Published in final edited form as:

J Affect Disord. 2019 June 15; 253: 285–291. doi:10.1016/j.jad.2019.04.035.

Examining the Relationship between Negative Affect and Posttraumatic Stress Disorder Symptoms Among Smokers Using Ecological Momentary Assessment

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Abstract

Background—Posttraumatic stress disorder (PTSD) and negative affect (e.g., anger, depression, anxiety), are highly co-occurring. It remains unclear whether changes in PTSD symptoms subsequently impact negative affect, or vice versa. This study assessed associations between moment to moment PTSD symptoms and negative affect in a sample of smokers with PTSD to determine directionality of this relationship.

Methods—Participants (N = 125) enrolled in two smoking cessation studies with co-occurring PTSD and cigarette use completed measures of PTSD and negative affect. Ecological momentary assessment (EMA) methodology was used to record symptoms during a one-week baseline period, during which participants smoked *ad lib*. Cross-lagged path analyses assessed PTSD symptoms and negative affect for directionality of their relationship, controlling for whether an EMA reading was smoking or non-smoking. Path analyses examined the lagged associations between PTSD symptoms and negative affect.

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Contributors

Jean Beckham and Patrick Calhoun were Principal Investigators for the research projects. Meredith Erwin and Paul Dennis designed the study for this manuscript. Meredith Erwin took lead in writing the manuscript and managed revisions. Paul Dennis undertook the statistical analysis, wrote the methods and results, and created the tables and figures. Jean Beckham, Patrick Calhoun, and Lara Coughlin all read and provided feedback concerning all sections of the paper. All authors contributed to and have approved the final manuscript.

Conflict of Interest

None declared.

Results—Results found PTSD symptom severity at T-1 was significantly related to negative affect levels at time T, but negative affect at time T-1 was not associated with PTSD symptom severity at time T. Results indicated the model retaining the cross-lagged effect of PTSD symptom severity on negative affect provided better fit to the data than other models.

Limitations—Limitations included use of self-report data, brief measures of symptoms, participants already had PTSD and/or MDD, participants were recruited from a specific clinical population, and use of DSM-IV data.

Conclusions—Results suggest PTSD symptoms drive day-to-day fluctuations in negative affect, and highlight the importance of evaluating negative affect in the treatment of PTSD.

Keywords

PTSD; negative affect; ecological momentary assessment; smoking

Introduction

Posttraumatic stress disorder (PTSD) has a lifetime prevalence rate of approximately 7% in the general population (Kessler et al., 1995), and is comprised of intrusion symptoms, avoidance symptoms, negative alterations in cognition and mood symptoms, and anxious arousal and reactivity symptoms (American Psychiatric Association, 2013). Negative affect, defined as a variety of negative emotional states (e.g., sadness, anger, anxiety; Beckham et al., 2002; Byllesby et al., 2016; Kotov et al., 2010), frequently co-occurs with PTSD (e.g., Contractor et al., 2014; Durham et al., 2015; Kotov et al., 2010). The relationship between PTSD and negative affect can be described as that of two overlapping but distinct constructs, primarily because their relationship remains after controlling for symptom overlap (Elhai et al., 2008; Grubaugh et al., 2010; Orth et al., 2008). Therefore, there are most likely other reasons that explain the co-occurring relationship between PTSD and negative affect.

The co-occurring relationship between negative affect and PTSD is posited to be one that develops temporally, with one construct preceding the other (Breslau, 2002; Ginzburg et al., 2010). It is possible negative emotionality arises from PTSD symptoms (Breslau, 2002), with past research showing negative emotions as secondary to PTSD symptoms, particularly in the context of anger, anxiety, and depression (e.g., Ginzburg et al., 2010; Kessler et al., 1995; O'Toole et al., 1998; Orth et al., 2008; Van Voorhees et al., 2018). In contrast, negative emotionality has been posited to create vulnerability for development of PTSD (Breslau, 2002), with some research finding PTSD to be secondary to depression (O'Toole et al., 1998); however, there appears to be less research in support of the second explanation. These mixed findings are cause for further investigation into clarifying the direction of the relationship between PTSD and negative affect.

Research has found individuals with greater PTSD symptomatology report greater negative affect (Cohn et al., 2014), and respond to stressful events with higher negative affect (Beckham et al., 2002). Individuals with PTSD can have particular trouble managing negative emotions (Beckham et al., 2003), and those with higher negative affect show increased risk-taking behaviors, such as alcohol use (Cohn et al., 2014). Additionally, co-

occurring PTSD and negative emotionality are correlated with poorer functioning, efficacy of treatment, and outcomes (Angelakis and Nixon, 2015; Bryant et al., 2003; Cahill et al., 2003; Olatunji et al., 2010). Due to the negative effects that can accompany co-occurring PTSD and negative affect, it is important to understand how these constructs can exacerbate each other over time.

There are few studies that have investigated temporal relations between *only* PTSD and negative affective states (e.g., anxiety, depression, anger) (Ginzburg et al., 2010; Orth et al., 2008; Van Voorhees et al., 2018). The few studies examining PTSD and negative affect's temporal relationships have found PTSD predicts anxiety and depressive disorders, but anxiety and depressive disorders do not predict PTSD (Ginzburg et al., 2010). Additionally, PTSD symptoms have been found to predict anger, both longitudinally (Orth et al., 2008) and moment-to-moment (Van Voorhees et al., 2018), but not vice versa. Besides the study by Van Voorhees et al. (2018), the majority of these prior studies examine this relationship over a larger time scale (e.g., weeks up to years; Van Voorhees et al., 2018). Few studies have investigated the moment-to-moment symptom and emotional experiences in those with co-occurring PTSD and negative affect. Therefore, examining this specific temporal relationship is warranted.

One such method of investigating temporal relationships between PTSD and negative affect is ecological momentary assessment (EMA). EMA involves the momentary sampling of mental health symptoms at various time points throughout the day and allows for a more refined view of daily symptoms beyond global recall (Van Voorhees et al., 2018). The majority of studies investigating only negative affect and PTSD symptoms have focused on clinical trajectories of both these constructs over several months after the occurrence of a trauma. By contrast, utilizing an EMA format enables the examination of momentary fluctuations in negative affect and PTSD symptomatology. While there are several studies that have investigated PTSD and negative affect along with other constructs in an EMA format (Cohn et al., 2014; Short et al., 2017), few have investigated the relationship between only PTSD and negative affect to determine the direction of this relationship (e.g., Van Voorhees et al., 2018). Specifically, Van Voorhees et al. (2018) showed that moment-to-moment PTSD symptoms predicted subsequent anger, but anger did not predict PTSD symptoms. Therefore, with this type of analysis we can elucidate the function of negative affect as a driver or product of PTSD symptoms at the proximal level.

Additional characteristics of the sample utilized in this study, a sample of cigarette smokers with PTSD, must be considered when examining the relationship between PTSD and negative affect. Cigarette smoking has been found to be highly prevalent in a sample of veterans with PTSD (60%; Beckham, 1999). Past research has found that smokers with PTSD reported higher levels of anxiety and depression (Beckham et al., 1995). Higher cravings, negative affect, and PTSD symptoms are associated with trauma-related imagery in groups with and without PTSD, and smoking is associated with decreased cravings, PTSD symptoms, and negative affect in the presence of trauma-related imagery (Beckham et al., 2007). Research has also shown that motivation to smoke can underlie relationships between negative affect and PTSD, in that individuals might be motivated to smoke to regulate negative emotionality experienced due to PTSD (Mahaffey et al., 2016) or acute stressors

(Cook et al., 2017). As PTSD and negative affect are reportedly high in cigarette smokers, examining the moment-to-moment relationship between these variables merits examination in this sample.

Aims and Hypotheses

Building on prior work exploring the temporal relationship between PTSD and negative emotions (Ginzburg et al., 2010; Orth et al., 2008; Van Voorhees et al., 2018), we employed novel EMA methods to explore if PTSD symptoms predict negative affect, or vice versa, if negative affect predicts exacerbations in PTSD symptoms in the context of trauma-exposed cigarette smokers. Prior research largely supports PTSD predicting negative affect across different time frames (Ginzburg et al., 2010; Kessler et al., 1995; Orth et al., 2008), and initial evidence suggests moment-to-moment PTSD symptoms predict anger (Van Voorhees et al., 2018). Given this prior research, we hypothesized that PTSD would predict moment to moment negative affect in a trauma-exposed, cigarette smoking sample, while controlling for whether a reading is smoking versus non-smoking.

Method

Participants

The present analyses are secondary to data collected from 125 smokers with PTSD enrolled in one of two smoking-cessation trials, prior to beginning active treatment. Forty-nine of these participants ($n = 25$ females) had enrolled in a study to determine the effects of nicotine patch preloading (i.e., wear nicotine patches prior to and/or during a quit attempt in order to diminish the effects of nicotine, and therefore its associated reward, derived from smoking) before and after a quit attempt among smokers with PTSD (Dennis et al., 2016). The other 76 participants ($n = 34$ females) had enrolled in a study examining the combined effects of nicotine patch preloading and low-nicotine cigarettes before and after a quit attempt among smokers with PTSD (Dedert et al., 2018).

Across both studies, eligible participants were between 18 and 70 years of age, generally healthy, spoke English, met DSM-IV criteria for current PTSD, and were currently smoking at least 10 cigarettes per day for at least a year. Participants were excluded for organic mental disorder, pregnancy, using other forms of nicotine, current substance abuse/dependence other than nicotine dependence, schizophrenia, lifetime but not current PTSD, current psychotic symptoms, unstable medical conditions, not being cleared by a study physician, or if they were going to be unstable on medications during the study period. The first study also included an exclusion criterion of meeting DSM-IV criteria for a diagnosis of bipolar disorder. The second study excluded participants for experiencing a current manic episode, or if they had a recent history of myocardial infarction. Both studies were approved by the Duke University School of Medicine Institutional Review Board (IRB) and the Durham VA IRB and Research and Development Committees. See Table 1 for participant demographic and clinical characteristics.

Procedures

Complete information on the procedures of each of the two studies is available elsewhere (Dedert et al., 2018; Dennis et al., 2016). In both studies, participants completed a screening and baseline session prior to the initiation of EMA data collection. During the baseline session, participants provided basic sociodemographic information, completed the Fagerström Test of Nicotine Dependence (Heatherton et al., 1991), the Structured Clinical Interview for the DSM-IV Axis I disorders (SCID; First et al., 1996), and the Clinician Administered PTSD Scale for the DSM-IV (CAPS; Blake et al., 1995).

Both studies used EMA to document changes in nicotine craving, PTSD symptomatology, and negative affect from baseline to pre-quit to post-quit. The present data are drawn from the approximately week-long baseline observation period during which participants smoked *ad lib*, prior to the pre-quit time period. During the *ad lib* period, participants responded to random alarms throughout the day and initiated their own assessments before and after smoking. The present analyses were conducted using EMA data from random alarms and self-initiated entries made prior to smoking. Random alarms in which participants indicated that they had just smoked and self-initiated entries in which participants were preparing to smoke were labeled smoking entries. The remaining random alarms were labeled non-smoking entries.

Measures

At baseline, psychiatric disorders were assessed using the Structured Clinical Interview for the DSM-IV Axis I disorders (SCID; First et al., 1996) and the Clinician Administered PTSD Scale for the DSM-IV (CAPS; Blake et al., 1995). Current diagnosis was determined by a 1-month timeframe for PTSD and major depressive disorder and a 3-month time frame for current substance abuse or dependence. Each rater was trained using SCID and CAPS standardized training (i.e., manual, videotapes, and co-ratings with a trained rater). Interrater reliability for diagnoses based on videotapes of patient interviews was excellent (Fleiss' kappa = .96) (American Psychiatric Association, 1994). Nicotine dependence was assessed with the Fagerström Test of Nicotine Dependence (Heatherton et al., 1991), a six-item self-report measure for which scores ranging from 0 to 4 indicate low dependence and scores 6 to 10 suggest high dependence.

PTSD symptoms were assessed with four questions corresponding to King et al. (1998) PTSD factors: "Right now, how much are you bothered by... disturbing thoughts, images, or feelings related to your traumatic event" (B cluster); "avoiding thoughts, activities, or feelings related to your traumatic event" (C1 cluster); "feeling distant or cut off from other people and/or feeling emotionally numb" (C2 cluster); "difficulty concentrating, feeling jumpy or easily startled, feeling overtly alert, or feeling irritable or angry?" (D cluster). Responses to each question ranged from 0 ("not at all") to 4 ("extremely"). A total PTSD symptom score was calculated by summing the four cluster scores ($\alpha = .87$).

Negative affect was assessed by four items from the *Minnesota Nicotine Withdrawal Scale* (MNWS; Hughes & Hatsukami, 1986). Specifically, participants indicated the extent to which they felt a) angry, irritable, and frustrated, b) anxious and nervous, c) depressed mood

and sad, and d) difficulty concentrating on a scale of 0 (“none”) to 4 (“severe”) (Cappelleri et al., 2005). Negative affect was calculated as the sum of these four scores ($\alpha = .91$). Diary entries were time-stamped to ensure temporal accuracy (i.e., participants could not delay or clump entries) and to assess protocol adherence.

Data Analysis

To explore the direction of the association between PTSD symptom severity and negative affect over time, we tested multilevel cross-lagged path models. These examined whether EMA measurements of PTSD symptom severity predicted negative affect at the following reading while controlling for the effects of previous negative affect and also whether negative affect predicted subsequent PTSD symptom severity after controlling for the effects of previous PTSD symptom severity. Multilevel modeling was used because the data were characterized by two levels of observations: those made at the level of each diary reading (Level 1) and those made at the level of each participant (Level 2). The advantage of path analysis over univariate approaches, such as multiple regression, is that multiple dependent variables can be examined simultaneously. Moreover, using fit indices, one can examine all viable paths in a path model and then determine whether elimination of one or more of those paths significantly reduces model fit. For instance, in the present study, we could examine not only the autoregressive effects of negative affect and PTSD symptom severity, but also whether previous negative affect predicted subsequent PTSD symptom severity and whether previous PTSD symptom severity predicted subsequent negative affect.

To disentangle between-person associations between negative affect and PTSD symptoms from the within-person associations, grand-mean standardized negative affect and PTSD symptom scores were generated by calculating each individual’s mean levels of negative affect and PTSD symptom severity across the observation period and *z*-scoring these in relation to those of the other participants in the sample. Individual-mean standardized scores were then calculated by using each individual’s mean negative affect and PTSD symptom levels and corresponding standard deviations to *z*-score the levels recorded at each reading. Because the effect of lagged variables likely diminished over time, we omitted lagged values that occurred more than 4 hours prior to the subsequent reading, which were approximately 13.6% of readings. The remaining readings had a mean interval of approximately 1.26 hours between them ($SD = 0.85$). Additionally, the minimum time interval between readings was 1.55 minutes.

Prior to conducting the cross-lagged analyses, we first examined the distribution of PTSD symptom severity and negative affect scores. Both were characterized by positively skewed distributions with a large number of zeros (28.4% for PTSD symptom severity, 33.7% for hostility/irritability). However, subsequent examination of quantile-quantile plots indicated that the normality assumption was not violated, although concern over that is likely a moot point in the context of multilevel models (Gelman and Hill, 2007). Nevertheless, the models were fitted in Mplus 7 using robust maximum likelihood estimation, which is capable of modeling non-normal data.

In the initial path analysis, we modeled PTSD symptom severity and negative affect scores as a function of their lagged values, covarying for whether or not a given reading was a

smoking reading. We then examined three additional models in which the cross-lagged paths were removed, first individually and then simultaneously. The Satorra-Bentler chi-square test was used to compare nested models. Bayesian information criteria (BIC; Schwarz, 1978) was consulted to aid in comparison of non-nested models. In using BIC values to compare models, a 10-point difference can be interpreted as “very strong” evidence (i.e., $p < .05$) of the superiority of the model with the smaller BIC (Kass and Raftery, 1995; Raftery, 1995). A difference of 6 to 9 points may be interpreted as “strong” support for a meaningful difference between the models.

Results

As indicated in Table 1, participants were largely middle-aged and Black with low nicotine dependence and a high incidence rate of current major depressive disorder (MDD). Across the sample, participants initiated a total of 11,414 EMA entries during the observation period but completed only 3737 (32.7%) due to mistakenly initiating entries, technical difficulties, and failure to finish entries. Of these, 1326 (35.5%) had complete data on current and lagged negative affect and PTSD symptomatology. Approximately 98.0% of these entries were smoking entries.

Path analyses examined the lagged associations between PTSD symptoms and negative affect while controlling for whether an EMA reading was smoking or non-smoking. Initial results examining the Level 1 effects from the cross-lagged model revealed strong lagged effects of negative affect at time T-1 on negative affect at time T (Cohen's $d = 0.44$, $p < .001$) and PTSD symptom severity at time T-1 on PTSD symptom severity at time T (Cohen's $d = 0.54$, $p < .001$) (see Figure 1). This model also indicated that the individual-mean standardized effect of negative affect at time T-1 was not associated with PTSD symptom severity at time T (Cohen's $d = 0.05$, $p = .56$); however, individual-mean standardized PTSD symptom severity at T-1 was significantly related to negative affect levels at time T (Cohen's $d = 0.35$, $p < .001$). Whether or not a given reading was a smoking – versus a non-smoking – reading had no bearing on PTSD symptom severity or negative affect. Level-2 results indicated that individual differences in negative affect and PTSD symptomatology were associated with momentary levels of negative affect and PTSD symptom severity, respectively. However, these effects were not crossed.

Dropping the path from negative affect at time T-1 to PTSD symptom severity at time T did not significantly diminish model fit in comparison to the fully cross-lagged model, Satorra-Bentler $\chi^2(1) = 0.37$, $p = .54$. However, dropping the path from PTSD symptom severity at time T-1 to negative affect at time T did significantly reduce model fit, Satorra-Bentler $\chi^2(1) = 19.74$, $p < .001$. Moreover, the BICs indicated that the model retaining the cross-lagged effect of PTSD symptom severity on negative affect provided a better fit to the data than the model retaining the cross-lagged effect of negative affect on PTSD symptom severity (see Table 2). Dropping both cross-lagged effects resulted in the poorest fitting model.¹

¹There was substantial conceptual overlap in the items used in this set of analyses to measure negative affect, cluster C2 PTSD symptoms, and cluster D PTSD symptoms. Thus, to mitigate the effects of this overlap, we performed the cross-lagged analyses using PTSD symptom severity based solely on B and C1 PTSD cluster scores. The results were nearly identical to the original analyses. Namely, the initial model indicated that the effect of negative affect at time T-1 was not associated with PTSD symptom severity at

Discussion

The purpose of this study was to utilize EMA to examine the moment-to-moment relationship between PTSD and negative affect, while controlling for smoking and non-smoking readings in individuals with co-occurring PTSD and smoking behaviors. We hypothesized that PTSD would predict negative affect over several hours and days during a baseline week-long time period during which participants smoked *ad lib*. Consistent with past research that has found support for PTSD predicting negative emotionality (Ginzburg et al., 2010; Kessler et al., 1995; O'Toole et al., 1998; Orth et al., 2008) and in line with our hypothesis, PTSD predicted moment-to-moment negative affect after controlling for smoking and non-smoking readings, suggesting that PTSD plays a role in the maintenance of negative affect among persons with PTSD.

The findings of our study are largely consistent with prior longitudinal research that found that PTSD predicted symptoms of anxiety, depression, and anger but not vice versa (Ginzburg et al., 2010; Orth et al., 2008), and extend on these findings by showing the occurrence of this relationship at the moment-to-moment level. Our findings are also consistent with those of a prior EMA study demonstrating that PTSD symptoms are predictive of subsequent anger, but not vice versa (Van Voorhees et al., 2018). Current results are consistent with current theoretical models of PTSD and subsequent negative emotional reactions (e.g., Chemtob et al., 1997; Ehlers and Clark, 2000; Foa et al., 1995). For example, the cognitive model of PTSD (Ehlers and Clark, 2000) posits that trauma symptoms develop and are maintained due to deficits in processing a trauma memory and negative appraisals of peritrauma and posttrauma reactions, which increase threat appraisals of regularly non-threatening stimuli. This leads to increased symptoms of hyperarousal and negative affect. These symptoms are maintained with continued use of avoidant cognitive strategies, such as rumination (e.g., Roley et al., 2015). Therefore, this implies that the continued cyclical relationship between PTSD symptoms increases the experience of negative affect. In relation to anger and PTSD, previous work suggests that anger may be a reaction to PTSD symptoms, either as a response to perceiving relatively neutral stimuli as threatening (Chemtob et al., 1997), or as an avoidant coping mechanism to eschew experiencing fear that results when confronted with trauma-related stimuli (Foa et al., 1995). Our results are in contrast to prior research that has found depression precedes PTSD (O'Toole et al., 1998). However, within the context of our study, we utilized a broad definition of negative affect which encompassed anger, anxiety, and depression. Therefore, research into PTSD and specific negative emotional states (e.g., depression or anxiety) is warranted to determine if PTSD drives only certain negative emotions versus general negative emotionality. Broadly, our results are in line with past research and current theories of PTSD, indicating that momentary PTSD symptoms increase the likelihood of experiencing worse negative affect in the near future.

time T ($p = .51$), whereas the effect of PTSD symptom severity at T-1 was significantly related to negative affect levels at time T ($p = .002$). Moreover, dropping the path from negative affect at time T-1 to PTSD symptom severity at time T did not significantly diminish model fit in comparison to the fully cross-lagged model, Satorra-Bentler $\chi^2(1) = 0.45$, $p = .50$.

There was a fairly high rate of current MDD in this sample, which raises the possibility that the results in this study could simply be a product of already existing comorbid symptomatology or symptom overlap. Past research has found that the rates of comorbidity between MDD and PTSD are the same, even after controlling for symptom overlap (Elhai et al., 2008; Grubaugh et al., 2010), which implies other mechanisms must be considered for the comorbidity of these two constructs, beyond negative emotionality. Therefore, the results of this study are likely not just an artifact of overlap between two co-occurring disorders. Current MDD could have had an effect on the relationship in this study; however, our definition of negative affect was broad and covered multiple types of negative emotionality, including anger, which is not part of MDD criteria. Additionally, past EMA research has found that PTSD symptoms appear to drive hostility and irritability (Van Voorhees et al., 2018). This suggests that PTSD predicts and exacerbates symptoms of anxiety, depression, and anger, and these negative emotions are likely to increase if PTSD symptoms worsen over time.

Smoking could play a role in the relationship between PTSD and negative affect. This analysis was carried out among a group of smokers with PTSD, a specific clinical population that might have characteristics that differ from that of other trauma-exposed populations. Cigarette smoking is highly prevalent in individuals with PTSD (Beckham, 1999), and smokers with PTSD report higher levels of anxiety and depression (Beckham et al., 1995). Past research has shown that smoking can temper negative affect following a negative mood induction task (Cook et al., 2017), and smoking is associated with decreased cravings, PTSD symptoms, and negative affect in the presence of trauma-related imagery (Beckham et al., 2007). The use of a sample of smokers could limit generalizability to other clinical populations. Therefore, future research should attempt to replicate these results in other types of clinical samples (e.g., without substance use, etc.).

The results from this study are particularly relevant when considered in the context of clinical work. This study highlights the importance of understanding the moment-to-moment relationship between PTSD and negative affect, as this can show how patients' day-to-day co-occurring symptomatology might be affected by life stressors, which can further complicate functioning. This study also supports current treatments for PTSD that initially primarily focus on traumatic stress. In particular, this study demonstrates a point of intervention in that targeting trauma-related cognitions and behaviors could result in subsequent drops in PTSD symptoms and negative emotionality, and improvements in functioning (Van Voorhees et al., 2018). It also underscores the need to continually assess for negative emotionality reactions or comorbid depressive and anxiety disorders, as these can possibly develop and complicate treatment as it progresses.

Limitations, Strengths, and Future Areas of Study

Certain limitations must be considered when interpreting the results of the current study. This research program relied on self-report data through EMA when documenting PTSD symptom severity and negative affect, which can introduce some bias in reporting of results. Second, we utilized a shortened measure of negative affect and PTSD, which do not specifically account for some symptoms associated with PTSD (e.g., trauma-related

amnesia) or negative affect (e.g., worry in anxiety). Future research should utilize more comprehensive measures of these symptoms, such as the PTSD Checklist for DSM-5 (Weathers et al., 2013), the Patient Health Questionnaire – 9 (for depression; Kroenke et al., 2001), the Generalized Anxiety Disorder – 7 questionnaire (Spitzer et al., 2006), and the Dimensions of Anger Reaction Scale-5 (Forbes et al., 2014). Third, another limitation in our sample was that participants already had PTSD and there was a high incidence rate of MDD, which does not allow us to speak to the full development of PTSD and negative affect. We are only able to speak to which seems to be driving moment to moment symptoms. Future studies should investigate PTSD symptoms and negative affect from peritrauma to an extended time frame post-trauma to provide a more comprehensive picture of the temporal relationship between trauma, PTSD, negative affect, and other associated disorders. Fourth, the sample in this study are all cigarette smokers, which could have a confounding effect on the relationship between PTSD and negative affect. As previously mentioned, future research should examine relations between these variables in other clinical samples. Last, this study utilized DSM-IV data for PTSD, which limits generalizability of the results. In order to have the most up-to-date results to assess relations between these constructs, future studies should use DSM-5 data for PTSD.

Despite the limitations, the study also has several strengths. Above all, it sheds light on the direction of the relationship between daily symptoms of PTSD and negative affect. It utilizes a sample of trauma-exposed participants with co-occurring PTSD and cigarette use. Last, the study uses EMA to investigate this relationship, which allows for a within-in person approach to understanding the relationship between these constructs in a clinical sample. Overall, the study is able to provide a foundation for future research investigating the relationships between DSM-5 PTSD and negative affect in trauma-exposed populations.

Acknowledgements

We would like to thank the participants who volunteered to participate in these studies.

Role of the Funding Source

This research was supported by a Department of Veterans Affairs Clinical Sciences Research and Development (CSR&D) Merit Review award and a grant from the National Institute of Health (R21CA128965). Funding sources had no role in the study design, data collection, analysis, and interpretation, manuscript writing, and the decision to submit results for publication. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health, the Department of Veterans Affairs or United States government, or any of the institutions with which the authors are affiliated.

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Highlights

- This study assessed the relationship between PTSD symptoms and negative affect.
- Moment to moment symptoms were examined using ecological momentary assessment.
- The direction of the relationship was assessed with cross-lagged path analyses.
- PTSD drove the relationship between momentary PTSD symptoms and negative affect.

Figure 1.

Multilevel cross-lagged model of negative affect and PTSD symptom severity. Level-1 effects represent the effects of time-varying factors on momentary negative affect and PTSD symptoms. Level-2 effects represent the effects of between-person variables on momentary negative affect and PTSD symptom severity. PTSD Sx = posttraumatic stress disorder symptoms; IMS = individual-mean standardized; GMS = grand-mean standardized * $p < .05$, ** $p < .01$

Table 1

Sample Demographics and Ecological Momentary Assessment (EMA) Characteristics

	Mean (SD)	Frequency (%)
<i>Baseline Characteristics</i>		
Age (years)	44.35 (10.19)	
Females		59 (47%)
Race		
Black		82 (66%)
White		35 (28%)
Other		7 (6%)
Veterans		43 (35%)
Nicotine dependence	2.95 (1.57)	
PTSD severity (CAPS)	51.34 (20.29)	
Current MDD		58 (46%)
<i>EMA Characteristics</i>		
Observation period (days)	8.47 (2.98)	
Random alarms (non-smoking)	21.62 (14.16)	
Random alarms (smoking)	10.10 (9.32)	
Self-initiated entries (smoking)	59.62 (35.21)	
Mean negative affect	3.64 (3.00)	
Mean PTSD severity	4.22 (2.95)	

Note. $N = 125$. PTSD = posttraumatic stress disorder; CAPS = Clinician-Administered PTSD Scale; MDD = major depressive disorder.

Table 2

Model Fit Statistics

Model	$\chi^2(df)^a$	AIC	BIC
Both Cross-lags	366.19 (3)	48731.76	48877.05
PTSD Symptoms -> Negative Affect	395.83 (4)	48730.34	48869.03
Negative Affect -> PTSD Symptoms	412.63 (4)	48759.46	48898.15
No Cross-lags	406.11 (5)	48766.90	48898.99

^a All models were significant at $p < .001$.