

Final Summary, Proposed Ecological Trauma Theory, Implications for Safe Interventions and Future Research

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Interventions and Future Research**

1. Final Summary

The broad aims of this thesis were to elucidate the critical determinants that underlie the development and maintenance of posttraumatic intrusion, hyperarousal and avoidance symptom clusters in PTSD and to outline a trauma theory for the development of safe and effective interventions. In order to facilitate these aims a literature review was conducted (Part I). Based on the literature review it was concluded that PTSD appeared to be a functional survival response to a life-threatening trauma that was maintained, or even enhanced, over time by post-trauma variables. An animal predator-prey model of survival as outlined by Kavaliers and Choleris (2001) was used as a framework for identifying potential factors to be investigated in the development and maintenance of posttraumatic symptom clusters (Part II: Papers 1-6).

Following a qualitative investigation into the existence of PTSD cross-culturally (paper one), factors derived from the animal predator-prey model were investigated in trauma populations exposed to large-scale natural or man-made disasters (papers three, four and six). The Impact of Events Scale-Revised (IES-R) was used as a measure of posttraumatic symptom clusters across these human trauma populations. Due to the cross-cultural nature of the studies, validation of a translated and modified version of the IES-R was included (papers two and six). Two animal experiments were also conducted to allow for experimental manipulation of factors considered problematic in human subjects (paper five). This final chapter (Part III) includes a summary and addresses the second aim of the thesis, which was to develop a trauma model from related research literature and the experimental findings of papers one to six. The implications for safe psychological interventions following traumatic events and future research are also discussed at the end of this chapter.

The first experimental paper in this thesis highlights the existence of PTSD in non-Western cultures. Congolese, Ugandan and Sudanese community leaders reported similar symptom clusters to those that characterise PTSD in Western culture. The Africans identified these cluster of symptoms as the ‘insanity’ or ‘madness’ that occurs after rape, war and other severe traumatic events. Despite this different label many of their astute observations regarding the ‘insanity’ matched Western knowledge of PTSD. Importantly they also raised the same concerns as can be found in Western research literature regarding a possible negative effect of brief psychological interventions on some trauma populations. Of interest is their observation that the ‘insanity’ was

rare in small rural communities where supportive social networks, individualised care, community-grieving customs and rituals that symbolised cleansing and a sense of shared community pain were present.

The second experimental paper validated a Sri Lankan version of the Impact of Events Scale Revised (IES-R-SL) for use in subsequent studies. Results revealed that the IES-R-SL had a similar factorial structure as the IES-R. High reliability co-efficients were found for the total scale and subscales. Identified factors and weaknesses were in accordance to that reported for English, French and Japanese versions of the IES-R. Analysis of data from the IES-R-SL across two time points (three weeks and six months post-tsunami) indicated that the IES-R-SL was less sensitive to the core PTSD construct of hyperarousal in the immediate aftermath of the tsunami. High levels of distress in the majority of subjects three weeks after the tsunami was thought to be the reason for the initial low sensitivity. Similar to other studies, correlations between the three subscales suggested that avoidance taps into a separate construct to hyperarousal and intrusion symptoms. Given the similarities to other translations of the IES-R it was concluded that the IES-R-SL could be scored according to the original IES-R for comparative purposes cross-culturally.

The aim of the third experimental paper was to conduct an exploratory study into maintaining and mitigating factors of posttraumatic hyperarousal, intrusion and avoidance symptom clusters. Specifically, to ascertain whether posttraumatic symptom clusters of intrusion, hyperarousal and avoidance are maintained by variables analogous to Kavaliers and Choleris's (2001) animal preparatory predator-prey responses (current risk of life-threat, threat exposure, individual resources to negotiate threat and environmental refuge from threat). A trauma population exposed to war atrocities (Africa) and a population exposed to the 2004 Asian tsunami (Sri Lanka) was administered the IES-R (Africa) or the IES-R-SL (Sri-Lanka) along with subjective ratings of perceived current risk of life-threat. Perceived current risk of life-threat was significantly correlated with intrusion, hyperarousal and avoidance symptom clusters in both the Sri Lankan and African population.

The African population was asked additional questions relating to the remaining preparatory predator-prey responses (threat exposure, individual resources to negotiate threat and environmental refuge from threat). Results revealed significant correlations between:

hyperarousal and exposure to life-threat; hyperarousal and current refuge from threat; intrusion and current refuge from threat; current perceived risk of life-threat and exposure to life threat; and current perceived risk of life-threat and current refuge from threat. The exploration of strategies used by the African participants revealed that, of the four strategies described by the Africans (spiritual, avoidance, family/friends and behavioural), only those who reported using spiritual strategies had significantly lower mean intrusion and perceived risk of life-threat scores than no strategy at all. Further, only those Africans who reported using spiritual strategies had significantly lower refuge scores (lower score = higher refuge) when compared to no strategy at all (i.e. no action taken). Based on the findings and available research a model was then outlined of how individual and environmental factors interact to produce variability in individual posttraumatic responses following a similar trauma.

The aim of the fourth experimental paper was to build on the knowledge gained in the previous study on the possible role of the environment in the development and maintenance of posttraumatic symptom clusters. This aim was addressed by administering the IES-R-SL to people-helpers exposed to the aftermath of the 2004 Asian tsunami at three weeks and six months post-tsunami. The people-helpers permanently resided in one of two geographical locations in Sri Lanka. One geographical location (Batticaloa) was on the east coast of Sri Lanka where the tsunami struck and the other location (Colombo) was on the opposite side of the country. Participants who permanently resided in the tsunami-affected location had significantly higher hyperarousal and avoidance scores at both time points. Intrusion scores were significantly higher in subjects living in the tsunami-affected location at six months only. The results were discussed in light of recent animal and human studies. It was proposed that in PTSD a cycle occurs in which an environment perceived to be dangerous reinstates learned fear to trauma reminders (cues), which in turn consolidates images and feelings associated with the trauma, which in turn confirms the perception that the environment is dangerous.

The aim of the fifth experimental paper was to explore the possibility raised in previous papers that the environment and discrete trauma cues interact to produce ongoing symptoms in PTSD. As experimentally manipulating these variables in humans was considered to be ethically problematic, investigation into fear-related context (environment) and discrete cue interactions was explored in animals. Context/cue interactions were explored using variations of forward and backward fear conditioning procedures (i.e. pairings of a brief tone and shock in a chamber) in

rats. Results revealed that (a) following CS (tone)-US (shock) pairings, the CS can be indistinguishable from a global context that is excitatory (i.e. all elements of the global context in which the CS-US was experienced have the ability to elicit fear), (b) learned associations that predict a local context of safety facilitate differentiation from a global excitatory context, (c) cues predictive of a threat modulate ongoing levels of fear to the context in which the cue was encountered, (d) levels of fear to the context modulate ongoing levels of fear to backward and delayed cues (e) measures of fear to a discrete cue associated with shock are significantly higher when re-exposed to the cue at one month post-shock compared to re-exposure at one day post-shock. The relationship between these findings and PTSD symptomatology was explained by expanding on Rudy, Huff and Matus-Amat's (2004) two-process model of hippocampal mediated contextual fear conditioning.

The aim of the sixth experimental paper was to indirectly explore whether: (a) a dangerous context maintains posttraumatic symptoms, (b) a failure to experience or identify associations that predict a local context of safety is associated with a global excitatory context and (c) a perceived global excitatory context is correlated with posttraumatic symptoms levels. This aim was achieved by administering a modified version of the IES-R and a questionnaire rating subjective levels of threat and safety across contexts (local to global) to Northern Ugandan High School students currently living in a dangerous context (a war zone with the constant threat of gunfire, abduction by rebel armies and/or rape). Confirmatory factor analysis and exploratory factor analysis was also conducted on the modified IES-R to validate its use. Exploratory factor analysis was also conducted on the questionnaire rating subjective levels of threat and safety to ascertain whether questions tapped into discrete constructs.

Results of the confirmatory factor analysis on the IES-R revealed that the data did not fit the traditional three-factor model of avoidance, intrusions and hyperarousal, nor did it fit a general distress or two factor model as suggested in the literature. Follow-up exploratory factor analysis revealed a six-factor solution better fitted the data. Due to the differences in identified factors to the original IES-R, a total score was used for correlational purposes rather than sub-scales as in other studies. Exploratory factor analysis on the questionnaire (14 questions) rating subjective levels of threat and safety across contexts identified three separate factors. These factors were identified as generalised safety, global threat and context-specific threat. Similar to the rat

studies, the factor analysis also revealed that a failure to experience a local context of safety was associated with a global perception of threat.

It was expected that the majority of students would exhibit PTSD-like symptoms if a dangerous context maintains PTSD symptom clusters. Results revealed that 99% of the students had posttraumatic symptoms of clinical significance. Significant correlations were identified between ratings of safety and threat across contexts and posttraumatic symptoms. Findings were discussed in light of animal studies that identified that exposure to a dangerous context re-establishes the excitatory value (i.e. ability to elicit fear) of a cue previously paired with a shock that had undergone extinction (i.e. no longer elicited fear). It was proposed that these phenomena occurs across species leaving trauma survivors who perceive the world to be globally dangerous vulnerable across contexts to trauma related cues (e.g., smells, sounds, sensations) triggering and consolidating memories (intrusions) of the trauma, which in turn reinforces the dangerous nature of the current environment and the need for avoidance and hyperarousal to detect and avoid threat. Risks associated with early psychological interventions (i.e. interventions within 72 hours of a traumatic event) were discussed in light of these findings.

Together, the papers that comprise this thesis offer an explanatory framework for the development and maintenance of posttraumatic symptoms following exposure to a life-threatening event. Consistent with the neurological data outlined in the introduction, the data contained in this thesis suggests that PTSD is underpinned by survival mechanisms. Following a life-threatening event survival mechanisms are maintained so that survival is enhanced in a dangerous environment. Specifically, this thesis identified that posttraumatic symptom clusters are a cross-species form of preparatory antipredator responses that are adopted after a life-threatening event. Interactions between trauma exposure (history and intensity), current perceived risk of life-threat, individual resources to negotiate threat and environmental refuge from threat determine the level and duration of intrusion, hyperarousal and avoidance symptoms adopted.

Papers five and six suggest that these symptom clusters are maintained in PTSD when exposure to a stressor of sufficient intensity embeds discrete features (e.g., smells, sounds, tactile sensations, objects, colours, emotions etc) associated with the traumatic event in a global excitatory context (i.e. there is no differentiation between safe and dangerous elements of the

global trauma context; all elements of the global context are fear eliciting). A failure to identify associations that predict safety maintains this global excitatory context and high levels of posttraumatic symptoms.

Based on the results it was proposed that a lack of differentiation from a global excitatory context results in retrieval of amygdala-driven trauma sequences across contexts. Retrieval of amygdala-driven trauma sequences across contexts (intrusions) confirms a global excitatory context, the ongoing presence of a life-threat and the ongoing need for vigilance to threat cues (hyperarousal symptoms) across contexts. If cues that predict the threat is nearby are detected, then rapid avoidance of threat is facilitated (avoidance symptoms). Successful avoidance of the feared life-threat increases the excitatory value of the trauma cues ensuring ongoing successful avoidance of the threat. If associations that predict safety are learned then posttraumatic symptoms are gradually inhibited in all contexts except those that contain specific associations that predict threat. If threat is not encountered upon exposure to contexts that contain specific associations that predict threat then new learning occurs and posttraumatic symptoms are inhibited in these contexts as well.

The above findings are not startling in light of existing knowledge. As outlined in the introduction existing theories of PTSD already propose a role for fear conditioning (Foa, Steketee and Rothbaum, 1989), memory processes (e.g., Brewin, Dagleish & Joseph (1996) and individual cognitive perceptions/beliefs regarding global threat (Foa and Rothbaum, 1998; Ehlers & Clark, 2000) in the development of PTSD. What this thesis does offer that is new is how these processes in the individual are connected to each other and why current treatments based on these theories are not beneficial to all PTSD sufferers. It also offers explanation for how the individual interacts with the environment to develop and maintain PTSD. In the following section, the knowledge outlined in the introduction (Part I) and the research knowledge gained from the six experimental papers (Part II), is expanded upon to address the second aim of the thesis, to develop a trauma theory with a conceptual framework for the future development of safe and effective interventions.

2. Proposed Ecological Trauma Theory

2.1 The development of posttraumatic intrusion, hyperarousal and avoidance symptoms

A healthy adult possesses a psychological boundary around the self that acts as a barrier between the self and the external environment (Cloud and Townsend, 1996). Exposure to a traumatic event of sufficient intensity can perforate this boundary. Figure 1 represents the hypothesised difference between a psychologically healthy adult and a recent trauma survivor.

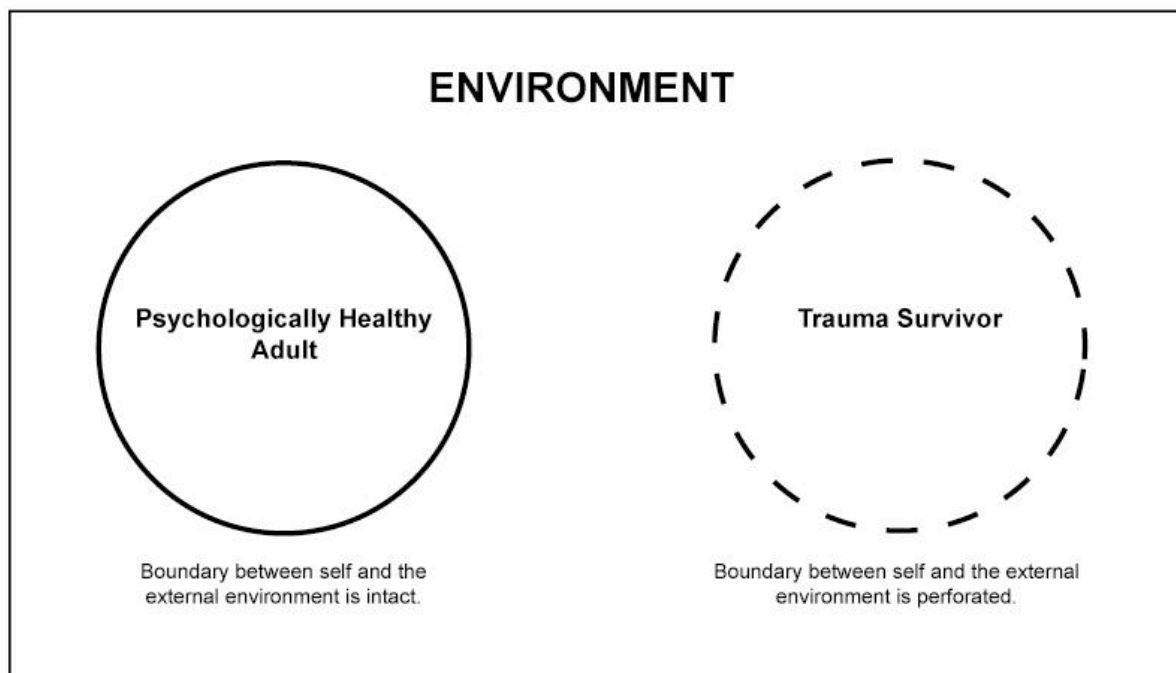


Figure 1. Representation of a psychological boundary between the self and the environment. Solid circle represents intact boundaries of a psychologically healthy adult who has not experienced recent trauma. Broken circle represents a recent trauma survivor with a perforated boundary.

An intact boundary enhances an individual's ability to deflect or filter irrelevant environmental information, maintain a stable internal state despite changes in the external environment and make self-determined optimal cortical driven responses to achieve personal goals. However, if a life-threat is still present, this mode of operating decreases survival chances. Critical environmental clues may not be detected due to filtering and defensive survival responses not initiated rapidly enough due to conscious evaluation and slower cortical-driven responses (refer to introduction pp. 19,20).

In contrast, perforation of this boundary following trauma exposure facilitates enhanced interaction between the self and the environment. The resulting interaction between the individual and the environment then determines optimal limbic and brainstem driven automatic intrusion, hyperarousal and avoidance survival responses. In the immediate aftermath of a life-threatening event the trauma survivor thus remains primed, ready to rapidly respond to environmental cues that predict the whereabouts of returning threat. Figure 2 represents the hypothesised interaction between the self and the environment in a psychologically healthy adult who has not undergone trauma and a recent trauma survivor.

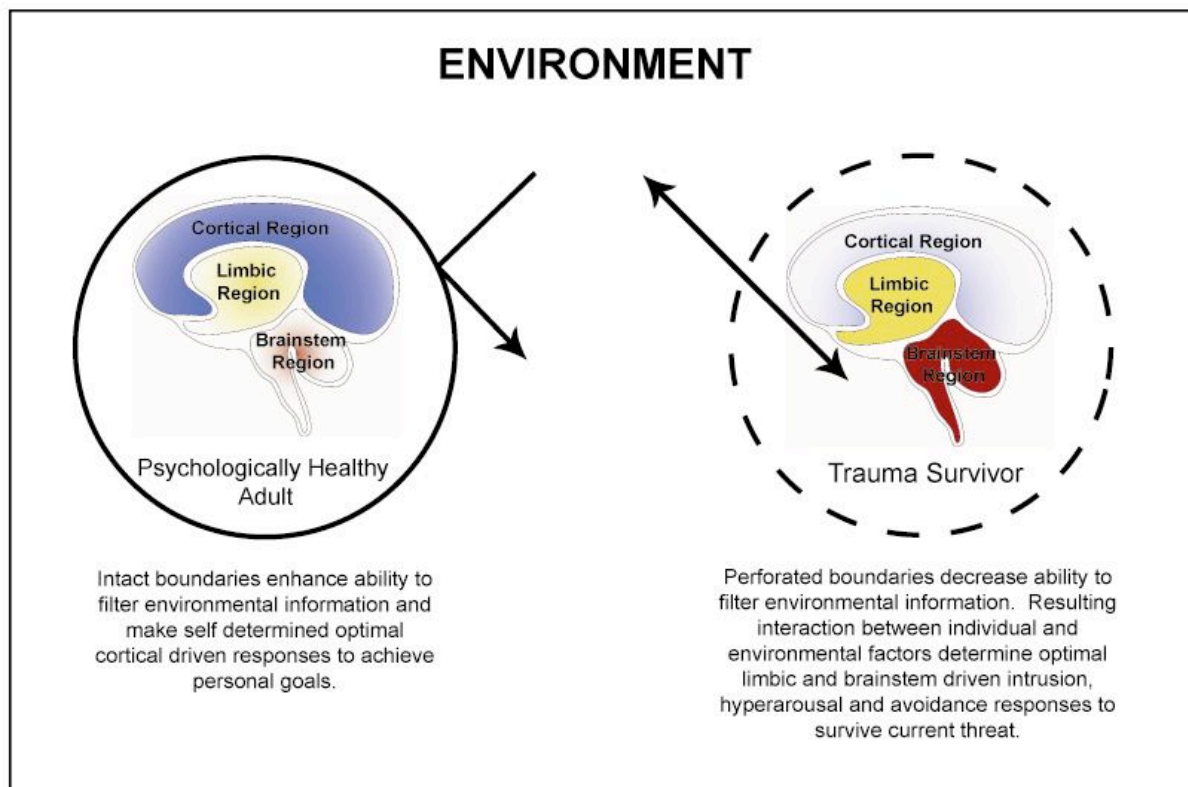


Figure 2. Representation of how the environment is hypothesised to interact with a psychologically healthy adult and a recent trauma survivor. Intact circle represents a psychologically healthy adult who has not undergone recent trauma. Broken circle represents a recent trauma survivor. Deeper colours in the brain represent dominant brain region activity. An intact boundary deflects environmental regulation. A perforated boundary facilitates increased environmental regulation of the individual.

The specific individual and environmental variables that interact to determine optimal levels of preparatory intrusion, hyperarousal and avoidance survival responses are exposure to a life-threatening event (experienced or witnessed), perceived current risk of life-threat, individual

resources to negotiate life-threat, and environmental refuge from life-threat. These four factors are cross-species forms of animal preparatory antipredator modulating variables as outlined by Kavaliers and Choleris (2001). In animals these variables are determined by concrete events. For example, past encounters with a predator, the prey animals escape record, access to physical shelter and modelling from conspecifics (Kavaliers & Choleris, 2001). In humans, due to higher order functioning, some aspects of these variables are also likely to be determined by individual idiosyncratic perceptions and beliefs (see De Houwer, Vandorpe & Beckers, 2005 for a discussion on human cognition processes and associative learning). This cognitive aspect of modulating variables may be why cognitive behaviour therapy (CBT), an intervention that challenges erroneous perceptions and beliefs has been shown to be efficacious in some PTSD cases (see van Etten & Taylor, 1998 and Harvey, Bryant & Tarrier, 2003 for reviews on the efficacy of CBT with PTSD).

Based on the ability of humans to engage in complex higher order functioning it is proposed that in humans the four modulators of intrusion, hyperarousal and avoidance responses are determined by the following factors. Exposure to life-threat is determined by the individuals' exposure history and the intensity of the exposure or exposures. Perceived current risk of life-threat is determined by the concrete presence of life-threats (distal or proximal) and/or perceived presence of life-threat derived from individual perceptions and beliefs regarding threat. Perceptions and beliefs regarding the current risk of life-threat may be constructed around past experiences, learning through others verbal communication or observing the fearful behaviour of others (see Delgado, Olsson & Phelps, 2006).

Individual resources are determined by available skills to negotiate threat (developmental, emotional, psychological, cognitive, spiritual, behavioural, physical), past learning regarding effectiveness of individual resources against threat and individual idiosyncratic perceptions and beliefs regarding one's resources (e.g., overestimating or underestimating individual resources). Environmental refuge is determined by current available geographical, social, emotional, psychological and spiritual refuge from threat, past experience regarding availability and effectiveness of refuge and individual idiosyncratic perceptions and beliefs regarding available refuge from threat (e.g., overestimating or underestimating available refuge).

The interaction between modulating variables then determines the level of intrusion, hyperarousal and avoidance symptoms that the trauma survivor will adopt following a life-

threatening event (experienced or witnessed). Figure 3 shows how the variables interact to produce the levels of intrusion, hyperarousal and avoidance responses adopted.

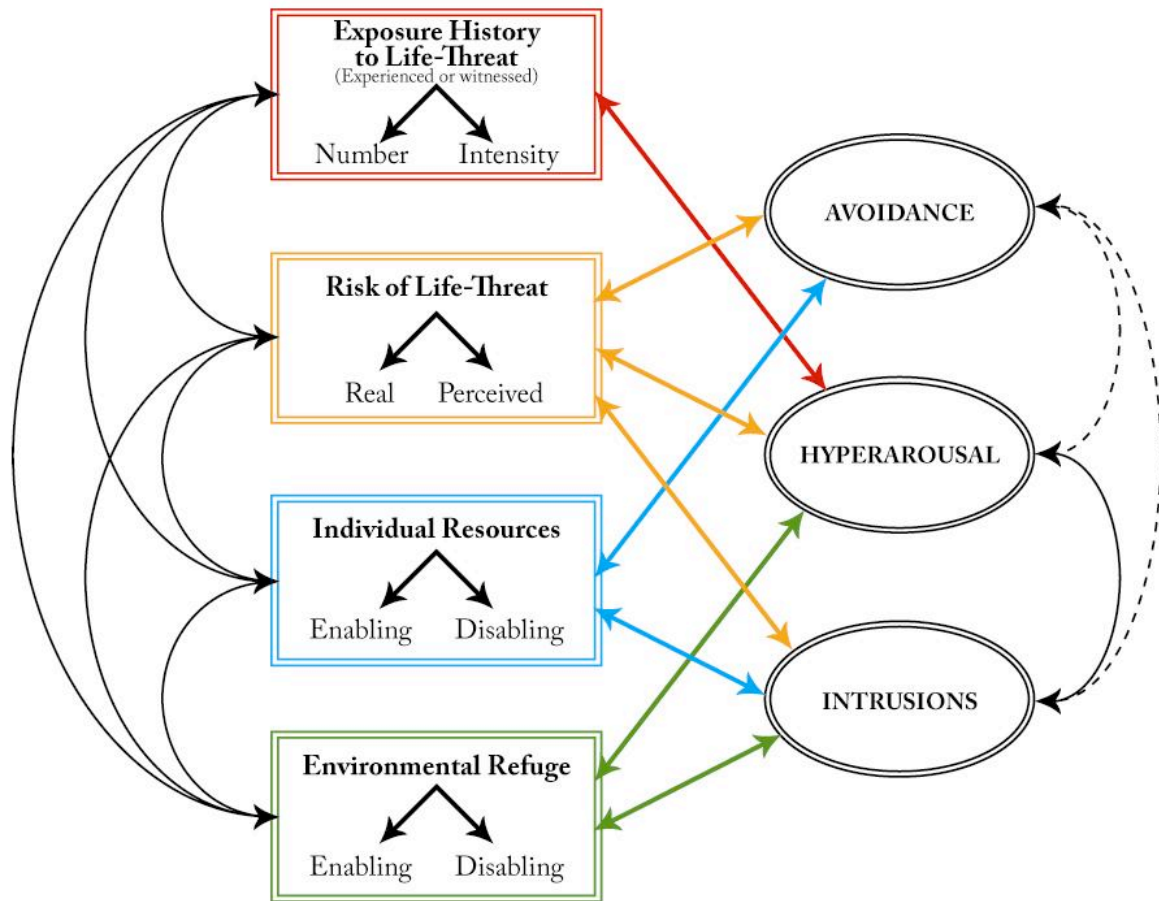


Figure 3. The ecological model of posttraumatic symptoms. In the immediate aftermath of a life-threatening event (experienced or witnessed) individual and environmental variables (real and perceived) interact to modulate the levels of posttraumatic avoidance, hyperarousal and intrusion symptom clusters adopted by the trauma survivor. Arrows represent ongoing dynamic interactions between variables, between symptoms, and between variables and symptoms. Broken arrows represent weaker interactions.

Based on the correlational data in chapter four, it is proposed that threat exposure, perceived current risk of life-threat and environmental refuge determines the level of hyperarousal adopted. The level of intrusion adopted is determined by perceived current risk of life-threat, individual resources to negotiate the threat and environmental refuge from threat. Intrusions and hyperarousal symptoms then interact to maintain an optimal state of vigilance to environmental cues that predict the whereabouts of potential threat. The level of avoidance adopted is determined by current perceived risk of life-threat and individual resources to negotiate threat. A

high risk of life-threat (real or perceived) and low individual resources to negotiate life-threat (real or perceived) results in a high level of avoidance to cues that predict the threat is nearby. Safe exposure to the feared threat results in reduced perceived risk, increased individual resources (e.g., mastery of fear, increased self efficacy), and a lower exposure ratio. The resulting interaction between the variables then determines the level of inhibition of posttraumatic symptoms. Thus identical exposure will not necessarily produce an identical reduction in posttraumatic symptom levels.

These modulating variables can either be enabling or disabling for the trauma survivor depending on the overall interaction between the variables. For example, individual resources that facilitate day-to-day functioning in the presence of threat are enabling only if they do not increase the level of exposure and risk of life-threat. An over reliance on individual resources may also be disabling if they limit or obscure access to available environmental refuge. Similarly, environmental refuge can be disabling if it limits or obscures available individual resources. For example, relying solely on significant others, military solutions, government or non-government organisations (NGO's), refugee and internally displaced peoples (IDP) camps for refuge may limit or obscure the trauma survivor's own resources and leave them disempowered to negotiate future threat.

A perceived high risk of life-threat in a dangerous context, although real, could be disabling if it paralyses the individual and increases the risk of exposure. In contrast, a perceived low risk of life-threat, although a misperception, may be enabling in a dangerous context if it facilitates the individual to function long enough to seek out refuge. Exposure to life-threat can also be enabling or disabling. In contrast to a disabling exposure that results in PTSD, a life-threatening experience of high intensity has been shown to trigger posttraumatic growth if the experience increases the individual's resources and affirms the availability of environmental refuge to the trauma survivor (see Linley, 2003; Linley & Joseph, 2003; Joseph & Linley, 2005; Cryder, Kilmer, Tedeschi & Calhoun, 2006 for a discussion on posttraumatic growth).

This enabling and disabling potential of modulating variables validates the concern expressed by many clinicians regarding the potential harm of some interventions following trauma (e.g., Raphael, 2000; Bisson, 2003; Devilly, Gist & Cotton, 2006; Devilly, Varker, Hansen, & Gist, 2007). Interventions could inadvertently alter modulating variables to have a disabling effect in

the trauma survivor's individual context. In order to develop safe interventions a more detailed understanding of how these variables interact to mitigate and maintain levels of posttraumatic symptoms in the weeks and months following a life-threatening event is required. Drawing on the data contained in this thesis and additional research knowledge, the underlining processes that determine whether posttraumatic symptoms are extinguished or maintained in the weeks and months following a life-threatening event are outlined below.

2.2 The extinction of posttraumatic hyperarousal, intrusion and avoidance symptoms

Exposure to a life-threatening event of high intensity initially embeds associated discrete trauma features (e.g., smells, sounds, tactile sensations, objects, colours, emotions etc) in a global context that is excitatory. Emotional responses are not contained and trauma cues have the ability to trigger amygdala-driven fear responses across contexts. Experiencing environmental refuge (e.g., support from family and/or community, participating in spiritual rituals and cultural practices, escape to a protected geographical space) provides a local context of safety. The experience of a local context of safety (i.e. refuge) provides a surrogate barrier between the self and the environment, containing emotional responses and differentiating the life-threatening event from a global excitatory context (see Figure 4).

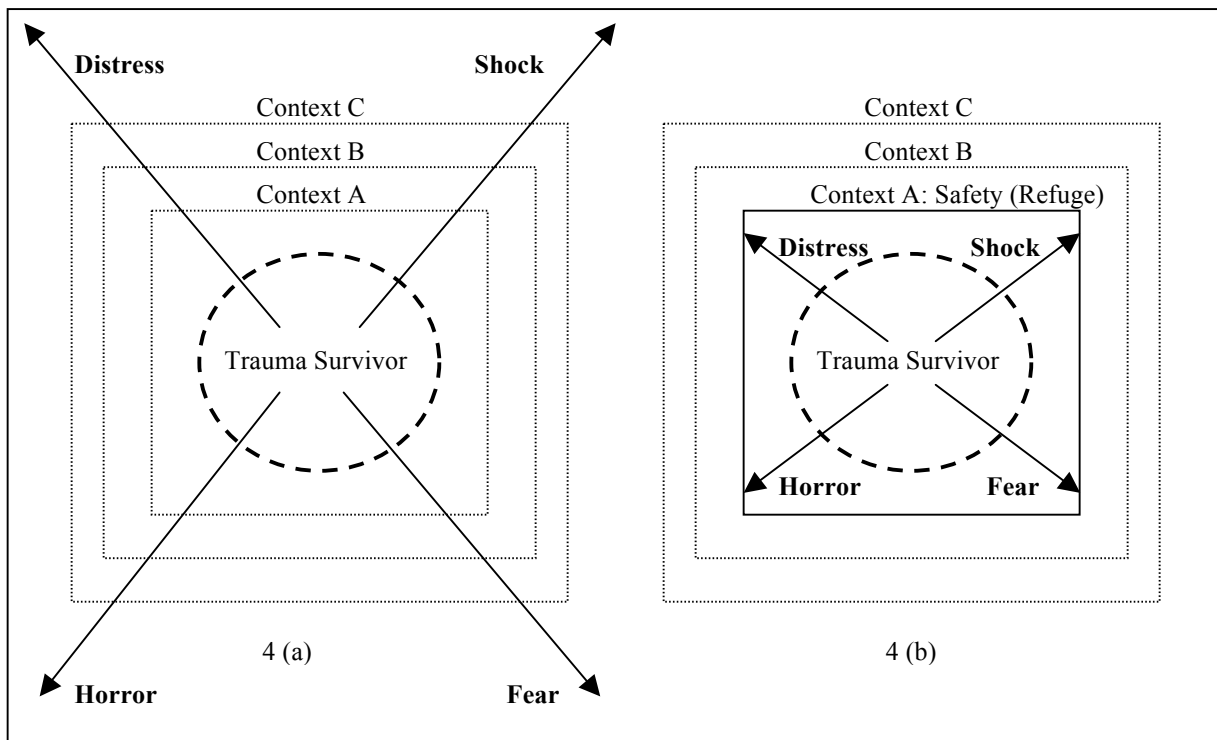


Figure 4. (a) Failure to experience a local context of safety (refuge) compromises containment of emotional responses and maintains a global excitatory context (b) Experiencing a local context of safety facilitates containment of emotional responses and differentiation from a global excitatory context.

An experience of safety (environmental refuge) facilitates the inhibition of symphathoexcitatory circuits and higher cortical processes to return to normal functioning (i.e. survival mechanisms are switched off). With restored cognitive functioning and a surrogate barrier between the self and the environment, perforations begin to heal and the ability of the individual to regulate their emotional world is restored. A return of higher cortical functioning also enhances conscious identification of associations that predict safety and threat (e.g., $A + B + C = \text{safety}$; $A + B - C = \text{threat}$). This differentiation between associations inhibits retrieval of an amygdala-driven global context - traumatic event sequence that underlies 're-living experiences'. For example, following an armed hold-up in an office building, learning associations of safety such as $A (\text{building}) + B (\text{man}) + C (\text{gun}) + D (\text{security uniform}) = \text{safety}$ and $A (\text{building}) + B (\text{man}) + C (\text{gun}) - D (\text{security uniform}) = \text{threat}$, differentiates these local contexts from a global excitatory context in which $A (\text{building}) = \text{threat}$, $B (\text{man}) = \text{threat}$, $C (\text{gun}) = \text{threat}$, across all contexts.

Identifying associations that predict safety (refuge) and threat (exposure) also increases individual resources and lowers perceived risk of life-threat, which in turn lowers avoidance of trauma related cues. Exposure to trauma related cues in the absence of threat then mitigates hyperarousal and intrusion symptoms further. Ongoing interactions between variables gradually inhibit posttraumatic symptoms. If threat still exists in a specific context then posttraumatic symptoms will be triggered in this specific context only.

If trauma cues are encountered in new contexts the individual is required to determine which learned associations (i.e. safety or threat) apply in 'this' context. This ability to differentiate between safety and threat in new contexts is facilitated by the hippocampus and the ventromedial prefrontal cortex working in concert (Milad et al. 2007). When trauma cues are encountered in a new context, these two brain structures work together to determine whether fear learning in context (1), or extinction learning in context (2) is the most appropriate learning for the current context (see Milad et al., 2007 for more details on this process). For example, context (1) $A + B + C + D = \text{safety}$ and context (2) $A + B + C - D = \text{threat}$ are evaluated. If context (1) is

determined to be more appropriate to the present context than context (2), then hippocampal-driven processing dominates and inhibits the ability of trauma cues to trigger amygdala-driven automatic survival responses in *'this'* context (see Corcoran, Desmond, Frey & Maren, 2005 for more details of the role of the hippocampus in context specific fear extinction). If context (2) is determined to be more appropriate to the present context, then amygdala-driven processing will dominate and trauma cues will trigger automatic survival responses in *'this'* context.

2.3 The maintenance of posttraumatic hyperarousal, intrusion & avoidance symptoms

The maintenance of posttraumatic hyperarousal, intrusion and avoidance symptoms across contexts and across time, as in PTSD, occurs when a global excitatory context is maintained. A global excitatory context is maintained by either a failure to experience environmental refuge (i.e. safety), or a failure to identify associations that predict safety. This failure results in ongoing disinhibition of sympathoexcitatory circuits, which in turn results in ongoing diminished cortical functioning and increased pre-attentive subcortical processes (Arnstein & Goldman-Rakic, 1998; Thayer & Brosschot, 2005). A failure to identify associations may then be further impaired by the reduced cortical functioning, or factors such as ambiguity, grief, depression, low intelligence quotient, pre-existing hippocampal damage or conflicting information introduced at debriefing procedure.

A global excitatory context that is maintained by a failure to identify associations that predict safety then results in discrete trauma features (e.g., smells, sounds, tactile sensations, objects, colours, emotions etc) being able to retrieve and consolidate amygdala-driven trauma sequences across all contexts. Retrieval of a trauma sequence by amygdala-driven processes, rather than hippocampal-driven processes, creates a re-living of the trauma rather than recall of a specific time-dependent memory. Amygdala-driven re-living adds to the excitatory value of discrete trauma features through a process known as sensitisation (see Pitman, Orr & Shalev, 1993; Siegmund & Wotjak, 2007), which in turn increases the excitatory value of the environment which then facilitates easier retrieval of amygdala-driven trauma sequences and so a debilitating cycle ensues.

Re-living across contexts, rather than context specific recall, then adds additional trauma exposures to the trauma survivors' exposure history, increases current perceived risk of life-

threat, lowers perceived individual resources to negotiate life-threat and lowers perceived environmental refuge from life-threat. As a result of these changes and ongoing interactions, the levels of intrusion, hyperarousal and avoidance responses increase. The increase in exposure, increase in perceived risk of life-threat and decrease in individual resources and environmental refuge gradually results in increased glucocorticoid receptor sensitivity, stronger negative feedback inhibition (see Yehuda, Golier, Yang & Tischler, 2004) and progressive sensitisation of the hypothalamic-pituitary-adrenal axis (see Yehuda, 1998), so that the individual's ability to respond to environmental stimuli and successfully avoid the increasing threat is enhanced (Yehuda, 1998). An increased ability to respond to environmental stimuli is also facilitated by a reduced pre-pulse inhibition (Grillon, Morgan, Southwick, Davis & Charney, 1996), diminished P50 sensory gating (Ghisolfi et al., 2004), and an exaggerated startle reflex (Grillon & Morgan, 1999).

The ongoing disinhibition of sympathoexcitatory circuits then results in secondary problems such as problems with verbal memory (Bustamante, Mellman, David & Fins, 2001), problems with working memory (Weber et al., 2005), difficulties encoding information, difficulties inhibiting irrelevant information (Cottencin et al., 2006; Vasterling et al, 2002), problems with overall executive functioning (Koso & Hansen, 2005; Kanagaratnam & Asbjørnsen, 2006), impaired learning (Saigh, Mroueh & Bremner, 1997; Scrimin, 2006), and increased risk of physical illness (Spiro, Hankin, Mansell, & Kazis, 2006; Lauterbach, Vora & Rakow, 2005).

As the disorder progresses, PTSD sufferers then begin to abuse alcohol and nicotine (Shipherd, Stafford & Tanner, 2005; Reynolds et al., 2005; Hapke et al., 2005; Koenan et al., 2006). A recent study by Feldner, Babson and Zvolensky (2007) identified that the greater the severity of the trauma exposure, the higher the risk of nicotine dependence. It may be that alcohol and nicotine are used as an attempt to dampen escalating levels of fear-related posttraumatic symptoms. A study by Curtin, Patrick, Lang, Cacioppo & Birbaumer (2001) offers some support for this hypothesis. They identified that alcohol effectively dampened anticipatory fear in undergraduate students. Unfortunately, alcohol and nicotine abuse only serves to maintain PTSD, as alcohol and nicotine impair the hippocampus (see Jatzko et al. 2006), which as mentioned previously, is an essential brain structure in differentiating between contexts that predict safety and threat.

3. The Ecological Trauma Theory and Current Research

3.1 Predictive and protective factors

The above ecological trauma theory is able to incorporate existing research knowledge and broaden our understanding of the findings in the research literature. Pre-trauma predictors, peri-trauma predictors, post-trauma predictors and protective factors associated with PTSD (highlighted in the introduction) are outlined in the first column in Tables 1, 2, 3 and 4. Their relationship to the proposed ecological trauma theory is outlined in the second column.

Table 1 (opposite page) suggests that pre-existing low individual resources, pre-existing low environmental refuge and prior trauma exposure leaves the individual vulnerable to PTSD following a life-threatening event. Prior trauma exposure would increase the number of trauma exposures and most likely increase the individual's perceived current risk of life-threat (i.e. the more traumas experienced the higher the perceived risk of current life-threat). However, pre-existing low individual resources and pre-existing low environmental refuge may be a significant predictor of PTSD only because they are predictive of low post-trauma individual resources and environmental refuge. This is an important issue for future prospective research studies.

Table 1. Pre-trauma predictors and their relationship to the ecological trauma theory.

Pre-trauma Predictors	Ecological Trauma Theory.
Prior psychological adjustment, (Ozer, Best, Lipsey & Weiss, 2003).	Low pre-existing individual resources (psychological)
Family history of psychopathology (Ozer, Best, Lipsey & Weiss, 2003).	Low environmental refuge pre-trauma (social, emotional, psychological). Pre-existing low individual resources as a result of poor modelling from family (due to psychopathology)
A harm-avoidance pre-trauma personality (Gil, 2005).	Limited individual resources to negotiate threat based on prior learning (i.e. avoidance dominant strategy used post-trauma). Avoidance increases excitatory value of trauma cues overtime (paper 6).
Prior trauma (Ozer, Best, Lipsey & Weiss, 2003).	High exposure (number).
Chronic environmental adversity (Koenen, Moffitt, Poulton, Martin & Caspi, 2006).	Low environmental refuge
Lower intelligence as measured by standardised measures of IQ (McNally & Shin; Vasterling et al., 2002; Koenen et al. 2006). After controlling for extent of combat exposure in Vietnam veterans, lower intelligence still emerged as a risk factor for PTSD (Macklin, 1998).	Low pre-existing individual resources (cognitive).
Low self-efficacy (Henrichs et al., 2005)	Low pre-existing individual resources (psychological).

The dominant peri-trauma predictor of PTSD in Table 2 is the intensity of the exposure to a life-threat. In animals the intensity of the adverse stressor or encounter with a predator triggers differential behavioural responses along an underlying continuum (Blanchard, Griebel & Blanchard, 2003). Fear-related responses are triggered at the upper end of the continuum and anxiety-related responses are triggered at the lower end of the continuum (Blanchard, 1993). It is likely given the similarity in modulating variables across species that a continuum of responses according to the intensity of the stressor also applies cross-species. A high intensity trauma exposure may therefore trigger high levels of fear-related posttraumatic responses. This is consistent with research that shows that the intensity of a stressor is encoded in the amygdala and that higher intensity threats trigger greater amygdala-driven fear learning (Rosen & Donley, 2006). Further, animal studies suggest that the higher the intensity of the exposure, the more likely that fear is generalised across contexts without differentiation (see Baldi, Lorenzini & Bucherelli, 2004). Thus a high intensity threat experience is more likely to result in the embedding of amygdala-driven trauma cues in a global excitatory context that regulates the individual's pre-attentive emotional and behavioural responses.

Table 2. Peri-trauma predictors and their relationship to the ecological trauma theory.

Peri-trauma Predictors	Modulating Variable
Peri-traumatic emotional responses and peri-traumatic dissociation (Harvey & Bryant, 2002; Ozer, Best, Lipsey & Weiss 2003). However, research by Holbrook Hoyt, Stein, and Sieber (2001) revealed that if perceived threat to life at the time of the trauma is taken into account, dissociation is not a significant predictor.	Exposure (high intensity).
Trauma severity (Bryant and Harvey, 1995; Brewin, Andrews & Valentine, 2000).	Exposure (high intensity).
Torture (Silove, Steel, McGorry & Mohan, 2002).	Exposure (repeated trauma and high intensity).
Higher life threatening trauma and trauma severity associated with chronicity of PTSD in Vietnam veterans (Roy-Byrne et al, 2004; Schnurr, Lunney & Sengupta, 2004).	Exposure (high intensity).
Threat exposure (Carr et al., 1997; Parslow, Jorm & Christensen, 2006)	Exposure (high intensity).
The extent of physical injuries sustained (Altindag, Ozen & Sir, 2005; Koren, Norman, Cohen, Berman & Klein, 2005).	Exposure (high intensity).
Loss or injury of a family member (Pfefferbaum, Nixon & Krug, 1999).	Exposure (high intensity) and perhaps loss of familiar refuge post trauma.
The experience of powerful emotions at the time of the trauma (Creamer, McFarlane & Burgess, 2005).	Powerful emotions are possibly a secondary effect of exposure to a high intensity trauma.
Fear of dying during traumatic event (Blanchard et al., 1997).	Exposure (high intensity).
Cognitive processing during trauma (assault) such as mental defeat, mental confusion and detachment (Dunmore, Ehlers, & Clark, 2001).	Low individual resources to negotiate exposure or a secondary effect of triggered survival mechanisms that diminish cognitive functioning (see Arnstein & Goldman-Rakic, 1998; Thayer & Brosschot, 2005)

Consistent with the Western research cited in Table 2, high intensity trauma exposure was also endorsed by the African community leaders (paper one) as one of the main contributors of the current high rates of PTSD in their people. They reported that current high rates of PTSD were due to the present war being different to other wars experienced. “Different = “*meaningless disfigurement, massacres and cruelty never witnessed before ... it makes no sense ... the sole purpose appears to be to terrorise inhabitants*” [statement by a Northern Ugandan woman that was endorsed by representatives from the Congo and Sudan]” (Dawson, 2005:106).

High intensity threats such as the massacres and other atrocities experienced and witnessed by these African communities (refer to paper 4) is also likely to reduce the possibility of

experiencing a local context of safety (i.e. environmental refuge) post-trauma. A large proportion of their existing refuges (i.e. political, military, spiritual, community, family etc) have been destroyed or severely compromised during the brutal attacks by rebel armies. They are also likely to feel powerless to negotiate the life-threat post-trauma (low individual resources). The witnessed overpowering strength and brutality of rebel armies with weapons compared to unarmed civilians lends itself to a sense of powerlessness should the threat (i.e. rebel armies) return. Further, these African communities are unlikely to trust that they can be safe in the future (i.e. increased current perceived risk) due to ongoing rebel presence.

Table 3. Post-trauma predictors and their relationship to the ecological trauma theory.

Post-trauma Predictors	Modulating Variable
Heart rate (HR) increased in car accident PTSD subjects when trauma specific cues were used. The severity of the accident and current symptomatology discriminated between PTSD subjects who did and did not respond with increased heart rate (Veazey, Blanchard, Hickling & Buckley, 2004).	Severity of accident = high exposure (intensity) High exposure increases likelihood of a global excitatory context. A global excitatory context allows retrieval of amygdala-driven trauma sequences over hippocampal driven trauma sequences upon presentation of trauma specific cues and by implication increased HR due to re-living rather than remembering experiences.
Hyperarousal in the immediate aftermath of trauma (Ginzberg et al, 2003; Kutz & Dekel, 2006). A prospective study by Schell, Marshall, and Jaycox (2004) with survivors of community violence found that hyperarousal was the predominant predictor of the subsequent severity of all other symptoms and poor recovery.	Exposure is the dominant peri-trauma predictor. Hyperarousal symptoms are the only posttraumatic symptom cluster correlated with exposure (paper three).
Initial PTSD symptom severity (Blanchard et al., 2003).	High exposure, high perceived risk, low individual resources and low refuge predict high symptom levels (paper 3)
After controlling for initial symptoms, trauma narrative disorganisation (repetition, non-consecutive chunks, and coherence) at one-week post trauma predicted trauma severity at 3 months post trauma (Jones, Harvey & Brewin, 2006).	Low individual resources (cognitive) to identify associations that predict safety and threat or a secondary effect of ongoing disinhibition of sympathoexcitatory circuits.
Emotion-based ex-consequencia reasoning (e.g., intrusions trigger anxiety and subject concludes “I feel anxious therefore there must be danger”) predicted both acute and chronic PTSD (Engelhard & Arntz, 2005; Engelhard van den Hout, Arntz & McNally 2002).	Low individual resources (emotional, cognitive) maintains a high perceived current risk of life-threat
Ongoing dissociation (Halligan & Yehuda, 2002)	Disabling individual resources
Lack of post trauma social support, (Ozer, Best, Lipsey & Weiss, 2003; Brewin, Andrews & Valentine, 2000).	Low environmental refuge (social).

Table 3. (Continued) Post-trauma predictors and their relationship to the ecological trauma theory.

Post-trauma Predictors	Modulating Variable
Avoidance behaviour post-trauma (Zoellner, Jaycox, Watlington & Foa, 2003; Marz & Sloan, 2005).	High levels of avoidance correlated with high levels of current perceived risk of life-threat and low levels of perceived individual resources (paper 3). Avoidance increases excitatory value of trauma cues overtime (paper 6).
Characteristics of intrusions such as perceived 'nowness', vividness of the memories, and level of distress are predictive of persistent PTSD (Speckens et al., 2006).	Retrieval of amygdala-driven trauma sequences (i.e. re-living not remembering) indicates maintenance of a global excitatory context and an increase in trauma exposures.
Emotional numbing post-trauma predicts pervasive PTSD symptom disturbance (Breslau, Reboussin, Anthony & Storr, 2005), chronic PTSD (Marshall et al., 2006) and comorbid major depression (Kashdan, Elhai & Frueh, 2006).	Disabling individual resources.
Lack of post trauma social support, (Ozer, Best, Lipsey & Weiss, 2003; Brewin, Andrews & Valentine, 2000).	Low environmental refuge (social).
Ongoing chronic system stress such as poverty, discrimination, poor social location, and social inequalities related to race, gender class (Kubiak, 2005).	Low environmental refuge (economic, emotional, geographical, structural).
Additional life stressors such as loss of job, serious illness, death or illness of close friends/family, broken relationships (Brewin, Andrews & Valentine, 2000; Maes, Mylle, Delmeire & Janca, 2001; Mayou, Ehlers & Bryant, 2002).	Additional exposures and low environmental refuge (economic, emotional).
Persistence of traumatic injury (Blanchard et al, 1997). Severity of injury (Schnyder, Moergeli, Klaghofer, & Buddberg, 2001).	Ongoing exposure and low individual resources (physical).
Negative interpretations of symptoms such as dissociation, anger and intrusive memories (Ehlers, Mayou & Bryant, 2003; Mayou, Ehlers & Bryant, 2002).	Low individual resources
Negative beliefs about self and world (Dunmore, Ehlers, Clark, 2001).	Low perceived individual resources and high perceived current risk of life-threat.
Lack of resilience, mastery and life satisfaction (Heilemann, Kury & Lee, 2005).	Low individual resources (emotional, psychological and spiritual).

Table 3 highlights the significant role of preparatory antipredator modulating variables in the maintenance of PTSD post-trauma. In contrast to the dominant role of one modulator (exposure) during the traumatic event, all four modulating variables feature in the existing research on post-trauma predictors. This offers explanation for why post-trauma variables were identified by

Schnurr et al. (2004) and Maes, Mylle, Delmeire & Janca, (2001) as contributing more than pre-trauma and post-trauma variables, all modulating variables are involved post-trauma.

Table 4 highlights the protective effect of access to environmental refuge and the protective effect of individual resources against the development of PTSD. Sadly, in circumstances such as that reported by many of the African communities researched in this thesis, loss of family, friends, communities, religious practices, placement in a displaced peoples camp (IDP camp) and ongoing war has left the individual dependent on outside aid (low individual resources) and open to regulation by a malevolent environment (low environmental refuge).

Table 4. Protective factors and their relationship to the ecological trauma theory.

Protective Factors	Modulating Variable
Social/emotional support following trauma (Joseph, 1999; Ozer et al, 2003; Vranceanu, Hobfoll & Johnson, 2007)	High environmental refuge (social, emotional).
Self-esteem and appraisal support following trauma (Hyman, Gold & Cott, 2003)	Individual resources are reinforced (emotional, psychological).
High sense of coherence (SOC). SOC refers to the ability to perceive a traumatic event as comprehensible, manageable and meaningful (Engelhard, vanden Hout & Vlaeyen, 2003).	High perceived individual resources (cognitive, emotional and spiritual).
Higher education associated with lower risk of PTSD (Schnurr, Lunney & Sengupta, 2004).	High individual resources (cognitive).
Higher intelligence quotient (IQ) was a predictor of resilience against PTSD in children and adolescents trauma survivors (Silva et al., 2000)	High individual resources (cognitive).
Personal hardiness and functional social support following trauma (King, King, Foy, Keane & Fairbank, 1999).	High individual resources (emotional) and high environmental refuge (social).
Supportive social networks, individualised care, community-grieving customs, rituals, shared community pain (paper 3).	High environmental refuge (social, emotional, psychological, spiritual).
Secure attachments (Fraley, Fazzari, Bonanno & Dekel, 2006)	High environmental refuge (social, emotional).

In summary, it would appear from the existing research literature on predictors of PTSD that low individual resources (cognitive and psychological), low environmental refuge (psychological) and exposure (history) are pre-existing vulnerabilities to develop PTSD following a life-threatening event. Correspondingly, protective factors are high individual resources (emotional, psychological, cognitive, spiritual) and high environmental refuge (social, emotional, psychological). The research suggests that high peri-trauma exposure levels

(intensity) underlie the development of PTSD. Subsequent exposures, high perceived/real current risk of life-threat, low perceived/real individual resources and low perceived/real environmental refuge then maintains PTSD.

The ecological trauma theory proposes that high intensity exposure is a critical determinant in the development of PTSD because a high intensity exposure to a life-threatening event initially embeds all trauma related cues in a global context that is excitatory (i.e. all elements of a global context have the ability to elicit fear). If a local context of safety is not experienced or associations that predict safety are not identified post-trauma then a global excitatory context is maintained. The maintenance of a global excitatory context then results in a pattern of interactions between modulating variables that maintains and even increases posttraumatic avoidance, intrusion and hyperarousal symptoms over time and across contexts.

3.2 PTSD and Comorbid Disorders

The ecological trauma theory also offers explanation for comorbid disorders such as borderline personality disorder, anxiety disorders and major depression identified in the introduction.

3.2.1 PTSD and Borderline Personality Disorder

Individuals with borderline personality disorder (BPD) appear to be regulated by environmental variables. They experience a shifting sense of self, emotional dysregulation, and abandonment fears to environmental triggers (Linehan, 1993). It may be that individuals with PTSD and co-morbid BPD have a greater perforation of the barrier between self and the environment due to greater exposure (history and/or intensity) and are therefore more vulnerable to environmental regulation over self-regulation. There is some evidence for this assertion in that high rates of individuals with BPD have experienced significant trauma in their lives (Yen et al., 2002; Golier et al., 2003). An invalidating environment has also been identified as a critical variable in the development of PTSD (Linehan, 1993). Thus high exposure coupled with a pre-trauma and post-trauma history of low environmental refuge may result in PTSD with comorbid BPD.

3.2.2 PTSD and Anxiety Disorders

Anxiety is marked by symptoms similar to that seen in PTSD. For example anxious individuals exhibit heightened vigilance to threat cues, intrusive thoughts and avoidance behaviours (Lau & Viding, 2006). It could be that the modulating variables of posttraumatic symptom clusters also determine the comorbidity of specific anxiety disorders. For example, failure to clearly identify the encountered life-threat may result in the individual consciously worrying about a range of potential threats as in generalised anxiety disorder. A child who perceives they have low individual resources may seek environmental refuge in a parent resulting in separation anxiety. Specific phobias may develop as a result of discrete cues/objects developing excitatory properties from an excitatory context as demonstrated in the animal experiments conducted as part of this thesis. Panic attacks may result from the presence of cues that predict the encountered life-threat is proximal.

The other explanation for why anxiety has a high comorbidity with PTSD is that anxiety may serve a preparatory survival function when threat is perceived to be distal so that a rapid switch to automatic survival responses is facilitated should the threat become proximal. There is some evidence for this hypothesis in that unconscious automatic survival responses such as evidenced in PTSD have also been shown to trigger more rapidly in high-anxious individuals compared to low-anxious individuals (see Bishop, Duncan & Lawrence, 2004). Similarly, a high level of anxiety in the home environment was identified as a significant predictor of PTSD in Palestinian school-aged children (Khamis, 2005). It may be that following a life-threatening event anxiety maintains the individual in a state of readiness so that there is minimal delay for switching to defensive survival responses.

3.2.3 PTSD and Major Depression

Major depression has been linked to learned helplessness; a learned response or belief that there is nothing one can do to change the current aversive circumstances (Sondaite & Zukauskienė, 2005; Day, Kane & Roberts, 2003). It may be that individuals with PTSD who have low perceived individual resources also develop major depression.

Thus high exposure (history and/or intensity) with low refuge (pre-trauma and post-trauma) may contribute to comorbid BPD, perceived non-discrete distal threat may contribute to comorbid anxiety disorders, and low perceived individual resources may contribute to comorbid

major depression. This possibility is an important focus for future research as individualised treatments that target specific modulating variables may be required for effective treatment of PTSD sufferers with comorbid disorders. General implications of the ecological trauma model for safe interventions are discussed in the next section.

4. Implications for Safe Interventions

The ecological trauma theory offers a theoretical framework for why psychological interventions that are implemented in the immediate aftermath of trauma appear to increase the risk of PTSD. Psychological debriefing procedures in the immediate aftermath of a major trauma are contraindicated within the ecological trauma theory because of the likelihood that the environment is still perceived as dangerous by a large percentage of survivors. For example, survivors of a tsunami, earthquake, rape or terrorists attacks are likely to have their assumptions of safety shattered by such an overwhelming experience and view the world as globally dangerous initially (see Janoff-Bulman, 1992 for discussion of shattered assumptions of safety following trauma). The introduction of trauma cues in an environment still perceived as dangerous could trigger retrieval and consolidation of amygdala-driven trauma sequences from the hippocampus, which in turn confirms the excitatory value of a global context. Access to additional information through group debriefing procedures may also clash with associations formed through individual experience and confuse the learning of associations that predict safety and threat. Previously neutral cues could also become excitatory through backward fear conditioning if presented in a context still perceived as dangerous, adding additional cues capable of triggering an amygdala-driven trauma sequence from the hippocampus.

Early interventions that assist in the identification of associations that predict safety would be the intervention of choice so that differentiation from a global excitatory context is promoted and retrieval of amygdala-driven trauma sequences avoided. Facilitating access to environmental refuge, such as family and friends, religious community and rituals, cultural practices, familiar objects and places, or a protective presence if the threat is still present could encourage identifying associations that predict safety. Interventions that focus on strengthening individual resources may also provide a safe intervention in the immediate aftermath of trauma. For example, education programmes that teach skills to differentiate self and others, differentiate self and the environment (i.e. repair trauma-induced perforations in the barrier between self and the

environment), promote conscious internal regulation of self and develop strategies/tasks that enhance safety and autonomy. Skills such as these are more likely to promote recovery in the immediate aftermath of trauma than exposure therapies that focus on trauma cues and risk retrieval of amygdala-driven trauma sequences.

As noted in the introduction, empirical evidence suggests that exposure and cognitive behavioural interventions are the most efficacious therapies in treating PTSD (see Rothbaum, Meadows, Resick & Foy, 2000 for a review of these therapies). However, it was also noted that these therapies are efficacious only in a sub set of PTSD sufferers. The ecological trauma theory offers a framework to differentiate between individuals who may benefit from these interventions and individuals who may not benefit initially. It also offers a framework to determine at what point in therapy to introduce exposure elements and cognitive challenging. For example, if the individual perceives a global excitatory context then exposure therapy would be contraindicated, as it would have the potential to trigger retrieval of amygdala-driven trauma sequences and inadvertently increase exposure levels. Providing a local context of safety and then assisting in the identification of associations that predict safety should be the initial preferred treatment in these individuals.

Cognitive behaviour therapy may also be ineffective if a global excitatory context is perceived due to diminished higher executive functioning and increased environmental regulation of limbic and brainstem regions. Again differentiation from a global excitatory context should be the focus of treatment in these individuals initially. Once differentiation has occurred through experiencing a local context of safety then inhibition of sympathoexcitatory circuits should occur. This inhibition restores cortical functioning (Thayer & Brosschot, 2005) which in turn enhances the individual's ability to identify associations that predict safety and threat, challenge any misperceptions and disabling beliefs associated with modulating variables (i.e. engage in CBT).

The following are brief treatment guidelines for safe trauma interventions indicated by the proposed ecological trauma theory.

1) Assessment of contextual fear (i.e. global or context specific).

If global contextual fear is assessed (i.e. all contexts are perceived as dangerous), then focus on developing and identifying associations that predict a local context of safety (real and imaginal) before discussing any trauma content so as to avoid retrieval of amygdala-driven trauma sequences (i.e. re-living).

2) Assessment of current perceived proximity of life-threat.

Obtain a subjective rating of current perceived risk of life-threat (e.g., 5-point Likert scale 0 = no threat; 1 = a little threat; 2 = moderate threat; 3 = large threat; 4 = extreme threat). If perceived risk of life-threat is high, then avoid exposure therapy until perceived risk is reduced. Identify feared life-threat (e.g., car accident, cancer, rebels). If perception of threat is real then increase individual and environmental resources to negotiate threat. If perception of threat does not appear to be based in reality for the current context, apply cognitive-behavioural therapy to challenge the erroneous perception. When perceived life-threat is reduced, and individual resources and environmental refuge is at a positive level introduce exposure therapy to promote new learning.

3) Assessment of trauma exposure: (a) obtain a trauma history (i.e. single trauma or multiple traumas experienced) and (b) intensity of trauma.

Increase individual and environmental resources to reduce future trauma exposures. If a trauma survivor has been exposed to childhood abuse (present or past) then individual resources to avoid future exposures and ability to utilise current environmental refuge may be severely compromised (see Gold, 2000). If real/perceived risk of life-threat has been reduced, individual resources are effective and environmental refuge is available, introduce safe exposure to trauma cues. Multiple safe exposures promotes relearning and extinction of fear behaviours by increasing the ratio of safe exposures to threatening exposures. Increasing exposure to positive experiences may also positively impact exposure history.

4) Assessment of pre-trauma functioning and current individual resources (cognitive, psychological, emotional, behavioural, spiritual).

Obtain a subjective rating of individual resources to negotiate feared life-threat and manage trauma symptoms (e.g., 5-point Likert scale 0 = no resources; 1 = a little resources; 2 = moderate

resources; 3 = large resources; 4 = abundant resources). If a low rating is distorted, challenge with cognitive-behavioural therapy. Strengthen existing enabling resources and challenge any existing disabling resources (e.g., dissociation, avoidance, isolation, drugs, alcohol). If a low rating is real, increase the individual's skills and strategies to negotiate the threat. (Note: Self-regulatory processes have been shown to defend against the threat of death, Gailliot, Scheichel & Baumeister, 2006). For example:

Emotional: Teach emotional regulation and grounding skills to increase control of trauma response. Increase positive emotions to decrease distress and promote the maintenance of refuge from significant people (see Bonanno, 2004 for benefits of promoting positive emotions following trauma).

Psychological: Assess for any comorbid psychological disorders and include in treatment plan.

Cognitive: Increase resources to identify associations that predict safety and threat. Identify cues that trigger posttraumatic symptoms (after emotional regulation skills have been taught). Teach thought challenging and strategic thinking.

Behavioural: Identify disabling behavioural strategies (e.g., avoidance, anger, alcohol, drugs) and challenge. Teach enabling behavioural strategies that will empower and promote autonomy.

Physical: Include physical strengths in assessment to increase sense of competency. Identify any physical injuries or limitations that may impact individual resources and refer to medical specialist if needed.

Developmental: Identify any developmental issues that may impact individual resources (e.g., developmental age of trauma survivor, poor development due to exposure history) and address in therapy.

Spiritual: Loss of faith or beliefs challenged due to trauma may need to be addressed in therapy or with spiritual leader.

5) Assessment of available environmental refuge (geographical, social, emotional, psychological, economic, spiritual).

Obtain a subjective rating of environmental refuge (e.g., 5-point Likert scale 0 = no refuge; 1 = a little refuge; 2 = moderate refuge; 3 = large refuge; 4 = abundant refuge). Identify specific areas of environmental refuge compromised. For example, are they living in a geographically

dangerous context, do they have a social network, have they lost significant family members or carers that would usually provide emotional and psychological refuge?

Increase access to the specific environmental resources compromised (e.g., family, friends, culturally appropriate resources, interpreters, government agencies, charities, refuges, police, spiritual community, cultural practices, familiar safe objects, places, people etc). Ensure therapist is skilled so that trust in environmental refuge is enhanced (i.e. ensure therapist is not easily overwhelmed, empowers rather than rescues, can avoid triggering re-living experiences). Providing education and skills to the trauma survivor's carers/support networks may also be beneficial to ensure they are an enabling refuge rather than a disabling refuge. For example, educate carers/support networks how to coach and help the trauma survivor contain/manage symptoms without diminishing trauma survivor's own resources or independence. If the rating given for environmental refuge appears distorted, then challenge with cognitive behavioural therapy.

6) Development of individualised treatment plans.

Even if the same trauma is experienced, every trauma survivor is likely to have a unique interaction of trauma exposures (number and intensity), current perceived risk of life-threat, perceived/real individual resources to negotiate threat and perceived/real environmental refuge from threat. Cultural factors should also be sensitively considered before psychological interventions are used to modify modulating variables. For example, in paper one, interventions shared by African rural community leaders that positively modulated posttraumatic symptoms were carried out in the context of local community.

5. Limitations of the Research

A major limitation of this study is that in the absence of clinical assessments or additional measures, PTSD cannot be assumed in the individuals in this study. However, the information gained from this thesis still adds valuable information to our understanding of posttraumatic responses as evidenced in PTSD. Another limitation is that the correlational design of the human studies does not allow direct tests of causality. Formal testing of the four modulating variables identified in this thesis and formal testing of the ecological trauma theory has yet to be conducted. Future research that utilises a prospective and/or longitudinal design will be

important to validate the modulators and the complex interactions between modulators and posttraumatic symptoms.

Caution should also be exercised in generalising the findings across all trauma populations, as the human subjects in this thesis were all trauma survivors from large-scale disasters of high intensity. Individual trauma that is not experienced as part of a large-scale disaster may produce a different pattern of responses. Caution should also be exercised with inferences drawn from the animal studies to the development and maintenance of PTSD in humans. Whilst contextual and cued fear in animals may mirror some aspects of PTSD, it cannot be equated to more complex aspects of PTSD evidenced in humans.

6. Future Research

The findings from the experiments conducted in this thesis open pathways for future research. They offer a theoretical framework for the development and maintenance of PTSD that can be formally tested by investigating predictive relationships between modulating variables and posttraumatic symptoms. Randomised controlled clinical studies can also be conducted that target specific modulating variables to ascertain the contribution of each variable and the most effective interventions for targeted variables.

Given that posttraumatic symptoms appear to be a cross-species form of animal survival responses, investigation into the neurobiology underlying survival responses in animals may offer valuable insight into the neurobiology of PTSD. For example, preliminary results from neurochemical experiments initially conducted as part of this thesis suggest that dopamine autoreceptor functioning is enhanced in rats that fail to differentiate a local context of safety from a global excitatory context. Further exploration of this preliminary finding and other neurochemical changes associated with animal survival responses may open pharmacological interventions for PTSD in humans.

The similarity between posttraumatic intrusion, hyperarousal and avoidance symptoms in humans and animal preparatory survival responses also suggests that investigation into defensive survival responses in animals beyond that studied in this thesis (i.e. freezing behaviour in rats) may provide valuable information. For example, risk assessment is a less fearful form of vigilance behaviour in animals than freezing and has been likened to anxiety in humans

(Blanchard, 1997; Blanchard, Hynd, Minke, Minemoto, & Blanchard, 2001). Whereas freezing is triggered by high intensity threat and low ambiguity, risk assessment is triggered by low intensity threat and high ambiguity. Therefore, it may be that the modulating variables investigated in this thesis operate across anxiety disorders with exposure to threat (number and intensity) being the critical variable between disorders marked by fear such as PTSD and disorders such as generalised anxiety disorder that is marked by constant worry (i.e. risk assessment). Future research conducted within the ecological trauma model may therefore not only provide a greater understanding into PTSD and safer trauma interventions but also offer understanding into a range of other anxiety disorders.

7. References

- Altindag, A. Ozen, S., Sir, A. (2005). One-year follow-up study of posttraumatic stress disorder among earthquake survivors in Turkey. *Comprehensive Psychiatry*, 46, 328-333.
- Arnstein, A. & Goldman-Rakic, P. (1998). Noise stress impairs prefrontal cortical cognitive function in monkeys. *Archives of General Psychiatry*, 55, 362-366.
- Baldi, E., Lorenzini, C. & Bucherelli, C. (2004). Footshock intensity and generalisation in contextual and auditory-cued fear conditioning in the rat. *Neurobiology of Learning and Memory*, 81, 162-166.
- Bishop, S., Duncan, J. & Lawrence, A. (2004). State anxiety modulation of the amygdala response to unattended threat-related stimuli. *The Journal of Neuroscience*, 24, 10364-10368.
- Bisson, J. (2003). Single-session early psychological interventions following traumatic events. *Clinical Psychology Review*, 23, 481-499.
- Blanchard, C. (1997). Stimulus, environmental, and pharmacological control of defensive behaviors. In M. Bouton & M. Fanselow (Eds.). *Learning, Motivation and Cognition*. (pp.283-303). Washington: American Psychological Association.
- Blanchard, C., Hynd, A., Minke, K., Minemoto, T., & Blanchard, R. (2001). Human defensive behaviors to threat scenarios show parallels to fear and anxiety-related defense patterns of non-human mammals. *Neuroscience & Biobehavioral Reviews*, 25, 761-770.
- Blanchard, C., Hynd, A., Minke, K., Minemoto, T., & Blanchard, R. (2001). Human defensive behaviors to threat scenarios show parallels to fear and anxiety-related defense patterns of non-human mammals. *Neuroscience & Biobehavioral Reviews*, 25, 761-770.
- Blanchard, D., Griebel, G., & Blanchard, R. (2003). The mouse defense test battery: pharmacological and behavioural assays for anxiety and panic. *European Journal of Pharmacology*, 463, 97-116.
- Blanchard, E., Hickling, E., Forneris, C., Taylor, A., Buckley, T., Loss, W. et al., (1997). Prediction of remission of acute posttraumatic stress disorder in motor vehicle accident victims. *Journal of Traumatic Stress*, 10, 215-234.

- Blanchard, E., Hickling, E., Malta, L., Jaccard, J., Devineni, T., Veazey, C., et al., (2003) Prediction of response to psychological treatment among motor vehicle accident survivors with PTSD. *Behavior Therapy*, 34, 351-363.
- Blanchard, R.J, Shepherd, J.K, Rodgers, R.J., Magee, L., Blanchard, D.C. (1993). Attenuation of antipredator defensive behaviour in rats following chronic treatment with imipramine. *Psychopharmacology*, 110, 245-253.
- Bonanno, G. (2004). Loss, trauma and human resilience. *American Psychologist*, 59, 20-28.
- Breslau, N., Reboussin, B., Anthony, J. & Storr, C. (2005). The structure of posttraumatic stress disorder. Latent class analysis in 2 community samples. *Archives of General Psychiatry*, 62, 1343-1351.
- Brewin, C., Andrews, B. & Valentine, J. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting Clinical Psychology*, 68, 748-766.
- Brewin, C.R., Dagleish, T., & Joseph, S. (1996). A dual representation theory of posttraumatic stress disorder. *Psychological Review*, 103, 670-686.
- Broman-Fulks, J., Ruggiero, K., Green, B., Kilpatrick, D., Danielson, C., Resnick, H., et al., Taxometric Investigation of PTSD: Data From Two Nationally Representative Samples. *Behavior Therapy*, 37, 364-380.
- Bryant R. & Harvey, A. (1995). Posttraumatic stress in volunteer firefighters: Predictors of distress. *Journal of Nervous and Mental disorders*, 183, 267-271.
- Bustamante, V., Mellman, T., David, D. & Fins, A. (2001). Cognitive functioning and the early development of PTSD. *Journal of Traumatic Stress*, 14, 791-797.
- Calhoun, L., Cann, A., Tedeschi, R & McMillan, J. (2000). A correlational test of the relationship between posttraumatic growth, religion, and cognitive processing. *Journal of Traumatic Stress*, 13, 521-527.
- Carr, V., Lewin T., Webster, R. & Kenardy, J. (1997). A synthesis of the findings from the quake impact study: a two-year investigation of the psychological sequela of the 1989 Newcastle earthquake. *Social Psychiatry & Psychiatric Epidemiology*, 32:123-136.

- Cloud, H. & Townsend, J. (1992). *Boundaries*. Grand Rapids, Michigan: Zondervan.
- Corcoran, K., Desmond, T., Frey, K. & Maren, S. (2005). Hippocampal inactivation disrupts the acquisition and contextual encoding of fear extinction. *The Journal of Neuroscience*, 25, 8978-8987.
- Cottencin, O., Vaiva, G., Huron, C., Devos, P., Ducrocq, F., Jouvent, R., Goudemand, M. & Thomas, P. (2006). Directed forgetting in PTSD: A comparative study versus normal controls. *Journal of Psychiatric Research*, 40, 70-80.
- Creamer, M., McFarlane, A. & Burgess, P. (2005). Psychopathology following trauma: The role of subjective experience. *Journal of Affective Disorders*, 86, 175-182.
- Cryder, C., Kilmer, R., Tedeschi, R. & Calhoun, L. (2006). An exploratory study of posttraumatic growth in children following a natural disaster. *American Journal of Orthopsychiatry*, 76, 65-69.
- Curtin, J., Patrick, C., Lang, A., Cacioppo, J. & Birbaumer, N. (2001). Alcohol affects emotion through cognition. *Psychological Science*, 12, 527-531.
- Dawson, J. (2005). African conceptualisations of posttraumatic stress disorder and the impact of introducing western concepts. *Psychology, Psychiatry, and Mental Health Monographs*, 2, 101-112. Sydney: New South Wales Institute of Psychiatry.
- Day, C., Kane, R. & Roberts, C. (2003). The prevention of depressive symptoms in rural Australian women. *Journal of Community & Applied Social Psychology*. 13, 1-14.
- De Houwer, J., Vandorpe, S. & Beckers, T. (2005). On the role of controlled cognitive processes in human associative learning. In A. Wills (Ed.). *New Directions in Human Associative Learning*. Mahwah, NJ: Lawrence Erlbaum, 41-63.
- Delgado, M. R., Olsson, A. & Phelps, E. A. (2006). Extending animal models of fear conditioning to humans. *Biological Psychiatry*, 73, 39-48.
- Devilley, G., Gist, R. & Cotton, P. (2006). Ready!! Fire!! Aim!! The Status of Psychological Debriefing and Therapeutic Interventions: In the Work Place and After Disasters. *Review of General Psychology*, 10, 318-345.

- Deville, G., Varker, T., Hansen, K. & Gist, R. (2007). An analogue study of the effects of Psychological Debriefing on eyewitness memory. *Behaviour Research and Therapy*, 45, 1245-1254.
- Dunmore, E., Clark, D. & Ehlers, A. (2001). A prospective investigation of the role of cognitive factors in persistent Posttraumatic Stress Disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy*, 39, 1063-1084.
- Ehlers, A. & Clark, D. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, 38, 319-345.
- Ehlers, A., Mayou, R. & Bryant, B. (2003). Cognitive predictors of posttraumatic stress disorder in children: results of a prospective longitudinal study. *Behavior Research and Therapy*, 41, 1-10.
- Engelhard, I. & Arntz, A. (2005). The fallacy of ex-consequencia reasoning and the persistence of PTSD. *Journal of Behaviour Therapy and Experimental Psychiatry*, 36, 35-42.
- Engelhard, I., van den Hout, M. & Vlaeyen, J. (2003). The sense of coherence in early pregnancy and crisis support and posttraumatic stress after pregnancy loss: a prospective study. *Behavioural Medicine*, 29, 80-84.
- Engelhard, I., van den Hout, M., Arntz, A. & McNally, R. (2002). A longitudinal study of intrusion-based reasoning” and posttraumatic stress disorder after exposure to a train disaster. *Behaviour Research and Therapy*, 40, 1415-1434.
- Engelhard, I., van den Hout, M., Arntz, A. & McNally, R. (2002). A longitudinal study of intrusion-based reasoning” and posttraumatic stress disorder after exposure to a train disaster. *Behaviour Research and Therapy*, 40, 1415-1434.
- Feldner, M., Babson, K. & Zvolensky, M. (2007). Smoking, traumatic event exposure, and post-traumatic stress: A critical review of the empirical literature. *Clinical Psychology Review*, 27, 14-45.
- Foa, E. & Rothbaum, B. (1998). *Treating the trauma of rape: Cognitive-behavioral therapy for PTSD*. New York: Guilford Press.

- Foa, E., Steketee, G. & Rothbaum, B. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy*, 20, 155-176.
- Forbes, D., Haslam, N., Williams, B. & Creamer, M. (2005). Testing the latent structure of posttraumatic stress disorder: A taxometric study of combat veterans. *Journal of Traumatic Stress*, 18, 647-656.
- Fraley, C., Fazzari, D., Bonanno, G. & Dekel, S. (2006). Attachment and psychological adaptation in high exposure survivors of the September 11th attack on the world trade center. *Personality and Social Psychology Bulletin*, 32, 538-551.
- Fraley, C., Fazzari, D., Bonanno, G. & Dekel, S. (2006). Attachment and psychological adaptation in high exposure survivors of the September 11th attack on the World Trade Center. *Personality and Social Psychology Bulletin*, 32, 538-551.
- Gailliot, M., Schmeichel, B. & Baumeister, R. (2006). Self-regulatory processes defend against the threat of death: Effects of self-control depletion and trait self-control on thoughts and fears of dying. *Journal of Personality and Social Psychology*, 91, 49-62.
- Gil, S. (2005). Pre-traumatic personality as a predictor of post-traumatic stress disorder among undergraduate students exposed to a terrorist attack: A prospective study in Israel. *Personality and Individual Differences*, 39, 819-827.
- Ginzberg, K., Solomon, Z., Koifman, B., Keren, G., Roth, A. & Kriwisky, M., et al., (2003). Trajectories of post-traumatic stress disorder following myocardial infarction: A prospective study. *Journal of Clinical Psychiatry*, 64 1217-1223.
- Gold, S. N. (2000). *Not trauma alone: Therapy for child abuse survivors in family and social context*. Philadelphia, PA: Brunner/Routledge.
- Golier, J., Yehuda, R., Bierer, L., Mitropoulou, V., New, A., Schmeidler, J., et al. (2003). The relationship of borderline personality disorder to posttraumatic stress disorder and traumatic events. *American Journal of Psychiatry*, 160, 2018-2024.
- Grillon, C. & Morgan, A. (1999). Fear-Potentiated Startle Conditioning to Explicit and Contextual Cues in Gulf War Veterans With Posttraumatic Stress Disorder. *Journal of Abnormal Psychology*, 108, 134-142.

- Halligan, S. & Yehuda, R. (2002). Assessing dissociation as a risk factor for posttraumatic stress disorder: A study of adult offspring of Holocaust survivors. *Journal of Nervous and Mental Disease*, 190, 429-436.
- Hapke, U., Schumann, A., Rumph, H., Ulrich, J., Konerding, U. & Meyer, C. (2005). Association of smoking and nicotine dependence with trauma and posttraumatic stress disorder in a general population sample. *The Journal of Nervous and Mental Disease*, 193, 843-846.
- Harvey, A. & Bryant, R. (2002). Acute stress disorder: A synthesis and critique. *Psychological Bulletin*, 128, 886-902.
- Harvey, A., Bryant, R. & Tarrier, N. (2003). Cognitive behaviour therapy for posttraumatic stress disorder. *Clinical Psychology Review*, 23, 501-522.
- Heilemann, M. Kury, F. & Lee, K. (2005). Trauma and posttraumatic stress disorder symptoms among low-income woman of Mexican descent in the United States. *The Journal of Nervous and Mental Disease*, 193, 665-672.
- Heinrichs, M. Wagner, D. Schoch, W., Soravia, L. Hellhammer, D. & Ehlert, U. (2005). Predicting posttraumatic stress symptoms from pretrauma risk factors: A 2-year prospective follow-up study in fire fighters. *American Journal of Psychiatry*, 162, 2276-2286.
- Holbrook, T., Hoyt, D., Stein, M., & Sieber, W. (2001). Perceived threat to life predicts posttraumatic stress disorder after major trauma: risk factors and functional outcome. *The Journal of Trauma*, 51, 287-292.
- Hyman, S., Gold, S. & Cott, M. (2003). Forms of social support that moderate PTSD in childhood sexual abuse survivors. *Journal of Family Violence*, 18, (5), 295- 300.
- Janoff-Bulman, R. (1992). *Shattered assumptions: Towards a psychology of trauma*. New York: Free Press.
- Jatzko, A., Rothenhöfer, S., Schmitt, A., Gaser, C., Demirakca, T., Weber-Fahr, W. et al., (2006). Hippocampal volume in chronic posttraumatic stress disorder (PTSD): MRI study using two different evaluation methods. *Journal of Affective Disorders*, 94, 121-126.

- Jones, C., Harvey, A. G. & Brewin, C. R. (2006). The organisation and content of trauma memories in survivors of road traffic accidents. *Behaviour Research and Therapy*, 45, 151-162.
- Joseph, S. & Linley, P. (2005) Positive Adjustment to Threatening Events: An Organismic Valuing Theory of Growth Through Adversity. *Review of General Psychology*, 9, 262-280.
- Kanagaratnam, P. & Asbjørnsen, A. E. (2006). Executive deficits in chronic PTSD related to political violence. *Journal of Anxiety Disorders*, doi:10.1016/j.janx-dis.2006.06.008.
- Kashdan, T., Elhai, J. & Frueh, B. (2006). Anhedonia and emotional numbing in combat veterans with PTSD. *Behavior Research and Therapy*, Article in Press.
- Kavaliers, M. & Choleris, E. (2001). Antipredator responses and defensive behavior: ecological and ethological approaches for the neurosciences. *Neuroscience and Biobehavioral Reviews*, 25, 577-586.
- Khamis, V. (2005). Post-traumatic stress disorder among school-age Palestinian children. *Child Abuse and Neglect*, 29, 81-95.
- King, D., King, L., Foy, D., Keane, T. & Fairbank, J. (1999). Posttraumatic stress disorder in a national sample of female and male Vietnam veterans: risk factors, war zone stressors, and resilience-recovery variables. *Journal of Abnormal Psychology*, 164-170.
- Koenan, K., Hitsman, B., Lyons, M., Stroud, L., Niaura, R., McCaffery, J., Goldberg, J., Eisen, S., True, W. & Tsuang, M. (2006). Posttraumatic stress disorder and late-onset smoking in the Vietnam era twin registry. *Journal of Consulting and Clinical Psychology*, 74, 186-190.
- Koenen, K., Moffitt, T., Poulton, R., Martin, J. & Caspi, A. (2006). Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort. *Psychological Medicine*, 37, 181-192.
- Koenen, K., Moffitt, T., Poulton, R., Martin, J. & Caspi, A. (2006). Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort. *Psychological Medicine*, 37, 181-192.

- Koren, D., Norman, D., Cohen, A., Berman, J. & Klein, E. (2005). Increased PTSD risk with combat related injury: A matched comparison study of injured and uninjured soldiers experiencing the same combat events. *American Journal of Psychiatry*, 162, 276-282.
- Koso, M & Hansen, S. (2005). Executive function and memory in posttraumatic stress disorder: a study of Bosnian war veterans. *European Psychiatry*, 21, 167-173.
- Kubiak, S. (2005). Trauma and cumulative adversity in women of a disadvantaged social location. *American Journal of Orthopsychiatry*, 475, 451-465.
- Kutz, I. & Dekel, R. (2006) Follow-up of victims of one terrorist attack in Israel: ASD, PTSD and the perceived threat of Iraqi missile attacks. *Personality and Individual Differences*, article in press.
- Lau, J. Y. & Viding, E. M. (2006). Anxiety-related biases in children's avoidant responses to a masked angry face. *Behaviour Research and Therapy*, Article in Press.
- Lauterbach, D., Vora, A & Rakow, M. (2005). The relationship between posttraumatic stress disorder and self-reported health problems. *Psychosomatic Medicine*, 939-97.
- Linehan, M. (1993). *Cognitive-Behavioral Treatment of Borderline Personality Disorder*. New York: Guildford Press.
- Linley, A. & Joseph, S. (2003). Positive change following trauma and adversity: A review. *Journal of Traumatic Stress*, 17, 11-21.
- Macklin, M., Metzger, L., Litz, B., McNally, R. Lasko, N. et al., (1998). Lower pre-combat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 66, 323-326.
- Maes, M., Mylle, J., Delmeire, L & Janca, A. (2001). Pre and post-disaster life events in relation to the incidence and severity of post traumatic stress disorder. *Psychiatry Research*, 105, 1-12.
- Maes, M., Mylle, J., Delmeire, L & Janca, A. (2001). Pre and post-disaster life events in relation to the incidence and severity of post traumatic stress disorder. *Psychiatry Research*, 105, 1-12.

- Marshall, R., Turner, J., Lewis-Fernandez, R., Koenan, K., Neria, Y & Dohrenwend, B. (2006). Symptom patterns associated with chronic PTSD in male veterans. *Journal of Nervous and Mental Disease*, 194, 275-278.
- Marx, B.P. & Sloan, D.M. (2005). Peritraumatic dissociation and experiential avoidance as predictors of posttraumatic stress symptomatology. *Behaviour Research and Therapy*, 43, 569-583.
- Mayou, R., Ehlers, A. & Bryant, B. (2002). Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study. *Behaviour Research and Therapy*, 40, 665-675.
- Mayou, R., Ehlers, A. & Bryant, B. (2002). Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study. *Behaviour Research and Therapy*, 40, 665-675.
- McNally, R. & Shin, L. (1995). Association of intelligence with severity of posttraumatic stress disorder symptoms in Vietnam combat veterans. *American Journal of Psychiatry*, 152, 936-938.
- Milad, M., Wright, C., Orr, S., Pitman, R., Quirk, G. & Rauch, S. (2007). Recall of fear extinction in humans activates the ventromedial prefrontal cortex and hippocampus I concert. *Biological Psychiatry*, Article in Press.
- Ozer, E., Best, S., Lipsey, T. & Weiss, D. (2003). Predictors of posttraumatic stress disorder an symptoms in adults: A meta-analysis. *Psychological Bulletin*, 129, 52-79.
- Parslow, R., Jorm, A. & Christensen, H. (2006). Associations of pre-trauma attributes and trauma exposure with screening positive for PTSD: An analysis of a community-based study of 2085 young adults. *Psychological Medicine*, 36, 387-395.
- Pfefferbaum, B; Nixon, S., Krug, R., Tivis, R., Moore, V., Brown, J., et al., (1999). Clinical needs assessment of middle and high school students following the 1995 Oklahoma City bombing. *American Journal of Psychiatry*, 156, 1069-1074.
- Pitman, R.K., Orr, S. P. & Shalev, A. Y. (1993). Once bitten, twice shy: beyond the conditioning model of PTSD. *Biological Psychiatry*, 33, 145-146.

- Raphael, B. (2000). Debriefing – science, belief and wisdom (pp. 351-359). In B. Raphael & J. Wilson (Eds.). *Psychological Debriefing: Theory, Practice and Evidence*. Cambridge: Cambridge University Press.
- Reynolds, M., Mezey, G., Chapman, M., Wheeler, M., Drummond, C. & Baldacchino, A. (2005). Co-morbid post-traumatic stress disorder in a substance misusing clinical population. *Drug and Alcohol Dependence*, 77, 251-258.
- Rosen, J. B. & Donley, M. P. (2006). Animal studies of amygdala function in fear and uncertainty: Relevance to human research. *Biological Psychiatry*, 73, 49-60.
- Rothbaum, B., Meadows, E., Resick, P., & Foy, D. (2000). Cognitive-behavioural therapy. In E. Foa, T. Keane & M. Friedman (Eds.), *Effective treatments for PTSD* (pp. 60-83). New York: The Guildford Press.
- Roy-Byrne, P., Arguelles, L., Vitek, M., Goldberg, J., Keane, T., True, W. & Pitman, R. (2004). Persistence and change of PTSD symptomatology. A longitudinal co-twin control analysis of the Vietnam era twin registry. *Social Psychiatry & Psychiatric Epidemiology*, 39:681-685.
- Ruscio, A., Ruscio, J. & Keane, T. (2002). The latent structure of posttraumatic stress disorder: A taxometric investigation of reactions to extreme stress. *Journal of Abnormal Psychology*, 111, 290-301.
- Saigh, P., Mroueh, M. Bremner, D. (1997). Scholastic impairments among traumatized adolescents. *Behaviour Research & Therapy*, 35, 429-436.
- Schell, T., Marshall, G. & Jaycox, L. (2004). All symptoms are not created equal: The prominent role of hyperarousal in the natural course of posttraumatic psychological distress. *Journal of Abnormal Psychology*, 113, 189-197.
- Schnurr, P., Lunney, C. & Sengupta, A. (2004). Risk factors for the development versus maintenance of posttraumatic stress disorder. *Journal of Traumatic Stress*, 17, 85-95.
- Schnyder, U., Moergeli, H., Klaghofer, R. & Buddberg, C. (2001). Incidence and prediction of posttraumatic stress disorder symptoms in severely injured trauma survivors. *American Journal of Psychiatry*, 158, 594-599.

- Scrimin, S. Giovanna, A., Capella, F., Moscardino, A., Stenberg, A. & Pynoos, R. (2006). Posttraumatic reactions among injured children and their caregivers 3 months after the terrorist attack in Beslan. *Psychiatry Research*, 141, 333-336.
- Shipherd, J., Stafford, J. & Tanner, L. (2005). Predicting alcohol and drug abuse in Persian Gulf War veterans: What role do PTSD symptoms play? *Addictive Behaviors*, 30, 595-599.
- Siegmund, A. & Wotjak, C. T. (2007). Hyperarousal does not depend on trauma-related contextual memory in an animal model of Posttraumatic Stress Disorder. *Physiology & Behavior*, 90, 103-107.
- Silove, D., Steel, Z., McGorry, P & Mohan, P (2002). The impact of torture on post-traumatic stress symptoms in war-affected Tamil refugees and immigrants. *Comprehensive Psychiatry*, 43, 49-55.
- Silva, R., Alpert, M., Munoz, D., Singh, S., Matzner, F., Dummit, S. (2000). Stress and vulnerability to posttraumatic stress disorder in children and adolescents. *American Journal of Psychiatry*, 157, 1229-1235.
- Speckens, A., Ehlers, A., Hackman, A. & Clark, D. (2006). Changes in intrusive memories associated with imaginal reliving in posttraumatic stress disorder. *Anxiety Disorders*, 20, 328-341.
- Spiro, A., Hankin, C., Mansell, D. & Kazis, L. (2006). Posttraumatic stress and health status: The veteran's health study. *Journal of Ambulatory Care Management. Ambulatory Care and Conflict*, 29, (1), 71-86.
- Thayer, J. & Brosschot, J. (2005). Psychosomatics and psychopathology: looking up and down from the brain. *Psychoneuroendocrinology*, 30, 1050-1058.
- Van Etten, M. & Taylor, S. (1998). Comparative efficacy of treatments for post-traumatic stress disorder: A meta-analysis. *Clinical Psychology and Psychotherapy*, 5, 126-144.
- Vasterling, J., Duke, L., Bariley, K., Constans, J., Allain, jr, A. & Sutker, P. (2002). Attention, learning and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. *Neuropsychology*, 16, 5-14.

- Veazey, C., Blanchard, E., Hickling, E. & Buckley, T. (2004). Physiological responsiveness of motor vehicle accident survivors with chronic posttraumatic stress disorder. *Applied Psychophysiology and Biofeedback*, 29, (1), 51-62.
- Vranceanu, A., Hobfoll, S. E. & Johnson, R. J. (2007). Child multi-type maltreatment and associated depression and PTSD symptoms: The role of social support and stress. *Child Abuse & Neglect*, 31, 71-84.
- Waschbusch, D., Sellers, D., LeBlanc, M., Kelley, M. (2003). Helpless attributions and depression in adolescents: The roles of anxiety, event valence and demographics. *Journal of Adolescence*, 26, 169-183.
- Weber, D., Clark, C., McFarlane, A., Moores, K., Morris, P. & Egan, G. (2005). Abnormal frontal and parietal activity during working memory updating in post-traumatic stress disorder. *Psychiatry Research: Neuroimaging*, 140, 27-44.
- Yehuda, R. (1998). Neuroendocrinology of trauma and posttraumatic stress disorder. In R Yehuda (Ed.) *Psychological Trauma*. (pp. 97-125). Washington, DC: American Psychiatric Press, Inc.
- Yehuda, R., Golier, J., Yang, R. & Tischler, L. (2004). Enhanced sensitivity to glucocorticoids in peripheral mononuclear leukocytes in posttraumatic stress disorder. *Biological Psychiatry*, 55, 1110-1116.
- Yen, S., Shea, M., Battle, C., Johnson, D., Zlotnick, C., Dolan-Sewill, R. et al., (2002). Traumatic exposure and posttraumatic stress disorder in borderline, schizotypal, avoidant, and obsess-compulsive personality disorders: findings from the collaborative longitudinal personality disorders study. *The Journal of Nervous and Mental Disease*, 190, 510-518.
- Zoellner, L., Jaycox, L., Watlington, C. & Foa, E. (2003). Are the Dissociative Criteria in ASD Useful? *Journal of Traumatic Stress*, 16, 341-350.