The Effect of Catastrophic Cognitions on Fear Acquisition and Extinction in Posttraumatic Stress Disorder

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I declare that this report is my own original work and that contributions of others have been duly acknowledged.

Kate Gray

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Abstract

This study examined the potential role of catastrophic cognitions in mediating threat expectancy during fear conditioning and extinction in Posttraumatic Stress Disorder (PTSD). It was hypothesised that participants with PTSD would display heightened catastrophic thinking and greater threat expectancy during fear extinction; the potential for catastrophic cognitions to mediate the relationship between PTSD symptom severity and threat expectancy during fear extinction was also assessed. Fifty-nine participants (21 PTSD, 19 TEC, and 19 NTEC) completed measures of catastrophic thinking (CCQ-M and PTSI) and the differential fear conditioning and extinction paradigm. The PTSD group demonstrated significantly greater trauma-specific catastrophic thinking than both control groups, but group differences in more generalised catastrophic cognition were non-significant. The PTSD group also exhibited more rapid fear acquisition and impaired fear extinction. Trauma-related catastrophic thinking did not mediate the relationship between PTSD symptoms and threat expectancy in the early extinction phase of the fear conditioning paradigm.

Most people experience *trauma* at some point in their lives, but the development of *Posttraumatic Stress Disorder* (PTSD) in response to trauma is relatively rare. Trauma is the emotional reaction to an extremely disturbing or stressful event such as an accident, interpersonal violence, terminal diagnosis, or natural disaster. The American Psychological Association (APA) describes trauma as resulting from "exposure to actual or threatened death, serious injury, or sexual violence" (Criterion A, APA, 2013, p. 271). Survivors respond differently to traumatic events, but in most cases these symptoms subside over the first week. However, for a minority, symptoms persist beyond one month or have a delayed expression, and the psychopathological response to trauma may be clinically identified as PTSD (APA, 2013). The projected lifetime risk for PTSD is 6.4% in Australia (Australian Bureau of Statistics, 2007), and individuals with PTSD are 80% more likely to meet diagnostic criteria for another mental disorder (APA, 2013). The Diagnostic and Statistical Manual of Mental Disorders (DSM) Fourth Edition Revised (DSM-IV-TR; APA, 2000) specifies that a cluster of re-experiencing, avoidance, and hyperarousal symptoms be present for diagnosis, and the Fifth Edition (DSM-5; APA, 2013) also indicates that negative changes in mood and cognition are experienced. However, the diagnosis of PTSD offers little insight into the mechanisms underlying its development and persistence. Two key models have emerged within the literature to explain why some people develop PTSD: a *biological model* which suggests PTSD evolves from impaired extinction of conditioned fear (Pitman et al., 2012); and a *cognitive model* which proposes that PTSD arises due to maladaptive appraisals and disturbed memory coding (Ehlers & Clark, 2000). Both models are well supported by empirical studies (for e.g., Bryant

& Guthrie, 2007; Lommen, Engelhard, Sijbrandij, van den Hout, & Hermens, 2013), but research investigating how the biological and cognitive models of PTSD overlap is scarce. As threat expectancy learning is critical for the acquisition and extinction of fear (Lovibond, Mitchell, Minard, Brady, & Menzies, 2009), the current study explores whether fear extinction learning is mediated by cognitive processes.

Cognitive Model of PTSD

Cognitive Schema Theory (Beck; Foa & Rothbaum). Beck expanded on Piaget's description of schemata as cognitive structures (Beck, 1967; Piaget, 1948) by considering their role in information processing. Schemata have since been described as core beliefs or stable cognitive patterns that form the basis for how an individual attends, interprets, codes, stores and retrieves an experience (Beck, Rush, Shaw, & Emery, 1979; Eysenck, 1992). Everybody develops schemata about themselves, others and the world and these affect emotional and behavioural responses. Individuals with catastrophic schemata would more readily interpret their external and internal environment in a way that is congruent with such schemata, thus perceiving threat even when faced with contradictory, ambiguous or neutral stimuli (Clark & Beck, 2010; Eysenck, 1992; Padesky, 1994). Maladaptive schemata thus play a central role in the development and maintenance of disorders such as PTSD (Padesky, 1994).

Foa and Rothbaum (1988) proposed that dysfunctional self schema ("I am totally incompetent") and dysfunctional world schema ("the world is completely dangerous") mediate the development of PTSD. They suggest that these schemata can emerge if the trauma either activates similar rigid prior beliefs about the world being dangerous and one being incompetent, or that the trauma violates a rigid belief

that the world is safe and one is capable so that the traumatic experience cannot be interpreted and assimilated as a unique experience. Foa, Ehlers, Clark, Tolin and Orsillo (1999) developed the Posttraumatic Cognitions Inventory (PTCI) to measure these negative trauma-related cognitions about the self, self-blame and the world. Bryant and Guthrie (2005) found that only pre-trauma maladaptive cognitions about the self predicted posttraumatic stress. Similarly, Moser, Hajcak, Simons and Foa (2007) only found negative cognitions about the self to be related to PTSD symptom severity in their study of trauma-exposed college students.

Cognitive Appraisal Model (Ehlers and Clark). Ehlers and Clark (2000) also conceptualised PTSD from a cognitive perspective, proposing that the development and maintenance of the disorder are dependent on a sense of persistent current threat caused by individual differences in two key cognitive processes: the first being *the appraisal of the trauma and/or its sequelae*. If the individual negatively appraises the trauma, their own response, or the reaction of others, then they are more likely to have a strong sense of current threat. Re-experiencing the trauma (through intrusive thoughts, nightmares or flashbacks) consolidates or amplifies this sense of threat (Dekel, Peleg, & Solomon, 2013), and avoiding trauma-related stimuli prevents individuals from realising the threat is no longer present.

The second key cognitive process implicated in Ehlers and Clark's (2000) model of PTSD relates to *how the trauma memory is coded and linked to other autobiographical memories*. Ehlers and Clark (2000) suggested that an inability to form a self-referential perspective when encoding the trauma memory means that its integration into biographical memory is inhibited, and intentionally recalling the event is made difficult. This lack of self-referential perspective may be due to the

extreme nature of the trauma and its violation of previously held beliefs about safety, predictability and controllability in relation to oneself, others, and the world in general (Foa, Steketee, & Rothbaum, 1989). The violation of these beliefs may be responsible for further promoting a sense of current threat.

PTSD sufferers tend to have fragmented memory of their trauma, with strong sensory impressions that are easily triggered, but poor conceptual processing of context and meaning (Ehlers & Clark, 2000). It is suggested that this fragmentation may be due to dissociation during trauma (van der Kolk & Fisler, 1995). The fragmented and highly arousing nature of these trauma memories is thought to reduce an individual's ability to contextualise flashbacks as autobiographical memories; instead they are vividly experienced as if occurring in the present (Brewin, 2007; Ehlers & Clark, 2000; Siegel, 1995).

Negative Appraisals and PTSD

Empirical research indicates that negative or catastrophic appraisals (before, during or in response to trauma and its sequelae) predict the development and maintenance of posttraumatic stress (Bryant & Guthrie, 2005; Dunmore, Clark, & Ehlers, 1997; Halligan, Michael, Clark, & Ehlers, 2003). For example, Bryant and Guthrie's (2005) study of 82 trainee fire fighters found that greater pre-trauma negative appraisals of oneself were associated with increased posttraumatic stress at follow-up. Conversely, a prospective study of police officers found higher evaluations of self-worth pre-trauma predicted reduced PTSD (Yuan et al., 2011). This suggests that pre-trauma negative cognitions about oneself being inadequate or hopeless if faced with a traumatic experience predict greater posttraumatic stress.

Longitudinal and cross-sectional studies have also suggested that negative appraisal of a traumatic experience predicts increased risk for developing PTSD (Dunmore, Clark, Ehlers, 2001; Ehlers Mayou, & Bryant, 1998; Ehring, Ehlers, & Glucksman, 2008; Halligan et al., 2003). Halligan et al. (2003) conducted two studies of assault victims, one cross-sectional (n = 81) and the other prospective longitudinal (n = 73), and found that negative appraisals of trauma memories (memory disorganisation and intrusive memories) predicted severity of PTSD symptoms and maintenance of the disorder. Dunmore et al. (2001) study of 57 assault victims established that negative beliefs about self and world were significant predictors of PTSD severity at 4 and 6-9 months after the assault.

Whilst this research is consistent with a cognitive model of PTSD, it only considers catastrophic appraisals anchored to trauma or a negative event experienced, thus gives no indication whether trait catastrophic thinking is a vulnerability factor in PTSD unrelated to the experience of trauma. In fact, Dekel et al. (2013) 17-year longitudinal study of Israeli combat veterans used a generalised measure of cognitions (the World Assumption Scale; WAS) to assess three core cognitions: benevolence of the world, the meaningfulness of the world (i.e., assumptions about control and justice) and self-worth or self-control. They found that PTSD symptoms predicted generalised negative cognitions about the self and the world, rather than vice versa. This suggests that whilst trauma-specific catastrophic thinking might predict PTSD symptoms, this is not the case for generalised negative cognition. These findings imply that generalised catastrophic thinking develops in response to PTSD, and is therefore not a vulnerability factor in PTSD development.

Biological Model of PTSD

An alternative model of PTSD is a biological model, based on Pavlov's (1927) classical conditioning theory. It proposes that PTSD develops from a *fear conditioning process* in which previously neutral stimuli present at the time of trauma become associated with intense fear and arousal. These then act as conditioned stimuli to elicit ongoing conditioned fear responses. For example, a song playing in the background during a traumatic event heard at a later time might prompt intense fear as if reliving the trauma, unless the individual learns that the music is of no threat. Avoidance of these conditioned stimuli inhibits *extinction learning* and maintains PTSD symptoms (Pitman et al., 2012). In this model, trauma memory is over-consolidated due to the release of stress hormones (noradrenaline and cortisol) during the traumatic event, leading to intrusive memories (Pitman et al., 2012).

Whilst fear conditioning is thought to be involved in the acquisition of PTSD, the biological model emphasises that impaired fear extinction learning is involved in its persistence. Fear conditioning alone cannot explain why some people develop PTSD and others do not when faced with the same trauma or one of similar severity (Orr et al., 2000); nor can it explain why about 50% of individuals with PTSD make a complete recovery within 3 months, yet others remain symptomatic for years (APA, 2013). Strong evidence has emerged suggesting that impaired fear extinction interferes with the gradual reduction in PTSD symptoms (as cited in Pitman et al., 2012).

Shin and Liberzon (2010) reviewed research on the neurocircuitry of anxiety disorders and neuroimaging studies of PTSD patients and found: a hyporesponsive

ventromedial prefrontal region (thought to regulate amygdala activity and thus fear response; for e.g., Bremner et al., 1999); a hyperresponsive amygdala (suggesting an increased fear response; for e.g., Chung et al., 2006); abnormal hippocampal activation and volumes (perhaps reflecting issues with dissociation, and memory coding and retrieval; for e.g., Bossini et al., 2008); and a hyperresponsive dorsal anterior cingulate cortex (associated with exaggerated fear learning; for e.g., Bremner et al., 2005). In a fear and extinction paradigm, positron emission tomographic imaging indicated that PTSD groups have increased amygdala activation during fear acquisition and reduced anterior cingulate functioning during extinction compared to controls, thus implicating these brain regions in PTSD (Bremner et al., 2005). Using fMRI, Milad et al. (2009) also found that hyperactivity in the dorsal anterior singulate and hypoactivity in the ventromedial prefrontal cortex (vmPFC) were related to impaired extinction in PTSD. Animal studies demonstrate that mechanisms of extinction learning are consistent across species in implicating the vmPFC in fear extinction (Milad and Quirk, 2002; Quirk & Mueller, 2008). Together these findings suggest that reduced activity in vmPFC reflects impaired extinction learning in PTSD, and that activation of the prefrontal cortex is required to extinguish fear.

Differential fear conditioning and extinction paradigm. The classical fear conditioning and extinction paradigm has been used extensively to investigate emotional learning and memory implicated in the development, symptomology and maintenance of anxiety disorders including PTSD, (Graham & Milad, 2011; Guthrie & Bryant, 2006; Sijbrandij, Engelhard, Lommen, Leer, & Baas, 2013). The acquisition or conditioning phase of the paradigm involves an aversive

unconditioned stimulus (US) such as a mild electric shock being repeatedly paired with an originally neutral stimulus (CS^+) so that it evokes a subsequent conditioned fear response when presented alone. The extinction phase involves the CS^+ and CS^- being randomly presented without the US. Another neutral stimulus (CS^-) is randomly presented during each phase, but never paired with the US. This allows the differential responses to the CS^+ and CS^- in each phase to be clearly observed (see Figure 1). Skin conductance response (SCR) or fear potentiated startle are typically monitored as measures of fear learning, and threat expectancy ratings are recorded prior to each trial (Lovibond et al., 2009). It is expected that individuals with anxiety disorders or PTSD would be faster fear conditioning to the CS^+, but slower to extinguish that fear.

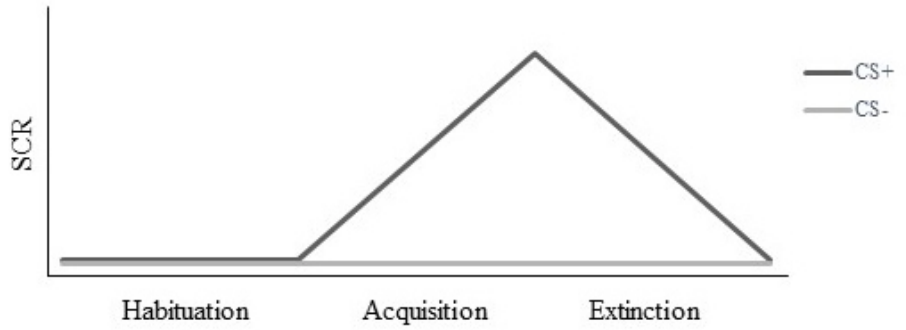

Figure 1. Expected SCR for the CS^+ and CS^- in the Habituation, Acquisition, and Extinction Phases of the Fear Conditioning and Extinction Paradigm.

Fear Acquisition and Extinction in PTSD

Whilst some studies report increased fear conditioning in PTSD (reflected by increased SCR to the CS^+ during the acquisition phase), not all have. The more robust finding is that fear extinction is impaired in PTSD, indexed by higher SCR

during the extinction phase. PTSD research using the differential fear-extinction paradigm has suggested that dysfunctional fear learning and extinction is: a vulnerability factor in the development of posttraumatic stress pre-trauma (Guthrie & Bryant, 2006; Lommen et al., 2013); a feature of the disorder (Peri, Ben-Shakhar, Shalev, 1997; Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007); and, a maintaining factor in PTSD (Milad et al., 2008; Sijbrandij et al., 2013; Orr et al., 2000; Peri, Ben-Shakhar, Orr, & Shalev, 2000). Relative to controls, PTSD groups were found to have higher skin conductance responses following the acquisition of fear conditioning (Orr et al., 2000) and reduced fear extinction (Peri et al., 2000).

Longitudinal and prospective studies have identified reduced fear extinction as a key factor in the development, severity and maintenance of PTSD symptoms. A study of 249 Dutch soldiers deployed to Afghanistan for 4 months (Lommen et al., 2013) found that reduced fear extinction prior to deployment (pre-trauma) was found to predict PTSD symptom severity on return (post-trauma), even after controlling for pre-trauma symptoms and risk factors. A longitudinal study of 144 of the same trauma–exposed soldiers found that impaired fear inhibition also predicted the maintenance of PTSD symptoms (Sijbrandij et al., 2013). Similar results were found in a study of 70 firefighters assessed during cadet training – pre-trauma reduced extinction predicted 31% of the variance in posttraumatic severity when assessed within 24 months of becoming active firefighters (post-trauma; Guthrie & Bryant, 2006).

Strong empirical evidence suggesting extinction learning is a predictive and maintaining factor in PTSD informs treatment methods that facilitate extinction learning. Exposure therapy mirrors this process by repeatedly exposing the

individual to the feared stimulus (though often with a graded approach), so that the individual learns that there is no aversive consequence thus reducing their fear response (Graham & Milad, 2011). Exposure therapy has proven effective in the treatment for anxiety disorders, including PTSD (Felmingham et al., 2007; Hofmann & Smits, 2008).

Cognitive Factors, Fear Conditioning and Extinction, and PTSD

Fear conditioning has traditionally been seen as a low-level bottom-up reflexive or biological process independent of awareness, but recently debate has arisen suggesting that it also depends on high-level top-down conscious cognitive processes (Chater, 2008; Lovibond et al., 2009; Lovibond & Shanks, 2002; Mitchell, De Houwer, & Lovibond, 2009). Lovibond and Shanks (2002) suggest that threat expectancy ratings in the fear conditioning and extinction paradigm may themselves reflect both unconscious biological and conscious cognitive processes. Lovibond and Shanks (2002) research review identified typical congruence between measures of threat expectancy, skin conductance or fear potentiated startle, and conscious awareness of the contingency presented in the fear-extinction paradigm, suggesting all three measures result from the same learning mechanism. Purkis and Lip (2001) used all 3 measures in a differential conditioning paradigm and found that there was no evidence of conditioned learning in the absence of cognitive contingency awareness (as assessed using an expectancy dial), confirming that these factors are important in extinguishing fear. Just as heightened threat expectancy is thought to characterise PTSD (Kimble, Batterink, Marks, Ross, & Fleming, 2012), catastrophic thinking, hypervigilance and exaggerated strartle responses are criteria used for PTSD diagnosis (APA, 2013), thus individuals with PTSD may demonstrate

heightened levels of each leading into each phase of the differential fear conditioning and extinction paradigm.

Whilst cogent biological and cognitive models of PTSD have been developed, and there is evidence supporting both models, very few studies have examined how cognitive and biological models may intersect. Recent research has revealed that cognitive factors can influence fear conditioning and extinction. Gazendam and Kindt's (2012) study of the effect of worrying on fear conditioning found that after initial fear acquisition, individuals randomly allocated to a "worry" condition displayed enhanced fear responses and impaired extinction compared to controls. In Lovibond, Mitchell, Minard, Brady, and Menzies' (2009) study participants in one group were given the opportunity to make an avoidance response in the extinction phase; they found that those who did so maintained higher shock expectancy ratings and strong SCRs compared to controls. Both studies suggest that a cognitive mechanism may account for conditioned responding and the failure to extinguish fear.

The Present Study

Given the evidence for both biological and cognitive models of PTSD, the lack of research integrating them, and the increasing debate regarding the role of cognitive processes in fear conditioning and extinction, the current study aims to use the differential classical conditioning paradigm to examine the influence of catastrophic thinking on fear conditioning and extinction in PTSD, as measured by threat expectancy. It is hypothesised that:

1. PTSD participants will have greater catastrophic thinking than the TEC and NTEC groups.

2. PTSD participants will display impaired fear extinction learning compared to the TEC and NTEC groups, as indicated by greater threat expectancy ratings in fear extinction.
3. Catastrophic thinking mediates the relationship between PTSD symptoms and fear extinction, as measured by threat expectancy in the early extinction phase.

Method

Participants

The sample comprised 59 participants (37 women, 22 men) aged between 17 and 63 (M = 27.66 years, SD = 12.96 years). Six individuals were excluded from the study as they did not complete the differential fear-extinction paradigm. Other exclusion criteria included: a history of substance dependence (or an AUDIT score of at least 16; Hays, Merz, & Nicholas, 1995; see Appendix A5), previous PTSD, neurological disorders or stroke, or traumatic brain injury; current psychosis or suicidal ideation; and, being medically unwell. Participants were recruited from first year psychology students who receive course credit for their participation. Others were recruited from the community via email advertising and received $50 for their time. Participants were categorised into three groups according to whether they were exposed to a Criterion A trauma (APA, 2000) using the Trauma Exposure Questionnaire (TEQ; Vrana & Lauterbach, 1994; see Appendix A1), and whether they had gone on to develop PTSD using to the PTSD Checklist – Civilian Version (PCL-C; Foa, Cashman, Jaycox, & Perry, 1997; see Appendix A2). To maximise sample sizes, subclinical PTSD participants with scores of at least 40 on the PCL-C

were included in the PTSD group. According to the National Centre for PTSD (2014), these participants met the PTSD cut-off score of 30 for a non-clinical setting; the cut-off is 50 in clinical settings.

- Group 1: Non-trauma exposed control (NTEC) were 19 participants (13 women, 6 men; age M = 22.84 years, SD = 2.78).
- Group 2: Trauma-exposed controls who did not develop PTSD (TEC group) were 19 participants (12 women, 7 men; age M = 25.05 years, SD = 2.78).
- Group 3: Trauma-exposed controls who did develop PTSD (PTSD group according to our classification above) were 21 participants (12 women, 9 men; age M = 34.38 years, SD = 2.64).

Design

The study assessed group differences using a between-groups univariate design for each of the demographic, clinical, and catastrophic cognition measures. A 3 (Group: NTE, TE, PTSD) x 2 (Stimuli: CS^+, CS^-) x 5 (Trial: 1, 2, 3, 4, 5) mixed factorial design was applied for threat expectancy ratings in the acquisition, early extinction, and late extinction phases in the fear conditioning and extinction task. The same design was applied in the habituation phase, only with 4 rather than 5 trials.

A mediation analysis was used to assess whether catastrophic thinking mediates the relationship between level of posttraumatic stress symptoms and threat expectancy during fear extinction.

Materials

Traumatic Events Questionnaire (TEQ; Vrana & Lauterbach, 1994; see Appendix A1). This is an 11-item questionnaire that assesses Criterion A (APA,

2000) trauma exposure, such as interpersonal assault, life-threatening accident, natural disasters, or combat experience. Questions are dichotomous (yes/no) in nature (for e.g., "Have you ever witnessed someone being badly injured or killed?"). The TEQ is a brief screener that identifies lifetime exposure to a traumatic event.

PTSD CheckList – Civilian Version (PCL-C; Weathers, Huska, & Keane, 1991; see Appendix A2). This is a 17-item standardised self-report measure rating PTSD symptomatology (based on DSM-IV-TR criteria; APA, 2000) over the last month. It uses a 5-point Likert scale ranging from 1 ("not at all") to 5 ("extremely"). The PCL-C provides an ordinal scale of PTSD symptom severity, allowing for PTSD diagnosis and has demonstrated good reliability and internal consistency (Foa et al., 1997); Cronbach's alpha is .92.

Depression, Anxiety and Stress Scale – 21 (DASS-21; Lovibond & Lovibond, 1995; see Appendix A3). This is a 21-item self-report measure of distress in the past week in relation to three subscales: depression (for e.g., "I felt that I had nothing to look forward to"), anxiety (for e.g., "I was aware of dryness in my mouth"), and stress (for e.g., "I found it difficult to relax"). It uses a 4-point Likert scale ranging from 0 ("did not apply to me at all") to 3 ("applied to me very much, or most of the time"). Total subscale scores are doubled and higher scores reflect greater subscale severity (scores above 21, 15 and 26 for depression, anxiety and stress subscales respectively are considered severe). The DASS-21 is a well-validated measure that has demonstrated good reliability and internal consistency (Henry & Crawford, 2005). Cronbach's alpha for each subscale is .91, .84, and .90 (Lovibond & Lovibond, 1995), respectively.

Medical questionnaire (see Appendix A4). This questionnaire is used to collect demographic information, and to screen for factors that might give rise to risk or need to exclude individuals from the study (for e.g., medical history, medication or substance use). This screener was formulated for use in other studies using the same participants, thus not all responses applied to this study.

The Alcohol Use Disorders Identification Test (AUDIT; World Health Organisation, 2001; see Appendix A5). The AUDIT is a 10-item self-report questionnaire designed to screen for potentially harmful alcohol consumption patterns. The AUDIT uses a 5-point Likert scale to measure the frequency of drinking or problems with drinking. A score of 16 or more is considered to be predict high-risk or harmful drinking. The AUDIT is a well-validated measure that has demonstrated good reliability and internal consistency (Cronbach's α = .83; Hays et al., 1995).

Differential fear conditioning and extinction task. This paradigm was run on a laptop computer using Inquisit 3.0.6.0 (2011) software, and threat expectancy data was collected as part of this task. A Powerlab 16/35 Recording Bare Electrode (MLADDB30) attached to the first dorsal interosseous muscle of the dominant hand generated a 500 millisecond mild electrical shock (the unconditioned stimulus; US) from PowerLAb 16/35 Stimulus Isolator (FE180).

Catastrophic Cognitions Questionnaire – Modified (CCQ-M; Khawaja, Oei, & Baglioni, 1994; see Appendix A6). This questionnaire is a 21-item self-report trait measure of negative appraisals in relation to various experiences. Its 3 subscales relate to different types of catastrophic cognitions: emotional (extent to which danger is related to emotional responses, for e.g., "being agitated"), physical (extent to

which physical hazards are interpreted as dangerous, for e.g., "being injured"), and mental (extent to which social anxieties are interpreted as dangerous, for e.g., "unable to think rationally"). It uses a 5-point Likert scale ranging from 1 (not at all dangerous) to 5 (extremely dangerous) to rate the participant's sense of danger. Scoring involves totalling the scores for each subscale and the total score gives a trait measure of catastrophic thinking. The subscales showed high internal consistency and good test-retest reliability. Cronbach's alpha for the emotional, physical and mental subscales was .83, .85, and .89 respectively (Khawaja et al., 1994).

The Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999; see Appendix A7). This is a 36-item self-report measure of negative appraisals in relation to a traumatic experience (those who had not experienced trauma were asked to relate the items to the most stressful experience they could recall). The PTCI uses a 7-point Likert scale ranging from 1 ("totally disagree") to 7 ("totally agree"). The total score is a sum of all scores and the subscale scores are the sum of the relevant scores divided by the number of items; scoring involves exclusion of 3 items. The 3 subscales are Negative Cognitions about Self (about one's capabilities and response to a negative event), Negative Cognitions the World (about world safety in response to a negative event), and Self Blame (for the harm caused by a negative event). All subscales showed excellent internal consistency and good test-retest reliability and Cronbach's alpha for each was .97, .88, and .89 respectively; along with .97 for the scale total score (Foa et al. 1999).

Procedure

Ethics approval was obtained from the University of Tasmania's Social Sciences Human Research Ethics Committee (see Appendix B). Study information was provided and informed consent was obtained for the study (see Appendix C). Medical, AUDIT, DASS, TEQ and PCL-C questionnaires were completed. Participants were then classified into PTSD, TEC or NTEC groups according to their TEQ and PCL-C results. The measures of catastrophic thinking (CCQ-M and PTCI) were administered after the differential fear conditioning and extinction paradigm. See Appendix A for questionnaires used. Some additional questionnaires were administered for other studies using the same participants.

In preparation for the fear conditioning and extinction paradigm, participants were asked to select a level of the US that they found "uncomfortable, but not painful". The electrical shock was administered to the 2nd and 3rd fingers of their non-dominant hand starting at 2mA and increased incrementally by .5mA until discomfort was reported.

The fear conditioning and extinction paradigm was then presented on a computer screen (see Figure 2) and comprised habituation, acquisition, early extinction and late extinction phases. The initial habituation phase involved 4 trials of each of the conditioned stimuli (CS): either a red or blue circle. The acquisition phase that followed involved each CS (CS^+ or CS^-) being presented 5 times, with the US administered immediately after each CS^+ presentation (100% reinforcement schedule). Finally, in the extinction phases (early and late extinction) the CS^+ and CS^- were both presented 5 times without the US. The CS^+ and CS^- colours were varied from red to blue in a randomised, counterbalanced order between participants

and groups. CS presentation was random, with the constraint that no CS occurred more that twice in succession. See Figure 1. Participants were told that they may experience a shock following the habituation stage. Each CS appeared in the centre of a white computer screen for 12 seconds, with an inter-trial interval of 12-21seconds. A saliva sample was taken between the early and late extinction phases for use in another study. Participants were required to rate threat expectancy on an 11-point Likert scale ranging from -5 (certain no shock), 0 (uncertain), to 5 (certain shock) displayed on the screen with each trial.

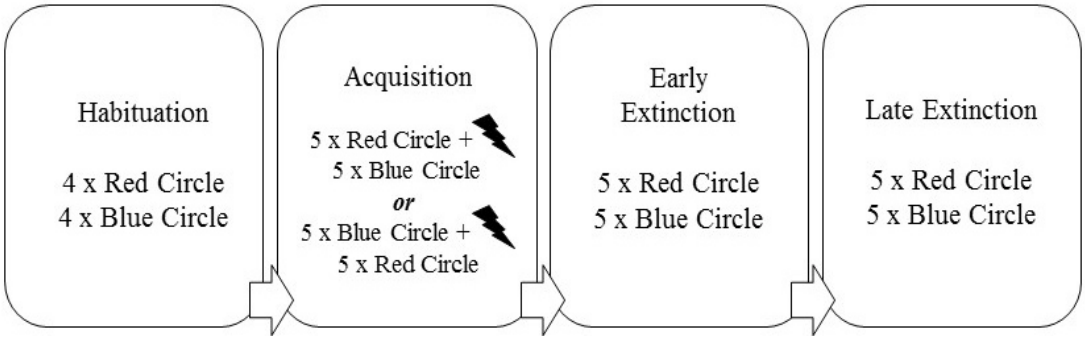

Figure 2. Trials presented within Each Phase of the Differential Fear Conditioning-Extinction Paradigm.

Analysis

The study assessed group differences for demographic and clinical measures using between-groups univariate analyses of variance (ANOVA), and 3 x 2 Chi Square test of independence for gender distribution. The Welch statistic was applied when the assumption of homogeneity of variance was not met. To assess whether the PTSD group had greater catastrophic cognitions, separate between-groups univariate ANOVA were conducted using the total and subscale scores of two catastrophic

cognition measures, the PTCI and CCQ-M. Sidak-adjusted post-hoc pairwise comparisons were used to examine differences between groups.

To assess whether the PTSD group displayed impaired fear extinction learning, threat expectancy measures were analysed using mixed factorial ANOVA and Sidak-adjusted post-hoc pairwise comparisons were employed where significant main effects and interactions were found. A 3 (Group: PTSD, trauma-exposed, no trauma) x 2 (Stimuli: CS^+, CS^-) x 4 (Trial: 1, 2, 3, 4) ANOVA was used for the habituation phase and a 3 (Group: PTSD, trauma-exposed, no trauma) x 2 (Stimuli: CS^+, CS^-) x 5 (Trial: 1, 2, 3, 4, 5) ANOVA was applied to the acquisition, early extinction and late extinction phases. Greenhouse-Geisser corrections were made when the assumption of sphericity was violated.

To assess whether catastrophic thinking mediated the relationship between PTSD symptoms and threat expectancy in extinction learning, a simple mediation analysis was conducted using Hayes' Process macro version 2.12.1 in SPSS Version 21 (Hayes, 2013). The simple mediation analysis (see Figures 3 & 4) assesses whether *X* [predictor variable: PTSD symptom severity (PCL total score)] affects *Y* [outcome variable: average threat expectancy ratings during extinction (EEAv)] indirectly through *M* [mediator variable Catastrophic Cognitions (PTCI total)] according to the following criteria being satisfied (Baron & Kenny, 1986):

1. *X* significantly predicts *Y* in the unmediated *total effect model* (see Figure 3). This model estimates $Y = i_1 + cX + e_1$ (regression coefficient *c* represents the *total effect* of *X* on *Y*, i_1 is the intercept, and e_1 is the residual).

2. *X* significantly predicts *M* in the simple mediation model (see Figure 4). This model estimates $M = i_2 + aX + e_2$ (regression coefficient *a* represents the relation of *X* on *M*, i_2 is the intercept, and e_2 is the residual).
3. M significantly predicts Y controlling for M (see Figure 4). This model estimates $Y = i_3 + c'X + bM + e_3$ (regression coefficient *c'* represents the *direct effect* of *X* to *Y* adjusted for *M,* and *b* is the effect of *M* to *Y* adjusted for *X*; i_3 is the intercept, and e_3 is the residual).
4. Baron and Kenny (1986) also specify that Y should not cause M and M should be free from errors in measurement.

Bootstrapping was used for the indirect mediation effects to reduce sample skewness and kurtosis common in smaller samples. It involves extensive resampling from the data and allows 95% confidence intervals and a measure of standard error to be calculated. Mediation is said to occur if all criteria have been satisfied and the indirect effect of *ab* (*where* $c = c' + ab$) is found to be significant as the bootstrapped 95% confidence interval does not include zero (Fritz & MacKinnon, 2007).

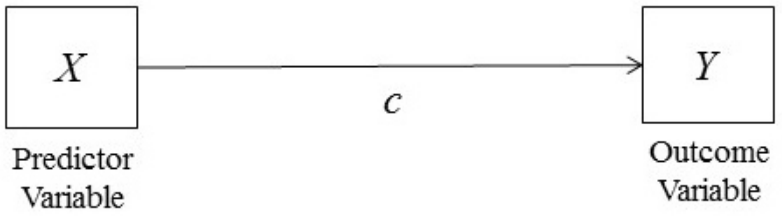

Figure 3. Total Effect Model of X on Y.

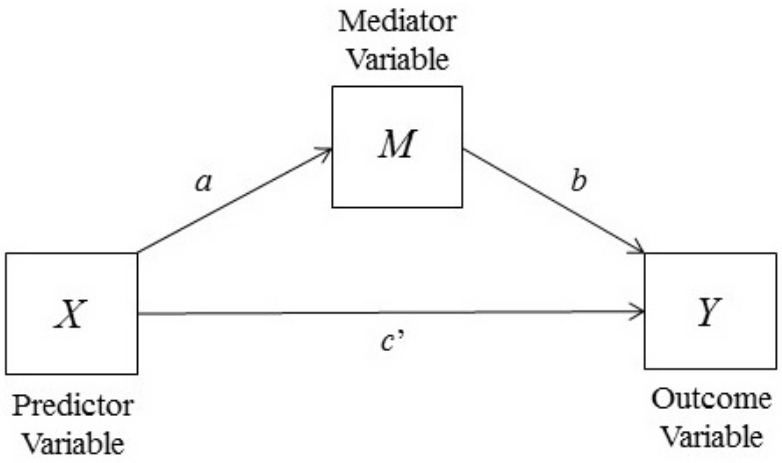

Figure 4. Simple Mediation Model.

Results

Data Preparation and Screening

There were no missing values among study completers for any measure tested. One extreme univariate outlier was observed in PTCI Self-blame and was replaced with a value equal to 3 standard deviations from the mean (Tabachnick & Fidell, 2001). Data was screened for skewness and kurtosis. A significant departure from normality was identified using the Shapiro-Wilks test for PTCI Self in the NTC and TEC groups, and for the PCL and all DASS subscales (all positively skewed distributions). Logarithmic transformations were conducted for all but the DASS and PCL (which are prone to positive skew due to the incidence of psychopathology within the population), however transformations did not affect the pattern of results or the conclusions.

Demographic and Clinical Measures

Table 1 displays the mean and standard deviation for clinical and descriptive data collected for each group (PTSD, TEC and NTEC). It includes results from univariate ANOVAs comparing the mean scores between groups on age, the PCL-C. and the subscales of the DASS, and from a 3 x 2 Chi Square test of independence for gender differences across groups. Significant group differences were found for all variables other than gender. According to Levene's test, the assumption of homogeneity of variance was not met for Age ($F(2.56) = 5.25$, $p=.008$), nor for the depression subscale of the DASS ($F(2.56) = 6.11$, $p=.004$), thus the Welch statistic was substituted in both cases.

Sidak-adjusted post-hoc pairwise comparisons (see Appendix D1) indicated a significant difference between the PTSD group and both the NTEC and TEC groups on the PCL-C (reflecting greater PTSD symptomology) and the DASS subscales, however there was no significant difference between the NTEC and TEC groups. Post-hoc comparisons of Age indicated that the PTSD group was significantly older that the NTEC and trending towards being significantly older than the TEC group. Although these results reflected the fact that older participants are more likely to have experienced trauma in their lifetime, further analyses controlled for Age as a covariate using repeated measures ANCOVA. High ratings on DASS depression are assumed in PTSD due to high comorbidity of disorders, thus further analyses will not control for DASS depression. According to the APA (2013), approximately 80% of people with PTSD have at least one comorbid psychiatric disorder, most commonly depression.

Table 1

Demographic and Clinical Measures Including Group Means (Standard Deviations), Test Statistics, Significance Levels, and Effect Sizes

Variable	PTSD	TEC	NTEC	*Test Statistic*	*p*	η_p^2
Age	34.38 (15.26)	25.05 (9.81)	22.84 (10.08)	$F = 4.11$	.024*	.16
PCL-C	51.71 (11.77)	24.84 (4.62)	20.47 (3.70)	$F = 95.43$	<.001**	.77
DASS						
- Depression	8.52 (5.58)	2.89 (3.75)	1.84 (3.20)	$F = 11.06$	<.001**	.33
- Anxiety	8.05 (3.51)	2.10 (2.08)	1.78 (2.22)	$F = 34.11$	<.001**	.55
- Stress	12.14 (4.60)	5.21 (2.8)	3.63 (4.03)	$F = 27.15$	<.001**	.49
Gender	12 F, 9 M	12 F, 7 M	13 F, 6 M	$\chi^2 = .54$	.761	

*$p < .05$. **$p < .01$. *Note.* η_p^2 = Effect size; Degrees of Freedom = 2; PCL-C = PTSD Check List Civilian Version; DASS = Depression, Anxiety and Stress Scales

Measures of Catastrophic Thinking

Table 2 displays the means, standard deviations and results from univariate ANOVAs comparing the means from each of the groups (PTSD, TEC and NTEC) for measures of catastrophic thinking and their subscales. No significant group differences were found for the Total CCQ-M, the CCQ-M subscales, or the PTCI Self-blame subscale. Significant group differences were found for the Total PTCI and its subscales PTCI World and PTCI Self.

Sidak-adjusted post-hoc pairwise comparisons (see Appendix D2) revealed a significant difference between the PTSD group and both the NTEC and TEC groups on the PTCI Total and PTCI Self subscale, but there was no significant difference between the NTEC and TEC groups. Posthoc comparisons identified that the PTSD and TEC groups had significantly higher PTCI world scores than the NTEC group,

and the PTSD group was trending towards having higher PTCI World scores than the TC group.

Table 2

Measures of Catastrophic Thinking Including Group Means (Standard Deviations), Test Statistics, Significance Levels, and Effect Sizes

Variable	PTSD	TEC	NTEC	*F*	*p*	η_p^2
CCQ Total	60.33 (11.39)	55.21 (8.88)	57.58 (12.97)	1.05	.358	.04
CCQ Mental	20.05 (5.11)	18.36 (4.98)	19.95 (6.59)	.55	.580	.02
CCQ Emotional	15.05 (4.13)	12.89 (3.49)	12.79 (4.28)	1.85	.167	.06
CCQ Physical	24.90 (4.49)	23.95 (3.95)	24.84 (4.63)	.29	.749	.01
PTCI Total	109.57 (35.34)	78.05 (28.27)	68.10 (30.66)	9.43	<.001**	.25
PTCI Self	2.97 (1.15)	2.01 (1.00)	1.82 (.94)	7.19	.002**	.20
PTCI World	4.52 (1.49)	3.54 (1.30)	2.45 (1.02)	12.79	<.001**	.31
PTCI Self-Blame	3.09 (1.53)	2.19 (1.06)	2.55 (1.47)	2.14	.128	.07

*$p < .05$. **$p < .01$. *Note.* η_p^2 = Effect size; Degrees of Freedom = 2; CCQ = Catastrophic Cognitions Questionnaire-Modified; PTCI = Posttraumatic Cognitions Inventory

Threat Expectancy in Fear Conditioning and Extinction

Habituation. A 3x2x4 repeated measures ANOVA in the habituation phase found no significant main group effect [$F(2, 56) = .97$, $p = .384$, $\eta p^2 = .03$]; a significant condition main effect [$F(1, 56) = 5.92$, $p = .018$, $\eta p^2 = .10$] which revealed that whilst participants did not expect a shock for both conditions (signified by negative values), there was less threat expectancy for the CS^- (M = -2.1, SD=.34) than for the CS^+ (M=-1.8, SD = .35); and a significant trial main effect [$F(1.77, 98.91) = 4.32$, $p = .020$, $\eta p^2 = .07$], following a Greenhouse-Geisser correction. See Figure 5.

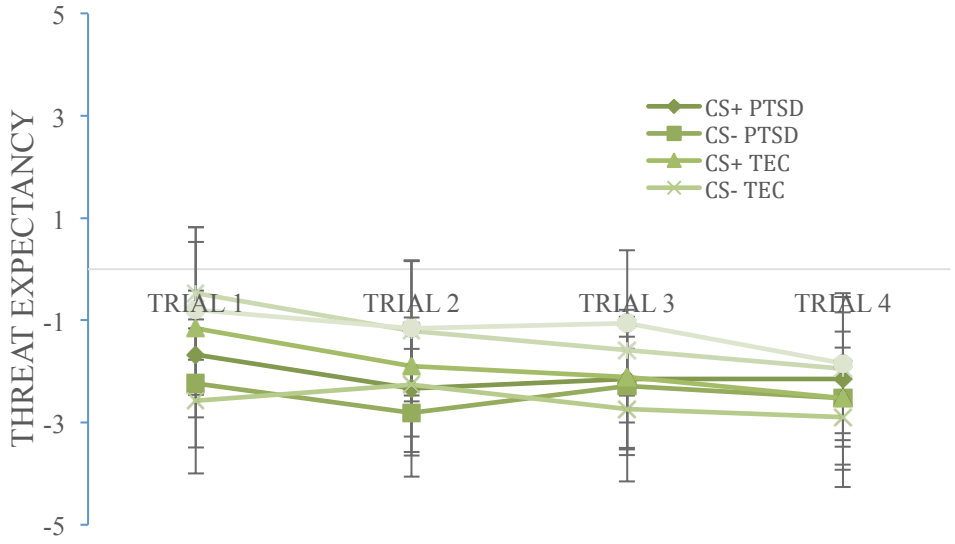

Figure 5. Threat Expectancy Ratings at Each Trial for each CS and Group in the Habituation Phase.

Sidak-adjusted post-hoc pairwise comparisons revealed that only the threat expectancy between trial 4 and trial 5 reduced significantly (see Appendix D3). A trend was identified for the condition by group interaction [$F(2, 56) = 2.74$ $p = .073$, $\eta p^2 = .09$] and post-hoc tests revealed this reduced threat expectancy to the CS^+ was present in the TEC group, but not NTEC group. The condition by trial interaction was non-significant following a Greenhouse-Geisser correction [$F(2.00, 112.20) = 7.69$, $p = .190$, $\eta p^2 = .03$]. The trial by group interaction [$F(6, 168) = .79$, $p = .577$, $\eta p^2 = .03$] and condition by trial by group [$F(6, 168) = .40$, $p = .880$, $\eta p^2 = .01$] interactions were also non-significant. Taking Age as a covariate removed all effects previously found.

Acquisition. A 3x2x5 repeated measures ANOVA in the acquisition phase found no significant main effect of group [$F(2, 56) = .07$, $p = .935$, $\eta p^2 < .01$], or of trial [$F(4, 224) = 1.85$, $p = .120$, $\eta p^2 = .01$], but there was a main effect of condition

[$F(1, 56) = 175.07$, $p < .001$, $\eta p^2 = .76$]. This indicates that threat expectancy ratings for the CS^+ were significantly greater than for the CS^- overall. See Figure 7. The condition main effect revealed that threat expectancy ratings were significantly greater for the CS^+ ($M = 2.905$, $SE = .22$) than the CS^- ($M = -2.854$, $SE = .26$). The main effect was superseded by a significant condition by trial interaction [$F(3.32, 185.74) = 56.56$, $p = < .001$, $\eta p^2 = .51$], following a Greenhouse-Geisser correction. Figure 6 reveals that the difference between threat expectancy ratings for the CS^+ and the CS^- increased across trials. Sidak-adjusted post-hoc pairwise comparisons revealed that there were significant condition effects at each trial, but the difference between the CS^+ and the CS^- was smaller at trial 1 than at later trials (see Figure 7 & Appendix D4). The condition by group [$F(2, 56) = .23$, $p = .794$, $\eta p^2 = .01$], trial by group [$F(8, 224) = .60$, $p = .775$, $\eta p^2 = .02$], and condition by trial by group [$F(8, 224) = 1.54$, $p = .144$, $\eta p^2 = .05$] interactions were non-significant. Taking Age as a covariate did not alter the pattern of findings.

Figure 6. Threat Expectancy Ratings at each Trial for CS^+ and CS^- during the Acquisition Phase.

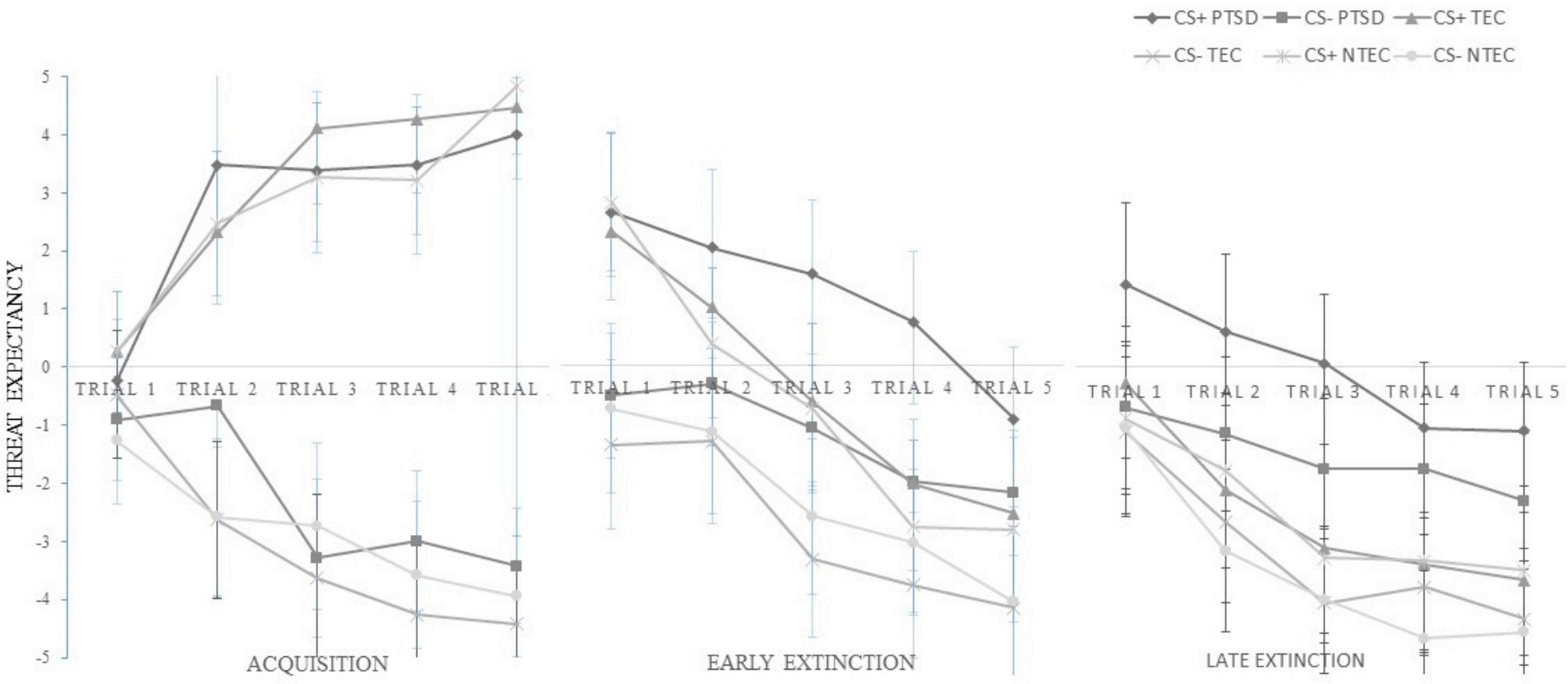

Figure 7. Threat Expectancy Ratings at each Trial for each CS and Group in the Acquisition, Early Extinction and Late Extinction Phases.

Early Extinction. A 3x2x5 repeated measures ANOVA in the early extinction phase revealed a significant main effect of group [$F(2, 56) = 5.44$, $p = .007$, $\eta p^2 = .16$]. See Figure 8. Sidak-adjusted post-hoc pairwise comparisons revealed that the PTSD group [$M = .005$, $SE = .35$) had significantly greater threat expectancy ratings than the TEC group [$M = -1.474$, $SE = .36$, $p = .015$, 95% CI (.24, 2.72)] and the NTEC group [$M = -1.368$, $SE = .36$, $p = .026$, 95% CI (.13, 2.61)]. The TEC and NTEC groups did not differ significantly [$p = .996$, 95% CI (-1.38, 1.17)], and both did not expect threat in the early extinction phase (see Figures 7 & 8).

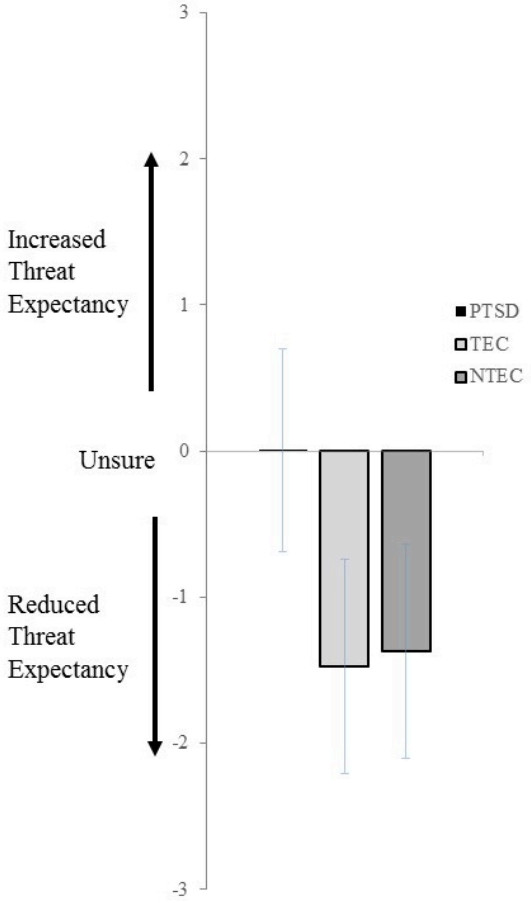

Figure 8. Mean Threat Expectancy Ratings for the PTSD, TEC and NTEC Groups during the Early Extinction Phase.

There was also a significant condition main effect [$F(1, 56) = 29.78$, $p = <.001$, $\eta p^2 = .35$] which revealed that there was significantly less threat expectancy with the CS^- than the CS^+. Furthermore, there was a significant trial main effect [$F(2.25, 126.01) = 49.04$, $p = <.001$, $\eta p^2 = .47$], following a Greenhouse-Geisser correction (see Figures 7 & 9). Sidak-adjusted post-hoc pairwise comparisons revealed that threat expectancy reduced over time across trials, and with the exception of trial 1 to trial 2, the differences between successive trials were significant (see Appendix D3). The main effect was superseded by a significant condition by trial interaction [$F(4, 224) = 5.01$, $p = .001$, $\eta p^2 = .08$]. See Figure 9. Sidak-adjusted post-hoc pairwise comparisons revealed that there were significant condition effects at each trial (see Appendix D4). When broken down by trial, it was revealed that fear extinction occurred more rapidly for the CS^+ than the CS^-, as the differences between all trials except 4 and 5 were significant for the CS^+, but only the difference between trials 2 and 3 was significant for the CS^- (see Appendix D5).

Figure 9. Condition by Trial Threat Expectancy Ratings during the Early Extinction Phase.

A marginal trend was identified for the trial by group interaction [$F(8, 224) = 1.78$, $p = .082$, $\eta p^2 = .06$], but the condition by group [$F(2, 56) = .37$, $p = .692$, $\eta p^2 = .01$], and condition by trial by group [$F(8, 224) = .76$, $p = .634$, $\eta p^2 = .03$] interactions were non-significant. Taking Age as a covariate did not alter the pattern of findings.

Late Extinction. A 3x2x5 repeated measures ANOVA in the late extinction phase found a significant main effect of group [$F(2, 56) = 6.39$, $p = .003$, $\eta p^2 = .19$]. See Figures 7 and 10. Sidak-adjusted post-hoc pairwise comparisons revealed that the PTSD group [$M = -.738$, $SE = .46$) had significantly greater threat expectancy ratings than the TEC group [$M = -2.700$, $SE = .48$, $p = .014$, 95% CI (.32,3.60)] and the NTEC group [$M = -2.863$, $SE = .48$, $p = .007$, 95% CI (.48, 3.77)]. The TEC and NTEC groups did not differ significantly [$p = .993$, 95% CI (-1.52, 1.84)].

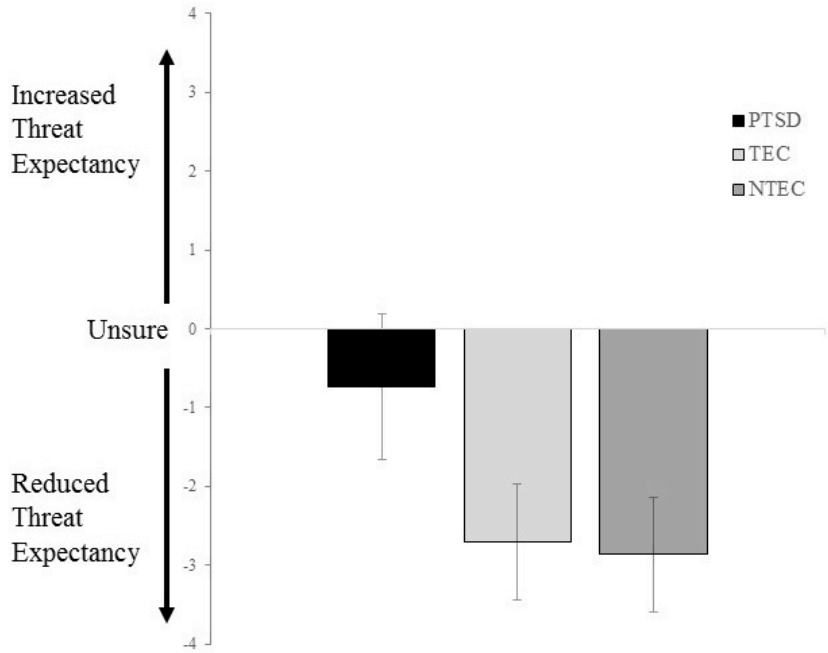

Figure 10. Mean Threat Expectancy Ratings for the PTSD, TEC and NTEC Groups during the Late Extinction Phase.

There was also a significant condition main effect [$F(1, 56) = 10.31$, $p = .002$, $\eta p^2 = .15$] which revealed significantly less threat expectancy with CS^- than CS^+. Finally, there was a significant trial main effect [$F(1.846, 103.37) = 36.28$, $p = <.001$, $\eta p^2 = .39$], following a Greenhouse-Geisser correction. Sidak-adjusted post-hoc pairwise comparisons revealed that threat expectancy reduced over time across trials, and the differences between successive trials were significant (See Appendix D3) with the exception of trial 4 to trial 5. A trend was identified for the condition by trial by group interaction [$F(8, 224) = 1.81$, $p = .076$, $\eta p^2 = .06$], but there was no significant condition by trial interaction following a Greenhouse-Geisser correction [$F(2.64,147.73) = .55$, $p = .623$, $\eta p^2 = .01$]. The condition by group [$F(2, 56) = .60$, $p = .551$, $\eta p^2 = .02$], and trial by group [$F(8, 224) = 1.14$, $p = .335$, $\eta p^2 = .04$] interactions were also non-significant. Taking Age as a covariate removed previous effects, except that of a significant main trial effect.

Mediation Analysis

Compared to the TEC and NTEC groups, the PTSD group was found to display significantly greater threat expectancy in early extinction and significantly higher levels of trauma-related catastrophic thinking on the total PTCI, but not significantly greater generalised catastrophic thinking on the CCQ-M. The simple mediation analysis (see Figure 12) was therefore performed using total PTCI as the mediator variable (*M*), PCL-C scores as the predictor variable (*X*), and a measure of average threat expectancy across trials for the CS^+ and CS^- for the early extinction phase (EEav) as the outcome variable (*Y*).

Prior to mediation analysis, the *c* and *a* pathways were tested for significance (see Figures 11 and 12). An increase in the PCL-C (*X*) was found to significantly

predict an increase in the EEav variable (*Y*; see Figure 11). An increase in the PCL-C (*X*) was found to significantly predict an increase in the total PTCI (*M*), as indicated by pathway *a* in Figure 12 (see Table 3).

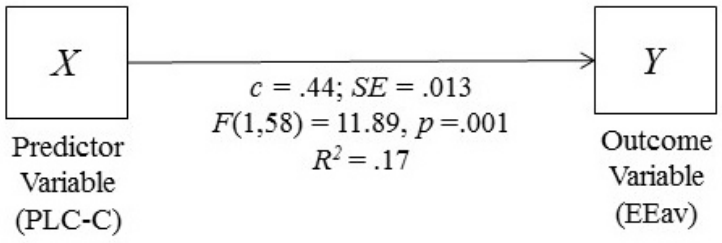

Figure 11. Total Significant Effect Model of PCL-C on EEav.

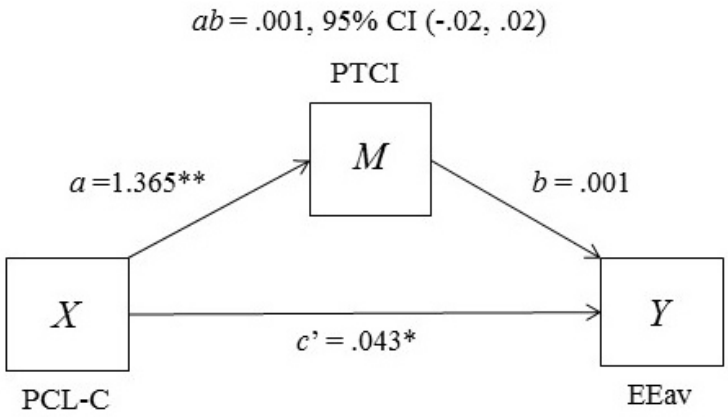

Figure 12. Simple Mediation Model for the Mediation Analysis of PTCI on the Direct Relationship Between PCL-C and EEav scores *(*$p < .05$, **$p < .01$).

The simple mediation model for the mediation analysis of PTCI on the direct relationship between PCL-C and EEav scores (see Figure 12) indicated a significant direct effect (*c'* pathway) of the PCL-C on the EEav variable, and a non-significant effect of total PTCI on the EEav variable (*b* pathway) as set out in Table 3. The indirect effect of PCL-C on EEav through total PTCI was non-significant as

bootstrapped 95% confidence intervals included zero (see Figure 12). Taking Age as a covariate reduced the significant direct effect (*c'* pathway) of the PCL-C on the EEav variable to trend level (p =.051).

Table 3

Model Coefficients for the Mediation Study of PTCI on the Direct Relationship Between PCL-C and EEav Scores

		Consequent						
		M (PTCI)				Y (EEAv)		
Antecedent		Coefficient	*SE*	*p*		Coefficient	*SE*	*p*
X(PCL-C)	*a*	1.365	.235	<.001**	*c'*	.043	.016	.011*
M (PTCI)	-	-	-	-	*b*	.001	.007	.906
Constant	i_1	41.011	8.616	<.001**	i_2	-2.407	.560	<.001**
		R^2 = .372, $F(1, 57) = 33.733$, $p < .001$**				R^2 = .173, $F(2,56) = 5.849$, $p = .005$**		

*$p < .05$. **$p < .01$. *Note.* PCL-C = PTSD Check List Civilian Version; PTCI = Posttraumatic Cognitions Inventory; EEAv = Measure of Average Threat Expectancy ratings for the CS^+ and CS^- across trials of the Early Extinction Phase.

Discussion

This study examined catastrophic thinking and threat expectancy in fear extinction in PTSD compared to controls, and addressed the question of whether catastrophic thinking may mediate the relationship between PTSD symptom severity and heightened threat expectancy in fear extinction. PTSD participants were found to have significantly greater catastrophic thinking than controls relating to their traumatic experiences (indexed by the PTCI score), but did not report differences in more generalised catastrophic thinking (total CCQ score). PTSD participants also

displayed significantly greater threat expectancy during fear extinction compared to controls, suggesting impaired fear extinction learning. Catastrophic thinking (as measured by the PTCI total score) was not found to mediate the relationship between PTSD symptoms (as measured by PCL-C scores) and fear extinction (as measured by threat expectancy ratings across the early extinction phase). These findings suggest that although impaired fear extinction and catastrophic cognitions of trauma are both associated with PTSD, catastrophic cognitions do not explain the relationship between PTSD symptoms and both heightened threat expectancy and impaired fear extinction learning. This study raises questions regarding some aspects of the cognitive model and highlights the need for the development of more integrative biological and cognitive models of PTSD.

Catastrophic Thinking in PTSD

The first hypothesis that PTSD participants will have greater catastrophic thinking than controls was partially confirmed; significant differences were found for the PTSD group relative to controls on the trauma-related PTCI measure (except for the Self-blame subscale), but no significant group differences were found for the more generalised CCQ-M measure. Together these findings suggest that heightened catastrophic thinking in PTSD is specific to trauma experiences rather than a more generalized trait and cognitive style.

The PTSD group scored significantly higher on the Total PTCI and and Self – subscale of the PTCI compared to both the NTEC and TEC groups, though there was no significant difference between the control groups. This suggests that negative appraisals about one's capabilities and response to trauma are more significant in PTSD than controls. Interestingly, both the PTSD and TEC groups scored

significantly higher on the World PTCI measures of negative appraisal compared to the NTEC group, but not compared to each other. This suggests that catastrophic thinking in relation to the world being unsafe or unpredictable is affected by exposure to trauma rather than specifically by PTSD diagnostic status. This is consistent with findings from Bryant and Guthrie's (2005) prospective study of fire-fighters in which pre-trauma catastrophic thinking on only the PTSI Self subscale predicted PTSD symptoms, but the World subscale was not found to be a significant predictor pre-trauma. Previous research also suggests there is increased trauma-related catastrophic thinking in PTSD, although a variety of measures have been used (Bryant & Guthrie, 2005; Dunmore et al., 2001; Halligan et al., 2003).

The Self-blame PTCI measure reflects the extent to which one believes they are to blame for the harm caused by the trauma, and group differences on this measure were found to be non-significant in the present study. Whist this finding is consistent with research finding that the Self-blame measure on the PTCI did not predict PTSD symptoms (Beck et al., 2004; Kolts Robinson, & Tracy, 2004), other research has suggested self-blame is important in PTSD (Foa et al., 1999; Frazier, Berman, & Steward, 2002; Laposa & Alden, 2003). Beck et al. (2004) suggested their non-significant Self-blame findings might be related to the nature of the trauma their participants experienced. Whilst their participants were victims of motor vehicle accidents who did not report excessive self-blame, nearly half of the participants in Foa et al. (1999) study were victims of assault whose scores on all three PTCI subscales were significantly greater than those of accident survivors. Startup, Makgekgenene and Webster (2007) found that victims of accidents, disasters and life-threatening illnesses produced the highest scores on all subscales of the PTCI,

and sexual assault victims reported the highest levels of self-blame. The present study used a mixed trauma sample, therefore the non-significant findings on the Self-blame measure of the PTCI may be due to differences in the type of trauma experienced.

No significant group differences were found for the CCQ-M, a generalised or trait measure of catastrophic thinking based on 3 types of negative appraisals (emotional, physical and mental) in relation to various experiences (rather than being specifically trauma-related). Previous research has tended to focus on a trauma-anchored measure only, but Dekel et al. (2013) used a general measure of negative cognition and found that increased PTSD symptoms amplified general maladaptive appraisals of the self and world over time. As this study was longitudinal and without control groups, a direct comparison cannot be made.

Threat Expectancy in the Fear Conditioning and Extinction Paradigm

The second hypothesis predicted that the PTSD group would have impaired fear extinction compared to the control groups. This was expected to be seen by higher threat expectancy ratings for the PTSD group in the fear extinction phase compared to controls, but no differences were predicted between groups in the fear conditioning phase. Differential responses to the CS^+ and CS^- followed the pattern expected in the differential fear conditioning paradigm, in that increased differential responses during fear conditioning and decreased differential responses during extinction were recorded. Therefore, the differential fear conditioning and extinction task resulted in valid fear conditioning and extinction. As hypothesised, the PTSD group maintained higher levels of threat expectancy for both the CS^+ and CS^-

throughout the extinction phases. Responses in each phase of the paradigm will be discussed in more detail below.

Habituation. A significant main effect of trial in the habituation phase revealed that threat expectancy reduced across trials. As participants were given instructions that they would not receive a shock during this phase, it was not surprising that fear reduction followed. There was a surprising condition main effect in the habituation phase that revealed that the CS^+ had greater threat expectancy than the CS^-. This was unexpected as the coloured circles for the CS^+ and CS^- were randomised across subjects and counterbalanced across groups. The size of this effect was small, and there was a trend for a group by condition effect that may shed some light on this main effect. The group by condition trend revealed that this heightened threat expectancy was observed in the trauma-exposed and PTSD groups, but not the NTEC group – this suggests there was generally greater threat expectancy (potentially related to anticipatory anxiety) in the trauma-exposed groups. However, the fact that these groups displayed less threat expectancy to the CS^+ is considered a random chance effect. It should be noted that all values were negative in this analysis, which reveals that threat was not expected on any trial (therefore, instructions were valid and this can be considered a valid baseline condition).

Acquisition. The significant main effect of condition on threat expectancy had a large effect size and indicated that participants were much more likely to expect a shock with the CS^+ than the CS^-. The condition by trial effect was also significant with a large effect size, indicating the threat expectancy was significantly larger for the CS^+ than the CS^- at every trial, and that this difference between conditions increased across successive trials (see Figure 7). This significant condition by trial

interaction confirmed that successful fear conditioning had occurred during the fear acquisition phase.

Extinction. As noted above, the second hypothesis that PTSD participants would display impaired fear extinction learning compared to controls was confirmed. This was seen by the group main effect in the extinction phase. Pairwise posthoc comparisons revealed that the PTSD group reported greater threat expectancy ratings compared to both controls groups (which did not differ), and this was of a moderate effect size. The finding that the PTSD group had impaired fear extinction learning is consistent with previous fear conditioning and extinction research, such as Sijbrandij et al. (2013) and Norrholm et al. (2011) studies that both reported fear-potentiated startle responses and threat expectancy ratings.

It is interesting to note that this effect of heightened threat expectancy in PTSD was evident to both the CS^+ and CS^-. This suggests that not only does the PTSD group expect threat with the danger signal, but it also has elevated threat expectancies in relation to safety signals. A positive value in threat expectancy (meaning actively expecting threat) was evidenced until trial 5 in the PTSD group, compared with trial 3 for both controls. In late extinction, the control groups maintained their sense of safety in relation to CS^+, but the PTSD group's sense of threat increased again and was positive until trial 4.

Early extinction findings were generally consistent with previous research (for e.g., Lommen et al., 2013), but late extinction findings differed somewhat. Typically, the late extinction phase or lengthened extinction phase indicates consolidation of previous extinction learning (for e.g., Gazendam & Kindt, 2012; Peri et al., 2000), however expectancy ratings for the CS^+ were raised in the PTSD group again in late

extinction, and examination of the graphs revealed a reinstatement of threat expectancy in late extinction. These findings may be explained by a methodological variation in the current study in which a saliva sample was taken for another research project between the early and late extinction phases. This may have resulted in increased uncertainty and a consequent elevation of threat expectancy. Interestingly, it appears that only the PTSD group responded to this disruption by shifting from a sense of safety in response to the CS^+ at the end of the early extinction phase (as measured by a negative mean threat expectancy at trial 5) to a sense of threat in response to the CS^+ at the start of late extinction phase (as measured by a positive mean threat expectancy at trial 1).

Does Catastrophic Thinking Mediate the Relationship between PTSD Symptoms and Fear Extinction?

The third hypothesis predicting that catastrophic thinking mediates the relationship between PTSD symptoms and fear extinction (as measured by threat expectancy in the early extinction phase) was not supported. In the present study, an average measure of threat expectancy for the CS^+ and CS^- across trials of the early extinction phase was used as the outcome variable in the mediation analysis due to a significant difference found for the PTSD group compared to controls. The total PTCI score was used as the mediator in this analysis as the PTSD group showed significantly greater catastrophic thinking according to this measure. Findings suggest that whilst there is a strong association between greater PTSD symptoms and both impaired extinction and heightened trauma-specific catastrophic thinking, difficulties in extinguishing fear are not explained by the heightened catastrophic cognition.

Studies analysing whether a cognitive mechanism accounts for the failure to extinguish fear are rare, and those involving PTSD have not been conducted to the author's knowledge. Gazendam and Kindt's (2012) study found that worry enhanced fear responses and impaired extinction, and Lovibond et al. (2009) found that avoidance responses in the extinction phase led to higher shock expectancy ratings being maintained; however, neither considered these variables in relation to PTSD.

As this was the first study of its kind, it is difficult to relate the current findings to previous research. However, given that it has been suggested that threat expectancy reflects both unconscious biological and conscious cognitive processes (Lovibond & Shanks, 2002), it could be argued that the fear-conditioning paradigm includes both cognitive and biological mechanisms involved in PTSD. Whilst catastrophic thinking may not mediate the relationship between PTSD symptoms and fear extinction, it is premature to reject the cognitive models of PTSD as other cognitive variables such as worry or avoidance may explain this relationship and further research is required.

Consistent with previous literature, a significant relationship was found between PTSD symptoms and both reduced fear extinction (for e.g., Guthrie & Bryant, 2006; Sijbrandij et al., 2013) and increased catastrophic thinking (for e.g., Bryant & Guthrie, 2005; Dekel, et al., 2013), but interestingly, the relationship between catastrophic thinking and reduced fear extinction was not significant. Including the non-significant self-blame subscale in the total PTCI measure used in the mediation analysis may have affected results, but when the mediation analysis was run again with only the self-blame subscale as the mediator variable, the findings were still non-significant.

Theoretical and Clinical Implications

Catastrophic thinking in PTSD. Partially in support of cognitive theory, the present study found that catastrophic thinking in the PTSD group was significantly greater than controls when trauma-specific, but not when generalised. These findings do not support schema theory unless the pre-trauma schemata are trauma-specific catastrophic cognitive patterns (Beck et al., 1979; Eysenck, 1992). Foa and Rothbaum (1988) build on schema theory by suggesting that if PTSD does not develop when trauma activates maladaptive schemata, then it develops when trauma violates a rigid belief about the predictability and safety of the world and of one being capable; the trauma cannot be interpreted and adaptively integrated as a unique experience. The current findings fit better with Foa and Rothbaum's theory (1988) as negative cognitions about the world were only significant in the group in which trauma had been experienced (PTSD and TEC groups), thus catastrophic cognitions or schemata about the world were not problematic pre-trauma in the PTSD group. As Ehlers and Clarke's (2000) cognitive model anchors catastrophic appraisals in PTSD to the traumatic event and its sequelae, the present study supports their theory in this respect. The current findings and previous research do implicate trauma-related catastrophic thinking in PTSD and perhaps suggest that adopting more evidence-based appraisals might be helpful in treating or preventing PTSD.

Fear Extinction Model of PTSD. The present study supported the biological model confirming that fear extinction is impaired in PTSD (Pitman et al., 2012). The persistence of heightened threat expectancy responses to the CS^{+} when no longer paired with electric shock implies that there is reduced capacity to extinguish aversive conditioned responses in PTSD.

Integrating cognitive and fear extinction models of PTSD. The third hypothesis tested a potential mechanism (catastrophic thinking) which may have integrated the cognitive and biological models of PTSD. Although the current study did not find that catastrophic thinking mediated the relationship between PTSD symptoms and fear extinction, this is the first study assessing this relationship and it is premature to reject any model on this basis of this or the potential to integrate models.

The persistence of PTSD according to Ehlers and Clark's (2000) cognitive model involves an ongoing sense of current threat due to catastrophic cognitions and disturbed memory coding, whereas the biological model suggests impaired extinction of conditioned fear is involved - both models implicate an ongoing sense of threat, and implicit memory and learning processes in the maintenance of PTSD. It is therefore reasonable to suggest that the cognitive and fear conditioning models are not mutually exclusive. However, further research is required to elucidate how the two models intersect.

Limitations and Future Research

Several limitations were identified in the current study. Firstly, the PTCI related responses to trauma, but not all participants had been exposed to trauma and NTEC participants were asked to fill in the PTCI in relation to a stressful event. Differences on this score may therefore have been associated with the relative severity of the distressful event to which responses related. Trauma varies in severity and therefore level of actual threat, but the relative severity and type of traumatic event was not measured in the present study. Future research would benefit from the inclusion of a measure of trauma severity, larger samples, and a PTSD group with greater PTSD

symptoms, as average PLC-C scores for the PTSD group (*M*=51.71) were only just within clinical guidelines (above 50 for diagnosis).

Secondly, as we were assessing the relationship between catastrophic cognitions and fear extinction, we selected threat expectancy ratings as a dependent measure as this has been linked to cognitive processes involved in fear extinction. However, most studies of fear conditioning and extinction utilise threat expectancy ratings in conjunction with SCR or startle responses (as indices of cognition and arousal respectively; Lovibond et al., 2009). Whilst some studies find a concordance between measures (see Lovibond & Shank, 2002), others do not concur (for e.g., Sijbrandj et al., 2013) and therefore further research is required comparing dependent measures or fear response. A mediating relationship of catastrophic cognitions between PTSD symptoms and fear extinction may be therefore be found if fear is measured by SCR, however further research is needed.

Thirdly, methodological considerations include the use of a100% reinforcement schedule of conditioned stimulus and unconditioned stimulus pairings in the acquisition phase, which appeared to cause a ceiling effect in the PTSD group. This has been found in some other previous studies with the same rate of reinforcement (for e.g., Bos, Beckers & Kindt, 2012). We selected a 100% reinforcement rate to ensure contingency learning, however, a partial reinforcement schedule in future research would likely reduce the chance of ceiling effects and delay acquisition and extinction. Such research might also shed light on whether the PTSD group maintains their level of threat expectancy even when faced with contradictory stimuli, as has been suggested in the past (Eysenck, 1992; Padesky, 1994).

Research would also benefit from a two-day testing paradigm to allow delayed recall test of fear extinction learning, as this has been found to be most robustly impaired in PTSD (Milad et al., 2008; Milad et al., 2009). This was beyond the scope of the present thesis and would require controlling for menstrual phase in women, as low levels of estrogen have been shown to specifically impair fear extinction recall (Milad, Igoe, Lebron-Milad, & Novales, 2009a; Milad et al., 2010).

Conclusion

The present study addressed the increasing debate about whether fear extinction learning is mediated by cognitive processing and applied this for the first time to PTSD. Specifically, this study examined whether catastrophic cognitions mediated the relationship between PTSD symptoms and fear extinction. In accordance with hypotheses, greater catastrophic cognitions were found in PTSD (although they were specific to the trauma experience) and impaired fear extinction (indexed by heightened threat expectancy ratings in the fear extinction phase). With findings in support of both cognitive and biological theory, the third hypothesis examined whether catastrophic thinking mediated the well-established relationship between posttraumatic symptoms and impaired fear extinction, but findings did not support this hypothesis. Whilst these findings do not support a significant interaction between biological and cognitive models in PTSD, further research is required as influential models suggest a potential integration of cognitive and biological models of PTSD (Ehlers & Clark, 2000). Future research would benefit from comparing several dependent measures of fear in conditioning (SCR, startle and threat expectancy), examining retention of fear extinction learning over time, and

investigating whether other cognitive variables mediate fear extinction learning in PTSD.

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Appendix A

Measures and Questionnaires

Appendix A1

Traumatic Experience Questionnaire (TEQ)

TRAUMATIC EXPERIENCE

ID: Mega_____ Date: _____

Below is a list of very traumatic or upsetting events that sometimes happen to people. Please indicate if any of these events have happened to you:

1. Have you ever had direct combat experience in a war?	Yes ☐	No ☐
2. Have you ever been involved in a life-threatening accident?	Yes ☐	No ☐
3. Have you ever been involved in a fire, flood or other natural disaster?	Yes ☐	No ☐
4. Have you ever witnessed someone being badly injured or killed?	Yes ☐	No ☐
5. Have you ever been seriously attacked, assaulted or molested?	Yes ☐	No ☐
6. Have you ever been threatened with a weapon, held captive, or kidnapped?	Yes ☐	No ☐
7. Have you ever been tortured or the victim of terrorists?	Yes ☐	No ☐
8. Have you ever experienced an extremely stressful or upsetting event?	Yes ☐	No ☐
9. Have you ever suffered a great shock because one of the events on the list happened to someone close to you?	Yes ☐	No ☐

If you are happy to be contacted for potential participation in a research study related to this questionnaire, please write your contact details below:

Name:_____

Email: _____

Mobile: _____

Appendix A2

PTSD CheckList – Civilian Version (PCL-C)

PTSD CheckList – Civilian Version (PCL-C)

~~**Client's Name:**~~ ID: Mega _____ Date: _____/_____/_____

Instruction to patient: Below is a list of problems and complaints that veterans sometimes have in response to stressful life experiences. Please read each one carefully, put an "X" in the box to indicate how much you have been bothered by that problem *in the last month*.

No.	Response	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
1.	Repeated, disturbing *memories, thoughts, or images* of a stressful experience from the past?					
2.	Repeated, disturbing *dreams* of a stressful experience from the past?					
3.	Suddenly *acting* or *feeling* as if a stressful experience *were happening* again (as if you were reliving it)?					
4.	Feeling *very upset* when *something reminded* you of a stressful experience from the past?					
5.	Having *physical reactions* (e.g., heart pounding, trouble breathing, or sweating) when *something reminded* you of a stressful experience from the past?					
6.	Avoid *thinking about* or *talking about* a stressful experience from the past or avoid *having feelings* related to it?					
7.	Avoid *activities* or *situations* because they *remind you* of a stressful experience from the past?					
8.	Trouble *remembering important parts* of a stressful experience from the past?					
9.	Loss of *interest in things that you used to enjoy?*					
10.	Feeling *distant* or *cut* off from other people?					
11.	Feeling *emotionally numb* or being unable to have loving feelings for those close to you?					
12.	Feeling as if your *future* will somehow be *cut short*?					
13.	Trouble *falling* or *staying asleep*?					
14.	Feeling *irritable* or having *angry outbursts*?					
15.	Having *difficulty concentrating*?					
16.	Being *"super alert"* or watchful on guard?					
17.	Feeling *jumpy* or easily startled?					

PCL-M for DSM-IV (11/1/94) Weathers, Litz, Huska, & Keane National Center for PTSD - Behavioral Science Division

Appendix A3

Depression, Anxiety and Stress Scale (DASS-21)

DASS21 *ID:* *Date:*

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

Appendix A4

Medical Questionnaire

Medical and History Questionnaire

ID: Mega_____ Date: _____

Medical History

Are you currently suffering from anxiety or depression? _____

Have you ever been diagnosed with a psychiatric disorder? _____

Are you currently receiving counselling or psychological problems? _____

Do you have heart condition or any other serious physical condition?

_____

Are you currently taking any prescription medication? If so, what medication?

_____

Have in the past taken any medication for psychological condition(s)? if so, what medication?

_____

Is there any possibility that you could be pregnant?

_____

Have you ever had or are you now suffering from any of the following (please circle):

Fits or convulsions	Yes	No
Epilepsy	Yes	No
Giddiness	Yes	No
Concussion	Yes	No
Severe head injury	Yes	No
Loss of consciousness	Yes	No

On average, how many cups of caffeinated drinks (coffee, coke or energy drinks) would you drink per day?

- ☐ None
- ☐ 1-2
- ☐ 3-4
- ☐ Over 5 cups

Smoking History

How often do you smoke cigarettes / tobacco / cigars / pipe? (please circle)

- ☐ Never
- ☐ Less than 5 per week
- ☐ Less than 5 per day
- ☐ 5 to 9 per day
- ☐ 10 to 19 per day
- ☐ 20 to 39 per day
- ☐ Over 40 per day

Do you or have you in the past used marijuana? (please circle) Yes No

a) Have you used marijuana in the last two weeks? Yes No

b) Have you used any other form of illicit drug in the last 6 months? Yes No

How often do you smoke marijuana?

- ☐ Never
- ☐ Less than 5 per week
- ☐ Less than 5 per day
- ☐ 5 to 9 per day
- ☐ 10 to 19 per day
- ☐ 20 to 39 per day
- ☐ Over 40 per day

Vision

Do you have any difficulties with vision? (Please specify)

_____

If yes, are these difficulties corrected (i.e. glasses / contacts)

_____

Appendix A5

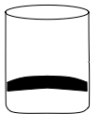
Alcohol Use Disorder Identification Test

Australian Government
Department of Veterans' Affairs

ID: Mega_____

Alcohol Screen (AUDIT)

THE RIGHT MIX

Light Beer 425ml 2.9% Alcohol	Full Strength Beer 285ml 4.9% Alcohol	Wine 100ml 12% Alcohol	Fortified Wine 60ml 20% Alcohol	Spirits 30ml 40% Alcohol	Full Strength Can or Stubbie 375ml 4.9% Alcohol
					

*The guide above contains examples of **one standard drink**.* A full strength can or stubbie contains ***one and a half standard drinks***.

Introduction

Because alcohol use can affect health and interfere with certain medications and treatments, it is important that we ask you some questions about your use of alcohol. Your answers will remain confidential, so please be as accurate as possible. Try to answer the questions in terms of **'standard drinks'**. Please ask for clarification if required.

AUDIT Questions Please tick the response that best fits your drinking.

						Score	Sub totals
		Never	*Monthly or less*	*2 - 4 times a month*	*2 - 3 times a week*	*4 or more times a week*		
1.	How often do you have a drink containing alcohol?	☐ Go to Qs 9 & 10	☐	☐	☐	☐	☐	
		1 or 2	*3 or 4*	*5 or 6*	*7 to 9*	*10 or more*		
2.	How many standard drinks do you have on a typical day when you are drinking?	☐	☐	☐	☐	☐	☐	
		Never	*Less than monthly*	*Monthly*	*Weekly*	*Daily or almost daily*		
3.	How often do you have six or more standard drinks on one occasion ?	☐	☐	☐	☐	☐	☐	☐
4.	How often during the last year have you found that you were not able to stop drinking once you had started?	☐	☐	☐	☐	☐	☐	
5.	How often during the last year have you failed to do what was normally expected of you because of drinking?	☐	☐	☐	☐	☐	☐	
6.	How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?	☐	☐	☐	☐	☐	☐	☐
7.	How often during the last year have you had a feeling of guilt or remorse after drinking?	☐	☐	☐	☐	☐	☐	
8.	How often during the last year have you been unable to remember what happened the night before because you had been drinking?	☐	☐	☐	☐	☐	☐	
			No		*Yes, but not in the last year*	*Yes, during the last year*		
9.	Have you or someone else been injured because of your drinking?		☐		☐	☐	☐	
10.	Has a relative, friend, doctor, or other health care worker been concerned about your drinking or suggested you cut down?		☐		☐	☐	☐	☐
							TOTAL	

Supplementary Questions	*No*	*Probably Not*	*Unsure*	*Possibly*	*Definitely*
Do you think you presently have a problem with drinking?	☐	☐	☐	☐	☐
	Very easy	*Fairly easy*	*Neither difficult nor easy*	*Fairly difficult*	*Very difficult*
In the next 3 months, how difficult would you find it to cut down or stop drinking?	☐	☐	☐	☐	☐

D0718 - 8/03 - P1 of 2

Appendix A6

Catastrophic Cognitions Questionnaire – Modified (CCQ-M)

Catastrophic Cognitions Questionnaire (CCQ)

ID: Mega_____ Date: _____

This questionnaire aims at measuring your beliefs and thoughts regarding the following items. Sometimes these items are believed to be DANGEROUS. Please read the items carefully, and choose a number from 1 to 5 from the scale given below to rate the extent you believe them to be dangerous to you. Write the number you choose in the box to the right of each item.

1	2	3	4	5
Not at all dangerous	A little dangerous	Quite dangerous	Very dangerous	Extremely dangerous

1	Feeling edgy	
2	Having an accident	
3	Mind not functioning normally	
4	Being miserable	
5	Being injured	
6	Unable to think rationally	
7	Feeling shaky	
8	Having a stroke	
9	Unable to control thinking	
10	Being agitated	
11	Being ill	
12	Losing memory	
13	Unable to relax	
14	Being suffocated	
15	Being mentally blocked	
16	Being alarmed	
17	Being attacked	
18	Being out of senses	
19	Being angry	
20	Losing sight	
21	Being mentally blurred	

Appendix A7

Posttraumatic Cognitions Inventory (PTCI)

Posttraumatic Cognitions Inventory (PTCI)

ID: Mega_____ Date: _____

We are interested in the kind of thoughts you may have had after a traumatic experience. Below are a number of statements that may or may not be representative of your thinking.
Please read each statement carefully and tell us how much you AGREE or DISAGREE with each by putting the appropriate number between 1 & 7 in the box to the right of the statement. People react to traumatic events in many different ways. If you have not experienced a traumatic event, please consider thoughts after the most stressful experience you can recall. There are no right or wrong answers to these statements.

1	2	3	4	5	6	7
Totally disagree	Disagree very much	Disagree slightly	Neutral	Agree slightly	Agree very much	Totally agree

1	The event happened because of the way I acted	
2	I can't trust that I will do the right thing	
3	I am a weak person	
4	I will not be able to control my anger and will do something terrible	
5	I can't deal with even the slightest upset	
6	I used to be a happy person but now I am always miserable.	
7	People can't be trusted	
8	I have to be on guard all the time	
9	I feel dead inside	
10	You can never know who will harm you	
11	I have to be especially careful because you never know what can happen next	
12	I am inadequate	
13	I will not be able to control my emotions, and something terrible will happen	
14	If I think about the event, I will not be able to handle it	
15	The event happened to me because of the sort of person I am	
16	My reactions since the event mean that I am going crazy	
17	I will never be able to feel normal emotions again	
18	The world is a dangerous place	
19	Somebody else would have stopped the event from happening	
20	I have permanently changed for the worse	
21	I feel like an object, not like a person	
22	Somebody else would not have gotten into this situation	
23	I can't rely on other people	
24	I feel isolated and set apart from others	
25	I have no future	
26	I can't stop bad things from happening to me	
27	People are not what they seem	
28	My life has been destroyed by the trauma	
29	There is something wrong with me as a person	
30	My reactions since the event show that I am a lousy coper	
31	There is something about me that made the event happen	
32	I will not be able to tolerate my thoughts about the events, and I will fall apart	
33	I feel like I don't know myself anymore	
34	You never know when something terrible will happen	
35	I can't rely on myself	
36	Nothing good can happen to me anymore	

Appendix B

Ethics Approval Letters

Social Science Ethics Officer
Private Bag 01 Hobart
Tasmania 7001 Australia
Tel: (03) 6226 2763
Fax: (03) 6226 7148
Katherine.Shaw@utas.edu.au

HUMAN RESEARCH ETHICS COMMITTEE (TASMANIA) NETWORK

12 September 2013

Assoc Prof Kim Felmingham
School of Psychology
Private Bag 30

Student Researchers: Daniel Zuj
Kate Gray
Ken Chia-Ming Hsu

Sent via email

Dear Assoc Prof Felmingham

Re: FULL ETHICS APPLICATION APPROVAL
Ethics Ref: **H0013304 - The Effect of Sleep on Emotional Memory and Fear Extinction in PTSD**

We are pleased to advise that the Tasmania Social Sciences Human Research Ethics Committee approved the above project on 8 September 2013.

This approval constitutes ethical clearance by the Tasmania Social Sciences Human Research Ethics Committee. The decision and authority to commence the associated research may be dependent on factors beyond the remit of the ethics review process. For example, your research may need ethics clearance from other organisations or review by your research governance coordinator or Head of Department. It is your responsibility to find out if the approval of other bodies or authorities is required. It is recommended that the proposed research should not commence until you have satisfied these requirements.

Please note that this approval is for four years and is conditional upon receipt of an annual Progress Report. Ethics approval for this project will lapse if a Progress Report is not submitted.

The following conditions apply to this approval. Failure to abide by these conditions may result in suspension or discontinuation of approval.

1. It is the responsibility of the Chief Investigator to ensure that all investigators are aware of the terms of approval, to ensure the project is conducted as approved by the Ethics

Committee, and to notify the Committee if any investigators are added to, or cease involvement with, the project.

2. Complaints: If any complaints are received or ethical issues arise during the course of the project, investigators should advise the Executive Officer of the Ethics Committee on 03 6226 7479 or human.ethics@utas.edu.au.

3. Incidents or adverse effects: Investigators should notify the Ethics Committee immediately of any serious or unexpected adverse effects on participants or unforeseen events affecting the ethical acceptability of the project.

4. Amendments to Project: Modifications to the project must not proceed until approval is obtained from the Ethics Committee. Please submit an Amendment Form (available on our website) to notify the Ethics Committee of the proposed modifications.

5. Annual Report: Continued approval for this project is dependent on the submission of a Progress Report by the anniversary date of your approval. You will be sent a courtesy reminder closer to this date. **Failure to submit a Progress Report will mean that ethics approval for this project will lapse.**

6. Final Report: A Final Report and a copy of any published material arising from the project, either in full or abstract, must be provided at the end of the project.

Yours sincerely

Katherine Shaw
Ethics Officer
Tasmania Social Sciences HREC

Social Science Ethics Officer
Private Bag 01 Hobart
Tasmania 7001 Australia
Tel: (03) 6226 2763
Fax: (03) 6226 7148
Human.ethics@utas.edu.au

HUMAN RESEARCH ETHICS COMMITTEE (TASMANIA) NETWORK

30 April 2014

Professor Kim Felmingham
Psychology
Private Bag 30

Sent via email

Dear Professor Felmingham

Re: APPROVAL FOR AMENDMENT TO CURRENT PROJECT
Ethics Ref: **H0013304 - The Effect of Sleep on Emotional Memory and Fear Extinction in PTSD**

- Addition of two investigators: Ms Pippa Cushing and Ms Emma Nicholson.
- Remove mention of the Zeo sleep monitor.
- Adding standardised tests of verbal function (the Rey Auditory Verbal Learning Test and the National Adult Reading Test), and to do a delayed recall of the RAVLT on Day 2.
- Expand recruitment of participants to an additional source: the Yoga Association of Tasmania.
- Revised Information Sheets and Consent Forms.

We are pleased to advise that the Chair of the Tasmania Social Sciences Human Research Ethics Committee approved the Amendment to the above project on 29 April 2014.

Yours sincerely

Katherine Shaw
Executive Officer
Tasmania Social Sciences HREC

Appendix C

Information Sheets and Consent Forms

Appendix C1

Information Sheet

Participant Information Sheet

The effect of sleep quality and cognitive factors on fear extinction in PTSD

Invitation
You are invited to participate in a research study examining the influence of sleep and cognitive factors on fear extinction in Posttraumatic Stress Disorder (PTSD). This study will be carried out in the Cognitive Neuroscience Laboratory at the School of Psychology, University of Tasmania (Hobart campus). The study is being conducted by the following people:

- Professor Kim Felmingham, supervisor and chief investigator, UTAS.
- Mr Daniel Zuj, PhD Candidate, UTAS.
- Ms Kate Gray, Masters student, UTAS.
- Mr Ken Chia, Masters student, UTAS.
- Ms Emma Nicholson, Masters student, UTAS.
- Ms Pippa Cushing, Honours student, UTAS.

What is the purpose of this study?
The purpose of this study is to investigate the effect of sleep quality and a number of cognitive variables on fear extinction in PTSD. Previous research has indicated that difficulty in extinguishing fear is an important influence on the severity of PTSD symptoms. Recent research has also revealed that quality of sleep and cognitive variables (styles of thinking) can predict the severity of symptoms in PTSD. Therefore, the present study also aims to determine the degree that sleep difficulties (and additional cognitive variables) effect performance on a fear extinction task in PTSD.

Why have I been invited to participate?
You have been invited to participate in this study as you are a psychology student and this project is being offered as part of research participation course credit. We are looking for volunteers who are not currently taking any medication and are aged between 18 and 55. You are eligible for participation in this study if you have never experienced a traumatic event OR you have experienced a traumatic event OR you have experienced a traumatic event and meet diagnostic criteria for PTSD.

What will I be asked to do?
You will be asked to engage in two testing sessions at the Cognitive Neuroscience Laboratory, School of Psychology, UTAS: an initial session where you will complete some questionnaires and view some emotional images from a standard picture series (some of these images will be negative and involve images of injury or violence, and may be mildly distressing). You will also be asked to do some tests of verbal memory and verbal function During the second testing session, you will be asked to complete a number of questionnaires, and to complete a behavioural task examining how your body arousal (sweat gland activity) reacts to a mild electrical stimulus that will be administered to your fingertips. You will first be asked to select a level of mild electrical stimulus that feels uncomfortable but not painful to you. This will be done by attaching a finger stimulator to your index finger and delivering the lowest level of electrical stimulus, the level of which will then be increased in small increments until you report that it feels uncomfortable but not painful. You will then be asked to complete the behavioural task. In this task, you will sit in front of a computer screen and small recording disks will be attached to your fingertips to measure your body arousal (via

skin conductance). You will be asked to watch a computer screen, on which you will see different coloured circles (red or blue) appear. Following the presentation of some of these coloured circles, you will receive an electrical stimulus, which will be set at the level you have previously chosen. You will also be asked to provide ratings on how much you are expecting to receive the electrical stimulus in the task. This behavioural task will last approximately 15 minutes.

Are there any possible benefits from participation in this study?
If you decide to participate in this research, you will gain experience in research procedures and also some knowledge of underlying mechanisms of anxiety and exposure therapy through the process of fear extinction. If you are enrolled in first year Psychology, you will also receive research participation credit of 2 hours for your participation. Furthermore, you will be involved in research that may provide a platform to better understand the mechanisms and processes involved in the extinction of fear within the context of PTSD, and this may also lead to more efficient and effective exposure treatments for anxiety disorders.

Are there any possible risks from participation in this study?
Prior to participation in this study, you will be asked to sign a consent form, which will evidence your agreement to participate. You may feel a small amount of arousal or discomfort from viewing the negative images or from the mild electrical stimulus. However, we expect this arousal or discomfort to be minimal, as these are standard images and are not graphic (similar to what would be seen on television crime shows) and the electrical stimulus level that is administered will have been selected by you to be uncomfortable but not painful. The technology used to administer this electrical stimulus is very safe and has been used in many previous studies with no adverse effects reported. There will be a researcher with you at all times, and you can discontinue the study at any time without penalty and it will not affect your relationship with the University of Tasmania or the School of Psychology.

What if I change my mind during or after the study?
Participation in this research is entirely voluntary. You may choose to withdraw from the study at any time without prejudice. Deciding to withdraw from this research at any time will not affect your academic standing in any way. You can also choose at this time to withdraw any data previously collected. Participants will be given copies of this information sheet and the statement of informed consent.

What will happen to the information when this study is over?
Your individual data will be treated confidentially and your name will be replaced by an ID number on all data. Data will be kept in a locked filing cabinet or on password secured computers at the School of Psychology at the University of Tasmania for a period of at least five years.

How will the results of the study be published?
Following completion of the research, the data obtained from this study will be published. However, no participant will be personally identifiable in these publications, as only group data will be published. A summary of the results of these experiments will be available on the University of Tasmania School of Psychology web page at www.utas.edu.au/psychology or will be available by contacting the researchers.

What if I have questions about this study?
The researchers will be available after the testing session to answer any questions you may have. If you have any questions, or would like any additional information regarding this research, please contact Daniel Zuj at Daniel.Zuj@utas.edu.au, or Professor Kim Felmingham at Kim.Felmingham@utas.edu.au.

This study has been approved by the Tasmanian Social Sciences Human Research Ethics Committee. If you have concerns or complaints about the conduct of this study, please contact the Executive Officer of the HREC (Tasmania) Network on (03) 6226 7479, or email human.ethics@utas.edu.au. The Executive Officer is the person nominated to receive complaints from research participants. Please quote ethics reference number [Hxxxxx].

Thank you for taking the time to consider this study.
If you wish to take part, please sign the attached consent form.
This information sheet is for you to keep.

Appendix C2

Consent Forms

Participant Consent Form

The effect of sleep quality and cognitive factors on fear extinction in PTSD

Participant Consent Statement:

1. I agree to take part in the study named above.
2. I have read and understood the Information Sheet for this study.
3. The nature and possible effects of the study have been explained to me.
4. Any questions that I have asked have been answered to my satisfaction.
5. I understand that the study requires me to attend the Cognitive Neuroscience laboratory at the School of Psychology twice – once to complete questionnaires and view emotional images, and once where my arousal responses will be recorded whilst I view different coloured circles and receive a mild electrical stimulus to my fingers. I understand that I can set the level of this mild electrical stimulus to feel uncomfortable but not painful prior to the task.
6. I understand that the study requires me to wear a sleep monitor on my forehead for the night preceding participation in this study. I understand that the sleep monitor should cause minimal amounts of discomfort, and is only required to record brain wave activity during deep sleep.
7. I understand that all research data will be treated as confidential. I agree that research data gathered for the study may be published provided that I cannot be identified as a participant.
8. I understand that my participation is voluntary and that I may withdraw from participation and/or withdraw my data at any time without prejudice to my academic standing.

Participant's name: _____

Participant's signature: _____ Date: _____

Investigator Statement

I have explained this research and the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation.

Investigator's name: _____

Investigator's signature: _____ Date: _____

Appendix D

Supplementary Results

Appendix D1

Table D1

Sidak-Adjusted Post-Hoc Pairwise Comparisons for Age and Clinical Measure ANOVAs Including p-values and 95% Confidence Intervals

			95% Confidence Intervals	
Variable	Comparison	*p*	Lower Bound	Upper Bound
Age	PTSD & TEC	.054	-.113	18.77
	PTSD & NTEC	.012*	2.09	20.98
	TEC & NTEC	.924	-7.46	11.88
PCL-C	PTSD & TEC	<.001**	20.80	32.95
	PTSD & NTEC	<.001**	25.16	37.32
	TEC & NTEC	.246	-1.86	10.59
DASS				
- Depression	PTSD & TEC	<.001**	2.24	9.02
	PTSD & NTEC	<.001**	3.29	10.07
	TEC & NTEC	.842	-2.42	4.53
- Anxiety	PTSD & TEC	<.001**	3.82	8.06
	PTSD & NTEC	<.001**	4.14	8.34
	TEC & NTEC	.978	-1.85	2.49
- Stress	PTSD & TEC	<.001**	3.88	9.98
	PTSD & NTEC	<.001**	5.46	11.56
	TEC & NTEC	.523	-1.54	4.70

*$p < .05$. **$p < .01$. *Note.* PCL-C = PTSD Check List Civilian Version; DASS = Depression, Anxiety and Stress Scales

Appendix D2

Table D2

Sidak-Adjusted Post-Hoc Pairwise Comparisons for Posttraumatic Cognitions Inventory ANOVAs, Including p-values and 95% Confidence Intervals

			95% Confidence Intervals	
Variable	Comparison	*p*	Lower Bound	Upper Bound
PTCI Total	PTSD & TEC	.008**	6.81	56.22
	PTSD & NTEC	<.001**	16.76	66.17
	TEC & NTEC	.709	-15.37	35.26
PTCI Self	PTSD & TEC	.015*	.15	1.77
	PTSD & NTEC	.003**	.35	1.97
	TEC & NTEC	.916	-.63	1.03
PTCI World	PTSD & TEC	.060	-1.98	.03
	PTSD & NTEC	<.001**	1.06	3.08
	TEC & NTEC	.035*	.06	2.12
PTCI Self-Blame	PTSD & TEC	.130	-.18	1.95
	PTSD & NTEC	.541	-.54	1.59
	TEC & NTEC	.805	-1.45	1.45

*$p < .05$. **$p < .01$. *Note.* PTCI = Posttraumatic Cognitions Inventory

Appendix D3

Table D3

Sidak-Adjusted Post-Hoc Pairwise Comparisons of Threat Expectancy Across Trials for Phases in which ANOVAs Identified a Significant Main Trial Effect

			95% Confidence Intervals	
Phase	Trial Comparison (*a* and *b*)^	*p*	Lower Bound	Upper Bound
Habituation	1 and 2	.486	-.30	1.22
	1 and 3	.441	-.30	1.30
	1 and 4	.048*	.01	1.65
	2 and 3	1.00	-.29	.37
	2 and 4	.228	-.11	.85
	3 and 4	.171	-.08	.73
Early Extinction	1 and 2	.080	-.05	1.43
	1 and 3	<.001**	.87	2.85
	1 and 4	<.001**	1.73	3.87
	1 and 5	<.001**	2.34	4.47
	2 and 3	<.001**	.44	1.89
	2 and 4	<.001**	1.21	3.00
	2 and 5	<.001**	1.75	3.67
	3 and 4	<.001**	.41	1.48
	3 and 5	<.001**	.91	2.19
	4 and 5	.005**	.13	1.08
Late Extinction	1 and 2	.002**	.32	2.09
	1 and 3	<.001**	1.16	3.11
	1 and 4	<.001**	1.37	3.48
	1 and 5	<.001**	1.60	3.72
	2 and 3	<.001**	.38	1.47
	2 and 4	<.001**	.55	1.89
	2 and 5	<.001**	.73	2.17
	3 and 4	.033*	.01	.56
	3 and 5	.002**	.14	.91
	4 and 5	.350	-.09	.57

*$p < .05$. **$p < .01$. *Note.* ^These figures are based on the mean difference in threat expectancy ratings over 2 trials (trial *a* minus trial *b*).

Appendix D4

Table D4

Sidak-Adjusted Post-Hoc Pairwise Comparisons of the Condition by Trial Interaction of Threat Expectancy for Phases in which ANOVAs Identified a Significant Condition by Trial Effect.

Phase	Trial	Condition Comparison (*a* and *b*)^	*p*	95% Confidence Intervals	
				Lower Bound	Upper Bound
Acquisition	1	CS^+ and CS^-	.018	.17	1.78
		CS^- and CS^+	.018	-1.78	-.17
	2	CS^+ and CS^-	<.001**	4.12	6.64
		CS^- and CS^+	<.001**	-6.54	-4.12
	3	CS^+ and CS^-	<.001**	5.50	8.10
		CS^- and CS^+	<.001**	-8.10	-5.50
	4	CS^+ and CS^-	<.001**	6.06	8.46
		CS^- and CS^+	<.001**	-8.46	-6.06
	5	CS^+ and CS^-	<.001**	7.50	9.24
		CS^- and CS^+	<.001**	-9.24	-7.50
Early Extinction	1	CS^+ and CS^-	<.001**	2.08	4.37
		CS^- and CS^+	<.001**	-4.37	-2.08
	2	CS^+ and CS^-	<.001**	.91	2.93
		CS^- and CS^+	<.001**	-2.93	-.91
	3	CS^+ and CS^-	<.001**	1.29	3.20
		CS^- and CS^+	<.001**	-3.20	-1.29
	4	CS+ and CS-	.001**	.67	2.31
		CS- and CS+	.001**	-2.31	-.67
	5	CS+ and CS-	.004**	.42	2.16
		CS- and CS+	.004**	-2.16	-.42

*$p < .05$. **$p < .01$. *Note.* ^These figures are based on the mean difference in threat expectancy between two conditions (condition *a* minus condition *b*).

Appendix D5

Table D5

Sidak-Adjusted Post-Hoc Pairwise Comparisons of the Trial by Condition Interaction of Threat Expectancy in the Early Extinction Phase

			95% Confidence Intervals	
Condition	Trial Comparison (*a* and *b*)^	*p*	Lower Bound	Upper Bound
CS^+	1 and 2	.005**	.28	2.41
	1 and 3	<.001**	1.18	3.51
	1 and 4	<.001**	2.24	5.11
	1 and 5	<.001**	2.94	5.80
	2 and 3	.028*	.06	1.94
	2 and 4	<.001**	1.19	3.46
	2 and 5	<.001**	1.86	4.19
	3 and 4	.001**	.42	2.22
	3 and 5	<.001**	1.06	2.98
	4 and 5	.414	-.33	1.73
CS^-	1 and 2	1.000	-.92	1.01
	1 and 3	.029*	.09	2.65
	1 and 4	<.001**	.71	3.16
	1 and 5	<.001**	1.11	3.76
	2 and 3	.001**	.42	2.23
	2 and 4	<.001**	.83	2.95
	2 and 5	<.001**	1.27	3.52
	3 and 4	.488	-.31	1.44
	3 and 5	.005**	.23	1.91
	4 and 5	.231	-.14	1.14

*$p < .05$. **$p < .01$. *Note.* ^These figures are based on the mean difference in threat expectancy ratings over 2 trials (trial *a* minus trial *b*).

Appendix E

SPSS Output