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The Temporal Dynamics of Posttraumatic Stress Disorder and Trauma-Related Negative Emotions

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THE TEMPORAL DYNAMICS OF POSTTRAUMATIC STRESS DISORDER AND
TRAUMA-RELATED NEGATIVE EMOTIONS

By

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Posttraumatic Stress Disorder is a psychological disorder that includes re-experiencing, avoidance/numbing and hyperarousal symptoms that sometimes develop following exposure to a traumatic event. A large body of theory and research suggests that negative emotions play an important role in the development and maintenance of the disorder. The present study explores the temporal dynamics of PTSD symptoms and trauma-related negative emotions using Ecological Momentary Assessment. Thirty-three participants who reported experiencing a past traumatic event and a clinical level of PTSD symptoms were recruited through mass screening days for introductory to psychology courses. Participants who endorsed a past traumatic event on the Trauma History Screen and scored at least a 44 on the PCL-C were contacted to participate in the study. Participants were asked to carry an Android device that prompted them to complete questions about PTSD symptoms and negative emotions over two weeks at six randomly selected times throughout each day (84 total assessments). The presence of significant trends and cyclical behaviors in PTSD symptom clusters were examined in three participants using time-series analyses. Finally, hypotheses about the relationship between trauma-related negative emotions and PTSD symptoms were tested using multi-level modeling. Results of the study broadly suggest that PTSD symptoms are dynamic over short periods of time.
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Dedication

This work is dedicated to my parents, Maurice and Lori.
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Chapter 1: Background

Posttraumatic Stress Disorder (PTSD) is a psychiatric construct that refers to three clusters of symptoms that sometimes develop following exposure to a traumatic event. The Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition Text-Revision (DSM-IV-TR; American Psychiatric Association [APA], 2000) specified that an individual must experience a traumatic event, criterion A, and report three separate symptom clusters, criteria B-D. Criterion A specifies that certain life-threatening events (A1) and emotional responses (A2) must precede and lead to the development of PTSD symptoms. More specifically, the DSM-IV-TR required that one respond emotionally to a potentially traumatic event with “fear, horror and/or helplessness.” Symptoms of PTSD include re-experiencing the event in a distressing fashion (criterion B), avoiding or numbing oneself to reminders of the event (criterion C) and prolonged and persistent hyperarousal after the event has ended (criterion D).

The Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition (DSM-5; APA, 2013) has introduced major changes to the PTSD diagnosis that are important to note. In an attempt to emphasize the central role of stress in the etiology of several disorders, trauma- and stressor-related disorders was added as a category. PTSD was moved from anxiety disorders to this new category not only to reflect that the symptoms are a reaction to experiencing a traumatic event, but also to indicate that fear is not always a central feature of the disorder. The DSM-5 notes that individuals who experience traumatic events and develop PTSD may instead present with depressed mood and negative thought patterns or dissociation and derealization as predominate symptoms (APA, 2013). Other important changes include the elimination of
peritraumatic emotional reactions from criterion A, the addition of a symptom cluster representing troublesome emotions and cognitions, and the inclusion of a specifier for predominately dissociative symptoms. The present study examines the temporal relationship between persistent negative emotions, a new PTSD symptom within DSM-5, and PTSD symptoms from DSM-IV-TR.

Researching PTSD is important because the prevalence rates of trauma exposure and PTSD are high in the United States. In an attempt to identify prevalence rates of various psychiatric illnesses in the U.S., Kessler et al. (1995) conducted the National Comorbidity Survey (NCS). The Diagnostic Interview Schedule was administered to a nationally representative group of 5,877 individuals ranging in ages from 15 to 54 to determine prevalence rates of different DSM disorders. The rate of individuals who had experienced at least one traumatic event in their lifetimes was 57%, and the lifetime prevalence rate for PTSD in this sample was 7.8%. A recent replication of the original NCS found slightly lower lifetime prevalence rates for PTSD at 6.8% (Kessler, Berglund, Demler, Jin, Merikangas, & Walter, 2005). Certain subgroups are at even greater risk for developing PTSD. For example, a recent summary of epidemiological research suggests that up to 16% of returning combat-deployed veterans will have a PTSD diagnosis (Gates et al., 2012).

Bonnano (2004) notes that several different trajectories can occur following trauma exposure including chronic PTSD, delayed PTSD, PTSD then recovery, and resilience. As noted above, most individuals who experience trauma will display enough resilience to buffer themselves from any harmful and lasting effects. In fact, in some cases it is not uncommon to
experience positive change or posttraumatic growth where an individual develops a greater appreciation for life, enhanced interpersonal relationships and deeper spirituality (Tedeschi & Calhoun, 2004). Because of the varying trajectories that may occur following trauma exposure, identifying risk or protective factors that may affect the course of PTSD is an important avenue for future research.

The current study explores the role that emotions play in temporal course of PTSD symptoms. First, relevant theoretical models of PTSD are reviewed in order to gain an understanding of how PTSD might change over time and how emotions might contribute to the disorder. Next, empirical research is reviewed regarding the relationship between emotions and PTSD symptoms. Finally, ecological momentary assessment is discussed as a data collection strategy that can elucidate some of the dynamical patterns of PTSD symptoms and emotions.

**Theoretical Models of PTSD**

In order to develop an understanding of the expected course of PTSD symptoms over time, a brief review of relevant theory is necessary. Despite a large base of theoretical models from which to choose, only three will be reviewed here because of the importance that they place on either emotions or on the temporal sequence of PTSD symptoms. The three models chosen are the Stress Response Syndromes model (Horowitz, 2001), the Cognitive Model of the Maintenance of PTSD (Ehlers & Clark, 2000), and the Schematic, Propositional, Analogic, Associative, Schematic Systems Model (SPAARS; Dalgleish, 2004; Power & Dalgleish, 1999). While there is significant overlap between these models, each offers a unique perspective on either temporal dynamics or trauma-related emotions.
Stress Response Syndromes (Horowitz, 2001). Whether or not it has been explicitly cited, Horowitz’s Stress Response Syndromes model of psychopathology following trauma exposure has been highly influential in the fields of trauma and PTSD research. Interestingly, despite its influence on many contemporary cognitive models of PTSD, the formulation of this model predates the existence of PTSD as a diagnosis. As a psychodynamic theory rooted in ego psychology, this model argues that humans have a psychological need to continually match new information to preexisting schemas of the self, others and the world. This pressing need is termed the “completion tendency.” If new information is incompatible with existing schemas, then attempts are made to revise both the new information and preexisting schemas until there is agreement. Horowitz considers stressful life events to be any event that presents information currently incompatible with preexisting schemas, and whenever an individual encounters an event that provides discrepant information, such as deaths, severe personal injuries or other stressful events, a period of time must elapse while the individual reconciles these discrepancies. Throughout the assimilation process, information about that event will be kept in “active memory” until the discrepancy is resolved.

Horowitz (2001) described the initial stage following trauma exposure as a “crying out” period in which individuals will alternate between periods of trauma intrusion and denial. Intrusions may take the form of recurrent thoughts, hallucinatory experiences, and nightmares based on details of the event. Denial or a state of disbelief develops as a means of coping with the highly distressing intrusive symptoms. Symptoms of denial include avoiding reminders of the event, social withdrawal and/or emotional numbing. Because information about the event is
stored in “active memory”, an individual experiences a state of chronic activation while the discrepancy is resolved. It is important to note that Horowitz views the “crying out” stage as a normative reaction to a highly stressful event, and the oscillation of intrusion and denial represents an individual’s “working through” the event/schema discrepancy. Individuals develop stress response syndromes, like PTSD or traumatic grief, because their attempts to integrate the trauma information into schematic representations are blocked.

Because Horowitz (2001) speaks broadly about reactions to extreme stress, it is important to consider how his model specifically applies to PTSD. The trauma intrusion and denial states can be easily conceptualized as the re-experiencing and avoidance/numbing symptom clusters. Thus, we might expect alternating periods of re-experiencing symptoms and then avoidance/numbing symptoms in individuals diagnosed with PTSD. Hyperarousal symptoms are hypothesized to result from trauma information being kept in “active memory” and will slowly decrease over time until the assimilation process ends.

Horowitz (2001) also considers negative emotions to be an important part of the clinical presentation of stress response syndromes. He notes that continuing anxiety may result from fears of recurrent intrusive symptoms, the possibility of the event reoccurring, and perceived loss of control over aggressive impulses. Shame and guilt may occur as the result of an increased sense of vulnerability, increased aggressive impulses, and survival of the event (e.g., survivor’s guilt). Intense hostility and anger may develop for the source of the trauma, at individuals exempted from the effects of trauma, and at oneself for feeling increased vulnerability. Because
each event is appraised in light of an individual’s unique schematic representations, the emotional reactions elicited by the traumatic event will widely vary among individuals.

The Horowitz research group (Becker, Horowitz, & Campbell, 1973; Horowitz, 1969; Horowitz & Becker, 1971; Horowitz, Becker, & Moskowitz, 1971) conducted a series of experimental studies that lend some support for the completion tendency concept. The experimental paradigm used in these studies involves showing a highly stressful video, one that arouses either intense negative or positive emotions, to a group of participants. To measure intrusive cognitions, participants are asked to complete two minute auditory tasks after which they list all mental contents and visual images that occurred during the task. It is assumed that an increase in intrusive mental content and cognitions related to the stressful video represent an individual’s attempt to assimilate the information presented in the video. Using the prototypical design of these experiments, Horowitz and Becker (1971) recruited 31 male college students and randomly assigned them to either view a stressful video followed by a neutral video or a neutral video followed by a stressful one. The stressful video depicted violent footage of the circumcision of a teenaged male, while the neutral video profiled the life of a long distance runner. Results indicated participants had more intrusive cognitions in general and greater recall of scenes from the stressful video compared to recall from the neutral video.

Cognitive Model of the Maintenance of PTSD (Ehlers & Clark, 2000). In an attempt to explain why only a small proportion of individuals develop PTSD following exposure to a traumatic event, Ehlers and Clark (2000) developed a cognitive model of PTSD that focuses primarily on the maintenance of PTSD symptoms. Similar to the stress response syndromes
model, Clark and Ehlers posit that most individuals develop at least below-threshold symptoms of PTSD following exposure to a traumatic event, and most of these individuals will see a reduction of symptoms over the course of a few months. However, for many individuals symptoms persist for years. Ehlers and Clark are primarily concerned with understanding the persistence of PTSD symptoms in the latter group.

Ehlers and Clark (2000) argue that PTSD symptoms persist after the traumatic event ends because people continue to experience a perceived sense of threat based on problematic appraisals of the event or the symptoms following the event. Individuals who develop persistent PTSD appraise the event in such a manner that they no longer view the traumatic experience as a time-limited event, but instead view it as having global implications about the future. Problematic appraisals about the event may include statements such as “nowhere is safe” or “I deserve that bad things happened to me” (Ehlers & Clark, 2000, p. 323). Furthermore, negative appraisals about trauma-related consequences, such as “my brain has been damaged” or “I’m dead inside” will also result in the perception of current threat because with these appraisals the individual believes that the trauma is still having an immediate negative impact.

The nature of the appraisal made will also have emotional consequences for the person experiencing a traumatic event. In agreement with Horowitz (2001) Ehlers and Clark (2000) note that most negative emotions, including shame, anger, guilt and fear, are likely to be reported alongside PTSD symptoms. Whether a specific emotion is experienced is dependent upon the type of traumatic event and individual differences in appraisals made about the meaning of the
event. For example, an individual is likely to experience anger if the event is appraised as being unfair or shame if severe and harsh appraisals are made about the self.

In addition to problematic cognitive appraisals, Ehlers and Clark (2000) argue that problems with trauma-related memories also contribute to a continued sense of threat. Problems in memory are typically observed in the findings that while individuals typically have a difficult time intentionally recalling details about the traumatic event, involuntary recall, a primary symptom cluster of the disorder, is often vividly detailed. Problematic trauma memories are characterized by poor integration into autobiographical memories, strong stimulus-stimulus and stimulus-response associations, and strong perceptual priming. Furthermore, negative appraisals are likely to be supported by memories about the event as retrieval attempts are likely to be biased by the appraisals themselves.

**The SPAARS Model** (Dalgleish, 2004; Dalgleish & Power, 2004; Power & Dalgleish, 1999). The SPAARS model argues that the complexity of emotional experiencing requires that researchers use multiple levels of cognitive processing systems to account for the production of emotion. According to Power and Dalgleish (1999) the construction of emotion begins with the occurrence of an event and follows with the sensory processing of that event (analogic level). After the sensory information is registered, the information can then be processed at three different levels: A higher-order schematic level, where the current information is appraised in light of schemas and current goals, an associative level which consists of an architecture of stimulus-stimulus and stimulus-response relationships based on past learning experiences, and a lower-level propositional level which is composed of language and immediate thoughts.
Emotion can be elicited directly via the schematic or associative levels. At the schematic level, emotion can be produced through an appraisal process where the event is considered in light of current goals. For example, if an event threatens one’s goals, then that individual will likely experience fear or anxiety. Emotion can be produced at the associative level through the presentation of stimuli that activates any of the S-S or S-R relationships within the associative network. Finally, the propositional level can lead to emotion indirectly through the schematic or associative system, as certain words or phrases may have a learned response at the associative level or may contribute to the appraisal process.

Although it was initially developed as a model to explain normal emotional functioning, the SPAARS model has been expanded to explain the development of emotional disorders, such as PTSD and depression (Dalgleish, 2004; Dalgleish & Power, 2004; Power & Dalgleish, 1999). Dalgleish (2004) posits that experiencing a traumatic event, one in which fear is the dominant emotion experienced, leads to significant changes at all levels of representation. At a schematic level, the event is evaluated in terms of its implications for ongoing goals, such as keeping one’s previously held schemas intact. Furthermore, information about the event is registered at all other levels, as sights, sounds and smells are kept at the analogical level, trauma-related thoughts are stored at the propositional level, and links between various aspects of the event and emotions are stored at the associative level.

After the event ends, trauma-related information stored at all levels is appraised as threatening to previously held schemas about the self, world and future. As a result, resources within the cognitive processing system are allocated towards assimilating information about the
event into those schemas. Dalgleish argues for a process similar to the one posited by the stress response syndromes model (Horowitz, 2001) as assimilating trauma-related information results in chronic activation of the traumatic event which produces the hyperarousal symptoms observed in PTSD. Furthermore, the assimilation process leads to recurrent intrusions of trauma-related information into consciousness, and the chronic activation of this information leads to biased perceptual processes in which individuals more quickly register and respond to information relevant to the traumatic event.

Thus far, the SPAARS model has conceptualized traumatic events as involving fear as the dominant emotion experienced; however, Dalgleish and Power (2004) contend that not only are other emotions likely to be present and affect the course of PTSD, but they may also sometimes be the dominant emotion experienced during some traumatic events. To describe the role of other emotions in PTSD, Dalgleish and Power differentiate between emotion specific and emotion non-specific components of the disorder. Their model argues that re-experiencing and avoidance primarily result from the cognitive system’s persistent attempts to resolve discrepancies between the traumatic event and previously held beliefs about the world and/or self (Dalgleish & Power, 2004). Depending upon the implications of the discrepancy, an individual may experience a myriad of emotions, including shame, anger, sadness or disgust. Therefore, re-experiencing and avoidance symptoms are seen as emotion-nonspecific symptoms of the disorder because they are the result of an assimilation process that occurs regardless of the dominant emotion experienced. The fact that the dominant emotion experienced during trauma is fear leads to persistent appraisals of threat throughout the assimilation process because trauma-
related, threatening information is chronically activated. Thus, the hyperarousal symptoms can be considered emotion-specific because they result appraisals of threat and a fear response.

Dalgleish and Power (2004) also suggest that PTSD is just one of many possible stress-related emotional disorders. When the dominant appraisal and emotion experienced during a traumatic event are an appraisal of threat and subsequent fear, PTSD is the likely result. However, if the dominant emotion experienced during a traumatic event is another emotion (e.g., sadness or anger), then one might expect a different set of emotion-specific symptoms in addition to the emotion non-specific re-experiencing and avoidance/numbing symptoms. For example, Dalgleish and Power conceptualize traumatic grief as a stress-related disorder in which the dominant emotion experienced during the event is sadness, resulting from appraisals of loss. Whereas it is common for individuals experiencing traumatic grief to cycle between re-experiencing and avoidance/numbing symptoms, it is highly uncommon for those individuals to report experiencing hyperarousal symptoms, such as an exaggerated startle response or persistent hypervigilance. Rather, research suggests that these individuals report chronic nostalgia and persistent yearning for the lost person (Raphael & Martinek, 1997). Dalgleish and Power posit that the symptoms noted above are likely to be specific to experiencing a traumatic event with appraisals of loss and intense sadness.

**Emotions and PTSD**

Examining the relationship between emotions and PTSD initially seems redundant, circular and unnecessary because at its core PTSD is very much an emotional disorder. Its previous classification as an anxiety disorder within the DSM-IV-TR indicated that anxiety,
typically considered a negative emotion or affective state, was a central feature of the disorder. Within DSM-5, it is acknowledged through its inclusion as a trauma- and stressor- related disorder that individuals with PTSD may present with a variety of presentations in addition to the previous fear-based model. Furthermore, the requirement of a traumatic event for diagnosis specifies that an individual experiences an event laden with a myriad of negative emotions. Regardless of whether specific emotions are required, a traumatic event absent of negative emotions is not likely to have a lasting impact on an individual. It is important to note, however, that changes to DSM-5 included the removal of peritraumatic emotional responses from the diagnostic criteria for a traumatic event.

Within theoretical models of PTSD, emotions are often designated as an important feature in the clinical presentation of the disorder. For example, the SPAARS model explains that PTSD symptoms result from and are maintained by specific (fear) and non-specific (shame, guilt, anger, sadness and disgust) trauma-related emotions. Furthermore, the cognitive model of PTSD presented by Ehlers and Clark (2000) similarly argues that persistent negative appraisals and their accompanying emotions prevent an individual from adaptively coping with the typical sequelae that follow traumatic event exposure. Based upon these models, one might hypothesize that significant relationships would exist between various emotions and PTSD symptoms, and this is indeed the case; however, the specific emotions experienced for an individual during and after a traumatic will be highly variable because of the subjective manner in which they perceive the event and its relationship to schemas about the self, world and future.
The role of emotions in PTSD has received a great deal of attention not only in the research literature but also in the development of the diagnostic criteria of the disorder. For example, within the DSM-IV the peritraumatic emotions of “fear, horror and/or helplessness” were given a central role in defining a traumatic event (APA, 2000), although research examining the utility of criterion A2 has been somewhat mixed. While a number of studies have found a significant relationship between criterion A2 and PTSD symptoms (Boals & Schuttler, 2009; Brewin, Andrews, & Rose, 2000; Dewey & Schuldberg, 2013), some argue that A2 lacks clinical utility and does not add to the predictive validity of criterion A1 (Friedman, Resick, Bryant, & Brewin, 2011). Despite the removal of criterion A2, DSM-5 continues to emphasize the importance of negative emotions in the presentation and maintenance of PTSD by adding problematic cognitions and emotions as a new symptom cluster, criterion D (APA, 2013). Symptoms of criterion D are a combination of the numbing symptoms previously included in DSM-IV-TR, and three new symptoms are added: persistent negative emotions, persistent negative appraisals about oneself, others, or the world, and distorted cognitions about the cause of the event and its consequences.

The following sections review the theoretical and empirical literature base for the role of specific emotions (e.g., anger, guilt and shame) and their relationship to the development and maintenance of PTSD. Although there is some preliminary research supporting the inclusion of disgust as a predictor of PTSD symptoms, it is not reviewed in the current study. Additionally, although not reviewed in the document, sadness was measured in the current study. While most studies examining emotions and PTSD have been cross-sectional in nature, an attempt will be
made to shed light on potential temporal relationships. Consistent with the SPAARS model, emotions will be conceptualized as an “experiencing mode” consisting of “an initiating event (external or internal), an interpretation, an appraisal of the interpretation especially in relation to goal relevance, physiological reaction, an action potential, conscious awareness, and overt behaviour” (p. 130, Power & Dalgleish, 1999). This definition was chosen because of its inclusive nature, which is necessary because measurement of emotion varies tremendously across research studies.

**Anger.** Historically, the field of psychology has defined anger in a variety of ways, including as a feeling state, an attitude towards others or even sometimes as hostile behavior (Berkowitz, 1999). Although Berkowitz believes that the multiple definitions of anger have led to a general confusion and lack of clarity in the literature base, these different definitions appear to be measuring separate but related components of the anger experience. The reviewed literature that follows uses the terms anger and hostility interchangeably.

The relationship between anger/hostility and PTSD symptoms has been well-documented across a wide variety of traumatic experiences. Researchers have found significant relationships between anger/hostility and PTSD symptoms in Iraq and Afghanistan war veterans (Jakupcak, et al., 2007), Vietnam war veterans (Taft, Street, Marshall, Dowdall & Riggs, 2007) disaster relief workers following 9/11 (Jayasinghe, Giosan, Evans, Spielman & Difede, 2008), police officers (Meffert, et al., 2008) and crime victims (Andrews et al., 2000; Orth, Cahill, Foa, & Maercker, 2008). Furthermore, individuals experiencing anger in addition to PTSD symptoms are more likely to suffer from lower levels of occupational and social functioning (Evans, Giosan, Patt,
Spielman, & Difede, 2006) and higher rates of suicide attempts and negative health outcomes (Ouimette, Cronkite, Prins, & Moos, 2004). Anger has also been demonstrated to impact recovery from trauma and PTSD negatively in combat veterans in treatment (Forbes, Parslow, Creamer, Allen, McHugh & Hopwood, 2008; Owens, Chard & Cox, 2008). Gender differences have also been noted with respect to clinical presentations of PTSD in victims of interpersonal assault (Galovski, Mott, Young-Xu, & Resick, 2010). Although men and women presented with similar clinical presentations of PTSD symptoms, men reported higher levels of state anger compared to women even though there were not gender differences in trait anger.

To understand better the magnitude of the relationship between anger, hostility and PTSD, Orth & Wieland (2006) conducted a meta-analytic review including published articles through 2003. The authors used “anger, hostility, PTSD, traumatic stress, posttraumatic stress” as search terms and found 189 relevant studies, which were reduced to 38 after applying exclusion criteria. Overall, the results showed large effect sizes for the relationship between anger/hostility and PTSD symptoms. Orth and Wieland found time since trauma to moderate the relationship between anger/hostility and PTSD, as the association between anger and PTSD was low at the time of the event, increased strongly in the months immediately following the event, and then began to decreases until no association is present. Type of event was also found to moderate the relationship, as military war experience yielded the strongest relationship \((r = .56)\) and criminal victimization had the weakest association \((r = .30)\).

Olatunji, Ciesielski and Tolin (2010) conducted a meta-analytic review to explore the specificity of anger in PTSD as opposed to other anxiety disorders. Their research was based on
the close relationship of anxiety/fear and anger in theoretical models. For example, Barlow (2002) views anger and fear responses as related emotions, as fear and anger represent flight and fight, respectively, in the physiological fight/flight response to threat. Olatunji et al. used anger, anxiety and the names of specific anxiety disorders as search terms, and included 28 studies after reviewing over 1,900 articles found using the search criteria. Articles included in the literature review were published prior to August, 2007. The authors were able to compare several control groups (non-clinical control, trauma-exposed control and psychiatric controls) to groups diagnosed with PTSD or other anxiety disorders on various measures of anger. Both the PTSD and other anxiety disorder groups had significantly higher overall anger scores compared to control groups. The mean effect size for PTSD was also significantly greater than all other anxiety disorders in predicting overall anger suggesting some specificity to the relationship between PTSD and anger.

Studies published since the completion of both of these meta-analyses have presented somewhat mixed support for the conclusions of the reviews. For example, analyzing data from the National Comorbidity Study – Replication, Hawkins and Cougle (2011) found anger experience to be significantly associated with all anxiety disorders while controlling for psychiatric comorbidity. However, when depression was added as a covariate, PTSD was not significantly associated with anger experience. Furthermore, PTSD was not found to be associated with anger expression. These findings are inconsistent with the Olatunji et al. (2010) review, and Hawkins and Cougle explain their surprising findings in light of the fact that a large proportion of the sample indicated “loss of a loved one” as their traumatic event.
Given the relationship between anger and PTSD, a pressing concern to researchers and clinicians is the level of anger in veterans returning from Iraq and Afghanistan. Several studies have examined anger and PTSD in this population and some important findings have been noted. Levels of anger and hostility have statistically differentiated between veterans diagnosed with PTSD and those who report sub-threshold symptoms or no symptoms at all (Jakupcak et al., 2007). Elbogen and colleagues (2010) examined three different facets of anger in Iraq/Afghanistan veterans, including aggressive impulses/urges, difficulty managing anger and problems controlling violence, and their relationship to PTSD symptom clusters. Results indicate significant relationships between all three types of anger and hyperarousal symptoms, although only aggressive impulses was associated with re-experiencing symptoms and difficulty managing anger was associated with avoidance symptoms. Finally, veterans with PTSD or traumatic brain injury who reported anger were more likely to be arrested than other veterans (Elbogen et al., 2012). Kulkarni, Porter and Rauch (2012) investigated the relationship between dissociation, anger and PTSD symptoms in veterans entering treatment for PTSD. Both anger and dissociation significantly predicted PTSD symptom scores, and anger significantly predicted avoidance/numbing and hyperarousal symptoms clusters.

**Guilt and Shame.** Although guilt and shame have historically been ignored in trauma research and treatment (Blum, 2008), recent research indicates that they are important factors in an individual’s response to traumatic events. Broadly speaking, shame is typically conceptualized as a severe, negative judgment about the self, while guilt refers to harsh judgments about one’s actions (Tangney, Miller, Flicker, & Barlow 1996). Shame and guilt are
included within the same section because they are similar constructs that are often examined within the same studies. Experiencing intense shame and/or guilt following a traumatic event places an individual at a greater risk for a myriad of other negative outcomes, including general emotion distress (Whiffen & MacIntosh, 2005), suicidality (Wilson, Drozdek, & Turkovic, 2006), and loss of interpersonal connectedness (Dorahy, 2010).

In order to elucidate the unique contributions of shame and guilt to PTSD, Lee, Scragg and Turner (2001) created models of PTSD for each emotion. Based on previous schematic models of PTSD (e.g., Horowitz, 2001), the authors argue that individuals have a strong need to match trauma-related information to schematic representations of the self, world and future. Lee and colleagues note that there are two pathways through which an individual might develop shame or guilt-based PTSD. First, the traumatic event may activate schemas about the self as incompetent. Second, the discrepancy between trauma-related information and schemas results in feelings of humiliation and anger upon thinking of the traumatic event. In this case, shame may develop if previously held schemas about the self are “shattered” by the extreme nature of the event.

Shame and guilt have been linked to PTSD symptoms in both victims and perpetrators of intimate partner violence (IPV; Beck, McNiff, Klapp, Olsen, Avery, & Hagewood, 2011; Hundt & Holloway, 2012; Kubany, Abueg, Owens, Brennan, Kaplan, & Watson, 1995; Sippell & Marshall, 2011; Street and Arias, 2001). Sippell and Marshall (2011) examined the relationship between IPV, PTSD symptoms and shame by using an emotional Stroop task in victims of IPV diagnosed with PTSD. The emotional Stroop task required participants to identify the color of a
word, either neutral or shame-related, by speaking into a microphone. Words were presented at a supraliminal and subliminal level, and the latency of response time was recorded. Participants who responded more quickly to shame-related stimuli were hypothesized to have internal shame-based schemas. Results showed that response time mediated the relationship between PTSD symptoms intensity and IPV perpetration frequency in both the supraliminal and subliminal conditions. Sippel and Marshall suggest that their findings indicate that shame-based internal schemas increase the likelihood of eliciting IPV by affecting perceptual processes, such as leading to perceptions of rejection in ambiguous situations, and behavioral inclinations, like avoiding a partner in order to minimize discomfort and protect self-esteem.

Hundt and Hollohan (2012) explored the relationship between IPV, PTSD, guilt, shame, and depression in male combat-veterans who primarily perpetrated IPV within their relationships. Two hundred and sixty four male combat veterans completed self-report measures and were divided into separate groups based on self-report of perpetrating IPV. The authors used discriminant function analysis to determine if each predictor could significantly predict membership of each group, and the model including all predictor variables was significant, correctly classifying 62% of all cases. Shame was found to be to best predictor of IPV within the model, and it also mediated the relationship between PTSD symptoms and IPV.

Platt and Freyd (2012) conducted an empirical study to understand better the contribution of negative underlying assumptions in the development of shame. Within their study negative underlying assumptions were considered to be the equivalent of negative schemas or assumptions about the self, world or future. Participants completed questionnaires assessing
shame-proneness, trauma history, negative underlying assumptions and PTSD symptoms before completing a simple problem solving activity. After completion of the activity, participants either received positive or negative feedback from an evaluator and state shame was measured using a self-report questionnaire. The results of this research found that the presence of a trauma history was related to higher rates of negative underlying assumptions, and individuals with greater negative underlying assumptions were more likely to respond to negative feedback with shame.

Beck and colleagues (2011) explored the relationship between shame, guilt, PTSD symptoms and abuse type in victims of IPV seeking treatment for PTSD. Results indicated a significant relationship between shame, guilt-distress and guilt-cognitions and PTSD symptoms. High levels of emotional abuse and dominance/isolation, as measured by a structured interview developed by the primary author, moderated the relationship between shame and PTSD symptoms. The authors conclude that the presence of high levels of shame and guilt should guide therapeutic interventions in that exposure-based, fear reduction interventions may not be sufficient.

In addition to IPV, a great deal of research has examined the role of shame and guilt in PTSD among war veterans (Andrews, Brewin, Stewart, Phillpott, & Hejdenberg, 2009; Beckham, Feldman, & Kirby, 1998; Kubany et al., 1995; Leskela, Dieperink, & Thuras, 2002; Wong & Cook, 1992). For example, studying Vietnam War veterans with chronic PTSD, Beckham and colleagues (1998) examined the relationships between atrocity exposure during war, PTSD symptoms, guilt and interpersonal violence. Results indicated that atrocity exposure was related to both PTSD symptoms and several guilt scales, including global guilt and cognitive
aspects of guilt. The correlation between PTSD symptoms and guilt was not reported. Wong and Cook (1992) compared veterans hospitalized for PTSD, depression and substance abuse on measures of self-esteem, internalized shame and depressive symptoms. Participants who had been previously hospitalized for PTSD scored higher in internalized shame as well as depressive symptoms compared to the depression and substance abuse groups.

Using a cross-sectional design, Leskela, Dieperink and Thuras (2002) examined the association between guilt and shame proneness and PTSD symptoms in former prisoner of war veterans. Potential participants were mailed a packet of questionnaires assessing current PTSD symptoms, combat exposure and guilt/shame proneness. Results of this correlational study found a significant relationship between shame proneness and PTSD symptoms, but not for guilt and PTSD symptoms.

Andrews and colleagues (2009) conducted retrospective interviews with combat veterans on disability for PTSD to explore factors that may differentiate between immediate and delayed-onset PTSD. Veterans with immediate-onset PTSD reported experiencing higher levels of shame and anger. The authors suggest although veterans are trained to cope with fear, horror and helplessness that may occur during a traumatic event, they are less equipped to handle other peritraumatic responses, such as shame and anger. Therefore, experiencing those emotions may result in soldiers becoming quickly overwhelmed and developing immediate PTSD.

The work on moral injury in veterans provides a promising new area of research conceptually similar to shame-based PTSD. Moral injury refers to harmful consequences, including but not limited to PTSD symptoms, that result from a soldier engaging in behaviors
that conflict with his or her deep-rooted moral views and expectations (Litz, Stein, Delaney, Lebowitz, Nash, Silva, & Maguen, 2009). Litz and colleagues (2009) argue that the unconventional nature of modern warfare, such as unmarked enemies and urban settings, creates an increasingly ambiguous moral and ethical situation for soldiers as it is more likely that soldiers will have to make decisions that lead to the death of non-combatants. Furthermore, Litz et al. argue that these settings as well as more frequent and longer deployments have led to an increase in soldiers who are likely to violate moral standards and suffer from a range of psychological, social and spiritual consequences.

Shame and guilt have also been implicated in other types of traumatic events, including indirect exposure to trauma through journalistic work (Browne, Evangeli, & Greenberg, 2012), the experience of motor vehicle accidents (Lowinger & Solomon, 2004), childhood sexual abuse (Feiring & Taska, 2005), stressful life events (Robinaugh & McNally, 2010), and in other mixed-trauma samples (Harman & Lee, 2010). Browne et al. found trauma-exposure and guilt cognitions and guilt cognitions and PTSD symptoms to be significantly correlated in journalists exposed to work-related trauma. Furthermore, guilt cognitions partially mediated the relationship between trauma-exposure and PTSD symptoms within this sample.

Using a non-clinical sample, Robinaugh and McNally (2010) examined the mediating role of autobiographical memory in the relationship between state shame and guilt and PTSD symptoms. Participants completed online surveys in which they were asked to recall a specific event associated with high levels of shame and guilt. After identifying an event, participants completed six questionnaires assessing memory, emotions and psychopathology. While a
significant correlation was found between shame and PTSD symptoms, guilt was not a significant predictor. Furthermore, the centrality of the event within participants’ autobiographical memory mediated the relationship between shame and PTSD symptoms, and visual perspective of the memory moderated that relationship.

Lowinger and Solomon (2004) explored the relationships between shame, guilt and PTSD symptoms within men convicted of killing another individual through reckless driving. The researchers matched the clinical group with a sample that was relatively similar in most demographic variables. Compared to the control group, individuals convicted of vehicular homicide were more likely to reported PTSD symptoms. Although queried retrospectively, participants reported high levels of PTSD symptoms immediately after the study, which declined to a moderate level at the time of the study. Furthermore, a significant relationship was found between guilt feelings and PTSD symptoms, and the course of guilt feelings peaked in the period after the event but before the trial, and dropped to low levels at the time of the study.

Feiring and Taska (2005) used a longitudinal study to examine shame’s role in the development and maintenance of PTSD following childhood sexual abuse. Victims of childhood sexual abuse between the ages of eight and fifteen were assessed upon discovery of childhood sexual abuse before treatment and at a one year and six year follow-up. To measure abuse-related shame, the authors developed four questions for this study, and general shame and guilt proneness were evaluated using a developed structured interview. The authors found that participants with high levels of shame at the first assessment were at greater risk for high levels
of shame at the six year follow-up. Furthermore, participants with high levels of shame at both follow-up assessments were more likely to report higher rates of intrusive PTSD symptoms.

Harman and Lee (2010) argue that shame may lead to and maintain PTSD symptoms by eliciting more self-critical and less self-reassuring thinking. The authors used a correlational design including 49 participants recently referred to treatment for PTSD. Participants completed measures assessing PTSD symptoms, shame, and self-critical/self-reassuring thinking styles. Results partially supported the hypotheses of the authors in that shame significantly predicted both PTSD symptoms and critical thinking styles.

**Ecological Momentary Assessment and Clinical Research**

Ecological Momentary Assessment (EMA) is a data collection strategy that allows for continuous, real-time collection of self-report data in a research participant's natural environment (Stone & Shiffman, 1994). EMA is an ideal research technique for psychological processes, such as thoughts, emotions, and behaviors that are thought to be dynamic in nature because ongoing collection of data in the real world may be more sensitive to temporal fluctuations than other research designs. This unique methodological approach has three primary advantages, including avoiding recall bias, enabling the study of processes over time and enhancing ecological validity (Stone, Shiffman, Atienza, & Nebeling, 2007).

Because there is increasing consensus both that memory heuristics can undermine the accuracy of retrospective recall and that psychiatric symptoms tend to be dynamic in nature, EMA is an ideal design for clinical research (Trull & Ebner-Priemer, 2009). The advantages of using EMA with clinical populations have led to a proliferation in EMA research across a wide
range of clinical conditions. EMA has been used to study various aspects of mood disorders (Ebner-Priemer & Trull, 2009), substance misuse (Litt, Cooney, & Morse, 1998), psychosis (Oorschot, Kwapil, Delespaul, & Myin-Germeys, 2009), schizophrenia (Granholm, Loh, & Swendson, 2008), panic disorder and specific phobia (Alpers, 2009), borderline personality disorder (Ebner-Priemer & Sawitzki, 2007), binge eating (Haedt-Matt & Keel, 2011) and chronic pain (Bruehl, Liu, Burns, Chont, & Jamison, 2012). As an example of its use in clinical research, Trull and colleagues (2008) used EMA to examine affective instability in groups of individuals diagnosed with BPD or a depressive disorder. Participants were given electronic diaries for 30 days and were randomly prompted to report their mood up to six times a day. The authors found that while the BPD and depressive disorders groups did not differ in mean levels of positive and negative affect, but did differ in degree of variability over time.

EMA may represent a particularly good approach to data collection in trauma research because prospective studies suggest that retrospective reports of traumatic events are inconsistent over time (Engelhard, van den Hout, & McNally, 2008; Lalande & Bonanno, 2011). To date, only a few published studies have used EMA to examine research questions about PTSD. Possemato and colleagues (2012) used EMA methods to monitor temporal fluctuations in PTSD symptoms and alcohol consumption. Participants were veterans who screened positive for hazardous drinking behaviors and sub-threshold PTSD symptoms on self-report measures. Each participant was provided with a cell phone with interactive voice response technology, which allowed researchers to randomly prompt participants via automated phone calls to complete pre-recorded assessments. Participants were randomly called four times throughout the day for 28
days. Although hypotheses or results related to PTSD symptoms or alcohol consumption were not included in the published study, information about compliance rates and participant reactivity were reported. In total, participants responded to 86% of the random prompts, and several variables, including lower avoidance symptoms, full-time employment and perceived benefit, predicted higher compliance rates. Furthermore, a significant decrease in PTSD symptoms was observed throughout the course of assessment symptoms (Possemato, Kaier, Wade, Lantinga, Maisto & Ouimette, 2012).

Pfaltz and colleagues (2010) used EMA employing electronic diaries to monitor the instability of physical anxiety symptoms in individuals with panic disorder, PTSD or no reported psychiatric diagnosis. After an initial meeting with the researchers, participants were given electronic diaries and asked to complete questionnaires on those devices at fixed times (9 AM, 12 PM, 3 PM, 6 PM, 9 PM) for seven days. All groups had high compliance rates, as participants completed, on average, 94% of the scheduled assessments. Results of this study indicated that individuals with PTSD and panic disorder experienced fluctuating physical anxiety symptoms compared to a control group. The authors speculated that their findings regarding physical anxiety symptoms could be attributable to recurrent panic attacks in participants with panic disorder and frequently occurring but highly variable re-experiencing symptoms in participants with PTSD (Pfaltz, Michael, Grossman, Malgraf, & Wilhelm, 2010).

**Hypotheses**

At present, there is an inadequate supply of research that examines the temporal patterns of PTSD symptoms over short periods of time. While many theoretical models of PTSD specify
how symptoms might change over time, longitudinal designs with only a few time points spanning several months or years may not adequately enable a researcher detect cyclical patterns occurring within a day or over several days. The current study advances past research by measuring PTSD symptoms several times per day for a two week period. Based on the research and theories reviewed thus far, three research questions were developed for the current study:

**Acceptability and Feasibility of the Protocol.** First, the possible effect of participating in an EMA protocol on subjective distress and overall PTSD symptom severity was evaluated through the use of paper and pencil questionnaires. Based on the results of Possemato and colleagues (2012), it was hypothesized that participants would experience minimal increases in subjective distress based on responses to a qualitative exit survey. Furthermore, it was expected that participants would experience a slight reduction in PTSD symptom severity, as assessed by self-report before and after participation in an EMA protocol. While previous studies have seen high response rates for EMA protocols (86-92%: Pfitz et al., 2010; Possemato et al., 2012), it was anticipated that response rates in the current study would be lower because electronic surveys were administered on a random interval sampling schedule compared to schedules with a fixed interval.

**Hypothesis 1.** A common theme across cognitive models of PTSD is that symptoms of the disorder represent an individual’s attempts to reconcile significant features of the traumatic event with previously held beliefs about the self, world or future. More specifically, Horowitz (2001) suggests that individuals alternate between periods of re-experiencing and avoidance/numbing symptoms. These fluctuating processes may represent an individual’s
individuals attempt to slowly integrate the meaning of the traumatic event into previously held beliefs about the self and the world. In contrast, cognitive models posit that hyperarousal symptoms represent the chronic activation of the traumatic memory. As an individual reconciles the differences between the traumatic event and previously held beliefs, hyperarousal symptoms are expected to decrease over time. If successful assimilation processes are present, one would expect downward trends for the severity of re-experiencing and avoidance symptoms as well.

It was hypothesized that significant downward trends would be present for all three symptom clusters over the two week assessment period. Furthermore, it was hypothesized that cyclical patterns, representing an individual alternating between assimilation and avoidance, would be present for the re-experiencing and avoidance symptom clusters but not the hyperarousal symptom clusters. Three individuals with high response rates who were not currently receiving treatment were included in time-series analyses that tested these hypotheses.

**Hypothesis 2.** Although a large body of research suggests that various emotions play an important role in the development and maintenance of PTSD symptoms, very few research studies, if any, have examined how trauma-related emotions change with PTSD symptoms over time. This project will be one of the first to examine the short-term temporal dynamics between trauma-related negative emotions and PTSD symptoms. It was hypothesized that current fear, shame, guilt and anger would be associated with concurrent changes in PTSD symptom severity. All participants who completed the study were included in the analyses for this hypothesis.

**Exploratory Analyses.** A growing body of research suggests that a variety of cyclic, oscillating, or self-regulative psychological processes, both normal and psychopathological,
exhibit properties of chaotic dynamics, including sensitivity to initial conditions, self-similarity, and unpredictability. Processes that have been studied include normal affect (Schuldberg & Gottlieb, 2002), mood fluctuations in bipolar disorder (Gottschalk & Whybrow, 1995), course in schizophrenia (Tschacher & Hashimoto, 1997), and other phenomena. To explore possible chaotic dynamics in temporal fluctuations in PTSD symptoms, time-series data from participants included in analyses for Hypothesis 1 were examined using nonlinear data-analytic tools.

Chapter 2: Method

Participants

Participants were recruited from an introductory to psychology participant pool at a large university located in the Mountain West. On separate mass screening days, participants completed a trauma event history checklist as well as a PTSD symptom checklist to determine initial eligibility for the study. The Trauma History Screen (THS) and PTSD Checklist – Civilian (PCL-C) were used to assess trauma history and symptom severity, respectively. Participants who endorsed a traumatic event on the THS and scored above a 44 on the PCL-C were contacted to participate in the study. Participants received course credit for their participation in the study, and they were also compensated by inclusion in a raffle to win a $25 gift card.

Thirty-three participants completed the study with recorded data. Four other participants took part in the study but were unable to complete the protocol due to various technological problems, including problems charging the device ($n = 1$), a broken device ($n= 1$) and data not recorded ($n= 2$). Information about the three participants chosen for the time-series analyses will be reported with the results for those analyses.
On the THS, participants first identified whether a particular traumatic event happened to them, and then indicated the number of times that the event occurred. Participants reported experiencing an average of 14.56 (SD = 20.12) potentially traumatic events, ranging from 1-114 events reported for each participant. After listing these events, participants were then asked to complete a box of more detailed information for each event that they considered to be “really emotionally bothersome.” Participants reported experiencing an average of 2.78 (SD = 1.31) events that were considered to be emotionally bothersome. The most commonly reported events were: sudden loss of a loved one or friend (n = 19); a really bad car, boat, train, or airplane accident (n = 10); some other sudden event that made [the person] feel very scared, helpless or horrified (n = 10); hit or kicked hard enough to injure, as a child (n = 9); and suddenly abandoned by spouse, partner, parent, or family (n = 9). See Table 2 for more results regarding the frequency of emotionally bothersome events.

Measures

**Demographic Survey Form.** A demographic form was included to collect relevant demographic information. Demographic information in the form included age, gender, education year, ethnicity, marital/partner status, and sexual orientation. Participants also were asked whether they were currently receiving any form of psychological treatment and the length of their treatment in weeks.

**PTSD Checklist – Civilian.** The PTSD Checklist - Civilian (PCL-C; Blanchard, Jones-Alexander, Buckley & Forneris, 1996) is a self-report inventory of PTSD symptoms. The
questionnaire contains 17 items assessing PTSD symptoms from the DSM-IV-TR. Each symptom is rated on a Likert scale from 1, “not at all” to 5, “extremely.” Scores on the PCL-C range from 17 to 85. Past research has found scores on the PCL-C to demonstrate adequate test-retest reliability ($r = .87$), internal consistency (Cronbach’s $\alpha = .91$), and clinical utility in college students (Adkins et al., 2008). Only participants who initially scored above a 44 (in addition to endorsing an event on the THS) were contacted to participate because this score has been recommended as a clinical cut-off for individuals who are likely to have PTSD (Blanchard et al., 1996).

In addition to being used as an initial screening measure, the PCL-C was also used to examine how participants’ PTSD symptoms were affected throughout participation in the study. The PCL-C was administered at three time points throughout the study: 1) at the initial screening; 2) the day before beginning the EMA protocol; 3) within a week of completing the EMA protocol.

Finally, the PCL-C was adapted to electronic form and included as a measurement of PTSD symptom severity within the EMA protocol. The first 17 items during each assessment correspond directly the items of the PCL-C, although slight changes were made to the measure so that participants could complete the questions on a smart phone device. Furthermore, symptom cluster scores were calculated during each assessment by adding the scores from the items for each symptom cluster and then dividing that number by the total number of items within the cluster. Reexperiencing symptoms correspond to items 1-5, avoidance/numbing symptoms to items 6-12, and hyperarousal symptoms to items 13-17.
Trauma History Screen. The Trauma History Screen (THS; Carlson, Smith, Palmieri, Dalenberg, Ruzek, Kimerling, Burling, & Spain, 2011) is a brief measure of exposure to highly stressful events that are usually associated with severe posttraumatic distress. In the first section of the measure, participants are asked to identify whether or not they have experienced 14 events that may lead to the development of PTSD. Participants were asked if the event “really bothered [them] emotionally.” If participants affirmatively answered the previous question, they were then asked to provide more detailed information related to the severity of the event. In an initial validation study, the THS demonstrated good temporal stability over periods of one week, two weeks and two months. In the current study, the THS was used to screen participants for past criterion A events.

Design

A prospective design using EMA data gathering techniques was used to collect data related to the proposed hypotheses. The purpose of this design was to capture and summarize the fluctuation of PTSD symptoms throughout the day for each participant. In order to accomplish this task, data were collected based on a stratified sampling schedule where participants were randomly prompted within six distinct, two hour time blocks from 10 AM to 10 PM. Data collection lasted for two weeks. The Android application used in this project was developed locally by Dr. Allen D. Szalda-Petree (See Figure 1 for screen shots of the application).

Procedure

Participants were recruited for the study using a mass screening day for introduction to psychology students. During each screening session, potential participants completed a packet of
measures, including informed consent, a demographic questionnaire, THS and PCL-C. At the end of the packet participants were asked to provide their contact information (email address and telephone number) if they would like to participate in the full study. Participants who included their contact information and met the inclusion criteria of the study were contacted by the PI to schedule an initial meeting.

At each initial meeting, informed consent was provided and the purpose of the study was thoroughly discussed with the participants. Important issues, such as battery life and participant compliance, were reviewed. Participants were also reassured that they could discontinue participation at any time without any penalty. At this point, participants were asked to complete the Posttraumatic Cognitions Inventory, Trauma Emotion Questionnaire and PCL-C. After completing these questionnaires, participants scheduled a final meeting with the PI to return the device, and complete some final questionnaires. Questions from the participants were addressed and resources/referrals were provided to participants for treatment resources within the community.

The first prompt to complete a survey occurred between 10 AM and 12 PM on the morning after the first scheduled meeting with the PI. Participants were notified of each assessment by an easily audible ringing noise and vibration of the device. Participants also had the option to silence the device so that they were only notified through vibration of the device; however, the volume automatically reset following a completed or missed prompt. If a participant did not respond to the prompt within 15 seconds, the device skipped the current scheduled assessment, and another prompt did not occur until the next time block. When
responding to a prompt, participants were given the choice to immediately begin the assessment or delay the assessment for 10 minutes. Participants were allowed to delay the survey twice, and they were forced either to enter the survey or to skip the assessment on the third prompt.

After being prompted and entering the survey on the Android device, participants were asked to complete 22 items that assessed current PTSD symptoms and trauma-related negative emotions (see Appendix A for list of items). The first 17 items corresponded to the items from the PCL, and participants were given the instructions to “please indicate how much you have been bothered by the following problems since your last assessment.” Reexperiencing symptoms correspond to items 1-5, avoidance/numbing symptoms to items 6-12, and hyperarousal symptoms to items 13-17. The final five items asked participants to rate current emotions (fear, anger, guilt, shame and sadness) related to the traumatic event. Each question was phrased as “how [insert emotion] are you feeling about the event.” Participants rated all items on a 5-point Likert scale ranging from “0, not at all” to “4, extremely.” The items were presented in the same order on every survey.

After the two week assessment period ended, the device thanked the participant for their involvement in the study and reminded participants to contact the principle investigator if they forgot their scheduled final meeting. Upon arriving for the final meeting, participants completed a PCL-C and exit interview questionnaire (see Appendix B). After completion of these forms, questions from the participant were answered, and participants were thanked for their involvement.

**Analytic Strategy**
Acceptability and Possible Impact of EMA Protocol. PCL-C scores over time and participant exit questionnaires for all participants who completed the EMA protocol were examined to evaluate the acceptability of using an EMA protocol with a trauma-exposed, PTSD symptomatic sample. A repeated-measures ANOVA with a Greenhouse-Geisser correction was used to evaluate the impact of participating in an EMA protocol on PTSD symptom severity. Time of administration (initial screening, day before EMA protocol, week after EMA protocol) was the independent variable in this analysis, and PCL-C scores were included as the dependent variable. Planned comparisons were also tested to examine whether PCL scores significantly differed between time 1 (initial screening) and time 2 (day before EMA protocol) and between time 2 and time 3 (week after EMA protocol), and a measure of effect size, Cohen’s $d$, was computed to evaluate the magnitude of the difference between groups. While the repeated-measures ANOVA broadly examined whether PTSD symptoms changed throughout the study, planned comparisons clarified whether or not these changes resulted from participation in the EMA protocol. If a non-significant change was observed from time 1 to time 2, but a significant difference in PCL-C scores was present from time 2 to time 3, it would provide preliminary evidence, in addition to the actual EMA data, that participating in the EMA protocol affected PTSD symptom trajectory.

In addition to the examination of change in PCL-C scores, common themes from participant exit questionnaire are presented to provide participants’ subjective reactions to completing the EMA protocol. These questionnaires specifically asked participants if any logistical problems were encountered during participation, how participation affected them
generally, and how it impacted their PTSD symptoms specifically. Common themes in each category were summarized, and example responses for each theme are discussed.

**Hypothesis 1.** The hypotheses that significant trends and cyclical patterns would be present for PTSD symptom clusters were evaluated in three participants using the recommendations and analytic strategies described by Warner (1996) and applied to each PTSD symptom cluster. Individual cases were chosen for the analyses, as opposed to the entire sample, because it was not anticipated that participants would have similarly spaced cycles, making aggregate analyses difficult. Furthermore, a large number of participants were not appropriate for these analyses because of their low response rates. The three participants were chosen because their high response rate (over 90%). was appropriate for the time-series analyses, and it was anticipated that their high number of responses would provide clearer depictions of time-related changes. Also, participants were selected who did not report receiving any form of psychological intervention following their traumatic event, so their symptom change would provide a better representation of how PTSD symptoms fluctuate naturally over time. Although this may represent a biased subsample, it was anticipated that the results of these cases would provide preliminary information for future research.

Based on the recommendations by Warner (1996), the following steps were taken to examine whether trends were present for all symptom clusters and cyclical patterns were present for reexperiencing and avoidance symptoms. First, means and variances were calculated for scores on each symptom cluster. Time-series graphs for each symptom cluster were plotted to screen for serious problems in the data or violations of assumptions. Time-series graphs were
then visually inspected to discern patterns in the data. Next, an autocorrelation function (ACF) was calculated to examine the correlation between two lagged time points of a time-series and to determine the presence of serial correlation within the dataset. For Hypothesis 1a, OLS regressions were used to examine the presence of linear, quadratic or cubic trends in the data and to determine the amount of variability explained by the identified trend. The identified trend was then removed by saving the residuals from the OLS regression analysis. Of note, parameters calculated for the regression analyses are not reported due to biased estimates resulting from the likely high autocorrelation among observations of PTSD symptom severity. A second ACF was conducted to determine if serial correlation was still present in the time-series. For Hypothesis 1b, spectral analyses were then conducted if the ACF plot indicated the presence of serial correlation after removal of the trend. These analyses examined the amount of variance explained by cycles present in the data. Because no predictions were made regarding specific features of the patterns (i.e. periods, amplitudes or phases), spectral analyses were conducted to determine what types of patterns might exist in the data.

**Hypothesis 2.** In order to test the hypothesis that trauma-related emotions would be positively correlated with PTSD symptom severity, a multi-level model was constructed with PTSD symptom severity as the outcome variable and trauma-related emotions entered as level-1 predictor variables. These analyses were conducted in IBM SPSS Statistics version 21 using the MIXED data analysis procedure, and the data analytic strategy was based on the recommendations of Singer (2003). Means and standard deviations were computed for all relevant variables. Based on suggestions by Nezlek (2001), descriptive statistics were calculated
for all measures using a “totally unconditional model” in which no predictor variables were included at any level of the model. This model provided between-days and within-persons estimates of means and variances. Secondly, an unconditional growth model, one in which only time was included as a level-one predictor variable, was conducted to evaluate whether individuals differ from each other in a linear fashion. Finally, a multilevel model with trauma-related negative emotions, including fear, anger, guilt and shame, was constructed to test the hypothesis that trauma-related emotions will be significantly associated PTSD symptom severity. Because the relationship between trauma-related emotions and PTSD symptom scores was not expected to differ across individuals, the slopes of these relationships were treated as fixed effects (See Figure 2 for a depiction of the equations defining the model).

**Exploratory analyses.** Additional analyses were conducted to explore for the presence of chaos in the symptom clusters for the three participants included in the analyses for Hypothesis 1. After removal of trends in the data for each of the participants individually, nonlinear indices were calculated for each PTSD symptom cluster using the Chaos Data Analyzer Professional Version 2.1 software package (Sprott & Rowlands, 1995). No predictions were made regarding the presence of chaos in the data.

**Chapter 3: Results**

**Acceptability of the Protocol and Changes in Participant Distress**

**PCL-C Scores over Time.** To understand how involvement in the study was associated with participant distress and overall PTSD symptom severity, PCL-C total scores from three separate time points in the study were compared. The PCL-C scores were collected at initial
screening (time 1), before completing the EMA protocol (time 2) and after completing the EMA protocol (time 3). To examine the association between participation in the EMA protocol and changes in PTSD symptom severity, a repeated measures ANOVA with a Greenhouse-Geisser correction was conducted with time (initial screening, first interview, second interview) as the independent variable and PTSD symptom severity as the dependent variable (PCL-C total score). Results of this analysis indicated that mean PCL-C scores differed significantly across time points ($p < 0.001$). Post hoc comparisons were computed using the Bonferroni correction. With three comparisons the cutoff for statistical significance using this correction is $p = .017$. Results of the contrasts revealed a slight reduction in PCL-C scores from the time 1 (initial screening: $M = 57.16$, $SD = 10.06$) to time 2 (interview before EMA protocol: $M = 55.72$, $SD = 10.87$), representing a small effect (Kirk, 1996: $d = .14$) which was not statistically significant, $t (31) = 1.043$, $p = .305$. However, PCL-C scores at time 3 (PCL-C after completing the EMA protocol) decreased to 46.97 ($SD = 13.19$), which was significantly different than the baseline screening ($t (31) = 4.205$, $p < 0.001$) and from the first interview ($t (31) = 4.388$, $p < 0.0001$). The decrease in means between the time 2 and time 3 represented a medium-large effect ($d = .72$). These results suggest that while there was only a slight difference in PTSD symptom severity at screening day and the first interview, which can be viewed as a pre-EMA baseline period, there was a large difference in symptom severity between the pre-EMA and post-EMA interview.

Exit Questionnaires. Responses to the exit questionnaires provided not only general feedback about the study, but also subjective impressions regarding changes to behaviors and PTSD symptoms. Surprisingly, most participants found that it was not difficult to participate in
the study, as 17 participants indicated that it was either “not difficult” or “easy” to complete the study. Some typical problems associated with participation were difficulties keeping the device charged, prompts occurring during class or work and increased experiencing of negative affect associated with being repeatedly reminded of a traumatic event.

When asked on the exit questionnaire how participation affected PTSD symptom severity throughout the study, the most common response was that participation increased awareness of PTSD symptoms and, in a small number of cases, intensified and worsened those symptoms. The following responses highlight those themes:

“It made me more aware of my symptoms. Sometimes, it would bring up symptoms when I hadn't been thinking about the accident.”

“It made it more prevalent that my experiences truly were affected and I was still dealing with the event.”

“It amped them up. They increased. It honestly made my life worse in many ways”

While this information may contrast with the quantitative data provided above, it is important to note that participants are reporting how they felt during the assessment period. It is unclear from some of the responses whether symptoms remained worse or more intense after the data collection ended, which is when the final PCL-C was completed. In some cases, participants
articulated that becoming more aware of the event and experiencing emotions associated with it actually reduced their PTSD symptoms. For example, below are three responses that highlight the benefits of participation:

“Sometimes it did cause more memories to come up but answering the questions helped to settle that or those emotions.”

“It made it easier to deal with because every time there was a survey I had to face my feelings.”

“I think it forced me to confront some emotions that I suppressed. Now I feel like I can think/talk about the experience more easily.”

**Hypothesis 1**

Three female participants were included in these analyses because their high response rate (over 90%) was appropriate for the time-series analyses, and all three denied current psychological treatment. The hypotheses that significant trends and cyclical patterns would exist for PTSD symptom clusters was tested using spectral analyses based on the work of Warner (1996). These analyses examined the amount of variance explained by trends and cycles for three separate individual cases. Analyses were conducted separately for each of the PTSD symptom clusters (re-experiencing, avoidance, hyperarousal) for the three participants selected, and it was anticipated that downward trends would exist for all symptom clusters, while temporal cycles
assessed via spectral analyses would be present for re-experiencing and avoidance/numbing symptom clusters.

**Participant A.** Participant A was a 22-year-old European American, heterosexual woman. On the THS she reported experiencing three previous traumatic events, including being attacked by a gun, knife or weapon, being forced to have sexual contact (as an adult) and experiencing the sudden death of family member or close friend. She indicated that her worst event was being raped at 17 years of age, and she denied currently receiving psychological treatment. Her PCL-C scores at time 1 (screening day), time 2 (pre-EMA interview) and time 3 (post-EMA interview) were 69, 66 and 41, respectively.

Participant A completed surveys for 78 out of 84 prompts (92.86%). The mean reexperiencing score for the series was $M = 1.46$ (SD = 0.57). An initial visual inspection of the time-series plot suggested the presence of curvilinear trend, although no consistent temporal cycles appeared to be present in the time-series data. Reexperiencing symptom severity initially decreased over the first 30 measurements, followed by a slight increase in severity over the next 30 measurements, and another decrease in severity over the remaining ones (see Figure 3.1).

A lagged autocorrelation function (ACF) was calculated to determine if any temporal patterns existed within the time series. The ACF calculates the correlation between two lagged time points of a time-series and indicates the presence of serial correlation within the dataset (Warner, 1996). The ACF indicated the presence of serial correlation for reexperiencing symptoms as lags were significantly different from zero for lags 1-21. Examination of the ACF
plot (see Figure 3.2 for an illustration of an ACF plot) suggests a trend within the data as closer lags are highly correlated and the strength of this correlation decreases gradually over time.

To account for this serial correlation, three statistical models (linear, quadratic and cubic trend models) were evaluated to determine which model best fit the data. A cubic trend model was selected because it explained 73% of the total variance of the time-series \( R^2 = 0.73 \): see Figure 3.3 for a graphical depiction of the model fitted to the data) compared to 17% for the linear trend model and 30% for the quadratic trend model. Of note, parameters are not reported for the regression analysis due to likely biased estimates resulting from high autocorrelation among observations of PTSD symptom severity. The cubic trend was removed from the time-series by saving the residuals from the regression analysis, and a second ACF was computed to determine if any serial correlation remained. Once again, the ACF indicated the presence of serial correlation. To account for any possible periodic components to the data, a spectral analysis was conducted after the removal of the cubic trend. The spectral analysis did not indicate the presence of any clear temporal cycles for re-experiencing symptoms (see Figure 3.4)

The mean avoidance/numbing score for the series was \( M = 1.01 \) (SD = 0.30). An initial ACF indicated the possibility of serial correlation, as a majority of lags 1-21 were significantly different from zero. Similar to the re-experiencing symptoms, a cubic trend best fit the data (see Figure 3.5: \( R^2 = 0.68 \)) followed by a quadratic trend model \( R^2 = 0.40 \) and a linear trend model \( R^2 = 0.28 \). An ACF was conducted to determine the presence of serial correlation after removing the cubic trend in the data. The ACF indicated the presence of serial correlation. To account for any possible periodic components to the data, a spectral analysis was conducted after
the removal of the cubic trend. The spectral analysis did not indicate the presence of any clear temporal cycles for avoidance symptoms (see Figure 3.6), so a spectral analysis was not conducted.

The mean hyperarousal score for the series was $M = 2.26$ (SD = 0.79). The ACF indicated the presence of serial correlation through 21 lagged time points, and a cubic model best fit the data, explaining 66% of the total variance (see Figure 3.7). The ACF did not indicate any significant serial correlation after removing the cubic trend.

**Participant B (2231).** Participant B was an 18-year-old European American, heterosexual woman. On the THS she reported experiencing one previous traumatic event, which was a really bad accident. She specified that this event was an “[all-terrain vehicle] wreck and life flight” at age 17, and she denied receiving any form of psychological treatment. Her PCL-C scores at time 1 (initial screening), time 2 (pre-EMA interview) and time 3 (post-EMA interview) were 56, 56 and 43, respectively.

Participant B completed surveys for 82 out of 84 prompts (97.62%). Her mean reexperiencing score was $M = 0.13$ (SD = 0.33). The mean avoidance/numbing score was $M = 0.28$ (SD = 0.26). Visual inspection of the plotted time-series for reexperiencing and avoidance symptoms suggested minimal variance for both variables, as Participant B reported near zero symptom scores on most measurements (Figure 4.1). However, despite the near zero scores on each survey, there were also sporadic elevations in symptom severity at several time points throughout the data collection period. ACF plots were constructed and suggested that significant serial correlation was not present in either time-series, so no further analyses were conducted.
The mean hyperarousal score was $M = 1.78$ (SD = 0.36). An ACF did indicate the presence of serial correlation, and a quadratic trend best fit the data, explaining 26% of the total variance (Figure 4.2: $R^2 = 0.26$).

**Participant C (2271).** Participant C was 22-year-old Asian American, heterosexual woman. On the THS she reported experiencing three previous traumatic experiences, including being attacked with a knife, gun or weapon, a sudden move or loss of home and possession, and being suddenly abandoned by spouse, partner, parent, or family. She indicated that her worst event was “sex without consent” when she was 17-years-old. Of note, she did not include this event on the THS that she completed during screening day even though this event occurred before her initial screening. She denied receiving any current psychological treatment. Her PCL-C scores at time 1 (initial screening), time 2 (pre-EMA interview) and time 3 (post-EMA interview) were 74, 72 and 32, respectively.

Participant C completed surveys for 82 out of 84 prompts (97.61%). The mean reexperiencing score for the series was $M = 0.30$ (SD = 0.55), which suggests very minimal levels of reexperiencing symptoms throughout the measurement period. Visual inspection of the time-series plot suggested the presence of curvilinear trend, although no temporal cycles appeared to be present within the data (Figure 5.1). Moderate levels of reexperiencing symptoms were initially present followed by a rapid reduction over the first 30 measurements (approximately four days), and remained nearly absent throughout the rest of the series except for a slight increase around measurement 70.
The ACF plot indicated the presence of serial correlation for reexperiencing symptoms as lags were significantly different from zero for lags 1-21. To account for this serial correlation, three statistical models (linear, quadratic and cubic trend models) were evaluated to determine which model best fit the data. A cubic trend model was selected because it explained 45% of the total variance of the time-series ($R^2 = 0.45$; see Figure 5.2 for a graphical depiction of the model fitted to the data) compared to 31% for the linear trend model and 42% for the quadratic trend model. The cubic trend was removed from the time-series by saving the residuals from the regression analysis, and a second ACF indicated that no serial correlation remained after removing the cubic trend. A spectral analysis was not conducted because of the absence of serial correlation.

The mean avoidance/numbing score for the series was $M = 0.74$ (SD = 0.53), suggesting minimal avoidance symptoms reported over the course of the measurement period. A visual inspection of the time-series suggested the presence of a curvilinear trend, but did not indicate the presence of any temporal cycles (see Figure 5.1). Moderate levels of avoidance symptoms were initially present. Avoidance symptom severity decreased slowly over the first 50 measurements after which symptom severity started to increase slowly until the measurement period ended. An initial ACF indicated the possibility of serial correlation, as a majority of lags 1-21 were significantly different from zero. Both the quadratic and cubic trend models explained 21% of the total variance ($R^2 = 0.21$) followed by a linear model ($R^2 = 0.13$). After removal of the quadratic trend, an ACF indicated no significant serial correlation for the series, so there was no attempt to fit a seasonal model to the data.
The mean hyperarousal score for the series was $M = 1.01$ (SD = 0.50). A visual inspection of the time-series suggested the presence of a curvilinear trend, but did not indicate the presence of any periodic components (See Figure 5.4). Moderate levels of hyperarousal symptoms were initially present, which decreased rapidly over the first 24 measurements (approximately 3 days). An initial ACF indicated the possibility of serial correlation, as a majority of lags 1-21 were significantly different from zero. A cubic model best fit the data, explaining 37% of the total variance ($R^2 = 0.37$), followed by a quadratic model ($R^2 = 0.29$) and linear model ($R^2 = 0.12$).

**Hypothesis 2**

Thirty-three participants completed the EMA protocol. Two participants were excluded from data analysis because over 85% of their responses were equal to zero. Therefore, thirty-one participants are included in the current analyses. A multi-level model was constructed to test the hypothesis that trauma-related emotions would be positively correlated with PTSD symptom scores. The overall response rate was 67.5% with participants completing, on average, $M = 56.71$ surveys (SD = 18.64). The number of completed EMA surveys ranged from 13-81 surveys. The response rate in the current study is much lower than previous research using EMA data collection strategies (86-92%: Pfitz et al., 2010; Possemato et al., 2012). This finding, however, is not surprising given that electronic surveys were administered on a random-interval sampling schedule compared to the fixed scheduling at a fixed interval used in previous research.

First, an unconditional means model was conducted to describe and partition the outcome variation in PTSD symptom scores. Maximum likelihood was used as the estimation procedure
and was the estimation procedure used in all subsequent models. The grand mean for PTSD symptom score was $\gamma_{00} = 20.57$, and variance components suggested statistically significant variability at the between- and within-person levels ($\tau_{00} = 141.53$ and $\sigma^2 = 64.18$, respectively). This finding indicates that an average person’s PTSD symptom scores vary over time and differ from person to person. The intra-class coefficient was calculated to provide an index of the proportion of variability accounted for at the within- and between-person levels. The intra-class correlation coefficient was computed as $(141.53 / [141.53 + 64.18]) = .68$, which suggests that 68% of total PTSD symptom score variability occurred at the between-person level.

An unconditional growth model was conducted to evaluate whether individuals differ from each other in a linear fashion. Results from this analysis indicated that the mean PTSD symptom score at initial measurement was $\beta_{00} = 23.19$. The mean growth per measurement was $\beta_{10} = -.03$, which was not statistically significant, $t (28.52) = -1.67, p = 0.11$. This finding indicates that PTSD symptom scores do not change linearly over time, on average. The statistically significant variance components suggest that individuals do vary significantly from these averages, and while the average trajectory may be flat, individual trajectories are not.

In order to test the hypothesis that trauma-related emotions would significantly predict PTSD symptom scores, trauma-related emotions (fear, anger, shame and guilt) were added to the model as predictor variables. Because the relationship between trauma-related emotions and PTSD symptom scores was not expected to differ across individuals, the slopes of these relationships were treated as fixed effects. Results from this analysis indicate a substantial
positive relationship between all trauma-related negative emotions and PTSD symptoms scores at $p < .0001$ (see Tables 4 and 5 for additional information on these analyses).

**Exploratory Hypotheses.**

Phase space plots were conducted to examine the possible presence of chaos in the three participants included in the time-series analyses. These analyses used the residual data from the time-series analyses after the trends were removed. Results from the phase space plots are presented in Figure 7, along with estimates of two chaos parameters, the Correlation Dimension (Grassberger & Procaccia, 1983) and Largest Lyapunov exponent (Wolf, Swift, Swinney, & Vastano, 1985). Visual inspection of these plots and examination of the chaos indices is suggestive of moderate-dimension (4 or 5) chaotic variability in the symptom ratings. However, it is wise to heed Sprott’s warning regarding joining “the legions of others who have published false claims of chaos in experimental data” (Sprott, 2003, p. 235).

**Discussion**

The current study uses a time-series design with EMA data collection techniques to examine the temporal dynamics of and relationships between PTSD symptoms and trauma-related negative emotions. The use of a time-series design is somewhat unique in PTSD research and offers a complementary approach to more traditional nomothetic research designs. Furthermore, the combination of idiographic and nomothetic research methods used in the current study takes advantage of the specificity of the former approach while still offering the generalizability of the latter. The primary finding of the current study is that PTSD symptoms are dynamic in nature; however, understanding how and why PTSD symptoms change over time has
proven to be a challenging endeavor. The results that completion of an EMA protocol might actually influence PTSD symptom trajectory will be introduced initially. Next, the results from the time-series analyses will be discussed in an attempt to understand how PTSD symptoms changed over time for three participants. Finally, the relationship between trauma-related emotions and PTSD symptoms will be considered in light of recent changes to the DSM-5 and implications for treatment.

**Impact of EMA Protocol on PTSD Symptom Severity**

Somewhat surprisingly, data from the present study indicates that participation in an EMA research protocol was associated with a reduction in PTSD symptom severity. This finding is based on significant pre to post reductions in PCL-C scores following completion of the EMA protocol. Given that this reduction followed a period of stable symptoms when no monitoring occurred, it provides initial evidence for a causal relationship between EMA participation and PTSD symptom reduction.

These results are consistent with growing body of literature suggesting that self-monitoring of PTSD symptoms can lead to a meaningful decrease in PTSD symptom severity, particularly in individuals with less severe symptoms. For example, previous research using EMA or other intensive longitudinal research designs have found that subjects, including both veterans and civilians, experience low levels of distress during participation (Pederson, Kaysen, Lindgren, Blayney, & Simpson, 2014) and in some cases may actually experience symptom reduction as the result of participation (Ehlers et al., 2003; Possemato et al., 2012; Reynolds & Tarrier, 1996).
A small but consistent body of literature suggests that self-monitoring of PTSD symptoms, specifically intrusive symptoms, can result in PTSD symptom improvement. For example, Reynolds and Tarrier (1996) presented a series of cases that examined the effect of monitoring the frequency and duration of intrusive symptoms in six patients diagnosed with PTSD. More specifically, patients were instructed to record each occurrence of an intrusive symptom (e.g., a flashback) in a diary for a three month period of time. At the conclusion of the monitoring period, four participants experienced full remission of PTSD symptoms, one participant experienced partial remission of symptoms, and one patient still met full diagnostic criteria.

In a follow-up study with a larger sample, Tarrier and colleagues (1999) examined the impact of self-monitoring of intrusive symptoms in a mixed-trauma sample of 116 patients referred from local health clinics. Before being randomized into more intensive treatment as a part of a larger outcome study, participants were asked to monitor intrusive symptoms by completing a diary at the end of each day that required them to rate the frequency and intensity of bad dreams, unwanted thoughts, flashbacks and trauma-related images. At the end of the self-monitoring phase 12 patients no longer met criteria for a PTSD diagnosis and follow-up assessments at 3- and 12-months suggested that individuals who improved through self-monitoring continued to maintain those therapeutic gains. The authors concluded that individuals with less severe PTSD symptom profiles are most likely to benefit from a self-monitoring intervention (Tarrier et al., 1999).
Ehlers and colleagues (2003) compared the effectiveness of self-monitoring, self-help through psychoeducational materials, and cognitive therapy for victims of motor vehicle accidents. Individuals who displayed significant levels of PTSD symptoms in the months following their motor vehicle accident were contacted to participate in the study ($N = 100$). Prior to being randomized to a specific treatment type, all participants completed a three week self-monitoring phase, which resulted in complete remission of PTSD symptoms in 12% of participants. While cognitive therapy resulted in the greatest level of PTSD symptom improvement, the self-monitoring group also experienced significant reduction in PTSD symptom severity and actually outperformed the self-help group.

The results of this body of literature should not be surprising in light of theories about recovery from PTSD. Foa and Kozak (1986) argue that most theoretical approaches to psychotherapy support the notion that avoidance of fear is a primary contributor to the development and maintenance of psychopathology. Emotional processing is a form of exposure shown to reduce pathological fear. By definition, it involves both evocation of the feared stimuli and information (i.e., cognitive and affective experiences) that is incompatible with fear response (Foa & Kozak, 1986). Prolonged Exposure and Cognitive Processing Therapy, both evidence-based treatments for PTSD, describe avoidance as a primary mechanism through which PTSD develops, and use exposure techniques to reduce PTSD symptom severity by promoting emotional expression. In Prolonged Exposure, two forms of exposure are used to treat PTSD symptoms, situational exposures and imaginal exposures, which involve confronting feared situations and the trauma memory, respectively. In Cognitive Processing Therapy, written
accounts of the trauma that are assigned as homework serve to disrupt an individual’s avoidance of thinking about the event.

Consistent with these treatments, a potential explanation for the reduction observed in PTSD symptom severity following EMA participation or other self-monitoring studies is that it serves as a form of exposure by reminding participants repeatedly throughout the day about their traumatic experience. Furthermore, by asking participants to rate negative emotions related to the event, the current EMA protocol may provide participants the opportunity to engage in emotional processing. Therefore, the reduction observed in the current study might result from the same emotional processing or habituation processes that occur during formal treatment, albeit in a lighter format.

There is some evidence to suggest that EMA protocols, in research studies examining psychological disorders other than PTSD, have facilitated emotional processing. For example, Kauer and colleagues (2012) examined the relationship between the self-monitoring of depression symptoms via mobile application and emotional self-awareness in a sample of adolescents and young adults. The sample was required to report their experience of depression symptoms four times per day for a period of at least two weeks. Their results indicated that objective self-monitoring of symptoms increased self-reported emotional self-awareness. Additionally, these gains mediated the relationship between the SM intervention and subsequent reductions in depression symptoms. Consistent with this finding, it is possible that EMA protocols induced basic emotional processing related to monitored PTSD symptoms, resulting in an increased tolerance of such symptoms in the present study. Certainly, some of the comments
from participants in the exit questionnaires support this interpretation, as most individuals noted increased awareness of PTSD symptoms and negative emotions resulting from their participation in the study. In more specific cases, participants actually communicated that confronting the event and trauma-related emotions made it easier to cope with them.

Aside from the implications associated with the decrease of PTSD symptoms in participants in this study, it is an equally important finding that participants’ PTSD symptoms, on average, did not increase as a result of EMA participation. This is a particularly salient result given the finding that researchers and members of Human Subject Review boards regularly worry that asking participants about exposure to trauma and related symptoms will cause harm (Finkelhor, Vanderminden, Turner, Hamby, & Shattuck, 2014). Despite this concern, this study adds to the extant literature suggesting that participation in EMA protocols that assess PTSD symptoms are not harmful for most participants (Pederson, et al., 2014; Possemato et al., 2012). However, an important caveat is that one participant did report a worsening of symptoms that occurred throughout her participation that was not only communicated in response to the exit questionnaire, but also observed as a 20 point increase in PCL-C scores from Time 2 to Time 3. Certainly, understanding factors that may predict an aversive response to participation is an important research area moving forward.

PTSD Symptom Change as a Function of Time
First, it was predicted that small downward trends would exist for all PTSD symptom clusters. Second, it was predicted that cyclical patterns would be present for both re-experiencing and avoidance symptom clusters. If supported, these hypotheses would have provided initial validation for the temporal sequence of PTSD symptoms noted in the Stress Responses Syndromes model posited by Horowitz (2001). Horowitz argued that individuals alternate between periods of re-experiencing and avoidance/numbing symptoms. As previously discussed, this fluctuating process may represent an individual’s attempt to slowly integrate the meaning of the traumatic event into previously held beliefs about the self and the world. In contrast, hyperarousal symptoms should decrease slowly over time without the fluctuating patterns observed for the other symptom clusters. Because it is thought to be unlikely that cyclical patterns will be the same for all participants, time-series analyses were conducted for three participants to determine if any cyclical behaviors might become discernible.

The hypothesis that downward trends would be present for all three symptom clusters did receive some support. In the three participants included in these analyses, seven of nine symptom cluster scores displayed a curvilinear trend. While the fit of these models widely varied, most of the time-series could be described as having a deceleration of symptom severity over the first half of the assessment period. This decrease in symptom severity was then followed by a smaller change in the second half of the assessment period. Given the length of time since the index trauma for each participant, it was anticipated that only small trends would be present; however, the trends observed for PTSD symptoms clusters for all three participants were much greater than initially anticipated.
Participant A provides an excellent example of the surprising change in symptom severity over time. Prior to participation in the EMA portion of the study, Participant A reported relatively stable symptoms on her first two PCL-C questionnaires with scores of 68 and 64, respectively. These high scores were also reflected in her initial surveys completed during the EMA assessment period (see Figure 3.1.), as her hyperarousal symptom scores were at or near the ceiling for the first eight assessments. However, during the first four days of her assessment period (first 24 surveys), her PTSD symptoms improved rapidly, and she mostly appeared to maintain those gains throughout the rest of the assessment period.

The change in symptom severity for all three participants, along with the analyses using PCL-C scores already described, does call into question whether EMA actually obtains an ecologically valid assessment of PTSD symptoms. While EMA studies may provide rich information about how cognitive, behavioral and emotional processes change in real-time, PTSD may present a serious challenge for EMA given that a primary feature of the disorder is the intentional avoidance of trauma-related stimuli, thoughts and memories. As already discussed, repeated prompts to provide information about current PTSD symptoms may disrupt a participant’s normal avoidance strategies and lead to a recording of symptoms that is not ecologically valid. For example, questions on the survey require participants to think about how the traumatic event is currently affecting them, and then rate how they feel about it. From this perspective, EMA could actually be described as a form of “light exposure” that provides participants an opportunity to habituate to their trauma memory and PTSD symptoms.
An important caveat to these findings is related to the fact that there might actually be something different about participants who are highly compliant compared to participants who are not complaint. It is clear from viewing the growth plots of other participants (see Figure 6), as well as the MLM results, that participants, on average, did not experience a significant reduction in PTSD symptoms throughout the assessment period. In some cases, participants symptoms worsened throughout the duration of the study, and future research would do well to understand these varied responses among participants.

The hypothesis that cyclical patterns would exist for reexperiencing and avoidance symptoms was not supported in this study. This finding is based on not only visual inspection of the data from the three subjects, but also through the use of spectral analysis, a type of time-series analysis that allows for the identification of cyclical behavior in a dataset. There are several reasons that may help explain why these hypotheses were not supported with the present data. First, self-report data on PTSD symptoms may not be appropriate for detecting cycles that may exist. Instead, this type of analytic strategy may be better suited for understanding physiological changes in individuals diagnosed with PTSD. Also, it is possible that self-report data obtained through EMA techniques may be able to capture cycling of re-experiencing and avoidance symptoms, but the design of the study did not adequately sample PTSD symptoms. For example, the current sampling schedule, six times daily for two weeks, might not be able to capture longer cycles that are greater than the two week assessment period. Finally, rather than being controlled by internal processes that allow for the integration of the trauma memory, it is possible that change in PTSD symptoms is more contingent upon external reminders of the
traumatic event. It is likely that PTSD symptoms worsen immediately following a cued reminder of the trauma (e.g., a particular image or scent), and avoidant coping strategies follow these exposures. Therefore, attempting to explain the variability of PTSD symptoms solely using temporal factors, such as cycles, is unlikely to be successful as much of the variability in PTSD symptom severity that occurs over time may be due to events that occur independent of time. The intermittent elevations in re-experiencing and avoidance symptoms clusters for Participant B might provide an example where this explanation is applicable.

Exploratory analyses revealed that deterministic chaos might be present in some of the PTSD symptom clusters for the time-series cases. Generally, the presence of deterministic chaos suggests that while a behavior or process may initially appear highly unpredictable, this unpredictability is bounded within the constraints of a particular system. The deterministic chaos in PTSD symptoms might reflect homeostatic processes that maintain and regulate a certain level of symptom severity regardless of how much symptoms appear to fluctuate over time. Symptoms are likely to be bounded within particular constraints until an individual is successful in either integrating the trauma memory or regulating trauma-related emotions.

**Temporal Relationships between PTSD Symptoms and Negative Emotions**

It was predicted that trauma-related emotions would be positively correlated with PTSD symptoms. This hypothesis was rooted in theoretical models and empirical research suggesting that negative emotions are an important part of the clinical presentation, as they often co-occur with other PTSD symptoms and predict the development of PTSD symptoms following trauma exposure. As predicted, momentary ratings of trauma-related negative emotions were
significantly correlated with PTSD symptom severity. To the author’s knowledge, this study is the first attempt to evaluate the relationship between PTSD symptoms and trauma-related emotions using EMA as a data-collection strategy. Furthermore, the support for the hypotheses provides more evidence strengthening the role of negative emotions in the maintenance of PTSD symptoms, especially anger as it was the strongest predictor of PTSD symptoms.

A primary implication of the finding that negative emotions are not only present but also co-vary with PTSD symptoms is that it supports the utility of the inclusion of persistent negative emotions as a symptom addition to the DSM-5. While there is strong support for the role of negative emotions in the development and maintenance of the disorder, there is limited published literature on the relationship between negative emotions and PTSD symptoms over short periods of time. This study fills that gap in the literature by exploring the temporal dynamics of PTSD symptoms and trauma-related negative emotions using EMA in a trauma-exposed, PTSD symptomatic sample. Furthermore, the finding that anger was the best predictor of PTSD symptoms also supports the change in the DSM-5 of PTSD from an anxiety-based condition to a more general trauma and stressor-related disorder.

Two possible mechanisms might explain the observed relationship between negative emotions and PTSD symptoms. First, it is possible that experiencing a traumatic event, which elicits high levels of stress and negative emotions, may disrupt an individual’s ability to regulate negative emotions in the future. In support of this idea, Seligowski and colleagues (2015) conducted a meta-analysis to evaluate the strength of the relationship between emotion regulation and symptoms of posttraumatic stress. In total, 57 studies were reviewed that met the
inclusion criteria of cross-sectional research studies examining the relationship between posttraumatic stress symptoms and qualities of emotion regulation. Results of the analysis suggested a strong relationship between posttraumatic stress symptoms and emotion dysregulation \((r = 0.53)\). Furthermore, several “negative” emotion regulation strategies were also strongly related to symptom severity, including thought suppression \((r = 0.47)\) and experiential avoidance \((r = 0.40)\). One limitation of this review is that it does not provide evidence of the temporal sequence of these variables, and emotion regulation difficulties might actually precede exposure to traumatic stress and serve as an important risk factor.

Another mechanism through which negative emotions may develop is increased negative cognitions related to the self, others or world. This explanation is consistent with many of the theoretical models of PTSD already discussed. For example, the Stress Response Syndromes model presented by Horowitz (2001) describes the development of negative emotions following trauma as resulting from new meaning developed about oneself following a traumatic experience. For example, high levels of anger are likely to be present in an individual who interprets PTSD symptoms as an indication of vulnerability or weakness.

Recent research suggests a strong relationship between specific cognitions and negative emotions. For example, Beck and colleagues (2015) examined the relationship between specific cognitions and negative emotions in victims of interpersonal violence seeking treatment at a trauma-focused clinic. Self-report measures were given to assess negative emotions and cognitive beliefs. Results indicated that negative thoughts about the world were associated with
increased guilt, negative thoughts about the self were associated with increased shame and depression, and shame and guilt were correlated with high levels of self-blame.

The addition of persistent negative emotions to the PTSD diagnostic criteria has important implications for the treatment of the disorder. To date, cognitive-behavioral treatments have primarily characterized PTSD as a fear-based disorder resulting from disruptions to fear circuitry in the brain. For example, Prolonged Exposure, which is rooted in emotional processing theory and related to exposure and response prevention approaches, assumes that PTSD develops when an individual becomes conditioned to respond to neutral stimuli with a fear response (Foa & Kozak, 1986). From this perspective, avoidance symptoms develop because previously neutral stimuli now elicit fear through their association to the traumatic event. Prolonged Exposure attempts to treat these symptoms through exposure to previously feared stimuli, so that the individual learns new information that is incompatible with the fear response, namely that the situations, people and/or places are relatively safe.

While Prolonged Exposure is a recommended and highly efficacious treatment for PTSD, treatment dropout and non-response are important issues facing clinicians and researchers moving forward (Schottenbauer et al., 2008). Understanding the specific negative emotions experienced by at trauma survivor may provide information that enhances treatment outcomes by allowing a clinician to select a more appropriate intervention, as specific emotion or symptom presentations may be better suited for different types of trauma treatments. For example, preliminary research suggests that avoidance symptoms may be specific to high levels of trauma-related fear, as trauma-related guilt, shame, anger and disgust were not associated with the
presence of avoidance symptoms in a trauma-exposed sample (Dewey, Schuldberg, & Madathil, 2014). Given the importance of avoidance in its rationale and its reliance on disruptions to fear circuitry, Prolonged Exposure might then be best suited for individuals who present with primary symptoms of high levels of fear and avoidance. In contrast, individuals who present with predominant feelings of shame and thoughts of self-blame might be better suited for treatments, such as Cognitive Processing Therapy, that focus on treating negative cognitions related to the self that develop as the result of traumatic experiences and may help explain the presence of those emotions.

**Limitations and Future Directions**

A primary limitation of the study is its somewhat inclusive approach to recruitment, as a standardized assessment was not used to determine whether participants met full diagnostic criteria for PTSD. As such, conclusions that can be drawn regarding clinical populations from the current results must be tempered. It is worth noting, however, the severity of posttraumatic stress in this sample of students. Using the recommended cut-off score of 44 on the PCL-C for a diagnosis of PTSD (Blanchard et al., 1996), the present sample appears to meet this threshold ($M = 57.16$, $SD = 10.06$) and exhibit PTSD symptoms of moderate severity.

There are several notable limitations related to the time-series analyses included in this study. First, these analyses are idiographic in nature and the results are not generalizable to the larger population of individuals with PTSD. Second, the assessment schedule may have made it difficult to detect temporal patterns that are present in PTSD symptoms. Third, self-report data of psychological processes may not be ideal for detecting temporal patterns, and more objective
recordings of trauma-related phenomena may be more sensitive to these patterns. Fourth, because of the small number of assessments, there is a possibility of observing chaotic behavior that is in fact not actually present. Finally, it is likely that fluctuations in PTSD symptoms over time are better understood through environmental factors, and this type of data was not collected for the present study.

The model constructed to test the second hypothesis is limited to explaining only 32% of the total variability in PTSD symptoms because only level-1 or within-subject variables were included. Future research would benefit from a larger sample size and the inclusion of important between-subject predictor variables. Finally, the multi-level model evaluating the relationship between trauma-related emotions and PTSD symptoms does not permit a discussion of the directionality of the relationship between these variables. Future research examining the time-lagged relationship among these variables will help disentangle the direction of these relationships.

Moving forward, there are two clear directions for the role of EMA and other technology-based data collection strategies in PTSD and traumatic stress research. First, these intensive, technologically-based data gathering techniques can be used to enhance and supplement current evidence-based interventions for trauma-related psychopathology. While the technological aspects of EMA are currently being used to inform our understanding of various clinical syndromes, like PTSD, these strategies can also be incorporated into current treatments as a means of enhancing patient compliance and understanding factors associated with better clinical outcomes.
Future research might involve using an EMA framework to examine the frequency and intensity of intrusive memories and other reexperiencing symptoms in treatment-seeking individuals. As an example of this approach, Kleim and colleagues (2013) conducted a study using electronic diaries to monitor intrusion symptoms in victims of motor vehicle accidents. Participants carried electronic diaries and reported real-time information about intrusive re-experiencing of their trauma. Results indicated that individuals with PTSD were no more likely to experience intrusions than individuals who did not meet criteria for the disorder; although participants with PTSD reported more vivid intrusive symptoms. The authors concluded that EMA, in this study, provided useful information about individual perceptual experiences of PTSD symptoms that may have been otherwise obscured by retrospective reporting.

The assessment strategy utilized by Kleim and colleagues could be expanded by using this protocol shortly before individuals begin an evidence-based treatment for PTSD. By including a frequency-based assessment of intrusive memories prior to the start of treatment, researchers would be able to describe the frequency and quality of intrusive memories over a one-week period which would significantly enhance our understanding of this clinical phenomena in treatment-seeking patients. Furthermore, this type of data could then be used to evaluate whether certain characteristics of intrusive memories predict treatment outcomes. Finally, this approach would benefit the clinician and patient directly by providing an unbiased recording of intrusive memories that can aid treatment planning.

Another future direction of using EMA in PTSD and traumatic stress research would be including this data collection strategy as part of a mixed-methodological, longitudinal design to
evaluate potential risk and protective factors for developing psychopathology following trauma stress exposure. Price and colleagues (2014) examined the feasibility of this idea by collecting information about PTSD symptoms through the use of daily text messages in individuals who recently experienced a traumatic injury. Participants were recruited from the emergency department of a level 1 trauma center where they were receiving treatment for their injuries. Text messages were sent daily for 15 days to assess PTSD symptoms. The authors concluded that text messaging represents a viable strategy for collecting real-time information about PTSD symptoms, as 81% of the total sample responded to at least one text message and the overall response rate was approximately 63%.

Ideally, an EMA framework could be used to collect self-report of behaviors and psycho-physiological recordings of arousal in individuals at multiple intervals following exposure to traumatic stress. This information could then be used to understand who might develop trauma-related psychopathology following trauma exposure. This approach would be consistent with National Institute of Health strategic research priorities by identifying potential biomarkers and behavioral indicators for different stages of illness and recovery following trauma exposure.

**Conclusions.** The current research study suggests that PTSD symptoms are highly dynamic in nature, and several conclusions can be drawn regarding how and why PTSD symptoms change over time. First, results indicate that completing an EMA protocol that requires self-monitoring of PTSD symptoms may actually influence how PTSD symptoms change over time. This finding is in line with previous research examining the relationship between self-monitoring and PTSD symptom change (Ehlers et al., 2003; Reynolds and Tarrier.
1996; Tarrier et al., 1999), and suggests that EMA protocols may have some therapeutic utility, even when not combined with formal intervention.

Second, data from three time-series cases suggest that PTSD symptoms fluctuate over time; however the variability in PTSD symptom clusters was not explained by temporal cycles. While this finding did not support hypotheses based on the Stress Response Syndromes model of PTSD, it is possible that limitations inherent in self-report data made it difficult to determine if cycles were actually present. There was some evidence that PTSD symptom clusters may display chaotic variability, which may broadly reflect the complex and “homeostatic” processes involved in people’s ongoing daily processes of emotion regulation.

Finally, results of the current study are consistent with previous literature documenting the importance of negative emotions in the development and maintenance of PTSD. A primary implication of the current study is that PTSD symptoms are dynamic in nature over short periods of time, and negative emotions co-vary with PTSD symptoms. This finding supports the inclusion of persistent negative emotions as a symptom addition to the DSM-5.
Footnotes

1Horowitz uses the term “stress response syndromes” to refer to a number of pathological reactions that may occur following exposure to a stressful life event. His use of this term predates the establishment of PTSD as a diagnosis, and he continues to use it in recognition of the many pathological reactions that may occur following trauma, including but not limited to PTSD and Traumatic Grief. The use of this term is actually consistent with the most recent revision for PTSD in DSM-5, which intended to broaden post-traumatic emotional responses beyond fear.

2Engelhard et al. (2011) mention measuring other peritraumatic emotions but do not specify which ones.
References


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Table 1. Descriptive Statistics for Demographic Characteristics of the Participants

<table>
<thead>
<tr>
<th>Variables</th>
<th>N = 33</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>8</td>
<td>75.8</td>
</tr>
<tr>
<td>Female</td>
<td>25</td>
<td>24.2</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>30</td>
<td>90.9</td>
</tr>
<tr>
<td>Asian/Pacific Islander</td>
<td>3</td>
<td>9.1</td>
</tr>
<tr>
<td><strong>Sexual Orientation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Straight</td>
<td>30</td>
<td>90.9</td>
</tr>
<tr>
<td>Bisexual</td>
<td>2</td>
<td>6.1</td>
</tr>
<tr>
<td>Something else</td>
<td>1</td>
<td>3.0</td>
</tr>
<tr>
<td><strong>Relationship Status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single/Not dating</td>
<td>29</td>
<td>87.5</td>
</tr>
<tr>
<td>Married</td>
<td>3</td>
<td>9.4</td>
</tr>
<tr>
<td>Separated</td>
<td>1</td>
<td>3.1</td>
</tr>
</tbody>
</table>
**Table 2. Frequency of Traumatic Events Reported on Trauma History Screen**

<table>
<thead>
<tr>
<th>Event</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden death of a family or close friend</td>
<td>19</td>
</tr>
<tr>
<td>A really bad car, boat, train, or airplane accident</td>
<td>10</td>
</tr>
<tr>
<td>Some other sudden event that made you feel very scared, helpless, or horrified</td>
<td>10</td>
</tr>
<tr>
<td>Hit or kicked hard enough to injure – as a child</td>
<td>9</td>
</tr>
<tr>
<td>Suddenly abandoned by spouse, partner, parent, or family</td>
<td>9</td>
</tr>
<tr>
<td>Forced or made to have sexual contact – as an adult</td>
<td>8</td>
</tr>
<tr>
<td>Sudden move or loss of home and possessions</td>
<td>7</td>
</tr>
<tr>
<td>Hit or kicked hard enough to injure – as an adult</td>
<td>5</td>
</tr>
<tr>
<td>Forced or made to have sexual contact – as a child</td>
<td>5</td>
</tr>
<tr>
<td>Seeing someone die suddenly or get badly hurt or killed</td>
<td>4</td>
</tr>
<tr>
<td>A really bad accident at work or home</td>
<td>3</td>
</tr>
<tr>
<td>Attacked with a gun, knife, or weapon</td>
<td>3</td>
</tr>
<tr>
<td>A hurricane, flood, earthquake, tornado or fire</td>
<td>1</td>
</tr>
<tr>
<td><strong>During military service – seeing something horrible or being badly scared</strong></td>
<td>1</td>
</tr>
</tbody>
</table>

*Note.* Participants ($N = 33$) were able to report more than one event.
Table 3. *Mean PCL-C Scores over Time*

<table>
<thead>
<tr>
<th>Time</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time 1 (Screening)</td>
<td>57.16</td>
<td>10.06</td>
</tr>
<tr>
<td>Time 2 (Before EMA)</td>
<td>55.72</td>
<td>10.87</td>
</tr>
<tr>
<td>Time 3 (After EMA)</td>
<td>46.97</td>
<td>13.19</td>
</tr>
</tbody>
</table>
Table 4. Fit Statistics for Multi-Level Model Examining the Relationship between Negative Emotions and PTSD Symptoms

<table>
<thead>
<tr>
<th>Information Criterion</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>-2 Log Likelihood</td>
<td>11031.82</td>
</tr>
<tr>
<td>Akaike’s Information Criterion</td>
<td>11051.82</td>
</tr>
<tr>
<td>Hurvich and Tsai’s Criterion</td>
<td>11051.96</td>
</tr>
<tr>
<td>Bozdogan’s Criterion</td>
<td>11116.16</td>
</tr>
<tr>
<td>Schwarz’s Bayesian Criterion</td>
<td>11106.16</td>
</tr>
</tbody>
</table>

Note. Smaller values represent a better fitting model.
Table 5. Summary of Multi-Level Model Analysis Evaluating the Relationship between Negative Emotions and PTSD Symptoms

<table>
<thead>
<tr>
<th></th>
<th>Estimate</th>
<th>SE</th>
<th>t</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>12.10</td>
<td>1.58</td>
<td>7.65*</td>
<td>8.89</td>
</tr>
<tr>
<td>Time</td>
<td>-0.1</td>
<td>0.01</td>
<td>-1.23</td>
<td>-0.04</td>
</tr>
<tr>
<td>Anger</td>
<td>3.28</td>
<td>0.22</td>
<td>15.04*</td>
<td>2.85</td>
</tr>
<tr>
<td>Fear</td>
<td>2.04</td>
<td>0.21</td>
<td>9.54*</td>
<td>1.62</td>
</tr>
<tr>
<td>Guilt</td>
<td>2.06</td>
<td>0.26</td>
<td>7.59*</td>
<td>1.53</td>
</tr>
<tr>
<td>Shame</td>
<td>1.63</td>
<td>0.27</td>
<td>6.23*</td>
<td>1.12</td>
</tr>
</tbody>
</table>

Note. *p < .0001
Figure 1. Screenshots of EMA Application on Android Device

If you have any technical problems, please contact Daniel Dewey at (313) 683-1415

Question 1
Please indicate how much you have been bothered by the following problems since your last assessment?
Repeated, disturbing memories, thoughts, or images of a stressful past event

Question 19
How angry are you feeling about the event?

3
Quite a bit

Not at all
Extremely
Figure 2. Multilevel Model to Examine the Relationship between Negative Emotions and PTSD Symptoms

Level 1

- PTSD
  \[ \text{PTSD}_{ij} = \beta_{0i} + \beta_{1i}\text{TIME}_{ij} + \beta_{2i}\text{FEAR}_{ij} + \beta_{3i}\text{GUILT}_{ij} + \beta_{4i}\text{SHAME}_{ij} + \beta_{5i}\text{ANGER}_{ij} + \varepsilon_{ij}. \]

Level 2

- Intercept: \( \beta_{0i} = \gamma_{00} + \zeta_{0i} \)
- TIME: \( \beta_{1i} = \gamma_{10} + \zeta_{1i} \)
- FEAR: \( \beta_{2i} = \gamma_{20} \)
- GUILT: \( \beta_{3i} = \gamma_{30} \)
- SHAME: \( \beta_{4i} = \gamma_{40} \)
- ANGER: \( \beta_{5i} = \gamma_{50} \)
Figure 3.1. *Time-Series Plots of PTSD Symptom Clusters for Participant A*
Figure 3.2. Autocorrelation Function Plot of Reexperiencing Symptoms for Participant A
Figure 3.3. Reexperiencing Time-Series with Cubic Trend for Participant A
Figure 3.4. Spectral Density Chart of Reexperiencing Symptoms for Participant A
Figure 3.5. Avoidance Time-Series with Cubic Trend for Participant A
Figure 3.6. Spectral Density Chart of Avoidance Symptoms for Participant A
Figure 3.7. Hyperarousal Time-Series with Cubic Trend for Participant A
Figure 4.1. *Time-Series Plots of PTSD Symptom Clusters for Participant B*
Figure 4.2. Hyperarousal Time-Series with Quadratic Trend for Participant B
Figure 5.1. Time-Series Plots of PTSD Symptom Clusters for Participant C
Figure 5.2. Reexperiencing Time-Series with Quadratic Trend for Participant C
Figure 5.3. Avoidance Time-Series with Quadratic Trend for Participant C
Figure 5.4. Hyperarousal Time-Series with Cubic Trend for Participant C
Figure 6.1. Empirical Growth Plot 1 for PTSD Symptoms over Time

Note. Lines represent growth plots for individual participants.
Figure 6.3. Empirical Growth Plot 2 for PTSD Symptoms over Time

Note. Lines represent growth plots for individual participants.
Figure 6.3. *Empirical Growth Plot 3 for PTSD Symptoms over Time*

*Note.* Lines represent growth plots for individual participants
Figure 6.4. Empirical Growth Plot 4 for PTSD Symptoms over Time

Note. Lines represent growth plots for individual participants
Figure 6.5. Empirical Growth Plot 5 for PTSD Symptoms over Time

*Note.* Lines represent growth plots for individual participants.
Figure 6.6. Empirical Growth Plot 6 for PTSD Symptoms over Time

Note. Lines represent growth plots for individual participants
Figure 7. Phase Space Plots of Residuals for 3 Participants and 3 Symptom Scales

<table>
<thead>
<tr>
<th></th>
<th>1105</th>
<th>2231</th>
<th>2271</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Avoidance</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Dimension</td>
<td>4.553 ± 0.564; LLE = 0.421 ± 0.246</td>
<td>4.570 ± 2.828; LLE = 0.408 ± 0.227</td>
<td>Correlation Dimension 5.415 ± 5.415*; LLE = 0.466 ± 0.202</td>
</tr>
<tr>
<td><strong>Re-experiencing</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Dimension</td>
<td>3.989 ± 0.039; LLE = 0.432 ± 0.210</td>
<td>5.383 ± 2.015; LLE = 0.370 ± 0.598*</td>
<td>Correlation Dimension</td>
</tr>
<tr>
<td><strong>Hyperarousal</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Dimension</td>
<td>2.126 ± 0.090; LLE = 0.432 ± 0.247</td>
<td>6.558 ± 6.558*; LLE = 0.445 ± 0.203</td>
<td>Correlation Dimension</td>
</tr>
</tbody>
</table>

**Notes:** LLE = Largest Liapunov Exponent (LLE estimate > 0 suggests deterministic chaos (Sprott & Rowlands, 1995). *Indicates that this value is uninterpretable.
Appendix A

List of EMA Questions

Participants responded to these items on a 5-point likert scale from “0, not at all” to “4, extremely.”

How much you have been bothered by each of the following [things] since your last assessment?

Re-experiencing symptoms
1. Repeated, disturbing memories, thoughts, or images of a stressful past event
2. Repeated, disturbing dreams of a stressful experience from the past
3. Suddenly acting or feeling as if a stressful experience were happening again (as if you were reliving it)
4. Feeling very upset when something reminded you of a stressful experience from the past
5. Having physical reactions (e.g., heart pounding, trouble breathing, or sweating) when something reminded you of a stressful experience from the past

Avoidance/numbing symptoms
6. Avoid thinking about or talking about a stressful experience from the past or avoid having feelings related to it
7. Avoid activities or situations because they remind you of a stressful experience from the past
8. Trouble remembering important parts of a stressful experience from the past
9. Loss of interest in things that you used to enjoy
10. Feeling distant or cut off from other people
11. Feeling emotionally numb or being unable to have loving feelings for those close to you
12. Feeling as if your future will somehow be cut short

Hyperarousal symptoms
13. Trouble falling or staying asleep
14. Feeling irritable or having angry outbursts
15. Having difficulty concentrating
16. Being “super alert” or watchful on guard
17. Feeling jumpy or easily startled
Trauma-Related Emotions
18. How sad are you feeling about the event?
19. How angry are you feeling about the event?
20. How guilty are you feeling about the event?
21. How ashamed do you feel about the event?
22. How fearful are you about the event?
Appendix B

Exit Questionnaire Questions

1. Please describe your experience of participating in this study. How difficult was it to participate?

2. Did carrying around the electronic device and completing the surveys affect your behavior throughout the day? If so, how did it affect you?

3. How did participating in this study affect the symptoms that you reported in the survey?

4. Any additional comments?