

Emotional lability and affective synchrony in posttraumatic stress disorder pathology[☆]

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ABSTRACT

This study examined the relations of PTSD pathology to both the lability of three specific emotions (anxiety, anger, self-conscious emotions [SCE]) and the extent to which changes in one emotional state co-occur with changes in another emotional state (i.e. affective synchrony). Moreover, given evidence that emotional responding in PTSD may be heightened in response to trauma-related cues, these relations were explored in the context of a trauma cue versus neutral cue. Trauma-exposed patients in residential substance use disorder treatment ($N = 157$) completed a diagnostic interview and two laboratory sessions involving presentation of neutral and individualized trauma scripts. State anxiety, anger, and SCE were assessed at five points throughout each laboratory session. Hierarchical linear modeling indicated that participants (regardless of PTSD status) exhibited greater lability of all emotions following the trauma script versus neutral script. Only anger lability was elevated among those with (versus without) a current PTSD diagnosis following the neutral script. Results also revealed synchrony (i.e., positive covariation) between each possible pair of emotions, regardless of PTSD status. Findings suggest that concurrent changes in anxiety and anger may be especially relevant to PTSD symptom severity.

1. Introduction

Difficulties in the experience and expression of emotion have been theorized to play a central role in the development and maintenance of posttraumatic stress disorder (PTSD) symptoms among individuals exposed to a traumatic event (e.g., Carlson & Dalenberg, 2000; Ehlers & Clark, 2000; Foa & Kozak, 1986; Litz, Orsillo, Kaloupek, & Weathers, 2000). For example, research indicates that trauma-exposed individuals with elevated PTSD symptoms report heightened emotional responding to emotionally evocative stimuli (e.g., Badour & Feldner, 2013; Orsillo, Batten, Plumb, Luterek, & Roessner, 2004) and reduced emotional responding to positive stimuli shortly after exposure to trauma cues (e.g., Litz et al., 2000). PTSD symptoms are also positively associated with broad deficits in emotion regulation (e.g., Tull, Barrett, McMillan, & Roemer, 2007).

One aspect of emotional dysfunction that has received less attention in PTSD is emotional lability (i.e., intense, frequent, and reactive shifts in emotional experience). Additional research in this area may be important for furthering our understanding of the possible ways in which specific negative beliefs about emotions develop among individuals

with PTSD, including the perception of emotions as being unpredictable or out-of-control (Price, Monson, Callahan, & Rodriguez, 2006). Such research may also assist in identifying vulnerability factors for the development of specific maladaptive behaviors (e.g., substance misuse; see Simons, Carey, & Gaher, 2004) among patients with PTSD. Although limited, research provides evidence of a positive association between PTSD symptoms and trait emotional lability (Lindley, Carlson, & Benoit, 2004; Marshall-Berenz, Morrison, Schumacher, & Coffey, 2011), and suggests that trait emotional lability may interfere with habituation to trauma-related cues in PTSD. Specifically, Dutton, Badour, Jones, Mischel, and Feldner (2016) found that repeated exposure to trauma cues facilitated reductions in state PTSD symptoms only in the context of low trait emotional lability. Additionally, experience sampling studies have revealed positive associations between PTSD pathology and negative emotion lability, with Newton and Ho (2008) finding that state lability of anxiety and anger over a one day period was associated with greater PTSD symptom severity among women exposed to interpersonal traumas. Similarly, Kashdan and colleagues found that Veterans with (vs. without) PTSD reported more frequent and intense shifting of negative emotions from day-to-day in a

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two-week daily diary study (Kashdan, Uswatte, Steger, & Julian, 2006). Despite this research, however, no studies to date have examined associations between PTSD symptom severity and changes in individual emotional states in response to trauma-relevant emotionally evocative cues. Research is also needed to investigate lability at the level of mixed emotions (i.e., affective synchrony) in the context of PTSD pathology.

Affective synchrony refers to the extent to which changes in one emotional state tend to co-occur with changes in another emotional state (see Rafaeli, Rogers, & Revelle, 2007). Originally used to refer to pleasant-unpleasant emotion mixtures, recent studies have begun examining the co-occurrence of similarly valenced emotions (given the prominence of unpleasant emotional states in psychopathology; Schoenleber et al., 2016; Scott et al., 2015). To date, only one study has considered affective synchrony in PTSD Kleim, Graham, Bryant, and Ehlers (2013) asked a sample of trauma-exposed individuals to report state levels of various unpleasant emotions (i.e., fear, helplessness, anger, guilt, and shame) following naturally occurring intrusive memories over the course of a week. Results suggested that emotions generally demonstrated synchrony (i.e., positive covariation) following intrusive trauma memories, with the exception of fear and shame, which evidenced de-synchrony (i.e., co-occurring changes in the opposite direction; Rafaeli et al., 2007). However, relations between PTSD symptom severity and emotion covariations were not reported, limiting our knowledge of how affective synchrony relates to PTSD pathology.

Notably, theory and research highlight the particular relevance of anxiety, anger, and self-conscious emotions (SCE; i.e., emotions elicited when individuals negatively evaluate their personal characteristics and/or behavior; cf. Tangney & Tracy, 2012) to PTSD pathology. Anxiety and fear are key elements of prominent theories of the development of PTSD (e.g., Foa, Steketee, & Rothbaum, 1989), and PTSD symptoms are positively related to anxiety intensity and lability (Newton & Ho, 2008). Anger is also common among individuals with traumatic exposure and PTSD pathology (e.g., Olatunji, Ciesielski, & Tolin, 2010; Orth & Wieland, 2006; see also McHugh, Forbes, Bates, Hopwood, & Creamer, 2012). Individuals with PTSD who report high levels of anger have more severe PTSD symptoms (see McHugh et al., 2012), and PTSD symptom severity is associated with anger lability (Newton & Ho, 2008). Furthermore, cognitive models of PTSD (e.g., Ehlers & Clark, 2000) suggest that anger may arise alongside other emotions as a result of negative appraisals related to the traumatic event and its consequences (e.g., “the world is a dangerous and unfair place”). Such appraisals have been found to be associated with trauma-related anger (Whiting & Bryant, 2007). Finally, negative appraisals of the self or behavior at the time of, or subsequent to, the event are thought to elicit shame and guilt (e.g., “I deserved what happened to me;” “what happened was my fault;” Ehlers & Clark, 2000). Indeed, several studies support the associations of PTSD pathology with SCE and SCE-related traits, such as shame (Andrews, Brewin, Rose, & Kirk, 2000) and shame-proneness (e.g., Andrews, Brewin, Stewart, Philpott, & Hejdenberg, 2009; Robinaugh & McNally, 2010; Schoenleber, Sippel, Jakupcak, & Tull, 2015).

Proposed functional relationships among these emotions in the context of PTSD pathology suggest that they may exhibit shared patterns of change over time. For example, anger in PTSD has been conceptualized as a reaction to the onset of anxiety in the presence of a perceived trauma-related threat (e.g., Foa, Riggs, Massie, & Yarczower, 1995; Feeny, Zoellner, & Foa, 2000). Consistent with this view, Novaco and Chemtob (1998) found that treatments that primarily target the anxiety-related symptoms of PTSD also reduce anger. Similarly, seminal theories of SCE (e.g., Lewis, 1971) and newer theories of SCE in the context of psychopathology (e.g., Bateman & Fonagy, 2004; Schoenleber & Berenbaum, 2012) assert that anger responses can reflect efforts to down-regulate SCE. Numerous studies have reported positive associations between SCE and anger responses (e.g., Heaven, Ciarrochi, & Leeson, 2010; Tangney, Wagner, Fletcher, & Gramzow, 1992), including among individuals exposed to potentially traumatic events

(e.g., Schoenleber et al., 2015), suggesting that anger and SCE may demonstrate synchronous change over time in trauma-exposed populations. Finally, although Kleim et al. (2013) found a negative covariation between state fear and shame, other research has demonstrated positive associations between trait anxiety and SCE among adolescents (Paulus, Vanwoerden, Norton, & Sharp, 2016) and between features of anxiety disorders and shame-related traits among adults (Fergus, Valentiner, McGrath, & Jencius, 2010; Schoenleber, Chow, & Berenbaum, 2014).

1.1. Aims of the present study

The present study examined the temporal dynamics of anxiety, anger, and SCE at the individual (i.e., emotional lability) and mixed (i.e., affective synchrony) emotion levels in a high-risk sample of trauma-exposed substance-dependent patients with and without PTSD. Such a sample is particularly relevant, as individuals with substance dependence experience elevated levels of both emotional dysfunction (e.g., Berking et al., 2011) and PTSD symptoms (e.g., Brady, Back, & Coffey, 2004). Moreover, given evidence that individuals with PTSD who use substances exhibit greater emotional reactivity than those with PTSD and no substance use (e.g., Beckham et al., 2007), the use of this sample increases the likelihood of observing fluctuations in emotions over time (thus providing a better context in which to test our hypotheses).

We expected individuals with a PTSD diagnosis to demonstrate greater lability of anxiety, anger, and SCE relative to those without PTSD. Moreover, in line with the theorized functional relationships among these emotions described above, we hypothesized that individuals with PTSD would generally report greater affective synchrony among anxiety, anger, and SCE over time. Furthermore, in light of existing research demonstrating the relevance of trauma-related stimuli to the heightened emotional responding seen in PTSD pathology (e.g., Badour et al., 2011), we hypothesized the between-group differences in lability and synchrony would be most prominent in the context of trauma-related cues (vs. neutral cues). Finally, given evidence that trauma-exposed individuals who do not meet full criteria for a diagnosis of PTSD may also exhibit difficulties in the experience and regulation of emotion (Jakupcak et al., 2007), we examined lability and synchrony in relation to PTSD symptom severity. Specifically, because the relevance of affective synchrony may depend on the extent to which each individual emotion is changing over time (e.g., Schoenleber et al., 2016), we hypothesized that synchrony between any pair of emotions would be more strongly related to PTSD symptom severity when the lability of one or both of the involved emotions was high.

2. Method

2.1. Participants

A total of 184 adults were recruited for a larger study on risk-taking propensity among individuals with PTSD receiving treatment for substance use disorders at a residential treatment facility in the Southern United States. The present study included the 157 participants (52.2% female) who reported experiencing at least one Criterion A traumatic event. Participants reported experiencing a variety of traumatic events, with the most common being sudden unexpected death of a loved one (20.4%), sexual assault (18.5%), assault with a weapon (17.2%), and physical assault (15.3%). Notably, 153 (97.5%) participants reported multiple types of traumatic events. All participants met criteria for alcohol dependence (27.4%), cocaine dependence (29.3%), or both (43.3%). Participants ranged in age from 18 to 59 years ($M = 34.4$ years; $SD = 9.9$) and were ethnically diverse (61.8% White; 34.4% African-American; 1.9% Latino/a; 1.3% Native-American). More than half of participants were single (64.3%) and unemployed (66.9%).

2.2. Measures

2.2.1. Clinician-administered PTSD scale (CAPS; Blake et al., 1995)

The CAPS was administered to obtain an interview-based assessment of DSM-IV (American Psychiatric Association (APA), 2000) PTSD diagnostic status, as well as a measure of PTSD symptom severity (i.e., sum of frequency and intensity ratings). In previous research, the CAPS has demonstrated good reliability and validity within various populations (e.g., Weathers, Keane, & Davidson, 2001). In this study, all interviews were conducted by bachelors- or masters-level clinical assessors trained to reliability with the principal investigator (MTT). Discrepancies were discussed as a group and a consensus was reached. In this sample, lifetime prevalence of PTSD was 59.9%, with 50 participants (31.8%) meeting criteria for current PTSD.

2.2.2. Positive and negative affect schedule (PANAS; Watson, Clark, & Tellegen, 1988)

To assess state unpleasant emotions, all participants completed the negative affect (NA) subscale of the PANAS five times during each of two lab sessions (see Procedures), for a total of 10 PANAS administrations. All items were rated on a 5-point scale (1 = “very slightly or not at all” to 5 = “extremely;” Watson et al., 1988). As has been found in previous research (Schoenleber et al., 2016), a principal components analysis using direct oblimin rotation indicated that the PANAS items reflect separable Anxiety (*nervous, afraid, scared*), Anger (*hostile, irritable*), and Self-Conscious Emotion (SCE; *ashamed, guilty*) components.¹ Mean scores for Anxiety, Anger, and SCE were therefore computed for each of the 10 PANAS administrations.

Scores reflecting the temporal dynamics of these emotions both at an individual level (i.e., lability) and at a mixed emotion level (i.e., synchrony) were then generated using the PANAS mean scores. Scores were computed for each of the two lab sessions (see Procedures) separately. First, lability of each type of emotion was computed as the mean squared successive difference (MSSD; Ebner-Priemer, Eid, Kleindienst, Stabenow, & Trull, 2009; von Neumann, Kent, Bellinson, & Hart, 1941). Though several methods of computing lability have been proposed (see Moskowitz & Zuroff, 2004), MSSD is unique in that it captures three contributors to affective lability: a) the effect of temporal dependency of scores; b) the amplitude, or degree of change; and c) the frequency of score changes. Therefore, MSSD scores represent the average fluctuation in each emotion over time, within each lab session. Second, synchrony scores for each pair of emotions (i.e., Anxiety-Anger, Anxiety-SCE, Anger-SCE) were computed for each of the two lab sessions to allow for examination of the interaction between individual-level and mixed-level temporal dynamics. Synchrony scores for each session were computed as the within-subject intraclass correlation (ICC; Shrout & Fleiss, 1979) using consistency agreement; all ICCs were transformed to Fisher’s *Z*’ scores for subsequent use. Thus, synchrony scores captured the degree to which two emotions changed together across the course of the laboratory procedures, within each lab session.

2.3. Procedures

All methods received prior Institutional Review Board approval from the University of Mississippi Medical Center and the Mississippi State Hospital. After providing written informed consent, all participants completed three study sessions. All procedures began 72 h or more after admission to the residential treatment facility to ensure that

¹ We conducted a principal components analysis using all of the PANAS NA items that do not reflect a general unpleasant emotional state (i.e. *distressed* and *upset* were not included). As in past work (Schoenleber et al., 2016), our analyses indicated that *jittery* cross-loaded on the Anxiety (loading = 0.607) and Anger (loading = 0.378) components and was therefore not included when computing Anxiety scores. All other items for Anxiety, Anger, and SCE loaded onto their respective components at or above 0.800, while not loading above 0.300 on either of the other components.

withdrawal symptoms would be negligible. Procedures involving the assessment of state emotions did not occur until at least 5 days post-admission. Session 1 consisted of participation in a diagnostic interview (which included the CAPS) and completion of questionnaires not relevant to the present study. During the interviews, detailed information regarding participants’ Criterion A events was gathered for use in the creation of 1-min personalized trauma script recordings, as has been done in previous research (e.g., Keane et al., 1998). A neutral script recording (a description of morning routine) was also created for use as a comparison for all participants in this study.

Session 1 was followed by two lab sessions, with session 2 occurring approximately 2 days after session 1 and session 3 occurring approximately 2 days after session 2. During one lab session (i.e., Trauma Script session), participants listened to their trauma script recording and were instructed to vividly imagine the events in the script with their eyes closed for one minute. Afterward, participants completed one of two computerized tasks assessing risk-taking propensity. Participants then listened to their trauma script a second time, and completed the other risk-taking task. Changes in emotions over the course of the session, rather than task performance, was the primary outcome of interest. Thus, participants completed the PANAS-NA subscale at baseline (before the initial script presentation), after the initial presentation (and before the first computerized task), after the first computerized task (and before the second presentation of the script), after the second script presentation (and before the second computerized task), and after the completion of the second computerized task, for a total of 5 administrations in each lab session. The second lab session followed the same procedures but involved presentation of the neutral script recording (i.e., Neutral Script session). Order of the lab sessions was counterbalanced across participants. Participants were reimbursed a total of \$55 for participating in all sessions.

3. Results

3.1. Emotion changes in response to trauma and neutral scripts

HLM 7.01 (Scientific Software International, 2001) was used to perform initial hierarchical linear modeling analyses, which accounted for the nesting of assessments within persons. A no intercept level 1 model, with two level 1 indicator variables that represented Trauma Script trial (Neutral = 0, Trauma = 1) and Neutral Script trial (Trauma = 0, Neutral = 1), was used to examine total-sample within-person mean differences for each emotion for each trial type (see Table 1). A level 2 indicator variable of CAPS-assessed current PTSD status (0 = No-PTSD, 1 = PTSD) was added to evaluate mean emotion scores within each session for individuals who met and did not meet PTSD diagnostic criteria. All models employed heterogeneous variance estimates for PTSD and No-PTSD groups. The multiple equation form of the models (see Raudenbush & Bryk, 2001) with the level 2 indicator is presented below:

$$\text{Level 1: } Y_{ij} = \beta_{1j}(\text{Trauma Trial}) + \beta_{2j}(\text{Neutral Trial}) + r_{ij}$$

$$\text{Level 2: } \beta_{1j} = \gamma_{10} + \gamma_{11}(\text{PTSD Status}) + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(\text{PTSD Status}) + \mu_{2j}$$

Within these models, Y_{ij} represents assessment *i* for person *j*, β_{1j} estimates the mean for individual *j* during Trauma Script trials, and β_{2j} estimates the mean for individual *j* during Neutral Script trials. These means become outcomes at level 2, where γ_{10} represents the group mean for individuals who did not meet PTSD diagnostic criteria during Trauma Script trials and γ_{11} represents the mean change following Trauma Script trials associated with meeting PTSD diagnostic criteria. The respective coefficients for β_{2j} (i.e., γ_{20} and γ_{21}) allow for examination of Neutral Script trials.

As shown in Table 1, participants reported significantly higher

Table 1
Differences in Emotion Means and Emotional Lability by PTSD Status and Trial Type.

Emotion	Total Sample			No PTSD			PTSD		
	Trial Type		$\chi^2(1)$	Trial Type		$\chi^2(1)$	Trial Type		$\chi^2(1)$
	Trauma	Neutral		Trauma	Neutral		Trauma	Neutral	
Anxiety	1.608	1.328	29.26***	1.503 ^a	1.268 ^c	13.82***	1.841 ^b	1.458 ^c	19.22***
Anger	1.770	1.455	31.44***	1.620 ^a	1.347 ^c	15.69***	2.106 ^b	1.690 ^d	19.33***
SCE	1.719	1.303	52.74***	1.683 ^a	1.238 ^b	41.33***	1.796 ^a	1.442 ^b	11.92***
MSSD Anxiety	1.118	0.234	59.78***	0.983 ^a	0.202 ^b	12.59***	1.416 ^a	0.335 ^b	15.75***
MSSD Anger	1.350	0.342	63.51***	1.131 ^a	0.239 ^b	26.28***	1.826 ^a	0.613 ^c	14.68***
MSSD SCE	2.093	0.287	54.25***	2.138 ^a	0.229 ^b	42.09***	1.995 ^a	0.462 ^b	10.80**

Note: SCE = Self-Conscious Emotions; PTSD = Posttraumatic Stress Disorder; MSSD = mean squared successive difference.

Note: n(No PTSD) = 105; n(PTSD) = 50.

Note: Significant ($p < 0.05$) between-group differences for each emotion and for a given trial type are indicated with different superscript letters (e.g., PTSD group reported significantly higher anxiety following trauma trials but not neutral trials).

** $p < 0.01$.

*** $p < 0.001$.

levels of all emotions following Trauma Script trials compared to Neutral Script trials, irrespective of PTSD status, all $ps < 0.001$. Moreover, analyses within each diagnostic group indicated that participants both with and without PTSD reported significantly higher levels of all emotions following Trauma Script trials compared to Neutral Script trials, all $ps < 0.001$. Participants with PTSD reported significantly more Anxiety after listening to the Trauma Script recording than those without PTSD, $\gamma_{11} = 0.338$, $t(153) = 2.47$, $p = 0.015$, though this relation was not significant following the Neutral Script recording, $\gamma_{21} = 0.190$, $t(153) = 1.75$, $p = 0.083$. By contrast, participants with PTSD reported more Anger than those without PTSD following both the Trauma, $\gamma_{11} = 0.486$, $t(153) = 3.52$, $p < 0.001$, and Neutral Script, $\gamma_{21} = 0.343$, $t(153) = 2.84$, $p = 0.005$, recordings. For SCE, there were no significant differences following either the Trauma, $\gamma_{11} = 0.113$, $t(153) = 0.82$, $p = 0.413$, or Neutral Script, $\gamma_{21} = 0.204$, $t(153) = 1.56$, $p = 0.102$, recording.

3.1.1. Emotional lability

Within-person lability scores were next considered using MSSD scores. Initially, squared successive difference scores (SSD; i.e., the square of $X_i - X_{i-1}$) were calculated within participants for each emotion. SSDs were entered into two level hierarchical linear models, as described above. Thus, γ_{10} represents the MSSD following Trauma Script trials for individuals not meeting PTSD diagnostic criteria and γ_{11} represents the change in MSSD associated with meeting PTSD diagnostic criteria. As displayed in Table 1, participants reported significantly more lability in all three emotions following Trauma Script trials compared to Neutral Script trials when examined in the total sample and both groups separately, all $ps < 0.01$. No significant between-group differences were found for Anxiety lability after listening to the Trauma Script, $\gamma_{11} = 0.433$, $t(153) = 1.31$, $p = 0.192$, or neutral script, $\gamma_{21} = 0.133$, $t(153) = 1.07$, $p = 0.284$. Likewise, no significant group differences were found for lability in SCE following either the Trauma, $\gamma_{11} = -0.143$, $t(153) = -0.30$, $p = 0.764$, or Neutral Script, $\gamma_{21} = 0.233$, $t(153) = 0.99$, $p = 0.325$ recordings. However, individuals with PTSD reported significantly more Anger lability following the Neutral Script recording than those without PTSD, $\gamma_{21} = 0.373$, $t(153) = 2.01$, $p = 0.046$, although no significant difference was found following the Trauma Script recording, $\gamma_{11} = 0.696$, $t(153) = 1.72$, $p = 0.088$.

3.1.2. Affective synchrony

We next considered the degree to which each pair of emotions (i.e., Anxiety-Anger, Anxiety-SCE, Anger-SCE) covaried over time within individuals by trial type. A contrast coded indicator of trial type (-1 = Neutral, +1 = Trauma), a within-person mean centered

predictor emotion, and an indicator of the trial type x predictor emotion interaction were entered at level 1. The level 2 PTSD indicator was initially retained in the HLM models:

$$\text{Level 1: } Y_{ij} = \beta_{0j} + \beta_{1j}(\text{Trial Type}) + \beta_{2j}(\text{Emotion 2}) + \beta_{3j}(\text{Interaction}) + r_{ij}$$

$$\text{Level 2: } \beta_{0j} = \gamma_{00} + \gamma_{01}(\text{PTSD Status}) + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{PTSD Status}) + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(\text{PTSD Status}) + \mu_{2j}$$

$$\beta_{3j} = \gamma_{30} + \gamma_{31}(\text{PTSD Status}) + \mu_{3j}$$

In this case, β_{0j} represents the mean emotion for individual j , controlling for all other predictors in the model. For individuals without PTSD, γ_{10} represents the within person effect of trial type, γ_{20} represents the within person relation between emotions 1 and 2 (e.g., between Anxiety and Anger) for individuals at their own mean of emotion 2, and γ_{30} represents the average within person effect of trial type by predictor emotion interaction. γ coefficients ending with 1 represent the change in these relations associated with meeting PTSD diagnostic criteria. However, initial analyses indicated no significant effect of PTSD diagnostic status on any level 1 predictor in the three affective synchrony models, all $ps > 0.07$, suggesting that individuals with and without PTSD reported similar patterns of relations between emotions during the laboratory session. Thus, the level 2 PTSD indicator was dropped from each model. As such, each γ coefficient reported below represents relations for the entire sample, irrespective of PTSD diagnostic status.

Replicating previous analyses, trial type was positively related with each emotion in the full sample (see Table 2). Results also indicated significant positive covariation (i.e., synchrony) between each pair of emotions across all trials (Anxiety-Anger: $\gamma_{20} = 0.451$, $t[154] = 8.10$, $p < 0.001$; Anxiety-SCE: $\gamma_{20} = 0.635$, $t[154] = 10.58$, $p < 0.001$; Anger-SCE: $\gamma_{20} = 0.411$, $t[154] = 10.84$, $p < 0.001$). The Anxiety x

Table 2
Associations between Emotions Over Time (i.e., Affective Synchrony) by Trial Type.

Emotion Pair	Intercept	Trial ^a	Predictor Emotion	Trial x Emotion
Anxiety-Anger	1.603	0.099***	0.451***	0.103 [†]
Anxiety-SCE	1.486	0.110***	0.635***	0.232***
Anger-SCE	1.466	0.134***	0.411***	0.253***

Note: SCE = Self-Conscious Emotions.

^a Contrast coded indicator of trial type (-1 = neutral, +1 = trauma).

[†] $p = 0.051$.

*** $p < 0.001$.

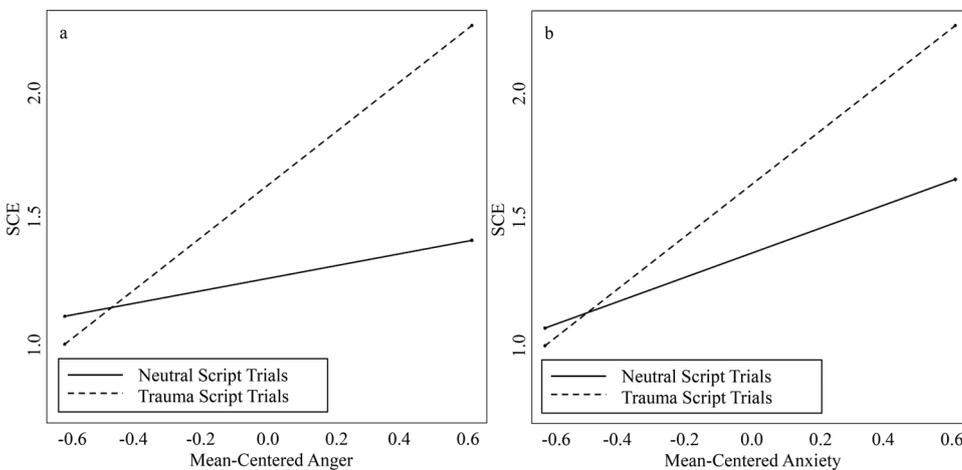


Fig. 1. Simple slopes illustrating average within-person SCE-Anger synchrony by trial type (panel a; left) and SCE-Anxiety synchrony x trial type (panel b; right). Anger and anxiety, respectively, are within-person mean centered and x-axis end-points represent -1 standard deviation and $+1$ standard deviation. SCE = self-conscious emotion.

Trial interaction term failed to reach significance in the Anxiety-Anger relation, $\gamma_{30} = 0.103$, $t(154) = 1.97$, $p = 0.051$, suggesting that although individuals reported more anger both in response to Trauma trials and when experiencing anxiety, this relation did not differ significantly by trial type. However, the predictor emotion x trial interaction was significant in the Anxiety-SCE, $\gamma_{30} = 0.232$, $t[154] = 4.80$, $p < 0.001$, and Anger-SCE, $\gamma_{30} = 0.253$, $t[154] = 5.49$, $p < 0.001$, relations, suggesting that affective synchrony varied by trial type for both pairings. Inspection of the simple slopes plots (see Fig. 1) indicated that both Anxiety-SCE synchrony and Anger-SCE synchrony were particularly strong at high levels of the predictor emotion during Trauma Script trials.

3.2. Interplay between lability and synchrony in relation to PTSD symptom severity

Synchrony's association with PTSD symptoms may be influenced by the degree to which the relevant emotions are changing over time at an individual level. As such, we next ran a series of hierarchical multiple regression analyses for each of the possible emotion pairs to examine the interactions of lability and synchrony in relation to PTSD symptom severity, using the CAPS total score as our outcome variable. Specifically, in each analysis, MSSD scores for both of the involved emotions (i.e., their labilities) and the ICC for that pair of emotions (i.e., synchronous change between them) were entered in Step 1, followed by all two-way interactions in Step 2, and the three-way interaction between both labilities and synchrony in Step 3. All independent variables were centered prior to creating interaction terms, and significant interactions were probed using simple slopes analyses, following Aiken and West (1991). Effect sizes (f^2) are provided below for all statistics with p -values ≤ 0.05 . Given the results of the HLM analyses, all of the regression analyses were run using scores from the Trauma Script sessions only.

3.2.1. Anxiety and anger

The results of the analysis examining Anxiety and Anger are presented in Table 3. In Step 1, neither the Anxiety nor Anger lability scores were significantly associated with PTSD symptom severity. However, Anxiety-Anger synchrony was significantly positively associated with PTSD symptom severity. Of all the two- and three-way interactions in Steps 2 and 3, only the interaction between Anger lability and Anxiety-Anger synchrony was significant ($\beta = 0.19$, $p = 0.02$, 95% CI: [.03, 0.33], $f^2 = 0.035$). Simple slopes analyses indicated that there was a non-significant negative association between Anxiety-Anger synchrony and PTSD severity when Anger lability was low ($\beta = -0.05$, $p = 0.70$, 95% CI: [-0.31, 0.21], $f^2 = 0.001$) but a significant positive association when lability was high ($\beta = 0.60$, $p = 0.001$, 95% CI: [.23,

0.90], $f^2 = 0.075$). Thus, shared patterns of change in Anxiety and Anger were associated with greater PTSD symptom severity, but only in the context of larger shifts in Anger over time (regardless of magnitude of shifting in Anxiety).

3.2.2. Anxiety and SCE

As shown in Table 3, a main effect for Anxiety lability ($\beta = 0.17$, $p = 0.050$, 95% CI: [.00, 0.33], $f^2 = 0.026$) was found in the analyses examining the combination of Anxiety and SCE in relation to PTSD symptom severity. No other variables, nor any of the interactions, were significantly associated with overall PTSD symptom severity.

3.2.3. Anger and SCE

Table 3 also shows the results of the analysis examining Anger and SCE. In Step 1, Anger lability was significantly associated with overall PTSD symptom severity ($\beta = 0.23$, $p = 0.006$, 95% CI: [0.06, 0.37], $f^2 = 0.051$), whereas SCE lability and Anger-SCE synchrony were not. As in the Anxiety-SCE analysis, no other main or interaction effects were found in relation to PTSD symptom severity.

4. Discussion

The goal of the present study was to examine individual- (i.e., lability) and mixed-level (i.e., synchrony) emotional changes over time within a sample of trauma-exposed substance-dependent patients with PTSD pathology. Results contribute to the literature on emotional functioning in PTSD pathology in multiple ways. First, our findings provide additional support for the heightened reactivity of unpleasant emotions among individuals with PTSD, particularly in response to trauma cue exposure; for example, evidence that trauma cues elicit greater SCE reactivity than neutral cues is consistent with existing theoretical and empirical literature on the relation between SCE and PTSD (e.g., Ehlers & Clark, 2000). Second, with regard to changes in emotion over time at the level of individual emotions, we found that trauma-exposed individuals with and without PTSD generally reported similar levels of anxiety, anger, and SCE lability. However, between-group differences in anger lability were found during the neutral script session. Third, at the level of mixed emotions, all possible pairs of emotion demonstrated synchronous change, particularly in response to trauma cue exposure. Additionally, results suggest that concurrent changes in anxiety and anger may be especially relevant to PTSD symptom severity.

Together with findings from past research, the present findings suggest that both context and emotion type matter when determining the importance of emotional lability to PTSD pathology. Lability of all emotions was greater in trauma script sessions than in neutral script sessions, suggesting that trauma-relevant contexts elicit greater changes

Table 3
Summary of Regression Analyses Examining Main and Interactive Relations of Emotional Lability and Synchrony to PTSD Symptom Severity.

	Outcome: CAPS Total Score								
	Anxiety ₁ -Anger ₂			Anxiety ₁ -SCE ₂			Anger ₁ -SCE ₂		
	β	ΔR^2	95% CI	β	ΔR^2	95% CI	β	ΔR^2	95% CI
<i>Step 1</i>		0.09**			0.04			0.05*	
Lability ₁	0.05		-0.11, 0.22	0.17 [†]		0.00, 0.33	0.23**		0.06, 0.37
Lability ₂	0.16		-0.01, 0.31	0.00		-0.17, 0.16	-0.02		-0.18, 0.14
Synchrony	0.19 [†]		0.03, 0.33	-0.13		-0.27, 0.02	0.04		-0.16, 0.27
<i>Step 2</i>		0.04			0.02			0.01	
Lability ₁ x Lability ₂	-0.21		-0.26, 0.03	0.00		-0.11, 0.12	-0.01		-0.13, 0.12
Lability ₁ x Synchrony	-0.06		-0.37, 0.23	0.14		-1.04, 2.51	0.10		-0.35, 0.61
Lability ₂ x Synchrony	0.30 [†]		0.06, 0.55	0.14		-1.54, 2.38	0.05		-0.10, 0.26
<i>Step 3</i>		0.00			0.02			0.02	
3-way Interaction	-0.08		-0.25, 0.16	0.20		-0.18, 1.94	0.24		-0.04, 0.58

Note: CAPS = Clinician-Administered PTSD Scale; SCE = Self-Conscious Emotions.

Note: Subscripts indicate which emotion's lability is presented; for example, in the first analysis, Anxiety Lability is presented in the first row under Step 1, followed by Anger Lability in the second row.

* $p \leq 0.05$.

** $p \leq 0.01$.

in these emotions over time. However, emotional responses to neutral contexts reveal the particular relevance of anger to PTSD. In the only past study to examine between-group differences in lability among trauma-exposed individuals, Kashdan et al. (2006) found that those with (vs. without) PTSD experienced greater negative affect lability in response to everyday (i.e., non-trauma-related) events assessed via daily diaries. Similarly, the only between-group difference we found was in the context of the neutral script session, rather than the trauma script session; individuals with PTSD reported greater anger lability than those without PTSD. Interestingly, Kashdan et al.'s (2006) findings were also somewhat specific to anger; although described as general negative affect, half of the items on their measure were anger terms (*angry, frustrated, irritable*). Thus, whereas trauma-exposed individuals – regardless of PTSD diagnostic status – experience similar lability of anxiety, anger, and SCE when exposed to trauma cues, only those who meet criteria for PTSD are additionally demonstrating increased anger lability in the context of non-trauma-related stimuli. These results suggest that (1) emotional lability among trauma-exposed individuals is not dependent on the presence of PTSD, and (2) anger lability in relation to non-trauma stressors may distinguish between trauma-exposed individuals with and without PTSD.

Compared with anger, the individual-level dynamics of anxiety and SCE were less relevant to PTSD pathology. Although the lability of both emotions was expectedly greater during the trauma versus neutral script session, there were no differences between those with and without PTSD in anxiety or SCE lability regardless of context. Moreover, SCE lability was not associated with PTSD symptom severity in any regression analyses. Similar to the one existing study considering anxiety lability in a trauma-exposed sample (Newton & Ho, 2008), the zero-order correlation between anxiety lability and overall PTSD symptom severity was small but statistically significant ($r = 0.16$, $p = 0.044$). Yet, although anxiety lability was associated with PTSD symptom severity in the anxiety-SCE analysis, this association was non-significant in the regression analysis that accounted for anger lability. Ultimately, at the level of individual emotions, the present study suggests that changes in anger are particularly important to PTSD symptoms among trauma-exposed individuals. Nonetheless, it is important to consider that this finding may be a consequence of examining these relations within a sample of patients with substance use disorders – a sample that may be more likely to exhibit externalizing features of PTSD (see e.g., Wolf, Miller, Harrington, & Reardon, 2012). As a result, the participants in this sample may be more likely to exhibit reactive anger relative to other emotions.

With regard to affective synchrony between emotions, anger lability

influenced the degree to which tandem changes in anxiety and anger were associated with PTSD symptom severity. Specifically, changes in anxiety were associated with PTSD symptom severity, but only when those changes co-occurred with large changes in anger. These findings are potentially consistent with cognitive models of PTSD (e.g., Ehlers & Clark, 2000) that suggest that trauma cues give rise to a variety of negative appraisals, which contribute simultaneously to anxiety and anger. As such, anxiety and anger would be expected to change together in response to trauma cues. The relevance of anxiety-anger synchrony may also provide support for the fear avoidance theory of PTSD, which posits that anger serves to down-regulate anxiety among individuals with PTSD (e.g., Foa et al., 1995). In contrast to cognitive models, this theory would suggest that anger is a response to the experience of anxiety, being activated almost simultaneously as a learned response that functions to reduce anxiety. However, anger may not be able to displace anxiety completely (or may even give rise to anxiety itself), resulting in these emotions exhibiting a shared pattern of change over the course of trauma cue exposure (with anger being the predominant emotion). Given that some theories (e.g., Foa et al., 1995) suggest a specific directional relation between anxiety and anger, future work will need to employ methods for capturing the onset and movement of these specific emotions in finer detail to clarify their specific relevance to PTSD pathology.

Finally, the shared dynamics of anger and SCE exhibited no meaningful relationships to PTSD pathology, even though SCE positively covaried with both anger and anxiety in response to trauma cues. What is most notable about this finding is that it is in stark contrast to the past work on affective synchrony in borderline personality disorder (BPD; Schoenleber et al., 2016; Scott et al., 2015), a disorder with which PTSD often co-occurs (e.g., Pagura et al., 2010). Whereas past research indicates that co-occurring increases in SCE and anger are particularly relevant to BPD symptom severity among adults and some adolescents (Schoenleber et al., 2016; Scott et al., 2015), PTSD symptom severity appears more strongly related to the mixture of anxiety and anger. Researchers have previously suggested that certain aspects of emotional responding other than emotional lability, such as emotional sensitivity, reactivity, or return to baseline, may distinguish between BPD and PTSD (Santangelo et al., 2014). Our results suggest an additional aspect of emotional responding that may be relevant: i.e., affective synchrony between emotional mixtures that are disorder-specific. That said, subsequent studies using larger samples, including both BPD- and PTSD-specific groups and a co-occurring BPD-PTSD group, will be useful for further elucidating the potential utility of mixed emotion dynamics in differentiating these disorders.

A variety of other directions for future research are also suggested by both the present results and the limitations of this work. Some limitations are related to our sample. First, despite the advantages of using a trauma-exposed sample when examining differences in emotional responding among individuals with and without PTSD, future research is needed to examine differences in emotional lability and affective synchrony as a function of trauma exposure. Second, despite the advantages of using a high-risk clinical sample, ours was currently engaged in residential treatment, which may limit the external validity of the results. Finally, the use of a substance use disorder sample may also have influenced our results, given the relation of substance use to emotional responding (Moberg, Bradford, Kaye, & Curtain, 2017). For example, certain substances (e.g., alcohol) have been found to be associated with increased emotional reactivity to trauma cues (e.g., Coffey, Saladin, Drobles, & Kilpatrick, 2002). Moreover, given the lack of variability in substance use severity within our sample, our ability to examine the associations of emotional lability and/or affective synchrony to substance use severity was limited. Thus, future work is needed to examine lability and synchrony among trauma-exposed clinical populations (1) in comparison to a non-trauma-exposed population, (2) in less restrictive treatment settings, and (3) without co-occurring substance use disorders. In addition, given its potential to distinguish among co-occurring forms of psychopathology, future research should examine the relevance of affective synchrony to individuals with versus without substance use disorders.

Other recommendations for future directions are based on limitations with our methodology. First, as data collection for this project began prior to the publication of the DSM-5, we used the DSM-IV version of the CAPS to assess PTSD and its symptoms. However, the symptoms have expanded and been reorganized in DSM-5 (APA, 2013), necessitating additional research examining lability and synchrony in relation to DSM-5 PTSD pathology. Second, our use of the PANAS NA subscale restricted the unpleasant emotions we were able to examine in this study. In future work, the addition of items assessing state sadness will be important, especially given the prevalence of depression among individuals with PTSD (e.g., Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Third, participants may conflate some emotions on the PANAS that scholars consider distinct; for example, our Anxiety scores actually reflect anxiety/fear and our SCE scores reflect shame/guilt. Although our preliminary analyses justify the use of these composites in the present study, future work may benefit from the use of instructions and/or measures that help participants learn to distinguish between these emotional states (particularly given evidence that PTSD is associated with poor emotional clarity; e.g., Tull et al., 2007). Indeed, differences in the specific emotion terms used in this study versus Kleim et al.'s (2013) study may have contributed to the differences in study results; specifically, whereas Kleim et al. found negative covariation between fear and shame, we found positive covariation between anxiety and SCE. In addition, as an alternative to relying on participants' understanding of differences between these specific emotions, it may be helpful for future research to use other methods of assessing emotional responding (e.g., facial affect coding, physiological measures). Finally, given findings suggesting the relevance of context to anger lability in this study and past work (e.g., Kashdan et al., 2006), future research in this area would benefit from the incorporation of experience sampling methods. Such methods can be used to assess emotional responses to both everyday events (which may be more emotionally evocative than our neutral script recording) and naturally arising trauma cues/intrusions, which will better reflect how individuals experience changes in individual and mixed emotional states in their real lives. This approach will additionally provide an opportunity to compare emotion dynamics in response to different cues and to consider the lability and synchrony of pleasant emotions among trauma-exposed individuals.

Despite limitations, the results of this study highlight the importance of assessing for and targeting anger in the treatment of PTSD pathology. Directly targeting anger during PTSD treatment may be

essential for some patients, given evidence that (a) pre-treatment anger is associated with premature dropout from prolonged exposure for PTSD (Rizvi, Vogt, & Resick, 2009), and (b) standard PTSD treatments do not sufficiently reduce anger (e.g., Stapleton, Taylor, & Asmundson, 2006). To this end, Chemtob, Novaco, Hamada, and Gross (1997) suggest that cognitive and behavioral techniques (e.g., developing hierarchies for anger exposure, restructuring anger-eliciting appraisals) can be used to purposefully address anger among individuals with PTSD, and Becker and Zayfert (2001) identify how specific DBT skills such as mindfulness may assist in addressing anger to facilitate engagement with prolonged exposure. The results of this study also suggest the importance of assessing mixed emotional experiences and the function of their component emotions. To this end, treatments aimed at increasing emotional clarity and awareness of the complexity of emotional responses to trauma cues may be particularly useful. Additionally, clinicians can use information about the shared patterns of change between emotions typically experienced by their patients to better determine the precise emotion regulation strategies that may be most useful. Although the present results warrant replication and our understanding of emotion dynamics in PTSD pathology is still incomplete, consideration of mixed emotions during treatment will enhance the ability of clinicians to address PTSD pathology and its consequences effectively.

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