Behaviour Research and Therapy 176 (2024) 104519

Contents lists available at ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Heart rate reactivity during trauma recall as a predictor of treatment outcome in cognitive processing therapy for PTSD

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ARTICLE INFO

Keywords:
Posttraumatic stress disorder
Cognitive processing therapy
Heart rate
Physiological arousal
Treatment outcomes
Treatment dropouts

ABSTRACT

Emotional engagement when recollecting a trauma memory is considered a key element of effective trauma-focused therapy. Research has shown that reduced physiological reactivity during trauma recall is associated with worse treatment outcomes for posttraumatic stress disorder (PTSD), but this has yet to be examined in a cognitively oriented treatment. This study examined whether pretreatment heart rate (HR) reactivity during trauma recall predicts PTSD symptom improvement and treatment dropout during Cognitive Processing Therapy (CPT) for PTSD. Participants were 142 women with PTSD secondary to interpersonal violence enrolled in one of two clinicals trials. HR reactivity reflected the mean increase in HR after listening to two 30-s scripts of the trauma memory prior to treatment. Linear mixed-effects models showed the effect of HR reactivity on change in total PTSD symptoms was not significant, but lower HR reactivity predicted less improvement in reexperiencing and avoidance and was associated with increased dropout. Findings suggest pretreatment physiological reactivity to the trauma memory may be a prognostic indicator of some elements of treatment response in CPT. Results tentatively support the importance of emotional activation during trauma recall in cognitive treatment of PTSD, though more research is needed to clarify how low HR reactivity impacts treatment.

1. Introduction

Trauma-focused psychotherapies such as prolonged exposure (PE) and cognitive processing therapy (CPT) have generated significant empirical support and are considered front-line treatments for PTSD (American Psychological Association, 2017; U.S. Department of Veterans Affairs Department of Defense, 2017). However, as many as 30–50% of patients receiving such treatments do not see clinically significant improvement (Steenkamp et al., 2015), highlighting the need to better understand the factors associated with treatment nonresponse. One promising predictor of treatment response may be the degree to which one is emotionally reactive to their worst trauma memory. Across trauma-focused interventions, activating the affective components of the trauma memory during recall (i.e., emotional engagement) is considered critical for ultimately reducing distress associated with the memory and facilitating more adaptive appraisals of the traumatic event (Foa et al., 2006; Resick Wachen, et al., 2017b; Schnyder et al., 2015). Blunted emotional reactivity during trauma recall, a likely indicator of poor emotional engagement, may therefore be an impediment to effective trauma processing and consequently result in reduced symptom change.

One indicator of PTSD-specific emotional reactivity that may be particularly relevant to treatment response is physiological arousal during voluntary trauma recall. Although physiology does not equate to emotional responding, assessing physiological reactivity provides an objective indicator of one component of the emotional response to a trauma memory and is a useful tool for characterizing the unique effects of recalling a traumatic memory among individuals with PTSD (Pineles et al., 2013; O'Kearney & Parry, 2014). An extensive body of literature indicates that individuals with PTSD are more likely to have elevated physiological reactivity during trauma recall than those without (Pole, 2007; O'Kearney & Parry, 2014), yet significant variability within PTSD

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https://doi.org/10.1016/j.brat.2024.104519
Received 16 November 2022; Received in revised form 5 March 2024; Accepted 8 March 2024
Available online 11 March 2024

samples exists. In fact, studies have found that 30–40% of individuals with clinician-diagnosed PTSD exhibit physiological responses during voluntary trauma recall that are indistinguishable from trauma-exposed individuals without PTSD (Orr & Roth, 2000; Pineles et al., 2013). For at least some individuals, such reduced reactivity may result from more chronic trauma exposure, particularly during childhood (D'Andrea et al., 2013; Iffland et al., 2020) and/or reflect processes related to dissociation or emotional blunting during trauma recall (Griffin et al., 1997; Lanius et al., 2002; Sack et al., 2012) which could negatively impact effective trauma processing during PTSD treatment.

To date, a number of studies examining exposure-based PTSD treatment have provided support for the hypothesis that lower physiological reactivity during voluntary trauma recall is associated with worse treatment outcome. In an early study by Pitman et al. (1996), smaller heart rate increases during an initial imaginal flooding session predicted less improvement in intrusive symptoms over the course of a six-session treatment. A similar result was found in a study on Written Exposure Therapy (WET), in which lower heart rate reactivity during the initial exposure session predicted less PTSD symptom improvement by the end of treatment (Wisco et al., 2016). Furthermore, a study by Tuerk et al. (2018) examining PE found that smaller increases in heart rate during the initial imaginal exposure session among patients receiving yohimbine, a drug used to stimulate sympathetic nervous system activity, predicted less improvement in PTSD symptoms at the next session and over the course of treatment. Finally, Wangelin and Tuerk (2015) showed that lower heart rate reactivity during trauma memory recall *prior* to treatment is also predictive of worse outcomes: using a widely employed script driven imagery (SDI) procedure in which individuals vividly imagine their trauma after listening to brief audio recordings narrating the events (Pitman et al., 1987), the researchers found that lower heart rate reactivity was associated with less reduction in PTSD symptoms during PE (Wangelin & Tuerk, 2015).

Taken together, the literature consistently shows that lower cardiac reactivity during voluntary trauma recall, whether measured prior to treatment or during an initial treatment session, predicts worse treatment outcome. However, several limitations to this research are worth noting. First, sample sizes of these studies have been small, with no study including more than 35 participants. Furthermore, these relationships have yet to be examined in a cognitively oriented treatment such as cognitive processing therapy (CPT). Although changes in symptom severity during CPT have been shown to be positively associated with changes in physiological reactivity to loud tones (Griffin et al., 2012), no studies have examined pretreatment physiological trauma memory reactivity as a predictor of CPT outcomes. Investigating such a relationship in a cognitively based treatment could inform whether reduced reactivity is a potential impediment to treatment response across trauma-focused interventions or is specific to treatments primarily consisting of exposure.

This focus on exposure therapies in the research reviewed thus far likely stems from the fact that such treatments are grounded in emotional processing theory (Foa et al., 2006; Foa & Kozak, 1986), which emphasizes the importance of fear activation and subsequent habituation during repeated exposures to the trauma memory. CPT, on the other hand, is based primarily on cognitive and social cognitive theory (Beck et al., 1979; McCann et al., 1988), thereby focusing on identifying and challenging maladaptive beliefs about the cause and meaning of the traumatic event, as well as the impact of the trauma on beliefs about oneself, others, and the world (Resick, Wachen, et al., 2017a). The emphasis on cognitive reappraisal strategies rather than repeated exposure to the trauma memory each session may make emotional engagement during trauma recall a less critical component of treatment. However, emotional activation during the application of cognitive techniques in CPT is still thought to be important for facilitating change in the meaning of the traumatic event and for the dissipation of natural affect associated with the trauma memory (Galovski et al., 2020; Resick et al., 2008). Moreover, although not framed as exposure, CPT involves intentional engagement with details of the trauma memory through recounting the events during cognitive work and can also involve a written account of the trauma memory. Thus, physiological trauma memory reactivity is also a plausible predictor of treatment response in CPT.

Another important direction for this line of research is to examine effects of physiological trauma memory reactivity on treatment completion. Dropout poses a substantial barrier to the efficacy of trauma-focused treatments, with average rates of attrition in studies of CPT and PE around 30% (Varker et al., 2021). Although contributors to dropout are heterogeneous, two frequently cited factors are emotional avoidance (Garcia et al., 2011; Zayfert et al., 2005) and lack of symptom improvement (Hoge et al., 2014; Holmes et al., 2019), both of which may be associated with reduced physiological reactivity during trauma memory recall. To date, only one study (Wangelin & Tuerk, 2015) has examined physiological trauma memory reactivity in relation to dropout, finding that individuals who demonstrated a *decrease* in heart rate during their trauma script were significantly more likely to drop out of therapy. Further investigations are clearly needed to better understand the impact of physiological reactivity during trauma recall on this important facet of PTSD efficacy.

Given the heterogeneous collection of symptoms that comprise PTSD (Galatzer-Levy & Bryant, 2013), examining the effect of heart rate reactivity on PTSD symptom clusters separately can also provide more specificity regarding the ways trauma memory reactivity may impact treatment outcomes. Reexperiencing and avoidance clusters have been conceptualized as reflecting heightened emotional responses to the trauma memory and associated stimuli, whereas hyperarousal and negative alterations in cognitions and mood (DSM-5 clusters D and E) capture a broader array of posttraumatic responses, many of which overlap with general dysphoria and anxious distress (Forbes et al., 2011, 2015; Hunt et al., 2018; Zoellner et al., 2014). Because emotional activation during trauma recall is thought to most directly impact adaptive processing of the traumatic memory itself (Foa & Kozak, 1986), physiological reactivity would be expected to have the strongest effect on symptom clusters that most clearly reflect emotional responses to the trauma memory, namely reexperiencing and avoidance. Recent evidence also suggests that changes in reexperiencing and avoidance symptoms during PTSD treatment may be particularly impacted by indices of baseline emotional engagement, reflected in greater insula activity during processing of negative emotional stimuli (Bryant et al., 2020). It remains an open question whether emotional engagement reflected by physiological reactivity during trauma recall would exhibit similarly distinct effects on PTSD symptom clusters.

The present study sought to address the gaps in the literature described above by examining whether pre-treatment cardiac reactivity during SDI (Pitman et al., 1987) predicts improvements in PTSD symptom severity and treatment dropout in a sample of 142 participants receiving CPT for PTSD. Because emotional engagement with the trauma memory is considered important across trauma-focused treatments (Schnyder et al., 2015), including during the application of cognitive techniques (Galovski et al., 2020; Resick et al., 2008), we hypothesized that lower cardiac reactivity would be associated with less symptom improvement during CPT and greater dropout from treatment. We also hypothesized that effects on PTSD symptom improvement would be most strongly evident on reexperiencing and avoidance symptom clusters. We expected weaker relationships, as indicated by the magnitude of effect sizes, with clusters more strongly overlapping with broad dysphoria and general distress, captured in the present study through the frequently used hyperarousal and numbing clusters (the latter of which reflects generalized emotional withdrawal, not numbing of trauma-specific reactions) derived from DSM-IV PTSD symptoms (King et al., 1998; Naifeh et al., 2008; Palmieri et al., 2007).

In addition, we conducted a second set of predictor analyses that included measures potentially associated with emotional blunting as covariates. The goal of this analysis was to assess whether cardiac

reactivity accounts for unique variance in treatment outcome beyond such factors, or whether shared variance between predictors could further inform possible explanations for reactivity-outcome relationships. Specifically, we examined: 1) dissociative symptoms, which have been associated with reduced physiological reactivity to trauma memory as well as treatment outcome in some studies on CPT (Resick et al., 2012); 2) defensive avoidance, which could lead to emotional disengagement during voluntary trauma recall (Kenny & Bryant, 2007), as well as reduced engagement in treatment; 3) a history of chronic childhood physical or sexual assault, which has been associated with blunted physiological responses (D'Andrea et al., 2013; Iffland et al., 2020); and 4) cardiac reactivity when imagining a stressful, non-trauma related scene, which has also been shown to be elevated in PTSD (McTeague et al., 2010), to examine whether any relationships with treatment outcomes are specific to trauma memory reactivity or can be attributed to broader patterns of emotional reactivity.

2. Method

2.1. Participants

Participants were 142 women with PTSD secondary to interpersonal violence who received CPT in one of two randomized controlled trials (Galovski et al., 2016; Resick et al., 2008) and completed a baseline psychophysiological assessment. Both trials were conducted at the same location and recruited from the same community population. Across both studies, participants had to be 18 years or older and meet DSM-IV-IR criteria for PTSD following interpersonal violence (i.e., sexual or physical assault) that had occurred at least 3-months previously, as measured by the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). An additional inclusion criterion in the Galovski et al. (2016) trial was clinically significant sleep impairment as indicated by a score of $\geq$3 on the CAPS sleep impairment item (D-1). Exclusion criteria across both studies consisted of psychosis, mania, developmental delay, significant imminent risk (e.g., active suicidality, significant self-harm, in abusive relationship, being stalked), and alcohol or other drug dependence. Psychiatric medications were allowed if participants had been on stable dose for the month prior to treatment. However, none of the participants reported use of medications that would alter cardiac activity (e.g., beta blockers, calcium channel blockers, etc.).

In the Resick et al. (2008) dismantling trial, which compared CPT with the written trauma account, CPT without the account, and a written account only condition, we examined data from those who received CPT with or without the account because the written account condition did not include any of the cognitive interventions constituting the core of CPT. Together, the Resick et al. (2008) and Galovski et al. (2016) trials included 192 participants who received CPT. However, 42 did not complete the psychophysiological assessment and 8 did not have valid heart rate due to equipment malfunction or excessive movement artifacts, leaving a final sample of $n = 142$. There were no significant differences in baseline PTSD severity, dissociation, defensive avoidance, index trauma type, or demographic variables between the sample analyzed in this study and those who received CPT and did not complete the psychophysiological assessment (all $ps > 0.05$).

The final sample had a mean age of 36.4 ($SD = 11.6$) and was 57% White, 41% Black, 1% American Indian/Alaska Native, and 1% other. The mean for years of education was 13.9 ($SD = 2.9$). The median annual income range was $10,001-$20,000. Frequency of index trauma type was as follows: 37.3% childhood sexual assault, 7.7% childhood physical assault, 28.9% adult sexual assault, 24.6% adult physical assault. In addition to their index trauma, the sample had an extensive history of interpersonal trauma: 63.4% of the sample had experienced at least three of the four trauma types described above, with 71.1% experiencing childhood sexual assault and 62.7% experiencing childhood physical assault. At pretreatment, 45.1% of the sample met criteria for major depressive disorder and 15.4% met criteria for panic disorder, as assessed by the Structured Clinical Interview for DSM-IV Disorders (First et al., 1996).

2.2. Measures and procedures

Participants completed an initial intake session that included diagnostic assessment of PTSD using the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995), self-report questionnaires, and creation of the trauma scripts used during the administration of the SDI task, which took place at a subsequent visit.

Script Driven Imagery (SDI). The SDI procedure (Pitman et al., 1987) is a commonly used and well-validated task to measure psychophysiological responses during mental imagery of a trauma memory. Numerous studies have shown that psychophysiological reactivity, including heart rate reactivity, during trauma imagery in this task is associated with PTSD severity (see Pole, 2007 for a meta-analysis) and PTSD diagnosis (Orr et al., 1998; Pineles et al., 2013; Pitman et al., 1987). Reactivity during stressful, non-trauma related imagery, however, does not appear associated with PTSD (Orr et al., 1998; Pineles et al., 2013).

Following standardized procedures used in prior research (Orr et al., 1998; Pitman et al., 1987), participants first created a script that detailed the events of their index trauma, which had been identified during administration of the CAPS. The assessor helped elicit further detail about the event as necessary to create a sufficiently vivid and coherent narrative. Participants were also provided a list of physiological descriptors associated with distress reactions (e.g., "palms sweaty", "heart racing" etc.) and were instructed to select any that applied to their response during the trauma. Next, the script was edited so that it used the second person and present tense (e.g., "You see the assailant") and could be divided into two 30-s segments of approximately 100 words, which together told the events of the trauma from beginning to end. Selected physiological descriptor words were also incorporated into the scripts. The segments were then read aloud by a female member of the research staff who was not the assessor for the creation of an audio recording.

During administration of the SDI task itself, which occurred at a separate visit from script creation and the CAPS assessment, participants listened to a total of six 30-s audio scripts while connected to heart rate monitoring equipment. This included the two personalized trauma scripts just described and four standard scripts: one describing a stressful but not traumatic, situation (i.e., feeling intense anxiety in a high-stakes public speaking situation), two neutral scripts and one positive script. The order sequence of the scripts was standardized as follows: neutral, non-trauma stressor, first trauma script, neutral, second trauma script, positive. Only the non-trauma stressor and trauma scripts were examined as predictors in this study. For each script, there were four consecutive 30-s periods: 1) baseline silence, 2) listening to the audio recording, 3) imagining the scene just described, and 4) recovery, plus an additional 30 s before the baseline period of the next script to provide time for HR to recover. Audio output was controlled via an audio receiver and decibel levels were aligned with normal voice levels (60 dB). Each participant received a hearing test prior to the SDI task to ensure all participants could hear the scripts.

Heart Rate Measurement and Scoring. Heart rate was measured via disposable Ag/AgCl leads attached to the left wrist and right ankle, which were connected to a Coulbourn bioamplifier (S75-01). Peak R-wave signals were detected and interbeat intervals were converted to heart rate. Data were continuously sampled at 500 Hz. Heart rate reactivity (HRR) was measured as the mean heart rate during the imagery period minus the mean heart rate during the baseline period immediately preceding the script. To reduce the variance associated with extreme scores, a square root transformation was performed on HRR values prior to analysis. Trauma Memory HRR was then calculated by taking the mean of the transformed HRR values from the two trauma scripts.

PTSD Symptoms. Severity of PTSD was assessed via the Posttraumatic Diagnostic Scale (PDS), a self-report measure containing 17 items, each corresponding to a DSM-IV symptom of PTSD (Foa et al., 1997). The frequency of each symptom is rated on a 4-point scale (0 = not at all or only one time, 3 = five or more times a week/almost always), with total scores ranging from 0 to 51. For PDS subscales, we examined four PTSD symptom clusters defined by King et al. (1998), which separates the DSM-IV avoidance cluster into an effortful avoidance factor consisting of trauma-specific situational and cognitive avoidance items (C1 and C2) and an emotional numbing factor (items C3–C7) that reflects broader affective responses associated with emotional and behavioral withdrawal (e.g., anhedonia, disinterest). This approach maps on more closely to DSM-5 clusters and has shown superior factor structure to the traditional DSM-IV three-factor solution (Elhai & Palmieri, 2011). Internal consistency for the PDS total and cluster scores in this sample was good to excellent: PDS Total: $\alpha = 0.94$; Reexperiencing: $\alpha = 0.89$; Avoidance: $\alpha = 0.80$; Numbing: $\alpha = 0.86$; Hyperarousal: $\alpha = 0.82$.

Dissociation. Dissociative symptoms were measured via the dissociation subscale of the Trauma Symptom Inventory (Briere, 1995), a self-report scale examining PTSD symptoms and associated features. The subscale contains nine items assessing various features of dissociation (e.g., depersonalization, derealization), which were rated regarding frequency in the last month on a 0 (never) to 4 (often) scale. The TSI-Dissociation subscale has shown strong reliability and criterion validity as demonstrated by associations with interpersonal trauma history in clinical samples (Briere et al., 1995). Internal consistency in the present sample was good ($\alpha = 0.81$).

Defensive Avoidance. The Defensive Avoidance subscale of the TSI consists of eight items assessing the tendency to avoid unpleasant thoughts and memories (Briere, 1995). In contrast to assessment of trauma-specific avoidance as measured through PDS items, the TSI Defensive Avoidance scale asks about avoidance of *any* unpleasant memories or painful reminders of one's past and has shown adequate discriminant validity with PTSD symptom clusters and total scores (McDevitt-Murphy et al., 2005). Internal consistency in the present sample was acceptable ($\alpha = 0.78$).

Childhood Trauma Chronicity. Chronicity of childhood trauma was assessed via the Lifetime Trauma Survey (Drescher et al., 1994), a self-report instrument in which respondents indicate whether they experienced a variety of different types of traumas, including childhood physical and sexual assault, along with the number of times each type of event occurred. Information was subsequently verified as part of a standardized clinician-administered trauma interview. Given the difficulty of identifying the exact number of instances in which repeated events such as childhood physical abuse occurred, trauma frequency was rated categorically as occurring either 0, 1, 2–11, 12–52, or 52+ times. For the purposes of this study, participants were categorized as having experienced chronic childhood trauma if they indicated experiencing 12 or more instances of either childhood physical or sexual assault.

Beck Depression Inventory-II (BDI). The BDI (Beck et al., 1996) is a widely used 21-item self-report instrument of depression severity, with scores ranging from 0 to 63, and scores of 29 or above suggesting severe depression. Internal consistency in this sample was strong ($\alpha = 0.89$).

2.3. Treatment

CPT was delivered over the course of twelve 60-min sessions, which took place weekly or twice-weekly (Galovski et al., 2016; Resick et al., 2008). CPT is a protocol-driven treatment that involves helping patients identify inaccurate trauma-related beliefs that maintain PTSD, learn strategies for challenging these beliefs, and develop more accurate, adaptive alternative ways of thinking (Resick, Wachen, et al., 2017). Therapy initially targets beliefs about why the traumatic event occurred (assimilated beliefs) before shifting focus to current and future beliefs that have been impacted by the trauma about the world, oneself, and others. The latter sessions specifically focus on beliefs related to several themes commonly impacted by trauma (i.e., safety, trust, power/-control, esteem, and intimacy). The majority of participants in this study received the version of CPT that included a written account of their most distressing traumatic event, with the exception of a subsample ($n = 24$) from Resick et al. (2008) that completed CPT without the account. A subsample of participants ($n = 42$) from Galovski et al. (2016) also received three sessions of sleep-directed hypnosis prior to beginning CPT, though this additional intervention also did not impact PTSD symptom improvement relative to those who did not receive the sleep intervention. Therapists were masters-level or doctoral-level clinicians trained and supervised by the principal investigator of each trial, and protocol adherence and competence were high (see details in Galovski et al., 2016; Resick et al., 2008).

2.4. Data Analytic approach

Analyses of improvement in PTSD symptoms as a function of baseline psychophysiological variables were conducted using growth curve modeling within a multilevel modeling framework (MLM; Gallop & Tasca, 2009), using SPSS v.28. Analyses were conducted on the intent-to-treat sample, using maximum likelihood estimation within MLM given its advantages in handling missing data (Grund et al., 2019). To estimate the slope of symptom change, time was examined as a level 1 predictor of PDS, and time data were nested within level 2 units (i.e., participants). The time variable was coded based on assessment point, which corresponded with where participants were in treatment (i.e., pre-treatment, before sessions 2, 4, 6, 8, 10 and 12, post-treatment, and at 6-month follow-up). Baseline was coded as 0, with each subsequent assessment point increasing by one. This approach was used because the number of sessions completed is typically considered a more important variable for modeling change in treatment studies than the mere passage of time (Kahn & Schneider, 2012; Resick et al., 2012). -The proportion of missing PDS data across available time points was 33%, with 1% missing data pre-treatment, 32% post-treatment, and 25% at follow-up. Patterns of missing data were examined in relation to baseline variables and demographics. Lower age and Trauma Memory HRR showed significant associations with missingness, and thus were included in all models. Such associations with missingness provide credibility to the assumption that data were missing at random and therefore suitable for use of maximum likelihood estimation to model missing values from observed data (Dziura et al., 2013).

Different change trajectories (i.e., linear, quadratic, cubic, logarithmic) were compared to identify the best-fitting growth curve based on AIC scores. Separate growth curves were modeled for PDS-Total and subscale scores. An autoregressive covariance matrix for repeated measures was used given correlations between repeated PDS assessments, though other covariance structures were examined and did not improve fit. To control for variability in the timing of assessments across individuals and between assessment points, the number of days since the baseline assessment at each timepoint was included as a time-varying covariate. Prior to examining predictors of interest, a dummy-coded study variable and its interaction with time was initially included in the model to determine whether the trajectory of symptom change differed between the two trials, though was only retained if significant. The same approach was taken regarding possible differences among participants receiving hypnosis sessions prior to CPT or among participants receiving CPT without the written account. Given that Trauma Memory HRR could be more strongly linked to outcomes when treatment involves a greater degree of trauma memory recall (e.g., through the written trauma account), follow-up analyses examining whether CPT type (account vs. no account) moderated Trauma Memory HRR effects were also conducted.

To test our primary hypotheses, pretreatment HRR during SDI with the trauma memory (Trauma Memory HRR) and its interaction with

Time were entered as fixed effects predicting PDS, with significant Predictor × Time interactions indicating an association with the slope of symptom change. Identical models were also run with each PDS subscale (defined by King et al., 1998, as discussed previously) as the outcome. Age and its interaction with time were included as a covariate in all models due to evidence that older age is associated with less HRR in emotionally evocative tasks (Uchino et al., 2010), and the association of age with missingness in the present data. For PDS total scores, and when HRR emerged as a significant predictor of PDS cluster scores, a second model was run to examine whether relationships held when controlling for covariates of interest (i.e., stressor HRR, dissociation, defensive avoidance, childhood trauma chronicity). Continuous predictor variables were mean-centered to improve interpretability of estimates. Effect sizes were calculated by examining the percentage reduction in unexplained variance when removing the predictor of interest, analogous to Cohen's f^2 (Selya et al., 2012), and were interpreted using Cohen's (1988) guidelines, with f^2 of 0.02, 0.15, and 0.35 representing small, medium, and large effects, respectively.

We examined Trauma Memory HRR as a predictor of dropout, defined as not completing all 12 sessions of the CPT protocol, in two ways. First, we conducted a logistic regression predicting dropout using the full sample, including those who were enrolled and randomized but never attended a CPT session (i.e., study dropout). Second, we conducted a survival analysis using Cox regression (Singer & Willett, 1993) to predict dropout only among those that attended at least one session of CPT (i.e., treatment dropout). This approach estimates a Hazard Ratio that reflects the risk of treatment dropout at any given point during treatment, rather than the end of treatment. This provides the advantage of greater power and sensitivity by accounting for time (i.e., number of sessions) until discontinuation, instead of treating early and late dropouts identically (Gros et al., 2018). We examined only those who attended at least one session for this analysis because factors that influence how long a patient remains in treatment once therapy is initiated may differ from those impacting dropout prior to the first session (Kehle-Forbes et al., 2016), and we were interested in whether Trauma Memory HRR specifically impacted dropout among patients who had some exposure to CPT. For prediction of both study and treatment dropout, we initially examined just Trauma Memory HRR as a predictor with baseline PDS as a covariate. We subsequently ran analyses including the above-mentioned covariates of interest (i.e., stressor HRR, dissociation, defensive avoidance, childhood trauma chronicity) to evaluate whether Trauma Memory HRR explained unique variance in likelihood of dropout.

We did not correct for multiple comparisons, as a relatively small number of planned tests were conducted based on a priori hypotheses (Althouse, 2016). Thus, all reported p values are uncorrected, with significance values interpreted alongside effect sizes.

3. Results

Means, SDs, and correlations among baseline variables are presented in Table 1. Notably, neither Trauma Memory HRR nor Stressor HRR showed any significant correlations with clinical measures, including reexperiencing symptoms measured by the PDS. Thirty-six percent of the sample showed no increase in heart rate in response to trauma scripts (i.e., had zero or negative values for Trauma Memory HRR). A repeated-measures ANOVA indicated that HRR during the imagery period significantly differed across scene type, $F(2.44, 344.27) = 24.26$, $p < 0.001$, $\eta_p^2 = 0.15$, with Bonferroni-adjusted post-hoc pairwise comparisons indicating significantly greater Trauma Memory HRR ($M = 2.51$, $SE = 0.47$) than Stressor HRR ($M = 0.86$, $SE = 0.32$), mean difference = 1.65, 95% CI [0.31, 3.01], $p < 0.001$, Neutral Scene HRR ($M = -1.25$, $SE = 0.21$), mean difference = 3.76, 95% CI [2.32, 3.01], $p < 0.001$, and Relaxing Scene HRR ($M = -1.04$, $SE = 0.39$), mean difference = 3.54, 95% CI [1.88, 5.20], $p < 0.001$. Thus, the sample as a whole demonstrated expected patterns of reactivity to the various scenes. Mean HRR

Table 1
Descriptive statistics and correlations between baseline variables.

	PDS Total	PDS Reexper.	PDS Avoid.	PDS Numbing	PDS Hyperar.	TSI Defens. Avoid.	TSI Dissociat.	BDI	Chronic Child Abuse	Resting HR	Trauma HRR	Stressor HRR
Mean (SD) or %	31.9 (9.3)	8.4 (3.4)	4.5 (1.6)	8.9 (3.7)	10.2 (3.0)	17.6 (4.5)	12.6 (5.9)	30.2 (10.6)	46%	76.1 (11.4)	2.5 (5.6)	0.9 (3.8)
n	141	141	141	141	141	141	141	141	136	142	142	142
PDS Total	–											
PDS Reexperiencing	0.80**	–										
PDS Avoidance	0.64**	0.52**	–									
PDS Numbing	0.79**	0.37**	0.36**	–								
PDS Hyperarousal	0.86**	0.59**	0.43**	0.61**	–							
TSI Def. Avoidance	0.40**	0.41**	0.46**	0.18*	0.31**	–						
TSI Dissociation	0.44**	0.26**	0.22**	0.42**	0.45**	0.39**	–					
BDI	0.62**	0.40**	0.29**	0.59**	0.57**	0.25**	0.43**					
Chron. Child Abuse	−0.09	−0.18*	−0.12	0.05	−0.06	−0.20*	0.05	0.01	–			
Resting HR	0.06	0.09	0.14	−0.06	0.07	0.09	0.04	0.12	−0.06	–		
Trauma HRR	0.01	0.08	0.08	−0.07	0.00	0.05	0.01	0.05	0.06	0.16	–	
Stressor HRR	0.06	−0.04	0.08	0.08	0.09	0.02	0.11	0.14	0.13	0.04	0.31**	–

Note: $^*p < 0.05$; $^{**}p < 0.01$. PDS = PTSD Diagnostic Scale; TSI = Trauma Symptom Inventory; HRR = Heart Rate Reactivity; BDI = Beck Depression Inventory-II.

during the listen, imagine, and recovery phases for each scene are presented and compared in Supplementary Materials.

3.1. Growth curve modeling

Growth curve modeling of PDS total scores indicated that a logarithmic trajectory of change fit the data best, indicating steeper declines in PTSD symptoms early in treatment that flattened over time. For models examining PDS subscales, the combination of a linear and cubic growth terms provided the best fit, though a logarithmic model had very similar fit and produced identical results, so results from the logarithmic model are described below for ease of interpretation (as it includes only one change parameter) and consistency across models.

Across models, the Study × Log Time interaction was not significant (all p's > 0.11), indicating no differences in the trajectory of change based on which clinical trial the participants were enrolled. Similarly, there were no significant differences in change trajectories for any outcomes between participants who received pre-treatment hypnosis sessions (p's > 0.33) or CPT without the written account (p's > 0.71) relative to rest of the sample. Accordingly, these variables were not included in subsequent models.

3.2. Change in PTSD symptoms

Table 2 shows the fixed effect estimates from models predicting PDS Total, first without covariates of interest (Model 1) and then with covariates included (Model 2). Results from Model 1 indicate show that the Trauma Memory HRR × Log Time interactions effect was not significant ($p = 0.086$), indicating that trauma memory HRR was not significantly associated with change in PDS total scores over time. This nonsignificant effect was in the expected direction (i.e., greater trauma memory HRR predicting a larger decline in PDS), with a small effect size (f2 = 0.031). Table 3 shows fixed effect estimates from models predicting PDS subscales, again without and with covariates. Results from models without covariates showed that Trauma Memory HRR × Log Time interactions significantly predicted both PDS reexperiencing and PDS avoidance ($p = 0.007$ and $p = 0.008$, respectively), indicating that

Table 2
Fixed effect estimates of multilevel models predicting PDS Total.

Predictor	Model 1			Model 2		
	Estimate	SE	p	Estimate	SE	p
Intercept	32.03	0.87	<0.001	32.28	0.98	<0.001
Log *Time*	−8.01	1.05	<0.001	−9.16	1.15	<0.001
Log *Days since Baseline*	−0.13	0.34	.695	−0.02	0.35	0.946
Age	−0.08	0.69	0.237	0.00	0.06	0.976
Trauma Memory HRR	0.06	0.46	0.891	−0.09	0.42	0.834
Stressor HRR				0.11	0.45	0.801
Chronic Child Abuse				−0.62	1.47	0.675
TSI Defensive Avoidance				0.55	0.18	0.002
TSI Dissociation				0.56	0.14	<0.001
Age × Log *Time*	−0.01	0.05	0.912	0.01	0.05	0.878
Trauma Memory HRR* × *Log Time	**−0.57**	**0.33**	**0.086**	**−0.49**	**0.32**	**0.131**
Stressor HRR × Log *Time*				−0.27	0.33	0.427
Chronic Child Abuse × Log *Time*				1.37	1.11	0.220
TSI Def. Avoidance × Log *Time*				−0.36	0.13	0.006
TSI Dissociation × Log *Time*				0.23	0.10	0.028

Note: HRR = Heart rate reactivity. PDS = Posttraumatic Diagnostic Scale; TSI = Trauma Symptom Inventory. HRR = Heart rate reactivity. Bolded row reflects predictor of primary interest for emphasis.

Table 3
Fixed effect estimates of multilevel models predicting PDS Subscales.

Predictor	Model 1			Model 2		
	Estimate	SE	p	Estimate	SE	p
PDS Reexperiencing						
Intercept	8.42	0.30	<0.001	8.71	0.36	<0.001
Log *Time*	−2.22	0.34	<0.001	−2.69	0.37	<0.001
Log *Days since Baseline*	−0.06	0.12	0.624	−0.02	0.13	0.906
Age	−0.02	0.02	0.514	0.01	0.02	0.689
Trauma Memory HRR	0.17	0.16	0.289	0.15	0.16	0.334
Stressor HRR				−0.13	0.16	0.442
Chronic Child Abuse				−0.47	0.53	0.383
TSI Defensive Avoidance				0.25	0.06	<0.001
TSI Dissociation				0.09	0.50	0.057
Age × Log *Time*	−0.00	0.01	0.852	0.00	0.01	0.987
Trauma Memory HRR* × *Log Time	**−0.25**	**0.09**	**0.007**	**−0.24**	**0.09**	**0.007**
Stressor HRR × Log *Time*				0.02	0.09	0.829
Chronic Child Abuse × Log *Time*				0.44	0.31	0.150
TSI Defensive Avoidance × Log *Time*				−0.14	0.04	<0.001
TSI Dissociation × Log *Time*				0.08	0.03	0.010
PDS Avoidance						
Intercept	4.42	0.14	<0.001	4.36	0.17	<0.001
Log *Time*	−1.20	0.18	<0.001	−1.40	0.20	<0.001
Log *Days since Baseline*	−0.06	0.07	0.367	−0.02	0.07	0.705
Age	−0.01	0.01	0.323	0.01	0.02	0.563
Trauma Memory HRR	0.06	0.07	0.420	0.03	0.07	0.692
Stressor HRR				0.02	0.08	0.816
Chronic Child Abuse				0.14	0.25	0.560
TSI Defensive Avoidance				0.17	0.03	<0.001
TSI Dissociation				0.00	0.02	0.958
Age × Log *Time*	0.00	0.01	0.557	0.01	0.01	0.273
Trauma Memory HRR* × *Log Time	**−0.14**	**0.05**	**0.008**	**−0.12**	**0.05**	**0.018**
Stressor HRR × Log *Time*				−0.07	0.05	0.196
Chronic Child Abuse × Log *Time*				0.10	0.17	0.555
TSI Defensive Avoidance × Log *Time*				−0.08	0.02	<0.001
TSI Dissociation × Log *Time*				0.06	0.02	<0.001
PDS Numbing						
Intercept	9.08	0.32	<0.001			
Log *Time*	−2.36	0.38	<0.001			
Log *Days since Baseline*	−0.04	0.13	0.749			
Age	−0.02	0.03	0.413			
Trauma Memory HRR	−0.17	0.17	0.320			
Age × Log *Time*	0.00	0.02	0.909			
Trauma Memory HRR* × *Log Time	**−0.08**	**0.11**	**0.471**			
PDS Hyperarousal						
Intercept	10.17	0.29	<0.001			
Log *Time*	−2.69	0.35	<0.001			
Log *Days since Baseline*	0.16	0.12	0.190			
Age	−0.04	0.02	0.098			
Trauma Memory HRR	0.03	0.15	0.825			

(*continued on next page*)

Table 3 (*continued*)

Predictor	Model 1			Model 2		
	Estimate	SE	p	Estimate	SE	p
Age × Log *Time*	−0.00	0.01	0.801			
Trauma Memory HRR* × *Log Time	**−0.14**	**0.11**	**0.209**			

Note: PDS subscales are those defined by King et al. (1998). Model 2 not run with PDS Numbing or Hyperarousal due to nonsignificant effect of Trauma Memory HRR × Log Time in Model 1. Bolded rows reflect predictor of primary interest for emphasis. HRR = Heart rate reactivity. PDS = Posttraumatic Diagnostic Scale; TSI = Trauma Symptom Inventory.

greater trauma memory HRR was associated with greater declines in reexperiencing and avoidance symptoms. Effect sizes indicated trauma memory HRR accounted for 14.4% of the variance in change in PDS reexperiencing (f2 = 0.144) and 6.1% of the variance in change in PDS avoidance (f2 = 0.061), reflecting medium and small effects, respectively. As hypothesized, these effect sizes were substantially larger than when examining the Trauma Memory HRR × Log Time interaction as a predictor of PDS numbing (f2 = 0.006) and PDS hyperarousal (f2 = 0.016), neither of which were significant (p's > 0.21). Across PDS variables, effects of Trauma Memory HRR × Log Time were not significantly moderated by CPT type (p's > 0.22), thus CPT type was not included in final models in the tables. Effect sizes for CPT with and without the account when predicting total PDS were f^2 = 0.018 and 0.034, respectively, both in the small range. Fig. 1 offers a graphical representation of model-predicted values of PTSD Total and cluster scores when trauma memory HRR is 1 SD above and 1 SD below the mean.

Model 2 in Tables 2 and 3 shows the results of follow-up analyses, in which we included stressor HRR, chronic child abuse, dissociation, defensive avoidance, and their interactions with Log Time as covariates in the prediction of PDS total, PDS reexperiencing and PDS avoidance. Dissociation and defensive avoidance emerged as significant predictors of symptom change of all three outcomes, with less dissociation and greater defensive avoidance each associated with a greater decrease in symptoms (see Table 3). However, the effect of Trauma Memory HRR on PDS Reexperiencing and PDS Avoidance remained significant, Changes in fixed effect estimates of Trauma Memory HRR × Log Time interactions from Model 1 to 2 were small, with a slight decrease in variance explained by PDS Total (f2 = 0.023, from 0.031) and PDS Avoidance (f2 = 0.056, from 0.061), and an increase for PDS Reexperiencing (f2 = 0.208, from 0.144). An additional sensitivity analysis including pretreatment BDI and its interaction with Log time did not meaningfully influence results, with Trauma Memory × Log Time remaining a significant predictor of PDS Reexperiencing (p = 0.006) and PDS Avoidance (p = 0.021), while the effect on PDS Total remained nonsignificant (p = 0.173).

3.3. Study and treatment dropout

Seventy-six (53%) of the 142 participants in the sample completed all 12 sessions of CPT, with no differences in treatment completion due to trial or treatment condition (all p's > 0.58). A logistic regression predicting study dropout indicated that holding age and baseline PDS constant, higher Trauma Memory HRR was associated with a significantly reduced likelihood of study dropout (p = 0.008; see Table 4).[1] The estimated odds ratio indicated a one unit increase in Trauma Memory HRR was associated with a 25% decrease in likelihood of study dropout. As seen in Table 4, including Stressor HRR, dissociation, defensive avoidance, and chronic child abuse as covariates did not meaningful impact the Trauma Memory HRR effect, nor were they significant predictors of dropout.

Among those who attended at least one session of CPT (n = 111), a Cox regression similarly indicated higher Trauma Memory HRR was associated with reduced risk of treatment dropout over the course of CPT, (p = 0.034; see Table 4). The estimated hazard ratio indicated that a one-unit increase in Trauma Memory HRR was associated with a 20% decrease in risk of treatment dropout. Again, the impact of Trauma Memory HRR did not meaningfully change after inclusion of Stressor HRR, dissociation, defensive avoidance, and chronic child abuse as covariates (see Table 4). Additional sensitivity analyses including BDI as an additional covariate produced identical effect estimates for Trauma Memory HRR on both study and treatment dropout.

4. Discussion

This study examined pretreatment heart rate reactivity during trauma memory recall as a predictor of treatment outcome in 142 women receiving CPT for PTSD secondary to interpersonal violence. Given that trauma memory reactivity may be an indicator of a patient's ability to emotionally engage in processing of the trauma, we expected greater trauma memory HRR to predict larger reductions in PTSD symptoms, particularly reexperiencing and avoidance symptom clusters. Results showed that although trauma memory HRR did not significantly predict change in total PTSD symptoms during CPT, lower HRR during trauma memory recall was associated with a smaller reduction in reexperiencing and avoidance symptoms. In addition, lower trauma memory HRR was associated with increased risk of dropout across the full sample and among those who attended at least one session of CPT.

The specific effects seen on reexperiencing and avoidance clusters are notable given that these symptoms reflect the elements of PTSD most directly associated with the trauma memory (Forbes et al., 2011; Gootzeit & Markon, 2011; Hunt et al., 2018). Importantly, the effect on such symptom clusters does not merely reflect that there was more room for improvement in reexperiencing or avoidance among those with greater trauma memory HRR (i.e., regression to the mean), since trauma memory HRR and PDS subscales were not significantly associated at baseline. Rather, results are consistent with the idea that emotional activation during trauma recall is particularly important for altering the affective elements of the trauma memory (Foa & Kozak, 1986; Resick et al., 2008), and therefore should have the most direct impact on symptoms most directly linked to the trauma memory and associated cues (e.g., intrusive memories, cognitive and situational avoidance). In contrast, changes in symptoms more distally related to trauma memory reactivity (i.e., generalized emotional numbing and hyperarousal clusters; King et al., 1998), were not impacted by variability in physiological reactivity during trauma recall. Such distinct effects on changes in reexperiencing and avoidance versus dysphoria or general distress symptoms have been shown in prior PTSD treatment literature (Bryant et al., 2020; Taylor et al., 2003), including a study showing that greater neural activity related to emotional processing predicted larger

[1] Some researchers have used a more lenient definition of CPT completion based on receiving an "adequate dose" of treatment, typically defined as 8–10 sessions (e.g., Alpert et al., 2020; Rizvi et al., 2009). For the present sample, defining study dropout as attending fewer than 10 sessions reduced the number of dropouts by 3. Greater Trauma Memory HRR continued to predict a reduced likelihood of dropout among the full sample, B = −0.28, SE = 0.11, p = 0.010, Exp(B) = 0.76, 95% CI = 0.62 to 0.94, and among those who initiated CPT, B = −0.22, SE = 0.11, p = 0.047, Exp(B) = 0.80, 95% CI = 0.64 to 0.997.

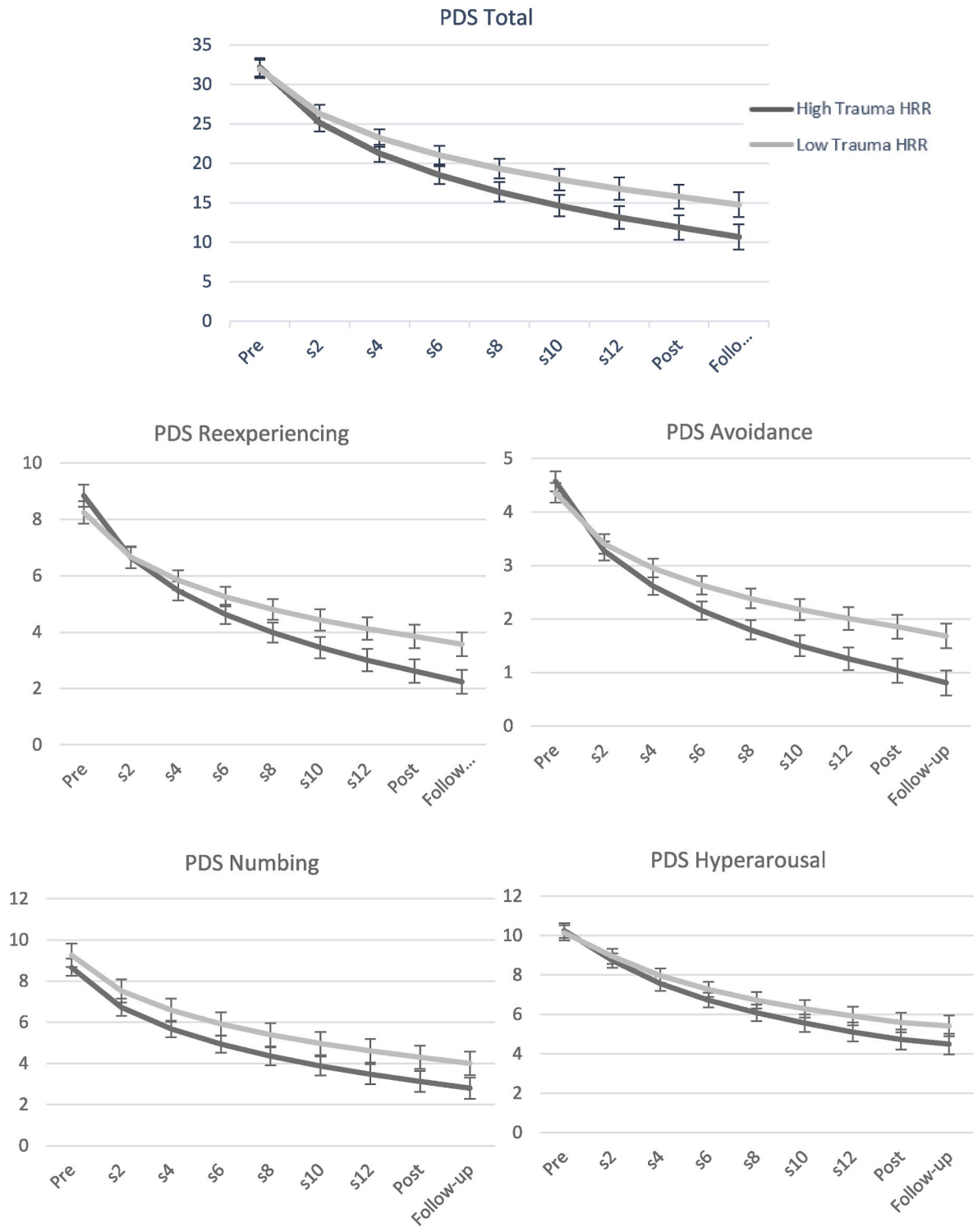

Fig. 1. Model-estimated change trajectories for PDS total and PDS symptom clusters at high (+1 SD) and Low (−1 SD) trauma memory HRR

improvements in reexperiencing and avoidance during PTSD treatment, but not dysphoria-related symptoms (Bryant et al., 2020). In the context of significant heterogeneity in PTSD symptoms (Galatzer-Levy & Bryant, 2013) and the challenges of identifying consistent predictors of PTSD efficacy (Fonzo et al., 2020), such results highlight the value of examining prediction of the most theoretically relevant symptom clusters.

This study is the first examination of the relationship between physiological reactivity during voluntary trauma recall and treatment outcomes in a cognitive treatment for PTSD. To the extent to which cardiac reactivity during SDI at baseline indicates an individual's capacity for emotional activation during treatment, findings support the notion that emotional engagement during trauma processing is an important element of CPT (Galovski et al., 2020; Resick et al., 2008), at least with regard to maximizing improvement in reexperiencing and avoidance symptoms. Findings also align with studies on emotional expression present in written trauma accounts in CPT, which have shown that greater expression of negative emotion in trauma narratives predicts greater likelihood of sudden symptom gains (Sloan et al., 2022)

Table 4
Effect of Trauma Memory HRR and covariates on Study Dropout and Treatment Dropout.

Predictor	Model 1				Model 2			
	B	SE	p	OR/HR (95% CI)	B	SE	p	OR/HR (95% CI)
Study Dropout								
Intercept	0.08	0.20	0.673	1.09 (NA)	0.21	0.26	0.420	1.23 (NA)
Age	−0.04	0.02	0.014	0.96 (0.93–0.99)	−0.04	0.02	0.018	0.96 (0.93–0.99)
Baseline PDS	0.00	0.02	0.842	1.01 (0.97–1.05)	0.00	0.02	0.891	1.00 (0.96–1.05)
Trauma Memory HRR	**−0.28**	**0.11**	**0.008**	**0.75 (0.61–0.93)**	**−0.28**	**0.11**	**0.010**	**0.75 (0.61–0.93)**
Stressor HRR					0.06	0.12	0.630	1.04 (0.83–1.31)
Chronic Child Abuse					−0.28	0.38	0.465	0.75 (0.36–1.56)
TSI Defensive Avoidance					−0.43	0.38	0.265	0.65 (0.31–1.38)
TSI Dissociation					0.31	0.34	0.369	1.36 (0.69–2.67)
Treatment Dropout								
Age	−0.03	0.02	0.059	0.97 (0.94–1.00)	−0.03	0.02	0.071	0.97 (0.94–1.00)
Baseline PDS	0.015	0.02	0.462	1.02 (0.98–1.06)	0.02	0.02	0.473	1.02 (0.97–1.06)
Trauma Memory HRR	**−0.23**	**0.11**	**0.034**	**0.80 (0.65–0.98)**	**−0.22**	**0.11**	**0.041**	**0.80 (0.65–0.99)**
Stressor HRR					0.03	0.11	0.774	1.03 (0.83–1.28)
Chronic Child Abuse					−0.49	0.37	0.185	0.61 (0.30–1.27)
TSI Defensive Avoidance					−0.31	0.38	0.412	0.73 (0.35–1.54)
TSI Dissociation					0.13	0.35	0.705	1.14 (0.57–2.28)

Note: Models predicting Study Dropout (logistic regression) include all participants ($n = 142$). Models predicting Treatment Dropout (Cox regression) include participants who attended at least one CPT session ($n = 111$), and account for time until discontinuation. Bolded rows reflect predictor of primary interest for emphasis. OR = Odds Ratio (of Study Dropout); HR = Hazard Ratio (of Treatment Dropout). HRR = Heart rate reactivity. PDS = Posttraumatic Diagnostic Scale; TSI = Trauma Symptom Inventory.

and less dropout (Alpert et al., 2020). Importantly, results do not indicate that low heart rate reactivity is by any means determinative of a poor response to CPT, given that significant associations emerged only at the symptom cluster level and effect sizes were modest (i.e., moderate for reexperiencing, small for avoidance). However, associations with dropout further support the idea that physiological activation to the trauma memory can be a prognostic indicator of benefit from CPT. One possibility for this latter finding is that patients with minimal physiological reactivity to the trauma memory at baseline may be particularly accustomed to blunted responses to the trauma memory (Sack et al., 2012), making the encouragement of affective expression during trauma processing particularly aversive, and consequently increasing chances of treatment discontinuation. Interestingly, Alpert et al., (2023) found therapist encouragement of affective expression to be associated with greater dropout in a sample of CPT patients overlapping with the current study sample. This could support the above hypothesis to the extent that therapists encouraged affective expression more with patients who were more emotionally blunted and already at greater risk of dropout. However, it also may indicate that such encouragement has deleterious consequences for treatment retention. Future research examining reasons for dropout in relation to such variables could help speak to these possibilities.

The associations between cardiac reactivity during trauma recall and treatment outcome in CPT partly parallel those in exposure-based treatments, where trauma memory reactivity has consistently predicted PTSD symptom improvement (Pitman et al., 1996; Tuerk et al., 2018; Wangelin & Tuerk, 2015; Wisco et al., 2016), suggesting the possibility of shared mechanisms of change. However, the fact that cardiac reactivity did not significantly predict total PTSD symptom change in this study could indicate that such reactivity plays a smaller role in recovery during PTSD treatments that rely more on processing the meaning of the event (i.e., a cognitive orientation) versus systematic exposure. Direct comparisons of the relationship between physiological reactivity and PTSD symptom reduction across cognitive and exposure-based treatments are needed to formally test this hypothesis. It is worth noting that the majority of participants in the current study completed a written account of their trauma memory as part of CPT, particularly in light of the fact that CPT is now commonly delivered without this account (Resick, Wachen, et al., 2017). It is possible results were driven to some degree by using this method of recounting the trauma narrative, which overlaps with elements of exposure-based treatments, particularly WET. That said, the mere assignment of the trauma narrative in CPT may not reflect the actual dose of exposure to the trauma memory. Compliance with the trauma narrative assignment tends to be variable (Stirman et al., 2018), and regardless of whether the written narrative is included in the protocol, the administration of CPT requires that the patient engages with the trauma memory by describing both the traumatic event and its impact in some detail. There was no evidence that CPT type moderated the effect of cardiac reactivity in the present study, though conclusions drawn from such an analysis are necessarily tentative given the small number of patients receiving CPT without the account ($n = 24$).

One important consideration for interpreting the results of this study involves better understanding what exactly low cardiac reactivity during SDI reflects. Although prior research has linked such low reactivity in PTSD samples to dissociation (Griffin et al., 1997; Lanius et al., 2002; Sack et al., 2012) and history of repeated childhood trauma (D'Andrea et al., 2013; Iffland et al., 2020), no association between those variables were seen at baseline in this study, nor were there significant relationships with defensive avoidance, total PTSD symptoms or PTSD symptom clusters. Moreover, including dissociation, chronic child abuse, and defensive avoidance as covariates in models predicting outcome did not influence results, indicating that trauma memory HRR explains unique variability in outcomes. Prior research has also found the low physiological reactivity to trauma imagery seen in subgroup of PTSD patients to extend to other stressful imagery (McTeague et al., 2010). However, effects of Trauma Memory HRR remained when Stressor HRR was included in the analysis, and Stressor HRR was not a significant predictor, further illustrating the specificity of trauma memory reactivity on treatment outcomes.

One possible reason for such results is that there may be varying reasons for low cardiac reactivity for different individuals, which in turn may correspond to different explanations for reactivity's impact on treatment outcomes. Rather than reflecting processes related to emotional or physiological blunting, some individuals' low cardiac reactivity may reflect trauma-related psychopathology that is less centrally characterized by trauma memory-specific autonomic arousal, and is more strongly defined by posttraumatic symptoms reflecting negative affect (e.g., guilt, dysphoria), general anxious arousal, and associated cognitions (Pineles & Orr, 2018; Ramage et al., 2016; Tuerk et al., 2018). In cases where PTSD is less centrally driven by trauma memory reactivity, a focus on emotional activation to the trauma memory in an intervention like CPT with the written account may be less well suited to a patient's needs, potentially contributing to the increased dropout and

reduced improvement in reexperiencing and avoidance symptoms seen in the present study. Future research would benefit from investigating possible subgroups of individuals who exhibit reduced HR reactivity during trauma recall, which could illuminate mechanisms of reactivity-outcome relationships and inform personalized approaches to maximizing treatment response among low reactors. For instance, some of these patients may benefit from techniques aimed at facilitating greater emotional activation, whereas others may benefit from less attention to recounting details of the trauma memory and more immediate focus on cognitive work (though it should be noted that this does not preclude emotional activation or trauma memory engagement).

Future research would benefit from including additional measures during the SDI task that could clarify reasons for reduced cardiac reactivity during pretreatment trauma recall, including subjective ratings of emotional distress, dissociation, cognitive avoidance, and task engagement, and additional physiological metrics like heart rate variability and skin conductance, which would allow for examination of the relative roles of parasympathetic and sympathetic nervous system activity implicated in dissociation, blunted stress reactivity and hyperarousal (D'Andrea et al., 2013; Sack et al., 2012). The reliance on heart rate reactivity as the sole indicator of emotional responding during SDI is a limitation of this study, particularly given the frequent discordance seen between and within physiological and subjective measures of reactivity (Lang et al., 2016; McTeague et al., 2010). As a result, the extent to which low heart rate reactivity to the trauma memory predicts outcomes because it reflects a specific form of physiological blunting vs. a broader pattern of emotional disengagement is unknown and in need of further research. It is also possible that low heart rate reactivity for some individuals reflected a lack of task engagement when asked to imagine their trauma memory, which was not formally measured, though trauma memory HRR was not associated with either of the two measures of avoidance used in this study.

A related limitation is that no data on physiological or subjective emotional reactivity during trauma processing in CPT sessions were included. As a result, we cannot definitively speak to how much cardiac reactivity during SDI at pretreatment was related to emotional reactivity during trauma processing in session. Future research in this area would benefit from linking pretreatment characteristics like trauma memory reactivity to in-session processes (e.g., physiological arousal during trauma processing) to clarify mechanisms and identify possible avenues for addressing barriers to treatment response. Such research could also add to the mixed body of findings on the relationship between changes in physiological reactivity during trauma recounting and treatment outcome, which has largely focused on exposure (Rupp et al., 2017; Yang et al., 2021). Moreover, it would help to address the possibility that extraneous factors associated with heart rate reactivity that were unaccounted for in this study (e.g., metabolic and social factors) were driving associations with treatment outcomes in the present study.

A third limitation is that combining data across two clinical trials with slight differences in study procedures inherently introduces noise into the data that can impact the ability to detect predictors of treatment outcomes. Although study settings, physiological equipment, and study investigators were the same and we confirmed there were no differences in outcomes across trials or variations in treatment procedures, less variability across the study sample and design could increase the capacity to detect effects. At the same time, drawing data from two studies enabled us to analyze a much larger sample of patients receiving CPT. This is an important strength of this study, particularly in the context of such limited sample sizes in prior research in this area.

A final limitation is that results from this sample of women with PTSD secondary to interpersonal violence may not generalize to other treatment-seeking samples with PTSD. In particular, the extensive history of interpersonal trauma in this sample, with the majority of the sample experiencing sexual and/or physical abuse in both childhood and adulthood, is notable. Given the previously demonstrated associations between childhood trauma and trauma chronicity with reduced physiological trauma memory reactivity (D'Andrea et al., 2013; Iffland et al., 2020), such a sample characteristic may have led to relatively high frequency of blunted heart rate reactivity. In fact, the sample did exhibit notably low levels of cardiac reactivity to the trauma memory compared to other PTSD samples undergoing SDI (e.g., Orr et al., 1998; Pineles et al., 2013): 36% of participants showed either a decrease or complete lack of increase in heart rate during trauma recall, which is not typically seen even in trauma-exposed samples without PTSD (e.g., Keane et al., 1998; Orr et al., 1998; Pineles et al., 2013). Accordingly, examination of such relationships in a wider variety of PTSD samples will be important.

In summary, this study demonstrates that lower heart rate reactivity during trauma recall prior to CPT does not predict reduction in PTSD symptoms as a whole, but is associated with less improvement in PTSD reexperiencing and avoidance symptoms and greater likelihood of treatment dropout. Thus, the relationship previously seen between trauma memory reactivity and response to exposure-based PTSD treatments is seen for some outcomes of a more cognitively oriented therapy such as CPT. To the extent that heart rate reactivity when recalling a trauma memory is an indicator of emotional engagement, these results support the idea that the capacity for emotional activation when revisiting such a memory contributes to CPT response. Additional research is needed to better clarify the various mechanisms by which heart rate reactivity during trauma recall may relate to treatment outcomes, which in turn could inform tailored approaches to mitigating suboptimal treatment response among those with low trauma memory reactivity.

Funding statement

This research was supported by funding from the National Center for Complementary and Integrative Health (NCCIH) grant R34-MH-074-937 awarded to Tara E. Galovski, the National Institute of Mental Health (NIMH) grant 2-R01-MH51509 awarded to Patricia A. Resick, and NIMH grant K23-MH129878 awarded to Joseph K. Carpenter. The contents of this research are the sole responsibility of the authors and do not necessarily represent the views of the NCCIH or NIMH.

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Joseph K. Carpenter: Conceptualization, Formal analysis, Writing – original draft. **Suzanne L. Pineles:** Conceptualization, Writing – review & editing. **Michael G. Griffin:** Data curation, Methodology, Project administration, Writing – review & editing. **Shivani Pandey:** Data curation, Visualization. **Kimberly Werner:** Data curation, Project administration, Writing – review & editing. **Natalia M. Kecala:** Data curation. **Patricia A. Resick:** Funding acquisition, Writing – review & editing. **Tara E. Galovski:** Conceptualization, Funding acquisition, Supervision, Writing – review & editing.

Declaration of competing interest

Dr. Resick receives royalties from Guildford Press for sales of the Cognitive Processing Therapy manual. The other authors have no competing interests to report.

Data availability

Data will be made available on request.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.brat.2024.104519.

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