



Research paper

Negative appraisals and fear extinction are independently related to PTSD symptoms



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ARTICLE INFO

Keywords:

PTSD
Fear extinction
Threat expectancy
Negative appraisals

ABSTRACT

Background: Considerable research has revealed impaired fear extinction to be a significant predictor of PTSD. Fear extinction is also considered the primary mechanism of exposure therapy, and a critical factor in PTSD recovery. The cognitive theory of PTSD proposes that symptoms persist due to excessive negative appraisals about the trauma and its sequelae. Research has not yet examined the relationship between fear extinction and negative appraisals in PTSD.

Methods: A cross-sectional sample of participants with PTSD ($n = 21$), and trauma-exposed controls ($n = 33$) underwent a standardized differential fear conditioning and extinction paradigm, with skin conductance response (SCR) amplitude serving as the index of conditioned responses. The Posttraumatic Cognitions Inventory (PTCI) was used to index catastrophic negative appraisals.

Results: Participants with PTSD demonstrated a slower decrease in overall SCR responses during extinction and greater negative appraisals compared to the group. A moderation analysis revealed that both negative trauma-relevant appraisals and fear extinction learning were independently associated with PTSD symptoms, but there was no moderation interaction.

Limitations: The current study was limited by a modest sample size, leading to the inclusion of participants with subclinical PTSD symptoms. Further, the current study only assessed fear extinction learning; including a second day extinction recall task may show alternative effects.

Conclusions: These findings indicate that negative appraisals and fear extinction did not interact, but had independent relationships with PTSD symptoms. Here we show for the first time in an experimental framework that negative appraisals and fear extinction play separate roles in PTSD symptoms.

1. Introduction

Posttraumatic Stress Disorder (PTSD) is characterized by persistent fear, intrusive memories, and negative appraisals about oneself and a traumatic event (Dunmore et al., 1999). Symptoms naturally subside in many trauma survivors, but persist as PTSD in approximately 2–10% of people (Atwoli et al., 2015; Ramchand et al., 2010). The cognitive theory of PTSD (Ehlers and Clark, 2000) proposes that two key mechanisms underlie the persistence of PTSD: negative appraisals relating to the trauma and its sequelae, and poorly elaborated autobiographical memories and conditioned fear responses which readily prime intrusive memories that are triggered by trauma remin-

ders (Ehlers and Clark, 2000).

A considerable body of research supports the idea that PTSD develops and is maintained by negative cognitions about a significant traumatic event (Dunmore et al., 1999, 2001; Ehlers et al., 1998, 2003). That is, traumatic experiences lead to negative appraisals about the trauma and its sequelae, causing feelings of current threat, persistent avoidance, and generalization of fear (Ehlers and Clark, 2000). Numerous studies have shown that acute post-trauma negative appraisals significantly predict increased PTSD symptoms in adults and children following a range of traumatic events (e.g., Dunmore et al., 2001; Ehlers et al., 1998, 2003; Ehring et al., 2008). Moreover, the tendency to engage in negative appraisals prior to a traumatic event

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predicts PTSD symptoms several years after exposure (Bryant and Guthrie, 2007).

A second influential model proposes that PTSD develops from impaired fear extinction learning and recall (Pitman et al., 2012). In this model, fear conditioning occurs during trauma, and re-exposure to trauma reminders and subsequent avoidance of reminders facilitate conditioned fear and fear generalization. Extinction typically occurs when the conditioned stimuli are presented in the absence of any aversive consequence, and in the context of trauma this typically involves experience of benign trauma reminders. In this sense, the minority of trauma survivors who experience persistent symptoms can be regarded as suffering impaired extinction (Davis and Myers, 2002). Considerable evidence demonstrates that individuals presenting with PTSD show impairments in fear extinction and extinction recall (e.g., Milad et al., 2008; Norrholm et al., 2011; Shvil et al., 2014; Zuj et al., 2016a). Recent studies have also found pre-trauma extinction learning significantly predicts increased post-traumatic stress (Guthrie and Bryant, 2006; Lommen et al., 2013; Orr et al., 2012). Further, psychophysiological indices of conditioned fear (e.g., fear-potentiated startle and skin conductance response) have been suggested as intermediate phenotypes and central factors of trauma- and stressor-related disorders (Briscone et al., 2014; Zuj et al., 2016b).

These models are not mutually exclusive, given that Ehlers and Clark (2000) also suggest that intrusive memories and conditioned fear responses triggered by trauma reminders are thought to reinforce negative appraisals, which act to maintain anxiety and sense of current threat. Further, longitudinal evidence has found that PTSD symptoms can be predicted by pre-trauma fear extinction learning (Guthrie and Bryant, 2006), and maladaptive pre-trauma negative appraisals (Bryant and Guthrie, 2007). Therefore, pre-trauma impaired extinction capacity and heightened negative appraisals may interact to potentiate fear responses following trauma. In an experimental framework, negative appraisals may moderate the relationship between fear extinction and PTSD symptoms. To our knowledge, no previous studies have examined fear extinction and negative appraisals in tandem, in relation to PTSD.

In the present study, we were interested in understanding the extent to which fear extinction learning and negative appraisals are uniquely and interactively related to PTSD symptom severity. On the basis of Ehlers and Clark's (2000) model, we predicted first that excessively negative appraisals (indexed by the Posttraumatic Cognitions Inventory (PTCI)) would be associated with increased PTSD symptom severity. Second, on the basis of considerable cross-sectional and longitudinal evidence we hypothesized that impaired fear extinction learning would be associated with PTSD symptoms. As yet, studies have not considered these two prevailing models in tandem, thus moderation analyses were conducted to test for a possible interaction between fear extinction learning and negative appraisals in influencing PTSD symptom severity.

2. Methods

2.1. Participants

Fifty-four participants aged 18–63 years ($M = 29.5$ years, $SD = 12.2$ years; 26 males and 28 females) comprised two groups: PTSD ($n = 21$) and trauma-exposed without PTSD (TC; $n = 33$). The PTSD and TC groups were classified on the basis of experiencing a criterion A stressor, whereby physical integrity was threatened (American Psychiatric Association, 2000, 2013) using the Traumatic Events Questionnaire (TEQ; Vrana and Lauterbach, 1994). Traumatic events included war-related combat ($n = 5$), life-threatening accident ($n = 21$), natural disaster ($n = 26$), witnessing a traumatic event ($n = 34$), assaulted or molested ($n = 21$), threatened or held captive ($n = 13$), and tortured or terrorist victim ($n = 2$). Mean years since trauma for the PTSD group was 10.1 years ($SD = 12.8$ years) and 10.5 years ($SD = 11$ years) for the TC group. Participants were classified into either the PTSD or TC group using the PTSD Checklist (PCL-C) for DSM-IV

(Weathers et al., 1994). DSM-IV diagnostic criteria used to estimate PTSD diagnosis consisted of at least one intrusive memory symptom, three avoidance behavior symptoms, and two hyperarousal symptoms (American Psychiatric Association, 2000). The PCL-C for DSM-IV was used as recruitment occurred prior to diagnostic instruments for the DSM-V being available. The University of Tasmania Social Sciences ethics committee approved the study protocol. Informed consent of the participants was obtained after the nature of the procedures had been fully explained.

2.2. Measures

2.2.1. PTSD checklist-civilian version

The PCL-C (Weathers et al., 1994) is a 17-item self-report measure that provides diagnostic information according to PTSD criteria on the DSM-IV (American Psychiatric Association, 2000). Responses are made on a 5-point Likert scale in regards to how often participants were distressed by each symptom in the past month, ranging from 1 (“Not at all”) to 5 (“Extremely”). The PCL-C for DSM-IV shows strong psychometric properties (Wilkins et al., 2011). The PCL-C total also provides an ordinal measure of symptom severity with a recommended clinical PTSD cut-off of 50, and subclinical PTSD of 40 (National Center for Posttraumatic Stress Disorder, n.d.). In the current sample, all participants in the PTSD group showed a PCL-C total score greater than 40%, and 52.4% of the PTSD group showed a PCL-C total score greater than 50.

2.2.2. Posttraumatic cognitions inventory

The PTCI (Foa et al., 1999) is a 33-item self-report measure of negative posttraumatic appraisals regarding the self, world, and self-blame. Responses are made on a 7-point Likert scale from 1 (“totally disagree”) to 7 (“totally agree”). The PTCI has demonstrated good psychometric properties (Foa et al., 1999).

2.3. Fear conditioning and extinction paradigm

The present study employed a standardized differential fear conditioning and extinction paradigm used previously (Zuj et al., 2016a, 2017). Findings from a subset of the participants in the current study have been reported elsewhere examining the impact of hours-since-waking (Zuj et al., 2016a) and cortisol reactivity (Zuj et al., 2017) on fear extinction learning in PTSD. The unconditioned stimulus (US) was a 500 ms mild electric shock delivered to the first interosseous muscle of the dominant hand, set to a level considered “highly annoying, but not painful” by each participant prior to the task (Orr et al., 2000). Conditioned stimuli were red and blue circles presented individually for 12 s on a computer screen. The testing protocol included four experimental phases: *habituation*, *acquisition*, *early extinction*, and *late extinction*. During *habituation*, participants were exposed to four trials of each colored circle (eight trials in total). During *acquisition*, one of the colored circles (CS+) was followed by the US (mild electric shock) immediately following CS+ offset on all five trials (100% reinforcement schedule; Orr et al., 2000) while the other colored circle was not reinforced on any of the five trials (CS-; ten trials in total). The *early extinction* phase consisted of five trials of the CS+ (with no reinforcement) and five trials of the CS- (ten trials in total), followed by a short break of no longer than one minute before participants completed the *late extinction* phase (e.g., Milad et al., 2005), which mirrored early extinction. Trial order was pseudo-random, with no more than two consecutive CS+ or CS- trials, and inter-trial intervals ranged from 12 to 21 s. Immediately following extinction, participants were asked which colored circle was associated with the mild electric shock, and all participants reported accurate contingency awareness.

2.4. Skin conductance response

Skin conductance level was measured through a 22 mV_{rms}, 75 Hz constant-voltage coupler (FE116, ADI Instruments) with bipolar electrodes on the intermediate phalange of the first and third fingers of the non-dominant hand, sampled at 512 Hz and stored at 64 Hz, and recorded in micro-siemens (μ S). Skin conductance response (SCR) amplitude to the CS+ and CS- was calculated by subtracting the mean SCL during the 2 s prior to stimulus onset from the maximum SCL during the 12 s stimulus duration. SCR values were square-root transformed, and the absolute value of negative scores was transformed and the negative sign replaced. Differential conditioned responding (DCR) was calculated by subtracting the SCR of the first CS- from the first CS+, for all trials (Menz et al., 2013; Zuj et al., 2016a). For use in moderation analyses, DCR change scores were calculated for the extinction phases by subtracting the trial 5 DCR from the trial 1 DCR, with larger values representing a greater change in differential fear responding across the experimental phase (i.e., better fear extinction performance).

2.5. US-expectancy ratings

During the 12 s stimulus presentation, participants were asked to rate their threat expectancy of the US on a 0–100 visual analogue scale (VAS; 0 “certain no electrical stimulus”; 100 “certain electrical stimulus”; as previously used by Lommen et al., 2013). Self-report US-expectancy ratings are often included in fear conditioning paradigms alongside objective physiological recordings (e.g., Kindt and Soeter, 2013; Norrholm et al., 2011; Vervliet et al., 2007).

2.6. Statistical analyses

Two (Group) \times 2 (CS) \times 5 (trial) mixed-model analyses of variance (ANOVA) were conducted for each phase (with four trials for habituation) to examine fear conditioning and extinction across groups. Analyses were identical for both SCR and US-expectancy data. Greenhouse-Geisser corrections were made for within-subjects variables where necessary. Brown-Forsythe *F*-ratio corrections were made where homogeneity of variance was violated. Pairwise comparisons were conducted with Bonferroni corrections or Games-Howell tests where appropriate. Moderation is a regression-based statistical technique that can be used in research investigating the circumstances by which a predictor variable is associated with an outcome, and analyses were conducted using the PROCESS macro for SPSS (Model 1; Hayes, 2013). An alpha level of $\alpha = .05$ was used for all tests of statistical significance. Effect sizes are reported as Cohen's *d* following the criteria of .2, .5, and .8 as small, moderate and large effects, respectively (Cohen, 1988). Partial-eta squared (η_p^2) are reported as effect sizes for mixed-model ANOVAs.

3. Results

3.1. Descriptives and clinical data

One-way ANOVA showed that there was a significant between-group difference in PCL-C total symptom severity, $F(1, 23.15) = 127.68, p < .001$, with the PTSD group displaying significantly elevated PTSD symptoms compared to the TC group. Further, the PTSD group displayed significantly higher levels of negative appraisals, and symptoms of depression, anxiety, and stress than the TC group (see Table 1 for demographic and inferential data). There were no significant group differences on age or alcohol use.

3.2. Threat expectancy

During the habituation phase, 2 (group) \times 2 (CS) \times 4 (trial) mixed-

model ANOVA revealed no significant main effects or interactions. During acquisition, there were significant main effects of CS and trial. These main effects were superseded by a significant CS \times trial interaction, $F(3.05, 146.36) = 99.90, p < .001, \eta_p^2 = .675, \epsilon = .762$, with differential threat expectancy to the CS+ and CS- increasing over trials, i.e., greater threat expectancy developed to the CS+ and reduced threat expectancy to the CS-. There was also a significant CS \times trial interaction during early extinction, $F(3.26, 159.66) = 7.02, p < .001, \eta_p^2 = .125, \epsilon = .815$, with differential threat expectancy decreasing over the phase. Specifically, by the end of the early extinction phase, participants were showing reduced differential threat expectancy between the CS+ and the CS-. During the late extinction phase there was a significant main effect of CS, $F(1, 49) = 20.52, p < .001, d = .40$, with greater threat expectancy reported to the CS+ compared to the CS- ($M = 35.82, 95\% \text{ CI}[28.50, 50.21], SD = 25.99$; and $M = 25.87 [19.20, 32.54], SD = 23.71$, respectively). Further, there was a significant main effect of trial, $F(2.39, 117.29) = 25.43, p < .001, \eta_p^2 = .342, \epsilon = .598$, with threat expectancy reducing over the experimental phase, irrespective of group and CS-type. During late extinction there was also a significant group main effect, $F(1, 49) = 7.42, p = .009, d = .88$, with the PTSD group displaying significantly greater threat expectancy than the TC group ($M = 39.85 [29.49, 50.21], SD = 23.08$; and $M = 21.84 [13.52, 30.16], SD = 18.48$, respectively).

3.3. SCR amplitude data

3.3.1. Habituation

During the habituation phase, mixed-model ANOVA showed a significant main effect of trial, $F(2.69, 139.61) = 2.97, p = .039, \eta_p^2 = .054, \epsilon = .895$, as SCR amplitude became smaller over the course of the phase as participants became habituated to the task (see Fig. 1).

3.3.2. Acquisition

During the acquisition phase, mixed-model ANOVA showed a significant main effect of CS, $F(1, 52) = 40.08, p < .001, d = .93$, with significantly larger SCR amplitude to the CS+ ($M = .90 [.75, 1.05], SD = .54$) than the CS- ($M = .56 [.41, .70], SD = .52$) reflecting acquisition of fear conditioning to the CS+. There was also a significant main effect of trial, $F(3.54, 184.31) = 10.96, p < .001, \eta_p^2 = .174, \epsilon = .886$, with SCR amplitude becoming smaller across the acquisition phase (see Fig. 1). No further main effects or interactions were significant during acquisition.

3.3.3. Early extinction

Mixed-model ANOVA revealed a significant main effect of CS, $F(1, 52) = 7.47, p = .009, d = .30$, showing that, pooled across group and trial, the CS+ continued to elicit a larger SCR ($M = .63 [.52, .74], SD = .40$) than the CS- ($M = .51 [.39, .62], SD = .41$). Further, there was a significant main effect of trial, $F(3.44, 178.93) = 27.40, p < .001, \eta_p^2 = .345, \epsilon = .860$, and a significant group \times trial interaction, $F(3.44, 178.93) = 3.17, p = .020, \eta_p^2 = .057, \epsilon = .860$. Test of simple main effects show that the PTSD and TC groups significantly differed in their responses on trial 2 of early extinction, $F(1, 52) = 4.73, p = .034, d = .61$, with the PTSD group producing a significantly larger SCR ($M = .76 [.54, .97], SD = .49$) than the TC group ($M = .46 [.28, .63], SD = .49$). Further, the PTSD group displayed no significant change from trial 1 to trial 2 ($p = .109$) and a significant difference from trial 2 to trial 3 ($p = .028$), with no significant differences between the trials thereafter ($ps > .05$). The TC group however displayed a significant reduction in responses from trial 1 to trial 2 ($p < .001$), with no further differences thereafter ($ps > .05$). Despite a small effect size, this interaction suggests that the PTSD group displayed a slower decrease in overall SCR amplitude during the early extinction phase compared to the TC group.

Table 1
Mean scores and standard deviations of demographic and clinical measures.

Measures	PTSD (n = 21)	TC (n = 33)	Test statistic	p
Demographic data				
- Age (years)	32.67 (14.62)	27.45 (10.05)	$F_{(1, 32.02)} = 2.05$.162
- Sex	13F, 8M	15F, 18M	$\chi^2_{(1)} = 1.39$.238
PCL-C				
- Total	52.52 (11.38)	23.39 (3.98)	$F_{(1, 23.15)} = 127.68$	< .001
- Intrusive	3.00 (1.22)	.27 (.52)	$F_{(1, 24.59)} = 93.54$	< .001
- Avoidance	4.24 (1.81)	.42 (.71)	$F_{(1, 23.93)} = 84.62$	< .001
- Hyperarousal	3.52 (1.03)	.39 (.66)	$F_{(1, 30.46)} = 153.76$	< .001
PTCI				
- Total	113.32 (36.91)	73.33 (28.90)	$F_{(1, 50)} = 18.81$	< .001
- Self	2.92 (1.21)	1.83 (.83)	$F_{(1, 27.81)} = 12.00$.002
- World	4.59 (1.33)	3.02 (1.23)	$F_{(1, 50)} = 18.59$	< .001
- Self-blame	3.23 (1.48)	2.16 (1.09)	$F_{(1, 50)} = 9.03$.004
DASS				
- Depression	9.43 (5.76)	2.21 (2.38)	$F_{(1, 24.41)} = 29.75$	< .001
- Anxiety	8.14 (4.26)	1.97 (1.91)	$F_{(1, 25.20)} = 39.12$	< .001
- Stress	13.62 (6.34)	4.88 (3.17)	$F_{(1, 26.44)} = 34.40$	< .001
AUDIT				
	6.86 (5.46)	6.12 (3.92)	$F_{(1, 33.04)} = .29$.596

Note: PCL-C = PTSD Checklist-Civilian version; PTCI = Posttraumatic Cognitions Inventory; DASS = Depression Anxiety Stress Scale; AUDIT = Alcohol Use Disorders Identification Test.

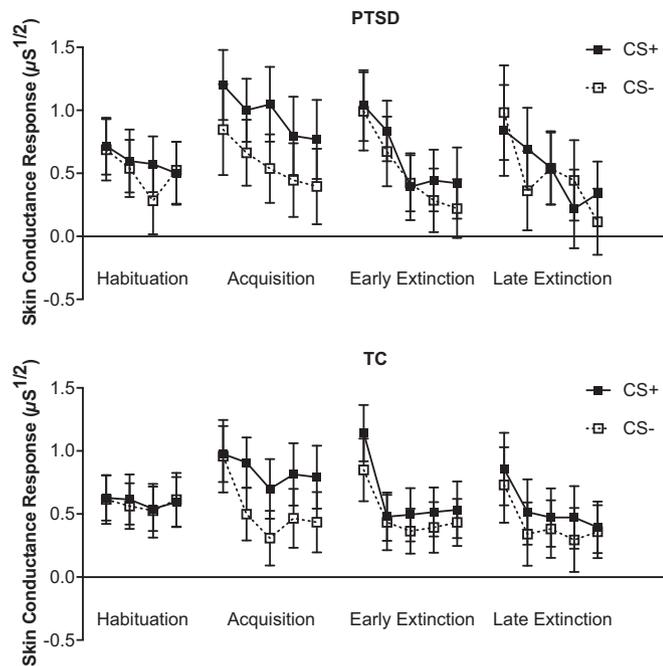


Fig. 1. Skin conductance responses to the CS+ and CS- throughout all phases of the differential fear conditioning and extinction paradigm are displayed separately for the PTSD group (top panel) and TC group (bottom panel). Vertical axes display square-root transformed SC responses ($\mu S^{1/2}$).

3.3.4. Late extinction

During late extinction there was no longer a significant difference in CS+/- responding, $F(1, 52) = 2.83, p = .099, d = .17$, however there remained a significant trial main effect, $F(3.33, 173.36) = 11.73, p < .001, \eta_p^2 = .184, \epsilon = .833$, with SCR amplitude becoming smaller over the experimental phase (see Fig. 1).

3.4. Fear extinction and negative appraisals: moderation analysis

To examine the relationship between fear extinction learning and negative appraisals in PTSD, a moderation analysis was conducted (Model 1; Hayes, 2013). PCL-C total was included as the outcome variable, and PTCI total was included as the moderator. As primary group effects in fear extinction learning were found during early

Table 2
Linear model of predictors of PCL total.

	b	SE b	t	p
Constant	33.81 [30.33, 37.29]	1.73	19.53	< .001
Negative appraisals	.28 [.18, .37]	.05	5.92	< .001*
Early extinction DCR change	-4.08 [-7.70, -.47]	1.80	-2.27	.028
Negative appraisals × early extinction	-.01 [-.11, .10]	.05	-.17	.865

Note: SE = Standard Error; Square brackets show 95% confidence intervals of b.
* Significant after including depression, anxiety, and stress as covariates.

extinction, an early extinction DCR change score was calculated and entered into the model as the predictor variable. The total model predicted a significant amount of variance in PTSD symptoms, $R^2 = .433, F(3, 48) = 12.20, p < .001$. As seen in Table 2, both negative appraisals and early extinction DCR change showed significant relationship with PTSD symptoms, however there was no significant interaction between negative appraisals and extinction. That is, negative appraisals did not moderate the relationship between fear extinction learning and PTSD symptom severity. The depression, anxiety, and stress scores of the DASS were included as covariates to ensure that the relationship between negative appraisals and PTSD symptoms was not inflated by an underlying negative affectivity bias. Depression, anxiety, and stress were significant covariates ($ps < .001$), however they did not change the relationship between negative appraisals and PTSD symptoms. Further, age was not a significant covariate ($p = .114$). Analyses were repeated with the self, world, and self-blame subscales of the PTCI as moderator variables, which yielded similar, albeit weaker patterns of effects (see Supplementary materials).

4. Discussion

The results of the present study suggest that both negative appraisals and fear extinction learning are associated with elevated PTSD symptoms. This study was the first to our knowledge to examine whether negative appraisals and fear extinction learning interact to influence PTSD symptoms. Moderation analyses revealed that negative appraisals did not moderate the relationship between fear extinction and PTSD symptoms, suggesting that fear extinction and negative appraisals do not interact in PTSD. These findings suggest that negative

appraisals and fear extinction play independent roles in PTSD symptoms.

Ehlers and Clark (2000) proposed that a key mechanism underlying the development of PTSD is excessive negative appraisals of the trauma and its sequelae (also see Ehlers et al., 2003). Using the PTCI as an index of trauma-related negative appraisals, our results confirm this prediction by finding that the PTCI total has a significant positive relationship with PTSD symptoms. This supports a wealth of prior studies indicating the importance of negative appraisals in PTSD (e.g., Dunmore et al., 1999, 2001; Ehlers et al., 1998). Analyses with the PTCI self, world, and self-blame subscales showed similar patterns with the PTCI total score (see supplementary materials). Participants in the current study experienced a range of interpersonal and disaster-related traumas, with many participants experiencing both. Future studies examining specific trauma populations may shed light on the contributions of individual trauma-types on negative appraisals and PTSD symptoms.

There is considerable evidence of PTSD-related impairments in fear extinction learning (Norrholm et al., 2011; Orr et al., 2000; Peri et al., 2000; Zuj et al., 2016a), and the findings of the current study provide some support for this notion. Specifically, we found that the PTSD group displayed a slower decrease in overall SCR amplitude during the early extinction phase, which is concordant with the idea of fear load (Norrholm et al., 2015). That is, PTSD is associated with greater expression of fear during the early stages of extinction learning, relative to trauma-exposed controls. Although the PTSD group showed significantly greater SCR amplitude during early extinction, the use of a 100% reinforcement schedule may have resulted in a small effect on extinction learning, with PTSD-related reductions in SCR amplitude appearing slower by a single trial, rather than showing a sustained conditioned fear response throughout the extinction phases. Further, we found that the PTSD and TC groups were both showing elevated SCR amplitude to the safety cue (CS-) in the early extinction phase. While the CS+ still produced significantly larger SCR amplitude than the CS-, greater responding to the CS- was not expected. This likely represents fear generalization at the onset of the extinction phases, where participants may be anticipating new US contingencies. Greater predictability of the US may also explain the trial main effect found during acquisition, where SCR amplitude gradually became smaller across the experimental phase as participants habituated to the task. Future investigations using a less predictable US reinforcement schedule may shed light on these issues (e.g., 60–62.5% reinforcement; Milad et al., 2009; Pace-Schott et al., 2013). Studies failing to find group differences in extinction learning have found PTSD to be associated with impaired recall of fear extinction learning 24 h later, compared to controls (e.g., Milad et al., 2008; Milad et al., 2009). Future research should employ 2-day extinction learning and recall designs to assess the interactive effects of negative appraisals and fear extinction on the recall of extinction learning. Importantly, based on the small effect of the current study, replications of these findings are needed to make robust conclusions.

Another important consideration when interpreting the current finding is that neutral CSs were used that had no relationship with the traumatic experiences of participants. This may account for, in part, the absence of a relationship between SCR extinction and negative appraisals. Future research employing fear conditioning and extinction paradigms with trauma-relevant stimuli (such as trauma films, as used by Pile et al., 2015) may show a relationship with negative trauma-related appraisals in a model of PTSD symptoms. Further, to ensure that the relationship between negative appraisals and PTSD symptoms was not being exaggerated by an underlying trait (i.e., negative affectivity), moderation analyses were repeated with depression, anxiety, and stress scores included as covariates. These analyses revealed no changes to the relationship between negative appraisals and PTSD symptoms, suggesting that this effect is not inflated by a negative affectivity bias.

The findings of the present study have potential implications for

treatment strategies. Previous research has found comparable treatment gains between exposure therapy alone, cognitive restructuring alone, or a combination of the two (Marks et al., 1998; Tarrier et al., 1999). Bryant et al. (2003), however, found that participants who underwent imaginal exposure combined with cognitive restructuring showed a greater reduction of PTSD symptoms than imaginal exposure alone. The findings of the current study provide preliminary evidence that both cognitive therapy for negative appraisals and exposure therapy for fear extinction are important elements to be included in treatment, as both have independent effects on PTSD symptoms (e.g., Bryant et al., 2003).

The cognitive model further hypothesizes that persistent PTSD is caused by fragmented and poorly contextualized trauma memories, and dysfunctional behavioral and cognitive strategies aimed at reducing feelings of threat, yet further exaggerating the problem (Ehlers et al., 2003). A limitation of the current study is that we were unable to examine these aspects of the model, and future research would benefit from investigating the role of extinction in the elaboration of trauma memories, and the nature of intrusive memories in PTSD. Specifically, the basolateral amygdala plays an important role in the expression and inhibition of fear (Milad and Quirk, 2012), and this neural substrate is also involved in emotional memory consolidation (e.g., Roozendaal et al., 2009; Roozendaal et al., 2008). This suggests a neurobiological basis for impaired extinction memory as a mechanism in the intrusive re-experiencing symptoms of PTSD. As fear extinction recall impairments are a seemingly reliable finding (e.g., Milad et al., 2008; Milad et al., 2009), specifically assessing the memory for fear extinction may show stronger convergences between extinction and cognitive models of PTSD.

The present study had some limitations, including a relatively modest clinical sample, the use of a cross-sectional design, and the lack of investigation of fear extinction recall. To further delineate the role of these potential mechanisms, future research is needed that employs longitudinal prospective designs to test the independent and interactive effects of negative appraisals and fear extinction learning (and recall of fear extinction learning) on PTSD symptom development over time. Employing a partial reinforcement schedule would test whether these relationships strengthen under conditions of less predictable threat, which may reduce the speed of extinction learning and enhance sensitivity of this measure. Further, there was a brief rest period between the early and late extinction phases, which likely explains the uptick in SCR amplitude from the last trial of early extinction to the first trial of late extinction (see Fig. 1). As there is some uncertainty regarding the temporal difference between acquisition and extinction phases before spontaneous recovery might occur (Norrholm et al., 2008), it is possible that this is a form of short-term spontaneous recovery. Future research would benefit from using a continuous fear extinction phase, which could be split into early and late trials for analysis purposes. Participants in the PTSD group were, on average, older than participants in the control group. Age showed no significant correlation with fear extinction learning, and had no effect on the moderation analysis when included as a covariate. Nevertheless, age is an important factor that should be considered in future research of biological fear processes.

In conclusion, as far as we are aware this is the first study to examine the interactive effects of negative, trauma-related appraisals and impaired fear extinction learning on PTSD symptomatology. We found evidence for an independent effect of both negative appraisals and fear extinction learning on PTSD symptoms. This supports the two key proposed mechanisms in a prevailing cognitive model of PTSD (Ehlers and Clark, 2000), but we did not find an interaction between cognitions and fear extinction learning. This highlights that there are both cognitive and biological processes involved in PTSD, and both may need to be addressed in treatment.

Role of the funding source

This project was supported by an NHMRC Project grant (APP1050848) and an NHMRC Program grant (APP1073041). Daniel Zuj was supported by an Australian Postgraduate Award from the Australian Government for the duration of this research.

Acknowledgements

We wish to thank Pippa Cushing, Amy Swindon, and Latifa Clark-Walters for assistance with data collection.

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.jad.2017.04.016>.

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