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## Trauma Exposure, Depressive Symptoms, and Responding to Positive Events and Affect in Young Adults

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Trauma Exposure, Depressive Symptoms, and Responding to Positive Events and Affect in  
Young Adults

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A dissertation submitted in partial fulfillment of the requirements for the degree of

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In

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School of Psychology, Family and Community

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### Abstract

The relationship between trauma exposure and responding to positive affect and events is unclear. Depression may co-occur with trauma exposure, and may also independently predict responding to positive affect and events. The purpose of this study was to examine the relationships between trauma exposure, depressive symptoms, and responding to positive affect and positive life events among young adults. Participants were 277 (84.8% female, 56.3% Caucasian) undergraduates ages 18-39 ( $M = 19.67$ ,  $SD = 2.22$ ). At baseline, ANCOVAs were used to examine the relationships between trauma exposed/non-trauma exposed groups, as well as high/low depressive symptom groups, on responding to positive affect, i.e., dampening or positive rumination. Trauma exposure was not associated with greater dampening ( $F[1, 270] = 1.80$ ,  $p = .181$ ), but was associated with greater positive rumination ( $F[1, 271] = 5.27$ ,  $p = .02$ ) at baseline. The high depressive symptom group reported greater dampening ( $F(1,270) = 30.77$ ,  $p < .001$ ) and less positive rumination ( $F(1,271) = 3.97$ ,  $p = .047$ ). There was no significant interaction between trauma exposure and depression symptoms in predicting dampening ( $F[1,270] = .83$ ,  $p = .36$ ) or positive rumination ( $F[1,272] = .53$ ,  $p = .47$ ). Prospective analyses using Hierarchical Linear Modeling software found that trauma exposure groups did not differ on reported positive affect either prior to (unstandardized coefficient =  $-.12$ ;  $t[125] = -.83$ ;  $p = .38$ ) or following best hourly event (unstandardized coefficient =  $-.01$ ;  $t[125] = -.05$ ;  $p = .96$ ), nor on reported dampening (unstandardized coefficient =  $.05$ ;  $t[125] = .54$ ;  $p = .59$ ) or positive rumination (unstandardized coefficient =  $.02$ ;  $t[125] = .24$ ;  $p = .81$ ). The high depressive symptom group reported less positive affect both prior to (unstandardized coefficient =  $-.06$ ;

$t[126] = -4.45; p < .001$ ) and following best hourly events (unstandardized coefficient =  $-.49$ ;  $t[126] = -3.34; p < .001$ ). Hypotheses were not supported by findings, and limitations may have impacted results; however, findings highlighted potential avenues for future research on the impact of trauma and depression on responding to positive affect.

## **Chapter I: Introduction and Literature Review**

The estimated prevalence of traumatic exposure in college students ranges from 66% - 84% (Arttime et al., 2019; Read et al., 2011), and the experiences endorsed by this population vary widely in type and intensity. Traumatic events are experiences of actual or threatened death, serious injury, or sexual violence (American Psychiatric Association [APA], 2013), and are typically associated with several adverse outcomes including posttraumatic stress disorder (PTSD) and depression (Breslau, 2008). While the link between trauma exposure and negative outcomes is well established, recent research has also identified potential positive outcomes such as posttraumatic growth (PTG), suggesting some individuals employ strategies that serve to amplify protective factors in response to trauma (Tedeschi & Calhoun, 2004).

Traumatic exposure affects how individuals respond to subsequent life events, which may in turn contribute to mental health outcomes. Individuals with trauma exposure show altered responsivity to subsequent negative or stressful life events, for example showing greater levels of emotional reactivity (Heleniak et al., 2015) and maladaptive coping behaviors (McLaughlin et al., 2009; Michl et al., 2013). Less is known about the impact of trauma on subsequent responsivity to positive events (PE). Research suggests that the ability to experience positive emotions following positive life events and the ability to employ emotion regulation strategies that serve to amplify positive emotions are adaptive and associated with better functioning (Algoe & Fredrickson, 2011; Quoidbach et al., 2015). While trauma exposure has been associated with an increased ability to identify positive stimuli (e.g., PTG; Stockton et al., 2011; Tedeschi & Calhoun, 2004), it is also linked with anhedonia and reduced emotional reactivity



broadly, which are shown to negatively impact the ability to experience or amplify/upregulate positive emotions (Ford et al., 2012; Kerig et al., 2016).

It is also important to differentiate the effects of trauma on the ability to experience or regulate positive emotions from the effects of depression. Depression may co-occur with trauma exposure, and/or may independently predict positive affect and responding to positive affect and events (Kerig et al., 2016). Identifying the unique impact of trauma exposure and its subsequent posttraumatic stress (PTS) symptoms on positive affect and responses to positive events (PE) requires separating trauma exposure from depression. The purpose of this study is to examine the relationships between trauma exposure, depressive symptoms, and responding to positive affect and positive life events among young adults. The aim is to differentiate the effects of trauma exposure and subsequent PTS symptoms from the effects of depression on responding to positive affect and positive life events using data from a prospective weekly diary study.

### **Positive Life Events and Positive Affect**

Positive affect (PA) is defined as “pleasurable engagement with one’s environment” (Watson & Clark, 1984) and is associated with the experience of positive emotions and mood-state. Research has demonstrated that the cultivation of positive affect and emotions can lead to a range of positive outcomes, including psychological growth, improved health, and more satisfying interpersonal relationships (Boehm & Lyubomirsky, 2008; Diener & Seligman, 2002; Isen, 1999; Lyubomirsky et al., 2005; Pressman & Cohen, 2005). The ability to generate PA in response to life events is of clinical interest and has been shown to be inversely related to the presence of mental illness (Beck et al., 2003; Cohen & Pressman, 2006).

There are two important aspects of PA in response to life events that are relevant for mental health outcomes. The first involves the magnitude and duration of PA experienced

following an event. Perhaps predictably, research has demonstrated that, over time, more frequent and longer-lasting experiences of PA are associated with higher levels of positive emotions, as well as with greater reported life satisfaction (Bryant, 2003; Langston, 1994; Quoidbach et al., 2010).

Research now focuses on the effect of actively fostering PA following life events using cognitive strategies. These strategies are thought to cultivate well-being by promoting positive emotions, thoughts, and behaviors rather than solely focusing on reducing negative stimuli (Parks & Biswas-Diener, 2013; Schueller et al., 2014). Individuals may upregulate PA by engaging in cognitive or behavioral processes that allow them to experience positive emotion above and beyond the effects of the situation itself. For instance, attending to certain pleasurable features of an event can increase positive emotions (Killingsworth & Gilbert, 2010; Quoidbach et al., 2010). Further, engaging in cognitive strategies that promote positive internal, stable, and global factors related to the role an individual played in the occurrence of the event may also promote PA (Cheng & Furnham, 2003; Rigby & Huebner, 2005; Sanjuan et al., 2008).

One PA-focused cognitive strategy, positive rumination, involves responding to PA with “...recurrent thoughts about positive self-qualities, positive affective experience, and one’s favorable life circumstances” (Feldman et al., 2008, p 509). Individuals who engage in positive rumination are better able to identify positive stimuli within a situation. Positive rumination has been linked to the cultivation and maintenance of PA in several contexts (e.g., Chan et al., 2011; Karanci & Erikam, 2007; Taku et al., 2009; Tedeschi & Calhoun, 2004; Watkins, 2008).

In contrast to cognitive strategies designed to upregulate PA, individuals may also engage in cognitive processes that serve to decrease or dampen PA following life events (Feldman et al., 2008). Individuals who engage in dampening tend to focus on negative features of PA in the

form of thoughts aimed at decreasing the positive emotion such as, “These feelings won’t last” or “I got lucky.” Engagement in dampening has been associated with poor mental health outcomes, including elevated depressive symptoms (Feldman et al., 2008; Nelis et al., 2015). Further, because dampening results in blunted PA, it may contribute to the presence of posttraumatic stress symptoms.

### **PA, Trauma, and Depression**

While increased PA has been linked to better overall well-being and improved mental and physical health, deficits in PA have been associated with greater psychopathology and negative outcomes (Breslau & Davis, 1992; Feldman et al., 2008; Frewen et al., 2012; Li et al., 2017). This may be especially true regarding negative outcomes related to traumatic events. An estimated 70-90% of individuals in the US report experiencing at least one traumatic event in their lifetime (Benjet, et al., 2016; Kilpatrick et al., 2013). As previously discussed, trauma exposure may be associated with both negative and positive outcomes, however negative outcomes such as PTSD and depression are more thoroughly researched. Notably, as negative outcomes of trauma, PTSD and depression are both characterized by the experience of low PA at the symptom level.

PTSD occurs when a traumatic event results in subsequent symptoms related to avoidance, cognitive or emotional changes such as emotional numbing, hyperarousal, and persistent re-experiencing of the traumatic event. Lifetime prevalence of PTSD is estimated to be 6.8%, with a 12-month prevalence of approximately 3.5% (APA, 2013; Kessler et al., 2005a; Kessler et al, 2005b). Emotional numbing was added to the diagnostic symptom criteria for PTSD in the latest edition of the *Diagnostic Statistics Manual of Mental Disorders* (5<sup>th</sup> ed.; DSM-5; APA, 2013). Emotional numbing in the wake of a traumatic event may dampen an

individual's awareness of distressing emotions, and may also serve to decrease the experience of positive emotions (Fetzner et al., 2012; Frewen et al., 2012; Litz & Gray, 2002). For example, Spahic-Mihajlovic, Crayton, and Neafsey (2005) found that individuals with PTSD rated pleasant pictures as less arousing and salient. In another study, men with combat-related PTSD were less interested in images of attractive women than controls (Elman et al., 2005), indicating a tendency for traumatized individuals to down-regulate emotional reactions to otherwise positive stimuli.

While most individuals who experience a traumatic event do not go on to develop PTSD, posttraumatic responses exist on a spectrum, and even subclinical symptoms of PTS can cause impairment and distress. PTS symptoms reflect cardinal symptoms of PTSD, to include negative changes in cognition/emotion and emotional numbing but may not meet certain symptom criteria to warrant a PTSD diagnosis (Galtzer-Levy, 2014). Research evaluating the presence of subthreshold PTS symptoms is limited; however, there is some evidence that between 15% to 30% of individuals who experience trauma report such symptoms (Hughes et al., 2011; Norris, 2007). Given the prevalence of PTS, examination of the subclinical effects of trauma exposure is warranted.

Another pervasive negative outcome of trauma is depression. It is estimated that approximately 7.1% of all US adults (an estimated 17.3 million people) experience a major depressive episode in a given year, and that prevalence of major depressive episode is highest among young adults between the ages of 18 and 25 (13.8%; McCance-Katz, 2019). It is characterized by depressed mood and/or anhedonia, as well as myriad symptoms related to concentration difficulties, feelings of hopelessness or guilt, lack of energy, weight changes, sleep disturbance, and decreased interest or pleasure. As a cardinal symptom of depression,

anhedonia—or the diminished ability to experience positive stimuli—is particularly implicated in depressed individuals’ inability to respond to PA (Werner-Seidler et al., 2013). Depressed individuals tend to report lower levels of PA in response to life events than their non-depressed counterparts (O’Hara, Armeli, Boynton, & Tennen, 2014; Werner-Seidler et al., 2013), potentially due to engagement in previously discussed regulation strategies that serve to dampen the experience of positive stimuli.

### **Theoretical Background: Effects of Trauma Exposure on Affective Responsivity**

Much of the research examining the impact of traumatic exposure focuses specifically on the impact of PTSD on emotional processing. For instance, Foa and Kozack (1986) developed the emotional processing theory to explain how anxiety, and later PTSD (Foa et al., 1989; Foa & Rothbaum, 1998), influences individuals’ ability to experience and process emotions. Their theory was developed using Lang’s bioinformational theory of fear (1977; 1979), in which fear is exemplified by memories made up of stimulus, response, and meaning-making elements designed to help individuals avoid danger (e.g., [a] an individual sees a bear; [b] they begin to tremble and sweat; [c] they determine they are in danger). Foa and Kozack (1986) proposed anxiety disorders are created via over-activation of these fear structures, such that they “involve excessive response elements” (p. 21), are resistant to change, and do not accurately represent reality.

Foa, Steketee, and Rothbaum (1989) further applied emotional processing theory to the fear structure of PTSD, proposing that these structures differ from those related specifically to anxiety in that they not only included excessive stimulus and response elements, but also pathological elements of meaning-making. For example, a survivor of a sexual assault who was attacked in a park may form associations between parks, the experience of fear, and behavioral

and physiological responses (e.g., sweating, heart racing, shaking). This event may also disrupt previously held beliefs about oneself and the world and create problematic meaning elements in their fear structure (e.g., “The world is not safe.”) Visiting a park may now activate this fear network, which in turn serves to increase hypervigilance (i.e., arousal symptoms of PTSD), to allow information from the memory-stored fear network to enter consciousness (i.e., intrusion symptoms of PTSD), and for the individual to attempt to suppress or avoid intrusive thoughts/memories (i.e., avoidance symptoms of PTSD; see Brewin & Holmes, 2003 for review). Thus, a previously neutral experience is now perceived as hostile and threatening.

Trauma-exposed individuals have reported greater numbers of subsequent negative life events than their non-trauma exposed counterparts (Jin et al., 2018), and showed altered responsivity to negative and stressful life events. Foa and Rothbaum (1998) posited that traumatized individuals often experienced two specific negative cognitions that served to maintain PTS symptoms: (a) the world is not safe and (b) the belief that one is incapable of managing the effects of the trauma. These negative cognitions are thought to produce maladaptive coping mechanisms that serve to maintain and exacerbate the effects of PTS symptoms, especially in the face of negative stimuli (Ehlers & Clark, 2000). Trauma exposure has been linked with greater engagement in maladaptive coping strategies such as substance abuse (Dell’Osso et al., 2013; Hruska et al., 2011; Kronish et al., 2012), disordered eating (Brewerton, 2007; Jacobi et al., 2004; Mattocks et al., 2012; Smyth et al., 2008), and risk-taking or promiscuous behaviors (Dell’Osso et al., 2011; Pat-Horenczyk et al., 2007). Further, trauma exposure has been associated with greater emotional reactivity to negative or trauma-related stimuli (Blomhoff et al., 1998; Metzger et al., 1997; Saar-Ashkenazy et al., 2015), as well as

increased avoidant thoughts and behaviors (Benotsch et al., 2000; Bryant & Harvey, 1995; Keane & Barlow, 2002).

Trauma exposure may also alter individuals' ability to experience and regulate positive experiences and affect (Ehring & Quack, 2010; O'Bryan et al., 2015; Tull et al., 2007). Research has demonstrated that individuals who endorse trauma exposure report less PA in response to subsequent, positively valenced stimuli (Frewen et al., 2012; Orsillo et al., 2004; Spahic-Mihajlovic et al., 2005). DePierro, D'Andrea, and Frewen theorized this may be due to the interference of increased NA in the face of potentially positive stimuli, a construct they termed Negative Affect Interference (NAI; 2014). The concept of NAI was first hypothesized by Meehl (1975), who posited that individuals who are unable to feel pleasure may experience secondary emotions of shame or guilt when comparing themselves to those who can. DePierro et al. suggested this secondary response may further increase the experience of NA because the individuals lack the necessary PA reserves in order to effectively buffer against negative emotionality (2014).

Certain emotion regulation difficulties have also been found to interfere with traumatized individuals' ability to experience PA. Emotion regulation is broadly defined as the ability to attend to and adjust emotional responding in the context of influencing subsequent behavior (Tull et al., 2007). Gratz and Roemer (2004) posited that effective emotion regulation involves an awareness, understanding, and acceptance of emotions, as well as the ability to control impulsive behaviors and adapt in the context of emotionally distressing situations. Trauma-exposed individuals often struggle with aspects of emotion regulation when faced with emotionally charged stimuli, which in turn may lead to an individual's experience of any emotion as unpredictable or uncontrollable (O'Bryan et al., 2015; Mennin, 2005; Tull et al.,

2007). As a result, both internal and external stimuli that lead to emotional arousal may begin to be feared and, ultimately, avoided. While most research focuses on the impact of arousal via negative stimuli, there is some evidence that prolonged inability to identify and accept positive emotion leads to increased NA as a result of the aforementioned secondary negative emotions (i.e., shame or guilt; Gratz & Roemer, 2004; Hayes et al., 1999; Salters-Pedneault et al., 2007). Further, avoidance of emotionally evocative situations may interfere with an individuals' ability to seek out and enjoy positive situations, indirectly diminishing the capacity for experiencing PA (see Nawijn et al., 2015).

## **Current Study**

### *Purpose*

In order to better understand the effects of trauma exposure on individuals' ability to respond to PA, it is imperative to parse out the comorbid effects of depression on responding. It is well established that PTSD and depression often co-occur following trauma. Research suggests between 30-50% of individuals diagnosed with PTSD also meet criteria for Major Depressive Disorder (MDD; e.g., Angelakis & Nixon, 2015; Creamer et al., 2001; Kessler et al., 2005b). In spite of the frequent co-occurrence, there is some evidence to support the position that PTSD and depression represent separate constructs when considering posttraumatic outcomes (Blanchard et al., 1998; O'Donnell et al., 2004), and thus, may exert unique effects on responding to PA.

Both depressed and traumatized individuals tend to experience reduced overall emotional reactivity, and these individuals may be less able to experience and amplify positive emotions following life events than their non-depressed or non-traumatized counterparts (Feeny et al., 2000). This may be due to the presence of shared cognitive vulnerabilities, such as engagement



in rumination or dampening (Michl et al., 2013; Werner-Seidler et al., 2013). While research shows deficits in overall responsiveness for individuals with trauma and depressive symptoms, it often conflates the effects of trauma and depressive symptoms on responding to life events. Research examining the effect of trauma on responding to PE separately from the effects of depression is lacking.

The primary purpose of this study was to better understand the effects of trauma exposure and depression on responding to positive affect and events. Because depression may co-occur with trauma exposure, and may also independently predict responding to PA, it is necessary to separate the effects of trauma exposure from those of depression. To that end, the purpose of this study was to examine responding to daily positive affect and events in trauma-exposed and non-trauma-exposed individuals who endorse high or low depressive symptoms.

### *Hypotheses*

I hypothesized that, compared to those who do not, individuals who endorsed exposure to trauma would (a) report less PA in response to daily positive events than non-trauma-exposed counterparts, and (b) report more dampening and less positive rumination in response to daily positive events than non-trauma-exposed counterparts, as is consistent with the literature. Given the dearth of literature focused on the effects of trauma exposure on positive affect and responding to PE, I did not make a specific hypothesis regarding differential effects of trauma exposure and/or depressive symptoms on responding.

## **Chapter II: Method**

### **Participant Characteristics**

Our sample was drawn from a larger sample of students taking part in a study examining stress, depression, and somatic symptoms. Participants were at least 18-years-old and enrolled in

undergraduate psychology courses at a private university in the Pacific Northwest. The total sample for this study at baseline included 277 (84.8% female) college undergraduates between the ages of 18 and 39 (mean age = 19.67, SD = 2.22). Approximately 56.3% of baseline participants identified as Caucasian, 13.4% Asian, 1.1% Native American, 5.4% African American, 3.6% Pacific Islander, and 5.8% other. In our sample, no participants identified as Hispanic/Latino. Of the 277 participants at baseline, 150 agreed to participate in the daily diary portion of the study.

### **Procedure**

The current study is part of an ongoing study at Seattle Pacific University (SPU) entitled Stress and Somatic Symptoms in Young Adults (SASSY). All study procedures and materials for SASSY were approved by the SPU institutional review board (IRB # 161702026R). For the purposes of this study, only pertinent procedures will be described in full.

Students who consented to participate were asked to complete baseline questionnaires via SONA and Qualtrics survey software, and were subsequently invited to participate in an in-person laboratory visit. Those who completed both baseline questionnaires and the laboratory visit were contacted via email to participate in ecological momentary assessment (EMA) using their personal mobile phones. Consenting participants were asked to download the RealLife Exp smartphone application that allowed them to complete six random daily questionnaires, every day, for seven days. Each EMA questionnaire took approximately 5-10 minutes to complete. In return for their participation, students received five research credits required by the university; in order to receive full credit, participants were required to complete a minimum of 75% of the EMA questionnaires.

EMA questionnaires inquired about state affect at the time of the assessment as well as use of emotion regulation strategies in response to the participant's self-reported best and worst event during the past hour. A combination of free response and Likert formats were used.

## **Measures**

### ***Trauma Exposure***

Trauma exposure was assessed at baseline using the Brief Trauma Questionnaire (BTQ; Schnurr et al., 1999). The BTQ is a 10-item self-report questionnaire designed to determine whether an individual has experienced an event that meets Criteria A for PTSD diagnostic criteria during the course of their lifetime. If a particular traumatic event has been endorsed, the respondent was asked (a) whether they thought their life was in danger or they might be seriously injured; and/or (b) whether they were seriously injured. In this study, individuals who endorsed at least one traumatic event (1) were compared to those that did not (0). The BTQ is considered to be a valid and reliable measure of trauma exposure (Sumner et al., 2015). In the present study,  $\alpha = .82$ .

### ***PTS Symptoms***

Symptoms of PTS were assessed at baseline using the PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013). The PCL-5 is a 20-item measure that assesses for DSM-5 symptom criteria for PTSD. Participants respond to items regarding how often, in the past month, they were bothered by 20 specific symptoms of PTSD (e.g., "...repeated, disturbing, and unwanted memories of the stressful experience"; "...having strong negative feelings such as fear, horror, anger, guilt, or shame"). Responses range from 0 (*Not at all*) to 4 (*Extremely*), with greater scores indicating greater symptom severity.

The PCL-5 was used in conjunction with the BTQ in post-hoc analyses to compare individuals who endorsed both traumatic exposure and high PTS with those that did not on reported outcomes. Participants who endorsed a score of 33 or more on the PCL-5 were considered high PTS, while those that scored 32 or below were considered low (Weathers et al., 2013). In post-hoc analyses, individuals who endorsed both traumatic exposure on the BTQ and high PTS symptoms made up the high trauma group (1), while those who did not were grouped as such (0). The PCL-5 has demonstrated strong convergent ( $r_s=.74$  to  $.85$ ) and discriminant ( $r_s=.31$  to  $.60$ ) validity as well as internal consistency ( $\alpha=.94$ ; Blevins et al., 2015). In the current study,  $\alpha = .96$ .

### ***Depressive Symptoms***

Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The CES-D is a 20-item measure, on which participants rate how often they experienced symptoms (e.g., “I felt depressed”; “I was bothered by things that don’t usually bother me”) over the past week on a scale of 0 (*Rarely or none of the time; less than 1 day*) to 3 (*Most or all of the time; 5-7 days*). Higher scores indicated a greater level of depressive symptomology. In previous studies, the CES-D has demonstrated internal consistency of  $\alpha= .92$  in a university population, and evidence of concurrent and discriminant validity with other symptom checklists (Umegaki & Todo, 2017). Recommended cut-off of 20 was used in this study to determine severity of depressive symptoms (i.e., High (1) vs Low (0); Vilgut et al., 2016). In our study,  $\alpha = .85$ , demonstrating good internal consistency.

### ***Responses to Positive Affect***

Participants’ responses to positive affect were measured using the Response to Positive Affect Scale (RPA; Feldman, et al., 2008). The RPA is a 17-item measure on which participants

rate how they respond to positive emotion (e.g., "...think 'I am living up to my potential'"; "...think 'this is too good to be true'") on a scale of 1 (*Almost never*) to 4 (*Almost always*). The RPA produces two subscales that measure specific patterns of responding to positive emotion. The Total Positive Rumination scale (nine items) measures the extent to which the individual employs cognitive strategies aimed at amplifying the effects of positive emotion, while the Dampening subscale (eight items) measures an individual's attempts to cognitively depress positive emotions. In previous studies, the Dampening and Positive Rumination subscales have demonstrated good internal consistency ( $\alpha = .83$  and  $.82$ , respectively; Nelis, et al., 2015; Nelis et al., 2016). The full RPA was administered (17 items) at baseline, and internal consistencies for Positive Rumination ( $\alpha = .89$ ) and Dampening ( $\alpha = .88$ ) subscales were satisfactory.

For daily EMA responding, participants completed a modified, nine-item version of the RPA following their best hourly events. This scale was designed to measure cognitive strategies employed immediately following the event that might serve to either dampen or amplify its emotional effects. Internal consistency for Positive Rumination and Dampening subscales following best hourly events was again satisfactory ( $\alpha = .84 - .86$  and  $.72 - .80$ , respectively)

### ***Positive Affect***

Participants' state experience of positive affect was assessed prior to reporting their best/worst experiences of the hour using a modified version of the Positive and Negative Affect Schedule-Expanded Form (PANAS-X; Watson & Clark, 1994). The modified PANAS-X is a 20-item measure on which participants report the extent to which they experience certain positive and negative emotions on a scale of 1 (*Very slightly or not at all*) to 5 (*Extremely*). In this study, only the General Dimension Scale of Positive Affect was used (10 items). The Positive Affect subscale assesses the extent to which an individual experiences positive emotions. This subscale

is scored by averaging these responses. The Positive Affect subscale is considered to be a psychometrically valid measure of positive affect in young adults ( $\alpha = .83 - .90$ ; Watson & Clark, 1999). In the present study,  $\alpha = .92 - .94$ )

Additionally, participants' experiences of positive affect following their best hourly event were measured using a modified version of the Positive and Negative Affect Schedule for Children (PANAS-C; Laurent et al., 1999). In the present study, only the four-item Positive Affect subscale was used to assess for positive emotions following participants' best hourly events on a scale of 1 (*Very slightly or not at all*) to 5 (*Extremely*). The Positive Affect subscale of the PANAS-C is considered to be psychometrically valid measure of positive affect in young adults ( $\alpha = .89$ ; Ebesutani et al., 2012). Chronbach's alpha in the present study ranged from .89 - .91.

### ***Data Analytic Plan***

This study examined the effect of depressive symptoms and trauma exposure on positive affect and cognitive emotion regulation in response to positive events. For planned analyses, I intended to identify four groups within my sample: trauma-exposed/non-exposed and high/low depressive symptoms. The trauma-exposed/non-exposed groups were defined using the BTQ, where those in the exposed group endorsed one or more traumatic events in their lifetime ( $n = 186$ ). Those that did not endorse traumatic exposure were grouped as such ( $n = 91$ ). Depressive symptom groups were defined by symptom endorsement on the CES-D, such that individuals who endorsed a score of 20 or more on CES-D at baseline were sorted into the high depressive symptom group ( $n = 158$ ); individuals that scored below 20 were placed in the low depressive symptom group ( $n = 115$ ; Vilgut, et al., 2016). Post-hoc analyses compared individuals who endorsed trauma exposure on the BTQ as well as high PTS symptoms (i.e., PCL-5 score of 33

and above;  $n = 47$ ) with those who did not ( $n = 226$ ) on reported responding to PA and PE using both baseline and daily EMA data.

To analyze baseline data, I ran two analyses of covariance (ANCOVA) to compare the differential effects of depressive symptoms and trauma exposure on baseline responding to positive affect (positive rumination and dampening). An ANCOVA measures the effect of two categorical independent variables (i.e., trauma exposure and depressive symptoms) on the dependent variable (i.e., dampening and positive rumination), while also testing the interaction effect of both independent variables. These analyses were performed in SPSS 26.0.

I then examined the effects of trauma exposure and/or depressive symptoms on state positive affect and cognitive responding to daily positive events using Hierarchical Linear Modeling software (HLM; Raudenbush et al., 2004). HLM allows for the measurement of within-person fluctuations in the outcome variable (i.e., dampening, positive rumination, hourly positive affect, and positive affect following best event) as a function of within-subject differences in the predictor variables (i.e., trauma group and depressive symptom group; Bolger et al., 2003). Regression analyses were run to determine differential effects of belonging to symptom-group (Level 2 [L2]) on responding to daily PA and PE (Level 1 [L1]) across 42 datapoints (i.e., PING).

## **CHAPTER III: Results**

### **Data Preparation and Missing Data**

#### ***Baseline Data***

Prior to statistical analysis, data were inspected and managed for missingness. The initial dataset at baseline consisted of 277 cases. Forty-one percent of the variables and 10% of the cases had some missing data. Ninety-nine percent of the values in the model had complete data.

Little's MCAR test was consistent with the assumption that data were missing completely at random ( $p = .23$ ). Visual inspection of the missing value patterns indicated a general pattern of missingness as defined by Enders (2010). Person-mean imputation was used to impute scale scores at the item level for participants who completed at least 80% of items in a measure, following Parent (2013) recommendations. In the present study, no single case was missing more than 19% of data at baseline, therefore the final N for baseline analyses was 277.

Tests of assumptions for ANCOVA analyses were conducted, including: (a) normal distribution of the data, (b) lack of outliers in the dataset, and (c) homogeneity of variances. Normality of baseline data was assessed through interpretation of skewness and kurtosis of study variables. Both dependent variables at baseline demonstrated either positive (dampening;  $\hat{\gamma} = .503$ ) or negative (positive rumination;  $\hat{\gamma} = -.54$ ) skew using Levene's test ( $p = .00$ ). Research demonstrates Levene's test may not be useful in large samples (e.g.,  $n > 85$ ), as even slight deviations from normality could be considered statistically significant. Per guidelines put forth by Kline (2016), interpretation of the skew statistic for each variable such that the absolute value of  $\hat{\gamma} < 3.0$  demonstrates sufficient evidence for a normal distribution. Neither variable demonstrated kurtosis. Thus, it is assumed the variables are sufficiently normally distributed.

Data were also analyzed for homogeneity of variances. For positive rumination, Levene's test was not significant ( $p = .30$ ), indicating equal variances across groups. For dampening, Levene's test was also not significant ( $p = .13$ ). Because there were no outliers in our dataset, the three assumptions needed for ANCOVA analyses were met.

### ***EMA Data***

One-hundred fifty participants agreed to participate in the daily diary portion of the study. EMA variables were also analyzed and managed for missingness using the multiple imputation



tools in SPSS. Missing value analysis indicated 100% of the variables and cases had some missing data. Fifty-one percent of the values in the weekly dataset had complete data. Inspection of missing value patterns indicated a pattern of missingness consistent with Ender's monotonic pattern (2010), and attrition toward the end of the week was most frequently observed. Little's MCAR test was non-significant, indicating weekly data were also missing completely at random ( $p = 1.00$ ). Again, individual scores were calculated when participants completed 80% of items in a measure. Nineteen participants did not complete any weekly data after completing demographic data. Comparison of these participants to those who completed data showed no significant differences regarding age, gender, and on all EMA measures. Thus, total number of participants for EMA analyses was 131.

### Descriptive Analyses

**Table 1**  
*Bivariate Correlations among Study Variables: Baseline*

	1	2	3	4	5	6	7	<i>M (SD)</i>
1. Gender N = 277	-							.91 (.56)
2. Age N = 275	-.03	-						19.67 (2.21)
3. PTS N = 277	.20**	-.01	-					19.48 (18.26)
4. BTQ N = 277	.03	.09	.31**	-				1.32 (1.39)
5. CES-D N = 277	.14*	-.06	.43**	.25**	-			23.76 (10.21)
6. RPA-D N = 274	.12	.33	.27**	.24**	.41**	-		16.10 (5.70)
7. RPA-PR N = 276	.08	.00	.04	.08	-.06	.10	-	27.20 (5.71)

Notes: BTQ = Brief Trauma Questionnaire (Trauma Exposure: Yes or No); PTS = Posttraumatic Stress Symptoms (PTSD Checklist-5 score 33 or above + BTQ: Yes, or no); CES-D = Center for Epidemiological Studies Depression Scale (Depression symptoms above or below cut off [20]); PANAS-X = Positive and Negative Affect Schedule – Expanded Form; RPA-D = Responses to Positive Affect-Dampening; RPA-PR = Responses to Positive Affect-Positive Rumination Total  
\* $p < .05$ , \*\* $p < .01$

### ***Baseline Data***

Bivariate correlations, means, and standard deviations for baseline variables are presented in Table 1. Depressive symptoms were positively correlated with gender, trauma exposure, and dampening. Dampening was also significantly positively correlated with trauma exposure. Contrary to hypotheses, trauma exposure was not negatively correlated with positive rumination.

### ***EMA Data***

**Table 2**  
*Bivariate Correlations among Study Variables: EMA*

	1	2	3	4	5	6	7	8	9	<i>M(SD)</i>
1. Gender N = 130	-									.93 (.45)
2. Age N = 130	-.02	-								19.52 (2.16)
3. BTQ N = 131	.12	.19*	-							1.27 (1.34)
4. PTS N = 130	-.25**	-.03*	.38**	-						.19 (.39)
5. CES-D N = 130	.23**	.01	.20*	.32**	-					23.41 (9.88)
6. RPA-PR N = 131	-.05	-.13	.01	.04	-.15	-				1.13 (.43)
7. RPA-D N = 130	-.07	-.02	.07	.01	.18*	.81**	-			.84 (.46)
8. PANAS-C N = 129	-.05	-.14	-.06	-.01	-.26**	.73**	.54**	-		3.18 (.85)
9. PANAS-X N = 131	-.07	-.05	-.06	-.22**	-.39**	.51**	.44**	.73**	-	2.61 (.78)

Notes: BTQ = Brief Trauma Questionnaire (Trauma Exposure: Yes or No); PTS = Posttraumatic Stress Symptoms (PTSD Checklist-5 score 33 or above + BTQ: Yes, or no); CES-D = Center for Epidemiological Studies Depression Scale (Depression symptoms above or below cut off [20]); PANAS-X = Positive and Negative Affect Schedule – Expanded Form; RPA-D = Responses to Positive Affect-Dampening; RPA-PR = Responses to Positive Affect-Positive Rumination Total; PANAS-C = Positive and Negative Affect Schedule for Children.

\* $p < .05$ , \*\* $p < .01$

Bivariate correlations, means, and standard deviations for EMA variables are presented in Table 2. Participants' age positively correlated with depressive symptoms, and gender significantly correlated with trauma exposure. Depressive symptoms and trauma exposure were positively correlated but, contrary to hypotheses, trauma exposure did not significantly correlate with any outcome measures. Depressive symptoms were positively correlated with dampening, and were negatively related to positive affect both at state level and in response to best hourly event. Outcome variables were all significantly positively correlated.

### Baseline Analyses

Baseline data were analyzed by two ANCOVAs using SPSS 26. In the first model, trauma exposure group (BTQ) and depressive symptom group (CES-D) were the independent variables. The dependent variable was trait dampening (RPA-D). In the second model, the independent variables remained the same, and reports of positive rumination (RPA-PR) was the measured outcome variable. Because it significantly correlated with depressive symptom groups, gender was entered as a covariate in both models.

### *Trauma Exposure and Depressive Symptom Groups on Dampening*

**Table 3**

*Means and Standard Deviations for ANCOVA: Dampening*

Trauma Exposure	Depression Symptoms	N	Mean (SD)
No Trauma Exposure	Low	45	12.96 (3.71)
	High	44	17.49 (6.16)
	Total	89	15.20 (5.53)

Trauma Exposure	Low	71	14.54 (5.46)
	High	114	17.79 (5.59)
	Total	185	16.54 (5.75)

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Table 3 provides a summary of means and standard deviations for independent variables as a function of the dependent variable. Results demonstrated a significant main effect of depression symptom group reports of dampening,  $F(1,270) = 30.77, p < .001$ , meaning individuals in high and low depression symptom groups significantly differed in their reports of dampening when responding to positive affect or emotions. The effect of gender was non-significant, indicating reports of dampening did not differ according to gender,  $F(1,270) = 2.48, p = .12$ . Follow-up pairwise comparisons revealed that individuals who reported high depression symptoms reported dampening significantly more than those who reported low depression symptoms ( $p < .01$ ). These results are consistent with the literature. There was a non-significant main effect of trauma exposure group on reports of dampening,  $F(1, 270) = 1.80, p = .181$ , indicating there was no difference in dampening across individuals in either trauma exposure group. Finally, the interaction term was not significant, such that the effect of depression symptom group on dampening did not change or differ according to trauma exposure group ( $F[1,270] = .83, p = .36$ ).

#### ***Trauma Exposure and Depressive Symptom Groups on Positive Rumination***

**Table 4**  
*Means and Standard Deviations for ANCOVA: Positive Rumination*

Trauma Exposure	Depression Symptoms	N	Mean (SD)
No Trauma Exposure	Low	45	27.13 (5.07)
	High	45	25.14 (5.36)
	Total	90	26.14 (5.28)
Trauma Exposure	Low	71	28.28 (5.21)

High	115	27.35 (6.21)
Total	186	27.71 (5.85)

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Table 4 provides a summary of means and standard deviations for symptom group independent variables as a function of the dependent variable, positive rumination. Simple main effect analyses found significant effect of trauma exposure group on reports of positive rumination ( $F[1, 271] = 5.27, p = .02$ ). There was also a significant main effect of depression symptom group on reporting positive rumination,  $F(1,271) = 3.97, p = .047$ ). Again, the effect of gender was non-significant  $F(1,271) = 2.03, p = .16$ . Pairwise comparisons were conducted to explore significant main effects. Analyses revealed individuals who endorsed trauma exposure were significantly more likely to report positive rumination than those who did not ( $p < .05$ ). Further, consistent with the literature, individuals who reported fewer depressive symptoms were also more likely to report positive rumination than their high depression symptoms counterparts ( $p < .05$ ). The interaction term was again non-significant, meaning the effect of depression symptom group on engagement in positive rumination does not differ as a function of trauma exposure group ( $F[1,272] = .53, p = .47$ ).

### **EMA Analyses**

The software package HLM 7.0 (student version) was used to analyze the hierarchically nested dataset of 131 individuals. Consistent with McCoach's recommendation (2010), models for each within-subject repeated measures outcome variable (i.e., dampening [DAMPEN], positive rumination [PR], hourly positive affect [PANAS-X], and positive affect following best event [PANAS-C]) were built in a step-wise fashion, beginning with an unconditional model. Sequential inclusion of predictor variables was performed, resulting in a full, final model. Trauma group, depression group, and the trauma x depression interaction were entered in L2 as

between-subject variables. Additionally, gender and age were initially included in the model as control variables, but did not achieve significance in any analyses and were thus trimmed from the final models.

***Model 1: Group Membership on Dampening in Response to Positive Events***

The first model tested whether membership in depression symptom or trauma exposure groups predicted dampening following positive events. Individual timepoint (PING) was entered at L1, group-centered, and depression symptom group was entered in L2 on the intercept, uncentered. Results indicated that depression group did not predict dampening (unstandardized coefficient =  $-.16$ ;  $t[126] = -1.92$ ;  $p = .06$ ). Trauma exposure group was also entered in L2, uncentered. Again, results indicated that trauma exposure did not predict dampening (unstandardized coefficient =  $.05$ ;  $t[125] = .54$ ;  $p = .59$ ). Finally, the interaction term (i.e., depression group x trauma exposure) was entered, uncentered, at L2. Similarly, the interaction of depressive symptoms and trauma exposure did not predict reported dampening (unstandardized coefficient =  $-.07$ ;  $t[124] = -.39$ ;  $p = .70$ ).

***Model 2: Group Membership on Positive Rumination in Response to Positive Events***

The second model tested whether group membership predicted positive rumination in response to daily positive events. PING was entered at L1, group-centered, and depression symptom group was entered, uncentered, at L2. In this model, depression group membership did not predict positive rumination (unstandardized coefficient =  $-.10$ ;  $t[126] = -1.25$ ;  $p = .21$ ). Trauma exposure group was entered at L2, and did not significantly predict positive rumination following positive events (unstandardized coefficient =  $.02$ ;  $t[125] = .24$ ;  $p = .81$ ). Lastly, the interaction term was entered, uncentered at L2; this was again not significant (unstandardized coefficient =  $-.02$ ;  $t[124] = -.13$ ;  $p = .90$ .)

***Model 3: Group Membership on State Positive Affect Prior to Positive Events***

The next model tested whether group membership predicted state-level positive affect. Again, PING was entered at L1, group-centered, and depression symptom group was entered in L2, uncentered. In this model, depression group significantly predicted state positive affect (unstandardized coefficient =  $-.06$ ;  $t[126] = -4.45$ ;  $p < .001$ ). Trauma exposure was entered at L2, uncentered, but this was not significant, indicating exposure group did not predict state positive affect (unstandardized coefficient =  $-.12$ ;  $t[125] = -.83$ ;  $p = .38$ ). The interaction term was then entered, uncentered, at L2. Again, the interaction of symptom group did not predict reported state positive affect (unstandardized coefficient =  $-.26$ ;  $t[124] = -.98$ ;  $p = .41$ ).

***Model 4: Group Membership on State Positive Affect in Response to Positive Events***

The final model tested whether symptom group predicted positive affect following positive events. PING was entered at L1, group-centered, and depression symptom group was entered at L2, uncentered. Similar to state positive affect, depression group significantly predicted positive affect immediately following positive events (unstandardized coefficient =  $-.49$ ;  $t[126] = -3.34$ ;  $p < .001$ ). Trauma exposure group was entered at L2, uncentered; this was not significant (unstandardized coefficient =  $-.01$ ;  $t[125] = -.05$ ;  $p = .96$ ). Finally, the interaction term was entered at L2. Again, the interaction of depressive symptoms and trauma exposure did not significantly predict engagement in positive affect following positive events (unstandardized coefficient =  $-.35$ ;  $t[124] = -1.16$ ;  $p = .25$ ).

**Post-hoc Analyses**

Planned analyses examined the effect of trauma exposure on responding to positive affect and events. While there is evidence to support that trauma exposure alone may sufficiently

predict responding to positive affect and events (DePierro et al., 2018), it may be that the experience of PTS symptoms following trauma exposure exacerbates the effects of trauma on cognitive responding. To that end, post-hoc analyses comparing individuals who endorse both trauma exposure (BTQ) *and* posttraumatic stress symptoms (PCL-5; 1) with those who did not (0), as well as previously defined depressive symptom groups, on responding to PA and PE were conducted.

### ***Post-hoc Baseline Analyses***

For baseline analyses, ANCOVAs were again conducted using SPSS, this time comparing the PTS groups with depression groups on reported dampening and positive rumination. Gender was again included as a covariate in both models.

### **PTS and Depressive Symptom Groups on Dampening.**

**Table 5**

*Post-hoc Means and Standard Deviations for ANCOVA: Dampening*

Trauma Symptoms	Depression Symptoms	N	Mean (SD)
No Trauma Symptoms	Low	110	13.76 (4.94)
	High	116	16.97 (5.42)
	Total	226	15.41 (5.42)
Trauma Symptoms	Low	5	17.20 (5.63)
	High	42	19.74 (6.15)
	Total	47	19.47 (6.09)

Table 5 provides a summary of means and standard deviations for independent variables on dampening. As before, results demonstrated a significant main effect of depression symptom group reports of dampening,  $F(1,273) = 4.37, p < .05$ , meaning individuals in high and low depression symptom groups significantly differed in their reports of dampening. The main effect of PTS group on reported dampening was also significant ( $F[273] = 5.28, p < .05$ ). The effect of



gender was again non-significant, indicating reports of dampening did not differ according to gender,  $F(1,273) = .76, p = .39$ .

Pairwise comparisons showed that individuals who reported high depression symptoms reported dampening significantly more than those who reported low depression symptoms ( $p = .04$ ), as previously demonstrated. Consistent with the literature, individuals who reported high PTS symptoms and trauma exposure also reported greater dampening than their counterparts ( $p = .02$ ). Taken together with previous analyses, the presence of PTS symptoms may moderate the relationship between trauma exposure and reports of dampening. Finally, the interaction term was not significant, such that the effect of depression symptom group on dampening did not change or differ according to PTS symptom groups ( $F[1,273] = .10, p = .75$ ).

#### **PTS and Depressive Symptom Groups on Positive Rumination.**

**Table 6**

*Post-hoc Means and Standard Deviations for ANCOVA: Positive Rumination*

Trauma Symptoms	Depression Symptoms	N	Mean (SD)
No Trauma Symptoms	Low	110	27.84 (5.24)
	High	118	26.41 (5.55)
	Total	228	27.10 (5.44)
Trauma Symptoms	Low	5	28.00 (4.18)
	High	42	27.64 (7.26)
	Total	47	27.68 (6.96)

Table 6 provides a summary of means and standard deviations for symptom groups on reported positive rumination. Contrary to previous analyses, neither PTS group ( $F[275] = .18; p = .68$ ) nor depressive symptom group significantly predicted reported positive rumination ( $F[275] = .57; p = .45$ ). The effect of gender was also not significant ( $F[275] = 1.52; p = .22$ ). Again, the

interaction term was not significant, indicating that the effect of PTS symptom group on positive rumination did not differ according to depression symptom groups ( $F[275] = .08; p = .78$ ).

***Post-hoc EMA Analyses.***

The student version of HLM 7.0 was again used to analyze daily EMA data. PTS group, depression group, and the PTS x depression interaction were entered in L2 as between-subject variables. For all models, PING was entered at L1. To avoid redundancy in text, the first step as outlined in previous models (i.e., depression group predicting outcomes) will not be repeated in forthcoming results.

**Post-hoc Model 1: Group Membership on Dampening in Response to Positive**

**Events.** The first model tested whether membership in depression symptom or PTS symptom groups predicted dampening following positive events. PING was entered at L1, group-centered. Depression symptom group was entered in L2 on the intercept, uncentered, and was not-significant. PTS group was next entered in L2, uncentered. Results indicated that PTS did not predict state dampening (unstandardized coefficient = .07;  $t[125] = .61; p = .55$ ). Finally, the interaction term (i.e., depression group x PTS group) was entered, uncentered, at L2. Similarly, the interaction did not predict reported dampening (unstandardized coefficient = .21;  $t[124] = -2.11; p = .47$ ).

**Post-hoc Model 2: Group Membership on Positive Rumination in Response to**

**Positive Events.** The second post-hoc model tested whether group membership predicted positive rumination in response to daily positive events. In this model, depression group membership did not predict positive rumination. PTS group was entered at L2, but did not significantly predict positive rumination following positive events (unstandardized coefficient =

.08;  $t[125] = .73$ ;  $p = .47$ ). Lastly, the interaction term was entered, uncentered at L2; this was again not significant (unstandardized coefficient =  $-.13$ ;  $t[124] = -.45$ ;  $p = .65$ ).

### **Post-hoc model 3: Group Membership on State Positive Affect Prior to Positive**

**Events.** The next model tested whether group membership predicted state-level positive affect. In this model, depression group significantly predicted state positive affect. PTS group was entered at L2, uncentered, but this was not significant (unstandardized coefficient =  $-.25$ ;  $t[125] = -1.46$ ;  $p = .15$ ). The interaction term was then entered, uncentered, at L2. Similar to previous model, the interaction of symptom group did not predict state positive affect (unstandardized coefficient =  $.39$ ;  $t[124] = .85$ ;  $p = .40$ ).

### **Post-hoc Model 4: Group Membership on State Positive Affect in Response to**

**Positive Events.** The fourth model tested whether symptom group predicted positive affect following positive events. Depression group significantly predicted positive affect immediately following positive events, but PTS group (entered at L2, uncentered) did not (unstandardized coefficient =  $.12$ ;  $t[125] = .60$ ;  $p = .56$ ). Finally, the interaction term was entered at L2. Again, the interaction of depressive and PTS symptoms did not significantly predict engagement in positive affect following positive events (unstandardized coefficient =  $.04$ ;  $t[124] = .07$ ;  $p = .94$ ).

## **Chapter IV: Discussion**

The primary purpose of the current study was to examine the differential impact of trauma exposure and depressive symptoms on cognitive responding to positive affect and events. While the relationship between depressive symptoms and cognitive responding to PA and PE is well established in the literature (e.g., Raes et al., 2012; Werner-Seidler et al., 2013), studies emphasizing the influence of trauma exposure on responding to PA and PE are sparse. Analyses were conducted to test the hypotheses that trauma-exposed individuals would (a) report less PA

prior to and following PE, and (b) report greater dampening and less positive rumination at both baseline and following PE than their non-trauma-exposed counterparts. Further, because depressive symptoms are often themselves sequelae of trauma exposure, while also influencing cognitive responding outside the context of trauma exposure, I sought to parse apart the unique effects of both. Given the dearth of literature examining this particular relationship, no hypothesis was made regarding the expected differential effects.

The influence of trauma exposure and depressive symptoms on reported dampening and positive rumination was examined at baseline. This relationship, as well as the relationship between symptom groups and state PA prior to and following PE, was also examined using daily EMA data.

#### **Do Trauma Exposed Individuals Report Less Positive Affect Following Positive Events?**

Depressive symptoms were significantly related to reported positive affect both prior to and following best hourly events, such that individuals in the high depressive symptom group reported less PA prior to and following PE, which is consistent with established literature. The relationships between trauma exposure group and reported PA both prior to and following best hourly events were not significant, and therefore, this hypothesis was not supported. Further, when reported PTS symptoms were included in the model via post-hoc analyses, these results were still non-significant. These findings were inconsistent with existing literature that indicates trauma exposure is associated with decreased PA overall, as well as following exposure to positively-valanced stimuli (Frewen et al., 2012; Spahic-Mihajlovic et al., 2005).

#### **Do Trauma Exposed Individuals Report Greater Dampening and Less Positive Rumination?**

Contrary to hypotheses, trauma exposure alone was not associated with reported dampening at either baseline or following best daily events. When PTS symptoms were included in the model along with trauma exposure, this relationship became significant at baseline only, such that individuals who endorsed both trauma exposure and high PTS symptoms reported greater dampening than those who did not. The presence of PTS symptoms may amplify the relationship between trauma exposure and dampening. Increased PTS in and of itself may also predict increased dampening of PA; research demonstrates that the presence of PTS is related to emotional numbing in response to positive stimuli (Frewen et al., 2012; Litz & Gray, 2002; Spahic-Mihajlovic et al., 2005). For the current study, it may be that the greater reported distress following traumatic exposure (i.e., PTS symptoms), the more likely an individual was to engage in dampening following PA at baseline.

While there was no significant difference between exposure groups on reported dampening at baseline, groups differed significantly on reports of positive rumination, such that individuals who reported trauma exposure endorsed greater positive rumination than their non-exposed counterparts. These results directly contradict our hypothesis that trauma exposed individuals would report less positive rumination, but suggest instead that positive rumination may play an important role in compensating for the negative effects of traumatic exposure on responding to PA. Research demonstrates that trauma exposure can lead to positive outcomes such as PTG, and engagement in positive rumination aimed at magnifying the effects of positive affect and emotions may lead to growth and better overall functioning (Chan et al., 2011; Tedeschi & Calhoun, 2004; Wozniak et al., 2020). It is possible that increased positive rumination allowed these individuals to amplify positive emotions so much so that their

experience of PA did not differ from that of their non-trauma-exposed counterparts. This would be an avenue for further research.

At baseline, the high depression group reported more dampening and positive rumination. Individuals who endorsed high depressive symptoms reported greater dampening in response to positive affect than those in the low symptom group, while individuals who endorsed low depressive symptoms reported greater positive rumination. This finding is consistent with the literature (Feldman et al., 2008; Nelis, Homes, & Raes, 2015). Results of EMA analyses demonstrated that neither depressive symptom group, trauma exposure group, nor the interactions thereof predicted reported dampening or positive rumination following PE, again contrary to our hypothesis.

The overall purpose of the present study was to differentiate the effects of trauma exposure on responding to PA from those of depression. The first hypothesis was not supported, such that trauma exposed individuals did not differ from non-trauma exposed individuals on reported PA both prior to or following PE. While the second hypothesis (i.e., trauma exposed individuals would report greater dampening and less positive rumination than non-exposed) was also not supported, the significant relationship between trauma exposure and reported positive rumination at baseline may have implications for future avenues of research. Further, the significant relationship between trauma exposure/high PTS symptoms and reported dampening at baseline may also warrant future examination. The results associated with depressive symptom groups largely supported existing research, such that high depressive symptoms were associated with greater reported dampening, less reported positive rumination, and decreased reported PA prior to and following positive life events.

## **Clinical Applications**

While hypotheses were unsupported by the results of the current study, current literature links trauma exposure with decreased positive affect overall as well as in response to positive stimuli (Ehring & Quack, 2010; Frewen et al., 2012; O'Bryan et al., 2015; Tull et al., 2007). Further, research indicates that increased dampening and decreased positive rumination are both strongly associated with greater depressive symptoms (Feldman et al., 2008; Nelis et al., 2015); PTS and depressive symptoms are often comorbid, which may be due to shared cognitive vulnerabilities such as engagement in dampening or rumination (Angelakis & Nixon, 2015; Creamer et al., 2001; Kessler et al., 2005a; Michl et al., 2013; Werner-Seidler et al., 2013). Therefore, clinicians should focus on assisting trauma-exposed individuals with the development of cognitive strategies that actively foster and amplify positive affect and emotions in an effort to combat the negative effects of exposure.

Active engagement in strategies aimed at upregulating positive affect and emotions, refocusing perspectives, and assimilating new information about self, world, and others following a traumatic event is associated with better overall functioning (Tedeschi & Calhoun, 2004; Stockton et al., 2011). Cognitive strategies such as deliberate rumination and reflection, positive refocusing, and future planning may lead to growth (Hussain & Bhushan, 2011; Kaye-Tzadok & Davidson-Arad, 2016). It is important when working with trauma exposed individuals to recognize the potential for deficits in PA, and that early implementation of these cognitive strategies promotes resilience and better prognoses overall.

## **Limitations**

There were several limitations in the current study that may have impacted results. First, participants were overwhelmingly Caucasian females, with a mean age of approximately 19, all

of whom were enrolled in university courses. Sample characteristics surely limit the generalizability of the results. Secondly, data were collected from a non-clinical sample. Self-report measures were used to identify PTS and depressive symptom groups but did not provide diagnostic information. Therefore, continued examination of these relationships in a more diverse, clinical sample is warranted.

The way in which trauma exposure was operationalized (i.e., lifetime traumatic events rather than trauma exposure within the last year) may have made traumatic exposure less salient regarding its impact on current cognitive and emotional functioning. For example, O'Donnell, Creamer, and Patterson (2004) found that early symptoms of depression following a trauma represented a distinct entity, with unique predictors and course for recovery. As overall psychological distress became more chronic over the subsequent 12 months, the distinction between PTS and depressive symptoms became almost indistinguishable. These results indicate that examination of the effects of traumatic exposure months or even years following the event or events likely inhibited our ability to identify unique effects of traumatic exposure on responding.

### **Future Directions**

Future research may build upon the present study and its limitations by first addressing the issue of generalizability. Examination of a more diverse sample of clinically relevant participants may yield different results than those of the current study, and provide a more accurate picture of the impact of trauma. A more precise definition of traumatic exposure, such as one that is more temporally-yoked, would also be appropriate. Given the findings of the O'Donnell et al. study (2004), this would be an important step toward potentially identifying unique trajectories of depression and sequelae of trauma exposure. Results of the current study



suggested the potential for moderative effects of cognitive strategies on the relationship between trauma exposure and responding to PA and PE. Moving forward, this would be an important avenue to pursue.

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