Psychotherapy Processes in PTSD Treatment: Trajectories of Positive and Negative Valence Systems

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Abstract

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Despite strong empirical support for the effectiveness of existing exposure-based and pharmacological treatments for trauma-related psychopathology (Cusack et al., 2016; Jeffreys et al., 2012; Sakaluk et al., 2019), far less is known about the processes driving clinical change. Existing research points to change processes involving both positively and negatively valenced affect, cognitions, and processing styles; yet past studies have been limited by overreliance on self-report measures, a dearth of longitudinal studies examining session-to-session change, and failures to integrate cognitive, affective, and processing variables in computational modeling approaches designed to study change. Although several studies have documented the role of decreases in negative processes in promoting recovery from trauma-related psychopathology (e.g., Cooper, Clifton, et al., 2017), inhibitory learning models of extinction processes during

exposure therapy increasingly point towards the importance of cultivating increases in positive processes as well (Craske et al., 2016, 2019; Zbozinek & Craske, 2017). In the present study, patients (N = 149) with primary PTSD enrolled in a clinical trial of prolonged exposure (PE) delivered alone or augmented with sertraline completed self-report measures of positive and negative affect (PA and NA) across ten sessions of treatment. Trained coders rated cognitive, affective, and processing psychotherapy change processes during psychotherapy sessions, coding both patient statements and behaviors at the first session and subsequent sessions after imaginal exposure to the trauma memory. Utilizing dynamic structural equation modeling, Study 1 examined temporal patterns of change in self-reported positive and negative affect and PTSD symptoms over treatment, and Study 2 examined patterns of change in positive and negative systems of psychotherapy process change processes and their relation to PTSD symptoms from session to session. In Study 1, positive affect increased moderately (d = 0.51) and NA decreased strongly (d = 0.78) across treatment sessions. Changes in PA and NA were generally reciprocal ($PA_t \rightarrow NA_{t+1}$: ES = -0.09, 95%CI = -0.15, -0.02; $NA_t \rightarrow PA_{t+1}$: ES = -0.20, 95%CI = -0.28, -0.13). However, changes in PTSD more strongly predicted next session negative affect ($PTSD_t \rightarrow NA_{t+1}$: ES = 0.50, 95%CI = 0.38, 0.60) and positive affect ($PTSD_t \rightarrow PA_{t+1}$: ES = -0.26, 95%CI= -0.34, -0.17) than the reverse. PE augmentation with an SSRI did not moderate temporal associations. In Study 2, positive system activation increased strongly (d = 1.42) and negative system activation decreased strongly (d = 0.98) over treatment sessions. Changes in positive and negative systems were reciprocal, with stronger effects of positive system activation on subsequent negative system activation than vice versa (positive $system_t \rightarrow$ negative $system_{t+1}$: ES = -0.25, 95% CI [-0.38, -0.11]); negative $system_t \rightarrow$ positive $system_{t+1}$: ES = -0.09, 95% CI [-0.16, -0.004]), and the effect of positive system changes on negative system changes was stronger with sertraline

augmentation (interaction ES = -0.42, 95% CI [-0.80, -0.10]). These findings were the opposite of the patterns observed for self-reported affect and PTSD. Decreases in PTSD symptoms predicted subsequent increases in positive ($PTSD_t \rightarrow$ positive $system_{t+1}$: ES = -0.44, 95% CI [-0.55, -0.37]) and decreases negative system activation ($PTSD_t \rightarrow$ negative $system_{t+1}$: ES = 0.38, 95% CI [0.26, 0.52]), respectively, but system changes did not predict subsequent changes in PTSD symptoms. Across studies, prolonged exposure produced substantial improvements in self-reported PA and NA as well as positive and negative emotions, cognitions, and processing styles during imaginal exposure processing. General affective changes may be more a consequence than a driver of PTSD improvement during PE, with improvements in NA and PA potentially linked to the extinction of negative emotional responses to trauma cues and increased engagement with rewarding activities, respectively. Increases in positive psychotherapy processes may be an especially robust indicator of improvement in treatment and may help patients overcome problems in psychotherapy indexed by increases in negative psychotherapy processes. The impacts of positive psychotherapy processes may be more pronounced with medication augmentation of psychotherapy.

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Psychotherapy Processes in PTSD Treatment: Trajectories of Positive and Negative Valence Systems

Exposure to traumatic events is nearly ubiquitous, and trauma-related psychopathology is both common and debilitating, with U.S. national lifetime prevalence estimates for posttraumatic stress disorder (PTSD) ranging from 6.1-8.3% (Goldstein et al., 2016; Kessler et al., 2012; Kilpatrick et al., 2013). Trauma-related psychopathology is associated with broad functional impairments, and approximately six in ten individuals with lifetime presence of PTSD receive mental health treatment (Goldstein et al., 2016). There is strong empirical support for both trauma-focused psychotherapies and selective serotonin reuptake inhibitors for the treatment of PTSD. Recent clinical practice guidelines for treating PTSD strongly recommend trauma-focused cognitive behavioral therapies (CBTs), including exposure-based therapies such as prolonged exposure (PE; Foa et al., 2007), as first-line treatments (American Psychological Association, 2019; Benedek et al., 2009), and considering SSRIs for patients with a preference for medication treatment (National Institute for Health and Care Excellence, 2018). Indeed, meta-analyses suggest large treatment effects for PE and other forms of trauma-focused therapies for PTSD (Cusack et al., 2016; Sakaluk et al., 2019; Watts et al., 2013).

Despite a wealth of evidence supporting the effectiveness of trauma-focused therapies for PTSD, the literature examining therapeutic change processes is less developed. Understanding how evidence-based treatments work – that is, elucidating processes of change in psychotherapy and their driving mechanisms – is critical to optimizing treatment effectiveness, addressing factors responsible for non-response to treatment, and overcoming obstacles to treatment dissemination and engagement (Kazdin, 2007; Miller, 2010). Knowledge of treatment

mechanisms can also facilitate cultural adaptations of existing treatments and new treatment development (e.g., Craske et al., 2016; Zoellner et al., 2018).

Exposure-Based Models of PTSD Treatment

Fear conditioning and extinction processes are thought to contribute substantially to the etiology and treatment of anxiety and traumatic-stressor disorders (Bouton, 2000; Bouton & Nelson, 1998; Craske et al., 2012; Grillon, 2008). For instance, a sexual assault survivor may encode associations between the assault itself, an unconditioned stimulus (US), with other, previously neutral stimuli, a conditioned stimulus (CS), present at or related to the assault, such as physical characteristics of the perpetrator (e.g., facial hair) or the location and circumstances of the assault (e.g., alone in a dorm room). After the assault, encountering a CS in the absence of the US may trigger a fear response by virtue of the CS's conditioned association with the US. Accordingly, treatments often aim to reduce conditioned fear responses, known as fear extinction, by presenting CS's repeatedly in the absence of the US, leading to declines in fear responses over time. Exposure therapies such as PE are often conceptualized as promoting fear extinction, as clients are repeatedly exposed to conditioned fear cues in order to promote new learning that attenuates fear responding (Craske et al., 2014; Gillihan & Foa, 2011).

A prominent model of anxiety disorders and the mechanisms underlying recovery, emotional processing theory (Foa et al., 2006; Foa & Kozak, 1986; Foa & McNally, 1996), conceptualizes fear as represented by network structures in memory that function as programs to escape or avoid danger. The model suggests that anxiety and traumatic-stress related psychopathology stem from pathological fear structures within memory (Lang, 1979), in which representations of feared stimuli (e.g., honking car horn), fear responses (e.g., racing heart; subjective fear), and their meaning (e.g., "I'm going to be in a car crash") are exaggerated or

inaccurate, leading to excessive fear responding. Exposure to trauma-related, fear CSs in the absence of aversive outcomes, together with emotional processing, in which the fear memory structure is activated and then modified by information inconsistent with the fear memory, leads to increasingly flexible, accurate, and adaptive fear structures (Foa & Kozak, 1986). While originally, emotional processing theory suggested that the elements and associations within fear structures are updated or re-written via exposure and emotional processing (Foa & Kozak, 1986), updates to the theory have increasingly emphasized the creation of new, non-fear associations that compete with the old fear associations, consistent with inhibitory learning models (e.g., Foa et al., 2006; Foa & McNally, 1996).

The inhibitory learning model of exposure-based psychotherapy complements emotional processing theory and makes unique predictions about the factors that lead to fear extinction (Bouton, 2004; Craske et al., 2008, 2014). According to the inhibitory learning model, the original, fear-excitatory CS-US association acquired during fear conditioning (e.g., during a traumatic event) remains intact, while secondary, fear-inhibiting CS-US associations are formed as the CS is encountered without the US. Thus, inhibitory learning involves learning that the CS no longer predicts the occurrence of the US (Bouton, 1993, 2004; Craske et al., 2014). Over repeated successful exposure to CSs where the US does not occur, the secondary, inhibitory CS-US association ambiguates the meaning of the CS, leading to decreased fear responding. Context serves to disambiguate the meaning of the CS. However, conditioned fear may be renewed as time passes since the end of extinction, if the US re-occurs, or if the CS is encountered in a different context from where extinction occurred. Inhibitory learning models are consistent with findings from both rodent and human studies demonstrating the role in fear extinction of the

ventromedial prefrontal cortex inhibiting threat responses from the amygdala (Kredlow et al., 2022; Milad et al., 2009; Quirk et al., 2007; Shin & Liberzon, 2010).

Processes of Change in PTSD Treatment

Both emotional processing theory and the inhibitory learning model suggest that changes in negative affective and cognitive processes such as fear and trauma-related beliefs over the course of exposure therapy serve as indicators of emotional processing or inhibitory learning, leading to reductions in psychopathology (Craske et al., 2012; Foa et al., 2006). Although most studies of emotional processing and inhibitory learning in exposure therapy have focused on changes in these negative processes, developments in the inhibitory learning literature suggest that increases in positive affective and related cognitive processes may facilitate recovery as well (e.g., Zbozinek & Craske, 2017). Accounting for both positive and negative change processes in exposure therapy may provide a more comprehensive picture of recovery during treatment for trauma-related psychopathology. Additionally, PTSD is often treated with SSRI medication, either alone or in combination with psychotherapy (Bandelow et al., 2012), highlighting the importance of understanding shared and distinct change processes across psycho- and pharmacotherapies.

Processes Involving Negative Affect and Cognitions

Anxiety and traumatic-stressor disorders may develop in part due to deficits in extinction or inhibitory learning (e.g., Craske et al., 2012; Indovina et al., 2011; Jovanovic et al., 2010), and thus treatment strategies that enhance inhibitory learning are thought to potentially facilitate recovery. Both emotional processing theory and inhibitory learning models suggest that emotional engagement, or fear activation, during exposure to the CS is critical for new learning to occur (Craske et al., 2008; Foa et al., 2006; Foa & McNally, 1996). Similarly, both emphasize

expectancy violation, whereby expectations of aversive events during exposure are not born out, and the importance of blocking avoidance of the CS during exposure, which are thought to interfere with new extinction learning. However, emotional processing theory and inhibitory learning diverge with respect to the role and nature of extinction, or reductions in fear within and across exposure trials. Originally, emotional processing theory maintained that reductions in fear during a given exposure trial (within-session extinction) and between exposure trials (between-session extinction) are necessary to promote extinction learning (Foa & Kozak, 1986; Foa & McNally, 1996); updates to emotional processing have de-emphasized the importance of within-session extinction in accordance with empirical findings (Brown et al., 2019). Within-session extinction of the fear response provides information incompatible with the fear structure, forming the basis for between-session extinction, which is thought to promote changes in meaning elements in the form of lowered expectancies of aversive outcomes (Craske et al., 2012). The inhibitory learning model posit that fear expression during extinction trials is a poor index of inhibitory learning, as inhibitory associations are learned independently of expressed fear during extinction trials. Rather, inhibitory learning is thought to be more dependent on context and time (Craske et al., 2008, 2012). Craske and colleagues (2012) have suggested that inhibitory learning is better indexed by independent outcome measures (e.g., symptom severity) obtained post-extinction.

Earlier iterations of emotional processing theory have typically referred to within- and between- session reductions in fear using the clinical term habituation, rather than extinction, and many studies of emotional processing theories mirror this terminology (Foa & Kozak, 1986; Foa & McNally, 1996). However, in the broader behavioral neuroscience field habituation refers to decreases in responding that likely does not involve learning (Furlong et al., 2016), whereas in

the exposure therapy literature typically habituation refers to decreases in conditioned responses that involve extinction learning (Rauch & Foa, 2006). Thus, this paper will refer to within- and between-session extinction, rather than habituation. Below, evidence for change processes tied to extinction learning during exposure treatment for PTSD is reviewed, followed by evidence for processes implicated more broadly by emotional processing and inhibitory learning models of treatment for trauma-related psychopathology.

Emotional Activation. There is mixed but generally strong evidence arguing that emotional engagement, or the activation of fear, anxiety, or distress during exposure trials, facilitates extinction learning during exposure therapy (Cooper, Clifton, et al., 2017). Emotional engagement has typically been measured subjectively via the Subjective Units of Distress scale (SUDs; Wolpe & Lazarus, 1966) administered throughout exposure exercises, physiologically via heart rate or skin conductance response (e.g., Pitman et al., 1996), and, rarely, behaviorally via facial fear ratings (Foa et al., 1995). For example, Foa and colleagues (1995) found that observer-rated facial fear (r = .78) and peak SUDs (r = .71) during imaginal exposure were associated with greater reductions in post-treatment PTSD symptoms, and Pitman and colleagues (1996) found that change in resting-to-peak heart rate (r = .70), but not skin conductance, was associated with fewer post-treatment re-experiencing symptoms. Using a subjective measure of emotional engagement, Rauch and colleagues (2004) found that post-treatment PTSD symptoms were associated with peak SUDs during the final imaginal exposure (r = .48), but not the first imaginal exposure in a trial of PE with or without cognitive restructuring. Harned and colleagues (2015) failed to find a relationship between pre to peak SUDs change averaged across sessions and loss of PTSD diagnosis post-treatment. Overall, studies have generally found moderate to strong relationships between greater emotional engagement during exposure and favorable

changes in PTSD post-treatment, but findings vary across measures of emotional engagement and the session in which engagement was assessed. Notably, these studies largely relied on subjective, self-report measures of distress and tended to utilize a single session of exposure to predict post-treatment outcomes, as opposed to examining the role of changes in emotional engagement from session to session, which may provide a more nuanced view of the dynamics of emotional engagement and symptom change over time.

Within-Session Extinction. Despite emotional processing theory's early assertion that within-session extinction is a necessary precursor of between-session extinction and subsequent changes in meaning and PTSD symptoms (Foa & Kozak, 1986; Foa & McNally, 1996), there is little evidence that within-session extinction is necessary for therapeutic change (Craske et al., 2008). Some studies have found positive, marginal trends for within-session extinction of heart rate during PE correlating with improvements in PTSD (e.g., Pitman et al., 1996), and several studies have failed to find a significant relationship between within-session extinction during the first imaginal exposure and treatment outcomes (Nacasch et al., 2015; Pitman et al., 1996; van Minnen & Foa, 2006; van Minnen & Hagenaars, 2002). However, a more recent study utilizing a mixed modeling approach found that greater within-session extinction during imaginal exposure was strongly associated with better symptom improvement at the next session and with superior treatment response (de Kleine et al., 2015). Nonetheless, most studies failed to find a significant relationship between within-session extinction and treatment outcomes.

Between-Session Extinction. Unlike within-session extinction, several studies point to the role of between-session extinction in therapeutic changes in anxiety and PTSD (Cooper, Clifton, et al., 2017). Several studies have found that between-session extinction, operationalized as the difference between peak or mean SUDs from the first and last exposures, was moderately

associated with PE outcomes (Gallagher & Resick, 2012; Harned et al., 2015; Nacasch et al., 2015; Rauch et al., 2004; van Minnen & Foa, 2006). Mean between-session change in peak SUDs was related to larger change over time and lower post-treatment PTSD (de Kleine et al., 2015; Rothbaum et al., 2014). However, Bluett et al. (2014) found that only approximately 35% of individuals experienced a reliable change in mean and peak SUDs from the first to final PE session; and while those with reliable change in SUDs had lower PTSD and depression at post-treatment, there were no differences in post-treatment PTSD diagnostic status between those who demonstrated reliable change and those who did not. These authors suggested that between-session extinction may not be a necessary ingredient for recovery and, in the absence of between-session extinction, increases in distress tolerance may mediate symptom improvement. In a review of mechanisms of change in PE, Cooper et al. (2017) noted that the observed relationships between between-session extinction and treatment outcomes may depend on the time in treatment at which it is assessed, as well as on the measurement modality. This points to the importance of repeated, multi-modal assessments of change processes over the course of treatment.

Negative Affect. Hallmark symptoms of PTSD include negative emotional responses to trauma cues as well as persistent negative emotional states, including fear, guilt, shame, and anger (American Psychiatric Association, 2013). Fear and anxiety are central to emotional processing and inhibitory learning models of exposure therapy (Craske et al., 2012; Foa et al., 2006) and are reflected in studies of fear activation and within- and between-session extinction. PTSD is associated with broader elevations in negative affect (NA; Badour et al., 2017), and several studies have examined the role of anger in PTSD treatment, with some studies finding that CBTs for PTSD strongly decreased anger over the course of treatment (e.g., Cahill et al.,

2003; Stapleton et al., 2006), and others finding that higher pre-treatment anger weakly predicted poorer PTSD outcomes (e.g., Foa et al., 1995; Forbes et al., 2008). Despite persistent negative emotional states being added as a PTSD symptom in the DSM-5 (American Psychiatric Association, 2013), there is a dearth of studies examining changes in NA over the course of treatment (e.g., Jerud et al., 2014) and the extent to which NA predicts PTSD outcomes.

Avoidance. Efforts to avoid stimuli that cue NA and internal experiences of distress are often implicated in the persistence of fear, as avoiding feared but objectively safe stimuli prevents opportunities for new extinction learning (e.g., Cooper, Clifton, et al., 2017; Craske et al., 2012; Pittig et al., 2018). Avoidance is thought to contribute directly to functional impairment by interfering with engagement in activities such as work, socializing, and ordinary tasks involved in basic functioning that are perceived as dangerous or threatening. In addition to avoidance of external stimuli, experiential avoidance of internal stimuli such as thoughts, emotions, memories, and physical sensations can interfere with functioning and wellbeing (Hayes et al., 1996) and with adaptive processing of emotional experiences, instead facilitating maladaptive processing styles such as worry or rumination (Brewin et al., 1996; Reynolds & Brewin, 1999). Avoidance of potentially rewarding activities, for instance an assault survivor who avoids getting together with friends for fear of being harmed, may increase risk for co-occurring depression symptoms by limiting positive experiences and blocking potential reinforcement of non-depressed behavior (Trew, 2011). Indeed, exposure therapies directly targeting avoidance of feared but safe stimuli are effective for anxiety and traumatic stressor-related psychopathology (e.g., Craske et al., 2014; Foa & McLean, 2016; Norton & Price, 2007; Rauch et al., 2012; Tolin, 2010) and depression (Grosse Holtforth et al., 2012; Hayes et al.,

2005; Hayes, Feldman, Beevers, et al., 2007), and behavioral activation therapy for depression directly targets behavioral avoidance to reduce depression symptoms (Martell et al., 2001).

Although there is a large body of evidence supporting the efficacy of exposure for anxiety and depression, few studies have explicitly examined whether changes in avoidance over the course of treatment mediate changes in trauma-related psychopathology. In one such study, Badour et al. (2012) found that higher levels of avoidant coping at intake predicted more severe PTSD at discharge from residential PTSD treatment, which in turn predicted higher avoidant coping at four-month follow-up. Although looking at depression and not PTSD, Hayes and colleagues (2005) used an in-session psychotherapy coding system called CHANGE (Hayes, Feldman, & Goldfried, 2007) to examine putative change processes in weekly essays about depression written by participants in a trial of an integrative, exposure-based therapy for depression. Peak levels of avoidance in the essays were associated with less improvement in depression and with more hopelessness and negative views of self, suggesting that higher levels of avoidance may inhibit therapeutic change.

Cognitive-Emotional Processing and Meaning Making. Although higher levels of avoidance may interfere with recovery processes in therapy, deeper and more adaptive forms of therapeutic processing may facilitate change. Indeed, the same study above (Hayes et al., 2005) also found that higher peak levels of observer-rated cognitive-emotional processing during exposure therapy, defined as exploring and questioning depression-related material with some insight or perspective shift, were associated with more improvement in depression symptoms and with increased expression of hope and positive view of self. There are strong conceptual parallels between cognitive-emotional processing and emotional processing as described in emotional processing theory applied to trauma-related psychopathology, which posits that emotional

processing drives the formation of adaptive fear structures, facilitating recovery (Foa & McLean, 2016).

Cognitive-emotional processing can involve attempts to make meaning of highly stressful events. There is a robust theoretical base for the processes and products of meaning-making in adjustment to stressful life events (e.g., Bonanno & Kaltman, 1999; Davis et al., 2000; Janoff-Bulman, 1992; Joseph & Linley, 2005; Lepore & Helgeson, 1998; Neimeyer, 2001; Taylor, 1983). As elaborated by Park (2010), despite a range of theoretical perspectives on the particulars of meaning-making processes and products, most agree that stressful or traumatic events can disrupt previously held global meaning structures, leading to processes of meaning-making that attempt to reconcile the discrepancy between previously held global meanings and the stressful event and its sequelae. When new meaning is made, meaning-making leads to better adjustment. Although attempts to make meaning are thought to be near-universal following highly stressful or traumatic events (e.g., Davis et al., 2000; Kernan & Lepore, 2009; Silver et al., 1983), meaning-making processes may not always lead to meanings made (e.g., Lehman et al., 1987; Updegraff et al., 2008), or meanings made may turn out to be maladaptive and increase, rather than decrease distress (e.g., Ehlers & Clark, 2000).

Cognitive-emotional processing may be viewed as a meaning-making process (Park, 2010), with theorists placing various degrees of emphasis on the cognitive and emotional aspects. Cognitive processing may involve the reconciliation of experiential data derived from a stressful event with pre-existing schemas about oneself, others, and the world, resulting in changes in beliefs related to the stressor (e.g., Creamer et al., 1992; Creswell et al., 2007; Foa et al., 1999; Janoff-Bulman, 1992; Williams et al., 2002). For example, following a sexual assault by a close acquaintance, the person who was assaulted may engage in a process of reconciling

previously held beliefs about the relative safety and danger of other people and her own perceived ability to detect dangerous individuals with the horror and unexpectedness of the assault. Emotional processing may involve experiencing and exploring one's emotions related to a stressor, involving affect regulation processes and attempts to understand what one is feeling (Ehlers & Clark, 2006; Foa et al., 2006; Rachman, 1980; Stanton et al., 2000). Evidence from expressive writing studies suggest that both cognitive and emotional aspects of processing facilitate meaning making (e.g., Hunt et al., 2007; Sloan et al., 2007), leading Hayes and colleagues (2007) to propose a unified cognitive-emotional processing construct involving exploring distressing material in a way that leads to new insights or perspective shifts.

Negative Trauma-Related Cognitions. Individuals with PTSD commonly report negative beliefs about oneself, others, and the world following a trauma, and the severity of these beliefs is associated with PTSD severity and has been proposed as a psychopathological mechanism underlying chronic PTSD (Ehlers & Clark, 2000; Foa et al., 1999; Resick et al., 2008). Changes in trauma-related beliefs have been shown to predict changes in PTSD symptoms across both cognitive and exposure-based treatments (e.g., Cooper, Clifton, et al., 2017; Foa & Rauch, 2004; Hagenaars et al., 2010; Kumpula et al., 2017; McLean et al., 2015; Nacasch et al., 2015; Zalta et al., 2014). Several of these studies (Cooper, Clifton, et al., 2017; Øktedalen et al., 2015; Zalta et al., 2014) found that decreases in trauma-related negative beliefs preceded subsequent decreases in PTSD symptoms, but not vice versa, while others found reciprocal relationships between trauma-related beliefs and PTSD symptoms over time (Kumpula et al., 2017; McLean et al., 2015), suggesting that cognitive change plays an important role in treatment for PTSD. However, these studies examined the end products of meaning-making processes – belief change – but not the meaning-making processes themselves, leaving

open the possibility that belief change is a marker, but not driver, of therapeutic change, consistent with the bidirectional temporal associations above. Although Hayes et al. (2007) highlighted the therapeutic role of cognitive-emotional processing in the effects of an exposure-based treatment for depression, this construct has yet to be studied in PTSD treatment, leaving a major gap in the literature characterizing how cognitive change occurs. Indeed, increased knowledge of the processes that promote adaptive meaning-making in individuals with PTSD may ultimately prove most useful to clinicians, who can promote those processes in clients to facilitate cognitive change.

Processes Involving Positive Affect and Cognitions

Positive Affect and Reward System Functioning. Much of the literature on processes of change in treatments for trauma-related psychopathology emphasize decreases in processes linked to NA and cognitions, including fear activation, fear extinction, and trauma-related beliefs. However, increased attention has begun to be paid to the role of positive affect (PA) and reward system functioning in the etiology and treatment of anxiety and traumatic stressor-related disorders. PTSD is associated with deficits in PA and reward system function (Nawijn et al., 2015), and between half to three-fourths of individuals with PTSD report anhedonia, even after controlling for co-occurring depression (Carmassi et al., 2014; Franklin & Zimmerman, 2001). Beckham et al. (2000) tracked individuals with and without PTSD every 30 min over a 14-hour period, finding that those with PTSD reported lower levels of positive affect (PA) than those without PTSD. Further, PTSD has been characterized as disrupted homeostasis of fear and reward systems (Stein & Paulus, 2009), with heightened avoidance associated with limbic hyperactivity, heightened anhedonia associated with striatal hypoactivity, and deficits in the integration of approach and avoidance associated with orbitofrontal cortex hypoactivity (e.g.,

Aupperle & Paulus, 2010; Nawijn et al., 2015). Excessive negative NA-driven avoidance and deficits in reward-driven approach may interact on psychological and behavioral levels as well via mechanisms of negative affect interference, whereby trauma-related NA pre-empts or intrudes upon otherwise rewarding experiences (DePierro et al., 2018), or via excessive NA-driven consumption of cognitive resources and attentional bias towards threat and away from reward (Litz & Gray, 2002). Individuals with PTSD may preferentially attend to and process trauma and threat-related stimuli, utilizing cognitive resources that could otherwise be used to process positive events. Thus, decreases in NA may facilitate subsequent increases in PA. Indeed, the short-term emotion dynamics literature suggests that PA and NA may dampen one another over time, with 1.5 to three times the level of PA required to exert a comparable effect on subsequent NA compared to NA on subsequent PA (Garland et al., 2010; Hollenstein, 2015; Husen et al., 2016; Kuppens & Verduyn, 2017).

Although excessive NA-linked processes and insufficient reward-driven processes may contribute to chronic PTSD, improvements in reward processing may facilitate recovery from PTSD. Increases in PA may enhance extinction learning and generalization during exposure therapy (Zbozinek & Craske, 2017), and treatment targeting PA in individuals with anxiety or depression resulted in improvements in PA, NA, and anxiety (Craske et al., 2019, 2023). One study of changes in emotion regulation and trait affect over the course of PTSD treatment with either PE or sertraline found that both treatments resulted in improvements in both NA and PA (Jerud et al., 2014). Additionally, stronger suppression of NA in the hours following increases in PA was found to predict response to depression treatment (Wichers et al., 2012). However, additional research is needed to clarify the temporal relationships among NA, PA, and PTSD symptoms over the course of treatment.

Positive Cognitions. In addition to positive affect, there is evidence that improvements in positive cognition may facilitate recovery from PTSD. Higher levels of hope pre-treatment have been associated with better treatment gains in psychotherapy (Cheavens et al., 2006; Geraghty et al., 2010), and Gilman and colleagues (2012) found that higher levels of hope at mid-treatment predicted mid-to-post treatment improvement in PTSD and depression symptoms during cognitive processing therapy, suggesting that increases in hope may be a mechanism of change, particularly in the latter stages of PTSD treatment. Furthermore, the social cognitive model of PTSD (Benight & Bandura, 2004) posits that increases in perceived coping self-efficacy mediate posttraumatic recovery. In this model, facilitating experiences of mastery in the selection and use of coping strategies disconfirms unrealistic fears and distorted beliefs and bolsters the belief that the individual can exercise control over what is feared. Self-efficacy is thought to be fostered via *in vivo* mastery of trauma-related situations activities and through imaginal exposure to trauma memories, as is done in PE, suggesting a potential mechanistic role for positive cognitions related to perceived self-efficacy in trauma-related situations (Benight & Bandura, 2004). This model also points to social support as an important recovery factor, as positive relationships with others may provide opportunities to learn effective coping skills and increase motivation to engage in beneficial activities (Benight & Bandura, 2004). Indeed, both PE and sertraline for PTSD have been shown to produce large and durable improvements in social functioning (Graham et al., 2020). In sum, positive cognitions related to hope, self and self-efficacy, and others may facilitate beneficial change in treatment for PTSD.

Conceptualizations of Positive and Negative Valence Systems in Psychotherapy

The National Institute of Mental Health's Research Domain Criteria (RDoC) identifies positive and negative valence systems as two organizing domains for constructs involved in

psychopathology research (Kozak & Cuthbert, 2016). Negative valence constructs include responses to acute, potential, and sustained threat, as well as frustrative nonreward and loss. These constructs involve the activation of defensive motivational systems that protect against harm or loss. Positive valence constructs include appetitive motivational systems and reward functioning (Kozak & Cuthbert, 2016).

Although first-line psychotherapeutic treatments for PTSD have traditionally emphasized targeting and decreasing negative valence processes, a growing body of research recognizes the potential mechanistic role of increases in positive processes. Though novel treatments (e.g., Craske et al., 2019) seek to test the impact of directly targeting positive processes, there is evidence that even treatments that predominantly target negative processes, such as PE, result in increases in positive processes. In line with conceptualizations of PTSD as characterized by an imbalance of approach and avoidance (Stein & Paulus, 2009), dynamic systems and network models of psychopathology (e.g., Cramer et al., 2016; Fried et al., 2016; Hayes et al., 2015; McNally, 2016) suggest that psychotherapy works by destabilizing pathological systems and building and strengthening adaptive systems (e.g., Hayes et al., 2015). In this context, recovery may be facilitated by transitions from unproductive processing, such as avoidance, that maintain and strengthen psychopathological negative systems, to productive cognitive-emotional processing, which helps facilitate adaptive meaning-making and strengthen positive systems (e.g., Hayes et al., 2005, 2015; Hayes, Hope, et al., 2007). Similarly, decreases in negative affect-related processes and increases in positive affect-related processes may be functionally linked, such that decreases in fear-driven avoidance may allow increased reward acquisition and improved reward processing (e.g., Craske et al., 2016; Zbozinek & Craske, 2017).

Alpert and colleagues (Alpert et al., 2021) provide an innovative example of how change processes suggested by emotional processing and inhibitory learning models can be studied through the lens of changes in positive and negative systems. In a study of trauma-focused CBT (TF-CBT) for youth, the authors coded in-session trauma narration and processing phases of TF-CBT for the extent to which positive and negative valence process systems, or networks, were activated across cognitive, emotional, behavioral, and physiological domains, with each domain constituting a node of the positive and negative networks. They found that curvilinear changes in multimodal negative network scores – reflecting a greater number of activated domains – predicted improvement in internalizing symptoms and PTSD symptoms following treatment, while linear increases in multimodal positive networks predicted improvement in externalizing symptoms. These findings are compelling in that they suggest potentially unique roles for changes in both positive and negative networks, each relating to distinct types of outcomes, and to the utility of examining in-session processes and content to better understand the trajectories and outcomes of therapeutic change processes. This approach may yield promising insights if applied to symptom change during treatment, as positive and negative valence systems may play distinct roles. Future studies ought to investigate the degree of dependence between positive and negative systems, and the extent to which their interaction versus independent effects impact trauma-related psychopathology.

Notably, studies of first-line PTSD treatments, such as PE, have yet to elucidate the relative independence or dependence of changes in positive and negative systems, the processes that drive changes in positive and negative systems, and their relative contributions to changes in PTSD symptoms over the course of treatment. Delineating the trajectories and degree of interplay between positive and negative systems during treatment, as well as their influence on

changes in PTSD, may provide valuable insights into how effective psychotherapies work and how they can become better targeted and improved.

Role of SSRI Medications

Cognitive behavioral therapies are recommended as first-line treatments for PTSD (American Psychological Association, 2019), however, psychotropic medications, particularly SSRIs such as sertraline, are also recommended, efficacious, and widely used to treat PTSD (American Psychological Association, 2019; Benedek et al., 2009; Comer & Figgitt, 2000; Williams et al., 2022). Psycho- and pharmacotherapeutic approaches are often combined, though clinical trials of combined psycho- and pharmacotherapy versus monotherapy have generally not found strong evidence for superior effects of pharmacotherapy augmentation (Hetrick et al., 2010). While a trial of PE plus paroxetine versus PE plus placebo found that those in the medication-augmented condition demonstrated significantly greater improvements in PTSD symptoms (OR = 12.6; Schneier et al., 2012), several others did not find significant differences in response for those in combined treatment conditions compared to those receiving psychotherapy alone (Rauch et al., 2019; Simon et al., 2008) or medication alone (Rothbaum et al., 2006).

SSRIs are thought to exert effects through manipulation of serotonin signaling and 5-HT neurons, potentially leading to more flexible responding during conditions of stress, uncertainty, or surprise (Roberts et al., 2020). There is also evidence that SSRIs modulate contextual fear expression and fear extinction (Heesbeen et al., 2023). One model of SSRI therapeutic action suggests both acute and long-term effects on multiple emotional and cognitive processes, including changes in self-referential attention and positive affect (e.g., Di Simplicio et al., 2012; Harmer, 2008). Several authors have highlighted the connections between neurobiological

changes effected by SSRIs and psychological constructs such as emotion regulation, with some evidence that SSRIs alter emotion regulation and repetitive negative thinking (e.g., Feurer et al., 2021; Harmer, 2012; Pringle et al., 2011). It is possible that the combination of psychotherapy and SSRIs may exert a synergistic effect on positive and negative systems and their interplay, where acute decreases in negative affect and negative self-referential processing co-occur with increased positive affect, leading to enhanced symptom reduction.

Evidence is mixed as to whether processes of change may differ in psychotherapy versus pharmacotherapy. Cooper et al. (2017) utilized a time-lagged modeling approach showing that decreases in trauma-related negative cognitions drove subsequent reductions in PTSD symptoms more robustly in PE than in sertraline. However, Allard et al. (2021) found that trauma-related guilt cognitions decreased equally after PE or sertraline, and that reductions in guilt predicted subsequent reductions in PTSD in both treatments. While trauma-related belief change may play a larger role in change processes in PE compared to pharmacotherapy, additional studies are needed to reconcile these mixed findings.

Measuring and Modeling Change Processes

Studies of change processes in treatments for PTSD have disproportionately relied on self-report measures. Although self-report measures are relatively easy to administer and less costly and resource-intensive compared to other measurement modalities, such as clinical interviews and observer-ratings of patient behaviors, they are subject to biases associated with their subjective and often retrospective nature. Although self-report assessments of PTSD symptoms reliably distinguish those with and without PTSD, they can potentially inflate severity scores and underperform in distinguishing PTSD symptoms from those of other mood and anxiety disorders, compared to structured clinical interviews (e.g., Engelhard, Arntz, & van den

Hout, 2007). Observer-ratings of putative change processes as they appear in-session through patient behaviors, as opposed to retrospective reports of change factors by the patients themselves, may offer a more proximal and less biased method of assessing change processes. Studies directly comparing the performance of self-report and observer-rating change may shed light on whether moving beyond self-report measures adds value to the study of change processes and their relation to psychopathology outcomes.

Examining the Role of Post-Exposure Processing

While several putative therapeutic change processes are purported to take place during exposure exercises (e.g., emotional activation; between-session extinction), others such as changes in trauma-related beliefs may be more strongly associated with the processing phase of treatment sessions that follows imaginal exposure. Indeed, during processing, patients can explore their reactions to exposure and the trauma and examine previously held trauma-related beliefs in-depth, facilitating cognitive change (Foa et al., 2019), and post-exposure processing may help elaborate on and consolidate new learning that occurs during exposure (Brown et al., 2019). Critically, however, very few studies have examined factors during post-exposure processing that facilitate therapeutic change in PE (e.g., Cox et al., 2020), meaning that the factors impacting the effectiveness of exposure processing largely remain obscured by a black box. Clarifying factors during processing that contribute to therapeutic change will help guide therapist decision-making during processing.

The Present Study

Models of therapeutic change emphasize both decreasing negative affect, trauma-related cognitions, and unproductive processing (Craske et al., 2014b; Ehlers & Clark, 2000; Foa et al., 2006; Hayes et al., 2005) and, more recently, increasing positive affect, perceived self-efficacy,

increased approach, and cognitive-emotional processing (Benight & Bandura, 2004; Craske et al., 2019; Foa et al., 2006; Hayes et al., 2005). The inhibitory learning model (e.g., Zbozinek & Craske, 2017) posit functional interactions between positive and negative valence processes. Together, several key questions to better understand processes of change in PTSD treatment are suggested by these models. First, how do positive and negative valence systems change over the course of treatment? Second, what is the relationship between changes in positive and negative valence systems? Third, to what extent do changes in negative and positive systems drive changes in PTSD symptoms across treatment? Fourth, how does the presence of an SSRI during exposure therapy differentially impact positive and negative valence system change in recovery?

Most studies of psychotherapy processes rely on self-report measures; these measures are less resource-intensive to implement compared to interview or observer-rated measures and reflect the patient's subjective experience. However, self-report measures are typically completed retrospectively, and observer-ratings of in-session patient behaviors may offer more proximal and less biased assessments of change processes. Further, observer-ratings of in-session behaviors based on therapy tapes can be much more nuanced and in-depth than self-report measures, assessing multiple facets of patient behaviors in the therapeutic context, leading to much richer change process data compared to self-report. The CHANGE coding system assesses a wide range of verbal and nonverbal patient and therapist in-session behaviors across several domains of cognitive and affective functioning (Hayes, Feldman, & Goldfried, 2007). In addition, observer-ratings can be tailored to specific therapeutic procedures or phases, shedding light on the roles of distinct therapeutic activities. The processing phase that follows imaginal exposure in PE, in which the patient and therapist discuss the clients' reactions to revisiting the trauma memory and thoughts and feelings about the trauma and its meaning, is thought to

promote new learning that drives symptom change (Foa et al., 2007). However, imaginal exposure processing is one of the least-studied components of PE, with many studies instead focusing on extinction-related processes measured during the imaginal exposure itself, such as fear activation and within- and between-session extinction (Cooper, Clifton, et al., 2017). Accordingly, the present study sought to elucidate change processes that occur during imaginal exposure processing using both self-report and observer-rated measures of change.

Changes in Self-Reported Positive and Negative Affect (Study 1) examined: (a) changes in self-reported PA and NA over the course of PE or PE plus sertraline for the treatment of PTSD; (b) whether within-person changes in NA preceded changes in PA or vice versa; and (c) whether within-person changes in PA and NA preceded changes in self-reported PTSD symptoms. It was hypothesized that NA would decrease and PA would increase linearly in both treatments (Sripada & Rauch, 2015; Zoellner et al., 2022), with potential augmentation effects of sertraline resulting in steeper rates of change in PE plus sertraline (e.g., Rauch et al., 2018; Schneier et al., 2012). Concerning temporal relationships between PA and NA, it was expected that decreases in NA would predict next-session increases in PA more strongly than vice versa. It was predicted that there would be reciprocal temporal relationships between increases in PA/decreases in NA and decreases in next-session PTSD symptoms. It was expected that session-to-session relationships between changes in affect and PTSD symptoms would be stronger in PE plus sertraline, given potential augmentation effects of the SSRI.

Changes in Observer-Rated Psychotherapy Process Positive and Negative Systems (Study 2**)** utilized in-depth psychotherapy coding using observer ratings of in-session change processes (CHANGE; Hayes, Feldman, & Goldfried, 2007) during the processing of imaginal exposure to examine: (a) changes in positive and negative psychotherapy change processes

during imaginal exposure processing over the course of PE or PE plus sertraline for the treatment of PTSD; (b) whether changes in positive change processes preceded changes in negative schange processes or vice versa; and (c) whether changes in positive and negative change processes preceded changes in self-reported PTSD symptoms. Positive and negative valence psychotherapeutic processes were conceptualized as systems comprised of cognitive, affective, and processing elements. Similarly to Study 1, it was hypothesized that positive and negative system activation during processing would linearly increase and decrease, respectively, over the course of treatment, with SSRI augmentation of PE producing steeper rates of change. With respect to temporal patterns of change in positive and negative system activation during processing, it was expected that decreases in negative system activation would predict next-session increases in positive system activation more strongly than vice versa. It was predicted there would be reciprocal temporal relationships between increases in positive system activation/decreases in negative system activation and decreases in next-session PTSD symptoms. Finally, it was predicted that session-to-session relationships between changes in positive and negative system activation and PTSD symptoms would be stronger in PE plus sertraline, given potential augmentation effects of the SSRI.

Method

Participants

Participants were 149 adults recruited at two urban universities through community advertising and referrals for a doubly randomized preference trial (NCT01600456) for adults (age 18-65 years) with PTSD, with participants first randomized to choice or no choice of treatment, and those in the no choice condition subsequently randomized to PE alone or augmented with sertraline. Inclusion criteria included a primary diagnosis of PTSD based on

DSM-IV criteria, with a minimum duration of 12 weeks since the traumatic event. Exclusion criteria included: a current diagnosis of schizophrenia or delusional disorder; medically unstable bipolar disorder, depression with psychotic features, or depression severe enough to require immediate psychiatric treatment (e.g., actively suicidal); severe self-injurious behavior or suicide attempt within the previous three months; no clear trauma memory or trauma before age of three; current diagnosis of alcohol or substance dependence within the previous three months; ongoing intimate relationship with the perpetrator (if trauma was an assault); unwilling or medically not advisable to stop current CBT or antidepressant medication, based on condition assignment; previous non-response to adequate trial of either PE or sertraline; medical contraindication for the initiation of sertraline (e.g., pregnancy/lactation); current high dose use of benzodiazepines; or sexually active female without acceptable birth control.

Given the focus of this study on treatment-related processes, participants with fewer than three time points of data were excluded from analyses in both Studies 1 and 2 to ensure participants had received a meaningful dose of treatment (DeRubeis, Gelfand, et al., 2014; Yang & Maxwell, 2014). In Study 1, 19 participants were excluded due to less than three data points of self-reported affect and/or PTSD symptoms. Among included participants (n = 130), 64.6% (n = 84) were missing one or fewer sessions (mean missing = 1.92; median = 0). There was no difference in baseline symptoms, PA, or NA between participants who were included and excluded from the sample. In Study 2, 38 participants were excluded due to less than three data points of self-reported PTSD symptoms or observer-rated, in-session therapeutic process variable scores. Among included participants (n = 111), 72.1% (n = 80) were missing one or fewer sessions (mean missing = 0.84; median = 0), and there were no differences in baseline PTSD symptoms, positive system activation, or negative system activation between participants

included and excluded from the sample. Sample characteristics for participants in Studies 1 and 2 are presented in Tables 1 and 2, respectively.

Measures

PTSD Symptom Scale – Interview for DSM-5 (PSS-I-5; Foa et al., 2016)

The PSS-I-5 consists of 20 items corresponding to the PTSD symptoms in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed; American Psychiatric Association, 2013) and was used to assess PTSD symptom presence and severity. Items were rated by independent evaluators on a four-point Likert scale according to the frequency and severity of symptoms over the past two weeks, ranging from 0 (*not at all*) to 3 (*5 or more times per week/very much*), yielding a total severity score and diagnosis. The PSS-I-5 has demonstrated high internal consistency ((α = .89) and high convergent validity (all *r*s > .72) with other well-validated measures of PTSD severity (Foa et al., 2016). Ten percent of cases were re-rated for interrater reliability on the PSS-I-5, with good inter-rater reliability for DSM-IV (17 items; ICC = .89) and DSM-5 (20 items; ICC = .83).

PTSD Symptom Scale – Self Report (PSS-SR; Foa et al., 1997)

The PSS-SR is a 17-item self-report instrument that assesses DSM-IV PTSD criteria A-F. Symptoms are rated from 0 (*not at all*) to 3 (*5 or more times per week/very much*) with respect to the primary trauma over the past two weeks. The PSS-SR has demonstrated high internal consistency (α = .92), good test-retest reliability (*r* = .87), and high convergent validity (82% agreement) with the SCID-IV for PTSD diagnosis (Foa et al., 1997).

Quick Inventory of Depressive Symptomatology – Clinical Rating (QIDS-C; Rush et al., 2003)

The 16-item QIDS-C is an interview measure assessing depression severity over the past two weeks. Items are rated on a 0 to 3 Likert scale, with anchors tailored to the specific item and

total scores ranging from 0 to 27. The QIDS-C has concurrent validity with other, longer measures of depression in patients with major depressive disorder (*r*s = .86-.96) and shows high sensitivity to symptom change (Rush et al., 2003). Ten percent of cases were re-rated for interrater reliability on the QIDS-C, with ICC = .98.

Structured Clinical Interview for DSM-IV (SCID-IV; First et al., 1995)

The SCID-IV is a diagnostic interview used to assess DSM-IV Axis I disorder criteria. It has acceptable joint interview interrater reliability (κ = .57 - 1.0; Zanarini et al., 2000) and was used to determine primary diagnosis and diagnostic co-occurrence. In this study, 10% of cases were re-rated for diagnostic reliability. Reliability for SCID diagnoses was acceptable: current MDD (κ = .73, *ppos* = .93, *pneg* = .80) and anxiety disorders (κ = 0.68, *ppos* = .86, *pneg* = .85).

Positive and Negative Affect Schedule (PANAS; Watson et al., 1988)

The PANAS is a self-report measure consisting of two, 10-item scales measuring positive affect (PA; e.g., excited, inspired) and negative affect (NA; e.g., upset, afraid). This study utilized the state version of the PANAS assessing affect over the past week. Each item consists of a word that describes different feelings and emotions which were rated on a 5-point Likert scale, from 1 (*very slightly or not at all*) to 5 (*extremely*), to which they have been experienced within the past week. The PANAS was completed at pre-treatment, before each session, and post-treatment. In the current sample, internal consistency was high for both PA and NA (respectively, α = .85, α = .92).

Change and Growth Experiences Scale (CHANGE; Hayes, Feldman, & Goldfried, 2007)

The CHANGE coding system was used to code psychotherapy sessions at weeks 1, 3, 4, 5, 8, and 10 in the imaginal exposure processing phase of PE; week 1 sessions were coded for the entire session since they did not include imaginal exposure. CHANGE is an observational

measure designed to capture a range of variables thought to be central to therapeutic change, such as cognitive-emotional processing, and has been used to code both narratives and therapy sessions in exposure-based cognitive therapy for depression (Hayes, Feldman, Beevers, et al., 2007), cognitive therapy for personality disorders (Hayes & Yasinski, 2015), and trauma-focused CBT for youths (Alpert et al., 2021). The CHANGE coding system was used to code the patient variables of cognitive-emotional processing, unproductive processing, avoidance, positive verbal emotion, negative verbal emotion, positive nonverbal emotion, negative nonverbal emotion, positive sense of hope, negative sense of hope, positive view of self, negative view of self, positive relationships, and negative relationships. CHANGE variables were coded on a 4-point scale from 0 (*not present*) to 3 (*high*).

Cognitive-Emotional Processing. This variable captured the extent to which the patient approached a problem and explored, tried to understand, challenge, and made meaning of it. Cognitive-emotional processing could begin as thinking about and questioning a problem area related to therapeutic goals or exposing oneself to new information and was then followed by insight or shift in perspective or meaning. For example, a patient response when asked about what was learned from imaginal exposure saying, “He was trying to humiliate me. It never occurred to me that he was trying to make me feel weak until today” would be coded as high on cognitive-emotional processing, as she realized for the first time that the negative comments about her made by the perpetrator probably weren’t true but were used to make her feel weak.

Unproductive Processing. In contrast to cognitive-emotional processing, unproductive processing captured the extent to which the patient approached a problem, explored, tried to understand, and made meaning of it but got stuck repetitively thinking about or analyzing a problem without significant insight or perspective shift. For example, a patient statement of, “I

can't stop thinking about everything and how I have failed in relationships. I've failed at everything. I am haunted by a list of failures." would be rated as high on unproductive processing, as the statement involved repetitive thoughts of guilt and failing relationships that were not viewed as therapeutically helpful.

Avoidance. Avoidance captured attempts to protect or defend oneself from problems or distressing experiences by pulling away, rather than moving toward them. Avoidance could take several forms, including emotional numbing, avoiding discussing certain topics, distraction, wandering off topic, or using humor to avoid or minimize a topic. Explicit statements of avoidance must have been made by the patient to be coded, for example, if during imaginal exposure processing a patient responded to a therapist's question about self-blame with, "I don't know… I don't want to talk about this anymore. I'm tired, I'm not going to talk about it." This statement would be coded as moderate avoidance.

Positive and Negative Verbal Emotion. The verbal emotion variables captured emotions that the patient verbally expressed about their own emotional state during the therapy session. Both specific emotion words and tone were considered to code the emotional valence. Although negative and positive verbal emotions were rated separately, they could co-occur, as it was possible to have a high level of both positive and negative emotion. Levels of verbal emotion coded during processing tended to reflect the patient's perceptions about how they felt during the imaginal exposure, as well as how they currently felt about the traumatic event and its sequelae. For example, "I felt like I got pretty upset. I remember gripping the arms of the chair" would be rated as negative, medium.

Positive and Negative Nonverbal Emotion. The nonverbal emotion variables captured the extent to which positive and negative emotions were activated during the therapy session, as

suggested by nonverbal patient behaviors (e.g., crying, laughing, voice wavering, eye contact, facial expressions, and gestures). Nonverbal emotions were coded independently of verbal emotions, as verbal and nonverbal emotional expressions could be incongruous. Apparent emotional numbness was coded as 0, reflecting the absence of emotion, and would instead be coded as avoidance.

Positive and Negative Sense of Hope. These variables captured the patient's capacity to see the possibility of change in the future, to recognize recent positive changes, recognition of feeling better, and/or an expression of commitment or determination to make changes. Hope could be directly related to treatment goals or be broader in scope, for instance a sense of hope or hopelessness about the world in general. For example, "I'm starting to think I might get better, that there might be a way out of this" would be coded as positive, low. Examples of negative hope statements include feeling stuck, trapped, having no way out, feeling tired of trying or a lack of commitment, or feeling permanently changed or damaged. For example, "I hate my life, and I can't see a way out" would be coded as negative, high.

Positive and Negative View of Self. Self variables captured the patient's self-concept and sense of worth, desirability, competence, and identity. Both general (e.g., feeling good about self for going out with friends) and treatment-specific (e.g., proud of self for completing imaginal exposure) selfviews were considered. Self statements must have been explicitly stated by the patient, not inferred judgments. For instance, stating "I messed up" without any elaboration would not imply a negative self-judgment. Positive self statements may have included self-acceptance, feeling of accomplishment and competence, clear sense of self, or feeling emotionally strong; negative self statements may have included low self-esteem, self-criticism, worthlessness, or feelings of failure. For example, "I feel so proud of myself. For the

first time in years, I was able to go to the mall. I feel like a new person" would be coded as positive, high.

Positive and Negative Relationships. These variables captured the patient's perceived quality of current interactions with others and their social support network. This could include immediate family, romantic partners, friends, co-workers, strangers, religious relationships (i.e., God), or people in general. They also captured memories about past relationships or fears about future relationships if the memories or fears were tied to current social functioning. The client-therapist relationship was not considered in these variables. Examples of positive relationship quality include encounters involving enjoyment or satisfaction, for example, "My friend really helped me get through the day last week, she was so supportive" would be coded as positive, high. Examples of negative relationship quality include encounters involving distress or dissatisfaction, or feelings of alienation, isolation, or loneliness activated in the absence of social encounters, for example, "I went to see my family for my father's birthday, but the criticism started immediately. I felt myself shut down" would be coded as negative, medium.

Coding Processes. Coding by volunteers, undergraduate and doctoral-level coders occurred at both study sites. All coders were trained to criterion (ICC $\geq$ .80 and within one point on the 4-point rating scale for all variables) through didactics, the CHANGE manual adapted for use with PE, and approximately 10 hours of practice coding tapes. Two coders rated each audio-visually recorded therapy session selected for coding, including sessions 1, 3, 4, 5, 8, and 10. For session 1, coders watched the entire session prior to coding; for sessions 3-10, coding occurred after each individual section (homework review, imaginal exposure, and processing). Coding of the processing section began as soon as the client opened their eyes or completed the narrative from imaginal exposure and ended once the therapist and client began discussing the homework

assignment for the next session. Coders only rated sessions from the other study site to reduce bias from knowing the therapists. Coders were masked to treatment condition and paired with each other a similar number of times to reduce drift or idiosyncratic pair ratings. Coding discrepancies and rater drift were addressed in weekly meetings with the coding team and biweekly, cross-site meetings. Consensus scoring by the coding team was used to reconcile ratings differing by 2 or more scale points. Once a year, tapes were submitted to Adele Hayes, Ph.D. (creater of CHANGE; Hayes, Feldman, & Goldfried, 2007) for criterion checks and coding feedback. Analyses in this study utilize consensus scores or average scores from the two coders in a pair. Inter-rater reliability, prior to consensus coding of imaginal exposure processing variables, was calculated for a subset of the cases, with good reliability (r = .90 - .96).

Positive and Negative System Activation. Positive and negative system activation was indexed by summing the scores of positively and negatively valenced affective, cognitive, and processing-related CHANGE variables. Specifically, positive system activation was operationalized as the sum of six CHANGE variables reflecting patient behaviors during processing of imaginal exposure: positive verbal emotion, positive nonverbal emotion, positive self-views, positive relationships, hopefulness, and cognitive-emotional processing. Negative system activation was operationalized as the sum of seven CHANGE variables: negative verbal emotion, negative nonverbal emotion, negative self-views, negative relationships, hopelessness, unproductive processing, and avoidance. Positive system activation scores could range from 0 to 24, and negative system activation scores could range from 0 to 28, with higher scores indicating higher levels of in-session positive and negative psychotherapy change processesPositive and negative system activation scores demonstrated marginally acceptable internal consistency, which was expected given the small number of items per system and the intentional selection of

variables assessing a range of positive and negative psychotherapy processes (positive system activation: mean α = .69, range α = .57 - .76; negative system activation: mean α = .65, range α = .54 - .74).

Interventions

Prolonged Exposure

PE was delivered following the treatment manual (Foa et al., 2007) in 10 weekly, 90-120 min sessions delivered by masters or Ph.D. level therapists with some prior CBT training. The protocol included psychoeducation about common reactions to trauma, breathing retraining, repeated *in vivo* exposures, repeated recounting of the trauma memories during session (i.e., imaginal exposure), and emotional processing of imaginal exposure. Homework assigned between sessions included reviewing psychoeducational handouts, listening to session recordings and imaginal exposure recordings, and completing *in vivo* exposures. After each imaginal exposure, 15-20 minutes of processing took place. During processing, the therapist encouraged the patient to talk about and explore their reactions to revisiting the trauma memory, as well as thoughts and feelings about the trauma and its meaning. Therapists used open-ended questions to get the patient to talk about their thoughts and feelings about the exposure. The therapist sought to normalize and contextualize the patient's thoughts, feelings, and behavior, and to help the patient recognize changes in distress and distress tolerance abilities, to examine expressions of unrealistic or maladaptive beliefs and to consolidate adaptive shifts in perspective and newfound insights (Foa et al., 2007).

PE training consisted of discussion, role playing, reviewing the treatment manual, viewing recordings of PE sessions and practicing treatment procedures. Weekly PE supervision was conducted, with sessions recorded and reviewed during supervision. Outside raters formally

assessed PE protocol adherence for 10% of cases using a detailed treatment integrity manual, with therapists completing 98% of essential components.

Prolonged Exposure Plus Sertraline

Participants who chose or were randomly assigned to receive sertraline in addition to PE met with a board-certified psychiatrist for six, up to 30-min sessions (Weeks 1, 2, 4, 6, 8, and 10), with the first session lasting 40 min, based on a treatment manual. Initial dosage was 25 mg/day, with a goal of 200 mg/day if indicated and tolerated, following a standard treatment algorithm. Pill counts and medication diaries assessed medication adherence.

Sertraline training consisted of review of the treatment manual, overview of treatment procedures, dosage schedules, and PTSD-relevant treatment information. Sertraline sessions were recorded. Adherence was conducted by an outside rater for 10% of sessions, using a checklist from the treatment manual, with providers completing 88% of essential components.

Procedure

The clinical trial was approved by each site's institutional review boards and pre-registered, but this study was not. Following recruitment and pre-screening of prospective participants via structured telephone screen, written informed consent was obtained. Independent evaluators masked to eventual condition assignment conducted semi-structured interviews to assess Criterion A trauma exposure, PTSD severity and diagnostic status (PSS-I), depression severity (QIDS-C), and other DSM-IV Axis I disorders (SCID-IV) to help determine eligibility.

Prior to randomization, eligible patients completed pre-treatment self-report measures (e.g., PANAS) and were shown digitized treatment rationales for PE alone and combined PE and sertraline and indicated their treatment preferences. Each rationale included background information, brief efficacy information, hypothesized treatment mechanisms of action, treatment

procedures, and side effects. The rationales were counterbalanced for gender and vocation of the presenter (psychologist or psychiatrist), as well as order.

Using a computer-generated urn sequence, randomization used a 1:1 ratio for choice vs. no choice, and in the no choice condition, a 1:1 ratio for PE vs. PE plus sertraline. Randomization was stratified based on current antidepressant status (yes/no) and current SCID-IV MDD diagnosis (yes/no). At the randomization visit, a research coordinator obtained the participant's condition assignment and stored it in a sealed envelope, which was then opened by the patient together with a PhD-level coordinator who had been masked to condition assignment. Potentially eligible patients were then randomized to "Choice" or "No Choice." Patients randomized to "Choice" were given their preferred treatment. Those randomized to "No Choice" were randomized to either PE alone or PE plus sertraline.

Following randomization procedures, patients received up to 10 sessions of PE or PE plus sertraline. At each session, patients completed self-report measures (e.g., PANAS). Sessions were video-recorded, and these recordings were later used for CHANGE coding. Upon the completion of treatment, independent evaluators masked to condition assignment conducted the post-treatment assessment (PSS-I, QIDS-C) and patients completed post-treatment self-report measures (e.g., PANAS). Follow-up assessments were conducted at 3, 6 (interview only), and 9 months.

Statistical Analysis

Dynamic structural equation modeling (DSEM; Asparouhov et al., 2018) was used to examine patterns of change in PA, NA, and PTSD symptoms across sessions 1-10 and moderation by treatment condition (Study 1). DSEM was also used in Study 2 to examine patterns of change in positive and negative systems of in-session change processes and PTSD

symptoms across sessions 1, 3-5, 8, and 10, and moderation by treatment condition. DSEM was chosen over a multilevel modeling (MLM) approach to examine multiple dependent variables within a single model, allowing direct examination of reciprocal relationships, and to avoid biases that can be introduced by MLM when estimating cross-lagged relationships (Asparouhov et al., 2018; Falkenström et al., 2022; McNeish & Hamaker, 2020). Further, DSEM allows for the separate examination of between-person (i.e., an individual's mean score across time points) and within-person (i.e., deviations from person-specific means at each time point) associations. This approach was chosen because the relationship of between-person variability in a therapeutic process variable (e.g., PA or positive system activation) and a subsequent outcome variable (e.g., PTSD symptoms) may stem from the influence of stable patient characteristics on both process and outcome variables, rather than reflecting a causal mechanism (Falkenström et al., 2013; Sasso et al., 2016). This study examined within-person, fixed, random, and autoregressive effects. SEM models were estimated in Mplus 8.4 (Muthén & Muthén, 2017) via Bayesian estimation using a Markov Chain Monte Carlo algorithm, utilizing uninformative priors. While a formal power analysis was not conducted, simulation studies suggest that the present sample sizes (Study 1: $N = 130$; $T = 10$; Study 2: $N = 111$, $T = 6$) approached the minimum recommended sample size for having at least 80% power to test lagged, with-person effects with random slopes (Schultzberg & Muthén, 2018), though other studies with similar research questions and number of observations have utilized DSEM (e.g., Gómez Penedo et al., 2021).

In Study 1, three models were estimated to examine cross-lagged relationships among PA and NA (Model 1), PA and PTSD symptoms (Model 2), and NA and PTSD symptoms (Model 3) across sessions 1-10. A similar approach was taken in Study 2: three DSEM models were estimated to examine cross-lagged relationships among positive and negative system activation

(Model 4), positive system activation and PTSD symptoms (Model 5), and negative system activation and PTSD symptoms (Model 6) across sessions 1, 3-5, 8, and 10. Models 1-6 included level 1 (within-person) effects of the lag-1 focal predictors and dependent variable cross-lagged associations and autoregressions, and level 2 (between-person) effects for each focal predictor. Models included random intercepts and slopes to allow average values, cross-lagged associations, and autoregressive effects to vary across individuals. Some authors have recommended detrending time-series data prior to DSEM analysis to avoid violating the stationarity assumption (McNeish & Hamaker, 2020), though others suggest that detrending for time may not be desirable for experimental, longitudinal studies, such as this one, where time trends are an intended effect of the study design (Thome, 2014; Wang & Maxwell, 2015). Therefore, because all variables in Studies 1 and 2 were expected to change over time due to treatment, to avoid removing the very effects that were of most interest in capturing, detrending time-varying variables was not employed. DSEM imputes missing values for each iteration of the Markov Chain Monte Carlo procedure based on values from other time points, parameter estimates from previous iterations, and error by sampling from the conditional posterior distributions (Asparouhov et al., 2018).

In Studies 1 and 2, two additional sets of three DSEMs (Models 1a – 3a; Models 4a – 6a) were fitted to test whether treatment condition moderated the cross-lagged associations between changes in affect/positive and negative systems and PTSD symptoms; these models were identical to Models 1-6 with addition of treatment condition (PE versus PE + sertraline) as a level 2 (between-person) predictor of cross-lagged slopes. For the treatment condition variable, PE alone was coded as 0 and PE plus sertraline was coded as 1. Significant interaction effects

were probed by examining simple slopes for PE and PE plus sertraline. Visual representations of Models 1-3 (Study 1) and Models 4-6 (Study 2) are provided in Figures 1-6.

Results

Study 1: Changes in Self-Reported Positive and Negative Affect

Means and standard deviations for PA, NA, and PTSD severity across each session are reported in Table 3. Correlations among PA, NA, PTSD severity, and depression severity, averaged across treatment sessions, are presented in Table 4. Correlations were as expected, with higher PA weakly to moderately associated with lower NA, PTSD severity, and depression severity, and higher NA moderately to strongly associated with higher PTSD and depression severity.

Linear mixed models with random slopes and intercepts were used to examine average change over time in PA and NA and whether changes over time were moderated by treatment condition. Positive affect increased moderately over sessions (b = 0.67, SE = 0.16, t(109.12) = 4.15, $p < 0.001$, 95% CI [0.35, 0.98], d = 0.51), and NA decreased strongly over sessions (b = -1.00, SE = 0.16, t(101.96) = -6.30, $p < 0.001$, 95% CI [-1.31, -0.68], d = 0.78). Neither changes in PA nor changes in NA over time were moderated by treatment condition.

Cross-Lagged Associations Among PA, NA, and PTSD Symptom Change

Results of DSEM Models 1-3 are presented in Table 7, including standardized model coefficients to provide a measure of effect size. In Model 1, session-to-session changes in PA and NA were reciprocally related. Decreases in NA at a session predicted small to medium sized increases in PA at the next session ($NA_t \rightarrow PA_{t+1}$: ES = -0.20, 95% credible interval [CI; -0.28, -0.13]), and increases in PA predicted small decreases in next-session NA ($PA_t \rightarrow NA_{t+1}$: ES = -0.09, 95% CI [-0.15, -0.02]). Notably, the $NA_t \rightarrow PA_{t+1}$ association was approximately twice the

size of the $PA_t \rightarrow NA_{t+1}$ relationship. Thus, while the cross-lagged relationship between NA and PA was reciprocal, a 1-unit decrease in NA predicted a 0.17-unit increase in PA at the next session, whereas a 1-unit increase in PA predicted only a 0.10-unit decrease in next-session NA.

In Model 2, changes in PA did not predict next-session PTSD symptoms, but decreases in PTSD symptoms predicted medium sized increases in next-session PA ($PTSD_t \rightarrow PA_{t+1}$: ES = -0.26, 95% CI [-0.34, -0.17]). A 1-unit decrease in PTSD severity predicted a 0.17-unit increase in next-session PA. In Model 3, session to session changes in NA and PTSD symptoms were reciprocally related, with decreases in NA at a session predicting small decreases in PTSD symptoms at the next session ($NA_t \rightarrow PTSD_{t+1}$: ES = 0.12, 95% CI [0.06, 0.18]), and decreases in PTSD predicting large decreases in next-session NA ($PTSD_t \rightarrow NA_{t+1}$: ES = 0.50, 95% CI [0.38, 0.60]). Notably, the $PTSD_t \rightarrow NA_{t+1}$ association was approximately four times the size of the $NA_t \rightarrow PTSD_{t+1}$ association. Thus, although NA and PTSD were reciprocally related from session to session, a 1-unit decrease in PTSD severity predicted a 0.35 unit decrease in NA at the next session, whereas a 1-unit decrease in NA predicted only a 0.16 unit decrease in next-session PTSD severity.

Across these three models as seen in Table 7, all random intercepts and slopes were significant, indicating the within-person, cross-lagged associations among PA, NA, and PTSD symptoms differed across participants. Thus, the cross-lagged, within-person associations among PA, NA, and PTSD may be moderated by between-person differences, such as treatment condition.

Finally, as seen in Table 8, across Models 1a – 3a, treatment condition did not moderate cross-lagged associations among PA, NA, and PTSD symptoms.

Summary of Changes in Self-Reported Positive and Negative Affect

In sum, self-reported PA and NA improved moderately to strongly over the course of treatment. Within-person, session-to-session improvements in NA and PA were reciprocally related, though the effect of improvements in NA on subsequent improvements in PA was twice as strong as PA on subsequent NA. Finally, improvements in PTSD symptoms predicted subsequent improvements in PA and NA much more strongly than the effects of improvements in affect on subsequent changes in PTSD.

Study 2: Changes in Observer-Rated Psychotherapy Process Positive and Negative Systems

Means and standard deviations for positive and negative system activation and PTSD severity across each session are reported in Table 5. Correlations among positive and negative system activation, PTSD severity, and depression severity, averaged across treatment sessions, are presented in Table 6. As expected, higher positive and lower negative system activation were weakly associated with higher PTSD and depression severity, with slightly stronger correlations among negative system activation, PTSD, and depression compared to positive system activation, PTSD, and depression. Higher positive system activation was weakly associated with lower negative system activation.

Linear mixed models with random slopes and intercepts were used to examine average change over time in positive and negative system activation and whether changes over time were moderated by treatment condition. Positive system activation increased strongly over sessions (b = 0.51, SE = 0.05, t(131.08) = 10.61, $p < 0.001$, 95% CI [0.42, 0.61], d = 1.42), and negative system activation decreased strongly over sessions (b = -0.34, SE = 0.05, t(142.96) = -7.27, $p <$ 0.001, 95% CI [-0.43 -0.25], d = 0.98). Neither changes in positive system activation nor changes in negative system activation over time were moderated by treatment condition.

Cross-Lagged Associations Among Positive and Negative Systems During Psychotherapy and PTSD Symptom Change

Results of DSEMs Models 4-6 are presented in Table 9, including standardized model coefficients to provide a measure of effect size. In Model 4, similar to self-reported PA and NA, session-to-session changes in in-session positive and negative systems were reciprocally related. Increases in positive system activation at one session predicted medium sized decreases in negative system activation at the next session (positive system$_t$ → negative system$_{t+1}$: ES = -0.25, 95% CI [-0.38, -0.11]), and decreases in negative system activation predicted small increases in next-session positive system activation (negative system$_t$ → positive system$_{t+1}$: ES = -0.09, 95% CI [-0.16, -0.004]). Notably, the positive system$_t$ → negative system$_{t+1}$ association was 2.78 times the size of the negative system$_t$ → positive system$_{t+1}$ relationship, which is the opposite pattern found for self-reported PA and NA, where improvements in NA more strongly predicted subsequent improvements in PA. Here, a 1-unit increase in positive system activation during a psychotherapy session predicted a 0.23-unit decrease in negative system activation during the next session, whereas a 1-unit decrease in negative system activation during a session predicted only a 0.10-unit increase in positive system activation at the next session.

In Models 5 and 6, changes in positive and negative systems during a psychotherapy session did not predict next-session changes in PTSD symptoms, but decreases in PTSD symptoms predicted medium sized increases in next-session positive system activation (PTSD$_t$ → positive system$_{t+1}$: ES = -0.44, 95% CI [-0.55, -0.37]) and medium sized decreases in negative system activation (PTSD$_t$ → negative system$_{t+1}$: ES = 0.38, 95% CI [0.26, 0.52]). Thus, improvements in PTSD severity preceded improvements in both self-reported PA and NA as well as improvements in positive and negative system activation during psychotherapy sessions.

However, whereas self-reported NA improved more strongly than PA following improvements in PTSD, in-session positive system activation improved more strongly than negative system activation following improvements in PTSD. A 1-unit decrease in PTSD severity predicted a 0.11-unit increase in positive system activation but only a 0.07-unit decrease in negative system activation during the next psychotherapy session.

Across these Models 4-6 as seen in Table 9, all random intercepts and slopes were significant, indicating the within-person, cross-lagged associations among positive and negative system activation and PTSD symptoms differed across participants. Thus, the cross-lagged, within-person associations among system activation and PTSD may be moderated by between-person differences, such as treatment condition.

Finally, as seen in Table 10, similar to Study 1 examining PA and NA, treatment condition did not significantly moderate most temporal associations among positive and negative system activation during psychotherapy sessions and PTSD symptoms. However, unlike the model examining associations between changes in PTSD and self-reported PA, treatment condition moderated the effect of positive system activation on subsequent negative system activation (interaction ES = -0.42, 95% CI [-0.80, -0.10]). The effect of increases in positive system activation during a psychotherapy session on subsequent decreases in negative system activation during a session was moderately stronger when PE was augmented with sertraline. In PE alone, the effect of changes in positive system activation on subsequent negative system activation did not reach statistical significance (positive system$_t$ → negative system$_{t+1}$ = -0.05, 95% CI [-0.25, 0.14]), whereas in PE plus sertraline a 1-unit increase in positive system activation predicted a 0.37-unit decrease in next-session negative system activation (positive system$_t$ → negative system$_{t+1}$ = -0.37, 95% CI [-0.54, -0.19]).

Summary of Changes in Observer-Rated Psychotherapy Process Positive and Negative Systems

In sum, similar to the improvements in self-reported PA and NA in Study 1, positive and negative in-session psychotherapy change processes d improved over the course of treatment. However, improvements in positive and negative change processes during psychotherapy sessions were larger than improvements in self-reported affect, and unlike self-reported affect, in-session positive chang processes improved to a greater degree than did negative processes. As was found for self-reported PA and NA, positive and negative change processes during sessions were reciprocally related from session-to-session, but unlike PA and NA, in-session positive processes improvements predicted stronger improvements in subsequent negative processes compared to the reverse. Further, the effect of positive processes on subsequent negative processes only reached significance when PE was augmented with sertraline. Similarly to Study 1, improvements in PTSD severity preceded improvements in positive and negative system processes during psychotherapy sessions, but unlike Study 1, changes in PTSD were more strongly associated with positive than negative processes.

Discussion

Study 1: Changes in Self-Reported Positive and Negative Affect

This study examined session-to-session patterns of change in self-reported positive and negative affect and PTSD symptoms during PE with or without sertraline augmentation. As hypothesized, negative affect decreased with a large effect and positive affect increased with a medium effect over treatment sessions, and these effects did not differ with sertraline augmentation of PE. Thus, PE may produce substantial improvements in positive emotionality, and augmenting PE with an SSRI does not appear to amplify this effect. Although changes in

self-reported PA and NA were related to each other from session to session, decreases in NA more strongly predicted increases in next session PA. Notably, PTSD symptom change consistently predicted small next session increases in positive affect and moderate next session decreases in negative affect and not as strong in the reverse direction. Accordingly, changes in affect may be more a consequence than a driver of PTSD symptom reduction, potentially arguing that PTSD-specific processes rather than broader negative affect or neuroticism effects underlie treatment change.

The strong reductions in NA observed across both treatment conditions are unsurprising. Exposure-based treatments for PTSD and anxiety disorders are linked with extinction learning models that promote reductions in negative emotional responses to conditioned stimuli. More interesting is that PA increased across both treatment conditions, at a moderate effect size, suggesting that PE may facilitate improvements in reward system functioning (e.g., Hoffman et al., 2022). Although not explicitly designed to target PA, there are several plausible pathways in PE through which PA may be improved. Avoidance of trauma-reminders could cause significant functional impairment and restrict access to rewards important for well-being (e.g., Winer et al., 2017). Once avoidance is decreased, rewards may be sought after more regularly, and PA may naturally increase. *In vivo* exposure explicitly encourages approach of previously avoided, trauma-related cues, and *in vivo* exposure exercises may entail behavioral activation ipso facto via the approach of activating activities, travel to therapeutically relevant locations, and re-engagement in social activities (Foa et al., 2019). Imaginal exposure may also increase PA by decreasing re-experiencing symptoms and increasing feelings of mastery. Good therapists often incorporate and highlight intrinsic, naturalistic, and more tangible rewards during therapy, including such feelings as relief or sense of accomplishment, praise from others, a stronger sense

of connection with friends or family, or even "treats," "prizes," or "rewards." Thus, both imaginal and *in vivo* exposure may directly encourage reward acquisition and, over time, lead to increases in PA. Reductions in distress experienced over the course of repeated exposure may also facilitate experiences of PA. Notably, augmenting PE with sertraline did not produce superior improvements in PA and NA. This is consistent with equivocal findings concerning the comparative efficacy of psychotherapy and combined psycho- and pharmacotherapy for PTSD (e.g., Hetrick et al., 2010; Rauch et al., 2019; Schneier et al., 2012) and suggests that adding SSRIs to exposure therapy may not confer meaningful benefit to improvements in PA and NA.

Decreases in NA preceded subsequent next-session increases in PA more strongly than vice versa, consistent with the broader literature on the relative potency of changes in NA on subsequent PA compared to vice versa (e.g., Garland et al., 2010; Hollenstein, 2015). One of the most plausible explanations is that exposure-based interventions directly target negative affect, almost by definition; whereas experiences of mastery and accomplishment during imaginal and *in vivo* exposure may occur (e.g., Benight & Bandura, 2004) in response to fear extinction and decreases in negative emotional responses to trauma cues. Similarly, shifting negative, trauma-related beliefs about oneself, others, and the world may set the stage for the adoption of new, positive, trauma-related beliefs (Ehlers & Clark, 2000; LoSavio et al., 2017). It is also possible that NA no longer interferes with the experiences of PA during potentially rewarding experiences (DePierro et al., 2018) or frees up cognitive resources to facilitate greater attention to and processing of rewarding experiences (Litz & Gray, 2002).

Remarkably, PTSD symptom change preceded decreases in NA and more strongly than the other way around, with the effect of PTSD on subsequent NA four times as strong as the reverse. The present results may argue that broadly targeting neuroticism and associated

temperamental characteristics, commonly seen linked with negative affect, as suggested by some (e.g., Barlow et al., 2021), may not be sufficient to produce clinically meaningful changes in trauma-specific symptoms such as re-experiencing, avoidance, or an exaggerated startle response. Rather, specifically targeting conditioned emotional responses to trauma cues and/or negative, trauma-related beliefs may be necessary for meaningful PTSD reduction. One of the key features that may be unique in PTSD compared to other anxiety and depressive disorders is the trauma memory. Specific treatment focus on the trauma memory and associated memory traces may be needed to promote hippocampal neurogenesis and better pattern separation (e.g., Besnard & Sahay, 2016). Indeed, inhibitory learning (Craske et al., 2014) and emotional processing (Foa et al., 2006; Foa & Kozak, 1986) highlight the importance of violating threat-related expectancies for trauma-related cues and the impact of their accompanying emotional responses, with these violations producing new, threat-inhibitory learning about the meaning or consequences of trauma-related stimuli. More broadly, these findings have implications for debates over common versus specific factors driving recovery across treatment modalities and disorders. While common factors like improvements in neuroticism (Barlow et al., 2014, 2021) and the therapeutic alliance (Baier et al., 2020; Buchholz & Abramowitz, 2020) may facilitate therapeutic change, improvements in PTSD symptoms preceding improvements in affect far more strongly than the reverse suggests that factors specific to PTSD, and perhaps specific to prolonged exposure therapy, play critical roles in recovery from PTSD.

Given that increases in PA predicted small subsequent decreases in NA, improvements in PA may play a role in building and maintaining momentum during exposure therapy, helping to generate approach motivation to engage in therapy activities (Zbozinek & Craske, 2017). Several authors have proposed explicit targeting of PA transdiagnostically, showing that improvements

in PA may enhance treatment engagement and outcomes (e.g., Cernasov et al., 2021; Craske et al., 2019; Zbozinek & Craske, 2017). In the present study, however, changes in PA did not significantly predict subsequent changes in PTSD, suggesting that improvements in PA may be more a consequence than a driver of treatment processes and PTSD symptom reduction. In addition, different approaches to targeting PA may differentially impact recovery processes; explicit strategies to amplify positive emotions such as imaginal recounting of positive experiences or gratitude exercises to savor positivity (Craske et al., 2023) may have a stronger impact than less direct targeting of PA through *in vivo* and imaginal exposure exercises. Importantly, there was significant variability in all of the cross-lagged and autoregressive associations among PA, NA, and PTSD, including in the association between PA and subsequent changes in PTSD. Though on average, PA is more a lagging than leading indicator of change in PE, for some individuals improvements in PA may do more to drive recovery. Future studies should explore moderators of the impact of changes in PTSD symptoms on subsequent PA, such as depression severity and specific facets of reward functioning, including reward hyposensitivity (e.g., Craske et al., 2023), to identify those most likely to benefit from additional targeting of PA. Positive emotions related to increased self-efficacy, mastery, and goal-oriented behaviors (e.g., pride, confidence, determination, excitement) may be particularly beneficial for inhibitory learning during exposure therapy (Zbozinek & Craske, 2017).

Sertraline augmentation of PE did not explain the variability in the observed associations among NA, PA, and PTSD symptoms. In PTSD, evidence is still mixed as to whether combined psycho- and pharmacotherapy approaches outperform psychotherapy alone (e.g., Hetrick et al., 2010; Rauch et al., 2019; Rothbaum et al., 2006; Schneier et al., 2012; Simon et al., 2008), and fewer studies still have examined how SSRI augmentation impacts processes of change in

exposure therapy (Allard et al., 2021; Cooper, Zoellner, et al., 2017). One explanation is that these distinct therapies actually modulate similar brain neurocircuitry surrounding fear expression and extinction (e.g., Heesbeen et al., 2023). Additionally, to the extent that trauma-specific processes drive change in PE relative to general changes in affect, SSRIs' broad impacts on emotion systems may be outweighed by PE's trauma-focused approach with respect to improvements in PTSD symptoms (e.g., MacNamara et al., 2016). Another explanation might be that there are specific people where this targeted augmentation is most important (e.g., DeRubeis, Gelfand, et al., 2014). Studies of individual difference variables related to reward functioning may help to identify individuals for whom SSRI augmentation is more helpful.

In conclusion, this study highlights temporal patterns of change in self-reported affect and PTSD symptoms during therapy for PTSD, with effects of PTSD symptom reduction on subsequent improvements in PA and NA being substantially stronger than the reverse effects. Although variation in temporal associations between changes in affect and subsequent PTSD leaves open the possibility that certain patients may benefit from targeting of affect more generally, these findings highlight the potential centrality of specifically targeting PTSD symptoms rather than broader affect. Indeed, engaging in standard PE designed to target PTSD symptoms appears sufficient to effect meaningful improvements in levels of positive and negative emotions. *In vivo* and imaginal exposure to trauma-related stimuli reduce negative emotional responses to and accompanying avoidance of trauma-related cues, which may facilitate increased reward acquisition and availability of cognitive resources for positive emotions. Prolonged exposure may also result in decreases in negative, trauma-related beliefs and increases in positive beliefs, which may translate to broader improvements in emotional functioning.

Study 2: Changes in Observer-Rated Psychotherapy Process Positive and Negative Systems

This study examined session-to-session patterns of change in positive and negative systems comprised of in-session, observer-rated patient emotions, cognitions, and processing styles, and their relationship to changes in PTSD symptoms during PE with or without sertraline augmentation. Over the course of treatment, positive and negative change processes during imaginal exposure processing increased and decreased, respectively, in a linear fashion and with large effects, consistent with observed changes in average self-reported positive and negative affect across treatment. Notably, improvements in observer-rated, in-session positive and negative psychotherapy processes were substantially larger than improvements in self-reported PA and NA. Further, improvements in positive processes during psychotherapy sessions outweighed improvements in negative processes, whereas the reverse pattern was found for changes in self-reported affect. This suggests that in-session patient processes offer insights into therapeutic change not captured by self-report measures completed outside of treatment sessions.

Changes in positive and negative change processes were reciprocally related over time, with the effect of higher positive processes on subsequent lower negative processes more than twice as strong as the reverse effect. Thus, improvements in positive in-session psychotherapy processes led improvements in negative in-session processes, in contrast with findings from Study 1 that improvements in self-reported NA preceded improvements in PA. This may suggest potential functional links between positive and negative in-session psychotherapy processes, with increases in approach-oriented processing, positive beliefs, and positive emotions playing an important role in inhibiting negative beliefs and cognitions and avoidant processing. This inhibition may play a particularly important role in the context of new learning that occurs

during exposure exercises and accompanying processing, compared to the relationship between positive and negative affect outside of session.

The effect of increased positive change processes on subsequent decreased negative change processeson was stronger with sertraline augmentation. Thus, sertraline augmentation may enhance the extent to which positive psychotherapy processes inhibit subsequent negative processes, possibly by boosting the effects of subjective reward perception during post-exposure processing. Finally, decreases in PTSD symptoms preceded medium increases and decreases in positive and negative processes, respectively, but neither positive nor negative processes subsequent changes in PTSD symptoms, suggesting that the observer-rated patient emotions, cognitions, and processing styles during imaginal exposure processing may be lagging rather than leading indicators of recovery during exposure therapy for PTSD. Together with findings from Study 1 that improvements PTSD led improvements in self-reported affect, this provides strong evidence that change processes specific to PTSD symptoms, such as extinction learning and changes in trauma-related beliefs, are more closely tied to recovery than are nonspecific change processes during exposure therapy for PTSD.

Larger improvements in positive patient emotions, cognitions, and processing styles across sessions of imaginal exposure processing relative to improvements in their negative counterparts stand in contrast to the larger improvements in self-reported NA relative to PA found in Study 1. Not only does PE appear to effect improvements in average levels of emotions, but it also produces robust improvements in adaptive processing of trauma-related material and accompanying improvements in trauma-related emotions and cognitions (Foa et al., 2006; S. Rauch & Foa, 2006). Furthermore, changes in positive and negative processes appeared to be mutually inhibitory over time. Processing of imaginal exposure during PE sessions offers

patients the opportunity to reflect on the experience of engaging in imaginal exposure and to create new ways of appraising the trauma and the trauma-related reactions upon approaching the trauma memory (Foa et al., 2019). The development of this new, more adaptive, trauma-related learning may be reflected in relative increases and decreases in positive and negative system activation over the course of treatment.

It is striking that, in contrast to relatively smaller changes in self-reported PA and smaller effects of PA on subsequent NA in Study 1, Study 2 revealed substantially larger improvements in positive compared to negative psychotherapy processes and stronger effects of positive processes on subsequent negative processes. Thus, positive, in-session patient processes during post-exposure processing appear to play a more prominent role in therapeutic change than either self-reported, average PA or negative, in-session patient processes. These discrepancies may be explained in part by the different processes captured by self-reported mean levels of affect and observer-rated in-session patient statements and behaviors. Positive psychotherapy patient processes, in particular, may capture strong indicators of progress in PE. Expressions of hopefulness during post-exposure processing may reflect patient perceptions that treatment is helping and increased confidence in completing treatment; hopefulness may also reflect increases in future-oriented thinking and posttraumatic growth, including the perception of new possibilities in life (e.g., Hagenaars & Van Minnen, 2010; Tedeschi et al., 2015). Increases in expressions of positive beliefs about oneself and relationships with others may reflect adaptive changes in trauma-related beliefs, increased social engagement and interpersonal connection, and increases in perceived self-efficacy and mastery, all of which are desirable and target outcomes of PE (e.g., Brown et al., 2019; Foa et al., 2019). Together with increases in cognitive-emotional processing, these factors may be picking up on new insights being generated by the patient, new

meanings made, and easier retrieval of new, adaptive learning relative to the retrieval of maladaptive learning.

Conversely, increases in negative patient psychotherapy processes may indicate that problems are occurring in PE. Increases in unproductive processing, avoidance, and hopelessness may reflect difficulties generating new trauma-related insights and be a sign that the patient is getting stuck processing their trauma. Indeed, hopelessness and negative beliefs about oneself may be more likely to occur when treatment is not progressing as the patient desires; increases in statements about relationship difficulties may indicate challenges in obtaining social support or strengthening interpersonal connections. Given that increases in positive psychotherapy processes predicted subsequent dampening of negative processes, increases in positive processes may indicate greater resilience to challenges in PE and even help patients overcome difficulties encountered in treatment. For example, a patient who increasingly believes they can do difficult things (i.e., engage in exposure and approach the trauma memory) and effectively tolerate distress may become less discouraged following a particularly difficult imaginal exposure exercise or new relationship difficulty, and they may be more likely to address these challenges with approach-oriented rather than avoidance-oriented coping strategies. Taken together, increases in positive patient processes during post-exposure processing may be a potent signal – moreso than changes in self-reported emotions outside of therapy sessions – to therapists that treatment is working, whereas increases in negative patient processes across sessions should raise alarms for therapists that adjustments may be needed to facilitate therapeutic response.

Although sertraline augmentation of PE did not significantly moderate temporal patterns of change in self-reported affect and PTSD symptoms in Study 1, increases in positive psychotherapy processes were more strongly associated with subsequent dampening of negative

psychotherapy processes when PE was augmented with SSRI medication. Thus, it appears that engaging in PE with a supplemental pharmacological aid may increase the potency of positive psychotherapy processes. This finding contrasts with previous studies in which pharmacological monotherapy (Cooper, Zoellner, et al., 2017) and augmentation of psychotherapy (Zoellner et al., 2017) were associated with decoupling of changes in putative therapeutic change processes, specifically improvements in negative trauma-related beliefs, and PTSD symptoms. Findings from these previous studies may suggest that engaging in psychotherapy without the aid of medication results in greater internal attributions of change, translating to increased self-efficacy that helps patients overcome challenges in treatment. Indeed, previous studies have found that individuals taking medication while engaged in psychotherapy experience smaller improvements in beliefs about coping abilities and lower confidence in maintaining treatment gains after acute treatment (e.g., Moradveisi et al., 2015; Schaumberg et al., 2013). However, this study suggests that SSRI augmentation may enhance the degree to which positive psychotherapy processes inhibit negative processes. Experiencing positive emotions during post-exposure processing may enhance exposure-related learning by encouraging semantic and relational processing and rehearsal of newly learned information (Zbozinek & Craske, 2017). The impact of positive emotions on learning may be strengthened by SSRIs, which have been shown to boost subjective reward perception during learning (Michely et al., 2020). Thus, SSRIs may increase the salience of rewards encountered during processing, such as experiences of mastery, which may in turn facilitate increased engagement in therapy activities and build motivation to overcome challenges.

Consistent with the findings from Study 1for self-reported negative and positive affect, neither changes in positive nor negative system activation during therapy sessions predicted

subsequent changes in PTSD symptoms; instead, reductions in PTSD symptoms predicted subsequent moderate next-session increases in positive and decreases in negative system activation. Changes in positive and negative emotions, cognitions, and processing styles during imaginal exposure may be consequences rather than drivers of changes in PTSD. Thus, as discussed above, treatment processes specific to PE and PTSD may lead to broader improvements in positive and negative psychotherapy processes during post-exposure processing, not the other way around. These findings particularly in regard to therapy-session content are partially consistent with others that have looked at the relationship between in-session CHANGE-coding processes and PTSD symptom change. Some studies have failed to find significant associations between outcomes of trauma-focused CBT and putatively adaptive psychotherapy processes such as expressions of overgeneralized trauma beliefs (Ready et al., 2015), rumination, and avoidance (Hayes et al., 2017), while another found that lower negative psychotherapy processes including, cognitive, emotional, behavioral, and physiological patient responses predicted greater PTSD improvement (Alpert et al., 2021). Few studies utilizing CHANGE coding have examined relationships between positive psychotherapy processes and PTSD outcomes, though greater cognitive-emotional processing and hopefulness, included among this study's positive psychotherapy processes, did predict improvement in depression (Abel et al., 2016; Hayes et al., 2014).

Therapists providing PE would be aided by future studies that can identify in-session factors that are not merely indicators of progress, but that predict subsequent improvements. It may be the case that in aggregating positive and negative psychotherapy processes, this study papered over unique contributions of specific psychotherapy processes, for example cognitive-emotional processing or hope, as demonstrated in other, non-PTSD studies (Abel et al., 2016;

Hayes et al., 2014). The high degree of between-patient differences in the temporal associations between self-reported affect and PTSD and between psychotherapy processes and PTSD also suggest that, for some individuals, these processes may predict change in PTSD. Given the central role of diminished positive affect in depression and high rates of co-occurrence of PTSD and depression, future studies should examine whether depression severity moderates the relationships between positive and negative processes and their impact on recovery (Clark & Watson, 1991; Peskin et al., 2019; Zoellner et al., 2014). Recent computational advances in identifying multivariate profiles of individual difference variables that impact treatment response may also facilitate efforts to optimally match individual patients with different treatment strategies (e.g., DeRubeis, Cohen, et al., 2014; van Bronswijk et al., 2021). As suggested by these studies, multimodal assessments including self-report, observer-rated, physiological, and cognitive neuroscience measures assessing both pre-treatment patient characteristics as well as in-session change processes will provide the most comprehensive insights. Finally, therapist behaviors during post-exposure processing may drive subsequent PTSD improvement; the CHANGE system codes for therapist behaviors, but they were not included in this study. Indeed, therapist competence in case conceptualization was found to predict sudden gains during depression treatment (Abel et al., 2016).

General Conclusions

The present studies examined positive and negative processes of therapeutic change in PE with or without sertraline augmentation for the treatment of PTSD, utilizing dynamic structural equation modeling to examine session-to-session patterns of change in self-reported positive and negate affect, observer-rated, positive and negative patient emotions, cognitions, and processing styles during the processing of imaginal exposure, and PTSD symptoms. Both PE with and

without sertraline were associated with substantial gains in positive affect and positive psychotherapy change processes and large reductions in negative affect and negative psychotherapy change processes; the medium to large increases in positive processes are notable given that PE was designed to most directly target negative, trauma-related emotional, cognitive, and behavioral responses (Foa et al., 2011). Improvements in positive processes suggest that the impacts of exposure therapy extend beyond reducing fear responses to trauma-related, conditioned stimuli. Indeed, engagement in exposure therapy may lead to increases in reward acquisition via increased engagement in rewarding activities, strengthening of interpersonal relationships through *in vivo* exposure, and increases in mastery, accomplishment, and perceived self-efficacy by completing challenging exposure exercises (Foa et al., 2019; Hoffman et al., 2022; Winer et al., 2017).

Across measurement modalities of self-report and observer ratings of in-session patient processes, changes in positive and negative affect and between positive and negative systems of psychotherapy change processes were reciprocally related, such that decreases in negative processes begat subsequent increases in positive processes, and vice versa. This consistent pattern across indices suggests that the improvements in positive and negative processes observed over the course of PE may be functionally linked. Indeed, PTSD may be characterized by an imbalance of approach and avoidance (Stein & Paulus, 2009), and PE may help individuals with PTSD achieve a more adaptive homeostasisbetween pursuing rewards and avoiding threats. With respect to self-reported PA and NA, decreases in negative responses to trauma cues and threat expectancies accomplished through *in vivo* and imaginal exposure may result in decreases in excessive avoidance motivation in the context of potential reward acquisition. Increased contact with rewards through behavioral activation, improvements in perceived coping abilities

and self-efficacy (Benight & Bandura, 2004; Cyniak-Cieciura et al., 2015), and strengthening of interpersonal bonds may result in increases in diminished approach motivation. With respect to in-session psychotherapy processes, increase in positive processes may be an especially strong indicator of favorable progress in treatment, and increases in hope, positive self-views, and adaptive insights may empower patients to overcome problems or challenges encountered during PE and indexed by elevated negative psychotherapy processes. Medication augmentation may enhance the benefits of increases in positive psychotherapy processes, possibly by boosting subjective reward perception and encoding and rehearsal of new learning during post-exposure processing (Michely et al., 2020; Zbozinek & Craske, 2017).

Notably, improvements in PTSD preceded changes in affect and psychotherapy processes. Reciprocal, mutually inhibitory relationships between positive and negative processes appear to be a robust characteristic of therapeutic change in PE with or without SSRI augmentation, consistent across both self-report and in-session change coding. One logical conclusion from these consistent findings is that targeting trauma-specific processes may be more effective than more broadly targeting positive and negative processes in general, as has been suggested by some (Barlow et al., 2021). Furthermore, examining the role of interactions or transactions between positive and negative processes and subsequent PTSD change may yield greater insight into treatment processes compared to examining positive and negative processes independently with respect to PTSD. The facilitative effect that decreases in NA may have on subsequent increases in PA, together with the effect increases in positive psychotherapy processes may have on patients ability to overcome challenges during treatment, may contribute to building and maintaining adaptive engagement in *in vivo* and imaginal exposure and accompanying improvements in PTSD. Additional research clarifying the processes through

which decreases in negative processes facilitate subsequent increases in positive processes, and vice versa, will inform treatment strategies to augment this effect, which may in turn enhance treatment response. Candidate processes of reciprocity between positive and negative processes may include the role of negative affect interference on positive affect (DePierro et al., 2018), enhancement of inhibitory learning via induction of positive affect (Zbozinek & Craske, 2017), enhancement of approach motivation via direct treatment of anhedonia (Craske et al., 2023), and the neural circuits supporting integration of approach and avoidance, particularly limbic, striatal, and orbitofrontal cortex activity (Aupperle & Paulus, 2010; Nawijn et al., 2015).

Finally, across both studies, there was significant between-patient variability in the relationships between positive and negative processes and PTSD symptoms. In other words, though on average affect and psychotherapy processes followed changes in PTSD, for some individuals the relationship may be reversed, and changes in affect and psychotherapy processes may in fact drive subsequent changes in PTSD. This highlights the importance of both examining within-person changes in addition to between changes, and of elucidating moderators of these relationships. Depression severity may be one such moderator (Clark & Watson, 1991; Peskin et al., 2019; Zoellner et al., 2014), and advances in computational strategies to identify multivariate profiles predicting treatment response may also aid in this work (e.g., DeRubeis, Cohen, et al., 2014; van Bronswijk et al., 2021).

Strengths of these studies include the comparison of a gold-standard psychotherapy for PTSD with and without pharmacological augmentation, session-by-session examination of changes in affect and psychotherapy processes and their relationship to changes in PTSD symptoms, and an analytic approach that facilitates examination of within-person effects and the testing of reciprocal temporal relationships within a single model. Previous studies of temporal

patterns of change during exposure therapy for PTSD have relied on MLM approaches (e.g., Cooper, Zoellner, et al., 2017), but MLM may yield biased estimates of cross-lagged effects compared to DSEM (Falkenström et al., 2022). Previous studies of change processes in exposure therapy have frequently relied on pre-post changes or only a limited number of measurements taken during the course of treatment, limiting insight into session-to-session patterns of change that may be most salient to clinicians. A key strength of this study is the multimodal assessment of putative change processes, the importance of which is reinforced by differences in the relationships between positive and negative affect and psychotherapy processes and their associations with PTSD symptoms. Other strengths include strong clinical measurement, standardized interventions, and experimental controls within a well-designed randomized clinical trial.

Despite its strengths, findings from this study should be considered in light of its limitations. The use of a primary PTSD sample, trauma-focused exposure therapy, and a single SSRI medication may limit generalizability to other anxiety, depression, and other traumatic stressor-related disorders, as well as to other forms of psycho- and pharmacotherapy. However, processes theorized to underlie exposure therapy are thought to operate transdiagnostically (Craske et al., 2012), and other SSRIs and other classes of antidepressants are generally thought to operate along shared mechanisms (Harmer et al., 2017; MacNamara et al., 2016). Though the inclusion PE with and without sertraline augmentation facilitates inferences about the impact of sertraline on change processes, the lack of a control condition, for instance of non-trauma focused treatment, precludes causal inferences about the impact of PE. That said, PE is a well-established treatment, with strong evidence of efficacy compared across psychotherapies (e.g., Sakaluk et al., 2019), arguing that the observed changes can be attributed to PE. Further, the

relationship between PE and sertraline has been studied in two larger randomized controlled trials showing efficacy (Rauch et al., 2019; Zoellner et al., 2019) and it is reasonable to benchmark observed changes in self-reported PTSD severity seen in this study (Table 3) with those trials. Finally, this study examined session-to-session changes in average levels of PA and NA and in psychotherapy processes; studies examining affective dynamics over even shorter time scales using intensive longitudinal data may yield different insights into the role of affective processes during PTSD treatment (Wichers et al., 2015).

Observer-rated measures of psychotherapeutic processes are few, likely in large part due to the increased resources required to implement them. Observer-rated measures may come with other limitations compared to self-report. For instance, distributions of individual CHANGE variables were highly skewed and suffered from low base rates of observed phenomena. Ultimately, some of this is inherent when studying patient behaviors during individual therapy sessions. Even if a patient behavior occurs with low frequency does not mean it cannot have a substantial impact on therapeutic change. Indeed, assessing these processes in-session, as they occur in real-time, may be the most proximal way to study change during psychotherapy. Further, observer-rated measures may complement self-report and physiological measurement to provide a more holistic portrait of change processes. Coding across the whole session, rather than the processing of imaginal exposure, specifically, may have improved statistical properties of the CHANGE variables; however, studying processing of imaginal exposure was a central aim of this study due to the relative dearth of research about it compared to exposure exercises themselves. More problematic is the possibility that these studies were underpowered to detect smaller effects (Schultzberg & Muthén, 2018), which may have obscured potential relationships

between affect and psychotherapy processes and subsequent changes in PTSD symptoms, or additional treatment moderation effects.

Clinically, therapists should be reassured that standard PE leads to substantial improvements in both positive and negative emotions as well as positive and negative in-session patient emotions, cognitions, and processing styles. Increases in positive psychotherapy processes during post-exposure processing, in particular, may be a powerful sign to clinicians that recovery is taking place and may also equip patients to better navigate challenges encountered during treatment. On the flipside, increases in negative psychotherapy processes during post-exposure processing, such as increased avoidance, unproductive processing, hopelessness, and negative self-views, may function as an alarm for potential problems in therapy and the need for course-correction. SSRI augmentation of PE may be considered for patients experiencing difficulties in PE, those this requires further study. Finally, self-report and observer-rated, in-session psychotherapy process variables may provide complementary information about patients' recovery during prolonged exposure for PTSD.

References

Abel, A., Hayes, A. M., Henley, W., & Kuyken, W. (2016). Sudden gains in cognitive–behavior therapy for treatment-resistant depression: Processes of change. *Journal of Consulting and Clinical Psychology*, *84*(8), 726–737. https://doi.org/10.1037/ccp0000101

Alexandra Kredlow, M., Fenster, R. J., Laurent, E. S., Ressler, K. J., & Phelps, E. A. (2022). Prefrontal cortex, amygdala, and threat processing: Implications for PTSD. *Neuropsychopharmacology*, *47*(1), 247–259. https://doi.org/10.1038/s41386-021-01155-7

Allard, C. B., Norman, S. B., Straus, E., Kim, H. M., Stein, M. B., Simon, N. M., Rauch, S. A. M., & Team, Prog. S. (2021). Reductions in guilt cognitions following prolonged exposure and/or sertraline predict subsequent improvements in PTSD and depression. *Journal of Behavior Therapy and Experimental Psychiatry*, 101666.

Alpert, E., Hayes, A. M., Yasinski, C., Webb, C., & Deblinger, E. (2021). Processes of change in trauma-focused cognitive behavioral therapy for youths: An approach informed by Emotional Processing Theory. *Clinical Psychological Science*, *9*(2), 270–283. https://doi.org/10.1177/2167702620957315

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). American Psychiatric Association. https://doi.org/10.1176/appi.books.9780890425596

American Psychological Association. (2019). Summary of the clinical practice guideline for the treatment of posttraumatic stress disorder (PTSD) in adults. *American Psychologist*, *74*(5), 596–607. https://doi.org/10.1037/amp0000473

Asparouhov, T., Hamaker, E. L., & Muthén, B. (2018). Dynamic structural equation models. *Structural Equation Modeling: A Multidisciplinary Journal*, *25*(3), 359–388.

https://doi.org/10.1080/10705511.2017.1406803

Aupperle, R. L., & Paulus, M. P. (2010). Neural systems underlying approach and avoidance in anxiety disorders. *Dialogues in Clinical Neuroscience*, *12*(4), 517–531. https://doi.org/https://doi.org/10.31887/DCNS.2010.12.4/raupperle

Badour, C. L., Blonigen, D. M., Boden, M. T., Feldner, M. T., & Bonn-Miller, M. O. (2012). A longitudinal test of the bi-directional relations between avoidance coping and PTSD severity during and after PTSD treatment. *Behaviour Research and Therapy*, *50*(10), 610–616. https://doi.org/10.1016/j.brat.2012.06.006

Badour, C. L., Resnick, H. S., & Kilpatrick, D. G. (2017). Associations between specific negative emotions and DSM-5 PTSD among a national sample of interpersonal trauma survivors. *Journal of Interpersonal Violence*, *32*(11), 1620–1641. https://doi.org/10.1177/0886260515589930

Baier, A. L., Kline, A. C., & Feeny, N. C. (2020). Therapeutic alliance as a mediator of change: A systematic review and evaluation of research. *Clinical Psychology Review*, *82*(November 2019), 101921. https://doi.org/10.1016/j.cpr.2020.101921

Bandelow, B., Sher, L., Bunevicius, R., Hollander, E., Kasper, S., Zohar, J., Möller, H.-J., Thibaut, F., Baranska-Rybak, W., Cubala, W. J., Fiellin, D., Kranzler, H. R., Moore, A., Rankans, E., Rasmussen, J., Saitz, R., Saravane, D., Schlaepfer, T. E., Tang, S. W., … Vega, J. (2012). Guidelines for the pharmacological treatment of anxiety disorders, obsessive-compulsive disorder and posttraumatic stress disorder in primary care. *International Journal of Psychiatry in Clinical Practice*, *16*(2), 77–84. https://doi.org/10.3109/13651501.2012.667114

Barlow, D. H., Curreri, A. J., & Woodard, L. S. (2021). Neuroticism and disorders of emotion: A

new synthesis. *Current Directions in Psychological Science*, *30*(5), 410–417. https://doi.org/10.1177/09637214211030253

Barlow, D. H., Sauer-Zavala, S., Carl, J. R., Bullis, J. R., & Ellard, K. K. (2014). The nature, diagnosis, and treatment of neuroticism. *Clinical Psychological Science*, *2*(3), 344–365. https://doi.org/10.1177/2167702613505532

Beckham, J. C., Feldman, M. E., Barefoot, J. C., Fairbank, J. A., Helms, M. J., Haney, T. L., Hertzberg, M. A., Moore, S. D., & Davidson, J. R. T. (2000). Ambulatory cardiovascular activity in Vietnam combat veterans with and without posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *68*(2), 269–276. https://doi.org/10.1037/0022-006X.68.2.269

Benedek, D. M., Friedman, M. J., Zatzick, D., & Ursano, R. J. (2009). Guideline watch (March 2009): Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder. *Focus*, *7*(2), 204–213.

Benight, C. C., & Bandura, A. (2004). Social cognitive theory of posttraumatic recovery: The role of perceived self-efficacy. *Behaviour Research and Therapy*, *42*(10), 1129–1148. https://doi.org/10.1016/j.brat.2003.08.008

Besnard, A., & Sahay, A. (2016). Adult hippocampal neurogenesis, fear generalization, and stress. *Neuropsychopharmacology*, *41*(1), 24–44. https://doi.org/10.1038/npp.2015.167

Bluett, E. J., Zoellner, L. A., & Feeny, N. C. (2014). Does change in distress matter? Mechanisms of change in prolonged exposure for PTSD. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*(1), 97–104. https://doi.org/10.1016/j.jbtep.2013.09.003

Bonanno, G. A., & Kaltman, S. (1999). Toward an integrative perspective on bereavement. *Psychological Bulletin*, *125*(6), 760–776. https://doi.org/10.1037/0033-2909.125.6.760

Bouton, M. E. (1993). Context, time, and memory retrieval in the interference paradigms of Pavlovian learning. *Psychological Bulletin*, *114*(1), 80–99. https://doi.org/10.1037/0033-2909.114.1.80

Bouton, M. E. (2000). A learning theory perspective on lapse, relapse, and the maintenance of behavior change. *Health Psychology*, *19*(1S), 57.

Bouton, M. E. (2004). Context and Behavioral Processes in Extinction. *Learning & Memory* , *11*(5), 485–494. https://doi.org/10.1101/lm.78804

Bouton, M. E., & Nelson, J. B. (1998). The role of context in classical conditioning: Some implications for cognitive behavior therapy. *Learning and Behavior Therapy*, 59–84.

Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A dual theory of posttraumatic stress disorder. *Psychological Review*, *103*(4), 670–686.

Brown, L. A., Zandberg, L. J., & Foa, E. B. (2019). Mechanisms of change in prolonged exposure therapy for PTSD: Implications for clinical practice. *Journal of Psychotherapy Integration*, *29*(1), 6–14. https://doi.org/10.1037/int0000109

Buchholz, J. L., & Abramowitz, J. S. (2020). The therapeutic alliance in exposure therapy for anxiety-related disorders: A critical review. *Journal of Anxiety Disorders*, *70*, 102194. https://doi.org/10.1016/j.janxdis.2020.102194

Cahill, S. P., Rauch, S. A., Hembree, E. A., & Foa, E. B. (2003). Effect of Cognitive-Behavioral Treatments for PTSD on Anger. *Journal of Cognitive Psychotherapy*, *17*(2), 113–131. https://doi.org/10.1891/jcop.17.2.113.57434

Carmassi, C., Akiskal, H. S., Bessonov, D., Massimetti, G., Calderani, E., Stratta, P., Rossi, A., & Dell′Osso, L. (2014). Gender differences in DSM-5 versus DSM-IV-TR PTSD prevalence and criteria comparison among 512 survivors to the L′Aquila earthquake.

Journal of Affective Disorders, *160*, 55–61. https://doi.org/10.1016/j.jad.2014.02.028

Cernasov, P., Walsh, E. C., Kinard, J. L., Kelley, L., Phillips, R., Pisoni, A., Eisenlohr-Moul, T. A., Arnold, M., Lowery, S. C., Ammirato, M., Truong, K., Nagy, G. A., Oliver, J. A., Haworth, K., Smoski, M., & Dichter, G. S. (2021). Multilevel growth curve analyses of behavioral activation for anhedonia (BATA) and mindfulness-based cognitive therapy effects on anhedonia and resting-state functional connectivity: Interim results of a randomized trial. *Journal of Affective Disorders*, *292*, 161–171. https://doi.org/10.1016/j.jad.2021.05.054

Cheavens, J. S., Feldman, D. B., Woodward, J. T., & Snyder, C. R. (2006). Hope in cognitive psychotherapies: On working with client strengths. *Journal of Cognitive Psychotherapy*, *20*(2), 135–145.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression : Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*(3), 316–336.

Comer, A. M., & Figgitt, D. P. (2000). Sertraline: A review of its therapeutic use in post-traumatic stress disorder. *CNS Drugs*, *14*(5), 391–407. https://doi.org/10.2165/00023210-200014050-00006

Cooper, A. A., Clifton, E. G., & Feeny, N. C. (2017). An empirical review of potential mediators and mechanisms of prolonged exposure therapy. *Clinical Psychology Review*, *56*, 106–121. https://doi.org/10.1016/j.cpr.2017.07.003

Cooper, A. A., Zoellner, L. A., Roy-Byrne, P., Mavissakalian, M. R., & Feeny, N. C. (2017). Do changes in trauma-related beliefs predict PTSD symptom improvement in prolonged exposure and sertraline? *Journal of Consulting and Clinical Psychology*, *85*(9), 873–882. https://doi.org/10.1037/ccp0000220

Cox, K. S., Wangelin, B. C., Keller, S. M., Lozano, B. E., Murphy, M. M., Maher, E. K., Cobb, A. R., & Tuerk, P. W. (2020). Emotional processing of imaginal exposures predicts symptom improvement: Therapist ratings can assess trajectory in prolonged exposure for posttraumatic stress disorder. *Journal of Traumatic Stress*. https://doi.org/10.1002/jts.22493

Cramer, A. O. J., van Borkulo, C. D., Giltay, E. J., van der Maas, H. L. J., Kendler, K. S., Scheffer, M., & Borsboom, D. (2016). Major Depression as a Complex Dynamic System. *Plos One*, *11*(12), e0167490. https://doi.org/10.1371/journal.pone.0167490

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, *46*(1), 5–27. https://doi.org/10.1016/j.brat.2007.10.003

Craske, M. G., Liao, B., Brown, L., & Vervliet, B. (2012). Role of inhibition in exposure therapy. *Journal of Experimental Psychopathology*, *3*(3), 322–345. https://doi.org/10.5127/jep.026511

Craske, M. G., Meuret, A. A., Ritz, T., Treanor, M., & Dour, H. J. (2016). *Treatment for Anhedonia: A Neuroscience Driven Approach*. *33*, 927–938. https://doi.org/10.1002/da.22490

Craske, M. G., Meuret, A. E., Echiverri-Cohen, A., Rosenfield, D., & Ritz, T. (2023). Positive affect treatment targets reward sensitivity: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*. https://doi.org/10.1037/ccp0000805

Craske, M. G., Treanor, M., Conway, C. C., Zbozinek, T., & Vervliet, B. (2014). Maximizing exposure therapy: An inhibitory learning approach. *Behaviour Research and Therapy*, *58*(3), 10–23. https://doi.org/10.1016/j.brat.2014.04.006

Craske, M. G., Treanor, M., Dour, H., Meuret, A., & Ritz, T. (2019). Positive affect treatment for

depression and anxiety: A randomized clinical trial for a core feature of anhedonia. *Journal of Consulting and Clinical Psychology*, *87*(5), 457–471. https://doi.org/10.1037/ccp0000396

Creamer, M., Burgess, P., & Pattison, P. (1992). Reaction to trauma: A cognitive processing model. *Journal of Abnormal Psychology*, *101*(3), 452–459. https://doi.org/10.1037/0021-843X.101.3.452

Creswell, J. D., Lam, S., Stanton, A. L., Taylor, S. E., Bower, J. E., & Sherman, D. K. (2007). Does self-affirmation, cognitive processing, or discovery of meaning explain cancer-related health benefits of expressive writing? *Personality and Social Psychology Bulletin*, *33*(2), 238–250.

Cusack, K., Jonas, D. E., Forneris, C. A., Wines, C., Sonis, J., Middleton, J. C., Feltner, C., Brownley, K. A., Olmsted, K. R., Greenblatt, A., Weil, A., & Gaynes, B. N. (2016). Psychological treatments for adults with posttraumatic stress disorder: A systematic review and meta-analysis. *Clinical Psychology Review*, *43*, 128–141. https://doi.org/10.1016/j.cpr.2015.10.003

Cyniak-Cieciura, M., Popiel, A., & Zawadzki, B. (2015). General self-efficacy level and changes in negative posttraumatic cognitions and posttraumatic stress disorder (PTSD) symptoms among motor vehicle accident survivors after PTSD therapy. *Studia Psychologiczne*, *53*(1), 18–29.

Davis, G., Wortman, C. B., & Darri, C. (2000). Searching for meaning in loss: Are clinical assumptions correct? *Death Studies*, *24*(6), 497–540. https://doi.org/10.1080/07481180050121471

de Kleine, R. A., Smits, J. A. J., Hendriks, G.-J., Becker, E. S., & van Minnen, A. (2015). Extinction learning as a moderator of d-cycloserine efficacy for enhancing exposure therapy

in posttraumatic stress disorder. *Journal of Anxiety Disorders*, *34*, 63–67. https://doi.org/10.1016/j.janxdis.2015.06.005

DePierro, J., D'Andrea, W., Frewen, P., & Todman, M. (2018). Alterations in positive affect: Relationship to symptoms, traumatic experiences, and affect ratings. *Psychological Trauma: Theory, Research, Practice, and Policy*, *10*(5), 585–593. https://doi.org/10.1037/tra0000317

DeRubeis, R. J., Cohen, Z. D., Forand, N. R., Fournier, J. C., Gelfand, L. A., & Lorenzo-Luaces, L. (2014). The Personalized Advantage Index: Translating Research on Prediction into Individualized Treatment Recommendations. A Demonstration. *PLoS ONE*, *9*(1), e83875. https://doi.org/10.1371/journal.pone.0083875

DeRubeis, R. J., Gelfand, L. A., German, R. E., Fournier, J. C., & Forand, N. R. (2014). Understanding processes of change: How some patients reveal more than others-and some groups of therapists less-about what matters in psychotherapy. *Psychotherapy Research*, *24*(3), 419–428. https://doi.org/10.1080/10503307.2013.838654

Di Simplicio, M., Norbury, R., & Harmer, C. J. (2012). Short-term antidepressant administration reduces negative self-referential processing in the medial prefrontal cortex in subjects at risk for depression. *Molecular Psychiatry*, *17*(5), 503–510. https://doi.org/10.1038/mp.2011.16

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behavior Research and Therapy*, *38*, 319–345. https://doi.org/10.1016/S0005-7967(99)00123-0

Ehlers, A., & Clark, D. M. (2006). *Predictors of Chronic Posttraumatic Stress Disorder: Trauma Memories and Appraisals.*

Engelhard, I. M., Arntz, A., & Van den Hout, M. A. (2007). Low specificity of symptoms on the post-traumatic stress disorder (PTSD) symptom scale: A comparison of individuais with

PTSD, individuals with other anxiety disorders and individuals without psychopathology. *British Journal of Clinical Psychology*, *46*(4), 449–456. https://doi.org/10.1348/014466507X206883

Falkenström, F., Granström, F., & Holmqvist, R. (2013). Therapeutic alliance predicts symptomatic improvement session by session. *Journal of Counseling Psychology*, *60*(3), 317–328. https://doi.org/10.1037/a0032258

Falkenström, F., Solomonov, N., & Rubel, J. A. (2022). How to model and interpret cross-lagged effects in psychotherapy mechanisms of change research: A comparison of multilevel and structural equation models. *Journal of Consulting and Clinical Psychology*, *90*(5), 446–458. https://doi.org/10.1037/ccp0000727

Feurer, C., Francis, J., Ajilore, O., Craske, M. G., Phan, K. L., & Klumpp, H. (2021). Emotion regulation and repetitive negative thinking before and after CBT and SSRI treatment of internalizing psychopathologies. *Cognitive Therapy and Research*, *45*(6), 1064–1076. https://doi.org/10.1007/s10608-021-10222-8

First, M., Spitzer, R., Gibbon, M., & Willams, J. (1995). *Structured clinical interview for DSM-IV Axis I disorders - Patient edition v.2 (SCID-I/P)*. Biometrics Research Department.

Foa, E. B. (2011). Prolonged exposure therapy: Past, present, and future. *Depression and Anxiety*, *28*(12), 1043–1047. https://doi.org/10.1002/da.20907

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, *9*(4), 445–451. https://doi.org/10.1037/1040-3590.9.4.445

Foa, E. B., Hembree, E. A., Rothbaum, B. O., & Rauch, S. (2019). *Prolonged Exposure Therapy for PTSD: Emotional Processing of Traumatic Experiences - Therapist Guide* (2 edn).

Oxford University Press. https://doi.org/10.1093/med-psych/9780190926939.001.0001

Foa, E. B., Hembree, E., & Rothbaum, B. (2007). *Prolonged Exposure Therapy for PTSD: Therapist Guide*. Oxford University Press. https://doi.org/10.1093/med:psych/9780195308501.001.0001

Foa, E. B., Huppert, J. D., & Cahill, S. P. (2006). Emotional processing theory: An update. In *Pathological anxiety: Emotional processing in etiology and treatment.* (pp. 3–24). The Guilford Press.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*(1), 20–35. https://doi.org/10.1037/0033-2909.99.1.20

Foa, E. B., & McLean, C. P. (2016). The Efficacy of Exposure Therapy for Anxiety-Related Disorders and Its Underlying Mechanisms: The Case of OCD and PTSD. *Annual Review of Clinical Psychology*, *12*(1), 1–28. https://doi.org/10.1146/annurev-clinpsy-021815-093533

Foa, E. B., McLean, C. P., Zang, Y., Zhong, J., Rauch, S., Porter, K., Knowles, K., Powers, M. B., & Kauffman, B. Y. (2016). Psychometric properties of the Posttraumatic Stress Disorder Symptom Scale Interview for DSM–5 (PSSI–5). *Psychological Assessment*, *28*(10), 1159–1165. https://doi.org/10.1037/pas0000259

Foa, E. B., & McNally, R. J. (1996). Mechanisms of change in exposure therapy. *Current Controversies in the Anxiety Disorders*, 329–343.

Foa, E. B., & Rauch, S. A. M. (2004). Cognitive changes during prolonged exposure versus prolonged exposure plus cognitive restructuring in female assault survivors with posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *72*(5), 879.

Foa, E. B., Riggs, D. S., Massie, E. D., & Yarczower, M. (1995). The impact of fear activation

and anger on the efficacy of exposure treatment for posttraumatic stress disorder. *Behavior Therapy*, *26*(3), 487–499. https://doi.org/10.1016/S0005-7894(05)80096-6

Foa, E. B., Tolin, D. F., Ehlers, A., Clark, D. M., & Orsillo, S. M. (1999). The Posttraumatic Cognitions Inventory (PTCI): Development and validation. *Psychological Assessment*, *11*(3), 303–314. https://doi.org/10.1037/1040-3590.11.3.303

Forbes, D., Parslow, R., Creamer, M., Allen, N., McHugh, T., & Hopwood, M. (2008). Mechanisms of anger and treatment outcome in combat veterans with posttraumatic stress disorder. *Journal of Traumatic Stress*, *21*(2), 142–149. https://doi.org/10.1002/jts.20315

Franklin, C. L., & Zimmerman, M. (2001). Posttraumatic stress disorder and major depressive disorder: Investigating the role of overlapping symptoms in diagnostic comorbidity. *The Journal of Nervous and Mental Disease*, *189*(8), 548–551. https://doi.org/10.1097/00005053-200108000-00008

Fried, E. I., van Borkulo, C. D., Cramer, A. O. J., Boschloo, L., Schoevers, R. A., & Borsboom, D. (2016). Mental disorders as networks of problems: a review of recent insights. *Social Psychiatry and Psychiatric Epidemiology*, *52*(1), 1–10. https://doi.org/10.1007/s00127-016-1319-z

Furlong, T. M., Richardson, R., & McNally, G. P. (2016). Habituation and extinction of fear recruit overlapping forebrain structures. *Neurobiology of Learning and Memory*, *128*, 7–16. https://doi.org/10.1016/j.nlm.2015.11.013

Gallagher, M. W., & Resick, P. A. (2012). Mechanisms of change in cognitive processing therapy and prolonged exposure therapy for PTSD: Preliminary evidence for the differential effects of hopelessness and habituation. *Cognitive Therapy and Research*, *36*(6), 750–755. https://doi.org/10.1007/s10608-011-9423-6

Garland, E. L., Fredrickson, B., Kring, A. M., Johnson, D. P., Meyer, P. S., & Penn, D. L. (2010). Upward spirals of positive emotions counter downward spirals of negativity: Insights from the broaden-and-build theory and affective neuroscience on the treatment of emotion dysfunctions and deficits in psychopathology. *Clinical Psychology Review*, *30*(7), 849–864. https://doi.org/10.1016/j.cpr.2010.03.002

Geraghty, A. W. A., Wood, A. M., & Hyland, M. E. (2010). Dissociating the facets of hope: Agency and pathways predict dropout from unguided self-help therapy in opposite directions. *Journal of Research in Personality*, *44*(1), 155–158.

Gillihan, S. J., & Foa, E. B. (2011). Fear Extinction and Emotional Processing Theory. In *Associative Learning and Conditioning Theory: Human and Non-Human Applications*. Oxford University Press. https://doi.org/10.1093/acprof:oso/9780199735969.003.0017

Gilman, R., Schumm, J. A., & Chard, K. M. (2012). Hope as a change mechanism in the treatment of posttraumatic stress disorder. *Psychological Trauma: Theory, Research, Practice, and Policy*, *4*(3), 270–277. https://doi.org/10.1037/a0024252

Goldstein, R. B., Smith, S. M., Chou, S. P., Saha, T. D., Jung, J., Zhang, H., Pickering, R. P., Ruan, W. J., Huang, B., & Grant, B. F. (2016). The epidemiology of DSM-5 posttraumatic stress disorder in the United States: results from the National Epidemiologic Survey on Alcohol and Related Conditions-III. *Social Psychiatry and Psychiatric Epidemiology*, *51*(8), 1137–1148. https://doi.org/10.1007/s00127-016-1208-5

Gómez Penedo, J. M., Hilpert, P., grosse Holtforth, M., & Flückiger, C. (2021). Interpersonal cognitions as a mechanism of change in cognitive behavioral therapy for generalized anxiety disorder: A multilevel dynamic structural equation model approach. *Journal of Consulting and Clinical Psychology*, *89*(11), 898–908. https://doi.org/10.1037/ccp0000690

Graham, B., Garcia, N. M., Bergman, H. E., Feeny, N. C., & Zoellner, L. A. (2020). Prolonged Exposure and Sertraline Treatments for Posttraumatic Stress Disorder Also Improve Multiple Indicators of Social Functioning. *Journal of Traumatic Stress*, *33*(4), 488–499.

Grillon, C. (2008). Models and mechanisms of anxiety: evidence from startle studies. *Psychopharmacology*, *199*(3), 421–437. https://doi.org/10.1007/s00213-007-1019-1

Grosse Holtforth, M., Hayes, A. M., Sutter, M., Wilm, K., Schmied, E., Laurenceau, J.-P., & Caspar, F. (2012). Fostering Cognitive-Emotional Processing in the Treatment of Depression: A Preliminary Investigation in Exposure-Based Cognitive Therapy. *Psychotherapy and Psychosomatics*, *81*(4), 259–260. https://doi.org/10.1159/000336813

Hagenaars, M A, & Van Minnen, A. (2010). Posttraumatic growth in exposure therapy for PTSD. *Journal of Traumatic Stress*, *23*(4), 504–508. https://doi.org/10.1002/jts.20551

Hagenaars, Muriel A, Minnen, A. van, & De Rooij, M. J. (2010). *Cognitions in prolonged exposure therapy for posttraumatic stress disorder*.

Harmer, C. J. (2008). Serotonin and emotional processing: does it help explain antidepressant drug action? *Neuropharmacology*, *55*(6), 1023–1028. https://doi.org/https://doi.org/10.1016/j.neuropharm.2008.06.036

Harmer, C. J. (2012). *Emotional Processing and Antidepressant Action* (pp. 209–222). https://doi.org/10.1007/7854_2012_210

Harmer, C. J., Duman, R. S., & Cowen, P. J. (2017). How do antidepressants work? New perspectives for refining future treatment approaches. *The Lancet Psychiatry*, *4*(5), 409–418. https://doi.org/https://doi.org/10.1016/S2215-0366(17)30015-9

Harned, M. S., Ruork, A. K., Liu, J., & Tkachuck, M. A. (2015). Emotional activation and habituation during imaginal exposure for PTSD among women with borderline personality

disorder. *Journal of Traumatic Stress*, *28*(3), 253–257. https://doi.org/10.1002/jts.22013

Hayes, A. M., Beevers, C. G., Feldman, G. C., Laurenceau, J.-P., & Perlman, C. (2005). Avoidance and processing as predictors of symptom change and positive growth in an integrative therapy for depression. *International Journal of Behavioral Medicine*, *12*(2), 111–122. https://doi.org/10.1207/s15327558ijbm1202_9

Hayes, A. M., Beevers, C. G., Feldman, G. C., Laurenceau, J. P., & Perlman, C. (2014). Avoidance and processing as predictors of symptom change and positive growth in an integrative therapy for depression. *An Exploration of the Health Benefits of Factors That Help Us to Thrive: A Special Issue of the International Journal of Behavioral Medicine*, *12*(2), 111–122. https://doi.org/10.4324/9781315799315-9

Hayes, A. M., Feldman, G. C., Beevers, C. G., Laurenceau, J.-P., Cardaciotto, L., & Lewis-Smith, J. (2007). Discontinuities and cognitive changes in an exposure-based cognitive therapy for depression. *Journal of Consulting and Clinical Psychology*, *75*(3), 409–421. https://doi.org/10.1037/0022-006X.75.3.409

Hayes, A. M., Feldman, G. C., & Goldfried, M. R. (2007). The Change and Growth Experiences Scale: A Measure of Insight and Emotional Processing. In *Insight in psychotherapy.* (pp. 231–253). American Psychological Association. https://doi.org/10.1037/11532-011

Hayes, A. M., Hope, D. A., & Hayes, S. (2007). Towards an understanding of the process and mechanisms of change in cognitive behavioral therapy: Linking innovative methodology with fundamental questions. *Clinical Psychology Review*, *27*(6), 679–681. https://doi.org/10.1016/j.cpr.2007.01.006

Hayes, A. M., Laurenceau, J. P., Feldman, G., Strauss, J. L., & Cardaciotto, L. (2007). Change is not always linear: The study of nonlinear and discontinuous patterns of change in

psychotherapy. *Clinical Psychology Review*. https://doi.org/10.1016/j.cpr.2007.01.008

Hayes, A. M., & Yasinski, C. (2015). Pattern destabilization and emotional processing in cognitive therapy for personality disorders. *Frontiers in Psychology*, *6*(FEB), 1–13. https://doi.org/10.3389/fpsyg.2015.00107

Hayes, A. M., Yasinski, C., Ben Barnes, J., & Bockting, C. L. H. (2015). Network destabilization and transition in depression: New methods for studying the dynamics of therapeutic change. In *Clinical Psychology Review*. https://doi.org/10.1016/j.cpr.2015.06.007

Hayes, A. M., Yasinski, C., Grasso, D., Ready, C. B., Alpert, E., McCauley, T., Webb, C., & Deblinger, E. (2017). Constructive and unproductive processing of traumatic experiences in trauma-focused cognitive-behavioral therapy for youth. *Behavior Therapy*, *48*(2), 166–181. https://doi.org/10.1016/j.beth.2016.06.004

Hayes, S. C., Wilson, K. G., Gifford, E. V., Follette, V. M., & Strosahl, K. (1996). Experiential avoidance and behavioral disorders: A functional dimensional approach to diagnosis and treatment. *Journal of Consulting and Clinical Psychology*, *64*(6), 1152–1168. https://doi.org/10.1037/0022-006X.64.6.1152

Heesbeen, E. J., Bijlsma, E. Y., Verdouw, P. M., van Lissa, C., Hooijmans, C., & Groenink, L. (2023). The effect of SSRIs on fear learning: a systematic review and meta-analysis. *Psychopharmacology*. https://doi.org/10.1007/s00213-023-06333-7

Hetrick, S. E., Purcell, R., Garner, B., & Parslow, R. (2010). Combined pharmacotherapy and psychological therapies for post traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews*. https://doi.org/10.1002/14651858.CD007316.pub2

Hoffman, S. N., Lyons, R. C., Stein, M. B., Taylor, C. T., & Norman, S. B. (2022). Changes in positive and negative affect following prolonged exposure for PTSD comorbid with alcohol

use disorder: Secondary analysis of a randomized clinical trial. *Behaviour Research and Therapy*, *155*, 104097. https://doi.org/10.1016/j.brat.2022.104097

Hollenstein, T. (2015). This time, it's real: Affective flexibility, time scales, feedback loops, and the regulation of emotion. *Emotion Review*, *7*(4), 308–315. https://doi.org/10.1177/1754073915590621

Hunt, M., Schloss, H., Moonat, S., Poulos, S., & Wieland, J. (2007). Emotional processing versus cognitive restructuring in response to a depressing life event. *Cognitive Therapy and Research*, *31*(6), 833–851.

Husen, K., Rafaeli, E., Rubel, J. A., Bar-Kalifa, E., & Lutz, W. (2016). Daily affect dynamics predict early response in CBT: Feasibility and predictive validity of EMA for outpatient psychotherapy. *Journal of Affective Disorders*, *206*, 305–314. https://doi.org/10.1016/j.jad.2016.08.025

Indovina, I., Robbins, T. W., Núñez-Elizalde, A. O., Dunn, B. D., & Bishop, S. J. (2011). Fear-Conditioning Mechanisms Associated with Trait Vulnerability to Anxiety in Humans. *Neuron*, *69*(3), 563–571. https://doi.org/10.1016/j.neuron.2010.12.034

Janoff-Bulman, R. (1992). Shattered assumptions: Towards a new psychology of trauma. In *Shattered assumptions: Towards a new psychology of trauma.* (pp. xii, 256–xii, 256). Free Press.

Jeffreys, M., Capehart, B., & Friedman, M. J. (2012). Pharmacotherapy for posttraumatic stress disorder: Review with clinical applications. *Journal of Rehabilitation Research and Development*, *49*(5), 703–716. https://doi.org/10.1682/JRRD.2011.09.0183

Jerud, A. B., Zoellner, L. A., Pruitt, L. D., & Feeny, N. C. (2014). Changes in emotion regulation in adults with and without a history of childhood abuse following posttraumatic stress

disorder treatment. *Journal of Consulting and Clinical Psychology*, *82*(4), 721–730. https://doi.org/10.1037/a0036520

Joseph, S., & Linley, P. A. (2005). Positive Adjustment to Threatening Events: An Organismic Valuing Theory of Growth through Adversity. *Review of General Psychology*, *9*(3), 262–280. https://doi.org/10.1037/1089-2680.9.3.262

Jovanovic, T., Norrholm, S. D., Blanding, N. Q., Davis, M., Duncan, E., Bradley, B., & Ressler, K. J. (2010). Impaired fear inhibition is a biomarker of PTSD but not depression. *Depression and Anxiety*, *27*(3), 244–251. https://doi.org/10.1002/da.20663

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, *3*(1), 1–27. https://doi.org/10.1146/annurev.clinpsy.3.022806.091432

Kernan, W. D., & Lepore, S. J. (2009). Searching for and making meaning after breast cancer: Prevalence, patterns, and negative affect. *Social Science & Medicine*, *68*(6), 1176–1182.

Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H.-U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research*, *21*(3), 169–184. https://doi.org/10.1002/mpr.1359

Kilpatrick, D. G., Resnick, H. S., Milanak, M. E., Miller, M. W., Keyes, K. M., & Friedman, M. J. (2013). National Estimates of Exposure to Traumatic Events and PTSD Prevalence Using DSM-IV and DSM-5 Criteria. *Journal of Traumatic Stress*, *26*(5), 537–547. https://doi.org/10.1002/jts.21848

Kozak, M. J., & Cuthbert, B. N. (2016). The NIMH Research Domain Criteria Initiative: Background, Issues, and Pragmatics. *Psychophysiology*, *53*(3), 286–297.

https://doi.org/10.1111/psyp.12518

Kumpula, M. J., Pentel, K. Z., Foa, E. B., LeBlanc, N. J., Bui, E., McSweeney, L. B., Knowles, K., Bosley, H., Simon, N. M., & Rauch, S. A. M. (2017). Temporal sequencing of change in posttraumatic cognitions and PTSD symptom reduction during prolonged exposure therapy. *Behavior Therapy*, *48*(2), 156–165. https://doi.org/10.1016/j.beth.2016.02.008

Kuppens, P., & Verduyn, P. (2017). Emotion dynamics. *Current Opinion in Psychology*, *17*, 22–26. https://doi.org/10.1016/j.copsyc.2017.06.004

Lang, P. J. (1979). A Bio-Informational Theory of Emotional Imagery. *Psychophysiology*, *16*(6), 495–512. https://doi.org/10.1111/j.1469-8986.1979.tb01511.x

Lehman, D. R., Wortman, C. B., & Williams, A. F. (1987). Long-term effects of losing a spouse or child in a motor vehicle crash. *Journal of Personality and Social Psychology*, *52*(1), 218.

Lepore, S. J., & Helgeson, V. S. (1998). Social constraints, intrusive thoughts, and mental health. *Journal of Social and Clinical Psychology*, *17*(1), 89–106.

Litz, B. T., & Gray, M. J. (2002). Emotional numbing in posttraumatic stress disorder: Current and future research directions. *Australian & New Zealand Journal of Psychiatry*, *36*(2), 198–204. https://doi.org/10.1046/j.1440-1614.2002.01002.x

LoSavio, S. T., Dillon, K. H., & Resick, P. A. (2017). Cognitive factors in the development, maintenance, and treatment of post-traumatic stress disorder. *Current Opinion in Psychology*, *14*, 18–22. https://doi.org/10.1016/j.copsyc.2016.09.006

MacNamara, A., Rabinak, C. A., Kennedy, A. E., Fitzgerald, D. A., Liberzon, I., Stein, M. B., & Phan, K. L. (2016). Emotion regulatory brain function and SSRI treatment in PTSD: Neural correlates and predictors of change. *Neuropsychopharmacology*, *41*(2), 611–618. https://doi.org/10.1038/npp.2015.190

Martell, C. R., Addis, M. E., & Jacobson, N. S. (2001). Depression in context: Strategies for guided action. In *Depression in context: Strategies for guided action.* (pp. xxx, 223–xxx, 223). W W Norton & Co.

McLean, C. P., Su, Y.-J. J., & Foa, E. B. (2015). Mechanisms of symptom reduction in a combined treatment for comorbid posttraumatic stress disorder and alcohol dependence. *Journal of Consulting and Clinical Psychology*, *83*(3), 655–661. https://doi.org/10.1037/ccp0000024

McNally, R. J. (2016). Can network analysis transform psychopathology? *Behaviour Research and Therapy*. https://doi.org/10.1016/j.brat.2016.06.006

McNeish, D., & Hamaker, E. L. (2020). A primer on two-level dynamic structural equation models for intensive longitudinal data in Mplus. *Psychological Methods*, *25*(5), 610–635. https://doi.org/10.1037/met0000250

Michely, J., Eldar, E., Martin, I. M., & Dolan, R. J. (2020). A mechanistic account of serotonin's impact on mood. *Nature Communications*, *11*(1), 2335. https://doi.org/10.1038/s41467-020-16090-2

Milad, M. R., Pitman, R. K., Ellis, C. B., Gold, A. L., Shin, L. M., Lasko, N. B., Zeidan, M. A., Handwerger, K., Orr, S. P., & Rauch, S. L. (2009). Neurobiological Basis of Failure to Recall Extinction Memory in Posttraumatic Stress Disorder. *Biological Psychiatry*, *66*(12), 1075–1082. https://doi.org/10.1016/j.biopsych.2009.06.026

Miller, G. A. (2010). Mistreating psychology in the decades of the brain. *Perspectives on Psychological Science*, *5*(6), 716–743. https://doi.org/10.1177/1745691610388774

Moradveisi, L., Huibers, M. J. H., & Arntz, A. (2015). The influence of patients' attributions of the immediate effects of treatment of depression on long-term effectiveness of behavioural

activation and antidepressant medication. *Behaviour Research and Therapy*, *69*, 83–92. https://doi.org/10.1016/j.brat.2015.04.007

Muthén, L. K., & Muthén, B. O. (2017). *Mplus User's Guide* (Eigth). Muthén & Muthén.

Nacasch, N., Huppert, J. D., Su, Y.-J., Kivity, Y., Dinshtein, Y., Yeh, R., & Foa, E. B. (2015). Are 60-minute prolonged exposure sessions with 20-minute imaginal exposure to traumatic memories sufficient to successfully treat PTSD? A randomized noninferiority clinical trial. *Behavior Therapy*, *46*(3), 328–341. https://doi.org/10.1016/j.beth.2014.12.002

National Institute for Health and Care Excellence. (2018). *Post-traumatic stress disorder*. NICE Guideline No. 116. https://www.nice.org.uk/guidance/ng116

Nawijn, L., van Zuiden, M., Frijling, J. L., Koch, S. B. J., Veltman, D. J., & Olff, M. (2015). Reward functioning in PTSD: A systematic review exploring the mechanisms underlying anhedonia. *Neuroscience and Biobehavioral Reviews*, *51*, 189–204. https://doi.org/10.1016/j.neubiorev.2015.01.019

Neimeyer, R. A. (2001). *Meaning reconstruction & the experience of loss.* American Psychological Association.

Norton, P. J., & Price, E. C. (2007). A Meta-Analytic Review of Adult Cognitive-Behavioral Treatment Outcome Across the Anxiety Disorders. *Journal of Nervous & Mental Disease*, *195*(6), 521–531. https://doi.org/10.1097/01.nmd.0000253843.70149.9a

Øktedalen, T., Hoffart, A., & Langkaas, T. F. (2015). Trauma-related shame and guilt as time-varying predictors of posttraumatic stress disorder symptoms during imagery exposure and imagery rescripting—A randomized controlled trial. *Psychotherapy Research*, *25*(5), 518–532.

Park, C. L. (2010). Making sense of the meaning literature: an integrative review of meaning

making and its effects on adjustment to stressful life events. *Psychological Bulletin*, *136*(2), 257–301. https://doi.org/10.1037/a0018301

Peskin, M., Wyka, K., Cukor, J., Olden, M., Altemus, M., Lee, F. S., & Difede, J. (2019). The relationship between posttraumatic and depressive symptoms during virtual reality exposure therapy with a cognitive enhancer. *Journal of Anxiety Disorders*, *61*, 82–88. https://doi.org/10.1016/j.janxdis.2018.03.001

Pitman, R. K., Orr, S. P., Altman, B., Longpre, R. E., Poiré, R. E., Macklin, M. L., Michaels, M. J., & Steketee, G. S. (1996). Emotional processing and outcome of imaginal flooding therapy in vietnam veterans with chronic posttraumatic stress disorder. *Comprehensive Psychiatry*, *37*(6), 409–418. https://doi.org/10.1016/S0010-440X(96)90024-3

Pittig, A., Treanor, M., LeBeau, R. T., & Craske, M. G. (2018). The role of associative fear and avoidance learning in anxiety disorders: Gaps and directions for future research. *Neuroscience and Biobehavioral Reviews*, *88*(March), 117–140. https://doi.org/10.1016/j.neubiorev.2018.03.015

Pringle, A., Browning, M., Cowen, P. J., & Harmer, C. J. (2011). A cognitive neuropsychological model of antidepressant drug action. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *35*(7), 1586–1592. https://doi.org/10.1016/j.pnpbp.2010.07.022

Quirk, G. J., Milad, M. R., Santini, E., & Lebron, K. (2007). Learning not to fear: A neural systems approach. In *Understanding Trauma: Integrating Biological, Clinical, and Cultural Perspectives* (pp. 60–77). Cambridge University Press. https://doi.org/10.1017/CBO9780511500008.006

Rachman, S. (1980). Emotional processing. *Behaviour Research and Therapy*, *18*(1), 51–60.

Rauch, S. A. M., Eftekhari, A., & Ruzek, J. I. (2012). Review of exposure therapy: a gold standard for PTSD treatment. *Journal of Rehabilitation Research and Development*, *49*(5), 679–687. https://doi.org/10.1682/jrrd.2011.08.0152

Rauch, S. A. M., Foa, E. B., Furr, J. M., & Filip, J. C. (2004). Imagery vividness and perceived anxious arousal in prolonged exposure treatment for PTSD. *Journal of Traumatic Stress*, *17*(6), 461–465. https://doi.org/10.1007/s10960-004-5794-8

Rauch, S. A. M., Kim, H. M., Powell, C., Tuerk, P. W., Simon, N. M., Acierno, R., Allard, C. B., Norman, S. B., Venners, M. R., Rothbaum, B. O., Stein, M. B., Porter, K., Martis, B., King, A. P., Liberzon, I., Phan, K. L., & Hoge, C. W. (2019). Efficacy of prolonged exposure therapy, sertraline hydrochloride, and their combination among combat veterans with posttraumatic stress disorder. *JAMA Psychiatry*, *76*(2), 117. https://doi.org/10.1001/jamapsychiatry.2018.3412

Rauch, S. A. M., Koola, C., Post, L., Yasinski, C., Norrholm, S. D., Black, K., & Rothbaum, B. O. (2018). In session extinction and outcome in Virtual Reality Exposure Therapy for PTSD. *Behaviour Research and Therapy*, *109*, 1–9. https://doi.org/10.1016/j.brat.2018.07.003

Rauch, S., & Foa, E. (2006). Emotional Processing Theory (EPT) and Exposure Therapy for PTSD. *Journal of Contemporary Psychotherapy*, *36*(2), 61–65. https://doi.org/10.1007/s10879-006-9008-y

Ready, C. B., Hayes, A. M., Yasinski, C. W., Webb, C., Gallop, R., Deblinger, E., & Laurenceau, J.-P. (2015). Overgeneralized beliefs, accommodation, and treatment outcome in youth receiving trauma-focused cognitive behavioral therapy for childhood trauma. *Behavior Therapy*, *46*(5), 671–688. https://doi.org/10.1016/j.beth.2015.03.004

Resick, P. A., Galovski, T. E., Uhlmansiek, M. O., Scher, C. D., Clum, G. A., & Young-Xu, Y. (2008). A randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. *Journal of Consulting and Clinical Psychology*, *76*(2), 243.

Reynolds, M., & Brewin, C. R. (1999). Intrusive memories in depression and posttraumatic stress disorder. *Behaviour Research and Therapy*, *37*(3), 201–215. https://doi.org/10.1016/S0005-7967(98)00132-6

Roberts, C., Sahakian, B. J., & Robbins, T. W. (2020). Psychological mechanisms and functions of 5-HT and SSRIs in potential therapeutic change: Lessons from the serotonergic modulation of action selection, learning, affect, and social cognition. *Neuroscience & Biobehavioral Reviews*, *119*, 138–167. https://doi.org/10.1016/j.neubiorev.2020.09.001

Rothbaum, B. O., Cahill, S. P., Foa, E. B., Davidson, J. R. T., Compton, J., Connor, K. M., Astin, M. C., & Hahn, C.-G. (2006). Augmentation of sertraline with prolonged exposure in the treatment of posttraumatic stress disorder. *Journal of Traumatic Stress*, *19*(5), 625–638. https://doi.org/10.1002/jts.20170

Rothbaum, B. O., Price, M., Jovanovic, T., Norrholm, S. D., Gerardi, M., Dunlop, B., Davis, M., Bradley, B., Duncan, E. J., Rizzo, A., & Ressler, K. J. (2014). A Randomized, Double-Blind Evaluation of <scp>d</scp> -Cycloserine or Alprazolam Combined With Virtual Reality Exposure Therapy for Posttraumatic Stress Disorder in Iraq and Afghanistan War Veterans. *American Journal of Psychiatry*, *171*(6), 640–648. https://doi.org/10.1176/appi.ajp.2014.13121625

Rush, A. J., Trivedi, M. H., Ibrahim, H. M., Carmody, T. J., Arnow, B., Klein, D. N., Markowitz, J. C., Ninan, P. T., Kornstein, S., Manber, R., Thase, M. E., Kocsis, J. H., & Keller, M. B.

(2003). The 16-Item quick inventory of depressive symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression. *Biological Psychiatry*, *54*(5), 573–583. https://doi.org/10.1016/S0006-3223(02)01866-8

Sakaluk, J. K., Williams, A. J., Kilshaw, R. E., & Rhyner, K. T. (2019). Evaluating the evidential value of empirically supported psychological treatments (ESTs): A meta-scientific review. *Journal of Abnormal Psychology*, *128*(6), 500–509. https://doi.org/10.1037/abn0000421

Sasso, K. E., Strunk, D. R., Braun, J. D., DeRubeis, R. J., & Brotman, M. A. (2016). A re-examination of process–outcome relations in cognitive therapy for depression: Disaggregating within-patient and between-patient effects. *Psychotherapy Research*, *26*(4), 387–398. https://doi.org/10.1080/10503307.2015.1026423

Schaumberg, K., Kuerbis, A., Morgenstern, J., & Muench, F. (2013). Attributions of change and self-efficacy in a randomized controlled trial of medication and psychotherapy for problem drinking. *Behavior Therapy*, *44*(1), 88–99. https://doi.org/10.1016/j.beth.2012.07.001

Schneier, F. R., Neria, Y., Pavlicova, M., Hembree, E., Suh, E. J., Amsel, L., & Marshall, R. D. (2012). Combined prolonged exposure therapy and paroxetine for PTSD related to the World Trade Center attack: a randomized controlled trial. *American Journal of Psychiatry*, *169*(1), 80–88. https://doi.org/https://doi.org/10.1176/appi.ajp.2011.11020321

Schultzberg, M., & Muthén, B. (2018). Number of subjects and time points needed for multilevel time-series analysis: A simulation study of dynamic structural equation modeling. *Structural Equation Modeling: A Multidisciplinary Journal*, *25*(4), 495–515. https://doi.org/10.1080/10705511.2017.1392862

Shin, L. M., & Liberzon, I. (2010). The Neurocircuitry of Fear, Stress, and Anxiety Disorders.

Neuropsychopharmacology, *35*(1), 169–191. https://doi.org/10.1038/npp.2009.83

Silver, R. L., Boon, C., & Stones, M. H. (1983). Searching for meaning in misfortune: Making sense of incest. *Journal of Social Issues*, *39*(2), 81–101.

Simon, N. M., Connor, K. M., Lang, A. J., Rauch, S., Krulewicz, S., LeBeau, R. T., Davidson, J. R. T., Stein, M. B., Otto, M. W., Foa, E. B., & Pollack, M. H. (2008). Paroxetine CR augmentation for posttraumatic stress disorder refractory to prolonged exposure therapy. *The Journal of Clinical Psychiatry*, *69*(3), 400–405. https://doi.org/10.4088/JCP.v69n0309

Sloan, D. M., Marx, B. P., & Epstein, E. M. (2007). Does altering the writing instructions influence outcome associated with written disclosure? *Behavior Therapy*, *38*(2), 155–168.

Sripada, R. K., & Rauch, S. A. M. (2015). Between-session and within-session habituation in prolonged exposure therapy for posttraumatic stress disorder: A hierarchical linear modeling approach. *Journal of Anxiety Disorders*, *30*, 81–87. https://doi.org/10.1016/j.janxdis.2015.01.002

Stanton, A. L., Kirk, S. B., Cameron, C. L., & Danoff-Burg, S. (2000). Coping through emotional approach: scale construction and validation. *Journal of Personality and Social Psychology*, *78*(6), 1150.

Stapleton, J. A., Taylor, S., & Asmundson, G. J. G. (2006). Effects of three PTSD treatments on anger and guilt: Exposure therapy, eye movement desensitization and reprocessing, and relaxation training. *Journal of Traumatic Stress*, *19*(1), 19–28. https://doi.org/10.1002/jts.20095

Stein, M. B., & Paulus, M. P. (2009). Imbalance of approach and avoidance: The Yin and Yang of anxiety disorders. *Biological Psychiatry*, *66*(12), 1072–1074. https://doi.org/10.1016/j.biopsych.2009.09.023

Taylor, S. E. (1983). Adjustment to threatening events: A theory of cognitive adaptation. *American Psychologist*, *38*(11), 1161.

Tedeschi, R. G., Calhoun, L. G., & Groleau, J. M. (2015). Clinical Applications of Posttraumatic Growth. In *Positive Psychology in Practice* (pp. 503–518). John Wiley & Sons, Inc. https://doi.org/10.1002/9781118996874.ch30

Thome, H. (2014). Cointegration and error correction modelling in time-series analysis: A brief introduction. *International Journal of Conflict and Violence*, *8*(2), 199–208. https://doi.org/https://doi.org/10.4119/ijcv-3055

Tolin, D. F. (2010). Is cognitive–behavioral therapy more effective than other therapies?A meta-analytic review. *Clinical Psychology Review*, *30*(6), 710–720. https://doi.org/10.1016/j.cpr.2010.05.003

Trew, J. L. (2011). Exploring the roles of approach and avoidance in depression: An integrative model. *Clinical Psychology Review*, *31*(7), 1156–1168. https://doi.org/10.1016/j.cpr.2011.07.007

Updegraff, J. A., Silver, R. C., & Holman, E. A. (2008). Searching for and finding meaning in collective trauma: results from a national longitudinal study of the 9/11 terrorist attacks. *Journal of Personality and Social Psychology*, *95*(3), 709.

van Bronswijk, S. C., DeRubeis, R. J., Lemmens, L. H. J. M., Peeters, F. P. M. L., Keefe, J. R., Cohen, Z. D., & Huibers, M. J. H. (2021). Precision medicine for long-term depression outcomes using the Personalized Advantage Index approach: cognitive therapy or interpersonal psychotherapy? *Psychological Medicine*, *51*(2), 279–289. https://doi.org/10.1017/S0033291719003192

van Minnen, A., & Foa, E. B. (2006). The effect of imaginal exposure length on outcome of

treatment for PTSD. *Journal of Traumatic Stress*, *19*(4), 427–438. https://doi.org/10.1002/jts.20146

van Minnen, A., & Hagenaars, M. (2002). Fear activation and habituation patterns as early process predictors of response to prolonged exposure treatment in PTSD. *Journal of Traumatic Stress*, *15*(5), 359–367. https://doi.org/10.1023/A:1020177023209

Wang, L. (Peggy), & Maxwell, S. E. (2015). On disaggregating between-person and within-person effects with longitudinal data using multilevel models. *Psychological Methods*, *20*(1), 63–83. https://doi.org/10.1037/met0000030

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063. https://doi.org/https://doi.org/10.1037/0022-3514.54.6.1063

Watts, B. V., Schnurr, P. P., Mayo, L., Young-Xu, Y., Weeks, W. B., & Friedman, M. J. (2013). Meta-analysis of the efficacy of treatments for posttraumatic stress disorder. *Journal of Clinical Psychiatry*, *74*(6), 541–550. https://doi.org/10.4088/JCP.12r08225

Wichers, M., Lothmann, C., Simons, C. J. P., Nicolson, N. A., & Peeters, F. (2012). The dynamic interplay between negative and positive emotions in daily life predicts response to treatment in depression: A momentary assessment study. *British Journal of Clinical Psychology*, *51*(2), 206–222. https://doi.org/10.1111/j.2044-8260.2011.02021.x

Wichers, M., Wigman, J. T. W., & Myin-Germeys, I. (2015). Micro-level affect dynamics in psychopathology viewed from complex dynamical system theory. *Emotion Review*, *7*(4), 363–367. https://doi.org/10.1177/1754073915590623

Williams, R. M., Davis, M. C., & Millsap, R. E. (2002). Development of the cognitive processing of trauma scale. *Clinical Psychology & Psychotherapy*, *9*(5), 349–360.

Williams, T., Phillips, N. J., Stein, D. J., & Ipser, J. C. (2022). Pharmacotherapy for post traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews*, *2022*(3). https://doi.org/10.1002/14651858.CD002795.pub3

Winer, E. S., Bryant, J., Bartoszek, G., Rojas, E., Nadorff, M. R., & Kilgore, J. (2017). Mapping the relationship between anxiety, anhedonia, and depression. *Journal of Affective Disorders*, *221*, 289–296. https://doi.org/10.1016/j.jad.2017.06.006

Wolpe, J., & Lazarus, A. A. (1966). *Behavior therapy techniques: A guide to the treatment of neuroses.*

Yang, M., & Maxwell, S. E. (2014). Treatment effects in randomized longitudinal trials with different types of nonignorable dropout. *Psychological Methods*, *19*(2), 188–210. https://doi.org/10.1037/a0033804

Zalta, A. K., Gillihan, S. J., Fisher, A. J., Mintz, J., McLean, C. P., Yehuda, R., & Foa, E. B. (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology*, *82*(1), 171–175. https://doi.org/10.1037/a0034735

Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., Morey, L. C., Grilo, C. M., Shea, M. T., McGlashan, T. H., & Gunderson, J. G. (2000). The collaborative longitudinal personality disorders study: Reliability of axis I and II diagnoses. *Journal of Personality Disorders*, *14*(4), 291–299. https://doi.org/10.1521/pedi.2000.14.4.291

Zbozinek, T. D., & Craske, M. G. (2017). The role of positive affect in enhancing extinction learning and exposure therapy for anxiety disorders. *Journal of Experimental Psychopathology*, *8*(1), 13–39. https://doi.org/10.5127/jep.052615

Zoellner, L. A., Lehinger, E. A., Rosencrans, P. L., Cornell-Maier, S. M., Foa, E. B., Telch, M.

J., Gonzalez-Lima, F., & Bedard-Gilligan, M. A. (2022). Brief imaginal exposure for PTSD: Trajectories of change in distress. *Cognitive and Behavioral Practice*. https://doi.org/10.1016/j.cbpra.2022.04.005

Zoellner, L. A., Pruitt, L. D., Farach, F. J., & Jun, J. J. (2014). Understanding heterogeneity in PTSD: Fear, dysphoria, and distress. *Depression and Anxiety*, *31*(2), 97–106. https://doi.org/10.1002/da.22133

Zoellner, L. A., Roy-Byrne, P. P., Mavissakalian, M., & Feeny, N. C. (2019). Doubly randomized preference trial of prolonged exposure versus sertraline for treatment of PTSD. *American Journal of Psychiatry*, *176*(4), 287–296. https://doi.org/10.1176/appi.ajp.2018.17090995

Zoellner, L. A., Telch, M., Foa, E. B., Farach, F. J., McLean, C. P., Gallop, R., Bluett, E. J., Cobb, A., & Gonzalez-Lima, F. (2017). Enhancing Extinction Learning in Posttraumatic Stress Disorder With Brief Daily Imaginal Exposure and Methylene Blue. *The Journal of Clinical Psychiatry*, *78*(7), e782–e789. https://doi.org/10.4088/JCP.16m10936

Zoellner, L., Graham, B., Marks, E., Feeny, N., Bentley, J., Franklin, A., & Lang, D. (2018). Islamic Trauma Healing: Initial Feasibility and Pilot Data. *Societies*, *8*(3), 47. https://doi.org/10.3390/soc8030047

Table 1

Baseline Sample Characteristics for Study 1 (Changes in Self-Reported Positive and Negative Affect; N = 130)

Participant Characteristics	PE (*n* = 67)		PE plus sertraline (*n* = 63)	
	%	*n*	%	*n*
Gender				
Female (cisgender/transgender)	79.1%	53	77.8%	49
Race				
White	59.7%	40	57.1%	36
Black or African American	29.9%	20	27.0%	17
Other Background	10.5%	7	15.9%	10
Education				
Not College Educated	68.7%	46	63.5%	40
Income				
Less than $20,000/yr	53.7%	36	52.4%	33
Trauma Type				
Adult Sexual Assault	31.3%	21	27.0%	17
Childhood Assault (e.g., CSA)	11.9%	8	23.8%	15
Adult Assault (non-sexual)	28.4%	19	20.6%	13
Accident (MVA, natural disaster)	6.0%	4	4.8%	3
Combat/war	3.0%	2	3.2%	2
Any current co-occurring diagnosis (SCID-IV)	77.6%	52	85.7%	54

	M	SD	M	SD
Age (years)	34.5	*11.7*	36.7	*12.2*
Time Since Trauma Exposure (years)	10.4	*13.6*	15.4	*12.9*
PTSD Symptoms (PSS-I-5)	34.5	*6.2*	37.0	*6.2*
Depression Symptoms (QIDS-C)	14.1	*4.5*	15.6	*4.2*
Positive Affect (PANAS)	27.4	*9.6*	26.2	*7.3*
Negative Affect (PANAS)	30.9	*8.4*	32.9	*7.8*

Note. CSA = Childhood Sexual Abuse, MVA = Motor Vehicle Accident, SCID-IV = Structured Clinical Interview for DSM-IV, PSS-I-5 = PTSD Symptom Scale – Interview for DSM-5, QIDS-C = Quick Inventory of Depressive Symptomatology – Clinical Rating, PANAS = Positive and Negative Affect Schedule.

Table 2

Baseline Sample Characteristics for Study 2 (Changes in Observer-Rated Psychotherapy Process Positive and Negative Systems; N = 111)

Participant Characteristics	PE (*n* = 58)		PE plus sertraline (*n* = 53)	
	%	*n*	%	*n*
Gender				
Female (cisgender/transgender)	82.8%	48	75.5%	40
Race				
White	56.9%	33	62.3%	33
Black or African American	34.5%	20	20.8%	11
Other Background	8.6%	5	16.9%	9
Education				
Not College Educated	69.0%	40	60.4%	32
Income				
Less than $20,000/yr	46.6%	27	41.5%	22
Trauma Type				
Adult Sexual Assault	32.8%	10	28.3%	15
Childhood Assault (e.g., CSA)	17.3%	10	30.2%	16
Adult Assault (non-sexual)	31.0%	18	22.6%	12
Accident (MVA, natural disaster)	6.9%	4	5.7%	3
Combat/war	1.7%	1	3.8%	2
Any current co-occurring diagnosis (SCID-IV)	77.6%	45	84.9%	45

	M	SD	M	SD
Age (years)	33.4	*11.4*	37.7	*12.6*
Time Since Trauma Exposure (years)	8.2	*10.2*	15.4	*13.6*
PTSD Symptoms (PSS-I-5)	34.6	*6.2*	36.7	*6.0*
Depression Symptoms (QIDS-C)	14.6	*4.4*	15.5	*4.5*

Note. CSA = Childhood Sexual Abuse, MVA = Motor Vehicle Accident, SCID-IV = Structured Clinical Interview for DSM-IV, PSS-I-5 = PTSD Symptom Scale – Interview for DSM-5, QIDS-C = Quick Inventory of Depressive Symptomatology – Clinical Rating.

Table 3

Study 1: Means and Standard Deviations of Self-Reported Affect and PTSD Severity at each Session

Variable	Session									
	1	2	3	4	5	6	7	8	9	10
Positive affect (PANAS)	24.65 *(7.51)*	25.07 *(8.85)*	26.55 *(8.61)*	27.24 *(9.43)*	27.62 *(8.54)*	27.54 *(9.08)*	28.66 *(9.39)*	29.95 *(9.60)*	29.74 *(9.10)*	31.78 *(9.02)*
Negative affect (PANAS)	31.33 *(7.99)*	27.19 *(8.64)*	27.54 *(8.85)*	26.82 *(9.66)*	25.15 *(9.21)*	23.85 *(9.64)*	22.13 *(8.98)*	21.89 *(8.94)*	19.94 *(7.42)*	19.81 *(8.16)*
PTSD severity (PSS-SR)	33.61 *(8.00)*	31.73 *(9.26)*	28.83 *(10.29)*	27.77 *(11.49)*	25.19 *(11.12)*	23.00 *(11.25)*	20.45 *(12.08)*	18.35 *(11.70)*	16.57 *(11.21)*	13.85 *(10.96)*

Note. Observed means are reported outside of parentheses; standard deviations are inside parentheses. PANAS = Positive and Negative Affect Schedule; PSS-SR = PTSD Symptom Scale, Self-Report.

Table 4

Study 1: Correlations among Self-Reported Affect, PTSD Severity, and Depression across Sessions

Variable	1	2	3
1. Positive affect (PANAS)	-	-	-
2. Negative affect (PANAS)	-0.14	-	-
3. PTSD severity (PSS-SR)	-0.22	0.64	-
4. Depression severity (QIDS-SR)	-0.29	0.52	0.71

Note. PANAS = Positive and Negative Affect Schedule; PSS-SR = PTSD Symptom Scale, Self-Report; QIDS-SR = Quick Inventory of Depressive Symptomatology – Self Report.

Correlation coefficients were calculated separately for each session 1-10, converted to z-scores using Fisher's *r* to z transformations and averaged across sessions. The mean z scores were then converted back to correlation coefficients so that values reflect the average correlations of scores over sessions 1-10.

Table 5

Study 2: Means and Standard Deviations of Positive and Negative Systems and PTSD Severity at each Session

Variable	Session					
	1	3	4	5	8	10
Positive system activation (CHANGE)	2.71 *(1.95)*	2.57 *(1.83)*	3.30 *(1.75)*	3.55 *(2.15)*	4.64 *(2.51)*	7.94 *(2.82)*
Negative system activation (CHANGE)	5.84 *(2.43)*	4.49 *(2.26)*	4.75 *(2.55)*	4.96 *(2.73)*	4.09 *(3.00)*	2.40 *(2.08)*
PTSD severity (PSS-SR)	33.65 *(8.13)*	28.70 *(10.43)*	27.54 *(11.62)*	25.28 *(11.11)*	18.63 *(11.84)*	14.05 *(11.04)*

Note. Observed means are reported outside of parentheses; standard deviations are inside parentheses. CHANGE = Change and Growth Experiences Scale; PSS-SR = PTSD Symptom Scale, Self-Report.

Table 6

Study 2: Correlations among Positive and Negative Systems, PTSD Severity, and Depression across Sessions

Variable	1	2	3
1. Positive system activation (CHANGE)	-	-	-
2. Negative system activation (CHANGE)	-0.08	-	-
3. PTSD severity (PSS-SR)	-0.16	0.24	-
4. Depression severity (QIDS-SR)	-0.12	0.22	0.72

Note. CHANGE = Change and Growth Experiences Scale; PSS-SR = PTSD Symptom Scale, Self-Report; QIDS-SR = Quick Inventory of Depressive Symptomatology – Self Report.

Correlation coefficients were calculated separately for each session, converted to z-scores using Fisher's *r* to z transformations and averaged across sessions. The mean z scores were then converted back to correlation coefficients so that values reflect the average correlations of scores over sessions 1, 3, 4, 5, 8, and 10.

Table 7

Study 1: Cross-Lagged Dynamic Structural Equation Models for Self-Reported Affect and PTSD Severity

Model 1: Positive and Negative Affect			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Within-patient associations			
$PA_t \rightarrow NA_{t+1}$	-0.10* *(0.05)*	-0.19, -0.00	-0.09
$NA_t \rightarrow PA_{t+1}$	-0.17* *(0.04)*	-0.25, -0.10	-0.20
$PA_t \rightarrow PA_{t+1}$	0.38* *(0.06)*	0.26, 0.50	0.36
$NA_t \rightarrow NA_{t+1}$	0.59* *(0.05)*	0.50, 0.68	0.59
Between-patient associations			
PA ↔ NA	6.90 *(8.57)*	-11.76, 22.08	0.16
Random effects	Coefficient (*SD*)	95% CI	ES
Level 1			
PA residual	30.39* *(1.77)*	27.06, 33.97	--
NA residual	32.42* *(2.00)*	28.92, 36.56	--
Level 2			
PA intercept	45.39* *(10.06)*	29.76, 69.09	--
NA intercept	42.22* *(14.71)*	19.26, 23.90	--
$PA_t \rightarrow NA_{t+1}$ slope	0.10* *(0.04)*	0.04, 0.20	--
$NA_t \rightarrow PA_{t+1}$ slope	0.05* *(0.02)*	0.02, 0.10	--
$PA_t \rightarrow PA_{t+1}$ slope	0.12* *(0.04)*	0.07, 0.20	--
$NA_t \rightarrow NA_{t+1}$ slope	0.06* *(0.02)*	0.02, 0.11	--

Model 2: Positive Affect and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Within-patient associations			
$PA_t \rightarrow PTSD_{t+1}$	-0.05 *(0.05)*	-0.14, 0.06	-0.06
$PTSD_t \rightarrow PA_{t+1}$	-0.17* *(0.04)*	-0.24, -0.10	-0.26
$PA_t \rightarrow PA_{t+1}$	0.27* *(0.06)*	0.21, 0.39	0.27
$PTSD_t \rightarrow PTSD_{t+1}$	0.78* *(0.04)*	0.69, 0.86	0.75
Between-patient associations			
PA ↔ PTSD	11.74 *(19.26)*	-32.43, 43.95	0.15
Random effects	Coefficient (*SD*)	95% CI	ES
Level 1			
PA residual	29.61* *(1.60)*	26.68, 33.05	--
PTSD residual	37.63* *(2.06)*	33.88, 41.99	--
Level 2			
PA intercept	64.57* *(17.70)*	39.02, 106.83	--
PTSD intercept	100.64* *(38.94)*	43.98, 199.68	--
$PA_t \rightarrow PTSD_{t+1}$ slope	0.04* *(0.02)*	0.01, 0.10	--
$PTSD_t \rightarrow PA_{t+1}$ slope	0.04* *(0.02)*	0.02, 0.09	--
$PA_t \rightarrow PA_{t+1}$ slope	0.11* *(0.04)*	0.05, 0.19	--
$PTSD_t \rightarrow PTSD_{t+1}$ slope	0.03* *(0.01)*	0.01, 0.06	--
Model 3: Negative Affect and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Within-patient associations			

$NA_t \rightarrow PTSD_{t+1}$	0.16* *(0.06)*	0.03, 0.26	0.12
$PTSD_t \rightarrow NA_{t+1}$	0.35* *(0.05)*	0.25, 0.44	0.50
$NA_t \rightarrow NA_{t+1}$	0.24* *(0.06)*	0.13, 0.37	0.24
$PTSD_t \rightarrow PTSD_{t+1}$	0.75* *(0.05)*	0.65, 0.87	0.74
Between-patient associations			
NA ↔ PTSD	37.69* *(20.72)*	3.58, 85.63	0.53
Random effects	Coefficient (*SD*)	95% CI	ES
Level 1			
NA residual	31.07* *(1.71)*	27.95, 34.65	--
PTSD residual	37.98* *(2.08)*	34.18, 42.30	--
Level 2			
NA intercept	41.26* *(17.56)*	12.39, 80.42	--
PTSD intercept	85.33* *(35.23)*	30.93, 166.61	--
$NA_t \rightarrow PTSD_{t+1}$ slope	0.05* *(0.03)*	0.01, 0.13	--
$PTSD_t \rightarrow NA_{t+1}$ slope	0.04* *(0.02)*	0.02, 0.10	--
$NA_t \rightarrow NA_{t+1}$ slope	0.09* *(0.04)*	0.04, 0.18	--
$PTSD_t \rightarrow PTSD_{t+1}$ slope	0.03* *(0.02)*	0.01, 0.07	--

Note. *$p < .05$; PTSD = posttraumatic stress disorder PA = positive affect; NA = negative affect; CI = credible interval; ES = effect size; $_t$ = time.

[a] Effect sizes represent the average of the standardized associations across clusters for each parameter.

Table 8

Study 1: Between-Patient Moderators of the Associations between Self-Reported Affect and PTSD Severity

Model 1a: Positive and Negative Affect			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Treatment condition*PA_t → NA_{t+1}	0.09 *(0.09)*	-0.08, 0.28	0.10
Treatment condition*NA_t → PA_{t+1}	0.05 *(0.08)*	-0.10, 0.20	0.07
Model 2a: Positive Affect and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Treatment condition*PA_t → $PTSD_{t+1}$	-0.02 *(0.07)*	-0.15, 0.12	-0.04
Treatment condition*$PTSD_t$ → PA_{t+1}	-0.02 *(0.07)*	-0.14, 0.10	-0.03
Model 3a: Negative Affect and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Treatment condition*NA_t → $PTSD_{t+1}$	-0.08 *(0.06)*	-0.21, 0.03	-0.14
Treatment condition*$PTSD_t$ → NA_{t+1}	-0.03 *(0.05)*	-0.14, 0.07	-0.06

Note. CI = credible interval; ES = effect size; PA = positive affect; NA = negative affect; PTSD = posttraumatic stress disorder; $_t$ = time. Treatment condition coded: 0 = prolonged exposure; 1 = prolonged exposure plus sertraline

[a] Effect sizes represent the average of the standardized associations across clusters for each parameter.

Table 9

Study 2: Cross-Lagged Dynamic Structural Equation Models for Positive and Negative Affect Systems and PTSD Severity

Model 4: Positive and Negative Systems (CHANGE)			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Within-patient associations			
$PSA_t \rightarrow NSA_{t+1}$	-0.23* *(0.07)*	-0.36, -0.08	-0.25
$NSA_t \rightarrow PSA_{t+1}$	-0.10* *(0.05)*	-0.19, -0.00	-0.09
$PSA_t \rightarrow PSA_{t+1}$	0.60* *(0.05)*	0.50, 0.70	0.59
$NSA_t \rightarrow NSA_{t+1}$	0.07 *(0.06)*	-0.03, 0.20	0.07
Between-patient associations			
PSA ↔ NSA	0.30 *(0.35)*	-0.55, 0.88	0.45
Random effects	Coefficient (*SD*)	95% CI	ES
Level 1			
PSA residual	5.50* *(0.36)*	4.86, 6.29	--
NSA residual	5.51* *(0.42)*	4.77, 6.40	--
Level 2			
PSA intercept	0.76* *(0.56)*	0.15, 2.36	--
NSA intercept	0.93* *(0.42)*	0.26, 1.87	--
$PSA_t \rightarrow NSA_{t+1}$ slope	0.09* *(0.05)*	0.02, 0.20	--
$NSA_t \rightarrow PSA_{t+1}$ slope	0.05* *(0.02)*	0.02, 0.22	--
$PSA_t \rightarrow PSA_{t+1}$ slope	0.01* *(0.01)*	0.00, 0.03	--
$NSA_t \rightarrow NSA_{t+1}$ slope	0.02* *(0.01)*	0.00, 0.05	--

Model 5: Positive System and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Within-patient associations			
$PSA_t \rightarrow PTSD_{t+1}$	-0.04 *(0.02)*	-0.08, 0.000	-0.09
$PTSD_t \rightarrow PSA_{t+1}$	-1.07* *(0.15)*	-1.38, -0.84	-0.44
$PSA_t \rightarrow PSA_{t+1}$	0.36* *(0.06)*	0.23, 0.45	0.36
$PTSD_t \rightarrow PTSD_{t+1}$	0.75* *(0.04)*	0.67, 0.83	0.75
Between-patient associations			
PSA ↔ PTSD	-0.13 *(0.37)*	-1.13, 0.26	0.25
Random effects	Coefficient (*SD*)	95% CI	ES
Level 1			
PSA residual	4.70* *(0.34)*	4.09, 5.43	--
PTSD residual	0.58* *(0.04)*	0.51, 0.68	--
Level 2			
PSA intercept	1.00* *(0.80)*	0.07, 2.93	--
PTSD intercept	0.46* *(0.28)*	0.02, 1.13	--
$PSA_t \rightarrow PTSD_{t+1}$ slope	0.001* *(0.001)*	0.001, 0.003	--
$PTSD_t \rightarrow PSA_{t+1}$ slope	0.04* *(0.04)*	0.003, 0.14	--
$PSA_t \rightarrow PSA_{t+1}$ slope	0.01* *(0.01)*	0.001, 0.02	--
$PTSD_t \rightarrow PTSD_{t+1}$ slope	0.003* *(0.002)*	0.001, 0.01	--
Model 6: Negative System and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Within-patient associations			

$NSA_t \rightarrow PTSD_{t+1}$	0.003 *(0.02)*	-0.03, 0.04	-0.01
$PTSD_t \rightarrow NSA_{t+1}$	0.73* *(0.13)*	0.50, 1.00	0.38
$NSA_t \rightarrow NSA_{t+1}$	0.05 *(0.06)*	-0.08, 0.16	0.05
$PTSD_t \rightarrow PTSD_{t+1}$	0.81* *(0.04)*	0.74, 0.88	0.82
Between-patient associations			
NSA ↔ PTSD	0.04 *(0.24)*	-0.31, 0.62	0.29
Random effects	Coefficient (*SD*)	95% CI	ES
Level 1			
NSA residual	5.51* (*0.38*)	2.65, 3.78	--
PTSD residual	0.56* *(0.04)*	0.48, 0.65	--
Level 2			
NSA intercept	0.49* *(0.40)*	0.09, 1.58	--
PTSD intercept	0.30* *(0.26)*	0.02, 1.00	--
$NSA_t \rightarrow PTSD_{t+1}$ slope	0.004* *(0.002)*	0.001, 0.01	--
$PTSD_t \rightarrow NSA_{t+1}$ slope	0.13* *(0.08)*	0.02, 0.32	--
$NSA_t \rightarrow NSA_{t+1}$ slope	0.02* *(0.02)*	0.003, 0.07	--
$PTSD_t \rightarrow PTSD_{t+1}$ slope	0.002* *(0.002)*	0.001, 0.01	--

Note. *$p < .05$; CHANGE = Change and Growth Experiences Scale; PTSD = posttraumatic stress disorder PSA = Change and Growth Experiences Scale - positive system activation; NSA = Change and Growth Experiences Scale - negative system activation; CI = credible interval; ES = effect size; $_t$ = time.

[a] Effect sizes represent the average of the standardized associations across clusters for each parameter.

Table 10

Study 2: Between-Patient Moderators of the Associations between Positive and Negative Systems and PTSD Severity

Model 4a: Positive and Negative Systems			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Treatment condition*PSA_t → NSA_{t+1}	-0.32* *(0.13)*	-0.56, -0.08	-0.42
Treatment condition*NSA_t → PSA_{t+1}	-0.16 *(0.08)*	-0.31, 0.01	-0.44
Model 5a: Positive System and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Treatment condition*PSA_t → $PTSD_{t+1}$	-0.01 *(0.03)*	-0.08, 0.04	-0.12
Treatment condition*$PTSD_t$ → PSA_{t+1}	0.34 *(0.27)*	-0.24, 0.78	0.52
Model 6a: Negative System and PTSD symptoms			
Fixed effects	Coefficient (*SD*)	95% CI	ES[a]
Treatment condition*NSA_t → $PTSD_{t+1}$	-0.06 *(0.03)*	-0.12, 0.001	-0.40
Treatment condition*$PTSD_t$ → NSA_{t+1}	0.29 *(0.24)*	-0.14, 0.74	0.30

Note. *$p < .05$; PTSD = posttraumatic stress disorder PSA = Change and Growth Experiences Scale (CHANGE) - positive system activation; NSA = Change and Growth Experiences Scale (CHANGE) - negative system activation; CI = credible interval; ES = effect size; $_t$ = time. Treatment condition coded: 0 = prolonged exposure; 1 = prolonged exposure plus sertraline

[a] Effect sizes represent the average of the standardized associations across clusters for each parameter.

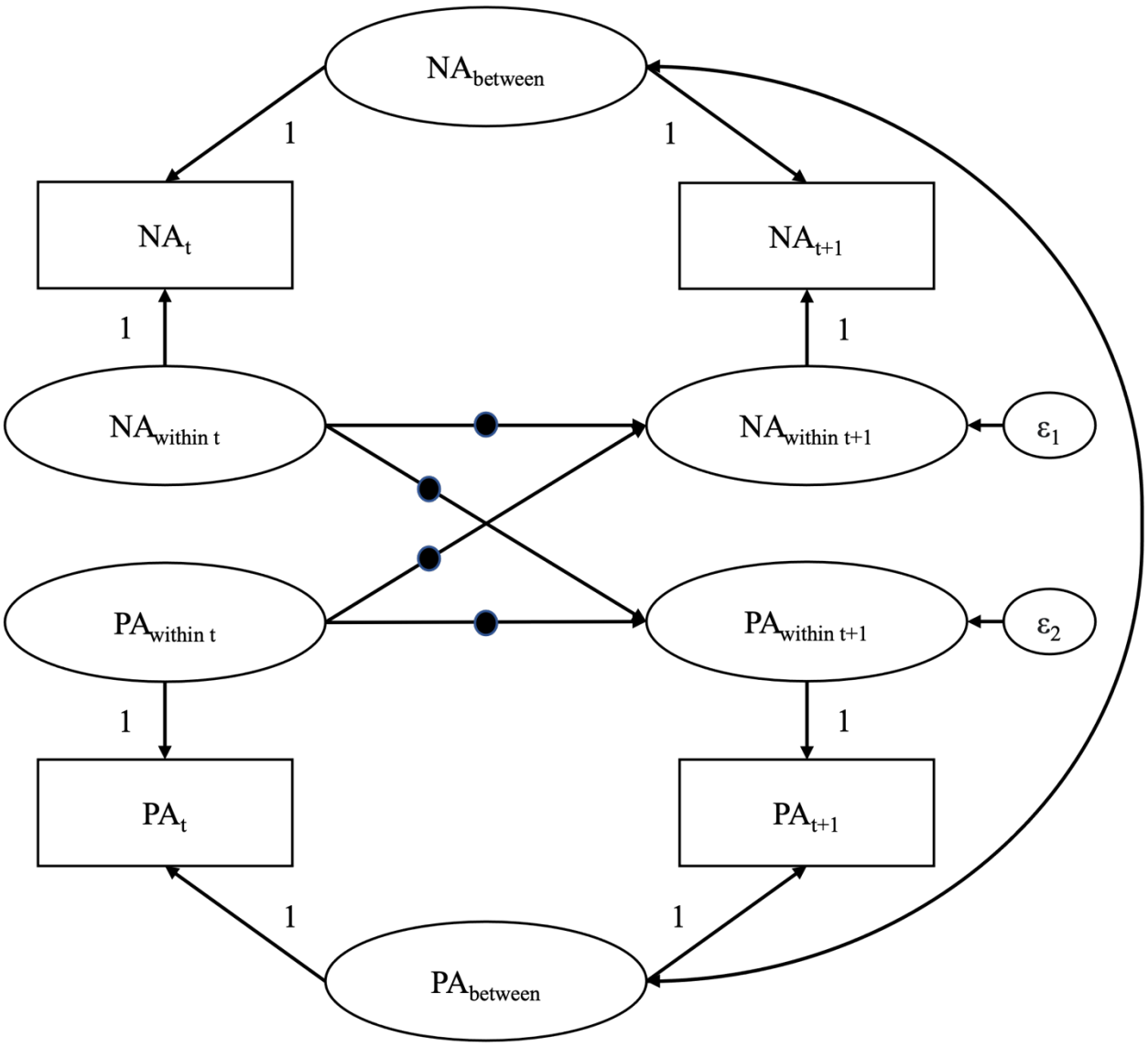

Figure 1. A visual representation of Model 1, the cross-lagged dynamic structural equation model examining the session-to-session association between within-person PA and NA change. *Note.* Circles depict disaggregated, latent within- and between-person variables, whereas rectangles depict observed or measured variables at consecutive time points (i.e., t and t +1). Paths with filled dots were allowed to vary between people (i.e., random slopes).
PA = Positive and Negative Affect Schedule – Positive Affect; NA = Positive and Negative Affect Schedule – Negative Affect.

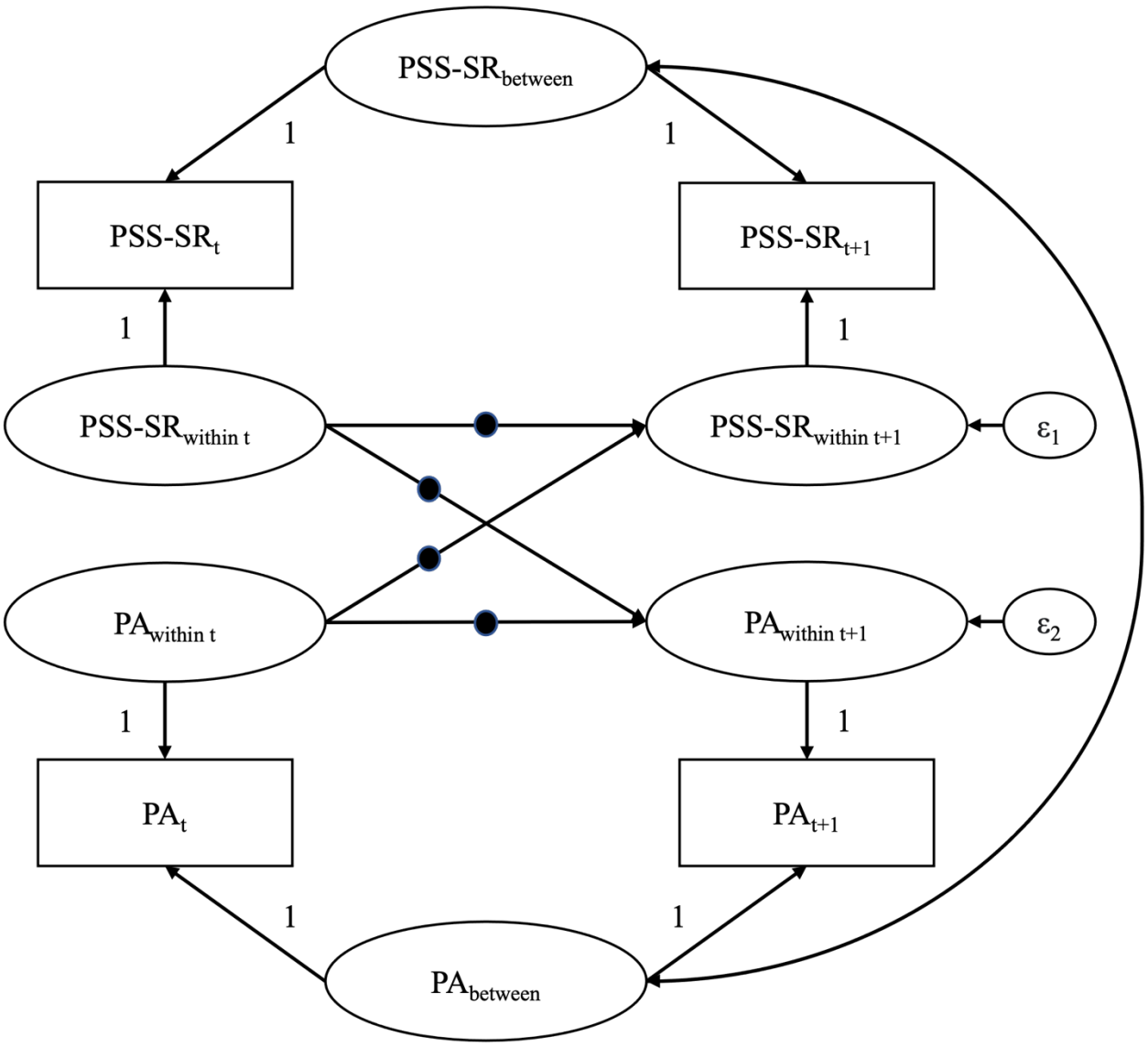

Figure 2. A visual representation of Model 2, the cross-lagged dynamic structural equation model examining the session-to-session association between within-person PA and PTSD symptom change.
Note. Circles depict disaggregated, latent within- and between-person variables, whereas rectangles depict observed or measured variables at consecutive time points (i.e., t and t +1). Paths with filled dots were allowed to vary between people (i.e., random slopes).
PA = Positive and Negative Affect Schedule – Positive Affect; PSS-SR = PTSD Symptom Scale – Self-Report.

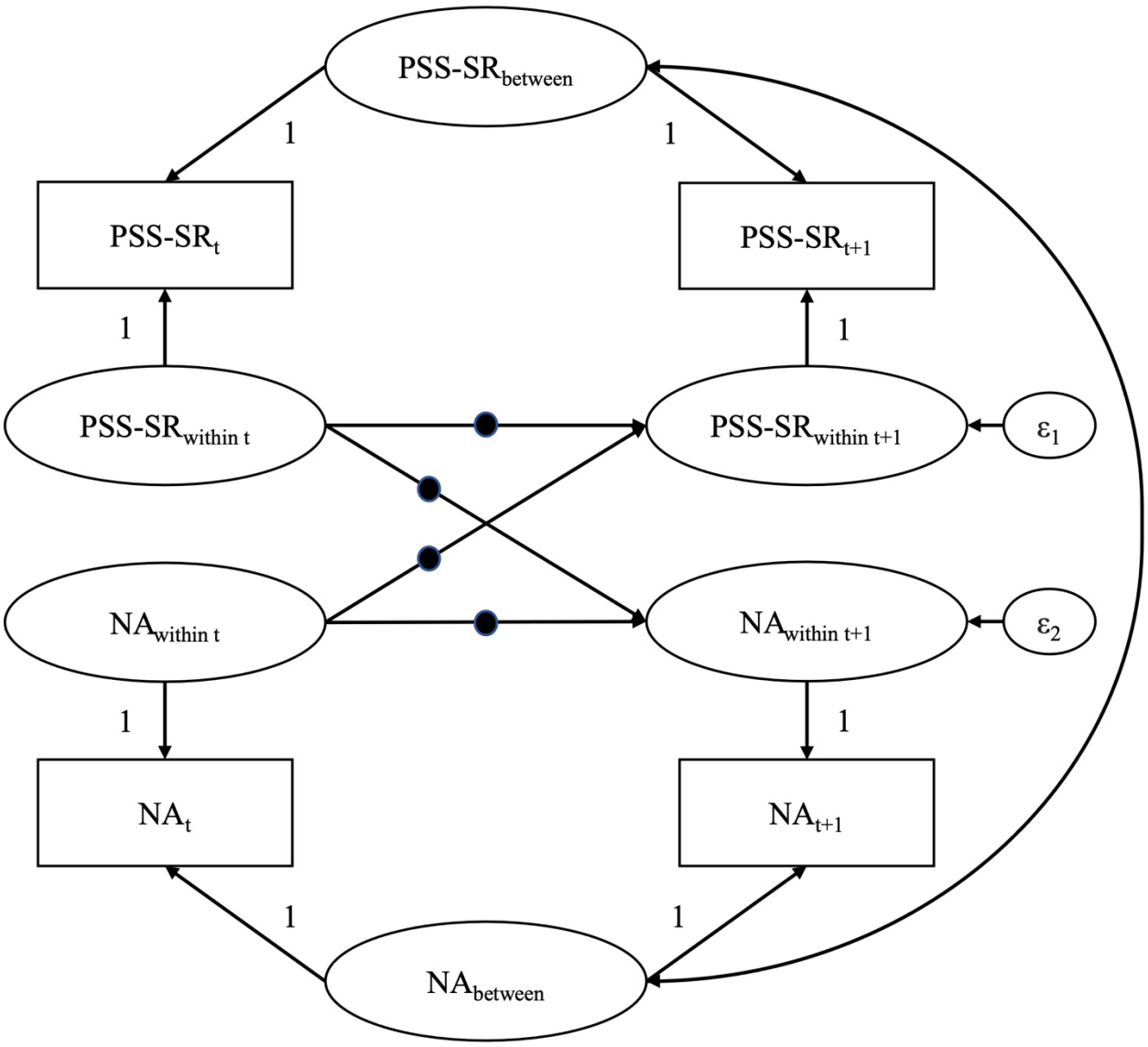

Figure 3. A visual representation of Model 3, the cross-lagged dynamic structural equation model examining the session-to-session association between within-person NA and PTSD symptom change.
Note. Circles depict disaggregated, latent within- and between-person variables, whereas rectangles depict observed or measured variables at consecutive time points (i.e., t and t +1). Paths with filled dots were allowed to vary between people (i.e., random slopes).
NA = Positive and Negative Affect Schedule – Negative Affect; PSS-SR = PTSD Symptom Scale – Self-Report.

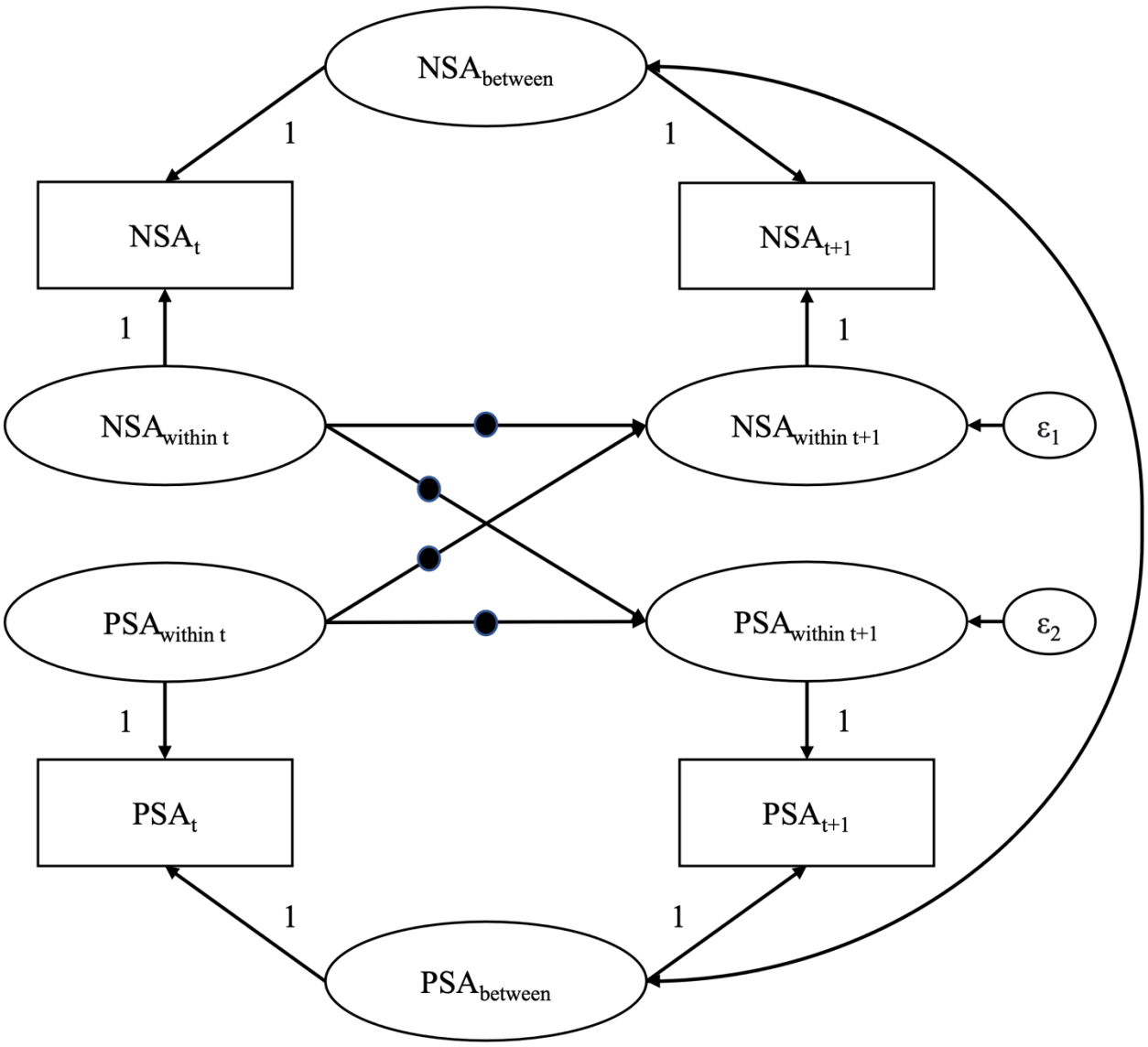

Figure 4. A visual representation of Model 4, the cross-lagged dynamic structural equation model examining the session-to-session association between within-person positive and negative system activation change.
Note. Circles depict disaggregated, latent within- and between-person variables, whereas rectangles depict observed or measured variables at consecutive time points (i.e., t and t +1). Paths with filled dots were allowed to vary between people (i.e., random slopes).
PSA = Change and Growth Experiences Scale - positive system activation; NSA = Change and Growth Experiences Scale - negative system activation.

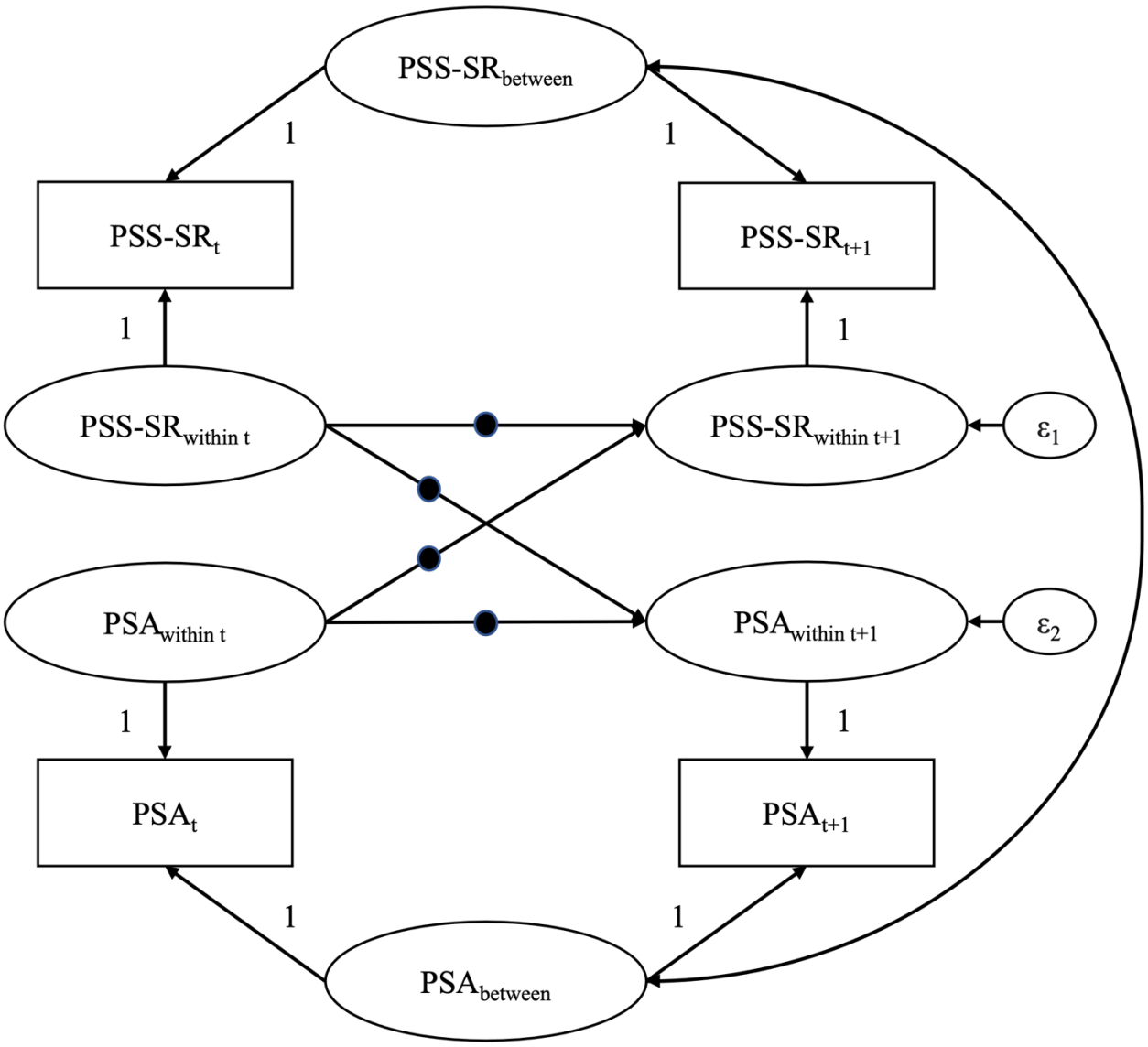

Figure 5. A visual representation of Model 5, the cross-lagged dynamic structural equation model examining the session-to-session associations between within-person positive system activation and PTSD symptom change.
Note. Circles depict disaggregated, latent within- and between-person variables, whereas rectangles depict observed or measured variables at consecutive time points (i.e., t and t +1). Paths with filled dots were allowed to vary between people (i.e., random slopes).
PSA = Change and Growth Experiences Scale - positive system activation; PSS-SR = PTSD Symptom Scale – Self-Report.

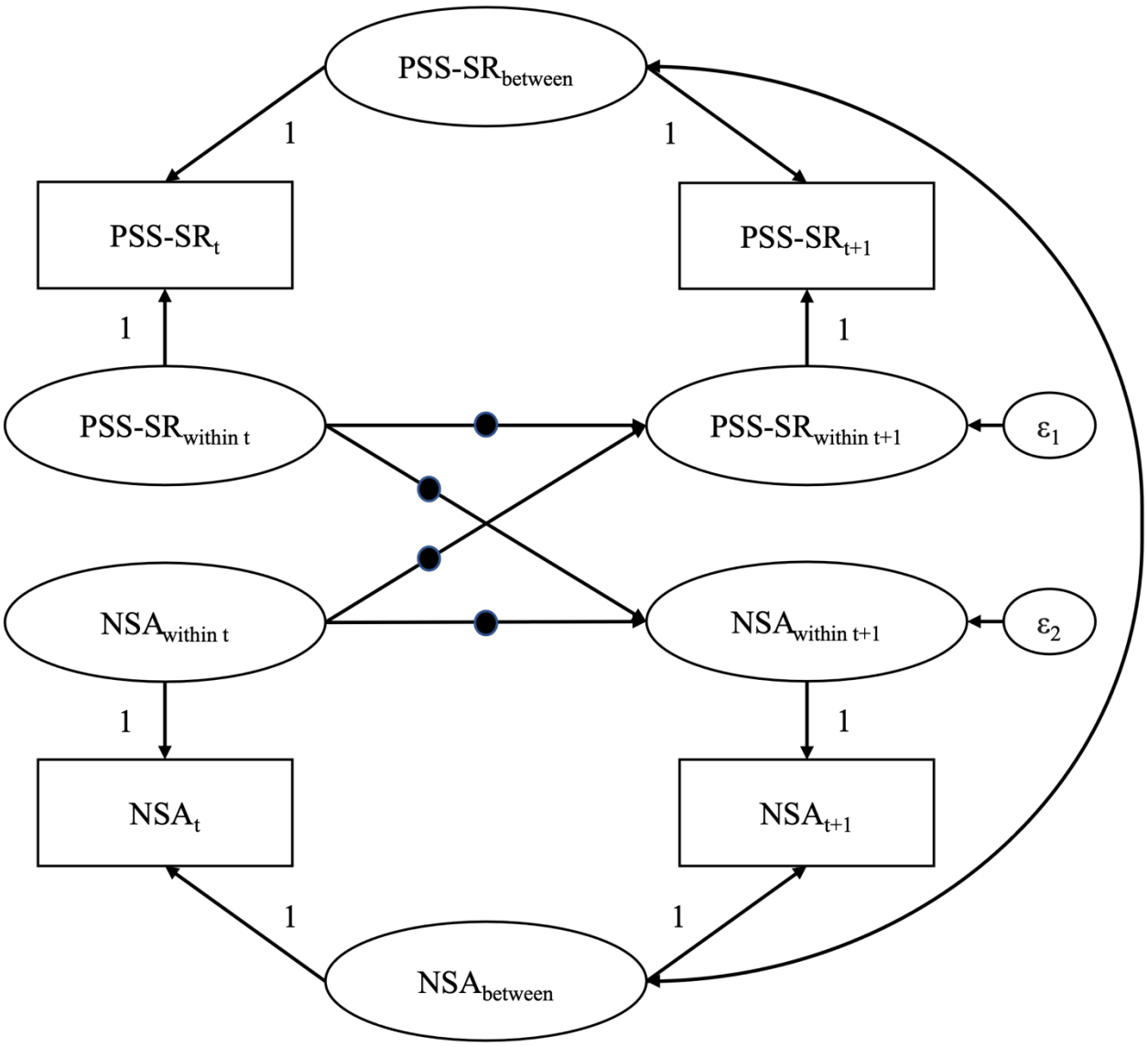

Figure 6. A visual representation of Model 6, the cross-lagged dynamic structural equation model examining the session-to-session associations between within-person negative system activation and PTSD symptom change.
Note. Circles depict disaggregated, latent within- and between-person variables, whereas rectangles depict observed or measured variables at consecutive time points (i.e., t and t +1). Paths with filled dots were allowed to vary between people (i.e., random slopes).
NSA = Change and Growth Experiences Scale - negative system activation; PSS-SR = PTSD Symptom Scale – Self-Report.