et al., 2013). Considering these mixed findings, future research may be needed to clarify precisely which components of emotion dysregulation are most related to PTSS. In addition, partially consistent with hypothesis, emotional clarity, awareness, and impulse control problems interacted with insomnia to predict PTSS. These findings indicate that, specifically, a lack of emotional clarity and awareness and an inability to control impulse problems when distressed were associated with a greater effect of insomnia on PTSS. For example, increased negative emotions and emotional reactivity associated with sleep loss may be exacerbated by a lack of understanding of one's emotions, causing further anxiety and distress when confronted with negative emotions related to posttraumatic stress. Additionally, an inability to control impulses when distressed in the context of sleep loss may lead to increased avoidance of emotional situations, such as trauma cues.

In terms of clinical implications, the current study, taken together with previous research, highlights the potential importance of targeting insomnia and emotion dysregulation in reducing PTSS. Even after receiving empirically supported PTSD treatment, a large proportion of individuals continue to meet criteria for PTSD or fail to reach good end-state functioning (e.g., 39%; Tarrier et al., 1999). Thus, identifying novel treatment targets for PTSD is an important goal for increasing treatment efficacy. Specifically, it may be helpful for clinicians to assess and consider targeting symptoms of insomnia and emotion dysregulation in individuals with PTSD. Indeed, previous research has found CBT-I is effective for individuals with PTSD in treating insomnia (Smith et al., 2005; Talbot et al., 2014). CBT-I may also reduce PTSS, although further research is needed considering mixed evidence and relative lack of randomized controlled trials (Baddeley and Gros, 2013; Margolies et al., 2013; Talbot et al., 2014). Research on emotion dysregulation interventions for PTSD is more limited. However, one study tested an emotion regulation intervention added to prolonged exposure (PE; Foa et al., 2007), and found it effective in improving emotion regulation and PTSD symptoms compared to a waitlist control (Cloitre et al., 2002). Although this research is promising, comparing such an intervention to an established treatment (e.g., PE) is necessary to determine whether targeting emotion regulation provides an added benefit for treatment outcomes.

The findings of the present study should be considered in the context of its limitations. First, our sample consisted of trauma-exposed smokers with and without PTSD. Future research should attempt to replicate these findings in a clinical sample of individuals with PTSD. We were also unable to determine whether individuals in our sample had a diagnosis of insomnia due the measure of insomnia used (IDAS - insomnia; Watson et al., 2007). Future research may benefit from ensuring the inclusion of individuals with clinical levels of insomnia. In addition, due to the cross-sectional design of the study, we are not able to make causal attributions regarding the relationships between these constructs. However, previous prospective research has indicated that both insomnia and emotion dysregulation predict elevated PTSS (Bardeen et al., 2013; Koren et al., 2002; Wright et al., 2011). Future

research should examine whether the interaction between insomnia and emotion dysregulation prospectively predicts elevated PTSS. Finally, we utilized self-report measures for each of our constructs of interest. Although we used well-validated measures, future research should consider multi-method designs, including objective measures of sleep (e.g., polysomnography).

Despite these limitations, the current study extends the literature on the relationship between insomnia and PTSS by demonstrating both insomnia and emotion dysregulation independently predict PTSS, and that those with elevated levels of emotion dysregulation may be more susceptible to the effects of insomnia on PTSS. In addition, these findings add to the emerging literature on the role of emotion dysregulation in elevated PTSS (e.g., Bardeen et al., 2013; Tull et al., 2007). These results also point to potential factors that may contribute to elevated PTSS among smokers, an important consideration as PTSS severity is linked to current smoking levels (Feldner et al., 2007). In sum, these results provide support for the independent and interactive roles of insomnia and emotion dysregulation in elevated PTSS among trauma-exposed smokers, and highlight the potential importance of targeting these factors in PTSD treatment.

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Figure 1. Interactive effects of insomnia and emotion dysregulation predicting posttraumatic stress symptoms

Note: DERS = Difficulties in Emotion Regulation Scale.

Table 1

Means, standard deviations, and zero-order correlations of all variables.

	1	2	3	4	5	6	7	8	9	10	11
1 Negative Affectivity	–	–	–	–	–	–	–	–	–	–	–
2 Number of Traumas	.20**	–	–	–	–	–	–	–	–	–	–
3 Insomnia	.55**	.23**	–	–	–	–	–	–	–	–	–
4 Emotion Dysregulation	.76**	.16*	.47**	–	–	–	–	–	–	–	–
5 Clarity	.55**	.04	.36**	.78**	–	–	–	–	–	–	–
6 Awareness	.21**	.04	.19**	.54**	.56**	–	–	–	–	–	–
7 Impulse Problems	.63**	.11*	.38**	.81**	.54**	.24**	–	–	–	–	–
8 Nonacceptance	.63**	.63**	.39**	.78**	.47**	.26**	.53**	–	–	–	–
9 Goals	.61**	.61**	.37**	.79**	.51**	.22**	.66**	.54**	–	–	–
10 Strategies	.78**	.78**	.46**	.88**	.60**	.25**	.72**	.66**	.70**	–	–
11 PTSS	.55**	.55**	.47**	.52**	.41**	.15*	.43**	.40**	.43**	.55**	–
Mean(SD)	19.29 (7.67)	3.05 (1.81)	13.27 (5.87)	75.60 (22.91)	10.26 (3.85)	14.74 (5.01)	10.47 (4.44)	12.18 (5.50)	13.08 (4.64)	14.86 (6.49)	7.24 (9.01)
Range	38	9	24	125	19	20	23	24	20	30	43

Note: $n=349$.*
= $p<.01$.**
= $p<.001$.