Psychophysiological Processes Involved in Traumatic Memory

Chia-Ying Chou

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I, Chia-Ying Chou confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

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Abstract

The pathways through which traumatic events are encoded into memory and subsequently retrieved affect the development of posttraumatic symptoms such as intrusion, as well as recovery from posttraumatic stress disorder (PTSD). This thesis examined how cardiovascular and hormonal processes are related to memory processing. Individual differences in traumatic history, as well as two cardiovascular stress response features, startle heart rate (sHR) and cardiac defence response (CDR), were investigated in this context as predictors and moderators. Relevant literature and the methods are reviewed in Chapter One and Chapter Two respectively. Chapter Three and Four adopted the trauma film paradigm to assess the memory encoding phase of trauma. The former found a dominant vagal activation during the analogue trauma, and identified a subgroup, in whom relationships between the psychological and physiological measures were different from the rest of the sample. The latter found increases in cortisol, and decreases in salivary alpha-amylase (sAA) levels, in response to the trauma film. Lower cortisol levels predicted greater vividness of intrusions. Individual differences in CDR and sAA levels moderated the relationship between cortisol and the frequency of intrusions. Chapters Five and Six examined PTSD patients' psychological and physiological reactions to voluntary retrieval of traumatic memories. Significant relationships between HR decreases and overall negative psychological states were found in the former. Associations between greater dissociation and a smaller suppression of cortisol were found in the latter. An overall discussion regarding the psychological and physiological activities at the memory encoding and retrieval phases, as well as the roles of trauma history, sHR and CDR in moderating these responses, are presented in Chapter Seven.

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The work presented in this thesis has given rise to the following publications:

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Chapter 1: Introduction to the psychophysiological processes involved in traumatic memory

1.1 Background

Trauma is one of the most inevitable phenomena in human life. Its psychological consequences, however, vary across individuals. One of the consequences of trauma is the development of posttraumatic stress disorder (PTSD; American Psychiatric Association, 1994). PTSD was first included in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM, American Psychiatric Association, 1994), with its diagnostic criteria continuously modified based on the latest research findings. According to the fifth edition of the DSM (American Psychiatric Association, 2013), diagnosis of PTSD is made when all of the following criteria are met. First, the individual was directly exposed to, witnessed, or learned about a close other's exposure to trauma, which involved actual or threatened death, serous injuries, or sexual violence. Second, trauma is persistently re-experienced in the form of intrusive memories, nightmares, flashbacks, or presentations of marked psychological or physiological distress after exposure to traumatic reminders. Third, the individual persistently and intentionally avoids trauma-related thoughts, feelings, or reminders. Fourth, negative alterations in cognitions or mood, such as inability to recall key features of the trauma, negative beliefs or expectations about self, self blame, negative emotions, detachment, or diminished interest, happen or worsen after the trauma. Fifth, alternation in arousal or reactivity is present in the form of irritation, self-destructive behaviors, hypervigilance, exaggerated startle, concentration or sleep difficulties. Sixth, these symptoms persist longer than a month, cause significant distress or functional impairment, and are not better explained by medication, substance use, or any other illnesses.

In a big scale national survey in the U.S. (Solomon & Davidson, 1997), the life-time prevalence rate of PTSD was 5% among men, and 10-12% among women. These data show that while almost everyone has suffered from at least one trauma in life, only a small portion of individuals developed PTSD. In other words, there is significant individual difference in the risk of PTSD development. Moreover, given the fact of being the most commonly adopted treatment for PTSD, the treatment outcome of cognitive-behavioural therapy (CBT) differs across patients with diverse symptom profiles (Lanius et al., 2010). Studies have linked the varieties of posttraumatic and treatment outcomes to the diversities of information processing. For example, the involvement of high-level cognitive functions has been suggested to be a protective factor at the memory encoding and early consolidation stages; The crucial role of sufficient emotional arousal during the retrieval of traumatic memory in therapy has been highlighted.

In addition to the cognitive and emotional aspects, psychophysiological stress-related responses have been examined in the PTSD literature. The fluctuations in heart rate (HR) and the amount of stress-related hormones released in response to traumatic stimuli have been associated with different levels of risk of developing PTSD. Moreover, their roles in predicting the effects of psychotherapy have been attended to in recent studies. By including physiological measures, these studies have provided more complete perspectives. The potential to use physiological activity to infer or predict trauma-related psychological status has been realised as well. In order to enable more sophisticated applications, the goal of this thesis is to examine the links between psychological and physiological reactions to trauma, as well as individual differences in these associations.

This chapter introduces the theories regarding the cognitive pathways involved in memory processes related to positive and negative posttraumatic outcomes (section 1.2). The

activity of the autonomic nervous system (ANS) as an immediate response to or long-term consequence of trauma will then be introduced within this theoretical context (section 1.3). Next, findings regarding the responses of the hypothalamic-pituitary-adrenal (HPA) axis to trauma will be introduced (section 1.4). Finally, the factors related to individual differences in posttraumatic psychophysiological responses and inconsistencies in the existing literature will be discussed (section 1.5), followed by a summary of the aims and hypotheses of this thesis (section 1.6).

1.2 The pathways of traumatic memory processing

1.2.1 The recent theories of PTSD

One of the most characteristic features of PTSD is the involuntary memories, which contain vivid sensory information and are experienced as if the incidents are occurring again in the present. Despite the intrusive and recurrent nature, the involuntary memories coexist with the inability to voluntarily recall episodic memories of trauma (Brewin, 2013). Given the diverse natures of the involuntary and voluntary memories, and many contradictory findings of trauma-related involuntary memories compared to ordinary autobiographic memories, two recent theories have suggested the existence of two separate and fundamentally distinct memory systems (Brewin, 2013; Brewin & Holmes, 2003)

First, according to the cognitive model proposed by Ehlers and Clark (2000), a range of negative appraisals, such as overgeneralisation of danger (e.g., others can see I am an easy target) and mistaken appraisal of one's own actions (e.g., I deserve it), contribute to poorly elaborated and poorly contexualised memories. These negative appraisals result in inadequate integration of traumatic memories with autobiographical memory and easily triggered re-experiencing of these memories. Both peri-traumatic (i.e., at the time of trauma) thought processes and pre-existing beliefs influence the formation of these negative appraisals. Among the peri-traumatic factors that may influence the memory encoding process of trauma, a distinction between data-driven processing and conceptual processing (Roediger & McDermott, 1993) has been highlighted. Applying Conway and Pleydell-Pearce's model of autobiographical memory (2000), Ehlers and Clark suggested a distinction between more general autobiographical knowledge and event-specific knowledge (ESK), which involves sensory information specific to a certain event. While data-driven processing focuses on sensory impressions, and leads to strong perceptual priming and memories that are not easily

voluntarily retrieved, conceptual processing focuses on the meaning and context of the incident, and facilitates the integration of traumatic ESK with autobiographical database (Ehlers & Clark, 2000).

Similar to Ehlers and Clark, another recent theory of PTSD, the dual-representation theory (DRT; Brewin, Gregory, Lipton, & Burgess, 2010; Brewin, Dalgleish, & Joseph, 1996), also proposed the existence of two memory systems. According to the DRT (Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996), traumatic memories are processed through two parallel but independent neurological pathways, each associated with different cognitive functions. The first pathway has the involvement of the hippocampus and medial temporal lobes (MTLs), which are related to the processing of spatio-temporal contextual information, and the formation of declarative memories. Processed with the support of these brain regions, the contextual memory (C-memory) and its representation, C-reps, contain spatio-temporal information and gist ideas (e.g., when, where, how, who) of an event. They enable the generation of episodic memories and meanings (e.g., the impact on one's life) of an event and are capable of being integrated into personal autobiographical memory.

Parallel to the C-memory, the sensation-based memory (S-memory) and its representations (S-reps) are formed and supported by lower level sensory cortices. S-memories contain sensory details (e.g., images, sound, smell), as well as physiological (e.g., heart pounding, palm sweating) and psychological feelings (e.g., fear, anger) imprinted during the event. Lacking the involvement of higher level brain regions, they are egocentric, viewpoint-dependent, and inflexibly depictive of the original experience.

In ordinary circumstances, S-reps quickly fade and become less active (Brewin et al., 2010). Differently, in stressful or emotionally salient situations, more enduring S-reps and C-reps are developed. With support of the brain structures which link the abovementioned brain

regions supporting the two memory systems, S-reps are able to associate with their corresponding C-reps in the MTLs. This association facilitates top-down control of the brain and, over time, allows salient emotional information to be processed and integrated with personal semantic memory system. However under extreme levels of subjective distress, brain regions related to semantic information processing, such as the hippocampaus, are impaired, whereas other regions related to primary emotional and sensory processing are activated (Elzinga & Bremner, 2002; Kensinger, Garoff-Eaton, & Schacter, 2007). As a result, stronger S-reps (compared to C-reps), and a weaker association between the two are formed. Due to a lack of top-down control and contextual information, vivid S-memories are triggered involuntarily and experienced as flashbacks.

Although DRT and Ehlers and Clark's model (2000) both propose two memory systems (i.e., S-memory and C-memory for the former, ESK and general autobiographical knowledge for the latter), with one of them involving more sensory information and less contexualisation than the other, there are substantial differences between the two theories. First, while DRT suggests that the S-memory system is the main source of intrusive memory, Ehlers and Clark have not identified either of the memory systems as being more closely related to the development of intrusion. Second, despite the fact that S-memory is strongly related to the development of intrusion, it is not considered harmful in DRT, as long as an equivalent level of processing is undertaken in the C-memory system. In contrast, in Ehlers and Clark's model, data-driven processing is generally regarded as a risk factor. Third, in DRT, the two commonly observed features of voluntary trauma memory, disorganisation and fragmentation, are believed to be related to one's ability to deliberately retrieve detailed and clear information in time, rather than the depth of processing in the C-memory system. They are therefore not considered risk factors based on the DRT. However, in Ehlers and Clark's

model, these features are signs of data-driven processing and lack of conceptual processing, and have been seen as risk factors.

1.2.2 Empirical evidence for the recent theories

Overall, despite the above-mentioned inconsistencies, the two recent theories, DRT (Brewin et al., 2010) and Ehlers and Clark's model (2000), both emphasise the contribution of several peri-traumatic factors to resilient vs. pathological outcomes. Their hypotheses about the association between peri-traumatic cognitive state and the development of posttraumatic memory symptoms have been examined with many studies adopting the trauma film paradigm (refer to Chapter Two for details). For example, in a study that manipulated information processing style during the memory encoding phase of the trauma film and immediately afterwards, conceptual encoding was found to relate to fewer intrusive symptoms, whereas data-driven processing after the film was related to more intrusions (Kindt, van den Hout, Arntz, & Drost, 2008).

Consistently, in a study where healthy participants were asked to carry out a visuospatial grounding task (i.e., construction of shapes out of plasticine) during a part of the trauma film viewing, less frequent intrusions were found from this part of the film (Stuart, Holmes, & Brewin, 2006). As the visuospatial grounding task was designed to occupy the cognitive resources in the S-memory system, its contribution to lessening the involuntary memories supported the DRT, which suggested the association between the S-memory system and PTSD memory symptoms. Moreover, to support the DRT in a different way, another set of studies, which applied a low dose of alcohol as a means to impair the cognitive functions of the C-memory system during memory encoding phase of the trauma film, resulting in more intrusive memories (Bisby, Brewin, Leitz, & Curran, 2009; Bisby, King, Brewin, Burgess, & Curran, 2010). Individuals with a generally less well functioning C-memory system, as assessed by an inability to shift viewpoint in a recognition memory test, also experienced more intrusive memories following a trauma film (Bisby et al., 2010).

Additionally, agreeing with these laboratory findings, peri-traumatic dissociation - a psychological state involving disruptions in the integration of consciousness - has been found in retrospective studies involving real-life traumas to significantly predict PTSD (meta-analysis by Ozer, Best, Lipsey, & Weiss, 2003). These data support the association between a less involvement of high-level cognitive functions, which is a mental state related to the S-memory system, and the development of PTSD. Furthermore, it has been found that the parts of trauma narratives retrieved by PTSD patients during flashbacks contained more S-rep characteristics such as sensory/movement details, and mention of primary emotions (e.g., fear, helplessness and horror) and use of the present tense. In contrast, narrative sections retrieved as ordinary episodic memories were more contextually bounded and contained more high-level meanings and secondary emotions such as guilt and anger (Hellawell & Brewin, 2004). Overall, these studies supported the existence of two parallel memory systems, as well as the association between the lack of the involvement of higher level cognitive functions and the development of intrusive memories.

1.3 Cardiovascular activities, trauma, and PTSD

1.3.1 Heart rate and the encoding of traumatic memory

Among many stress-related biological markers, HR is the most well studied in clinical psychology. It is an indicator of the balance between autonomic nervous systems (ANS) and an objective index of many important mental states (e.g., orienting, freezing, dissociation, and fight/flight response). While a HR increase is generally regarded as an indicator of active action or defense, the meaning of a HR decrease is more debatable (Graham & Clifton, 1966). Some studies considered the HR decrease as reflecting an orientating response, which has major effects on learning and perceptual processes (e.g., Sokolov, 1960). However, other studies involving aversive stimuli such as loud noise or electric shocks have yielded an alternative explanation for the HR decrease as a sign of inhibited defense (Graham & Clifton, 1966), or passive avoidance (Richter, Schumann, & Zwiener, 1990).

In the PTSD literature, it has been reported that PTSD patients tend to present heightened resting HR comparing to healthy control groups (e.g., Blechert, Michael, Grossman, Lajtman, & Wilhelm, 2007; Cohen et al., 1997). Moreover, considering the significance of peri-traumatic cognitive states, such as dissociation, in predicting PTSD symptoms (meta-analysis by Ozer, Best, Lipsey, & Weiss, 2003), HR at the time points close to the trauma (e.g., during the ambulance transport, or the initial presentation to the emergency department) have been examined in PTSD studies as an index to infer the peri-traumatic mental states (e.g., Bryant, Salmon, Sinclair, & Davidson, 2007; Kraemer, Moergeli, Roth, Hepp, & Schnyder, 2008). Most studies related HR at the time between the victims' arrival in hospital and the day of discharge to their PTSD symptoms a month posttrauma and onward. These studies generally found positive correlations between the two (e.g., Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2008; Bryant et al., 2007; De

Young, Kenardy, & Spence, 2007; Kraemer, Moergeli, Roth, Hepp, & Schnyder, 2008; Zatzick et al., 2005). However, among studies examining HR at time points even closer to the accidents (e.g. at the accident sites or during the ambulance transport), inconsistent results have been reported. Some studies did not find a significant discrepancy in HR between victims who did and did not develop PTSD (e.g., Buckley et al., 2004; Ostrowski, Christopher, & Delahanty, 2007). Others found lower early-stage HR in individuals later predicted higher PTSD symptoms (Blanchard, Hickling, Galovski, & Veazey, 2002; O'Donnell, Creamer, Elliott & Bryant, 2007). Due to the variation in the sampled time points and the types of trauma, consistent results were not found in terms of whether the increase or decrease during the initial stage of trauma predicts the development of PTSD.

To better control for variability and investigate phenomena during the time when traumatic information is being processed, the trauma film paradigm has been developed (reviewed by Holmes & Bourne, 2008; refer to Chapter Two for details). In a previous study applying the trauma film paradigm (Holmes, Brewin, & Hennessy, 2004), a traumatic film of real life footage from road traffic accidents was shown to a non-clinical sample. HR was monitored throughout the whole process of film viewing and examined for its association with later intrusive memory about the film. The study found a decrease in HR during film viewing. Larger decreases were predictive of an increased number of involuntary memories. Moreover, the mean HR during the film sequences matching the contents of participants' subsequent involuntary memories was significantly lower than the mean HR during the sequences that did not occur involuntarily to the participants' minds.

The reduction in HR was interpreted in this study (Holmes, Brewin, & Hennessy, 2004) as a representation of fear bradycardia (a brief freezing-like response characterised with a HR reduction) or the freezing response found in animals in the face of overwhelming

threats. However, as described earlier, because a decreased HR may also be an indicator of increased orienting, clarification is still needed. Specifically, further studies should investigate whether the decrease in HR during trauma film viewing is a product of the temporary shut-down of higher-level cognitive function (similar to the freezing response), or alternatively an indicator of orienting. Such clarification is of research interest, because the former may strengthen S-memory processes, whereas the latter is an important element in the C-memory system (Brewin et al., 2010).

1.3.2 Heart rate, the voluntary retrieval of traumatic memory, and dissociation

1.3.2.1 Heart rate and voluntary memory retrieval in exposure therapy

According to the DRT, successful treatment of PTSD requires the ability to hold the S-memories in focal attention, which allows the formation of corresponding C-reps, and strengthens the association between the two systems (Brewin et al., 2010). Consistent with these principles, voluntary retrieval of traumatic memories is a key element of exposure-based psychotherapy for PTSD. It has been established that full activation of the traumatic memory and emotional engagement during the voluntary memory retrieval are essential for therapeutic effects to occur in exposure therapies (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989).

Physiological reactions have been applied in recent studies to facilitate understanding of the mental states during voluntary retrieval. In a study (Halligan, Michael, Wilhelm, Clark, & Ehlers, 2006) examining reactions to a recall task modelled on imaginal reliving (Foa & Rothbaum, 1998), trauma victims with PTSD showed a smaller HR increase (relative to the baseline level) compared to victims without PTSD. Moreover, smaller HR increases during the recall task were found to predict less PTSD symptom reduction 6 months later. These findings supported the hypothesised association between incomplete retrieval of traumatic memories and limited physiological activation during voluntary traumatic memory retrieval (Foa, Molnar, & Cashman, 1995). However, it was unclear whether the smaller level of HR increases found among the PTSD patients (Halligan et al., 2006) was related to their higher baseline HR, compared to the victims without PTSD.

1.3.2.2 Dissociation and response to exposure therapy

Dissociation is a psychological state involving disruptions in the integration of consciousness, with depersonalisation and derealisation as its main components (American Psychiatric Association, 2013). Depersonalisation includes out-of-body experiences, perceptions as if things are not happening to self, and feelings as if oneself is not real. Similarly, derealisation includes dream-like perceptions, and feelings as if the world is not real. Both components are typically accompanied by an attenuation of emotional experience (American Psychiatric Association, 2013). The fifth edition of DSM (American Psychiatric Association, 2013) has proposed that the presence of these two components of dissociation indicates a distinct subtype of PTSD, in addition to other dissociation-related symptoms that are listed as core PTSD symptoms, such as flashbacks and dissociative amnesia.

It has been suggested that PTSD patients with strong dissociative symptoms tend to have early and chronic traumatic experiences, more complex PTSD symptoms and complex comorbidity, compared with those with less dissociation symptoms (Steuwe, Lanius, & Frewen, 2012; van der Kolk et al., 1996). Similarly, studies have reported peri-traumatic dissociation as a predictor of the chronic suffering of post-traumatic distress (e.g., Bremner et al., 1992).

In terms of the influence on memory processing, as a way to detach from overwhelming emotions, dissociation prevents the activation, and hence deeper processing of traumatic memories (Brewin et al., 2010). While being asked to recall personal traumatic experiences, PTSD patients with more dissociative symptoms exhibit abnormally high activations in brain areas involved in arousal modulation and emotional regulation. However, patients with fewer dissociative symptoms exhibited abnormally low activations in the same brain areas (Lanius et al., 2010). These findings of differences in physiological reactions echo

the suggestion that PTSD patients with and without dissociative symptoms may respond differently to exposure-based therapy (reviewed by Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012). Moreover, dissociation's potential negative effects on exposure therapies have been suggested (Lanius et al., 2010).

1.3.2.3 Heart rate, dissociation, and voluntary retrieval of traumatic memory

Dissociation has been found to mediate the psychophysiological responses to traumatic cues. For example, lowered HR responses were found to be associated in delinquent adolescents with greater dissociative symptoms while describing their free association thoughts and most stressful life events (Koopman et al., 2004). Moreover, in a study of rape victims (Griffin, Resick & Mechanic, 1997), peri-traumatic dissociation was found to be a critical variable that was related to HR responses to the experimental manipulation (talking about the rape experience) within 2 weeks posttrauma. Among victims with high levels of peri-traumatic dissociation, a decreased HR was found. Contrastingly, among those with low peri-traumatic dissociation, an increased HR was evident instead. Moreover, examination of skin conductance levels among the former group showed a significant discrepancy between self-report distress and biological stress reactions to the recall -- when high distress levels were reported, low physiological arousal was shown. Overall, this study suggested that individuals who were highly dissociative peri-trauma responded to the task of voluntarily retrieving traumatic memories with suppressed psychological and physiological patterns compared to those who were less dissociative.

Nonsignificant associations between HR and dissociation, nevertheless, have been found in some studies (e.g., Halligan et al., 2006; Kaufman et al., 2002; Nixon, Bryant, Moulds, Felmingham, & Mastrodomenico, 2005). The different prevalence of dissociative symptoms between victims of repeated life adversities (i.e., Koopman et al., 2004) and single-exposure trauma (i.e., Halligan et al., 2006) may explain part of the inconsistent findings (explained in more details in 1.5.1). Moreover, the difference in the elapsed time of trauma between studies contributes to the inconsistency. Since most studies have focused on studying peri-traumatic dissociation (e.g., Griffin, Resick, & Mechanic, 1997; Halligan et al.,

2006; Kaufman et al., 2002; Nixon et al., 2005), the elapsed time involved in studies varies between less than one month and more than ten years, depending on the time when the studies took place. For example, in a study (Kaufman et al., 2002) including veterans of the Vietnam War, the elapsed time is more than ten years. Given this, individuals' peri-traumatic dissociation levels were no longer a significant factor correlated with their HR during the recall task in the experiment.

To overcome this issue and draw attention to closer time points, two studies (Halligan et al., 2006; Koopman et al., 2004) have assessed dissociative symptoms presented just prior to the experimental procedures. However, inconsistencies still prevail. The need of direct assessment of an acute state of dissociation happening during the manipulation of memory retrieval in study procedures has therefore been suggested (Halligan et al., 2006; Sack, Cillien, & Hopper, 2012). In a study assessing cardiovascular activities during script-driven trauma imagery, PTSD patients who reported high reexperiencing and high dissociative states exhibited lower HR, compared to those who reported high reexperiencing but low dissociative states (Sack, Cillien, & Hopper, 2012). More investigations of such acute dissociative reactions and their associations with psychophysiological activities are needed.

1.3.3 Heart rate variability, trauma and PTSD

As another cardiovascular index of the ANS, the strength of heart rate variability (HRV) in separately estimate the activation levels of the vagal and sympathetic nervous systems has been gaining research attention. Specifically, by providing information about how the variance (power) of heart rate is distributed as a function of frequency, power spectrum analysis (PSA) of HRV is a method for quantifying the activity of the ANS functions (Cohen, Matar, Kaplan, & Kotler, 1999). The high frequency component of HRV (i.e., 0.15-0.5 Hz; HF-HRV) is considered as a marker of vagal activity. The low frequency HRV (i.e., 0.04-0.15 Hz; LF-HRV) is suggested by some studies as a marker of the SNS, especially when it is expressed in normalised units. However, due to the fact that an absolute power decrease of LF-HRV is found in some conditions associated with sympathetic activation, some authors consider LF-HRV as a parameter including both sympathetic and vagal influences. Consequently, the LF/HF ratio is considered by some as reflecting sympatho/vagal balance, and by others as indexing the modulation of the sympathetic nervous system (Task Force, 1996).

The sympathetic system is considered to be associated with the fight or flight response (Thayer & Lane, 2009). On the other hand, similar to the discussion about the decreasees in HR, debates still exist in the interpretation of vagal activity. Some studies have shown a relationship between higher vagal activation and better performance on executive tasks (e.g., Hansen, Johnsen, & Thayer, 2003). However, in animal studies, vagal dominance was also observed in a state of fear bradycardia in the initial exposure to threatening stimuli (Bradley & Lang, 2007). Accordingly, it is still unclear whether increases in vagal activation represent the functioning of a higher-level cognitive process, or its temporary break-down.

HRV has been studied in the trauma and PTSD literatures since the last two decades. In the examination of resting HRV, some studies have found lower levels of HF-HRV, higher LF-HRV and LF/HF ratio among PTSD patients (e.g., Blechert, Michael, Grossman, Lajtman, & Wilhelm, 2007; Cohen et al., 1997; Hauschildt, Peters, Moritz, & Jelinek, 2011). However, nonsignificant differences between PTSD patients and the control groups have also been suggested (e.g., Sahar, Shalev, & Porges, 2001; Woodware, Kaloupek, Schaer, Martinez, & Eliez, 2008).

Regarding the HRV response to trauma-related stimuli, findings vary across studies with different designs, in terms of how traumatic memories were retrieved. For example, in a study, where videos of different emotional valences (i.e., neutral, positive, negative, and traumatic) were played to trauma victims with PTSD, trauma victims without PTSD, and non-trauma-exposed controls, the PTSD group showed lower vagal activation during all videos than the non-trauma-exposed controls (Hauschildt et al., 2011). An overall (i.e., across all groups and all videos) lower vagal activation was found to be associated with an overall greater state dissociation in this study (Hauschildt et al., 2011). Similarly, in another study that engaged PTSD patients in script-driven imagery, vagal activation significantly decreased during the exposure to a personalised trauma script, which was after a neutral script (Sack, Hopper, & Lamprecht, 2004). Both studies suggested an association between PTSD and a limited vagal response to the exposure of traumatic stimuli.

However, when PTSD patients were asked to voluntarily retrieve their traumatic memories, the reactive HRV varied across studies with different designs. For example, when reliving was not required during the voluntary verbal retrieval of traumatic memories, LF-HRV and HF-HRV did not significantly change from baseline to the memory retrieval phase among PTSD patients (Cohen et al., 2000; Cohen et al., 1998). In contrast, in another study

that asked female PTSD patients to describe a traumatic incident in vivid detail, a significant decrease of HF-HRV, which suggests a decrease in vagal activation, was found. Moreover, compared to the healthy control group, PTSD patients' HF-HRV reduction was significantly greater (Keary, Hughes, & Palmieri, 2009). Such inconsistency suggests that different instructions for voluntary retrieval between studies may induce different psychological responses and hence different findings in HRV. Additionally, the lengths of retrieval (over 15 minutes in Cohen et al., 2000 and 1998, and 4 minutes in Keary et al., 2009), as well as the gender of participants may account for part of the controversy.

1.4 Hypothalamic-pituitary-adrenal axis and traumatic stress

1.4.1 Basal hypothalamic-pituitary-adrenal axis activity, trauma, and PTSD

The hypothalamic-pituitary-adrenal (HPA) axis is a major part of the neuroendocrine system. It regulates and controls the stress response and many other physiological and mental processes such as metabolism, immune system function, digestion, mood and memory (Jones & Moller, 2011). The 'stress hormone', cortisol, is the HPA axis' major output which is released under the stimulation of both physical (e.g., illness, temperature extreme) and psychological stress (Bowirrat et al., 2010). Salivary cortisol has been used to reflect HPA activities in studies because of its close correlation between the concentrations of serum cortisol and ease of examination (Kirschbaum & Hellhammer, 1994). It has been widely applied in studies as an objective indicator of stress with its increased secretion consistently being demonstrated under stressful conditions or manipulations (Bowirrat et al., 2010; Het, Ramlow, & Wolf, 2005; Takai et al., 2004). However, as an objective measure of stress, cortisol levels are not always consistent with self-report distress (e.g., Fergus, Rabenhorst, Orcutt, & Valentiner, 2011).

In the context of trauma and posttraumatic stress disorder (PTSD), cortisol has been widely studied. However, a meta-analysis (Klaassens, Giltay, Cuijpers, van Veen, & Zitman, 2012) has suggested nonsignificant effects of past traumatic experiences and PTSD on cortisol due to inconsistent findings of the reviewed studies. Regarding the effect of the history of traumatic experiences on resting cortisol, some found a heightening effect when comparing trauma survivors to their pre-trauma sampling (Kotozaki & Kawashima, 2012) or control groups (Steudte et al., 2011); Some found lower cortisol levels compared to control (e.g., Witteveen et al., 2010), while others found no significant effect (e.g., Klaassens, Giltay, van Veen, Veen, & Zitman, 2010).

Similarly, the effect of PTSD on cortisol has also been found to be inconsistent. Some studies reported lower resting cortisol levels in PTSD patients than in control groups (e.g., Mason, Giller, Kosten, Ostroff, & Podd, 1986; Yehuda, Boisoneau, Mason, & Giller, 1993; Boscarino, 1996), some observed higher levels (e.g., Steudte et al., 2011; Suglia, Staudenmayer, Cohen, & Wright, 2010), whereas others found no significant difference between PTSD patients and control groups (e.g., LeBlanc et al., 2011; Witteveen et al., 2010). A meta-analysis (Meewisse, Reitsma, de Vries, Gersons, & Olff, 2007) has associated the findings of low basal cortisol levels among PTSD patients with abuse-related trauma and gender (i.e., female). Additionally, other parameters of trauma (e.g., the elapsed time), and the profile and severity of PTSD, should be taken into account to better clarify these relationships.

1.4.2 Reactive HPA axis activity, trauma, and PTSD

Few studies have examined the effects of past traumatic experiences on the reactions of the HPA axis to later traumatic stimuli. In a study investigating blood cortisol within 51 hours after rape, women who had a previous history of assault were found to have lower cortisol levels than those who did not (Resnick, Yehuda, Pitman & Foy, 1995). However, in another study including police academy recruits (87% male), the participants with childhood (i.e., before age 14) traumatic experiences did not have significantly different levels of cortisol secretion in response to a traumatic film depicting real-life officers encountering highly stressful incidents (Otte et al., 2005). It has been suggested that the impact of previous trauma on the HPA axis' response to a later stressor is associated with levels of current psychopathology (Cohen, Zohar, & Matar, 2003; Otte et al., 2005). The diverse findings between the two studies may due to the possibility that the sample in Resnick et al. (1995) was more symptomatic than Otte et al. (2005). However, as the impact of previous trauma on reactive cortisol levels has been found in a study involving healthy college students (Luecken, 1998), in addition to current psychopathology, differences in the type of stressor/trauma, gender, age at and elapsed time of trauma should account for the inconsistent findings (Otte et al., 2005).

To examine the relationship between PTSD and the stress responses of HPA axis, a study including war-related PTSD patients found lower levels of corticotropin-releasing hormone (CRH) in the cerebrospinal fluid (CSF) and cortisol in plasma during the viewing of a war-related film, compared to the viewing of a neutral film. Moreover, the decrease of CRH level significantly correlated with subjective worsening of mood (Geracioti et al., 2008). As CRH concentrations have been shown to be elevated at basal level among chronic war-related PTSD patients (Baker et al., 1999; Geracioti et al., 2001), these findings of suppressed HPA

axis during symptom provocation might be a result of a feedback inhibition due to a readily increased brain glucocorticoid receptor occupancy at baseline (Geracioti et al., 2008).

With a different design, a study including adult female survivors of childhood abuse has shown heightened cortisol levels in response to exposure to personalised trauma scripts among those with PTSD, compared to those without PTSD (Elzinga, Schmahl, Vermetten, van Dyck, & Bremner, 2003). As an overall heightened cortisol level has been found among PTSD patients in comparison to their non-PTSD and non-traumatised control groups in a study examining both basal and reactive cortisol levels (Liberzon, Abelson, Flagel, Raz, & Young, 1999), it is unclear whether the increased cortisol level found in the aforementioned study (i.e., Elzinga et al., 2003) reflects a general neuroendocrine excitation of PTSD patients, or their specific response to trauma cues.

Concerning the effects of threat coping strategies involved in different types of trauma, a recent study has investigated salivary cortisol levels in response to an interview about traumatic experiences among war- and torture-related PTSD patients with and without a rape history (Gola et al., 2012). An increased cortisol secretion was found among the ones with a rape history, whereas a decreased cortisol level was shown among those without a rape history. These findings remained after the effect of gender was controlled. As one of the types of trauma characterised by a sense of inability to escape, rape has been associated with the involvement of passive coping strategies such as a shut-down response (Bradley & Lang, 2000; Gola et al., 2012). Animal studies have demonstrated an association between passive coping strategies and higher glucocorticoid releases when encountering threatening stimuli (Korte, Koolhaas, Wingfield, & McEwen, 2005). The authors therefore suggested that the heightened cortisol levels found among the patients with a rape history during the symptom provoking interview might be a replay of the initial neuroendocrine activity related to peri-

traumatic dissociation (Gola et al., 2012). However, as dissociation was not directly assessed in this study, further investigation is needed to support the above arguments.

1.4.3 Cortisol, traumatic memory, and PTSD

Cortisol‘s influences on brain regions involved in memory such as the hippocampus have been reported (Bowirrat et al., 2010). Hence, its relationship with memory has been a topic of research interest (Het, Ramlow, & Wolf, 2005). Animal studies have consistently shown that acute administrations of glucocorticoid receptor agonists before or immediately after inhibitory avoidance training enhance learning (Roozendaal, Okuda, de Quervain, & McGaugh; 2006; Roozendaal, Quirarte, & McGaugh, 2002). However, findings in human studies are relatively inconsistent. In a meta-analysis, administering cortisol before the encoding phase has shown a nonsignificant effect on memory among healthy participants (*d* = .22; Het, Ramlow, & Wolf, 2005). In most of the studies that had adopted recall tasks (cued or free recall) insignificant, or slightly positive effects of cortisol were found (Abercrombie, Kalin, Thurow, Rosenkranz, & Davidson, 2003; Buchanan & Lovallo, 2001; de Quervain, Roozendaal, Nitsch, McGaugh, & Hock, 2000; Hsu, Garside, Massey, & McAllister-Williams, 2003; Rimmele, Domes, Mathiak & Hautzinger, 2003). Interestingly, in a study that assessed recognition memories of faces and objects (Monk & Nelson, 2002), a significantly adverse effect of cortisol administered before learning was shown. Moreover, across studies giving cortisol at different phases, the effect sizes of those that adopted recognition memory tasks were found to be lower than those that assessed recall memory (Het, Ramlow, & Wolf, 2005). The involvement of different brain regions (review paper by Buckner & Wheeler, 2001; Rugg & Yonelinas, 2003) between the two kinds of memory tasks was suggested to explain these different effects of cortisol (Het, Ramlow, & Wolf, 2005).

In the PTSD literature, the differences in cognitive processes and neural mechanisms between intrusive memory and other non-symptom-related memories have been addressed (Brewin, Gregory, Lipton & Burgess, 2010). As memory processing in PTSD is different

from ordinary autobiographical memories (Brewin, 2007; Brewin, 2013; Golier & Yehuda, 1998), applying the abovementioned non-PTSD related findings in predicting cortisol's effect on traumatic memory requires careful considerations regarding the cognitive and neural mechanism underlying different kinds of memory tasks. Like most forms of episodic and semantic memories which are based on recollection, performance in a recall task requires the involvement of both MTL and the cortex associated with supporting retrieval contents. On the other hand, recognition memory may be based on judgments of familiarity, which involves less hippocampal activation, and not necessarily on recollection (Buckner & Wheeler, 2001; Rugg & Yonelinas, 2003). Considering the involuntary nature of intrusion and the suggested reduced involvement of the hippocampus in its development (Brewin et al., 2010), it might be relatively more appropriate to apply the findings of studies that involved recognition rather than recall memory to predict the effect of cortisol on PTSD related memory phenomena.

Consistent with this, an insufficient release of cortisol in the immediate aftermath of trauma has been suggested to be the cause of failure to downregulate catecholamines, such as norepinephrine and epinephrine, generated by excitation of the SNS under stress (Golier & Yehuda, 1998). As catecholamines were associated with memory enhancement (Roozendaal et al., 2006), a low level of cortisol secretion post trauma has hence been hypothesised to contribute to over-consolidation of the traumatic memories and hence the memory symptoms in PTSD (Yehuda & Harvey, 1997).

In order to examine this hypothesis, two prospective studies have investigated the association between cortisol levels and the subsequent development of PTSD in adult trauma victims in the early aftermath of motor vehicle accidents (Delahanty, Raimonde, & Spoonster, 2000; McFarlane, Atchison, & Yehuda, 1997). Supporting the hypothesis, both

studies reported lower cortisol levels among victims who subsequently developed PTSD than victims who did not. Moreover, a low secretion of cortisol was found to mediate the effects of injury severity and prior trauma history on the development of PTSD after motor vehicle accidents (Delahanty, Raimonde, Spoonster, & Cullado, 2003). However, inconsistent results have been found. In one study (McFarlane, Atchison, & Yehuda, 1997), the effect of cortisol level was no longer present after controlling for the duration between the accidents and the blood sampling. In studies involving adolescents aged between 7 and 18, the opposite findings were presented – positive associations were demonstrated between cortisol level within the first 24 hours and PTSD development between 1 and 6 months post trauma (Delahanty, Nugent, Christopher, & Walsh, 2005; Kolaitis et al., 2011; Pervanidou et al., 2007). In considering the aforementioned contradictory findings, recent studies have suggested the investigation of the roles of personality traits (e.g., neuroticism, extraversion; Savic, Knezevic, Damjanovic, Spiric, & Matic, 2012) and coping styles (e.g., emotion-oriented, task-oriented, avoidant-oriented; LeBlanc et al., 2011) in the relationship between cortisol and PTSD.

1.4.4 Effects of the sympathetic nervous system on cortisol and memory

In addition to the HPA axis, traumatic events are usually characterised by SNS activation. As part of the outcome of SNS arousal, an increase of noradrenergic activity has been found to mediate the relationship between cortisol and memory (Roozendaal et al., 2006). For example, the application of β-adrenergic receptor blockers has been found to prevent memory enhancement induced by cortisol in both animals and humans (e.g., Cahill, Prins, Weber, & McGaugh, 1994; McGaugh & Roozendaal, 2009; Roozendaal et al., 2006).

As another measure of SNS activity, salivary alpha-amylase (sAA), an oral cavity enzyme, has been examined. In a study with healthy participants, alpha-amylase level increased soon after a stressful film (corneal transplant surgery) began to be shown, and returned to baseline just after the film commenced (Takai et al., 2004). In a study examining both implicit and explicit memory of emotionally neutral words (Hidalgo et al., 2012), it was shown that while stress induced cortisol did not significantly affect any of the memory tasks, sAA was found to enhance the effect of a priming task on implicit memory, but did not affect the explicit word recall task. The role of sAA in moderating the effect of cortisol on memory has not been examined. This thesis related sAA to cortisol level and trauma-related memory processing for the first time to more completely examine the effects of the two stress-related biological systems and their interactions.

1.5 Individual differences related to diverse psychophysiological responses to trauma

1.5.1 Traumatic history and dissociation

The characteristics of trauma (e.g., frequency, duration, and onset age) have been associated with different presentations of post-traumatic symptoms (van der Kolk et al., 1996). In line with this suggestion, two types of trauma have been proposed (Terr, 1991). The first type refers to long-lasting and repeated trauma such as chronic war or abuse. The second type refers to conditions of single exposure such as road traffic accidents and natural disasters.

Greater vulnerability to developing and maintaining PTSD among the survivors of the first type of trauma has been established (see the meta-analysis by Brewin, Andrews, & Valentine, 2000). Moreover, an association between repeated exposure to trauma and the tendency to engage in extreme psychophysiological responses has been demonstrated. In an animal study, rats exposed to multiple stressors were found to develop over-sensitised neural activity and over-reactions to stimuli (Rau & Fanselow, 2007). Elevations in skin conductance (Giesbrecht, Merckelbach, ter Burg, Cima, & Simeon, 2008), HR (Koopman et al., 2004), and neuroendocrine responses (Otte et al., 2005) have also been shown among individuals who experienced childhood maltreatment and abuse in response to different threatening cues, such as aversive auditory probes, recollection of stressful experiences and a stressful video respectively. In a study adopting a non-threatening manipulation (i.e., riding a stationary bike), less vagal regulation of the heart and insufficient vagal re-engagement to return to a calm physiological state were also observed in adults with prior abusive experiences (Dale et al., 2009). Overall, physiological reactions characterising a more activated defensive system and a lower threshold of fight/flight behaviours have been associated with survivors of long-lasting and repeated traumatic experiences.

Despite such heightened physiological activity, individuals with the first type of traumatic experience have shown a tendency to adopt passive threat coping strategies such as dissociation, denial and numbing. In contrast, individuals with the second type of traumatic experience tend to have more re-experiencing symptoms (Lanius et al., 2010; van der Kolk et al., 1996). Although peri-traumatic dissociation was common, continuous dissociative symptoms are less observed in the latter population (Lanius et al., 2010).

The different psychophysiological reactivity patterns and symptom profiles between individuals with diverse traumatic experiences may contribute to the inconsistencies in studies examining the psychophysiological reactions to trauma cures. For example, as described in 1.3.2, the studies involving rape victims (Griffin, Resick, & Mechanic, 1997) and delinquent adolescents (Koopman et al., 2004) have shown significant negative correlations between peri-traumatic dissociation and HR during the voluntary recall of trauma; However, studies involving survivors of motor vehicle accidents (MVA) and physical assault (PA) victims (Halligan et al., 2006; Nixon et al., 2005) did not show significant correlations between these two measures. It is noteworthy that although some of the MVA and PA survivors in these studies had reported peri-traumatic dissociation, unlike the rape victims and adolescents with many early-life adversities, dissociation may not be a major feature in the symptom profile of these survivors of single-incident trauma. By the time when the retrieval task was introduced in the studies, little dissociation might have been induced, and therefore not be reflected by the HR responses.

1.5.2 Startle response

Just as reactions to the exposure to trauma and traumatic cues have been identified as important factors leading to adaptive or pathological outcomes, so neurobiological features associated with different stress coping styles have been widely explored. For example, to study fear and threat responses, startle responses have been examined to index the defensive motivational system of the brain, with exaggerated startle being indicative of hyperexcitable fear circuits (Rosen & Schulkin, 1998; Vaidyanathan, Patrick, & Cuthbert, 2009). In the PTSD literature, congruent with the fact of being part of the diagnostic criteria in the DSM-IV (American Psychiatric Association, 1994), an increased startle response has been found in PTSD patients (e.g., Butler et al., 1990; Cuthbert et al., 2003; Ladwig et al., 2002). However, a normal or reduced startle has also been reported. Related to the previous section, the diverse nature of traumatic experiences among the participants in different studies may account for the inconsistent results. Agreeing with the animal studies finding decreased startle reflex with repeated exposure to stressors (e.g., Davis, 1996), chronic exposure to traumatic experiences and PTSD symptoms has been associated with a lack of heightened startle reflex (Morgan & Grillon, 1998). Supporting this argument, a reduced startle response has been found in women suffering from chronic interpersonal violence (Medina, Mejia, Schell, Dawson, & Margolin, 2001). Accordingly, a subgroup of PTSD patients with physiological suppression, instead of heightened reactivity, has been suggested (Medina et al., 2001).

While the increase or decrease of startle response as an outcome of trauma and PTSD has been widely studied, little has been examined regarding its role in predicting different stress coping mechanisms and in turn the development of PTSD. In an animal study, rats with pre-existing exaggerated startle response showed more PTSD-like symptoms after stress stimulation (Rasmussen, Crites, & Burke, 2008). An examination has also been done in a

human study (Pole et al., 2009) that assessed pre-trauma startle reactivity of police academy cadets. This study showed that greater startle, indicated by elevated skin conductance, was predictive of more severe posttraumatic symptoms related to police duties a year later. However, startle HR response (sHR) and eye-blink electromyogram recorded at the same time did not show congruent results. Overall, startle responses have been shown to potentially moderate the effect of traumatic stress on PTSD symptom development. However, more studies are needed to further examine this mechanism, as well as the relations between startle responses as a trait and other psychophysiological responses to trauma cues.

1.5.3 Cardiac defence response and traumatic psychophysiological reactions

A neurobiological indicator of stress coping behaviour, the cardiac defence response (CDR; Turpin & Siddle, 1978), has been widely examined in the literature on anxiety disorders. The CDR is a HR response to a sudden loud noise, characterised by two pairs of HR accelerations and decelerations. The first component occurs within the first 10-s after the noise, whereas the second one usually takes place between 20-s and 45-s post-stimulus in the absence of external stimuli.

Given the fact that the first acceleration is triggered by a sudden strong sensory stressor and gets weaker after repetitive exposures, the first component of CDR has been regarded as a startle response in reaction to the sudden noise (Graham & Slaby, 1973). However, more research attention has been given to the second component (e.g., Fernández & Vila, 1989). Individuals who show the second acceleration are classified as Accelerators, whereas those who do not are termed Decelerators (Eves & Gruzelier, 1984). In contrast to Decelerators, who present a baroreceptor modulating response that lowers HR, Accelerators initiate an active inhibition of the baroreceptor modulation (Eves & Gruzelier, 1984) similar to the responses of animals in the acute phase of threat (e.g., Sakaguchi, LeDoux, & Reis, 1983). Moreover, a number of indices of increased adrenergic activity, such as reductions in EKG, T-wave amplitude (Contrada et al., 1989) and an increase in forearm girth (Turpin & Siddle, 1978), have been shown to be coincident with the second component of CDR. Considering the above, this component has been suggested as a human example of the sympathetically mediated fight-flight defensive response (Richards & Eves, 1991; Turpin, 1979; Turpin & Siddle, 1978).

Regarding the physiological characteristics of these two groups in response to threats, a study (Richards & Eves, 1991) using the Strelau Temperament Inventory (STI; Strelau,

Angleitner, Bantelmann, & Ruch, 1990) demonstrated that Accelerators' central nervous system reacts to stressors with less capability, persistence, and flexibility, and is more likely to pass into extreme coping states, such as transmarginal inhibition – a shutdown bodily reaction to an overwhelming stressor originally described by Pavlov (1927). Moreover, considering the nonsignificant difference in the subjective rating of stress levels between Accelerators and Decelerators, it is hypothesised that the Accelerators are individuals who are closer to the physiological position to confront or escape from a stressor, regardless of the amount of subjective fear they experience relative to the Decelerators (Richards & Eves, 1911).

Supporting the above notions, López, Poy, Pastor, Segarra, and Moltó (2009) applied a fear conditioning paradigm and found that, in response to the CS+, Accelerators showed 1) greater HR deceleration and 2) a heightened startle response to electrical shocks. The former represented a freezing-like response similar to bradycardia in animals; The latter suggested heightened reactions to unpleasant stimuli similar to the hyperarousal found in individuals with PTSD. Moreover, the finding that the heightened startle response not only presented in the acquisition stage, but also continued throughout the following three extinction blocks (when the aversive stimulus was no longer present) implied that Accelerators are not only more sensitive to threat but also take longer to recover from the effects of threat.

In addition to the distinct physiological responses to threat in Accelerators and Decelerators, studies have reported that the two groups differ in psychological traits and personality. Accelerators are more introverted, with higher neuroticism traits (Richards & Eves, 1991). Moreover, they are more commonly found among populations with phobia, chronic worry and type A personality (Delgado et al., 2009; Robles Ortega, Marfil, Reyes del Paso, 1995; Ruiz-Padial, Sánchez, Thayer, & Vila, 2002). Considering the above

physiological and psychological characteristics distinguishing Accelerators and Decelerators, it may be beneficial to study CDR as a moderating factor in the context of trauma and PTSD, along with a more sophisticated consideration of individual differences.

1.6 Aims of this thesis

1.6.1 Research questions

In order to provide a more sophisticated picture of psycho-physiological response in the context of trauma, this thesis aimed to examine the following two questions:

Question 1:

How do the ANS and HPA axis respond to the encoding of a trauma? How are these responses associated with the development of involuntary memories?

Question 2:

How do the ANS and HPA axis respond to the voluntary retrieval of traumatic memory? How are these responses associated with emotional engagement?

The psychological implications of these physiological responses were clarified by associating their fluctuations with acute psychological states. Taking account of the inconsistent results found in previous studies as well as their suggestions, a history of multiple trauma, dissociation, sHR, and CDR were examined as potential moderators in answering the two research questions.

1.6.2 Research designs and hypotheses

The research paradigms, and physiological and psychological measurements involved in this thesis are introduced in Chapter Two. In order to answer the first research question, Chapters Three and Four describe the application of an analogue traumatic stimulus to examine the ANS and HPA axis reactions, respectively, among healthy participants during the encoding phase of a trauma. In regard to the second research question, Chapters Five and Six included PTSD patients and assessed the ANS and HPA axis activities, respectively, in response to voluntary retrieval of traumatic memories.

In Chapter Three, as HR decelerations during exposure to an analogue trauma have been found to predict the development of involuntary memories (Holmes, Brewin & Hennessy, 2004), a replication of this result as well as associations between HR decreases, a history of multiple trauma, and dissociation were predicted. As heightened (Pole et al., 2009) and suppressed startle responses (Morgan & Grillon, 1998) have both been suggested to be predictive of more severe PTSD symptoms, exaggerated and suppressed sHR were both hypothesised to correlate greater psychological distress in response to the analogue trauma. Moreover, since Accelerators have been suggested to more easily engage in extreme threat defence responses (Richards & Eves, 1991), they were predicted to show a greater HR reduction during the analogue trauma. This represents a more extreme and passive psychophysiological reaction, as the current study design prevents an active defence taking place.

In Chapter Four, an increase in cortisol was predicted as a stress response of the HPA axis to the analogue trauma (Bowirrat et al., 2010; Het, Ramlow, & Wolf, 2005; Takai et al., 2004). Based on a previous hypothesis (Yehuda & Harvey, 1997), an association between lowered cortisol secretion and the development of involuntary memories was predicted.

Moreover, because the association between passive threat coping strategies and higher glucocorticoid releases in response to threat has been suggested (Gola et al., 2012; Korte et al., 2005), individuals who were more dissociative, with a history of multiple trauma, and with suppressed sHR were predicted to have greater cortisol secretion after the analogue trauma. Additionally, based on the literature (Richards & Eves, 1991), the presentation of a highly activated HPA axis among the Accelerators was hypothesised.

In Chapter Five, following previous findings (Halligan et al., 2006), an increase in HR was predicted during the voluntary retrieval of traumatic memories. Additionally, as the association between dissociation and smaller HR increases in response to the recall of trauma has been suggested (Griffin, Resick & Mechanic, 1997), low emotional activations, a history of multiple traumas, dissociation, as well as a restricted sHR were hypothesised to be predictive of smaller HR increases during voluntary memory retrieval. Following the previous study showing Accelerators' greater decreases in HR at the presentation of a threatening stimulus (López et al., 2009), a greater drop in HR during voluntary recall was predicted among these individuals.

In Chapter Six, as decreases in HPA axis activity have been found among PTSD patients in response to symptom provoking stimuli (Geracioti et al., 2008), a decrease in cortisol level was hypothesised after the retrieval of a traumatic memory. Moreover, taking account of the moderating role of passive threat coping strategies suggested in the literature (Gola et al., 2012), the patients with more severe dissociative symptoms, a history of multiple traumas, and suppressed sHR were predicted to show greater activation of the HPA axis, compared to the less dissociative patients. No specific hypothesis was made regarding the role of CDR in this context.

Chapter 2: Introduction to the research methods

This chapter first introduces the trauma film paradigm, which was used in Chapter Three and Four to examine the encoding of traumatic memory, and its related memory measures in 2.1. Next, the methods adopted in Chapter Five and Six to facilitate the voluntary retrieval of traumatic memories, as well as the identification of peri-retrieval psychological states are introduced in 2.2. All physiological measures involved in this thesis are introduced in 2.3, followed by the psychological measures in 2.4.

2.1 The trauma film paradigm and related memory assessments

The trauma film paradigm is an experimental tool adopted in trauma-related studies since the 1960s (e.g., Lazarus & Alfert, 1964). Its basic methodological components involve 1) baseline assessment of pre-existing vulnerabilities or traits, 2) viewing a short film depicting traumatic events, 3) pre-, peri-, and post-film assessment of state psychological and/or physiological measures, and 4) tracking of intrusive memories with the intrusion diary. These designs enable the investigation of psychological and physiological peri-traumatic mechanisms. In a review study (Holmes & Bourne, 2008), the capacity of the trauma film paradigm to induce intrusions in the laboratory has been demonstrated. Moreover, the amplifying and attenuating factors of the intrusive memories created in the laboratory are in line with those found in studies involving real life trauma (Holmes & Bourne, 2008). Overall, the validity of adopting the trauma film paradigm has been well supported, despite the limitations of using an analogue situation to represent real-life trauma. Details of the materials adopted in our studies are summarised in the following sections.

2.1.1 Viewing of the trauma film

We adopted a 13-min-40-sec trauma film applied in a previous study (Holmes, Brewin, & Hennessy, 2004). This film contains real-life footage from traumatic car accidents. It was presented to participants on a 28.5-x-40-cm computer monitor. The sound was played with headphones. The traumatic film consists of five scenes of different car accidents containing horrific images of emergency service personnel working to extract trapped victims and move dead bodies, injured individuals screaming, and body parts among vehicle wreckage. Before each scene, there was a brief narration (voiceover, without images), introducing the context of the accident and background of the victims involved. Electrocardiography (ECG) data was recorded throughout the film viewing. Participants were asked to stay still during the film and were told that any movement might result in artifacts in ECG recording. Moreover, they were also asked to watch the film concentrating as much as possible, imagining themselves being present and witnessing the occurrences firsthand.

2.1.2 Assessments of traumatic memories

Intrusion diary

The intrusion diary and relevant methods applied in the previous study (Holmes, Brewin, & Hennessy, 2004) were adopted. Participants were instructed to use a tabular diary to record involuntary memories (i.e., intrusions) of the trauma film for 7 days after the film viewing. They were informed of the definition of intrusion as "unintended and spontaneous, rather than deliberate, memories/thoughts/images about the film that easily capture attention and may interfere with ongoing activities." Participants were asked to note the timing of every intrusion, a brief description of each intrusion's contents, and whether the intrusive contents were mainly images, thoughts, or a mixture of both. Additionally, ratings of vividness and distress level (0 = not at all, 10 = extremely) of each intrusion were included.

The frequency of intrusive thoughts was the number of times when intrusions that took the form of mainly thoughts occurred over the week. Similarly, the frequency of imagery intrusions was calculated by summing up the numbers of times when pure imagery intrusions and those of a mixture of both images and thoughts occurred. The vividness of intrusion was derived from averaging the vividness ratings of the occurrences that were mainly images or a mixture of both images and thoughts.

In order to enhance the completion of the diary, participants were advised to carry the diary with them and record each occurrence as soon as possible. A text message was also sent at 9 p.m. each day as a reminder for checking the completion of the diary for that particular day. In the follow-up experimental session, a diary compliance rating was made.

Identification task

Similar to the previous approach (Holmes, Brewin, & Hennessy, 2004), based on the description of participants in the intrusion diary, the parts of the film, where intrusions were likely to be from were replayed to them. Participants were asked to point out the exact timing in the video where their intrusions were from. This was used to locate the intrusive and non-intrusive sequences of the film for each participant.

Recognition task

Recognition memory was assessed for each scene in the traumatic film following the procedures of Bisby, Brewin, Leitz, and Curran (2009). A questionnaire involving 30 multiple-choice questions, with one correct and three plausible choices were administered. For each scene of the trauma film, 6 questions were asked. Three of these questions tapped gist recognition memory (e.g., the cause of the accident), while the remaining three tapped detailed recognition memory (e.g., the colour of the shirt that a victim wears).

2.2 Voluntary retrieval of memories and the peri-retrieval psychological states

2.2.1 Instructions for the voluntary retrieval of memories

The voluntary retrieval task was developed in the current study, with reference to a previous method (Halligan et al., 2006). It involved verbal recollection of two types of memories. The participants were first asked to recall and speak about a neutral memory for 5 minutes. This procedure served as a baseline measure as well as a preparation for the following recollection of traumatic memory. The instructions used to facilitate the voluntary retrieval of a neutral event were given in both verbal and written form as below:

> *'There are many routines in daily life, which are familiar to us, but do not cause significant emotional reactions to us. Examples of routines include tidying the house, doing the laundry, walking or taking a bus/tube/train ride to work or supermarkets... etc. Now, please choose a routine that you are familiar with, but do not significantly emotionally react to.*
>
> *When you are ready, with your eyes open, please take yourself back to the time when you last did this. Begin just before it started. Go through everything that happened from start to finish. Include details about the surroundings. Describe everything you remember seeing, smelling, hearing, doing, feeling, and thinking about at each point in time.*
>
> *You will be given 5 minutes for this task. Please try your best to keep recalling during this period of time. A timer will ring to remind both of us when the time is up.'*

After the neutral retrieval, participants were asked to recall and speak about a piece of traumatic memory. They were given the option to choose an incident they felt comfortable to

talk about, which did not have to be the most stressful one. Similar instructions to these used in a previous study (Halligan et al., 2006) were given in both verbal and written form as below.

> *'In one of the questionnaires you have completed, you identified a (a few) traumatic event(s) that you had experienced. With your eyes open, I would like you to take yourself back to the time of one of the events, and remember it as vividly as you can. Begin just before it took place. Go through everything that happened from start to finish. Include details about the surroundings. Describe everything you remember seeing, smelling, hearing, doing, feeling, and thinking about at each point in time.*
>
> *You will be given 15 minutes for this task. Please try your best to keep recalling during this period of time. A timer will ring to remind both of us when the time is up.'*

ECG data were collected throughout both types of memory retrieval. The participants were asked to sit facing a camera. Video recording was performed with their permission. With respect to the ethical issues of inducing traumatic memories in the laboratory, we note that previous studies (e.g., Halligan et al., 2006; Hellawell & Brewin, 2004;) adopting similar methods did not report ongoing distress subsequent to the end of the experiments. Moreover, the instructions for this task are similar to the ones used in exposure therapies, which are safe and suitable to be applied to PTSD patients. Patients recognised as not suitable for exposure therapies by their doctors or therapists were not recruited. Moreover, before the study, all participants were fully informed of the nature of the tasks and of their right to withdraw at any time. After the experiments, participants were carefully debriefed and were encouraged to

contact the investigator and/or their doctors/therapists if distress occurred at any time as a result of the experiment.

2.2.2 Identification of peri-retrieval dissociation and flashback

After both voluntary retrieval tasks, participants were asked to watch the video taken during the retrieval of traumatic memory, in order to point out the exact periods of time when they had flashbacks and when they experienced dissociation during the retrieval. The instructions for this task and the definitions of flashback and dissociation were developed in the current thesis. They were fully explained in verbal and written formats as below.

'Please identify the times when these two mental states occurred to you while you were just recalling the memory of the traumatic incident.

1. *Flashbacks*

   During the recall, were there times when you felt that the original vivid feelings or memories of the event (e.g., images, sounds, smells, emotional and physiological feelings) were coming back to you, as if you were experiencing the event again?

2. *Dissociation*

   During the recall, were there times...

   when you felt as if the surrounding environment (this room) was unreal, or

   when you felt as if you were looking at things from outside of your body, or

   when you felt blanked out and it was difficult to make sense of what was going on?'

These procedures were used to locate the reexperiencing and dissociative sequences during the voluntary retrieval for each participant. In circumstances when the dissociative state during the retrieval was a repetition of dissociation which had originally occurred during the recalled trauma, a mixture of flashback and dissociation was recorded.

2.3 Physiological measures

2.3.1 Psychophysiological reactivity test

In order to assess sHR responses and the CDR, a psychophysiological reactivity test (Eves & Gruzelier, 1984; López et al., 2009; Turpin & Siddle, 1978) was conducted at the beginning of each study. Participants were told that the aim of this task was to examine the effect of sound on relaxation and therefore they might encounter an unexpected loud noise. However, the only thing they needed to do was to try to be relaxed. After the instruction, a 6-min resting period was given with a startle probe (i.e., a white noise which was 500-ms long, and 110dB loud with instantaneous risetime) presented through a set of headphones at the end, and followed by an HR recording of 80-s. Participants were categorised into different sub-groups of sHR and CDR, based on their HR patterns over the first-10-second and the 20^{th}-to-45^{th}-second post-startle periods, respectively. Statistical methods and results of the grouping are summarised in the methods section of the following chapters.

2.3.2 Physiological data acquisition

Cardiovascular activity: HR and HRV

The ECG signal was recorded from two disposable electrodes attached to the participants' chest. It was sampled continuously at 512 Hz with the Actiwave Cardio system (Camntech, Cambridge, UK). The ECG data were examined and derived using VivoSense software (VivoNoetics, San Diego, CA, USA). Artifacts in the data due to misdetected R-waves were easily recognized as outliers from the average HR curve and were manually deleted and interpolated using the facility of the software (Halligan et al., 2006). Data with more than 0.3% corrected R-R intervals were excluded (Hodson, Harnden, Roberts, Dennis, & Frayn, 2010; Vaile et al., 2001).

HR and HRV parameters were derived for selected time periods (see the Methods section of each chapter for details). The frequency domain indices of HRV were selected. The power spectrum density (PSD) of the R-R intervals was computed using a Fast Fourier Transformation, which decomposes the variance in the frequency domain (ms^2/Hz). Following the guidelines for frequency-domain computations of HRV (Task Force, 1996), spectral power was divided into low-frequency (LF-HRV, 0.04-0.14 Hz), and high-frequency (HF-HRV, 0.15-0.40 Hz). Because of the controversy about a possible parasympathetic contribution to the level of LF power (Task Force, 1996), the ratio of LF/HF was calculated as an index of sympathovagal balance. Moreover, because total power varies greatly between participants, power was determined in both absolute units and normalised units. The power of normalised units was calculated by dividing the absolute power of a given component by the total power minus the very low frequency (VLF-HRV, 0.01-0.04 Hz) component.

Hormonal and SNS activity: salivary cortisol and alpha-amylase

Salivary samples were collected with salivettes (Sarstedt, Leicester, UK). Participants were asked to chew each salivette for 2 minutes. Samples were then stored at -20C before the biochemical analysis took place. In order to control for circadian fluctuations (Nater et al., 2007), studies were arranged after 1:30 p.m. After thawing, saliva was centrifuged at 3000 rpm for 5 minutes before free cortisol was analysed using an immuno-assay with time-resolved fluorescence detection (Dressendorfer, Kirschbaum, Rohde, Stahl, & Strasburger, 1992). The level of salivary alpha-amylase was analysed using a kinetic colorimetric test (Nater et al., 2006).

2.4 Psychological measures

2.4.1 Life adversity, trauma, and subclinical PTSD symptom assessment

Life Stressor Checklist – Revised (LSC-R)

The LSC-R (Wolfe & Kimerling, 1997) is a 31-item self-report measure commonly used to assess participants' life adversity and trauma history. Thirty traumatic life events, including natural disasters, war, death of a loved one, physical, and sexual assaults etc are assessed. A yes/no question is first asked to inquire whether one has experienced a certain kind of event. For an endorsed event, the number of times when such adversity happened is asked, as well as the ages when it first and last occurred. Moreover, the belief that oneself/a loved one was in danger of serious harm is assessed. In the last question, respondents are given a chance to identify any unlisted life adversities.

Posttraumatic Stress Diagnostic Scale (PDS)

The PDS (Foa, 1995) is a 49-item self-report measure of traumatic experiences and related PTSD symptoms commonly adopted in trauma research. The first section presents a short checklist identifying potential traumatic events experienced by respondents. The second section asks respondents to indicate one of these events, which has troubled them the most at the time of answering the questionnaire. They then identify the elapsed time and rate Criterion A for PTSD in DSM-IV (American Psychiatric Association, 1994) based on the aftermath caused by the specified event. In the third and fourth sections, Criteria B, C, D, and E for PTSD are also rated according to the event picked out in section two. PTSD symptom severity is indicated by the sum of all the items for Criteria B, C, and D. The scores range between 0 and 51, with the higher scores indicating greater severity. The validity of the PDS (Foa, Cashman, Jaycox & Perry, 1997) has been supported by good diagnostic agreement

with the Structured Clinical Interview for DSM-IV (Spitzer, Williams, Gibbon, & First, 1990). Satisfactory reliability was found in the current sample (Cronbach's $\alpha = .86$).

Structured Clinical Interview for DSM-IV (SCID)

The SCID (First, Gibbon, Spitzer, Williams, & Benjamin, 1997) is a standardised semi-structured interview designed to identify the presence of Axis I psychopathology. It has been widely adopted in psychology research, and was applied in Chapter Five and Six to assess PTSD, the relevant comorbidity, and exclusion conditions (i.e., psychotic disorders).

2.4.2 Psychological trait and state measures

State Trait Anxiety Inventory (STAI)

The STAI (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) is a widely used self-report scale for anxiety. The first subscale measures state, whereas the second subscale measures trait anxiety. They each have 20 items, and the anxiety levels are indicated by the sum of all items in the corresponding subscale. The scores range between 20 and 80 with higher scores indicating greater anxiety. The validity of the STAI has been supported by its ability to discriminate high vs. low stress situations and its agreement with other anxiety assessment tools (Metzger, 1976; Spielberger, 1989). Satisfactory reliability was found in the current sample (Cronbach's α range between .94 and .96).

Dissociative Experiences Scale (DES)

The DES (Carlson & Putnam, 1993) is a 28-item questionnaire designed to examine trait dissociation. It is composed of three aspects of dissociation: Amnesia (e.g., finding oneself in a place and having no idea how he or she got there), Depersonalization-derealization (e.g., feeling ones body does not seem to belong to him or herself), and absorption (e.g., finding oneself so involved in a fantasy or daydream that it feels as though it were really happening). For each item, participants are required to indicate the percentage of time when they have a given experience in daily life (range between 0 and 100%). A tendency toward dissociation is indicated by averaging the percentage scores with higher scores indicating more frequent occurrence of dissociation. The DES is widely adopted in trauma-related research. Evidence suggesting its good validity has been reviewed by Dubester and Braun (1995). Satisfactory reliability was found in the current sample (Cronbach's $\alpha = .94$).

Peritraumatic Dissociative Experiences Questionnaire (PDEQ)

The PDEQ (Marmar, Weiss, & Metzler, 1997) is a measure that assesses dissociative status during an index event. This widely applied measure has 10 items. The responses are given on a 5-point Likert-type scale (from *not at all true* to *extremely true*). It was applied in Chapters Five and Six to assess peri-traumatic dissociation that happened during the most distressing trauma identified in the second section of the PDS.

Dissociative State Scale (DSS)

The DSS (adapted from Bremner et al., 1998) is a 19-item self-report measurement commonly applied to assess state dissociation. Its covered areas include depersonalisation, derealisation, and amnesia. For each item, participants are required to rate on a five-point scale anchored with 0 (not at all) and 4 (extremely) based on their feeling at the particular moment in time when they are given the measure. The level of state dissociation was indicated by the sum of all items. The scores range between 0 and 76, with higher scores indicating greater levels. Satisfactory reliability was found in the current sample (Cronbach's α range between .86 and .92). Validity has been supported by the findings that healthy participants scored lower than PTSD patients (Bremner et al., 1998).

Mood Rating Scale

The mood rating scale is an 11-point visual analogue scale (0 = not at all, 10 = extremely) designed in the current thesis to assess participants' state of fear, calm, and feeling of threat. Higher scores indicate stronger feelings of a given mood.

Chapter 3: ANS, traumatic memory, and individual differences

3.1 Introduction and hypotheses

3.1.1 What does a peri-traumatic HR decrease indicate and predict?

Involuntary memories are a hallmark of PTSD, but little is known of what causes them. Previous research has suggested HR falls during the encoding of trauma stimuli that later return as intrusive memories (Holmes, Brewin, & Hennessy, 2004). However, further investigations in terms of the ANS's contribution to HR fluctuation, as well as the psychological implication of such HR reduction, have not been conducted.

In the defence cascade model (Bradley & Lang, 2000) a cardiac deceleration representing an orienting and information gathering response has been suggested to occur at the initial proximity of a threat. With an approach of threat, an increased HR associated with the activation of the SNS is indicative of an active defense (fight or flight). However, when escape becomes, or is perceived as impossible, a passive defence mode featured by physical deactivations such as freezing or bradycardia is more likely to be observed (Kaada, 1987). This model suggests a decrease of cardiac activity in both the initial and final stages of defence. As such, it was unclear whether the decrease in HR during trauma film viewing (Holmes, Brewin, & Hennessy, 2004) was a product of a temporary shut-down of higher-level cognitive function (similar to the freezing response), or an indicator of orienting.

Dissociation is a passive threat coping response, which commonly occurs peri- and posttrauma. Due to its associated limited involvement of high-level cognitive function, it has been hypothesised to lead to weaker C-reps and to impede the normal integration of S-reps and C-reps (Brewin et al., 2010), and has been found to impair memory performance (Brewin, Ma, & Colson, 2013; Brewin & Mersaditabari, 2013) and to reliably predict PTSD

symptoms (Ozer, Best, Lipsey, & Weiss, 2003). Considering the significant correlation between dissociation and the development of PTSD, the current study sought to examine its relationship with the cardiovascular activity related to trauma film viewing. Other trauma-related psychological measures, such as subclinical PTSD symptoms, trait and state anxiety, acute states of fear and calm were also examined.

In order to investigate these questions, the study adopted the trauma film paradigm, with ECG data being recorded among healthy participants. Cardiovascular activity (i.e., the SNS, vagal system, HR) during the trauma film was examined. Following the approach in a previous study (Griffin, Resick, Mechanic, 1997), peri-film HR levels were related to 1) pre-existing psychological traits (i.e., dissociation and anxiety) and subclinical PTSD symptoms, and 2) peri-film psychological states (i.e., state dissociation, anxiety, calm and fear). Moreover, to replicate the previous findings (Holmes, Brewin & Hennessy, 2004), we examined 3) the relationships between peri-film HR decreases and the memories for the trauma film. Considering the clinical importance of the vividness of intrusion and the fact that it has received relatively little research attention compared with intrusion frequency, both aspects of memory for the film were assessed.

It was hypothesised that participants with high pre-existing subclinical PTSD symptoms would show higher HR during the film, as higher HR on exposure to traumatic stimuli has been well established in PTSD patients (e.g., Adenauer et al., 2010). Similarly, because anxiety has been considered to be associated with the activation of the sympathetic nervous system under stress (e.g., Takai et al., 2004), individuals with higher trait and state anxiety were predicted to present with higher HR during the film. Contrastingly, based on previous findings associating dissociation and lowered HR (e.g., Griffin, Resick, Mechanic, 1997), both trait and state dissociation were expected to correlate with lower HR during film

viewing. Moreover, following the previous study (Holmes, Brewin & Hennessy, 2004), a correlation between HR decreases and more involuntary memories was predicted.

3.1.2 Are there different underlying processes for C- and S-memories?

Diverse memory representations and processes have been proposed in the DRT (Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996). The memory products of each process contain different formats and contents. For example, involuntary images of trauma have been considered as a product of the S-memory system. In contrast, despite being a form of intrusion, the semantic nature of intrusive thoughts suggests the involvement of higher-level cognitive functions and a more prominent involvement of C-reps. Supporting these arguments, a previous study (Hagenaars, Brewin, van Minnen, Holmes, & Hoogduin, 2010) found that whereas intrusive images were increased by the laboratory manipulation of freezing behaviour (i.e., non-movement; Hagenaars, van Minnen, Holmes, Brewin, & Hoogduin, 2008) and were associated with negative peri-traumatic psychological states, intrusive thoughts did not show the same patterns. This finding has been regarded as evidence suggesting differences underlying the processes supporting involuntary sensory images and verbal thoughts (Hagenaars et al., 2010).

Other evidence for diverse memory processes has been found in a previous study separately examining gist and detail recognition memory for a trauma film (Bisby et al., 2009). The results showed that, while a moderate dose of alcohol was related to the development of intrusions, it decreased recognition memory for gist, but not detail information about the trauma film. Gist ideas (e.g., when, where, how, who) and abstract meanings (e.g., the impact on one's life) of an incident have been suggested to be important aspects of C-memories, whereas sensory details involved in an incident were considered part of S-memories (Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996). The existing results therefore suggested a selective impairing effect of alcohol on C-memories.

In order to further examine the abovementioned memory diversity and to investigate the distinct characteristics of C-memories and S-memories, involuntary memories taking the form of abstract thoughts vs. sensory formats (e.g., images, sounds), as well as recognition memory for gist vs. sensory details were examined and related to the ANS activities separately. Significant correlations between involuntary images and detailed recognition memory, as well as between involuntary thoughts and gist recognition memory were predicted. Because a decrease in HR during the trauma film viewing has been related to the strengthening of S-memories (Holmes, Brewin & Hennessy, 2004), it was hypothesised to relate to greater involuntary images and detailed recognition memories, but not to intrusive thoughts or gist recognition memories.

3.1.3 Are there individual differences in peri-traumatic HR and its implications?

As introduced in 1.5.2 and 1.5.3, extreme levels of sHR, and the presentation of a secondary HR acceleration in the CDR pattern (i.e., being classified as an Accelerator) have been associated with greater vulnerability to develop PTSD (Pole et al., 2009), as well as stronger and more enduring fear reactions (López et al., 2009). The third aim of the study was therefore to examine the roles of these psychophysiological features in the process of traumatic memory. Specifically, this study examined 1) whether individuals with different sHR levels, and CDR patterns varied in their HR, psychological states, and memory in response to the trauma film. 2) The moderating roles of these factors in the relationships between HR, psychological states and traumatic memories were also examined.

While the current study attempted to associate individuals' defence patterns to a startle stimulus with their defence responses to the trauma film, HR during the film viewing was predicted to be positively correlated with the level of sHR. Based on the findings of more severe symptoms among individuals with heightened and suppressed sHR (Morgan & Grillon, 1998; Pole et al., 2009), stronger psychological impacts of the trauma film were expected among these individuals. Similarly, as a greater vulnerability to extreme fear responses has been demonstrated among the Accelerators, they were expected to show more negative psychological consequences related to the trauma film. On the other hand, because of the lack of existing studies, specific predictions were not made regarding the moderating roles of traumatic experiences, sHR, and CDR in the correlations between HR and the psychological state and memory measures.

3.2 Methods

3.2.1 Participants and procedures

Non-smoking healthy native English speakers aged between 18 and 40 with a body mass index range of 17.5 to 30 were recruited. Volunteers with a history of cardiovascular illness, any other significant physiological illness, or currently taking any medication including contraceptives, were excluded. Because the materials in this study contained graphic footages from car accidents, those who had experienced/witnessed serious road traffic accidents, had close others seriously injured/killed in road traffic accidents, and individuals with a history of any mental disorder were excluded. Eighty-seven participants passed the inclusion and exclusion criteria and completed the study. They were paid 15 pounds as a reward for their participation. All participants provided written informed consent. This study was approved by the UCL Psychology and Language Sciences Ethics Committee (Appendix 9 and 10).

Priori power calculations based on a 3 (groups) by 3 (times) mixed design ANOVA, and a stepwise multiple regression (5 predicting variables overall) with an effect size of 0.18 and a power of 0.8 suggested a sample size of 66 and a sample size of 57, respectively. Among the 87 volunteers who have completed the study, the data from 10 of them were excluded due to a high number of artifacts in the ECG data (i.e., more than 3% corrected R-R intervals; Hodson, Harnden, Roberts, Dennis, & Frayn, 2010; Vaile et al., 2001). Another 13 participants were excluded because of procedural issues (e.g., failed to keep the intrusion diary at the end of each day, experienced actual traumatic or stressful events between the two study sessions, errors in the volume setting of the auditory startle trigger in the psychophysiological reactivity test). This resulted in a final sample size of 64 (male = 33). Subgroups were defined at the data analysis stage based on sHR and CDR patterns in the

psychophysiological reactivity test. See 3.3.3 for details regarding the classification approaches.

All participants were asked to be free of caffeine, alcohol and medication 24 hours before the study to avoid the effects of these substances. As shown in Figure 3.1, sub-clinical PTSD symptoms and the trauma-related traits (i.e., anxiety and dissociation) were assessed at the beginning. Next, participants were fitted with the ECG electrodes and given the psychophysiological reactivity test, after which they were reassured that no more sudden noise would be delivered. Another 6-minute pre-film resting period was then introduced with no task other than resting given in the first 2 minutes, and the pre-film psychological state measures (i.e., anxiety, dissociation, fear, and calmness) given at the third minute. The trauma film was then shown to the participants with a 10-min post-film resting period. The peri-film psychological state measures were given at the very beginning of this period and finished within the first 4 minutes, leaving the next 6 minutes pure rest. The post-film psychological state measures were given at the end of this resting period. Participants were instructed in the usage of the intrusion diary and asked to keep it for 7 days at the end of the first session. A text message was sent at 9 p.m. each day to remind the participants to check the completion of the diary for that particular day. In the follow-up session on the 8th day, the diary was returned and participants carried out the recognition and identification tasks.

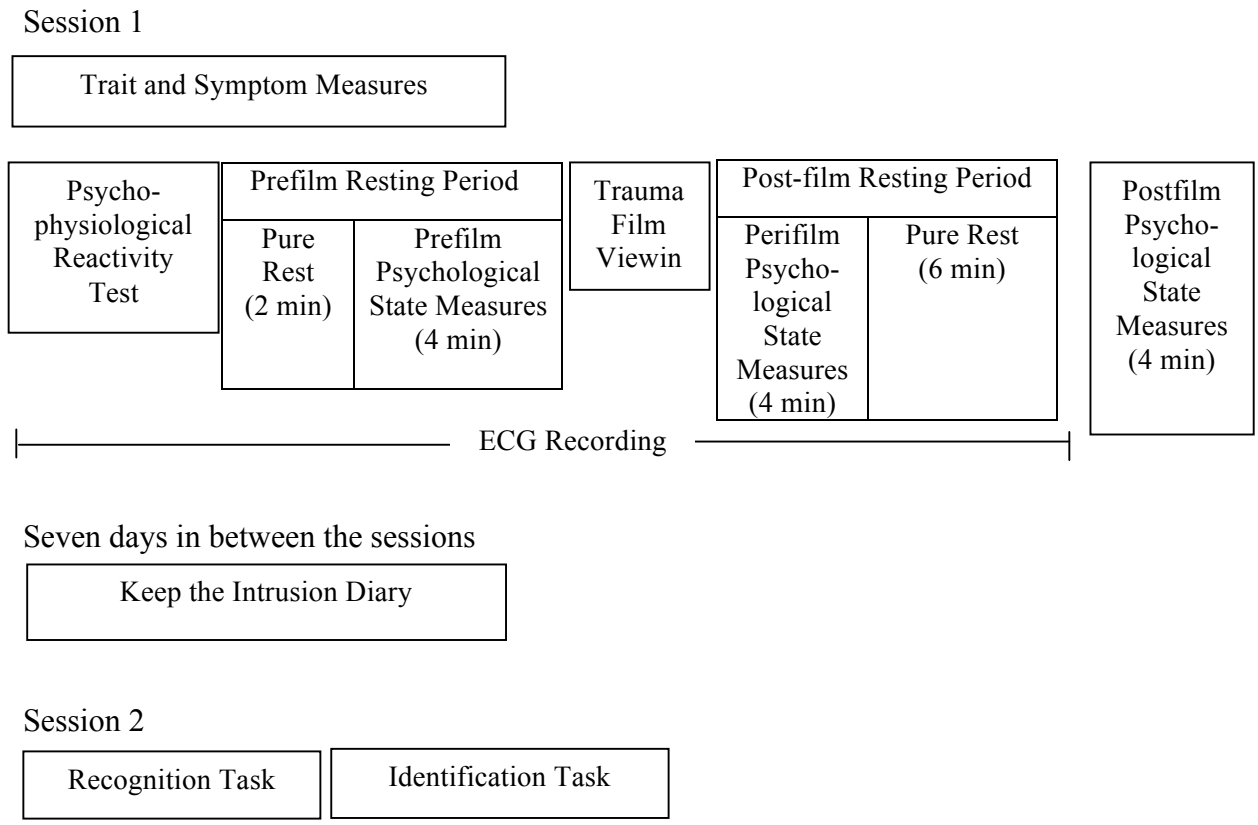


Figure 3.1 *Timeline of the Procedures*

3.2.2 Analytic strategy

All statistical analyses were performed with SPSS version 18 (SPSS Inc, Chicago, IL, USA). Scores greater than 3 standard deviations above the mean were changed to one unit larger than the greatest non-extreme score in the given variable, whereas scores smaller than 3 standard deviations below the mean were changed to one unit smaller than the smallest non-extreme score (Tabachnick & Fidell, 1996). For example, an outlying score of 27.0 would be changed to 11.5 if the highest non-extreme score 10.5. The variables and number of cases with such changes were: trait dissociation (2 cases); pre-film anxiety (1 case); pre- (2 cases), peri- (2 cases) and post-film dissociation (1 case); frequency of intrusive images (2 cases); and pre-film HR (1 case). Normality of distributions was examined by dividing the absolute values of skewness by the standard error of skewness. For variables with values larger than 3 from this calculation, square root transformation was performed.

As movement affects cardiovascular activities (Mulder, 1992), the calculation of HR and HRV indices at the pre- and post-film phases excluded periods when the participants were filling questionnaires. This means, for the pre-film and post-film phases respectively, only the data in the first 2 minutes and the last 6 minutes were included. Mean pre- and post-film HR were calculated by averaging HR during these periods, whereas mean peri-film HR was derived from averaging HR throughout the whole film. The pre-film HRV was derived from the first 2 minutes of the pre-film phase. However, in order to make equal length for HRV calculation, the peri- and post-film HRV were derived from averaging the data of six 2-min segments and three 2-min segments within the given periods.

Among those who had at least one imagery intrusion and were able to identify the film sequences that later intruded (n = 54), HR was averaged during these sequences and the sequences that did not intrude separately. Two forms of HR change were calculated: The

'overall peri-film HR change' was estimated by subtracting the pre-film HR from the peri-film HR. To calculate the 'intrusive sequence HR change', the mean HR in the non-intrusive sequences was first subtracted from the mean HR in the intrusive sequences of the film. In order to control for the overall amount of variation caused by the film, this HR difference score was then divided by the absolute value of the 'overall peri-film HR change'.

Given the extensive literature linking dissociation to lower HR, as well as the lower HR found during intrusive sequences by Holmes et al. (2004), 1-tailed t-tests were applied to examine the differences between pre- and peri-film HR, as well as between HR during the intrusive and non-intrusive sequences. Pearson's correlations were used to examine the association between HR and psychological states at the corresponding phases (e.g., pre-film HR with pre-film anxiety, peri-film HR with peri-film fear). Pearson's correlations were also performed to examine whether the 'overall peri-film HR change' and/or 'intrusive sequence HR change' were predictive of the measures of intrusive and recognition memory (i.e., intrusive thoughts and images, gist and detail recognition memory). Further, the relationships between these memory measures were examined by Pearson's correlations.

Next, to classify participants by their sHR and CDR patterns, Ward's hierarchical cluster analysis was adopted. This method has been used in previous studies (López et al., 2009; Milligan & Isaac, 1980) for its ability to produce clusters with similar numbers in small data sets. Following López et al. (2009), the variables used in this analysis were the second-by-second HR changes during the first 10 seconds (for sHR groups), and the 20-to-45-sec (for CDR groups) after the onset of the white noise in the psychophysiological reactivity test (relative to the mean HR in the 15-sec pre-stimulus period). One-way ANOVAs and chi-square tests were conducted initially to examine the differences of demographic, physiological, and psychological characteristics between the groups.

To examine the effects of the trauma film on different groups, two-way (group x time) mixed design ANOVAs were performed on HR and HRV indices, and one-way ANOVAs were used to examine group differences in memories for the trauma film. Given the multiple levels on the Group and Time factors as well as the specific design of the study, tests of linear and quadratic effects replaced tests of main effects. For all of the F tests, linear and quadratic effects were examined. Homogeneity of variance was assessed by Levene's statistic, while sphericity was examined with Mauchly's test. When the assumption of sphericity was not met, the uncorrected degrees of freedom, Epsilon (Greenhouse-Geisser), corrected F, and corrected p values were reported. Finally, to investigate the moderating effects of the type of traumatic experiences, sHR, and CDR on the relationships between HR, psychological states, and intrusive memories, stepwise multiple regressions were used.

A few exploratory analyses were performed. First, 2-tailed t-tests were used to compare the frequency of intrusive thoughts vs. images, the performance in gist vs. detail recognition memory, and recognition memory for the scenes that later became intrusive versus those that did not. Second, two-way (group x time) mixed design ANOVAs were used to examine the effect of the film on psychological states and individual differences in such reactions. Similar to the two-way mixed design ANOVAs on HR and HRV, tests of linear and quadratic effects replaced main effects.

3.3 Results

3.3.1 Relationships between involuntary and recognition trauma memories

The intrusive sequences in the film were readily identified by most of the participants, except for two whose involuntary images were too vague and difficult to specify. On average, materials from 2.08 (*SD* = 1.23) scenes in the trauma film involuntarily occurred. The overall duration of the intrusive sequences reported by each participant ranged between 2 to 173 seconds (*M* = 47.09; *SD* = 40.40). Descriptive data for the memory measures are summarised in Table 3.1. Involuntary images were significantly more frequently reported than thoughts ($t(63) = 7.97$, $p < .001$). Gist information was recognised significantly better than details ($t(63) = 20.01$, $p < .001$). Detail recognition memory was better for the scenes that involuntarily occurred than those that did not ($t(53) = 2.79$, $p < .01$). However, the performance of gist recognition memory was not significantly different ($t(53) = 1.49$, $p = .14$).

As for the relationships between the memory measures (see Table 3.2), the frequency of imagery involuntary memories was significantly positively correlated with detail, but not with gist recognition memory. A marginally significant partial correlation was found between the frequency of imagery involuntary memories and detail recognition memory after controlling for gist recognition memory ($r = .24$, $p = .06$). Frequency of involuntary thought was not significantly correlated with either type of recognition memory.

3.3.2 Relationships between HR, psychological states and traumatic memories

Descriptive data for HR, psychological states, and memory measures in the overall sample are summarised in Table 3.1. None of the correlations between HR and psychological states at the corresponding phases (e.g., pre-film HR and pre-film anxiety; post-film HR and post-film dissociation) was significant (largest r = -.18, p = .16).

Comparisons of HR levels at different phases with 1-tailed t-tests showed that the difference between pre- and peri-film HR ($t(63) = 1.27$, $p = .21$) was not significant. However, the drop in HR associated with intrusive versus non-intrusive sequences was significant ($t(53) = -2.33$, $p < .05$).

The 'overall peri-film HR change' did not show any significant correlations with the memory measures (see Table 3.2). Nevertheless, 'intrusive sequence HR change' significantly and negatively correlated with the frequency of imagery involuntary memory and detail recognition memory. The more reduction in HR during the intrusive relative to the non-intrusive sequences, the greater frequency of involuntary images and better recognition of details were. The correlation between 'intrusive sequence HR change' and frequency of involuntary images was marginally significant after controlling for vividness level and frequency of involuntary thoughts (r = -.32, p = .05). The partial correlation between 'intrusive sequence HR change' and detail recognition memory remained significant after controlling for gist recognition memory (r = -.42, $p < .01$).

Table 3.1 *Mean and Standard Deviations* of *All Variables by Phase: Data of the Overall Sample*

	N	*Mean (SD)*
Biological and background characteristics		
Age	64	24.98 (4.71)
Years in education	64	16.86 (2.09)
Body mass index (kg/m^2)	64	22.19 (3.04)
Psychological traits		
Trait anxiety (20-80)	64	39.02 (10.36)
Trait dissociation (%)	64	14.69 (10.52)
Subclinical PTSD symptom (0-51)[a]	51	4.56 (5.46)
Heart rate		
Pre-film	64	77.92 (9.56)
Peri-film	64	77.32 (9.46)
Post-film	64	78.35 (9.21)
Heart rate variability[b]		
Low Frequency Heart Rate Variability (ln)		
Pre-film	62	.67 (.20)
Peri-film	62	.65 (.17)
Post-film	62	.70 (.17)
High Frequency Heart Rate Variability (ln)		
Pre-film	62	.30 (.18)
Peri-film	62	.32 (.16)
Post-film	62	.27 (.16)
Low Frequency/High Frequency Ratio		
Pre-film	62	3.84 (3.56)
Peri-film	62	3.43 (2.57)
Post-film	62	4.29 (3.26)
Psychological states		
State anxiety (20-80)		
Pre-film	64	35.95 (9.54)
Peri-film	64	49.20 (11.51)
Post-film	64	41.67 (11.84)
State dissociation (0-76)		
Pre-film	64	4.95 (5.08)
Peri-film	64	7.39 (7.60)
Post-film	64	5.52 (6.74)
Fear (0-10)		
Pre-film	64	1.38 (1.75)
Peri-film	64	3.34 (2.85)
Post-film	64	1.33 (1.95)
Calmness (0-10)		
Pre-film	64	6.25 (2.61)
Peri-film	64	4.08 (2.73)
Post-film	64	5.59 (2.66)
Memory measures		
Intrusive memory		
Frequency of involuntary image	64	3.94 (2.79)
Frequency of involuntary thought	64	0.92 (1.26)
Vividness of involuntary image (0-10)[c]	56	4.76 (2.18)
Recognition memory		
Gist (0-15)	64	11.92 (1.77)
Detail (0-15)	64	6.64 (1.80)

a. Only the participants who had had at least one traumatic experience answered this questionnaire.
b. Heart rate variability results are expressed in normalised units.
c. Only the participants who had had at least one intrusive image answered this question.

Table 3.2 *Pearson's Correlation Coefficients between Memory Measures and Heart Rate Changes*

		Intrusive memory			Recognition memory	
		Frequency _image	Frequency _thought	Vividness _image	Gist	Detail
Intrusive memory	Frequency_image	-	-	-	-	-
	Frequency_thought	-.05 (64)	-	-	-	-
	Vividness_image	.00 (56)	-.12 (56)	-	-	-
Recognition memory	Gist	.15 (64)	-.04 (64)	.18 (56)	-	-
	Detail	.27 (64)*	-.14 (64)	.16 (56)	.31 (64)*	-
ΔHR	Overall peri-film ΔHR	.08 (64)	-.17 (64)	.07 (56)	.07 (64)	-.02 (64)
	Intrusive sequence ΔHR[a]	-.35 (54)*	-.04 (54)	-.01 (54)	.05 (54)	-.40 (54)**

* $p < .05$; ** $p < .01$.

Note. Frequency_image = frequency of intrusive images; Frequency_thought = frequency of intrusive thoughts; Vividness_image = vividness of intrusive images; ΔHR = HR change; Numbers in the brackets indicate sample sizes.

a. Only the participants who had had at least one intrusive image and were able to identify the intrusive sequence(s) in the trauma film were included in this analysis.

3.3.3 Classification of startle groups

To categorise participants by sHR response, both two- and three-cluster solutions were applied in Ward's hierarchical cluster analysis. The former resulted in non-equivalent sample sizes (50 participants with sHR reponse and 13 without). The latter resulted in a group with exaggerated and long lasting sHR (n = 14), a group with medium (n = 31), and a group with restricted sHR (n = 19). They were termed High Startle Group (HSG), Medium Startle Group (MSG), and Low Startle Group (LSG) respectively (see Figure 3.2) and were used in the following analyses. A 3 (group) x 11 (time: the 0- to 10-s interval after the white noise onset) mixed design ANOVA on HR change showed a significant time by group interaction ($F(20, 610) = 14.57$, $p < .001$) and main effects of Time ($F(10, 610) = 17.08$, $p < .001$) and Group ($F(2, 61) = 113.10$, $p < .001$). The results indicated a significant distinction between the three groups in HR over the first 10 seconds after the startle probe.

No significant difference was found between the three groups in gender (HSG: male = 4, female = 10; MSG: male = 20, female = 11; LSG: male = 9, female = 10, $X^2(2) = 5.18$, p = .08), age ($F(2, 61) = 1.51$, $p = .23$), years in education ($F(2, 60) = .48$, $p = .62$), BMI ($F(2, 61) = .09$, $p = .92$), trait anxiety ($F(2, 60) = .55$, $p = .58$), or subclinical PTSD symptoms ($F(2, 48) = 1.34$, $p = .27$). There were significant linear effects of group in baseline (i.e., pre-film) HR ($F(1, 61) = 4.03$, $p < .05$) and trait dissociation ($F(1, 61) = 7.29$, $p < .01$), with the LSG having higher baseline HR and reporting more trait dissociation than the HSG. However, the linear effects of group on baseline HRV indices were not significantly (largest $F(1, 60) = 1.69$, $p = .20$). The descriptive data are summarised in Table 3.3.

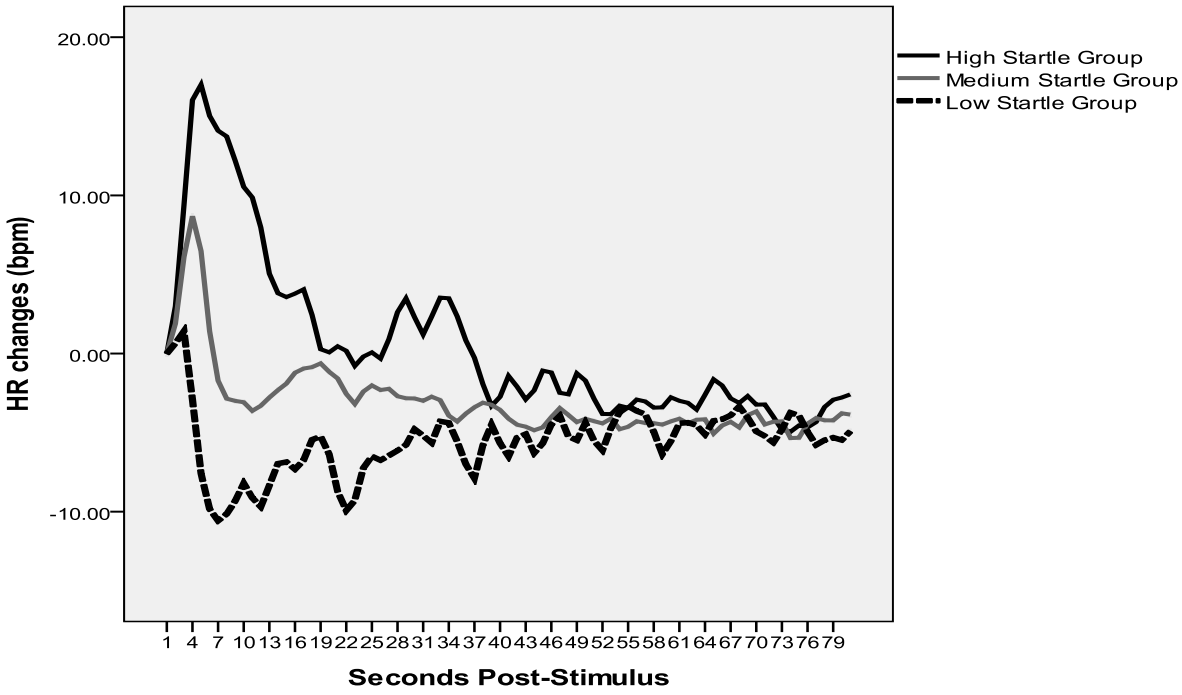


Figure 3.2 *Startle Heart Rate Response by Group*

Table 3.3 *Mean and Standard Deviations* of *All Variables by Phase and sHR Group*

	High Startle Group		**Medium Startle Group**		**Low Startle Group**	
	n	*Mean (SD)*	*n*	*Mean (SD)*	*n*	*Mean (SD)*
Biological and background characteristics						
Age	14	23.07 (3.50)	31	25.45 (5.20)	19	25.63 (4.49)
Years in education	14	16.38 (1.56)	31	17.06 (2.14)	19	16.84 (2.36)
Body mass index (kg/m^2)	14	21.88 (3.34)	31	22.28 (3.07)	19	22.27 (2.92)
Psychological traits						
Trait anxiety (20-80)	14	36.43 (7.61)	31	39.77 (12.14)	19	39.72 (8.93)
Trait dissociation (%)	14	9.30 (6.77)	31	14.81 (11.03)	19	18.46 (10.65)
Subclinical PTSD symptom (0-51)[a]	10	2.00 (1.94)	25	5.68 (6.04)	16	5.63 (6.52)
Heart rate						
Pre-film	14	74.83 (9.39)	31	77.05 (9.59)	19	81.46 (6.97)
Peri-film	14	74.48 (9.89)	31	76.46 (10.18)	19	80.12 (7.20)
Post-film	14	74.65 (9.68)	31	77.94 (9.42)	19	81.73 (7.64)
Heart rate variability[b]						
Low Frequency Heart Rate Variability (ln)						
Pre-film	12	1.25 (.07)	29	1.23 (.09)	19	1.21 (.10)
Peri-film	12	.79 (.12)	29	.81 (.10)	19	.78 (.14)
Post-film	12	1.23 (.07)	29	1.23 (.07)	19	1.19 (.09)
High Frequency Heart Rate Variability (ln)						
Pre-film	12	.26 (.16)	29	.30 (.18)	19	.33 (.20)
Peri-film	12	.33 (.16)	29	.30 (.15)	19	.33 (.18)
Post-film	12	.25 (.14)	29	.25 (.14)	19	.33 (.20)
Low Frequency/High Frequency Ratio						
Pre-film	12	1.94 (.84)	29	1.78 (.82)	19	1.67 (.94)
Peri-film	12	1.68 (.67)	29	1.81 (.66)	19	1.68 (.73)
Post-film	12	1.98 (.72)	29	2.06 (.74)	19	1.74 (.81)
Psychological states						
State anxiety (20-80)						
Pre-film	14	35.14 (9.79)	31	36.55 (9.70)	19	35.58 (9.55)
Peri-film	14	48.00 (12.30)	31	49.90 (10.98)	19	48.95 (12.30)
Post-film	14	38.21 (10.45)	31	43.23 (11.44)	19	41.68 (13.41)
State dissociation (0-76)						
Pre-film	14	3.36 (2.90)	31	6.19 (5.82)	19	4.11 (4.71)
Peri-film	14	4.86 (4.15)	31	8.29 (9.09)	19	7.79 (6.75)
Post-film	14	4.07 (4.46)	31	6.06 (8.33)	19	5.68 (5.20)
Fear (0-10)						
Pre-film	14	1.07 (1.38)	31	1.65 (1.99)	19	1.16 (1.57)
Peri-film	14	3.93 (2.92)	31	3.26 (2.61)	19	3.05 (3.24)
Post-film	14	0.86 (1.46)	31	1.52 (2.06)	19	1.37 (2.11)
Calmness (0-10)						
Pre-film	14	6.57 (2.10)	31	6.03 (2.69)	19	6.37 (2.89)
Peri-film	14	3.86 (2.96)	31	3.77 (2.47)	19	4.74 (2.98)
Post-film	14	6.07 (2.64)	31	5.26 (2.48)	19	5.80 (3.01)
Memory measures						
Intrusive memory						
Frequency_image	14	3.50 (4.33)	31	3.97 (2.63)	19	4.79 (3.65)
Frequency_thought	14	0.71 (1.07)	31	1.06 (1.26)	19	0.84 (1.43)
Vividness_image (0-10)[c]	10	5.38 (1.84)	27	4.64 (2.17)	17	4.60 (2.42)
Recognition memory						
Gist (0-15)	14	11.36 (1.74)	31	12.03 (1.92)	19	12.16 (1.50)
Detail (0-15)	14	6.79 (1.31)	31	6.52 (1.84)	19	6.74 (2.10)

Note. Frequency_image = frequency of intrusive images; Frequency_thought = frequency of intrusive thoughts; Vividness_image = vividness of intrusive images.

a. Only the participants who had had at least one traumatic experience answered this questionnaire.
b. Heart rate variability results are expressed in normalised units.
c. Only the participants who had had at least one intrusive image answered this question.

3.3.4 Effects of sHR on traumatic memory processing

Group by time (3 x 3) mixed design ANOVAs on HR and HRV indices showed significant quadratic effects of time on HR ($F(1, 60) = 4.53$, $p < .05$), LF-HRV ($F(1, 57) = 2402.10$, $p < .001$), HF-HRV ($F(1, 57) = 12.37$, $p < .01$), and LFHF-ratio ($F(1, 57) = 7.80$, $p < .01$). Post-film HR was significantly higher than peri-film ($p < .05$). Both pre- and post-film LF-HRV were significantly higher than peri-film ($p < .001$ for both). Post-film HF-HRV was significantly lower than peri-film ($p < .001$). Post-film LFHF-ratio was significantly higher than peri-film ($p < .001$). A significant linear effect of group was found on HR (but not any of the HRV indices) with the LSG presenting significantly higher HR than HSG ($p < .05$). None of the interaction effects between group and time was significant (largest $F(4, 114) = 1.57$, $p = .20$; See Table 3.3 for descriptive data).

For the psychological state measures, all the quadratic effects of time were significant ($F(1, 61) = 81.48$, $p < .001$ for state anxiety; $F(1, 61) = 19.27$, $p < .001$ for state dissociation; $F(1, 61) = 49.83$, $p < .001$ for fear; and $F(1, 61) = 46.12$, $p < .001$ for calmness). Peri-film dissociation was significantly higher than pre- ($p < .01$) and post-film ($p < .001$); peri-film calmness was significantly lower than pre- and post-film ($p < .001$ for both). In contrast, peri-film fear was greater than pre- and post-film ($p < .001$ for both). Peri-film anxiety was significantly higher than post- and pre-film ($p < .001$ for both). Moreover, post-film anxiety was significantly greater than pre-film ($p < .001$). There were no significant linear effects of group or group by time interactions on any of these state measures (largest $F(4, 122) = 2.01$, $p = .11$). Scores on the memory measures were not significantly different between the three groups (largest $F(2, 61) = 1.41$, $p = .33$). The descriptive data are summarised in Table 3.3.

To examine sHR as a potential moderator, peri-film HR and sHR group (i.e., dummy variables comparing either HSG or LSG with the other two groups) were entered in the first

step, followed by their interactions in the second step to predict the different peri-film psychological states[1]. As shown in Table 3.4, the interaction terms significantly increased the variance of peri-film anxiety, fear, and calmness explained by the model. In predicting peri-film dissociation, the second model as a whole did not significantly increase the variability accounted for. However, the interaction between peri-film HR and LSG (compared with the HSG and MSG) significantly contributed to the prediction of peri-film dissociation as well as all the other psychological states (i.e., anxiety, fear, and calmness). Additionally, both peri-film HR and its interactions with LSG and HSG (compared with the other two groups) showed significant effects on predicting peri-film anxiety.

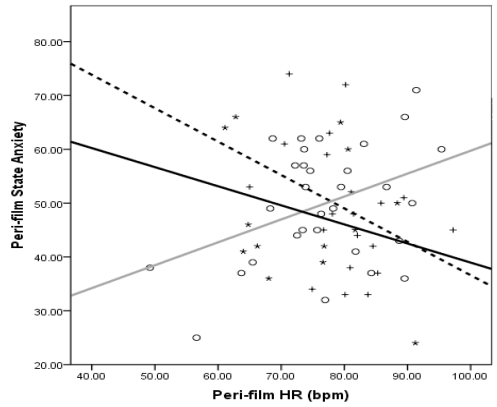
To clarify the above significant findings, the relationships between peri-film HR and psychological states were examined separately in different groups. As shown in Figure 3.3, while a trend level negative correlation between peri-film HR and dissociation was shown in the LSG (r = -.41, p = .08), the other two groups did not show the same pattern (r = .04, p = .90 for the HSG; r = .29, p = .11 for the MSG). In contrast, peri-film HR was significantly positively associated with anxiety among the MSG (r = .40, p < .05), but the associations in the HSG (r = -.29, p = .32) and LSG (r = -.36, p = .13) were in the opposite direction. In the LSG, peri-film HR was significantly negatively associated with fear (r = -.49, p < .05). However, a significant positive correlation between the two variables was found in the MSG (r =.39, p < .05), and the HSG (r =.13, p = .67) did not show a clear association. Finally, the patterns of correlation between peri-film HR and calmness in the HSG (r =.36, p = .21) and LSG (r =.38, p = .11) were found to be opposite to the MSG (r = -.25, p = .17).

[1]Multiple regressions with 'overall peri-film HR change' and Group entered in the first step, followed by their interactions in the second step, were also performed, but did not significantly predict the different psychological states peri-film.

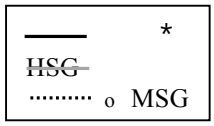
Table 3.4 *Multiple Regressions with Peri-film Psychological States as Dependent Variables and Peri-film Heart Rate, Group, and their Interaction as Independent Variables*

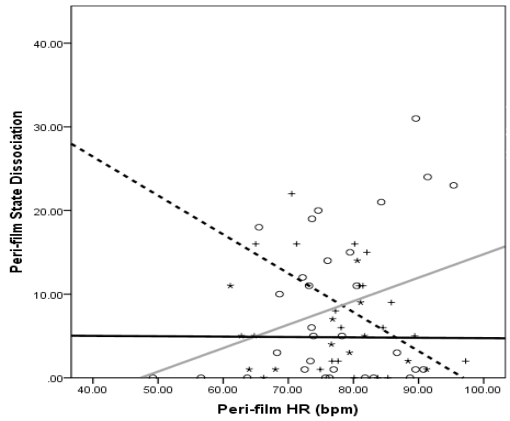
	B	*SE B*	β
Dependent variable: Peri-film Anxiety			
Step 1: R^2 = .01, *p* = .94, *df1* = 3, *df2* = 60			
Constant	49.96	2.12	
Peri-film Heart Rate	.61	1.64	.05
Low Startle Group	-1.15	3.46	-.05
High Startle Group	-1.76	3.81	-.06
Step 2: ΔR^2= .13, *p* < .05, *df1* = 2, *df2* = 58			
Constant	50.27	2.01	
Peri-film Heart Rate	4.36	2.03	.35*
Low Startle Group	.14	3.37	.01
High Startle Group	-3.43	3.74	-.12
Peri-film Heart Rate x Low Startle Group	-10.72	4.26	-.36*
Peri-film Heart Rate x High Startle Group	-7.99	3.79	-.32*
Dependent variable: Peri-film Dissociation			
Step 1: R^2 = .02, *p* = .76, *df1* = 3, *df2* = 60			
Constant	2.26	.29	
Peri-film Heart Rate	.17	.22	.10
Low Startle Group	.09	.47	.03
High Startle Group	-.24	.52	-.06
Step 2: ΔR^2= .09, *p* = .07, *df1* = 2, *df2* = 58			
Constant	2.29	.28	
Peri-film Heart Rate	.53	.28	.31
Low Startle Group	.30	.47	.09
High Startle Group	-.31	.52	-.08
Peri-film Heart Rate x Low Startle Group	-1.40	.59	-.35*
Peri-film Heart Rate x High Startle Group	-.49	.53	-.14
Dependent variable: Peri-film Fear			
Step 1: R^2 = .02, *p* = .72, *df1* = 3, *df2* = 60			
Constant	32.83	5.19	
Peri-film Heart Rate	2.93	4.03	.10
Low Startle Group	-2.98	8.50	-.05
High Startle Group	7.39	9.34	.11
Step 2: ΔR^2= .15, *p* < .01, *df1* = 2, *df2* = 58			
Constant	33.44	4.88	
Peri-film Heart Rate	10.14	4.93	.33*
Low Startle Group	2.32	8.17	.04
High Startle Group	7.05	9.07	.10
Peri-film Heart Rate x Low Startle Group	-32.86	10.33	-.45**
Peri-film Heart Rate x High Startle Group	-6.35	9.21	-.10
Dependent variable: Peri-film Calmness			
Step 1: R^2 = .03, *p* = .63, *df1* = 3, *df2* = 60			
Constant	37.89	4.96	
Peri-film Heart Rate	1.75	3.85	.06
Low Startle Group	9.07	8.12	.15
High Startle Group	1.24	8.92	.02
Step 2: ΔR^2= .10, *p* < .05, *df1* = 2, *df2* = 58			
Constant	37.22	4.78	
Peri-film Heart Rate	-6.19	4.82	-.21
Low Startle Group	6.42	8.00	.11
High Startle Group	4.85	8.88	.07
Peri-film Heart Rate x Low Startle Group	22.36	10.12	.32*
Peri-film Heart Rate x High Startle Group	17.14	9.02	.29

Note. Low Startle Group = the Low Startle Group compared against the High and Medium Startle Group; High Startle Group = the High Startle Group compared against the Low and Medium Startle Group.
*p < .05; **p < .01.

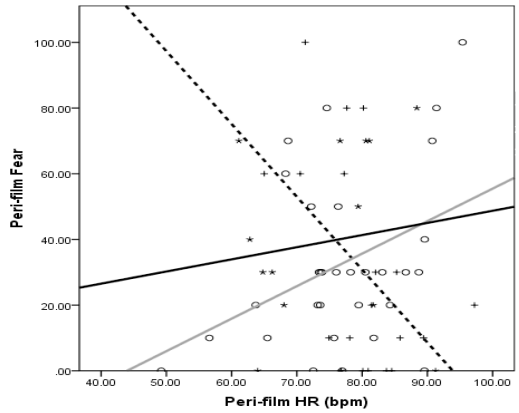


A. Relationships between Peri-film Anxiety and Heart Rate by Group

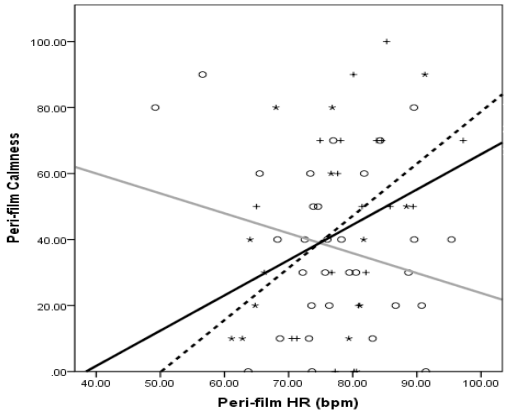




B. Relationships between Peri-film Dissociation and Heart Rate by Group



C. Relationships between Peri-film Fear and Heart Rate by Group



D. Relationships between Peri-film Calmness and Heart Rate by Group

Figure 3.3 *Relationships between Peri-film Psychological States and Heart Rate by Group.*

Similar analyses were used to examine the effect of sHR in the relationship between 'overall peri-film HR change' and the intrusive memory measures (i.e., frequency of intrusive images and thoughts, vividness of intrusive images). The second step significantly increased the variance in vividness of intrusive images explained by the model ($\Delta R^2 = .18$, $p < .01$) with the interaction between 'overall peri-film HR change' and the LSG (compared with the HSG and MSG) being the significant predictor ($\beta = .47$, $p < .01$). For the LSG, a greater HR decrease peri-film was associated with less vivid intrusive images ($r = .64$, $p < .01$), whereas for the HSG ($r = -.27$, $p = .45$) and MSG ($r = -.19$, $p = .35$) significant correlations were not found (see Figure 3.4). No moderating effects of sHR were found in the relationships between 'overall peri-film HR change' and both intrusion frequency measures (largest ΔR^2 of the second step = .05, $p = .44$).

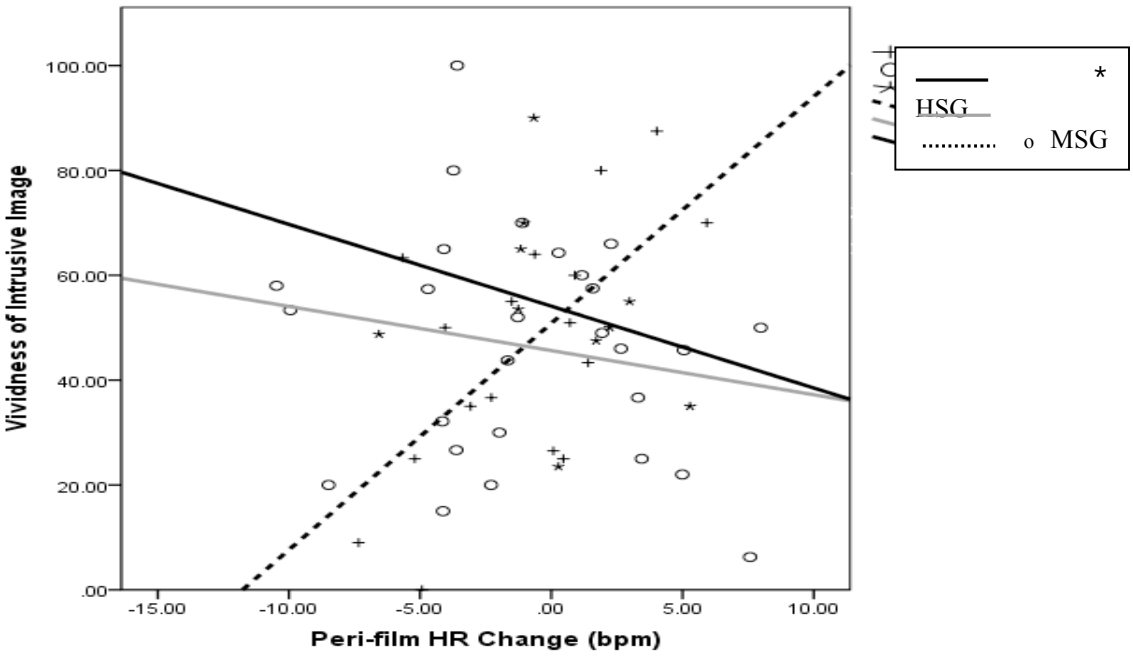


Figure 3.4 *Relationships between Vividness of Intrusive Image and Overall Peri-film Heart Rate Change by Group.*

3.3.5 Classification of CDR groups

To categorise participants based on the CDR, consistent with previous research (López et al., 2009), a three-cluster solution was first tested in Ward's hierarchical cluster analysis, but was rejected for producing an imbalanced distribution of sample size for each group (2 extreme Accelerators, 28 Accelerators and 34 Decelerators). A two-cluster solution was then tested and resulted in two groups with equivalent sample size– Accelerators (n = 30), who showed clear HR increase, and Decelerators (n = 34), who showed a HR decrease during this period (see Figure 3.5). This grouping result was used in the following analyses. A 2 (group: Accelerators vs. Decelerators) x 26 (time: the 20- to 45-s interval after the white noise onset) mixed design ANOVA on HR change found significant effects of group (F(1, 62) = 67.86, p < .001) and group by time interaction (F(25, 1550) = 4.20, p < .001). The time effect was marginally significant (F(25, 1550) = 1.90, p = .08). The results indicated that the grouping method was effective in distinguishing individuals with different CDR patterns.

Between Accelerators and Decelerators, there were no significant differences in gender distribution (Accelerator: male = 17, female = 13; Decelerator: male = 16, female = 18, X^2(1) = .59, p = .44), age (t(62) = .35, p = .73), years in education (t(61) = -.03, p = .97), BMI (t(62) = .27, p = .79), trait anxiety (t(61) = -.13, p = .89), trait dissociation (t(62) = -.87, p = .39), and subclinical PTSD symptoms (t(49) =.52, p = .60). Similarly, no significant group differences were found on baseline HR and the HRV indices (largest t(62) = .96, p = .34). The descriptive data are summarised in Table 3.5.

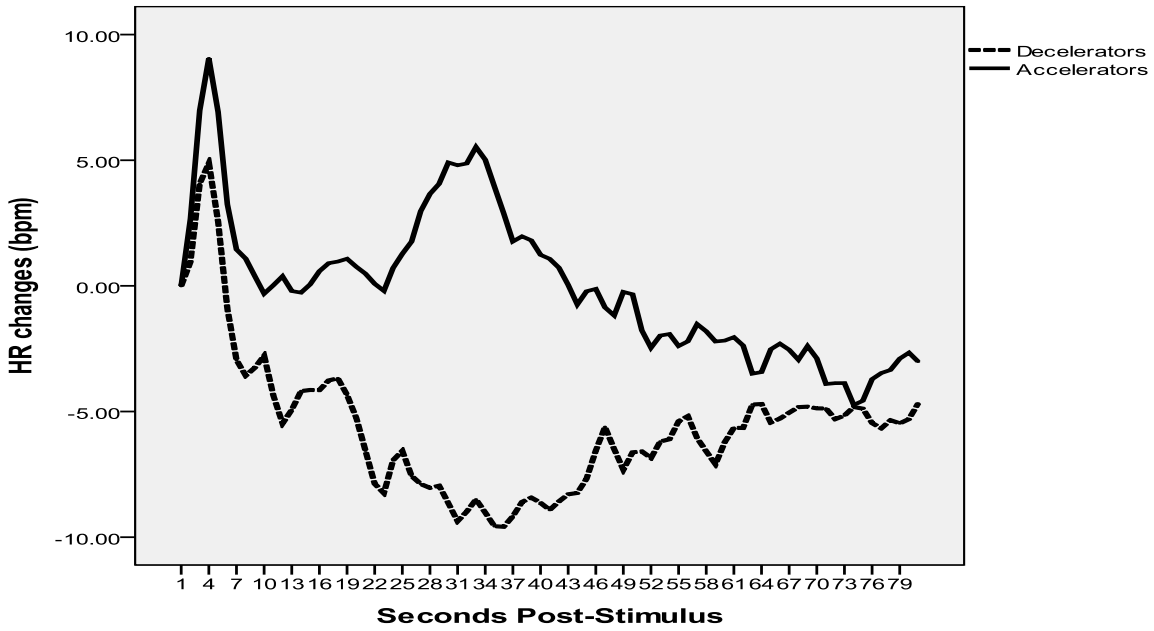


Figure 3.5 *Cardiac Defence Response by Group.*

Table 3.5 *Mean and Standard Deviations* of *All Variables by Phase and CDR Group*

	Accelerators		Decelerators	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Biological and background characteristics				
Age	30	24.77 (4.80)	34	25.18 (4.69)
Years in education	30	16.87 (2.13)	33	16.85 (2.09)
Body mass index (kg/m^2)	30	22.08 (3.37)	34	22.29 (2.77)
Psychological traits				
Trait anxiety (20-80)	29	39.21 (8.85)	34	38.85 (11.63)
Trait dissociation (%)	30	16.11 (12.02)	34	13.43 (8.98)
Subclinical PTSD symptom (0-51)[a]	25	4.40 (5.24)	26	5.46 (6.26)
Heart rate				
Prefilm	30	76.70 (8.68)	34	79.00 (10.29)
Perifilm	30	76.57 (8.40)	34	77.97 (10.39)
Postfilm	29	76.42 (7.92)	34	80.01 (10.00)
Heart rate variability[b]				
Low Frequency Heart Rate Variability (ln)				
Pre-film	29	.66 (.21)	34	.68 (.19)
Peri-film	29	.66 (.19)	32	.65 (.16)
Post-film	28	.71 (.20)	33	.70 (.16)
High Frequency Heart Rate Variability (ln)				
Pre-film	29	.31 (.19)	34	.28 (.16)
Peri-film	29	.31 (.17)	32	.32 (.15)
Post-film	28	.26 (.18)	33	.28 (.15)
Low Frequency/High Frequency Ratio				
Pre-film	29	4.00 (4.22)	34	3.82 (2.87)
Peri-film	29	3.83 (2.78)	32	3.30 (2.55)
Post-film	28	4.62 (3.46)	33	4.18 (3.17)
Psychological states				
State anxiety (20-80)				
Prefilm	30	35.90 (8.70)	34	36.00 (10.35)
Perifilm	30	49.37 (11.71)	34	49.06 (11.50)
Postfilm	30	41.13 (10.61)	34	42.15 (12.96)
State dissociation (0-76)				
Prefilm	30	5.60 (5.44)	34	4.38 (4.74)
Perifilm	30	8.13 (7.82)	34	6.74 (7.46)
Postfilm	30	6.27 (7.79)	34	4.85 (5.71)
Fear (0-10)				
Prefilm	30	12.00 (14.72)	34	15.29 (19.73)
Perifilm	30	34.00 (26.99)	34	32.94 (30.10)
Postfilm	30	10.00 (13.90)	34	16.18 (23.23)
Calmness (0-10)				
Prefilm	30	60.67 (27.28)	34	64.12 (25.24)
Perifilm	30	41.00 (24.40)	34	40.59 (29.94)
Postfilm	30	57.33 (22.73)	34	54.71 (29.87)
Memory measures				
Intrusive memory				
Frequency of intrusive images	30	3.87 (2.75)	34	4.00 (2.86)
Frequency of intrusive thoughts	30	1.07 (1.46)	34	.79 (1.07)
Vividness of intrusive images (0-10)[c]	24	49.64 (22.17)	30	46.00 (21.72)
Recognition memory				
Gist (0-15)	30	11.97 (1.85)	34	11.88 (1.72)
Detail (0-15)	30	6.77 (1.68)	34	6.53 (1.93)

a. Only the participants who had had at least one traumatic experience answered this questionnaire.
b. Heart rate variability results are expressed in normalised units.
c. Only the participants who had had at least one intrusive image answered this question.

3.3.6 Effects of CDR on traumatic memory processing

In examining the impact of the trauma film on Accelerators and Decelerators, mixed design ANOVAs did not find a significant effect of group (largest $F(1, 58) = .11$, $p = .75$), or time by group interaction (largest $F(2, 116) = 1.27$, $p = .29$) on HR and the HRV indices. Similarly, the effects of group and group by time interactions were not significant for any of the psychological state measures (largest $F(1, 62) = 1.33$, $p = .25$ for the group effect, largest $F(2, 124) = 1.01$, $p = .36$ for the group by time interaction). Finally, the group differences on the memory measures were not significant between Accelerators and Decelerators (largest $t(52) = -.61$, $p = .55$). See Table 3.5 for the descriptive data.

The moderating role of CDR in the relationships between peri-film HR and different peri-film psychological states were investigated with multiple regressions. Peri-film HR and CDR were entered in the first step, followed by the interaction of the two in the second step. The models did not significantly account for the variance of peri-film anxiety, dissociation, fear, and calmness in either the first (largest $\Delta R^2 = .04$, $p = .34$) or the second step (largest $\Delta R^2 = .06$, $p = .06$). The effect of CDR in the relationship between 'overall peri-film HR change' and the intrusive memory measures was also examined. In the regression models with 'overall peri-film HR change' and CDR in the first step, and the interaction of the two in the second step, a significant power of the predictors was not found (largest $\Delta R^2 = .03$, $p = .50$ for the first step; largest $\Delta R^2 = .01$, $p = .49$ for the second step).

3.4 Discussion

3.4.1 Trauma memory and peri-traumatic HR reduction

Adopting the same paradigm and similar methodology, our data provide the first replication of an important previous finding (Holmes et al., 2004) – HR during the encoding phase of the intrusive film sequences is lower than it is during the non-intrusive sequences. Moreover, the examination of HRV demonstrated that this HR fluctuation was contributed by both of the ANS and their balance.

We showed for the first time that the extent of HR reduction during intrusive sequences correlated with the frequency of intrusive images and also with recognition memory for details. Consistent with the DRT, which distinguishes between detail memory linked to viewpoint-dependent images (S-reps) and gist memory linked to contextualized episodic memories (C-reps), the positive associations between HR reduction during intrusive sequences, frequency of intrusive images, and detail memory were independent of gist memory. Moreover, our results also support the notion that the correlates of intrusive images and thoughts tend to be different (c.f., Hagenaars et al., 2010). Specifically, while the correlations between the frequency of involuntary images, HR, and detail recognition memory were significant, the correlations between involuntary thoughts and these variables were not significant. Additionally, two parameters of intrusions, namely frequency and vividness, were examined separately in the current study. Interestingly, the correlation between the two was not significant and they were related in different ways to changes in HR. The results suggest that the cognitive processes underlying the two parameters are different.

The current study failed to replicate Holmes et al.'s (2004) finding of a significant relationship between 'overall peri-film HR change' and intrusion frequency. This may be due

to the relatively smaller mean HR reduction (0.45bpm) in the current study compared to the previous one (4.24bpm). A factor contributing to the smaller HR reduction is likely to be the lower baseline HR (77.83bpm) in the current study relative to the 81.94bpm reported by Holmes et al. (2004). Presentation of the initial startle probe may have caused participants to be in a watchful state, resulting in this lowered baseline HR. However, as all participants had gone through the same procedures, its impact on the group effect and group by time interaction should be relatively small.

3.4.2 Individual differences in sHR, psychological states, and trauma memory

The current study assessed sHR as a psychophysiological trait indicating different stress defence styles. For the first time, a group of individuals (the LSG) has been identified who react to an unexpected threat with an inhibited cardiovascular response instead of the typical startle response characterised by a sharp HR increase. These individual differences echo the dual defensive behaviours observed in animal threat responses (Bradley & Lang, 2000; Kaada, 1987) and the reduced startle response of women suffering from chronic interpersonal violence (Medina et al., 2001). Accordingly, the LSG may represent individuals tending to adopt a passive stress coping strategy. Our finding that the LSG reported higher trait dissociation supports this hypothesis. Interestingly, we found higher overall HR among the LSG than HSG, despite their suppressive sympathetic response to threat (the startle probe). This finding is consistent with previous studies showing more severe hyperarousal symptoms among people with a dissociative subtype of PTSD (Ginzburg et al., 2006), as well as a positive association between acute dissociative symptoms and salivary cortisol level in PTSD patients (Koopman et al., 2003). The implication is that increased basal stress responses coexist with inhibition of reactive stress responses.

Although the groups did not differ in their psychological responses to the trauma film, individual differences were found in the relationship between peri-film HR and the psychological states. Agreeing with the existing literature, a high level of HR in the MSG correlated with increased fear and anxiety, implying a readiness to take active defensive actions in a threatening situation (Graham & Clifton, 1966), whereas a low level of HR correlated with a relatively calmer state. In contrast, for the LSG, it is lower HR that is suggestive of greater subjective distress. The lower these individuals' HR, the more anxious, fearful, and dissociative they are. Accordingly, for the MSG, lower HR may be a sign of

orientation and information gathering similar to the cognitive activities happening at the second stage of the defense cascade model (Bradley & Lang, 2000). However, for the LSG, low HR during the film viewing may indicate passive defense behaviour that can be found in the final stage of the model when predators have arrived and active coping is perceived as unavailable or useless (Bradley & Lang, 2000). In contrast, the HSG group did not show a consistent pattern of response.

The patterns of sHR did not relate to diverse phenomena of intrusion or moderate their relationships with HR. An unexpected finding was that greater overall peri-film HR reduction in the LSG was associated with lower vividness of intrusive images. Given the finding of negative correlation between HR and dissociation in the LSG, this result suggests a link between greater dissociation and lower vividness among these individuals. It may be related to the study by Ginzburg and colleagues (2006) which included adult female survivors of childhood sexual abuse and found a group of individuals who were less bothered by intrusive memories but suffered from greater dissociation symptoms.

3.4.3 Individual differences in CDR

Consistent with the previous literatures (Turpin & Siddle, 1978), diverse cardiovascular responses to a startle probe were found, with some individuals showing an unexpected HR acceleration (i.e., the Accelerators) and the others not (i.e., the Decelerators). Disagreeing with the previous study showing the Accelerators' vulnerability in fear responses (López et al., 2009), significant individual differences on any of the psychological and physiological reactions to the trauma film between the Accelerators and Decelerators were not found in the current study. Moreover, the CDR was not shown to play a significant moderating role in the relationships between the above measures.

The inconsistencies between the current study and the previous one (López et al., 2009) may be due to the differences in the characteristics of the adopted paradigms. In the fear conditioning paradigm, which was used in the previous study, pictures paired with electric shocks for several trials were used as the threat-provoking materials. In other words, these stimuli are associated with direct and concrete consequences. Contrastingly, the trauma film is experienced from an observer's point of view. Viewers only estimate the threatening level at the time when the film is presented and may not perceive it as an immediate threat. Accordingly, instead of provoking an extreme defence response, the trauma film may be more likely to induce a watchful and orienting response as described in the second stage of the defence cascade model (Bradley & Lang, 2000). In other words, compared to the fear conditioning paradigm, the trauma film triggers a relatively preliminary defence response.

To sum up, the current study examined the responses of Accelerators and Decelerators to the trauma film and the results supply more in depth additions to the defence coping profiles suggested in López and colleagues' study (2009). Specifically, Accelerators are individuals who tend to show stronger and long-lasting physiological fear responses to

stimuli that have been experienced as threatening. However, at the assessing or encoding stage of non-direct threat, Accelerators do not have biased judgments or a lowered threshold of active defence reactions. This lack of distinction in the initial processing stage may be one reason for the negative finding on intrusion frequency and recognition memory in the current study. However, considering the differences in the materials and assessment involved in both paradigms, alternative explanations need to be considered. For example, the group differences might be only restricted to physiological threat and/or immediate reactions, but not revealed in memory tested after a relatively longer period of time.

Chapter 4: HPA axis, traumatic memory, and individual differences

4.1 Introduction and hypotheses

4.1.1 How does trauma affect resting and reactive levels of cortisol?

The hypothalamic-pituitary-adrenal (HPA) axis and its major product, cortisol, have drawn research attention in the PTSD literature because of their essential roles in stress coping (Jones & Moller, 2011). The question, whether traumatic experiences in the past have an impact on the HPA axis and its responses to a later trauma, has been studied (e.g., Klaassens et al., 2012). However, due to the variance between the studies regarding the characteristics of past traumas (e.g., the elapsed time) and the study conditions (e.g., the type of trauma each study assessed cortisol's response to), this question has not been given consistent answers to.

This study aimed to clarify the impact of past traumatic experiences on resting and reactive cortisol levels with more sophisticated measures. In addition to investigating the effect of whether one has had a traumatic experience or not on the HPA axis, we also addressed the inconsistency associated with participants' past traumatic experiences. Specifically, trauma-related factors (i.e., the elapsed time and subclinical PTSD symptoms related to the most distressing past traumatic experiences), as well as pre-existing psychological traits (i.e., trait dissociation and trait anxiety) were accounted for in the investigation of the relationships between a past trauma and cortisol level at rest, and after a new traumatic event. In order to examine reactive cortisol with a standardised traumatic stimulus, the trauma film paradigm (Lazarus et al., 1965) was adopted, and the saliva samples were collected before, during and after the film. This design prevented possible inconsistency related to the diversity of the type of trauma that the HPA axis responded to in the previous literature.

As elevations of cortisol levels have been consistently found in reactions to stress (Takai et al., 2004), increased cortisol was expected in response to the trauma film. On the other hand, because previous studies have yielded contradictory results, no specific hypotheses were made concerning the relationships between resting cortisol level, whether or not one has traumatic experiences, and the pre-existing psychological traits. However, considering the attenuating effect of past trauma on cortisol reactions to a later trauma found previously (Resnick et al., 1995), participants with traumatic experiences happening more recently were predicted to show lower cortisol levels in response to the film. Moreover, because lowered cortisol secretion in the memory consolidation stage of trauma has been hypothesised to be associated with PTSD memory symptoms (Yehuda & Harvey, 1997), participants with greater pre-existing subclinical PTSD symptom severity were predicted to show lowered cortisol levels at the post-film measurement.

4.1.2 How do trauma-related cortisol responses relate to the development of intrusive memories?

Cortisol is not only a major psychophysiological index of stress, but also an essential element in the endocrinological process of memory. Its role in the development of trauma-related memory symptoms has therefore been an important issue in the PTSD literature. It has been hypothesised that an insufficient release of cortisol after a trauma is a cause of over consolidation of traumatic memories, and hence the related memory symptoms (Yehuda & Harvey, 1997). However, the attempts to examine this hypothesis have yielded inconsistent findings due to variability related to the studied populations, types of trauma, and sampling timings of cortisol (e.g., Delahanty et al., 2005; Delahanty, Raimonde, & Spoonster, 2000; McFarlane et al., 1997).

This study aimed to investigate Yehuda and Harvey's hypothesis (1997) with the trauma film paradigm, in order to minimise the variability caused by the above-mentioned factors in real life traumas. Moreover, as activation of the sympathetic nervous system (SNS) has been found to moderate the enhancing effect of cortisol on memory (Roozendaal et al., 2006), salivary alpha-amylase (sAA), an indicator of the SNS activation, was assessed as a possible moderator in the relationship between cortisol levels and the development of intrusive memory. Additionally, considering the cardiac defence response (CDR) and startle hear rate (sHR) as embodying different stress reactive patterns of the SNS to threats, these two physiological traits were examined as possible moderators. Finally, in addition to frequency, the vividness of intrusive memory was also assessed in consideration of its potential clinical significance.

Based on the previous hypothesis (Yehuda & Harvey, 1997), associations between low levels of cortisol and more frequent and vivid intrusions were hypothesised. Considering

the stressful nature of the trauma film, increased sAA levels were predicted in response to the film. Because adrenergic activation has been shown to mediate the effect of cortisol on memory (Roozendaal et al., 2006), it was predicted that the effect of cortisol on memory would only be shown when a sufficient level of sAA was released. Because the Accelerators (i.e., who show a secondary heart rate peak) have been suggested to be a group of individuals with higher anxiety related vulnerability (Delgado et al., 2009; Ruiz-Padial et al., 2002), they were predicted to show a greater frequency and vividness of intrusive memories. Similarly, as exaggerated sHR has been commonly reported by PTSD patients, the High Startle Group (HSG) was predicted to have greater intrusive memories. Moreover, as a higher level of sHR is regarded as a sign of a greater activation of the SNS in response to threat, the HSG was expected to show a stronger correlation between cortisol levels and the intrusive memory measures, given the enhancing effect of SNS activation found on the influence of cortisol on memory (Roozendaal et al., 2006).

4.2 Methods

4.2.1 Participants and procedures

This study was approved by the UCL Psychology and Language Sciences Ethics Committee (Appendix 1 and 2). The participants of this study were from the same cohort as Chapter Three. All of them provided written informed consent.

Priori power calculations based on a 3 (groups) by 3 (times) mixed design ANOVA, and a stepwise multiple regression (10 predicting variables overall) with an effect size of 0.18 and a power of 0.8 suggested a sample size of 66 and a sample size of 78, respectively. Among the 87 participants who passed the inclusion and exclusion criteria and completed the study, only the 69 participants who took part in the afternoon (i.e., between 1:30 p.m. and 6 p.m.) were included in order to control for the circadian fluctuations of cortisol and sAA (Nater, Rohleder, Schlotz, Ehlert and Kirschbaum, 2007). Another 10 were excluded due to procedural failures (e.g., failed to follow important instructions, experienced actual traumatic or stressful events between the two experimental sessions, and contamination of the saliva samples). This resulted in a final sample size of 59 (male = 32; age range = 18 to 37, M = 24.16, SD = 4.22). In the analyses involving sHR and CDR, the sample size decreased to 46 after excluding participants with high amount of artifacts in their ECG data. There were 10, 23, and 13 participants in High, Medium, and Low Startle Group, respectively. Twenty Accelerators and 26 Decelerators were identified.

The same procedures summarised in Chapter Three were introduced to the current sample. Additionally, the participants' saliva was collected three times. As illustrated in Figure 4.1, the first sample (i.e., the pre-film sample) was given at the beginning of the third minute of the baseline resting period. The peri-film sample was collected at the beginning of the final scene (the 11th minute of the film), whereas the post-film sample was collected

immediately after the 10-minute post-film resting period. The procedures regarding the use of the intrusion diary were the same as described in Chapter Three.

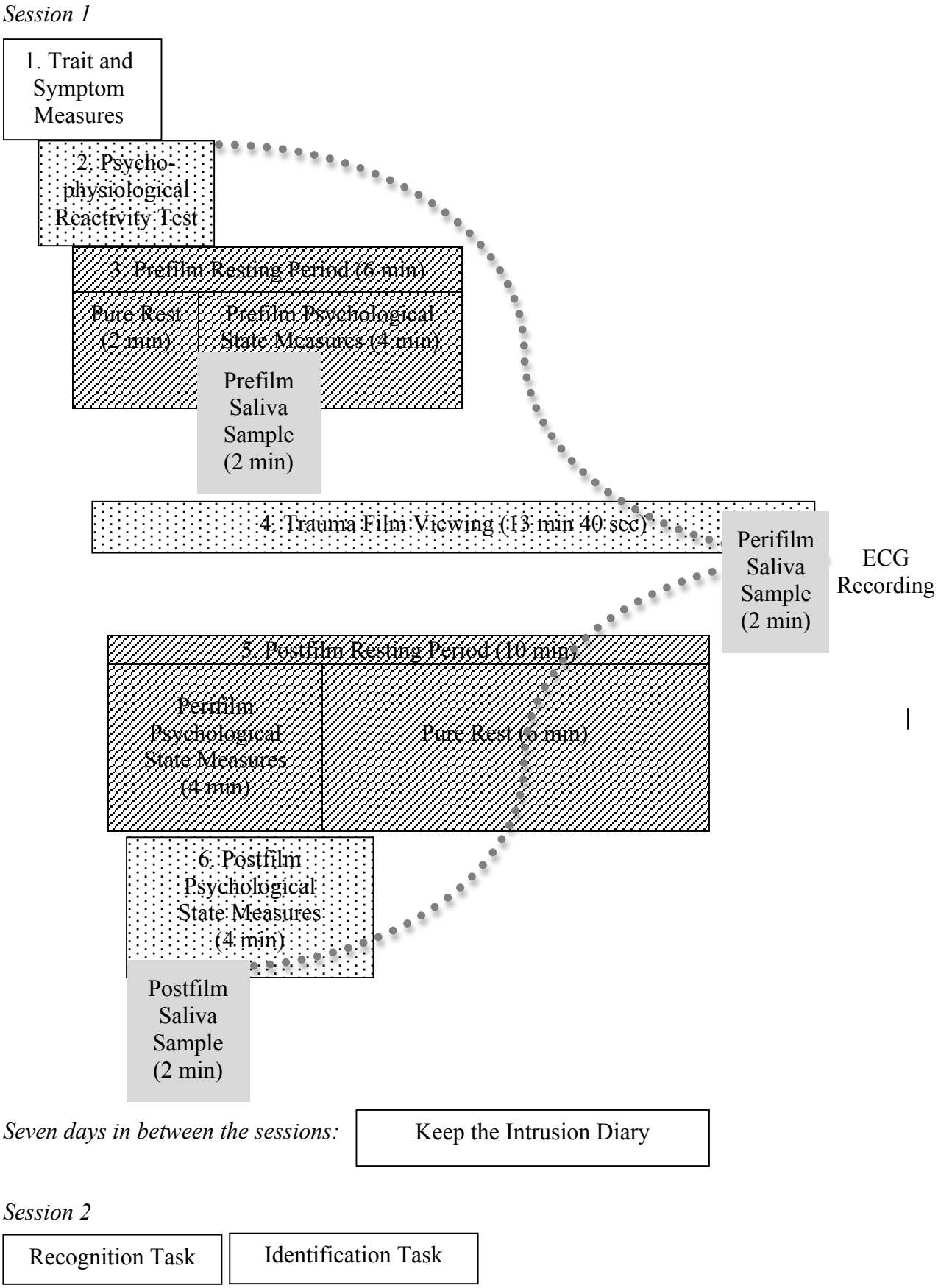


Figure 4.1 *Timeline of the Procedures*

4.2.2 Analytic strategy

All statistical analyses were performed with SPSS version 18 (SPSS Inc, Chicago, IL, USA). Adjustments for outliers and skewed distributions were performed in the same way as described in Chapter Three.

In order to examine the effect of past trauma on the cortisol and sAA levels in response to the trauma film, mixed design 3 (time: pre-, peri- vs. postfilm) x 2 (group: with vs. without past traumatic experience) ANOVAs on cortisol and sAA levels were performed. Next, among the individuals who had experienced at least one trauma, stepwise multiple regressions were used to examine the effects of the trauma-related factors (i.e., elapsed time of trauma and subclinical PTSD symptom) and pre-existing psychological traits (i.e., trait anxiety, and trait dissociation) on cortisol and sAA at different phases. As significant variance in cortisol levels has been associated with gender and age (reference), these two factors were entered in the first step in order to control for their effects in the model. Moreover, in the models predicting cortisol and sAA levels at the peri- and post-film phases, pre-film levels of the variable of interest were entered in the first step.

In order to examine the effects of sHR and CDR, a 3 (group: HSG vs. MSG vs. LSG) x 3 (time: pre- vs. peri- vs. post-film), and a 2 (group: Accelerators vs. Decelerators) x 3 (time: pre- vs. peri- vs. post-film) mixed design ANOVAs on the cortisol and sAA levels in response to the trauma film were performed. Additionally, stepwise multiple regressions were performed in order to investigate the relationships between cortisol levels at the peri- and post-film phases and intrusive memories. Moderating effects of sHR, CDR and sAA levels were examined with these regression models as well.

For all of the F tests, linear and quadratic effects were examined. Homogeneity of variance was assessed by Levene's statistic, while sphericity was examined with Mauchly's

test. When the assumption of sphericity was not met, the uncorrected degrees of freedom, Epsilon (Greenhouse-Geisser), corrected F, and corrected p values were reported.

4.3 Results

4.3.1 Associations between trauma, psychological traits, cortisol, and sAA levels

In the assessment of past traumatic experiences, 19 participants had not experienced any trauma. Among the remaining 39 who had experienced at least one traumatic event, 14 had experienced the occasion that they rated as the most stressful one before age 18. On average, 1.16 types of traumas (SD = 1.31) had been experienced. The average age when the most stressful event occurred was 20.13 years (range between 14 and 33; SD = 4.86). The mean elapsed time was 4.74 years (SD = 1.27).

Descriptive data of the cortisol and sAA levels at different phases are summarized in Table 4.1. Mixed design 3 (time) x 2 (group: with vs. without past traumatic experience) ANOVAs demonstrated a significant quadratic effect of time on cortisol ($F(1, 56) = 14.50$, $p < .001$), and a significant linear effect of time on sAA levels ($F(1, 56) = 16.50$, $p < .001$). Post-film cortisol levels were significantly higher than peri-film ($p < .01$), whereas post-film sAA levels were significantly lower than pre- ($p < .001$) and peri-film ($p < .05$). However, significant differences in cortisol ($F(1, 56) = .29$, $p = .60$) and sAA levels ($F(1, 56) = .83$, $p = .37$) were not found between the individuals who had experienced at least one traumatic incident, and those who had not. Similarly, the effects of the time by group interaction on cortisol ($F(2, 112) = .58$, $p = .49$), and sAA levels ($F(2, 112) = .15$, $p = .84$) were nonsignificant.

Table 4.1 *Mean and Standard Deviations of Cortisol and sAA Levels by Phase: Data of the Overall Sample*

	N	*Mean*	*SD*
		Salivary Cortisol	
Pre-film	59	8.69	4.06
Peri-film	59	8.12	4.22
Post-film	59	9.91	5.72
		Salivary Alpha-amylase	
Pre-film	59	51.37	42.87
Peri-film	59	45.71	41.25
Post-film	59	38.94	32.07

Among the individuals who had experienced at least one traumatic incident, influences of the trauma-related factors (i.e., elapsed time and subclinical PTSD symptoms related to the most stressful incident), and two pre-existing psychological traits (i.e., trait dissociation and trait anxiety) on baseline (i.e., pre-film) cortisol and sAA levels were examined with stepwise multiple regressions, with age and gender entered in the first step, and the relevant predictors in the second step. The model did not significantly predict baseline cortisol level (ΔR^2 of the first step = .01, p = .78; ΔR^2 of the second step = .10, p = .52). However, as shown in Table 4.2, the second step significantly increased the variance of baseline sAA levels explained by the model. Higher trait dissociation and lower trait anxiety significantly predicted a lower baseline sAA level.

Similar stepwise multiple regressions were applied to examine the effects of the above-mentioned variables on predicting cortisol and sAA levels at the peri- and post-film phases, with their baseline levels as another fixed variable entered in the first step. As shown in Table 4.3, the elapsed time of trauma was found to be significantly predictive of peri-film cortisol level, with a more recent trauma predicting a lower level of cortisol peri-film. A consistent finding was shown in the model predicting post-film cortisol levels. Additionally, more severe subclinical PTSD symptoms were found to be predictive of lower post-film cortisol levels. Overall, the second step significantly increased the variance of post-film cortisol level explained. On the other hand, while peri- (ΔR^2 = .56, p < .001) and post-film sAA levels (ΔR^2 = .82, p < .001) were significantly predicted by pre-film sAA levels; entering the other predictors in the second step did not significantly increase the variances of peri- (ΔR^2 = .03, p = .66) and post-film sAA levels (ΔR^2 = .02, p = .55) explained by the models.

Table 4.2 *Multiple Regressions Predicting Pre-film sAA Level*

	B	*SE B*	β
Dependent variable: Pre-film sAA Level			
ΔR^2= .01, *p* = .79			
Constant	6.41	1.49	
Age	.32	.46	.12
Sex	.23	1.02	.04
ΔR^2= .37, *p* < .01			
Constant	6.62	1.56	
Age	-.17	.41	-.06
Sex	-.72	.91	-.12
Elapsed time of trauma	.16	.48	.05
Subclinical PTSD symptoms	1.14	.97	.18
Trait dissociation	-1.80	.47	-.62**
Trait anxiety	1.13	.48	.41*

*$p < .05$; **$p < .01$

Table 4.3 *Multiple Regressions Predicting Peri-, and Post-film Cortisol Levels*

	B	*SE B*	β
Dependent variable: Peri-film Cortisol Level			
ΔR^2= .56, *p* < .001			
Constant	.70	.40	
Age	-.07	.07	-.12
Sex	-.02	.15	-.02
Pre-film cortisol level	1.52	.24	.74***
ΔR^2= .09, *p* = .16			
Constant	.78	.43	
Age	-.09	.07	-.16
Sex	-.09	.15	-.07
Pre-film cortisol level	1.64	.24	.80***
Elapsed time of trauma	.18	.08	.28*
PTSD symptoms	-.19	.16	-.15
Trait dissociation	-.05	.08	-.08
Trait anxiety	.06	.08	.10
Dependent variable: Post-film Cortisol Level			
ΔR^2= .18, *p* = .07			
Constant	1.19	.72	
Age	-.04	.12	-.05
Sex	.27	.26	.16
Pre-film cortisol level	1.12	.43	.41*
ΔR^2= .22, *p* < .05			
Constant	1.64	.73	
Age	-.09	.11	-.12
Sex	.10	.25	.06
Pre-film cortisol level	1.30	.41	.48**
Elapsed time of trauma	.30	.14	.36*
PTSD symptoms	-.58	.27	-.33*
Trait dissociation	-.19	.13	-.23
Trait anxiety	.19	.13	.25

*$p < .05$; **$p < .01$; ***$p < .001$

4.3.2 Relationships between sHR, cortisol, sAA, and intrusion

The data describing cortisol and sAA levels of each sHR group are summarised in Table 4.4. A set of 3 (group: HSG vs. MSG vs. LSG) x 3 (time: pre- vs. peri- vs. post-film) mixed design ANOVA on cortisol and sAA was performed. The effects of group ($F(2, 43) = 1.09$, $p = .35$), and group by time interaction ($F(4, 86) = .25$, $p = .84$) were nonsignificant on cortisol levels. Similarly, these effects were nonsignificant on sAA levels ($F(2, 43) = .47$, $p = .63$ for group effect; $F(4, 86) = 1.08$, $p = .37$ for group by time interaction).

Multiple regressions were used to examine the effects of sHR, peri- and post-film sAA and cortisol levels on predicting the vividness and frequency of intrusion. As shown in Tables 4.5 and 4.6, sex, age, whether one has experienced a trauma, as well as cortisol and sAA levels at the pre-film phase were controlled in the first step. Next, peri-, or post-film cortisol and sAA levels, as well as the sHR groups were entered in the second step, followed by their interactions in the third step. Results showed that a younger age and lower peri-film cortisol levels were significantly predictive of higher vividness of intrusion. Moreover, the second and third steps of the model which included post-film cortisol and sAA levels, sHR groups and the interactions of them significantly increased the amount of variance of the vividness of intrusion interpreted. In addition to a younger age and lower post-film cortisol levels, that were significantly predictive of higher vividness of intrusion, the interaction between post-film cortisol level and LSG (compared with the other two groups) significantly contributed to the predicting effect of the model.

Table 4.4 *Mean and Standard Deviations of Cortisol and sAA Levels by Phase and sHR Group*

	High Startle Group			**Medium Startle Group**			**Low Startle Group**		
	n	*Mean*	*SD*	*n*	*Mean*	*SD*	*n*	*Mean*	*SD*
Salivary Cortisol									
Pre-film	10	7.72	5.55	23	8.86	3.90	13	8.81	3.95
Peri-film	10	6.55	3.68	23	8.32	3.72	13	8.28	4.87
Post-film	10	8.04	6.53	23	10.40	4.38	13	9.92	6.22
Salivary Alpha-amylase									
Pre-film	10	51.75	44.95	23	51.73	43.22	13	36.14	33.53
Peri-film	10	50.27	48.77	23	38.32	30.75	13	39.22	49.93
Post-film	10	36.77	37.40	23	37.42	30.36	13	31.82	36.02

Table 4.5 *Multiple Regressions Predicting Vividness of Intrusion with Peri-film Cortisol, sAA Levels, and sHR*

	B	*SE B*	β
ΔR^2= .20, *p* = .15			
Constant	77.21	25.66	
Age	-5.78	3.20	-.29
Sex	-.53	7.33	-.01
Trauma	-11.06	8.28	-.22
Pre-film cortisol level	-17.10	9.61	-.28
Pre-film sAA level	2.40	10.76	.03
ΔR^2= .15, *p* = .16			
Constant	73.15	24.79	
Age	-6.68	3.22	-.34*
Sex	-2.28	7.65	-.05
Trauma	-12.52	8.36	-.25
Pre-film cortisol level	11.55	15.77	.19
Pre-film sAA level	11.98	16.30	.17
Peri-film Cortisol Level	-34.08	14.97	-.60*
Peri-film sAA Level	-7.26	14.53	-.12
Low Startle Group	9.06	8.08	.18
High Startle Group	3.05	9.78	.09
ΔR^2= .10, *p* = .20			
Constant	111.90	37.75	
Age	-7.73	3.16	-.39*
Sex	-1.38	7.45	-.03
Trauma	-8.30	8.97	-.16
Pre-film cortisol level	6.79	15.55	.11
Pre-film sAA level	14.54	15.90	.21
Peri-film Cortisol Level	-25.00	30.61	-.44
Peri-film sAA Level	-23.60	40.93	-.40
Low Startle Group	58.91	30.19	1.19
High Startle Group	8.19	32.47	.16
Peri-film Cortisol Level x Peri-film sAA Level	16.92	40.48	.31
Peri-film Cortisol Level x Low Startle Group	-59.69	34.92	-1.10
Peri-film Cortisol Level x High Startle Group	-3.27	32.71	-.06

Note. Trauma = a nominal variable representing whether or not one has experienced at least one trauma; Low Startle Group = the Low Startle Group compared against the High and Medium Startle Groups; High Startle Group = the High Startle Group compared against the Low and Medium Startle Groups.
*$p < .05$; **$p < .01$

Table 4.6 *Multiple Regressions Predicting Vividness of Intrusion with Post-film Cortisol, sAA Levels, and sHR*

	B	*SE B*	β
ΔR^2= .20, *p* = .15			
Constant	77.21	25.66	
Age	-5.78	3.20	-.29
Sex	-.53	7.33	-.01
Trauma	-11.06	8.28	-.22
Pre-film cortisol level	-17.10	9.61	-.28
Pre-film sAA level	2.40	10.76	.03
ΔR^2= .23, *p* < .05			
Constant	60.10	34.93	
Age	-6.51	3.00	-.33*
Sex	1.68	7.24	.04
Trauma	-8.84	7.89	-.17
Pre-film cortisol level	-4.65	9.80	-.08
Pre-film sAA level	-2.46	21.11	-.04
Post-film Cortisol Level	-11.74	3.52	-.51**
Post-film sAA Level	2.53	6.47	.12
Low Startle Group	7.11	7.56	.14
High Startle Group	-.86	8.69	-.02
ΔR^2= .14, *p* < .05			
Constant	108.66	47.48	
Age	-8.33	2.83	-.42**
Sex	3.11	6.86	.07
Trauma	-6.33	7.84	-.12
Pre-film cortisol level	4.13	9.86	.07
Pre-film sAA level	2.46	19.75	.04
Post-film Cortisol Level	-2.44	7.97	-.11
Post-film sAA Level	8.23	11.41	.40
Low Startle Group	65.28	22.73	1.32**
High Startle Group	2.70	23.05	.05
Post-film Cortisol Level x Post-film sAA Level	-17.46	27.25	-.35
Post-film Cortisol Level x Low Startle Group	-71.63	26.70	-1.32*
Post-film Cortisol Level x High Startle Group	-2.30	27.59	-.03

Note. Trauma = a nominal variable representing whether or not one has experienced at least one trauma; Low Startle Group = the Low Startle Group compared against the High and Medium Startle Groups; High Startle Group = the High Startle Group compared against the Low and Medium Startle Groups.
*p < .05; **p < .01; ***p < .001

To clarify the moderating effect of sHR, the relationships between post-film cortisol and vividness of intrusion, when pre-film cortisol and sAA levels were fixed, were examined separately in different groups. As shown in Figure 4.2, while a marginally significant negative correlation was found between post-film cortisol level and vividness of intrusion in the LSG (r = -.66, p = .05), the correlations of the two variables were nonsignificant in the MSG (r = -.33, p = .18) and HSG (r = -.41, p = .36).

Similar multiple regressions were used to predict the frequency of intrusion. The model including peri-film levels of cortisol and sAA did not show significant predictive effects (ΔR^2 in the first step = .09, p = .54; ΔR^2 in the second step = .04, p = .81; ΔR^2 in the third step = .04, p = .65). However, when post-film levels of cortisol and sAA were included in another model, a significant effect of the correlation between the two physiological indices on predicting the frequency of intrusion was found (Table 4.7). The results suggested an amplifying effect of the two indices on each other in predicting the frequency of intrusion.

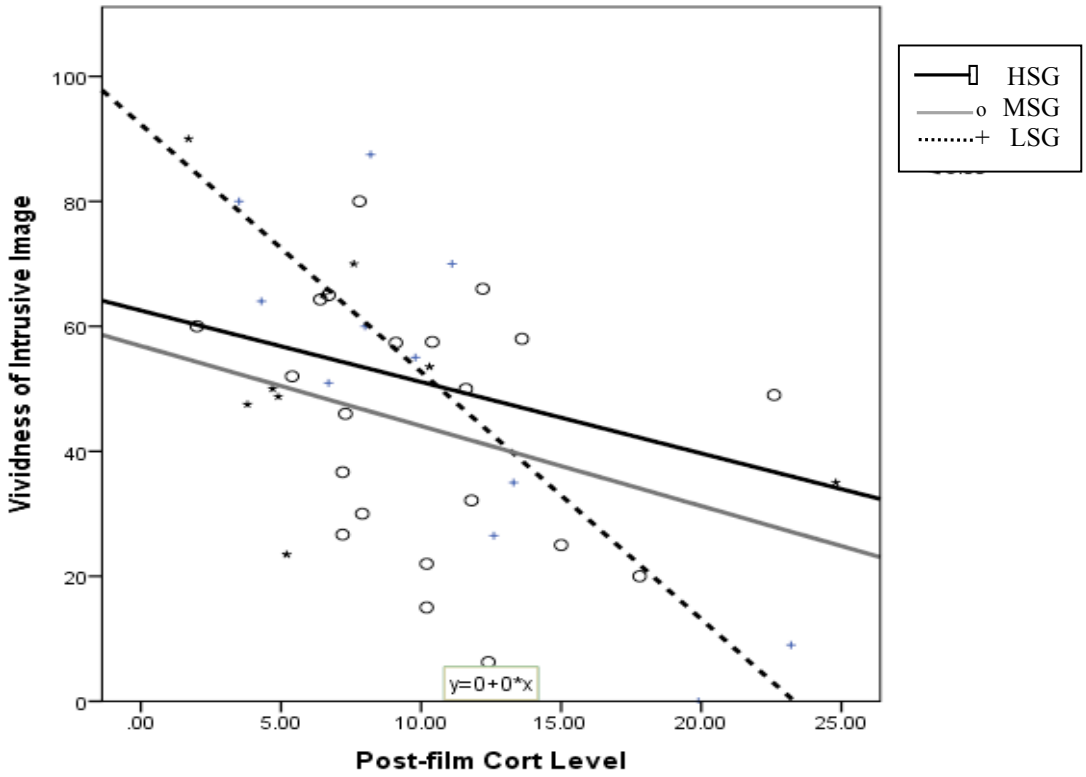


Figure 4.2 *Relationships between Vividness of Intrusive Image and Peri-film Cortisol Levels by sHR Group*

Table 4.7 *Multiple Regressions Predicting Frequency of Intrusion with Post-film Cortisol, sAA levels and sHR*

	B	*SE B*	β
ΔR^2= .09, *p* = .54			
Constant	1.06	2.94	
Age	.40	.41	.16
Sex	.34	.87	.06
Trauma	-.51	.96	-.08
Pre-film cortisol level	1.81	1.15	.24
Pre-film sAA level	.44	1.32	.05
ΔR^2= .04, *p* = .77			
Constant	.21	4.65	
Age	.36	.44	.14
Sex	.32	1.00	.06
Trauma	-.52	1.03	-.09
Pre-film cortisol level	2.31	1.34	.31
Pre-film sAA level	.39	2.94	.05
Post-film Cortisol Level	-.47	.51	-.16
Post-film sAA Level	.13	.93	.05
Low Startle Group	.88	1.05	.15
High Startle Group	.16	1.20	.02
ΔR^2= .13, *p* = .14			
Constant	-5.90	6.48	
Age	.35	.44	.14
Sex	-.13	1.00	-.03
Trauma	.20	1.10	.03
Pre-film cortisol level	1.88	1.45	.25
Pre-film sAA level	.06	2.96	.01
Post-film Cortisol Level	-2.40	1.18	-.83
Post-film sAA Level	-2.91	1.68	-1.10
Low Startle Group	2.08	3.22	.35
High Startle Group	.79	3.53	.12
Post-film Cortisol Level x Post-film sAA Level	8.92	4.06	1.44*
Post-film Cortisol Level x Low Startle Group	-1.25	3.88	-.18
Post-film Cortisol Level x High Startle Group	-.02	4.14	-.00

Note. Trauma = a nominal variable representing whether or not one has experienced at least one trauma; Low Startle Group = the Low Startle Group compared against the High and Medium Startle Groups; High Startle Group = the High Startle Group compared against the Low and Medium Startle Groups.
*$p < .05$; **$p < .01$; ***$p < .001$

4.3.3 Relationships between CDR, cortisol, sAA, and intrusion

The data describing cortisol and sAA levels of the Accelerators and Decelerators are summarised in Table 4.8. Significant group effects were not found on cortisol ($F(1, 44) = 0.02$, $p = .88$), or sAA ($F(1, 44) = .00$, $p = .96$). Similarly, the effects of group by time interaction on cortisol ($F(2, 88) = .40$, $p = .59$) and sAA ($F(2, 88) = .97$, $p = .37$) were not significant.

Stepwise multiple regressions were used to examine the relationships between CDR, peri- and post-film sAA and cortisol levels, and the vividness of intrusions. Sex, age, whether one has experienced a trauma, as well as cortisol and sAA levels at the pre-film phase were controlled in the first step. Next, peri- or post-film cortisol and sAA levels, as well as CDR were entered in the second step, followed by their interactions in the third step. The model including peri-film cortisol and sAA levels did not show a significant effect in predicting the vividness of intrusion (ΔR^2 in the first step = .20, $p = .15$; ΔR^2 in the second step = .08, $p = .37$; ΔR^2 in the third step = .03, $p = .48$). However, as summarised in Table 4.9, the model including cortisol and sAA levels at the post-film phase showed that a younger age and lower post-film cortisol level were significantly predictive of higher vividness of intrusion.

Similar multiple regressions were used to predict the frequency of intrusion. As summarised in Table 4.10, peri-film cortisol level and its interaction with CDR significantly predicted the frequency of intrusion. To clarify the effect of the interaction term, correlations between peri-film cortisol and frequency of intrusion were conducted separately among the Accelerators and Decelerators. As shown in Figure 4.3, a lower peri-film cortisol level significantly predicted more frequently occurring intrusions among the Accelerators ($r = .53$, $p < .05$). However, the correlation between these two variables was nonsignificant among the Decelerators ($r = -.04$, $p = .84$).

Table 4.8 *Mean and Standard Deviations of Cortisol and sAA Levels by Phase and CDR Group*

	Accelerators			**Decelerators**		
	n	*Mean*	*SD*	*n*	*Mean*	*SD*
Salivary Cortisol Levels						
Pre-film	20	8.66	3.86	26	8.55	4.58
Peri-film	20	7.55	2.81	26	8.21	4.82
Post-film	20	9.21	4.63	26	10.17	5.97
Salivary Alpha-amylase Levels						
Pre-film	20	51.64	47.89	26	44.02	35.10
Peri-film	20	41.79	43.05	26	40.69	38.97
Post-film	20	33.76	33.54	26	37.18	32.99

Table 4.9 *Multiple Regressions Predicting Vividness of Intrusion with Post-film Cortisol, sAA Levels and CDR*

	B	*SE B*	β
$\Delta R^2 = .20$, $p = .15$			
Constant	77.21	25.66	
Age	-5.78	3.20	-.29
Sex	-.53	7.33	-.01
Trauma	-11.06	8.28	-.22
Pre-film cortisol level	-17.10	9.61	-.28
Pre-film sAA level	2.40	10.76	.03
$\Delta R^2 = .21$, $p < .05$			
Constant	65.10	34.06	
Age	-6.89	3.04	-.35*
Sex	1.27	6.83	.03
Trauma	-6.42	7.59	-.13
Pre-film cortisol level	-2.92	9.75	-.05
Pre-film sAA level	-5.72	20.53	-.08
Post-film Cortisol Level	-11.70	3.56	-.51**
Post-film sAA Level	3.06	6.41	.15
Cardiac Defence Response	-1.70	6.45	-.04
$\Delta R^2 = .00$, $p = .98$			
Constant	60.46	48.27	
Age	-6.80	3.18	-.34*
Sex	1.24	7.07	.03
Trauma	-6.21	8.10	-.12
Pre-film cortisol level	-2.89	10.39	-.05
Pre-film sAA level	-5.55	21.28	-.08
Post-film Cortisol Level	-9.87	12.99	-.43
Post-film sAA Level	2.70	11.66	.13
Cardiac Defence Response	.85	15.63	.02
Post-film Cortisol Level x Post-film sAA Level	.64	28.90	.01
Post-film Cortisol Level x Cardiac Defence Response	-2.39	13.33	-.10

*$p < .05$; **$p < .01$

Table 4.10 *Multiple Regressions Predicting Frequency of Intrusion with Peri-film Cortisol, sAA Levels and CDR*

	B	***SE B***	**β**
ΔR^2= .09, *p* = .54			
Constant	1.06	2.94	
Age	.40	.41	.16
Sex	.34	.87	.06
Trauma	-.51	.96	-.08
Pre-film cortisol level	1.81	1.15	.24
Pre-film sAA level	.44	1.32	.05
ΔR^2= .03, *p* = .77			
Constant	-.30	3.84	
Age	.41	.45	.16
Sex	.39	.93	.07
Trauma	-.68	1.00	-.11
Pre-film cortisol level	2.74	1.93	.37
Pre-film sAA level	-.07	1.97	-.01
Peri-film Cortisol Level	-.49	.76	-.17
Peri-film sAA Level	.19	.66	.07
Cardiac Defence Response	.58	.90	.11
ΔR^2= .14, *p* = .05			
Constant	5.37	4.78	
Age	.29	.43	.11
Sex	.26	.88	.05
Trauma	-.20	.98	-.03
Pre-film cortisol level	2.09	1.84	.28
Pre-film sAA level	.25	1.87	.03
Peri-film Cortisol Level	-3.97	1.59	-1.40*
Peri-film sAA Level	-1.52	1.24	-.55
Cardiac Defence Response	-3.20	2.15	-.59
Peri-film Cortisol Level x Peri-film sAA Level	2.48	1.42	.81
Peri-film Cortisol Level x Cardiac Defence Response	3.23	1.58	1.19**

*$p < .05$; **$p < .01$

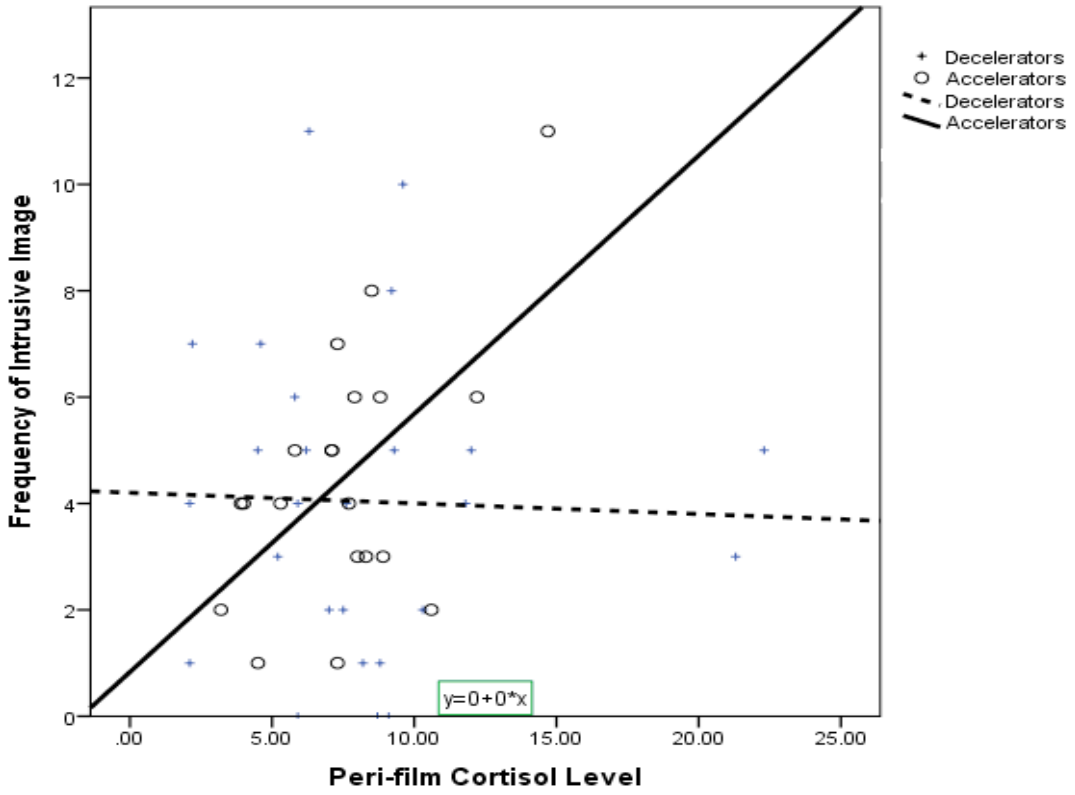


Figure 4.3 *Relationships between Frequency of Intrusive Image and Peri-film Cortisol Levels by CDR Group*

Similarly, when cortisol and sAA levels at the post-film phase were examined (Table 4.11), a significant and negative correlation between post-film cortisol level and the frequency of intrusion was found. However, the role of the interaction between cortisol level and CDR was replaced by a more dominant effect of the interaction between post-film cortisol and sAA levels at this stage. The results suggested a significant amplifying role of post-film sAA level in the correlation between post-film cortisol and the frequency of intrusion.

Table 4.11 *Multiple Regressions Predicting Frequency of Intrusion with Post-film Cortisol, sAA levels and CDR*

	B	*SE B*	β
$\Delta R^2 = .09$, $p = .54$			
Constant	1.06	2.94	
Age	.40	.41	.16
Sex	.34	.87	.06
Trauma	-.51	.96	-.08
Pre-film cortisol level	1.81	1.15	.24
Pre-film sAA level	.44	1.32	.05
$\Delta R^2 = .04$, $p = .65$			
Constant	.89	4.48	
Age	.40	.44	.16
Sex	.58	.94	.11
Trauma	-.46	1.00	-.08
Pre-film cortisol level	2.22	1.33	.30
Pre-film sAA level	-.84	2.87	-.10
Post-film Cortisol Level	-.41	.51	-.14
Post-film sAA Level	.46	.92	.17
Cardiac Defence Response	.69	.91	.13
$\Delta R^2 = .16$, $p < .05$			
Constant	-2.40	5.91	
Age	.37	.42	.15
Sex	.39	.88	.07
Trauma	.01	.97	.00
Pre-film cortisol level	1.52	1.28	.20
Pre-film sAA level	-1.90	2.70	-.22
Post-film Cortisol Level	-4.01	1.64	-1.39*
Post-film sAA Level	-2.32	1.49	-.88
Cardiac Defence Response	-1.01	2.03	-.19
Post-film Cortisol Level x Post-film sAA Level	8.99	3.71	1.45*
Post-film Cortisol Level x Cardiac Defence Response	1.91	1.72	.65

*$p < .05$; **$p < .01$

4.4 Discussion

4.4.1 The impacts of trauma on resting and reactive cortisol levels

The current study, adopting the trauma film paradigm, was the first one to examine the HPA axis' response with the trauma film paradigm. As predicted, cortisol secretion increased as a result of the stressful nature of the trauma film. This finding indicates a reaction of the current sample consistent with the findings in previous studies regarding the stress response of the HPA axis (Takai et al., 2004). However, decreased, rather than increased sAA levels were found in response to the film viewing. This may be due to the nature of the study design. With the trauma film paradigm, the participants might not perceive themselves in a threatening situation where a highly aroused SNS or 'fight/flight response' is normally triggered. Instead, as the participants were asked to try as much as they could to sit still throughout the film, they were more likely to respond to extreme stress with passive coping strategies. The finding of a negative correlation between trait dissociation and baseline sAA level in the study supports this argument. Individuals who are more prone to adopt a shut-down coping mechanism tend to have a less activated SNS.

The current study investigated the associations of cortisol with different trauma related characteristics. Consistent with the study which included healthy veterans (Klaassens et al., 2010), the resting cortisol of trauma victims and non-traumatised individuals did not vary significantly. Similarly, neither the elapsed time of trauma, subclinical PTSD symptom severity, trait anxiety, nor trait dissociation significantly related to resting cortisol levels.

On the other hand, the current study was the first one to explore the impact of trauma on the reaction of the HPA axis to a later stressful situation. As predicted, a shorter elapsed time from the most stressful past trauma was associated with lower cortisol levels peri- and post-film. Individuals with more severe subclinical PTSD symptoms released less cortisol at

the early memory consolidation stage of a traumatic event. Overall, the results supported the previous study (Resnick et al., 1995) by indicating that the more individuals suffer from the impact of a prior traumatic event, the smaller amount of cortisol was released in response to new stressor. Considering the protective function of spontaneous cortisol increase in a stressful situation, the current study suggests a possible vulnerability in the HPA axis among people who had recently experienced a trauma and who suffer more severe posttraumatic distress.

4.4.2 Cortisol and the development of intrusive memories: Examination of moderators

The current study assessed the vividness and frequency of intrusion in relation to cortisol levels at the peri- and post-film phases. Results showed that higher vividness of intrusive images was significantly predicted by lower cortisol secretion in response to the trauma film. Similarly, when the effect of CDR was included in the multiple regression models, lower peri- and post-film cortisol levels were predictive of more frequent intrusive memories. The nonsignificant associations between cortisol and the frequency of intrusion in the regression models including sHR suggested a less robust relationship between cortisol and the frequency of intrusion, compared to its vividness. However, overall, the findings support Yehuda and Harvey's (1997) hypothesis arguing the insufficiency of cortisol release in the immediate aftermath of trauma as a cause of over-consolidation of traumatic memories.

Moderating roles of other psychophysiological measures were found in the relationships between cortisol and intrusive memories. Although a significant positive correlation between cortisol secretion and the frequency of intrusion was not found at the peri-film phase, when the amount of sAA secretion became higher at the post-film phase, an enhancing effect of cortisol, and an amplifying effect of sAA, on the frequency of intrusion was shown. This finding supported the previous studies which suggested a mediating effect of noradrenergic activation on the relationship between corticoids and memory (Bryant, McGrath, & Felmingham, 2013; Cahill et al., 1994; McGaugh & Roozendaal, 2009; Roozendaal et al., 2006). Cortisol's enhancing effect on memory only occurs when the SNS is activated at the same time. Additionally, it is interesting to note that the moderating role of sAA only increased and became significant when the overall cortisol level significantly rose in the post-film phase. This suggests two hypotheses needing further investigation. First, the moderating effect of the SNS only becomes visible when cortisol levels are higher. Second,

intrusion frequency is only affected by cortisol under the moderating influence of the SNS during the memory consolidation, but not encoding phase.

Significant effects of sHR have been found on predicting the vividness of intrusion. The vividness of intrusive images was higher among the participants with restricted level of sHR (i.e., LSG). Moreover, sHR was found to moderate the relationship between post-film cortisol level and the vividness of intrusion, with the LSG showing greater negative association between the two variables. In other words, while a worsening effect (i.e., more vivid) of an insufficient post-film cortisol release on the involuntary memory symptom has been established, the LSG appeared to be affected even more negatively than the MSG and HSG. Taking account the finding of higher trait dissociation among individuals in the LSG (See Chapter Three), the current finding may be regarded as further evidence of the LSG's greater vulnerability to developing intrusive memories.

Additionally, a significant moderating role of the CDR has been shown in the relationship between peri-film cortisol level and frequency of intrusion. Contradicting the overall pattern in the whole sample showing a negative association between cortisol and intrusion, among the Accelerators higher peri-film cortisol was shown to predict a higher frequency of intrusive image, whereas this correlation was nonsignificant among the Decelerators. A high potential of the Accelerators to initiate a fight/flight response (Richards & Eves, 1991), and to have more persistent negative impact from aversive stimuli (López et al., 2009) have been suggested in the literature. Although the overall intrusive frequency was not significantly higher among the Accelerators than Decelerators, the current finding still suggested a higher sensitivity to stress among the Accelerators. Whereas cortisol level, as an index of the level of stress, did not affect the amount of intrusion occurring in the Decelerators, those Accelerators who were more distressed during the film viewing had

significantly more long-lasting impacts resulting from the film. In sum the results highlighted the importance of addressing individual differences in the prediction of the effects of cortisol on intrusive memory.

4.4.3 The vividness of intrusion as an alternative measure

The current study assessed the vividness of intrusive images in addition to the frequency of them. Diverse findings have been shown between the two measures. Across all groups, higher vividness of intrusion was predicted by lowered cortisol secretion at both the peri- and post-film phases. However, the correlations between cortisol levels and the frequency of intrusion vary with individual differences in the CDR and sAA levels. At the peri-film phase, this correlation differs between the Accelerators and Decelerators; At the post-film phase, an enhancing, rather than decreasing effect of cortisol secretion on the frequency of intrusion was revealed when the sAA levels were high.

The discrepancy between findings in the vividness and frequency of intrusions suggests that the HPA axis affects the two memory phenomena through different mechanisms. Specifically, two characteristics of cortisol have been studied: 1) as an objective indicator of stress, and 2) as a regulator of the consolidation-related neuromodulators. The results suggest that the frequency of intrusion is associated with the first characteristic and the vividness of intrusion is associated with the second. In other words, the frequency of intrusion is predicted by stress intensity (as indicated by cortisol and sAA levels), whereas the vividness of intrusion is more directly affected by cortisol's regulating effect on the neuromodulators that relate to memory consolidation.

The incongruent findings between the two measures of intrusive memory highlighted a few issues needing more sophisticated considerations in future research and clinical applications. First, separate examinations of the quality (i.e., vividness) and quantity (i.e., frequency) are needed in research and clinical practice in order to specify the assessment of intrusive memories. Second, diversity of the assessment of PTSD symptom severity has existed in research. For instance, the PDS (Foa, 1995) used in the current study assesses the

frequency of symptoms, whereas the other widely used tool, the Impact of Event Scale (IES-R; Weiss & Marmar, 1997), asks about the distress level created by each symptom. To better reflect the clinical phenomena, it is crucial to find out the most clinically significant measurement for research applications. Third, the beneficial effects of applying cortisol in the treatment and prevention of PTSD have been shown in recent studies (e.g. Bowirrat et al., 2010; Schelling et al., 2001; Schelling et al., 2004; Suris, North, Adinoff, Powell, & Greene, 2010). In the current study, different relationships between cortisol and the two measures of intrusive images, as well as the moderating effect of personal characteristics (i.e., CDR) were present. Specifically, although an increase of cortisol is associated with decreased vividness, it is correlated with more frequent occurrences of intrusion in individuals with higher proneness to extreme physiological defensive reactions (i.e., Accelerators). Accordingly, more sophisticated studies considering different domains of the PTSD memory symptoms and personal characteristics are needed to investigate the possibility of administering cortisol as a PTSD therapy.

Chapter 5: Cardiovascular responses, and voluntary retrieval of traumatic memory

Following the examination of the memory encoding phase of trauma, Chapter Five and Chapter Six focus on the voluntary retrieval of traumatic memory. We included individuals with PTSD in this part of investigation. Similar to Chapter Three and Chapter Four, several pre-existing psychological and physiological features were associated with the psychological and physiological responses during the voluntary retrieval of trauma. Detailed descriptions of the goals and hypotheses are provided in the next section.

5.1 Introduction and hypotheses

5.1.1 What are the psychological implications of HR fluctuations during voluntary retrieval of traumatic memory?

Voluntary memory retrieval is a crucial part of exposure based psychotherapy for PTSD. Psychophysiological reactions related to this process have been studied in terms of their associations with therapeutic outcomes (e.g., Halligan et al., 2006; Lanius et al., 2010). Moreover, correlations between physiological responses and psychological states during voluntary retrieval have been examined, in order to understand the mechanisms through which these physiological phenomena relate to diverse posttraumatic and psychological treatment outcomes. For example, in a previous study (Halligan et al., 2006), a lower level of HR increase in response to voluntary recall of a trauma has been shown to predict a worse recovery outcome 6 months later. However, an association between such cardiovascular phenomena and psychological distress experienced during voluntary recall was not evident (Halligan et al., 2006).

Two major sources of variation might have contributed to this nonsignificant result. First, the association between HR and psychological distress may vary between PTSD patients and healthy individuals, so that including both participants with and without a PTSD diagnosis in the study might have introduced unnecessary variance. Second, HR and subjective feelings of distress might be related both to the distressing nature of traumatic memories, as well as to the action of giving a speech. In other words, using a silent resting period as a baseline, in contrast to the trauma recalling task, may have introduced a confounding variable – the action of giving a speech - and therefore reduced the statistical power in Halligan and colleagues' study (2006).

To further investigate this question following Halligan and colleagues' study (2006), we only included adult individuals with a current PTSD diagnosis. A memory retrieval procedure mimicking the methods used in exposure-based therapy was adopted and compared with another memory retrieval period focussed on neutral memory. Differences in HR, as well as in the psychological states related to traumatic memory processing (i.e., state dissociation, and fearful, threatened and calm feelings) between the two memory retrieval conditions were assessed and related to each other. In order to examine the proportional contributions of sympathetic and vagal nervous systems, indices of HRV were also calculated and associated with the psychological states. Additionally, as individual differences in sHR responses and CDR patterns have been found to moderate the physiological and psychological reactions at the encoding phase of traumatic memory in Chapter Three and Chapter Four, their roles in the retrieval phase were examined with the current research designs.

Because a smaller amount of HR increase has been associated with a lower level of arousal, lower HR increases were predicted to relate to lower emotional arousal, and hence smaller increases in fearful and threatened feelings, smaller decreases in calmness, and greater increases in state dissociation. As LF-HRV and LF/HF ratio have been associated with sympathetic activation and fight or flight responses, they were predicted to positively relate to the levels of fearful and threatened feelings, and negatively relate to calmness and state dissociation ratings. Due to inconsistent findings regarding the psychological implications of increased HF-HRV (e.g., Bradley & Lang, 2007; Hansen, Johnsen, & Thayer, 2003), specific hypotheses about the relationships between HF-HRV and psychological state fluctuations were not made. Finally, as healthy individuals with suppressed sHR have been found to be more dissociative (See Chapter Three), PTSD patients with the same cardiovascular characteristics were predicted to react to the retrieval of traumatic memory

with a greater level of state dissociation. Moreover, as sHR was found to moderate the relationships between psychological states and cardiovascular responses at the memory encoding phase (See Chapter Three), similar effects were also expected in the memory retrieval phase. On the other hand, Accelerators have been suggested to have a more sensitive link between physiological and psychological reactions, and a greater tendency to engage in extreme psychophysiological responses to stress (Richards & Eves, 1991). As a result, the Accelerators in the current patient sample were expected to have stronger correlations between HR increases and elevated fearful and threatened feelings, as well as stronger correlations between HR decreases and state dissociation, in comparison to the Decelerators.

5.1.2 How do HR fluctuations relate to flashbacks and dissociations during voluntary recall of traumatic memory?

According to the dual representation theory (DRT; Brewin et al., 2010), the ability to retrieve and hold sensation-based memory (S-memory) in focal attention has been suggested to be crucial in the treatment for PTSD. Following this hypothesis, the ability to trigger flashback memories through a voluntary retrieving process in therapy, without switching into a dissociative state, were thought to be beneficial for recovery. It is therefore of research interest to explore the psychophysiological indices that can identify the states of flashbacks and dissociations.

The attempts to associate dissociation with the cardiovascular responses to voluntary recall of trauma have shown inconsistent results (e.g., Griffin, Resick, & Mechanic, 1997; Kaufman et al., 2002), which may be related to the varieties in the studied samples and the time elapsed since the targeted traumas (See detailed descriptions in 1.3.2 and 1.5.1). On the other hand, whereas overall PTSD symptoms have been related to HR variations in response to voluntary recall of traumatic memory, flashbacks as an expression of PTSD symptoms, as well as a state triggered by a voluntary recollection of trauma, have not been directly examined.

In order to explore this topic in the current study, PTSD patients were asked to identify the sequences when they were experiencing flashbacks or dissociation through reviewing the video taken during the voluntary recall. As mixed states of flashback and dissociation have been suggested (Lanius, Bluhm, Lanius, & Pain, 2006), these were also identified in the current study. Mean HR during these sequences were compared with that of the rest of recall period (i.e., pure recall sequences). Increases in HR were expected during the flashback sequences, as higher arousal was hypothesised to be involved. Following the

preliminary findings reported in Lanius and colleagues' study (2006), a higher mean HR during the sequences with a mixture of flashback and dissociation was predicted. A significant difference of HR between the dissociative and 'pure recall' sequences was not expected.

5.1.3 Do factors associated with the development of PTSD relate to emotional and psychophysiological fluctuations during voluntary recall of trauma?

Emotional and psychophysiological arousal during voluntary retrieval of traumatic memory have been associated with the treatment outcomes of PTSD (Halligan et al., 2006). Their relationships with factors which contribute to various levels of risk to develop PTSD were therefore of research interest. For example, individuals with repetitive and long-lasting traumas, as well as with greater peri-traumatic dissociation, have been suggested to have worse posttraumatic symptomatology and poorer treatment responses (Bremner et al., 1992; van der Kolk et al., 1996). These factors were examined in the current study, together with trait dissociation, PTSD and depression symptom severities, in terms of their associations with the cardiovascular and psychological phenomena occurring in the voluntary memory retrieving procedures. Moreover, as voluntary retrieval is a key element of PTSD treatment, we were also interested in the effect of psychotherapy (i.e., the amount of time when one has been receiving psychotherapy) on the cardiovascular and psychological state variations related to the recall task.

It was predicted that PTSD patients with greater trait dissociation and previous peri-traumatic dissociation should report longer periods of dissociation, smaller levels of psychological and cardiovascular arousal, and greater increases in state dissociation during the trauma recall (in comparison to the neutral recall). Following this, as repetitive and long-lasting traumas have been associated with a greater tendency to dissociation (van der Kolk, et al., 1996), individuals who had been exposed to more types of adversities were expected to report greater levels of dissociation and lower increases of HR in response to the trauma recall. On the other hand, because psychotherapy, PTSD, and depression symptoms were

explored in the current study for the first time, no specific hypotheses were made regarding their effects on HR during the voluntary retrieval of traumatic memory.

5.2 Methods

5.2.1 Ethics, participants and procedures

This study was reviewed by the NRES committee (London Bridge). In addition to minor modifications on the materials (e.g., simplifying the language in the information sheet) and procedures (e.g., sending some of the questionnaires to participants prior to the study meeting), the committee suggested to limit the source of participant recruitment to clinic referrals, instead of both clinic referrals and public advertisement (Appendix 11). Ethical approval was granted after the modifications were made (Appendix 12).

Based on the suggestions of the NRES committee, recruitment of the current study was initially through referrals from the Posttraumatic Stress Clinic and the Improving Access to Psychological Therapies Services of the Camden and Islington Foundation Trust, London, UK. However, in order to enhance the efficiency of recruitment, an amendment application was submitted and approved by the same NRES committee half way through the study (Appendix 13). Following this amendment, advertisement to the general public was included as an additional source of recruitment.

For all the volunteers, a detailed explanation regarding the study was given after they gave permission to the experimenter to contact them on the telephone. Assessment regarding the PTSD symptoms together with all the inclusion/exclusion criteria were performed over the phone. The inclusion criteria included a current diagnosis of PTSD based on the Structured Clinical Interview for DSM-IV (SCID), age between 20 and 65, and a fluent English speaking skill. The exclusion criteria included a current diagnosis of schizophrenia or other psychotic disorders, a recent history of attempted suicide or active suicidal plans. Concerning the involvement of physiological assessment in the current study, a current diagnosis of cardiovascular or neurological diseases, as well as substance-related disorders

based on DSM-IV were applied as another exclusion criteria. Additionally, in order to ensure that a dissociative state was related to trauma or PTSD, volunteers with a current diagnosis of any dissociative disorders were not included. Similarly, the volunteers who were recognised as too dissociative to complete study procedures by their clinicians or the experimenter were not included. All volunteers were given sufficient time to decide if they were willing to take part before a testing session was booked, and were informed of their rights to withdraw their participation in the study at any time.

Priori power calculations based on a t-test, and a stepwise multiple regression (3 predicting variables overall) with an effect size of 0.15 and a power of 0.8 suggested a sample size of 27 and a sample size of 55, respectively. There were 77 volunteers who expressed an interest and went through the screening procedures. Thirty-one of them did not meet the diagnostic criteria for PTSD; One met a current diagnosis of schizophrenia; One met a current diagnosis of Cannabis dependence; One had a brain injury and suffered from epilepsy. Among the 43 volunteers who passed all the inclusion/exclusion criteria, 16 withdrew before the scheduled session. All of the 27 volunteers that participated completed the study. All of them gave written informed consent (Appendix 14 and 15). They were paid 10 pounds per hour for their participation. There were 5 participants excluded at the data analysis stage due to a high number of artifacts in the ECG data (i.e., more than 3% corrected R-R intervals; Hodson et al., 2010). This resulted in a final sample of 22 (7 males), aged between 25 and 61. The descriptive data of background information and psychological characteristics are summarised in Table 5.1 (see 5.3). Ten participants had comorbid major depressive disorder. Generalised anxiety disorder (n = 5), obsessive compulsive disorder (n = 3), specific phobia (n = 3), social phobia (n = 2), agoraphobia without history of panic disorder (n = 2), panic disorder with (n = 1), and without agoraphobia (n = 2) were present in this sample.

All participants were asked to complete four questionnaires assessing trauma history and related symptoms before attending the study session. The questionnaires included: Life Stressor Checklist –Revised (LSC-R), Posttraumatic Stress Diagnostic Scale (PDS), Peritraumatic Dissociative Experiences Questionnaire (PDEQ), and Dissociative State Scale (DES). Detailed descriptions of these measures are provided in 2.4. Another questionnaire addressing the use of medication, contraceptives, cigarettes, alcohol, caffeine, and illicit drugs, as well as female participants' menstrual cycle and menopause symptoms were also completed before the study session. All participants were asked to avoid illicit drugs and alcohol 7 days, and vigorous exercise 3 days before the study session. They were also asked to refrain from caffeine and nicotine 3 hours before the study. Medication was advised to be taken as usual.

At the beginning of the study, the purposes, procedures, and risks of the study were verbally explained, before a written informed consent was given by the participants. The participants were instructed about the way to wear ECG electrodes. Most of the participants attached the ECG electrodes by themselves, unless assistance was needed and permission was given for the experimenter to do so.

The psychophysiological reactivity test for sHR and CDR was introduced as the first task in the study (refer to 2.3.1 for details). After this test, participants were instructed about the procedures to recall a neutral memory for 5 minutes, and a traumatic memory for 15 minutes (refer to 2.2.1 for detailed instructions). Their permission to be videotaped during these recalls was asked again (they were asked at the phone interview session for the first time). For two participants who did not want to be videotaped, an audiotape was used as a replacement. Psychological states (i.e., state dissociation, fear, threatened and calm feelings) were assessed immediately after the neutral and trauma recalls, and 15 minutes after the

trauma recall. After these, the video taken during the recall of the traumatic memory was played to the participants for the identification of the flashback and dissociative episodes (refer to 2.2.2 for detailed instructions).

At the end, a complete debriefing of the study was given. A leaflet detailing sources of support and treatment as well as the experimenters' details was given to the participants before they left. Participants were encouraged to contact the experimenter if any negative effects occurred after the study or if they wished for any advice concerning treatment. A phone call within 2 days was pre-arranged with all volunteers to confirm how they were feeling after the study and whether further support was needed.

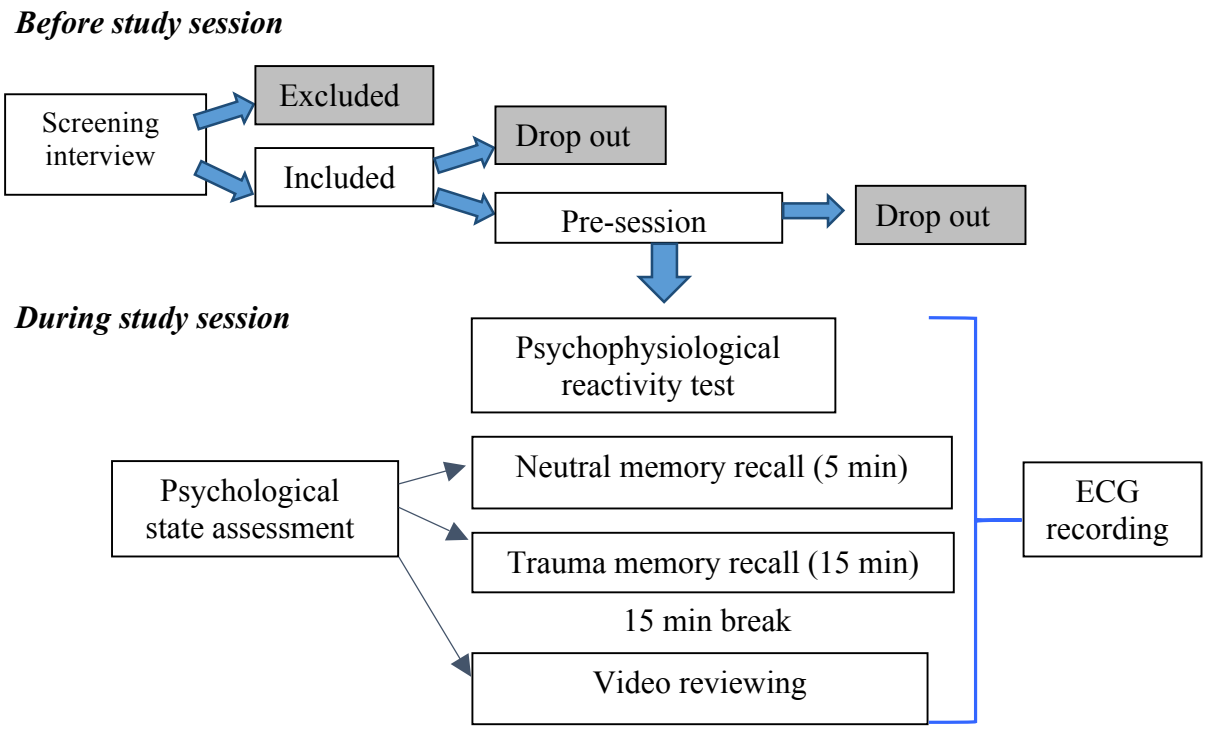


Figure 5.1 *Timeline of the Procedures*

5.2.2 Analytic strategy

All statistical analyses were performed with SPSS version 18 (SPSS Inc, Chicago, IL, USA). Adjustments for outliers and skewed distributions were performed in the same way as described in Chapter Three. Pearson's correlations were used to examine the relationships between life adversities, the trauma-related symptoms (i.e., PTSD symptoms, depression symptoms, and trait dissociation), duration of psychotherapy, and durations of flashbacks and dissociations during the trauma recall.

T-tests were applied to compare the mean levels of HF-HRV, LF-HRV, LFHF-ratio, and HR between the neutral and trauma recalls. In order to make equal length for HRV calculations and comparisons, the mean HRV levels of the trauma recall period were derived from averaging the means of three 5-min segments in the 15-min period. A one-way ANOVA was applied to examine possible HR variations across the three 5-min segments in the trauma recall period. Moreover, t-tests were conducted to examine the fluctuation in HR during the flashback and dissociation periods, in comparison to the 'pure recall' periods (i.e., periods without flashbacks or dissociation) preceding them. Next, Pearson's correlations were used to examine the relationships between the levels of change in HR and HRV (between the trauma and neutral recalls, and between flashbacks and pure recall periods for HR), life adversities, the dissociation measures, trauma-related symptoms, and duration of psychotherapy.

One-way repeat-measure ANOAVs were performed to examine the variance of psychological states (i.e., state dissociation, fearful, threatened, and calm feelings) across different stages of the study (i.e., Time: immediately after the neutral and trauma recalls, and 15 minutes after the trauma recall). Given the multiple levels of the Time factor, tests of linear and quadratic effects replaced tests of main effects. Using the state measure after the

neutral recall as a baseline, the change levels of these psychological states after the trauma recall were examined with Pearson's correlations, in terms of their relationships with life adversities, the trauma-related symptoms, and duration of psychotherapy.

Next, using the same approach as described in 3.2.2, the participants were classified based on their sHR and CDR patterns with Ward's hierarchical cluster analysis. T-tests and chi-square tests were adopted to examine the differences in demographic, physiological and psychological characteristics between the groups. Pearson's correlations were used to examine the relationships between the grouping and the fluctuations of cardiovascular and psychological states responses related to the trauma recall. Finally, step-wise multiple regressions were conducted to examine the effects of HR, HRV, and group measures on predicting the psychological state changes related to the trauma recall.

5.3 Results

5.3.1 Life adversities, trauma-related symptoms, and their relationships with flashbacks and dissociations during trauma recall

Descriptive data for the adversity- and symptom-related measures are summarised in Table 5.1. The number of types of adversities that one had experienced was significantly and positively correlated with PTSD symptom severity ($r = .66$, $p < .01$), peri-traumatic dissociation ($r = .53$, $p < .05$), trait dissociation ($r = .45$, $p < .05$), and depression level ($r = .48$, $p < .05$).

During the trauma recall, most participants ($n = 20$) reported flashbacks. Overall, the mean percentage of time within the recall session when flashbacks occurred was 34.65% ($SD = 29.72\%$). This percentage was not significantly related to the duration of psychotherapy one had received ($r = .11$, $p = .63$), the number of types of adversity ($r = .23$, $p = .34$), peri-traumatic dissociation ($r = .25$, $p = .27$), or overall PTSD symptoms ($r = .36$, $p = .12$). However, higher trait dissociation ($r = .55$, $p < .01$) and depression levels ($r = .44$, $p < .05$) were significantly correlated with a longer duration of flashbacks during the recall.

Nine participants reported having dissociation during the trauma recall. Across the whole sample, the mean percentage of time within the recall session when dissociation occurred was 5.62% ($SD = 10.00\%$). It was not significantly related to the abovementioned psychotherapy, adversity, dissociation, and symptom related measures (largest $r = .34$, $p = .13$). On the other hand, 10 participants reported a state with a mixture of flashbacks and dissociation. The mean percentage of time within the recall session when it occurred was 3.98% ($SD = 6.69\%$). This percentage was significantly and positively correlated with the number of types of adversities one had experienced ($r = .51$, $p < .05$), PTSD symptoms ($r = .48$, $p < .05$), and depression levels ($r = .45$, $p < .05$). However, its correlations with the

duration of psychotherapy (r = .02, p = .92), peri-traumatic dissociation (r = .18, p = .44), and trait dissociation (r = .20, p = .39) were nonsignificant.

Table 5.1 *Mean and Standard Deviations* of *All Variables by Phase*

	N	*Mean (SD)*
Background information		
Age	*22*	*42.36 (10.31)*
BMI (kg/m^2)	*21*	*24.91 (6.32)*
Years in education	*21*	*15.00 (2.35)*
Duration of therapy[a]	*21*	*2.00 (1.18)*
Traumatic experiences and related symptoms		
Nr. adversities	*20*	*11.85 (6.23)*
Peri-traumatic dissociation (10-50)	*21*	*31.43 (9.67)*
Trait dissociation (%)	*21*	*85.80 (55.61)*
PTSD symptom (0-51)	*20*	*32.60 (10.19)*
Depression symptom (0-63)	*22*	*26.91 (15.25)*
Cardiovascular responses		
Heart rate (beat per minute)		
Neutral recall	*22*	*76.63 (7.72)*
Trauma recall	*22*	*76.59 (7.97)*
High frequency heart rate variability (ln)		
Neutral recall	*22*	*301.84 (320.55)*
Trauma recall	*22*	*418.44 (488.05)*
Low frequency heart rate variability (ln)		
Neutral recall	*22*	*1473.45 (1572.72)*
Trauma recall	*22*	*1658.75 (1587.15)*
Low frequency/high frequency ratio		
Neutral recall	*22*	*6.40 (5.04)*
Trauma recall	*22*	*6.13 (4.29)*
Psychological state responses		
State dissociation (0-76)		
Neutral recall	*21*	*15.38 (18.63)*
Trauma recall	*22*	*22.59 (19.67)*
Recovery	*22*	*14.68 (17.04)*
Fear (0-10)		
Neutral recall	*21*	*1.90 (2.70)*
Trauma recall	*22*	*4.18 (3.45)*
Recovery	*22*	*2.27 (2.14)*
Threatened (0-10)		
Neutral recall	*21*	*1.52 (2.58)*
Trauma recall	*22*	*3.00 (3.60)*
Recovery	*22*	*2.09 (2.64)*
Calm (0-10)		
Neutral recall	*21*	*4.33 (3.06)*
Trauma recall	*22*	*3.36 (2.42)*
Recovery	*22*	*3.82 (2.65)*

Note. BMI = body mass index; Nr. adversities = number of types of adversities.

a. The raw data was transformed into ordinal data with 1 equals to 'never received psychotherapy', 2 equals to 'received psychotherapy for one to ten weeks', 3 equals to 'received psychotherapy for eleven weeks to one year', and 4 equals to 'received psychotherapy for more than one year'.

5.3.2 Cardiovascular and psychological responses to trauma recall

Descriptive data for HRV and HR at different phases of the study are summarised in Table 5.1. A significantly higher HF-HRV level was found during the trauma than the neutral recall phases ($t(21) = -2.18$, $p < .05$). However, the differences in LF-HRV ($t(21) = -1.20$, $p = .24$) and LFHF-ratio ($t(21) = .37$, $p = .72$) were nonsignificant. Related to the regulating effect of increased HF-HRV, a significant time effect on HR was found in an one-way ANOVA, with the 10-15 min segment (M = 75.53, SD = 7.38) of the trauma recall showing significantly lower HR than the 5-10 (M = 76.93, SD = 8.88, $p < .05$), and the 0-5 segments (M = 77.32, SD = 8.02, $p < .01$).

In consideration of the overall descending trend of HR during the trauma recall, only the first 5 seconds of each flashback and dissociative period, were included in the calculation of the mean HR during flashback and dissociative periods. With the same consideration, only the last 5 seconds of the 'pure recall' periods right before a flashback or a dissociative period were included in the calculation of the mean HR during 'pre-flashback' and 'pre-dissociation' periods. Five participants who had reported flashbacks, but had none of their flashback periods following a 'pure recall' period were excluded from this part of analysis, due to a lack of comparison baseline. Similarly, five and six participants who had reported dissociation and a mixture of flashbacks and dissociation respectively, but with none of these periods following a pure recall phase, were not included in this part of analysis. Results showed that the mean HR during flashback periods (M = 76.52, SD = 8.68) was significantly higher than during the pre-flashback pure recall periods (M = 74.92, SD = 7.72; $t(19) = -2.37$, $p < .05$). However, due to small sample sizes, differences in HR between the dissociative and pre-dissociation pure recall periods (n = 6), as well as between the mixed periods and the preceding pure recall periods (n = 3) were not analysed.

The change levels of HRV (between trauma and neutral recalls) were not significantly correlated with the duration of psychotherapy, the adversity-, dissociation- and symptom-related measures (largest $r = .27$, $p = .25$ for HF-HRV, largest $r = -.25$, $p = .27$ for LF-HRV, largest $r = .21$, $p = .34$ for LFHF-ratio). Similarly, the change level of HR (between trauma and neutral recalls) did not significantly correlate with any of these measures (largest $r = -.23$, $p = .31$). However, looking specifically at HR during the flashback periods revealed a significant negative correlation between HR and the duration of time one has been receiving psychotherapy ($r = -.47$, $p < .05$). Individuals who have been receiving psychotherapy for longer durations showed lower HR during the flashback periods. Mean HR during flashbacks did not significantly correlate with any of the adversity-, dissociation- and symptom-related measures (largest $r = -.13$, $p = .60$).

Descriptive data for the psychological state measures at different phases of the study are summarised in Table 5.1. A set of one-way repeated measure ANOVAs (phases: neutral recall, trauma recall, recovery) showed significant quadratic effects of time on state dissociation ($F(1, 26) = 36.25$, $p < .001$), fear ($F(1, 26) = 18.71$, $p < .001$), and threat ($F(1, 26) = 12.23$, $p < .01$). A significant linear effect of time on threat was also found ($F(1, 26) = 4.34$, $p < .05$). Post hoc analyses showed that state dissociation was significantly higher after trauma recall than the other phases ($p < .001$). Rating of fear was significantly stronger after the trauma recall than the neutral recall ($p < .001$), and recovery ($p < .01$). Similarly, the feeling of threat was significantly stronger after trauma recall than neutral recall ($p < .01$), and the recovery period ($p < .05$). Moreover, threatened feeling was still stronger after the recovery period than after the neutral recall ($p < .05$).

The amount state dissociation increased after trauma recall (compared to neutral recall) was significantly greater among the individuals who had experienced more types of

adversities ($r = .46$, $p < .05$); but it was not significantly correlated with the duration of psychotherapy, or any of the dissociation- and symptom-related measures (largest $r = .33$, $p = .09$). The amount fear increased after trauma recall (compared to neutral recall) was significantly greater among the individuals who had been receiving psychotherapy for a longer duration of time ($r = .48$, $p < .05$); nevertheless the associations between the levels of fear and the adversity-, dissociation- and symptom-related measures were nonsignificant (largest $r = .18$, $p = .39$). Changes in feelings of threat (largest $r = .28$, $p = .15$) and calm (largest $r = .22$, $p = .29$) did not significantly correlate with any of the above-mentioned measures.

5.3.3 Classification of groups by startle heart rate

Similar to Chapter Three, the second-by-second HR during the psychophysiological reactivity test was used to analyse the patterns of sHR. Two participants were excluded from this part of analyses, as well as the analyses involving CDR in 5.3.4, due to a high number of artifacts in ECG data (i.e., more than 3% corrected R-R intervals) in the psychophysiological reactivity test. HR categorise participants by sHR responses, both two- and three-cluster solutions were applied in Ward's hierarchical cluster analysis. The latter resulted in non-equivalent sample sizes with $n = 1$ in one of the groups. The former resulted in a group with restricted sHR ($n = 12$) and a group with exaggerated sHR ($n = 8$). These two groups were used in the following analyses. They were termed High Startle PTSD Group (HSPG) and Medium Startle PTSD Group (MSPG) respectively (see Figure 5.2) to distinguish from HSG and MSG in Chapter Three and Chapter Four. A 2 (groups: MSPG vs. HSPG) x 11 (time: the 0- to 10-s interval after the white noise onset) mixed design ANOVA showed significant effects of time ($F(10, 180) = 4.84$, $p < .01$), group ($F(1, 18) = 27.05$, $p < .001$), and time by group interaction ($F(10, 180) = 9.80$, $p < .001$). These results indicated significant distinctions between the MSPG and HSPG in HR over the first 10 seconds after the startle probe.

A significant difference was found between the MSPG and HSPG in gender (HSPG: male = 0, female = 8; MSPG: male = 6, female = 6, $X^2(1) = 5.71$, $p < .05$). The differences in age ($t(18) = -1.87$, $p = .08$), BMI ($t(17) = -.38$, $p = .71$), years in education ($t(17) = .88$, $p = .39$), and duration of receiving psychotherapy ($t(17) = -.74$, $p = .47$) were nonsignificant between the two groups. The HSPG had experienced significantly more types of adversity ($t(16) = -2.17$, $p < .05$). They reported significantly greater peri-traumatic dissociation ($t(17) = -2.42$, $p < .05$) than the MSPG. However, the group differences in trait dissociation ($t(17) =$

-1.48, p = .16), PTSD symptom severity (t(16) = -1.91, p = .07), and depression level were nonsignificant (t(18) = -.81, p = .43; See Table 5.2 for descriptive data).

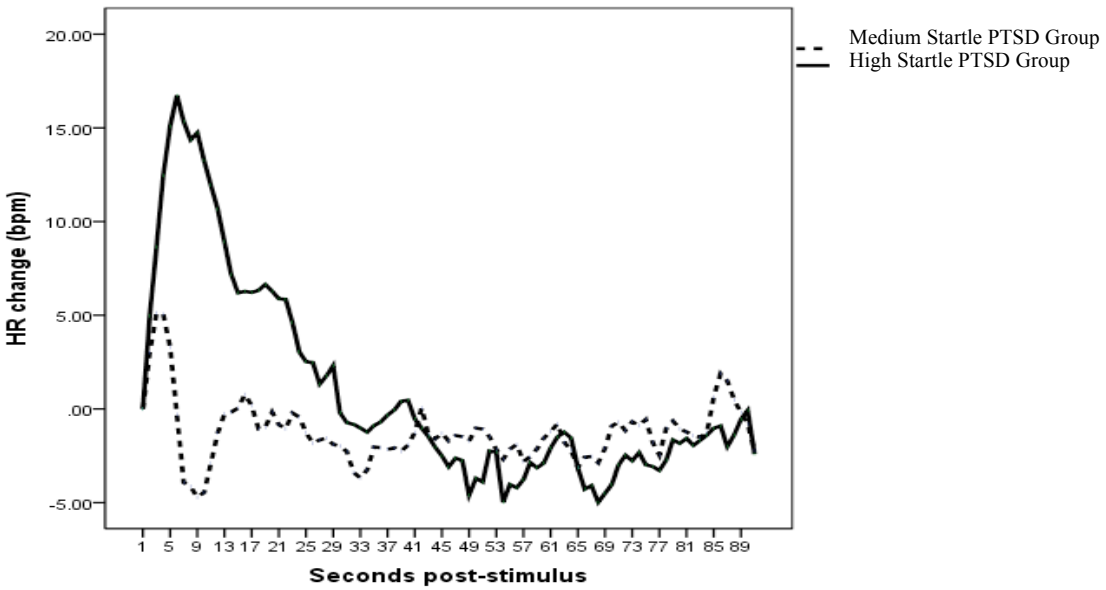


Figure 5.2 *Startle Heart Rate Response by PTSD sHR Group*

Table 5.2 *Mean and Standard Deviations* of *All Variables by sHR Group*

	High Startle PTSD Group		Medium Startle PTSD Group	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Background information				
Age	*8*	*47.50 (10.41)*	*12*	*39.25 (9.15)*
BMI (kg/m^2)	*8*	*26.00 (6.80)*	*11*	*24.91 (6.43)*
Years in education	*8*	*14.50 (3.07)*	*11*	*15.45 (1.63)*
Durations in therapy[a]	*8*	*2.13 (1.25)*	*11*	*1.73 (1.10)*
Traumatic experiences and related symptoms				
Nr. adversities	*7*	*15.86 (5.73)*	*11*	*9.64 (6.05)*
Peri-traumatic dissociation (10-50)	*7*	*38.14 (4.78)*	*12*	*27.83 (10.58)*
Trait dissociation (%)	*8*	*109.85 (44.64)*	*11*	*71.00 (63.54)*
PTSD symptom (0-51)	*7*	*37.29 (5.62)*	*11*	*28.36 (11.40)*
Depression symptom (0-63)	*8*	*30.25 (16.59)*	*12*	*24.25 (15.85)*
Cardiovascular responses				
Heart rate (beat per minute)				
Neutral recall	*8*	*75.06 (6.65)*	*12*	*78.22 (8.96)*
Trauma recall	*8*	*74.38 (6.47)*	*12*	*79.20 (8.67)*
High frequency heart rate variability (ln)				
Neutral recall	*8*	*165.87 (150.61)*	*12*	*295.63 (317.37)*
Trauma recall	*8*	*200.85 (184.64)*	*12*	*392.65 (447.92)*
Low frequency heart rate variability (ln)				
Neutral recall	*8*	*673.57 (485.92)*	*12*	*1743.55 (1883.34)*
Trauma recall	*8*	*904.76 (764.98)*	*12*	*1716.01 (1509.09)*
Low frequency/high frequency ratio				
Neutral recall	*8*	*5.45 (3.77)*	*12*	*7.47 (6.00)*
Trauma recall	*8*	*5.83 (3.89)*	*12*	*6.87 (4.78)*
Psychological state responses				
State dissociation (0-76)				
Neutral recall	*8*	*22.25 (24.79)*	*11*	*11.91 (13.92)*
Trauma recall	*8*	*29.13 (25.15)*	*12*	*20.25 (16.49)*
Recovery	*8*	*17.13 (18.61)*	*12*	*14.00 (17.95)*
Fear (0-10)				
Neutral recall	*8*	*2.63 (3.70)*	*11*	*1.55 (2.02)*
Trauma recall	*8*	*4.75 (4.03)*	*12*	*4.08 (3.34)*
Recovery	*8*	*2.00 (2.56)*	*12*	*2.25 (2.05)*
Threatened (0-10)				
Neutral recall	*8*	*2.50 (3.78)*	*11*	*0.82 (1.25)*
Trauma recall	*8*	*3.25 (4.20)*	*12*	*2.92 (3.65)*
Recovery	*8*	*2.25 (3.15)*	*12*	*1.75 (2.45)*
Calm (0-10)				
Neutral recall	*8*	*3.63 (3.62)*	*11*	*4.91 (2.81)*
Trauma recall	*8*	*3.63 (2.92)*	*12*	*3.42 (2.31)*
Recovery	*8*	*4.38 (2.97)*	*12*	*3.75 (2.63)*

Note. BMI = body mass index; Nr. adversities = number of types of adversities.
a.The raw data was transformed into ordinal data with 1 equals to 'never received psychotherapy', 2 equals to 'received psychotherapy for one to ten weeks', 3 equals to 'received psychotherapy for eleven weeks to one year', and 4 equals to 'received psychotherapy for more than one year'.

5.3.4 Classification of groups by cardiac defence response

Ward's hierarchical cluster analysis was applied to categorise participants based on the CDR. A three-cluster solution was first tested, but rejected for producing an imbalanced distribution of sample sizes, with $n = 1$ in one of the groups. A two-cluster solution was then tested and resulted in two groups with relatively equivalent sample sizes. A group of individuals showing an exaggerated startle response, and an immediate and long-lasting secondary increases in HR was identified and termed PTSD Accelerators ($n = 6$). The other group, which had smaller startle responses to begin with, and a weaker and shorter secondary peak was termed PTSD Decelerators ($n = 14$; see Figure 5.3). A 2 (groups: PTSD Accelerators vs. PTSD Decelerators) x 26 (time: the 20- to 45-s interval after the white noise onset) mixed design ANOVA showed a significant time by group interaction ($F(25, 450) = 3.74$, $p < .001$), and main effects of time ($F(25, 450) = 3.84$, $p < .001$) and Group ($F(1, 18) = 15.09$, $p < .01$). The results indicated significant distinctions between PTSD Accelerators and PTSD Decelerators in HR over the period of 20-45s after the startle probe.

The gender distributions between PTSD Accelerators and PTSD Decelerators did not differ significantly (PTSD Accelerators: male = 2, female = 4; PTSD Decelerators: male = 4, female = 10, $X^2(1) = .05$, $p = .83$). Similarly, the differences in age ($t(18) = 1.24$, $p = .23$), BMI ($t(17) = -.30$, $p = .77$), years in education ($t(17) = .56$, $p = .58$), and duration of receiving psychotherapy ($t(17) = -.15$, $p = .88$) were nonsignificant between the two groups. There were no significant group differences in the numbers of types of adversities experienced ($t(16) = .81$, $p = .43$). The group differences in peri-traumatic dissociation ($t(17) = -.66$, $p = .52$), trait dissociation ($t(17) = -.67$, $p = .51$), PTSD symptom severity ($t(16) = .52$, $p = .61$), and depression level were nonsignificant too ($t(18) = .12$, $p = .90$; See Table 5.3 for descriptive data).

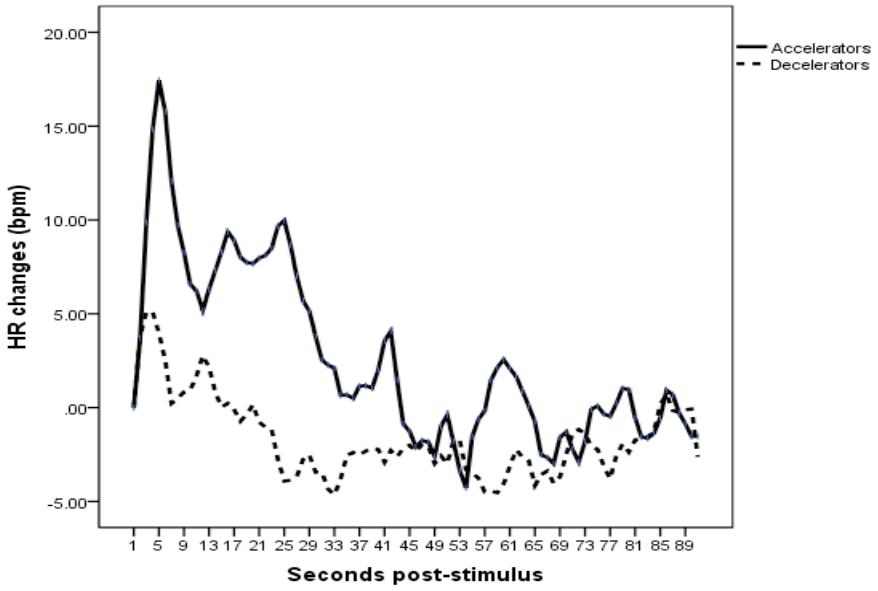


Figure 5.3 *Cardiac Defence Response by PTSD CDR Group*

Table 5.3 *Mean and Standard Deviations* of *All Variables by CDR Group*

	Accelerators		Decelerators	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Background information				
Age	*6*	*46.83 (8.08)*	*14*	*40.71 (10.82)*
BMI (kg/m^2)	*6*	*24.50 (3.15)*	*13*	*25.77 (7.56)*
Years in education	*6*	*15.50 (2.88)*	*13*	*14.85 (2.12)*
Durations in therapy[a]	*6*	*1.83 (1.17)*	*13*	*1.92 (1.19)*
Traumatic experiences and related symptoms				
Nr. adversities	*6*	*13.83 (7.60)*	*12*	*11.17 (6.12)*
Peri-traumatic dissociation (10-50)	*6*	*29.33 (10.67)*	*13*	*32.69 (10.09)*
Trait dissociation (%)	*6*	*73.97 (36.77)*	*13*	*93.54 (66.44)*
PTSD symptom (0-51)	*6*	*33.67 (7.34)*	*12*	*30.92 (11.79)*
Depression symptom (0-63)	*6*	*27.33 (11.52)*	*14*	*26.36 (17.96)*
Cardiovascular responses				
Heart rate (beat per minute)				
Neutral recall	*6*	*79.15 (10.30)*	*14*	*76.02 (7.17)*
Trauma recall	*6*	*77.97 (9.53)*	*14*	*76.98 (7.71)*
High frequency heart rate variability (ln)				
Neutral recall	*6*	*203.85 (153.78)*	*14*	*260.81 (305.80)*
Trauma recall	*6*	*248.95 (179.73)*	*14*	*344.64 (431.65)*
Low frequency heart rate variability (ln)				
Neutral recall	*6*	*981.30 (613.59)*	*14*	*1458.81 (1825.56)*
Trauma recall	*6*	*1272.32 (834.41)*	*14*	*1442.59 (1485.92)*
Low frequency/high frequency ratio				
Neutral recall	*6*	*5.69 (2.92)*	*14*	*7.08 (5.97)*
Trauma recall	*6*	*6.03 (3.15)*	*14*	*6.63 (4.90)*
Psychological state responses				
State dissociation (0-76)				
Neutral recall	*6*	*11.17 (15.66)*	*13*	*18.62 (20.98)*
Trauma recall	*6*	*19.00 (18.31)*	*14*	*25.86 (21.35)*
Recovery	*6*	*12.83 (16.29)*	*14*	*16.29 (18.89)*
Fear (0-10)				
Neutral recall	*6*	*2.00 (3.03)*	*13*	*2.00 (2.83)*
Trauma recall	*6*	*3.00 (3.69)*	*14*	*4.93 (3.45)*
Recovery	*6*	*2.00 (3.10)*	*14*	*2.21 (1.85)*
Threatened (0-10)				
Neutral recall	*6*	*2.00 (3.16)*	*13*	*1.31 (2.53)*
Trauma recall	*6*	*2.50 (3.73)*	*14*	*3.29 (3.91)*
Recovery	*6*	*2.17 (3.37)*	*14*	*1.86 (2.48)*
Calm (0-10)				
Neutral recall	*6*	*4.17 (3.76)*	*13*	*4.46 (2.99)*
Trauma recall	*6*	*4.83 (2.79)*	*14*	*2.93 (2.23)*
Recovery	*6*	*4.33 (3.78)*	*14*	*3.86 (2.28)*

Note. BMI = body mass index; Nr. adversities = number of types of adversities.
The raw data was transformed into ordinal data with 1 equals to 'never received psychotherapy', 2 equals to 'received psychotherapy for one to ten weeks', 3 equals to 'received psychotherapy for eleven weeks to one year', and 4 equals to 'received psychotherapy for more than one year'.

5.3.5 Group differences in cardiovascular and psychological reactions to trauma recall

This section examines the effects of the above-mentioned group differences on the cardiovascular and psychological responses to voluntary recall of trauma, as well as the relationships between the two. Pre-existing differences (i.e., gender, number of types of adversities, peri-traumatic dissociation) have been found between the LSPG and HSPG. Because the sample size of the current study was not sufficient to account for the variance caused by these factors, the sHR groups were not included in this part of analysis.

The change levels of HF-HRV, LF-HRV, LFHF-ratio and HR between trauma and neutral recalls were not significantly correlated with the CDR groups (largest $r = .28$, $p = .23$). The categorisation of CDR group was not significantly related to the difference in HR between the flashback and 'pure recall' periods ($r = .08$, $p = .75$). Similarly, the correlations between the CDR groups and change levels (between trauma and neutral memory recalls) of state dissociation, fear, threatened, and calmness feelings were nonsignificant (largest $r = -.31$, $p = .20$).

In terms of the associations between the cardiovascular and psychological reactions to trauma recall, as shown in Table 5.4, a greater HR decrease during the trauma recall (compared to neutral recall) was significantly predictive of greater increases of fearful, threatened, and calmness feelings after recalling a trauma. Moreover, being identified as a PTSD Accelerator was predictive of less increases of fearful and threatened feelings related to the trauma recall.

Similar multiple regressions were performed to examine the effects of HRV on predicting changes in psychological states. Greater increases in HF-HRV and greater decreases in LF-HRV significantly predicted greater increases in state dissociation and threatened feelings (Table 5.5 and 5.6). Moreover, as shown in Table 5.7, a decrease in the

LFHF-ratio during trauma recall significantly predicted an increase in state dissociation. The same set of predictors did not significantly predict changes in other psychological states (largest R^2 = .20, p = .17 at step one; largest ΔR^2 = .12, p = .12 at step two).

Table 5.4 *Multiple Regressions with Changes in Psychological States as Dependent Variables, Heart Rate Change and CDR Group as Independent Variables.*

	B	*SE B*	β
Dependent variable: State dissociation change			
Step 1: R^2 = .16, p = .24, df1 = 2, df2 = 16			
Constant	3.00	.80	
HR change	-.39	.23	-.42
CDR	.17	.46	.09
Step 2: ΔR^2 = .05, p = .34, df1 = 1, df2 = 15			
Constant	2.82	.82	
HR change	-1.31	.95	-1.40
CDR	.23	.47	.12
HR change x CDR	.52	.53	1.00
Dependent variable: Fear change			
Step 1: R^2 = .65, p < .001, df1 = 2, df2 = 16			
Constant	-3.05	1.69	
HR change	-2.42	.48	-.79***
CDR	3.36	.98	.53**
Step 2: ΔR^2 = .01, p = .65, df1 = 1, df2 = 15			
Constant	-3.23	1.78	
HR change	-3.36	2.08	-1.09
CDR	3.43	1.02	.54**
HR change x CDR	.53	1.15	.31
Dependent variable: Threatened change			
Step 1: R^2 = .46, p < .01, df1 = 2, df2 = 16			
Constant	-3.09	2.14	
HR change	-2.04	.60	-.65**
CDR	3.01	1.24	.47*
Step 2: ΔR^2 = .00, p = .88, df1 = 1, df2 = 15			
Constant	-3.16	2.27	
HR change	-2.42	2.64	-.78
CDR	3.04	1.30	.47*
HR change x CDR	.22	1.46	.13
Dependent variable: Calmness change			
Step 1: R^2 = .18, p = .21, df1 = 2, df2 = 16			
Constant	1.90	2.81	
HR change	-.99	.79	-.30
CDR	-1.51	1.64	-.22
Step 2: ΔR^2 = .16, p = .08, df1 = 1, df2 = 15			
Constant	.77	2.68	
HR change	-6.71	3.12	-2.02*
CDR	-1.08	1.54	-.16
HR change x CDR	3.26	1.73	1.75

*$p < .05$; **$p < .01$.

Note. HR change = the level of HR during trauma recall minus the level of HR during neutral recall.

Table 5.5 *Multiple Regressions with Changes in Psychological States as Dependent Variables, HF-HRV Change and CDR Group as Independent Variables.*

	B	*SE B*	β
Dependent variable: State dissociation change			
Step 1: R^2 = .26, p = .09, df1 = 2, df2 = 16			
Constant	3.32	.72	
HF-HRV change	.45	.19	.51*
CDR	-.04	.41	-.02
Step 2: ΔR^2 = .11, p = .13, df1 = 1, df2 = 15			
Constant	3.40	.69	
HF-HRV change	1.53	.71	1.75*
CDR	-.04	.40	-.02
HF-HRV change x CDR	-1.14	.72	-1.28
Dependent variable: Fear change			
Step 1: R^2 = .27, p = .09, df1 = 2, df2 = 16			
Constant	-.95	2.37	
HF-HRV change	1.22	.62	.42
CDR	1.97	1.36	.31
Step 2: ΔR^2 = .01, p = .71, df1 = 1, df2 = 15			
Constant	-.88	2.44	
HF-HRV change	2.12	2.49	.74
CDR	1.96	1.39	.31
HF-HRV change x CDR	-.95	2.54	-.32
Dependent variable: Threatened change			
Step 1: R^2 = .30, p = .07, df1 = 2, df2 = 16			
Constant	-1.34	2.39	
HF-HRV change	1.34	.62	.46*
CDR	1.86	1.37	.29
Step 2: ΔR^2 = .00, p = .88, df1 = 1, df2 = 15			
Constant	-1.37	2.47	
HF-HRV change	.95	2.53	.32
CDR	1.86	1.41	.29
HF-HRV change x CDR	.41	2.58	.14
Dependent variable: Calmness change			
Step 1: R^2 = .10, p = .43, df1 = 2, df2 = 16			
Constant	2.81	2.84	
HF-HRV change	-.21	.74	-.07
CDR	-2.15	1.63	-.31
Step 2: ΔR^2 = .14, p = .12, df1 = 1, df2 = 15			
Constant	3.15	2.71	
HF-HRV change	4.19	2.77	1.34
CDR	-2.17	1.55	-.32
HF-HRV change x CDR	-4.65	2.83	-1.46

*$p < .05$.

Note. HF-HRV change = the level of HF-HRV during trauma recall minus the level of HF-HRV during neutral recall.

Table 5.6 *Multiple Regressions with Changes in Psychological States as Dependent Variables, LF-HRV Change and CDR Group as Independent Variables.*

	B	*SE B*	β
Dependent variable: State dissociation change			
***Step 1:* R^2 = .23, *p* = .12, *df1* = 2, *df2* = 16**			
Constant	3.29	.74	
LF-HRV change	-.43	.19	-.48*
CDR	-.02	.42	-.01
***Step 2:* ΔR^2 = .13, *p* = .10, *df1* = 1, *df2* = 15**			
Constant	3.40	.70	
LF-HRV change	-1.67	.73	-1.89*
CDR	-.03	.40	-.01
LF-HRV change x CDR	1.30	.74	1.45
Dependent variable: Fear change			
***Step 1:* R^2 = .23, *p* = .12, *df1* = 2, *df2* = 16**			
Constant	-1.01	2.42	
LF-HRV change	-1.12	.64	-.39
CDR	2.01	1.38	.32
***Step 2:* ΔR^2 = .01, *p* = .64, *df1* = 1, *df2* = 15**			
Constant	-.91	2.48	
LF-HRV change	-2.34	2.59	-.80
CDR	2.00	1.42	.32
LF-HRV change x CDR	1.27	2.63	.43
Dependent variable: Threatened change			
***Step 1:* R^2 = .29, *p* = .07, *df1* = 2, *df2* = 16**			
Constant	-1.41	2.37	
LF-HRV change	-1.38	.63	-.47*
CDR	1.93	1.36	.30
***Step 2:* ΔR^2 = .00, *p* = .86, *df1* = 1, *df2* = 15**			
Constant	-1.47	2.46	
LF-HRV change	-.95	2.57	-.32
CDR	1.93	1.40	.30
LF-HRV change x CDR	-.45	2.60	-.15
Dependent variable: Calmness change			
***Step 1:* R^2 = .11, *p* = .40, *df1* = 2, *df2* = 16**			
Constant	2.85	2.83	
LF-HRV change	.35	.75	.11
CDR	-2.18	1.62	-.32
***Step 2:* ΔR^2 = .15, *p* = .11, *df1* = 1, *df2* = 15**			
Constant	3.25	2.68	
LF-HRV change	-4.32	2.80	-1.37
CDR	-2.20	1.53	-.32
LF-HRV change x CDR	4.89	2.84	1.53

*$p < .05$.

Note. LF-HRV change = the level of LF-HRV during trauma recall minus the level of LF-HRV during neutral recall.

Table 5.7 *Multiple Regression with Change of State Dissociation as a Dependent Variables, Change in LFHF-ratio and CDR Group as Independent Variables.*

	B	*SE B*	β
Dependent variable: State dissociation change			
Step 1: R^2 = .44, p < .01, df1 = 2, df2 = 16			
Constant	3.56	.63	
LFHF-ratio change	-.58	.16	-.67**
CDR	-.19	.36	-.10
Step 2: ΔR^2 = .12, p = .12, df1 = 1, df2 = 15			
Constant	3.64	.60	
LFHF-ratio change	-1.66	.67	-1.92*
CDR	-.22	.34	-.12
LFHF-ratio change x CDR	1.17	.71	1.29
Dependent variable: Fear change			
Step 1: R^2 = .10, p = .43, df1 = 2, df2 = 16			
Constant	-.72	2.63	
LFHF-ratio change	-.34	.68	-.12
CDR	1.78	1.50	.28
Step 2: ΔR^2 = .06, p = .30, df1 = 1, df2 = 15			
Constant	-.52	2.62	
LFHF-ratio change	-3.40	2.94	-1.20
CDR	1.68	1.50	.27
LFHF-ratio change x CDR	3.29	3.09	1.10
Dependent variable: Threatened change			
Step 1: R^2 = .14, p = .30, df1 = 2, df2 = 16			
Constant	-.96	2.62	
LFHF-ratio change	-.76	.67	-.26
CDR	1.59	1.50	.25
Step 2: ΔR^2 = .01, p = .66, df1 = 1, df2 = 15			
Constant	-.87	2.69	
LFHF-ratio change	-2.09	3.02	-.72
CDR	1.55	1.54	.24
LFHF-ratio change x CDR	1.43	3.17	.47
Dependent variable: Calmness change			
Step 1: R^2 = .20, p = .17, df1 = 2, df2 = 16			
Constant	2.44	2.70	
LFHF-ratio change	.98	.69	.32
CDR	-1.95	1.54	-.29
Step 2: ΔR^2 = .12, p = .12, df1 = 1, df2 = 15			
Constant	2.76	2.57	
LFHF-ratio change	-3.66	2.88	-1.19
CDR	-2.11	1.47	-.31
LFHF-ratio change x CDR	5.01	3.02	1.54

*p < .05; **p < .01.

Note. LFHF-ratio change = the level of LFHF-ratio during trauma recall minus the level of LFHF-ratio during neutral recall.

5.4 Discussion

5.4.1 Cardiovascular indicators of the psychological arousal induced by voluntary retrieval of traumatic memory

Emotional engagement during an exposure-based psychotherapy of PTSD is an essential element related to a successful treatment outcome (Foa, Molnar, & Cashman, 1995). As an indicator of emotional arousal, smaller HR increases in response to voluntary recall of a traumatic memory at an early stage post trauma have been related the development of PTSD and poorer recovery (Halligan et al., 2006). However, the psychological implications of HR fluctuation were unclear. The current study aimed to further investigate the associations between the variations in HR and psychological states related to the recall of traumatic memory. Measures of HRV were assessed in order to clarify the roles which the sympathetic nervous system and vagal system each play.

Significant increases in vagal activity, as indicated by elevated HF-HRV levels, were found during the recollection of a trauma, in comparison to recall of a neutral daily routine. This finding was inconsistent with a previous study, which similarly recorded HRV while PTSD patients were asked to voluntarily and vividly recall trauma, but found an association between decreased vagal activity during trauma recall and PTSD (Keary, Hughes, & Palmieri, 2009). A few differences in study designs may have contributed to the inconsistency. First, we compared HRV during trauma recall with neutral recall, whereas the previous study (Keary, Hughes, & Palmieri, 2009) compared the HRV between a period of trauma recall and another period of pure resting. Since effects on HRV of the action of speaking, and of the cognitive activities involved in memory recall itself have been demonstrated (e.g., Hauschildt et al., 2011), the inconsistent findings between the two studies may partially be explained by this difference in study design. Moreover, Keary and

colleagues (2009) drew their conclusion about the association between greater decreased vagal activity and PTSD by comparing the HRV patterns of PTSD patients and healthy controls. As we only included PTSD patients in the current study, it is unclear how healthy individuals may respond to our study design, relative to our current sample. Therefore, it is hard to compare our findings with Keary and colleagues (2009). Finally, as the previous study (Keary, Hughes, & Palmieri, 2009) only included females, gender differences in the response of the cardiovascular system to traumatic stimuli may play a role in the inconsistent findings.

Additionally, in the current study, a greater increase in vagal activity, a greater decrease in sympathetic activation, and correspondingly, a greater decrease in the LFHF ratio were found to be associated with a greater increase of state dissociation and feeling of threat. These results, together with the finding of heightened state dissociation during the trauma recall, suggested a passive coping mechanism triggered by the voluntary retrieval of traumatic memory and indicated by a dominant vagal activation. Notably, supporting the previous study which did not find a significant correlation between HR variations during voluntary retrieval of trauma and self-report numbing (Halligan et al., 2006), change in HR did not significantly predict state dissociation in the current study. Overall, the findings suggest that HRV, in contrast to HR, may be a more sensitive indicator of dissociation.

Associations have been shown between HR and mood fluctuations related to the voluntary recall of a trauma in the current study. Ratings of fear and threat increased in response to the recall of trauma. Contrary to the hypotheses, however, greater levels of these increases were indicated by smaller increases (or greater decreases) in HR during the voluntary recall of a trauma (compared to a neutral memory). These findings supported a previous study (Holmes et al., 2004), in which HR decreases under stress were viewed as a

psychophysiological reaction to great levels of threat and fear, similar to freezing behaviours observed in animals. Additionally, a decrease in calmness ratings was found after the recollection of trauma, with a larger level of such decrease being significantly predicted by a greater level of HR increase. As a greater decrease in calmness indicates greater emotional arousal, its association with a larger amount of HR increase suggested a positive association between emotional and physiological arousal.

A few factors related to the study designs may contribute to the inconsistency between the current and the previous study (Halligan et al., 2006), which showed nonsignificant correlations between HR and subjective feelings related to the recollection of trauma. First, agreeing with the hypotheses in 5.1.1, by including PTSD patients and adopting a neutral recall task as a baseline, the current study might have eliminated unnecessary variation associated with levels of PTSD symptoms, and different tasks (i.e., pure rest vs. verbally recall a memory). Second, by separately enquiring about different kinds of psychological states, instead of rating a general feeling of distress, the current study might have adopted a more specific, and therefore more sensitive measurement to investigate this topic.

Overall, associations between HR, HRV, and trauma-related psychological states have been shown. Additionally, a significant elevation in HR has been found while flashbacks occurred during the recollection of trauma. This finding suggest the validity of utilising HR as an indicator of flashbacks in an exposure therapy session to monitor and assess the mental states related to beneficial and unfavourable treatment outcomes. Overall the results highlight the practical potentials of these cardiovascular indices in clinical settings.

5.4.2 sHR, cardiac defence response, and their relationships with the psychological arousal induced by voluntary retrieval of traumatic memory

Following the previous chapters, sHR and CDR were examined among the current PTSD patient sample. Agreeing with the literature suggesting diverse patterns of startle among PTSD patients (Morgan & Grillon, 1998), a group showing exaggerated sHR (i.e., HSPG), and another group with moderate sHR (i.e., MSPG) were found. Moreover, indirectly supporting a previous study, which indicated an association between heightened startle eye blink levels and greater childhood abuse experiences (Jovanovic et al., 2009), more experiences of life adversities were found among the HSPG. Additionally, in the current study, being in the HSPG was found to relate to two other well-established risk factors for PTSD: being female, and experiencing greater levels of peri-traumatic dissociation. Because startle is a psychophysiological feature that shows a large variability between individuals, and a high consistency across time, it has been suggested to be a good study marker or screening risk factor for PTSD (Morgan & Grillon, 1998). Given the current findings on its associations with other risk factors, further replications are of research interest.

Different from the patterns found among the healthy individuals in Chapter Three, a group with only a sudden suppression, but without a sHR response (i.e., the Low Startle Group) was not found among the patient sample. Considering the finding of a higher trait dissociation among the LSG in the healthy sample, the absence of a group resembling the LSG might be related to one of our exclusion criteria, which excluded volunteers with high levels of dissociation. Studies including PTSD patients with a broader range of dissociation level should be conducted in the future, in order to more completely examine the patterns of sHR and its role as a risk factor within this population.

Examinations of the role of CDR in predicting the psychological and physiological responses to trauma recall did not locate significant group differences in the HR and HRV fluctuations related to the trauma recall and flashbacks during the recall. Inconsistent with the hypotheses, a significant moderating effect of the CDR was not found in the relationship between HR and psychological state variations either. However, CDR was found to be predictive of the levels of emotional arousal induced by the recollections of trauma. The PTSD Accelerators reported significantly smaller increases in fearful and threatened feelings related to the trauma recall (compared to the neutral recall), which suggested a restricted level of emotional arousal. As emotional engagement has been well established as the key to a successful treatment of PTSD, further investigations regarding the association between CDR and indices of treatment effects are of research interest.

5.4.3 Life adversities, dissociations, psychotherapy, and symptoms affecting the emotional and psychophysiological arousals during the recollections of trauma

Levels of emotional and psychophysiological arousals have been regarded as essential factors linked to the outcomes of psychotherapy for PTSD (Foa, Molnar, & Cashman, 1995; Halligan et al., 2006; van Minnen & Hagenaars, 2002). The current study investigated the associations between these reactions and other associates of PTSD. Inconsistent with our hypotheses, trait dissociation and peri-traumatic dissociation did not significantly relate to the levels of increases in the state dissociation resulting from the trauma recall. Moreover, these dissociation measures, which targeted the time prior to the study, also did not significantly correlate with the cardiovascular responses to the trauma recall. These findings highlight the state dependent nature of dissociation, and support the previous suggestion about the need to assess an acute state of dissociation when relating it to any study manipulations, such as recalling a trauma in the current case (Halligan et al., 2006; Sack, Cillien, & Hopper, 2012). Furthermore, these negative findings echo a previous study (Hagenaars, van Minnen, & Hoogduin, 2010), in which PTSD patients with different pretreatment severity of dissociation and depression were found to benefit similarly from exposure-based therapy.

A significant association was found between having been exposed to more life adversities and a greater increase of state dissociation resulting from the trauma recall. This finding supported our hypothesis and a previous study which suggested long lasting traumatic experiences as a risk factor for the development of the tendency to switch into passive defensive mechanisms, such as dissociation (van der Kolk, et al., 1996). However, impacts of multiple life adversities on the cardiovascular responses to the recollection of trauma were not significant.

In examining the effects of the symptom and treatment related factors, agreeing with the previous study (Hagenaars, van Minnen, & Hoogduin, 2010), PTSD and depression symptom severities were not significantly related to the psychological state changes, or cardiovascular reactions to the trauma recall. However, a longer duration of psychotherapy one has received significantly predicted a greater amount of increase in fear related to the trauma recall. Moreover, a longer duration in psychotherapy was also related to a smaller increase in HR during the flashback periods. These findings may suggest the effects of psychotherapy on emotionally engaging the clients, and reducing the physiological reactions resulted from psychological distress. Nevertheless, as many confounding variables, such as the types of therapy and the severity of PTSD, have been involved in the current design, replications with a better controlled sample are needed to further examine this topic.

Chapter 6: Cortisol and sAA levels during voluntary memory retrieval of trauma: investigation of individual differences

6.1 Introduction and hypotheses

Given the important roles of cortisol in stress coping and memory processing, investigations regarding the impacts of trauma and PTSD on one's later responses to new traumatic stimuli, and therapeutic procedures involving traumatic memory retrieval are crucial for determining potential psychophysiological vulnerabilities among the PTSD populations. In Chapter four, we found that healthy individuals with more recent experiences of, and severer psychological impacts from trauma responded to a later stressor with a less activated HPA axis response. This finding suggests a higher level of risk among this population, as an insufficient cortisol release during memory encoding process has been suggested to be a risk factor associated with the development of intrusive traumatic memories (Yehuda & Harvey, 1997). As reprocessing of traumatic memory is an essential part of exposure based psychotherapies for PTSD, it is of research interest to examine the level of cortisol in response to these therapies among PTSD patients.

Moreover, in Chapter Four, a higher level of cortisol has been found to be associated with the development of more frequent intrusions among those who are more prone to fight/flight response to stress (i.e., the Accelerators of cardiac defence response (CDR)), and those who had greater salivary alpha-amylase (sAA) level that indicated greater activation in the sympathetic nervous system (SNS) during trauma. Based on these findings, it is important to investigate the SNS response to procedures of exposure therapy, as well as the role of CDR and other potential sources of individual differences.

As reviewed in 1.4.2, it has been found that in response to an interview about trauma, war- and torture-related PTSD patients with rape experiences showed heighted cortisol

release, while patients without rape history showed decreases in cortisol secretion (Gola et al., 2012). The authors suggested that as peri-traumatic dissociation is commonly found among rape victims, it and posttraumatic dissociative symptoms that later developed were likely to be moderating factors underlying the association between rape history and a heightened HPA axis response during the trauma interview (Gola et al., 2012).

Based on the existing findings, the current chapter was interested in the impact of exposure-based therapies on cortisol and sAA levels among PTSD patients. Specifically, we aimed to examine the patterns of reactive cortisol and sAA levels in response to voluntary traumatic memory retrieval, which is a procedure commonly involved in exposure-based psychotherapies. In order to do so, we adopted a study design similar to Chapter Five. In addition to the procedures involved in Chapter Five, salivary cortisol and sAA samples were collected after the neutral and trauma recalls. Similar to the previous chapters, potential sources of individual differences were examined: We investigated, first, the relationships between previous life adversities, PTSD symptoms, duration of psychotherapy, and the change levels of cortisol and sAA in response to the neutral vs. trauma recall. Second, the group differences in cortisol and sAA levels between individuals with different cardiovascular threat response features (i.e., the PTSD Accelerators, and PTSD Decelerators) were examined. Additionally, following Gola and colleagues (2012), the current study explored the correlations between past experiences of rape, dissociation (i.e., trait dissociation, peri-traumatic dissociation, whether or not one had dissociative experiences during the voluntary memory retrieval, and an overall rating for state dissociation), and the reactive cortisol and sAA levels during the memory retrieval procedures.

Due to variations in study designs (see detailed review in 1.4.2), inconsistent results regarding the responses of the HPA axis to trauma-related stimuli have been found among

PTSD patients (e.g., Elzinga et al., 2003; Geracioti et al., 2008; Gola et al., 2012). On the other hand, sAA level during voluntary retrieval of traumatic memory has not been examined to date. As such, a directional hypothesis was not made in our study, in terms of the variation of cortisol and sAA after the neutral and trauma recalls.

However, based on the previous findings (Gola et al., 2012), individuals with rape experiences and those with greater dissociation levels were predicted to have greater cortisol increases related to the trauma recall. Moreover, as a significant association between greater trait dissociation and lower sAA levels has been found in Chapter Four, correlations in the same direction were hypothesised between sAA levels and the dissociation measures in the current study. On the other hand, although CDR Accelerators were suggested to be prone to extreme threat coping behaviours in the literature (Richards & Eves, 1991), the PTSD Accelerators and PTSD Decelerators did not show significant differences in terms of their dissociative experiences in Chapter Five. Given this, a directional hypothesis was not made about their reactive cortisol patterns in the current study.

The relationships between baseline cortisol and sAA levels, past traumatic experience, and subclinical PTSD symptom severity were nonsignificant in our investigation in Chapter Four. However, the associations between the recentness and severity of previous trauma and reactive cortisol levels were significant. Although these findings were relevant to the current investigation, a different population (i.e., PTSD patients) and different memory processing mechanism (i.e., voluntary memory retrieval) were included in the current chapter. As a result, specific hypotheses were not made for the associations between cortisol and sAA levels, previous life adversities, PTSD symptom severity, and the duration of receiving psychotherapy.

6.2 Methods

The participants of this study were from the same cohort as Chapter Five. All of them gave written informed consent (Appendix 14 and 15). Priori power calculations based on a 2 (groups) x 2 (times) mixed design ANOVA with an effect size of 0.25 and a power of 0.8 suggested a sample size of 34. Among the 27 volunteers that completed the study, one had too little saliva collected for analysis, and the other one dropped and contaminated the samples. Another participant was excluded due to unusually high levels of cortisol measured (i.e., >110 nmol/L). Therefore, the sample size of the analyses involving sAA levels was 25 (male = 11; age range = 20 to 61, M = 41.60, SD = 10.82), whereas the sample size of the analyses involving cortisol levels was 24 (male = 10; ages range = 20 to 61, M = 41.25, SD = 10.91). In the analyses involving CDR, the sample sizes decreased to 18 and 17, respectively, after excluding participants with high amounts of artifacts (i.e., more than 3% corrected R-R intervals; Hodson et al., 2010) in their ECG data. The procedures in Chapter Five were applied to the current sample, with the saliva sampling immediately after the neutral and trauma recalls as additional elements. Detailed descriptions of the relevant experimental manipulation, physiological and psychological measures are introduced in 2.2, 2.3 and 2.4.

All statistical analyses were performed with SPSS version 21 (SPSS Inc, Chicago, IL, USA). Adjustment for outliers and skewed distributions were performed in the same way as the previous chapters. Pearson's correlations were first used to examine the levels of cortisol and sAA after the two types of recalls (i.e., neutral and trauma recalls) in relation to sex and age, as well as the relationships between the cortisol and sAA levels in these two memory retrieval conditions.

Following these, one-way ANOVAs were applied to the overall sample to examine the levels of cortisol and sAA after the two types of recalls (i.e., neutral and trauma recalls).

Next, 3 sets of 2 (group: PTSD Accelerators vs. PTSD Decelerators as defined in 5.3.4; individuals with vs. without rape history; individuals who identified dissociative episodes vs. those who did not identify such episodes during the trauma recall) x 2 (time: neutral vs. trauma recalls) mixed design ANOVAs were performed to examined the variance of cortisol across different subgroups and tasks. The same analyses were applied to examine the variance in sAA levels. Homogeneity of variance was assessed by Levene's statistic, while sphericity was examined with Mauchly's test. When the assumption of sphericity was not met, Greenhouse-Geisser ε was reported.

Moreover, partial correlations were used to investigate the relationships between the numbers of type of life adversity, PTSD symptom severity, duration of psychotherapy, dissociation measures (i.e., peri-traumatic dissociation, trait and state dissociation), and cortisol and sAA levels after trauma recall, with their levels after neutral recall controlled.

Finally, as exploratory analyses, Pearson's correlations were applied to examine the relationships between the abovementioned adversity-, dissociation-, and symptom-related variables, and the levels of cortisol and sAA after the neutral as well as trauma recalls. Moreover, the relationships between all physiological measures assessed among this sample, including cortisol, sAA, HR and HRV, in the two recall conditions (e.g., change level of HR and change level of cortisol) were examined with Pearson's correlations. Change levels of the physiological measures were calculated by subtracting the level after the neutral recall from the level after the trauma recall. Given a large number of analyses, a more stringent alpha level ($p < .01$) was applied to these exploratory analyses.

6-3 Results

Descriptive data for cortisol and sAA levels are summarised in Table 6.1 and Table 6.2. Significant and positive correlations between the secretion levels after the two memory retrieval conditions (i.e., neutral and trauma recall) were found for both cortisol (r = .87, p < .001) and sAA (r = .74, p < .001). The levels of cortisol after both types of recall were not significantly correlated with sex or age (largest r = -.09, p = .67). However, a significant correlation between being female and a lower sAA secretion after the neutral recall was found (r = -.47, p < .05; M = 37.36, SD = 25.32 for female; M = 75.75, SD = 48.27 for male), although sAA level at this stage was not significantly correlated with age (r = -.12, p = .55), and sAA level after the trauma recall was not significantly associated with sex (r = -.32, p = .12) or age (r = -.27, p = .18). Due to this finding, sex was included as a covariate and controlled in the following analyses involving sAA level after neutral recall.

A trend for lower levels of cortisol was found after the trauma recall, in comparison to the neutral recall (F(1, 23) = 3.90, p = .06), but not for the levels of sAA (F(1, 23) = .21, p = .65). When the overall sample was divided into a group with a rape history and another group without such a history, the effects of group, and group by time interaction were nonsignificant for cortisol (F(1, 21) = .08, p = .79 for group effect; F(1, 21) = .00, p = .95 for group by time interaction) and sAA levels (F(1, 21) = 1.03, p = .32 for group; F(1, 21) = .15, p = .71 for group by time interaction).

In contrast, examining the role of the cardiac defence response (CDR) showed a significant group by time interaction for cortisol level (F(1, 15) = 6.49, p < .05), although the main effect of group (F(1, 15) = 1.80, p = .20) was nonsignificant. Neither the group by time interaction (F(1, 15) = .41, p = .53), nor the main effect of group (F(1, 15) = .44, p = .52) were significant on sAA level.

Similarly, separating the sample by whether one has experienced dissociation during the recall yielded a significant group by time interaction for cortisol level ($F(1, 22) = 5.01$, $p < .05$), despite a nonsignificant group effect ($F(1, 22) = .05$, $p = .83$). However, neither the group by time interaction ($F(1, 22) = .37$, $p = .55$), nor the main effect of group ($F(1, 22) = 2.80$, $p = .11$) were significant for sAA level.

Post hoc analyses suggested that while a significantly lower level of cortisol was found among the PTSD Accelerators after the trauma recall, in comparison to the neutral recall ($t(5) = 4.00$, $p < .05$); the difference among the PTSD Decelerators was nonsignificant ($t(10) = -.88$, $p = .40$). The levels of cortisol after neutral ($t(15) = .60$, $p = .56$) and trauma ($t(15) = -.49$, $p = .63$) recalls were not significantly different between the PTSD Accelerators and PTSD Decelerators. Moreover, the participants who reported dissociation during the trauma recall did not show a significant difference in their cortisol levels across the two types of recall ($t(11) = -.06$, $p = .96$). However, those without dissociation had a significantly lower cortisol level at the end of the trauma recall than the neutral recall ($t(11) = 4.85$, $p < .01$). The levels of cortisol after neutral ($t(22) = -.74$, $p = .47$) and trauma ($t(22) = .29$, $p = .77$) recalls were not significantly different between those with and without dissociation.

Table 6.1 *Mean and Standard Deviations of Cortisol Levels after Neutral and Trauma Recalls by Group*

	Overall sample			
	N		*Mean (SD)*	
Neutral recall	24		8.22 (3.57)	
Trauma recall	24		7.49 (3.65)	
	PTSD Accelerators		**PTSD Decelerators**	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Neutral recall	6	9.22 (4.47)	11	8.10 (3.20)
Trauma recall	6	7.63 (3.81)	11	8.59 (3.83)
	With Rape history		**Without rape history**	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Neutral recall	11	8.37 (3.16)	12	7.93 (4.15)
Trauma recall	11	7.58 (3.18)	12	7.19 (4.25)
	Dissociation during recall		**Without dissociation during recall**	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Neutral recall	12	7.68 (3.49)	12	8.77 (3.72)
Trauma recall	12	7.71 (4.05)	12	7.27 (3.37)

Note. Dissociation during recall = participants who reported dissociation during the trauma recall; Without dissociation during recall = participants who did not report dissociation during the trauma recall.

Table 6.2 *Mean and Standard Deviations of sAA Levels after Neutral and Trauma Recalls by Group*

	Overall sample			
	N		*Mean (SD)*	
Neutral recall	25		54.25 (41.19)	
Trauma recall	25		61.17 (48.30)	
	PTSD Accelerators		**PTSD Decelerators**	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Neutral recall	6	30.25 (25.84)	12	49.39 (38.23)
Trauma recall	6	50.45 (62.64)	12	57.43 (44.35)
	With Rape history		**Without rape history**	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Neutral recall	11	50.49 (37.02)	13	58.37 (46.98)
Trauma recall	11	63.84 (53.12)	13	60.80 (47.52)
	Dissociation during recall		**Without dissociation during recall**	
	n	*Mean (SD)*	*n*	*Mean (SD)*
Neutral recall	13	44.98 (40.16)	12	64.29 (41.60)
Trauma recall	13	47.69 (40.20)	12	75.77 (53.67)

Note. Dissociation during recall = participants who reported dissociation during the trauma recall; Without dissociation during recall = participants who did not report dissociation during the trauma recall.

Partial correlations (with cortisol level after neutral recall controlled) showed that the associations between the number of types of life adversity, PTSD symptom severity, duration of psychotherapy, and level of cortisol after trauma recall were nonsignificant (largest r = -.19, p = .40). However, state dissociation was significantly correlated with the level of cortisol after trauma recall (r = .44, p < .05), despite that significant partial correlations between cortisol level after the trauma recall, peri-traumatic dissociation (r = .07, p = .76), and trait dissociation (r = .27, p = .21) were not found. Moreover, when similar partial correlations were applied to examine the relationships between sAA level after trauma recall and the above measures, no significant correlation was shown (largest r = -.32, p = .14).

The exploratory Pearson's correlations showed that the duration of psychotherapy, numbers of type of adversity, trait dissociation, peri-traumatic dissociation, and PTSD symptom severity were not significantly associated with cortisol or sAA levels after the neutral recall (largest r = -.27, p = .22 for cortisol; largest r = .18, p = .38 for sAA), trauma recall (largest r = -.32, p = .13 for cortisol; largest r = -.16, p = .46 for sAA), or change levels (largest r = .27, p = .20 for cortisol; largest r = -.35, p = .09 for sAA). Similarly, state dissociation was not significantly correlated with cortisol (largest r = .22, p = .30) or sAA level (largest r = -.19, p = .37) at the corresponding condition (e.g., change level of state dissociation and change level of cortisol).

Pearson's correlation coefficients between the physiological measures (i.e., HR, HRV, cortisol and sAA levels) at the corresponding conditions are summarised in Table 6.3. As shown, in response to the trauma recall, a higher cortisol level was significantly correlated with lower high frequency HRV (HF-HRV), higher low frequency HRV (LF-HRV), and higher low frequency/high frequency ratio (LFHF-ratio).

Table 6.3 *Pearson's Correlation Coefficients between Physiological Measures*

		Cortisol level			*sAA level*		
		Neutral recall	Trauma recall	Change level	Neutral recall	Trauma recall	Change level
sAA level	Neutral recall	-.03	.	.	.	.	.
	Trauma recall	.	-.06	.	.	.	.
	Change level	.	.	.23	.	.	.
Hear rate level	Neutral recall	.53	.	.	-.05	.	.
	Trauma recall	.	.42	.	.	.07	.
	Change level	.	.	.17	.	.	-.11
HF-HRV level	Neutral recall	-.36	.	.	.31	.	.
	Trauma recall	.	-.61**	.	.	.30	.
	Change level	.	.	.19	.	.	.02
LF-HRV level	Neutral recall	.35	.	.	-.45	.	.
	Trauma recall	.	.61**	.	.	-.30	.
	Change level	.	.	-.16	.	.	-.04
LFHF-ratio	Neutral recall	.41	.	.	-.29	.	.
	Trauma recall	.	.72**	.	.	-.11	.
	Change level	.	.	-.34	.	.	-.33

Note. HF-HRV level = high frequency heart rate variability level; LF-HRV = low frequency heart rate variability level; LFHF-ratio = low frequency/high frequency ratio.
**$p < .01$.

6.4 Discussion

6.4.1 Rape history, dissociation, and cortisol response to trauma recall

The current study examined the level of cortisol and sAA variations after voluntary recall of trauma among PTSD patients. Overall, a significant change in cortisol and sAA levels was not found in response to the trauma recall, in comparison to the neutral recall. Regarding the individual differences associated with specific previous trauma, inconsistent with the previous study (Gola et al., 2012), a significant difference in the patterns of reactive cortisol level was not found between individuals with and without a rape history. The inconsistent results may be related to diversities in experimental design between the two studies: First, while the previous study (Gola et al., 2012) compared cortisol levels before and after a trauma related interview, we used the recollection of a neutral memory as the baseline measure. It is likely that the act of recollection itself has caused cortisol variation, regardless the emotional valence of the recalled contents. Second, the memory retrieval was induced with an interview in the previous study (Gola et al., 2012), whereas a less directive approach was adopted in the current study. It should be examined whether different memory retrieval mechanisms and psychological states are involved and triggered by the two methods. Finally, while the previous study (Gola et al., 2012) focused on a population with war and torture-related PTSD, we did not restrict the traumatic background related to the PTSD symptoms of our sample. Such diversity in our studied population may have created greater variance.

Although the above-mentioned inconsistent results have been found, by directly examining dissociation our data supported Gola and colleagues' argument (2012) that passive defence reactions, such as dissociation, may be the underlying mechanism in the relationship between a rape history and heightened glucocorticoid reactions to traumatic stimuli. Specifically, we found that, regardless of the baseline cortisol level after the neutral recall,

individuals reporting higher levels of state dissociation after the trauma recall showed smaller drops in cortisol level after the trauma recall, compared to those who reported lower state dissociation. Additionally, given a nonsignificant difference in cortisol level at baseline (i.e., after the neutral recall) between those who experienced dissociation during the trauma recall and those who did not, the former did not show a significant cortisol decrease in response to the trauma recall, whereas the latter did. Confirming this, a partial correlation between the change level of cortisol and whether one has experienced dissociation, with cortisol level after the neutral recall under control, showed a marginally significant association between the experience of dissociation and a smaller decrease of cortisol after the trauma recall ($r = .41$, $p = .05$). Overall, our findings demonstrated a smaller reduction in the activity of the HPA axis among the individuals who were more dissociated.

The discrepancies between the findings related to the examinations of rape and dissociation-related factors suggested a more powerful effect of dissociation on reactive cortisol level than the effect related to the type of traumatic background. Moreover, as trait dissociation and peri-traumatic dissociation were also investigated in our study, the negative findings of these measures suggest that: it is dissociation that happens close to the timing of the targeted traumatic event that has a significant effect on the cortisol secretion related to the event. In contrast, dissociation that happened in a previous trauma, or is a general personality trait, do not have significant effects.

Overall, our results consistently showed a more limited reduction in cortisol level among those who were more dissociative during the trauma recall. It is of research interest to further examine the treatment outcome (i.e., PTSD symptom reduction) of exposure-based therapies among these more dissociative individuals, and how such outcome associates with cortisol level. Specifically, we have found a significant association between lower cortisol

level and the development of more vivid intrusive memories (see Chapter Four). Previous literature has also suggested a subtype of PTSD patients who suffer from less intrusive, but more dissociative symptoms (e.g., Lanius et al., 2010). Future studies should investigate: 1) whether those who are more dissociated in exposure therapy tend to have less intrusive symptoms, and 2) how does this phenomenon relate to their long-term therapeutic outcome. Moreover, given that the current study only included patients with moderate level of dissociation, future studies are needed to clarify: 1) whether individuals with severer dissociation symptoms have different profiles of reactive cortisol in response to the recollection of trauma, and, similarly, 2) how is their cortisol reaction associated with the treatment outcome of exposure-based therapies.

6.4.2 CDR and cortisol response to trauma recall

The current study examined the effect of CDR on reactive cortisol and sAA levels. While a significant effect of CDR on sAA level was not found, compared to PTSD Decelerators, PTSD Accelerators were found to have greater decreases in cortisol levels in response to the trauma recall. Regarding the potential contribution of the baseline cortisol level, our result did not show a significant difference in cortisol level at the neutral recall between the two groups. Similarly, when a partial correlation between CDR and the change level of cortisol was performed, after the cortisol level at the neutral recall was controlled in the analysis, the correlation between CDR and the cortisol variation was still significant (r = .54, $p < .05$). These findings consistently suggested a greater reduction in cortisol level among the PTSD Accelerators, compared to PTSD Decelerators, during the trauma recall, and such result was not related to the difference in the baseline cortisol level between the two groups.

Recollection of trauma in exposure-based therapies activates traumatic memory for further memory processing, including re-encoding and re-consolidation. Based on the previous hypothesis (Yehuda & Harvey, 1997) and our findings (see Chapter Four) regarding the contribution of insufficient cortisol release to the over-consolidation of traumatic memory and its greater vividness, the current data highlighted potential vulnerability of the PTSD Accelerators. Specifically, this subgroup of PTSD patients may develop more vivid intrusive memories through exposure-based therapy. However, since the association between low cortisol secretion and vivid traumatic memory has only been shown among healthy participants encountering an analogue trauma, empirical data should be collected among PTSD patients in real-life therapy settings in order to examine this hypothesis.

6.4.3 Correlations between HRV and cortisol

As part of an exploratory investigation, we examined the relationships between different physiological activities (i.e., HR, HRV, cortisol and sAA levels) during the voluntary retrieval of traumatic memory. Overall, our data showed a consistent pattern of arousal across the two physiological systems that we examined by finding associations between a more activated HPA axis, indexed by a higher cortisol level, and a more greatly aroused cardiovascular system, which was indexed by higher LF-HRV, LFHF-ratio, and lower HF-HRV.

Overall, the current study provided a general profile of the associations between different physiological activities when PTSD patients voluntarily recall traumatic memories. However, due to a small sample size and hence limited statistical power, the findings discussed in this and the previous sections should be generalised with caution. This is especially true for the negative results. In order to validate the interesting findings in the current study, future replications with bigger sample sizes are required.

Chapter 7: General discussion

This thesis examined the reactions of a few indices of the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis in the memory encoding and retrieval phases of trauma. Potential individual differences associated with psychological traits, pre-existing trauma history, as well as two cardiovascular features related to stress defence - startle heart rate response (sHR) and cardiac defence response (CDR) - were taken into account. The aims of these investigations were to explore whether these physiological measures predicted the development of PTSD-like memory symptoms and what moderated these effects, as well as likely responses to exposure-based therapy procedures.

The important findings will be summarised in 7.1, followed by separate discussions on the results related to the ANS and HPA axis in 7.2 and 7.3, respectively. The discussions in both sections intend to summarise the contributions of the current thesis through answering the following questions: First, how do people respond to trauma psychologically and physiologically? Second, how do activities of the ANS and HPA axis inform us about the psychological states involved in the process of traumatic memory? Third, how do these physiological activities relate to the development of PTSD memory symptoms? Fourth, how do they predict responses to exposure-based therapies? Significant roles of individual differences will be highlighted in each part of discussion. Finally, the overall achievement and limitations of this thesis will be summarised in 7.4, with suggestions of future research directions.

7.1 Summary of findings

7.1.1 Individual differences in cardiovascular stress defence features

7.1.1.1 Individual differences in startle heart rate

The examination of sHR among our healthy participants found three subgroups with different responses to a startle trigger: the High Startle Group (HSG), Medium Startle Group (MSG), and Low Startle Group (LSG), who showed exaggerated, moderate, and suppressed sHR, respectively, with the LSG having higher trait dissociation than the rest. Among the PTSD patient sample, two subgroups, the High Startle PTSD Group (HSPG) and Medium Startle PTSD Group (MSPG), with sHR similar to the HSG and MSG, respectively, were found. The HSPG experienced more types of life adversity, and greater peri-traumatic dissociation during the most distressing trauma they encountered. In contrast to the healthy sample, a subgroup with suppressed sHR was not found in the patient sample. This has been related to the fact that patients who were considered to be highly dissociative have not been included in our studies, in order to ensure their capacity to complete the experimental procedures.

To further compare the sHR patterns between the healthy and PTSD samples, t-tests were conducted. First of all, baseline HR (i.e., mean HR during the 15 seconds before the onset of startle probe) did not significantly differ between the two samples (M = 78.69, SD = 10.07 for the healthy sample; M = 75.44, SD = 8.57 for the patient group; $t(82) = 1.30$, p = .20). Moreover, the amount of overall HR increase (i.e., the area under curve) during the first 10 seconds after the startle probe did not vary significantly between HSG in the healthy sample (M = 120.84, SD = 52.38), and HSPG in the patient sample (M = 127.55, SD = 41.66; $t(20) = -.31$, p = .76), or between MSG (M = 10.31, SD = 28.96) in the healthy sample, and MSPG in the patient sample (M = -3.95, SD = 62.59; $t(41) = 1.03$, p = .31). These results

suggest a congruency in sHR among individuals who were categorised into the same sHR groups, regardless of PTSD diagnosis.

7.1.1.2 Individual differences in cardiac defence response

In addition to sHR, individual differences in CDR patterns were assessed. Both the healthy and PTSD samples yielded two subgroups: Accelerators vs. Decelerators for the former sample, the PTSD Accelerators vs. PTSD Decelerators for the latter. However, a few differences in the patterns of the two CDR components (i.e., the first and second peaks of HR) were visible between the corresponding groups in the healthy and patient samples. First, the amount of overall HR increase in the first peak of CDR was significantly higher among the PTSD Accelerators (M = 104.46, SD = 58.34), compared to the healthy Accelerators (M = 31.50, SD = 80.96; $t(34) = -2.09$, $p < .05$). Second, as shown in Fig. 3 in 5.3.4 and Fig. 5 in 3.3.5, among the PTSD Accelerators, the second component of CDR appeared before HR returned to baseline after the first peak. Moreover it starts earlier (i.e., between the 13th and 33rd seconds after the startle probe) than it does among the healthy Accelerators (i.e., between the 22nd and 45th seconds after the startle probe).

In terms of the comparison between the PTSD and healthy Decelerators, the amount of overall HR increase during the first element of CDR did not differ significantly between the two samples (M = -5.33, SD = 66.16 for the healthy sample; M = 24.73, SD = 85.27 for the patient group; $t(46) = -1.31$, $p = .20$). However, while HR kept dropping after the first peak (i.e., the 10th second after the startle probe) among the healthy Decelerators (see Fig. 5 in 3.3.5), another minor HR increase appeared among the PTSD Decelerators before the drops occurred (see Fig. 3 in 5.3.4).

Overall, while the CDR patterns of the healthy sample mimicked those found in the literatures involving non-clinical samples, an earlier and stronger first peak of HR was found among the PTSD Accelerators, and a weak and shorter secondary peak of HR was found among the PTSD Decelerators. These discrepancies may suggest a more hyper-vigilant

cardiac defensive pattern among PTSD patients, in comparison to healthy individuals. Further replications regarding the differences in CDR between healthy and PTSD populations, as well as their psychological associates are needed.

7.1.2 Psychological and physiological responses to traumatic stimuli

As summarised in Table 7.1, increases in state dissociation, state anxiety, fear, and decreases in calm have been shown among our healthy participants during an analogue traumatic event. Among the PTSD patients, greater levels of state dissociation, and fearful and threatened feelings, were generally found during the voluntary memory retrieval of trauma, in comparison to the voluntary memory retrieval of a neutral event. Moreover, those who had been in psychotherapy for a longer period of time reported stronger increases in fear. Those with more life adversities had more increases in state dissociation. Furthermore, the PTSD Accelerators showed less increases in all negative emotions during the trauma recall, which suggests a restricted level of emotional arousal.

In terms of the reactions of the ANS, during the memory encoding phase of trauma, a dominant vagal activity was consistently indicated by the cardiovascular indices (i.e., heightened HF-HRV, lowered LF-HRV, LF/HF ratio, and HR) and decreased sAA level among our healthy sample. Similarly, when PTSD patients were asked to voluntarily retrieve a piece of traumatic memory, a stronger vagal modulation (compared to the recall of a neutral memory), and an associated gradual decrease in HR were found. However, significant individual differences in the level of cardiovascular variation were not shown in relation to any trait, or traumatic history related factors.

As for the reactions of the HPA axis, during the memory encoding phase of trauma, healthy participants showed an activation of the HPA axis indexed by increased cortisol levels. Interestingly, those who had a more recent traumatic experience and were more severely affected by a previous trauma were prone to less cortisol secretion when encountering the new traumatic stimulus in our study. Moreover, during a voluntary memory retrieval of trauma, a significant increase in cortisol level was not found among our PTSD

patient sample as a whole. However, individual differences associated with the CDR were found. PTSD Accelerators showed a greater difference in cortisol level between the two types of recall (i.e., trauma vs. neutral), which may suggest a more extreme defence mechanism.

Table 7.1 *Summary of Main Findings: Psychological and Physiological Reponses to Traumatic Stimuli*

	Overall sample	Pre-existing individual differences		
		Traumatic history and dissociation	**sHR[a]**	**CDR[b]**
Memory encoding phase among healthy individuals				
Psy.	❖Increased state dissociation, state anxiety, and fear	❖More life adversity predicted greater increase in state dissociation		
ANS	❖Increased vagal activation, and decreased SNS activation			
HPA axis	❖Increased cortisol level	❖Recent and high impact trauma predicted lower cortisol increase		
Memory retrieval phase among PTSD patients				
Psy.	❖Increased state dissociation, fear, and threatened feelings			❖PTSD Accelerators: Less increase in negative moods
ANS	❖Increased vagal activation			
HPA axis	❖Significant variation not found			❖PTSD Accelerators: Greater reduction in cortisol level

Note. Psy. = psychological response; ANS = autonomic nervous system; HPA axis = Hypothalamic-pituitary-adrenal Axis; sHR = startle heart rate; CDR = cardiac defence response; SNS = sympathetic nervous system

a. Three groups were found in the healthy sample: Low Start Group, Medium Startle Group, and High Startle Group. Two groups were found in the PTSD patient sample: Medium Startle PTSD Group, and High Startle PTSD Group.

b. Two groups were found in the healthy sample: Accelerators and Decelerators. Two groups were found in the PTSD patient sample: PTSD Accelerators and PTSD Decelerators.

7.1.3 How do the ANS and HPA axis indicate psychological states?

During the memory encoding phase of trauma, significant individual differences were found in the relationships between HR and different psychological states. As summarised in Table 7.2, among the individuals with suppressed sHR (i.e., the LSG), lower HR observed at this phase suggested greater fear and state dissociation. However, among those with medium sHR (i.e., the MSG), lower HR was indicative of a less anxious and fearful state.

In the memory retrieval phase of trauma, a greater overall decrease in HR among PTSD patients was found to be a sign of a more fearful and threatened state. However, when inspecting HR fluctuation moment-to-moment, increases in HR during the trauma recall were associated with the occurrence of flashbacks. Interestingly, among those who had received psychotherapy for a longer period of time, flashbacks were not accompanied by HR levels as high as in those who had not received or had received shorter periods of psychotherapy. This finding suggested a physiologically calming effect of psychotherapy. Additionally, examinations of HRV showed that a higher activation of the vagal system, a greater suppression of sympathetic activation, and a stronger dominance of the former are indices of an overall more dissociative state during the recollection of trauma.

The level of cortisol did not show significant relationships with the psychological states during memory encoding among the healthy participants. However, among the patient sample, less variation in the activity of the HPA axis between the recall of a trauma and a neutral event was found among those who were more dissociative during the trauma recall. This finding may suggest an association between dissociation and a less flexible HPA axis.

Table 7.2 *Summary of Main Findings: Psychological States, the ANS and HPA Axis Responses*

	Overall sample	Pre-existing individual differences		
		Traumatic history and dissociation	**sHR[a]**	**CDR[b]**
Memory encoding phase among healthy individuals				
ANS	❖Significant relationship not found		❖LSG: Low HR indicated great fear and state dissociation ❖MSG: Low HR indicated low anxiety and fear ❖HSG: Significant relationship not found	
HPA axis	❖Significant relationship not found			
Memory retrieval phase among PTSD patients				
ANS	❖Overall pattern: - Greater HR reduction suggested greater fearful and threatened feelings - Stronger vagal dominance indicated greater state dissociation ❖Periodic pattern: - HR increases suggested flashback			
HPA axis	❖Smaller variation in cortisol level found among patients with greater dissociation during trauma memory retrieval			

Note. Psy. = psychological response; ANS = autonomic nervous system; HPA axis = Hypothalamic-pituitary-adrenal Axis; sHR = startle heart rate; CDR = cardiac defence response; LSG = Low Startle Group; MSG = Medium Startle Group; HSG = High Startle Group; HR = heart rate

a. Three groups were found in the healthy sample: Low Start Group, Medium Startle Group, and High Startle Group. Two groups were found in the PTSD patient sample: Medium Startle PTSD Group, and High Startle PTSD Group.

b. Two groups were found in the healthy sample: Accelerators and Decelerators. Two groups were found in the PTSD patient sample: PTSD Accelerators and PTSD Decelerators.

7.1.4 How do the ANS and HPA axis predict the development of intrusive memories?

Adopting the trauma film paradigm, we found that: a greater decrease in HR during the memory encoding phase of certain traumatic episodes that later intruded was predictive of more frequent intrusions and better recognition of the detailed information related to the trauma (refer to Table 7.3). While a significant association between HR and the vividness of intrusion was not shown in the overall sample, individual differences were found after taking account of the group differences in sHR. Among the LSG, as a greater suppression in peri-traumatic HR was indicative of greater state dissociation, it was also predictive of less vividness of intrusive images. However, this correlation was not significant among the HSG and MSG.

In terms of the relationship between cortisol and intrusion, the association between lower cortisol secretion post-trauma, and greater vividness of intrusion has been found in our overall sample. Individuals in the LSG were found to show an even stronger association of this kind. Moreover, they were found to have more vivid intrusive memories compared to the other subgroups. In terms of the relationship between cortisol level and the frequency of intrusion, significant associations were not found until the CDR was taken into consideration. While an overall negative association was shown between peri-traumatic cortisol level and the frequency of intrusion, among the Accelerators, a higher cortisol level was predictive of the development of more frequent intrusions. However, immediately post-trauma, this moderating effect of CDR was replaced by sAA level. Our data showed that when cortisol and sAA responses are both activated, more frequent intrusions developed. This finding supported previous studies which suggested that an enhancing effect of cortisol on memory only occurs when SNS arousal is present (e.g., Bryant, McGrath, & Felmingham, 2013; Cahill et al., 1994; McGaugh & Roozendaal, 2009; Roozendaal et al., 2006).

Table 7.3 *Summary of Main Findings: Intrusive Memory, Peri-traumatic ANS and HPA Axis Responses*

		Pre-existing individual difference		
	Overall sample	**Traumatic history and dissociation**	**sHR[a]**	**CDR[b]**
Memory encoding phase among healthy individuals				
ANS	❖Greater HR decrease during the intrusive sequence of trauma film predicted more frequent intrusion, and better detailed recognition memory ❖Significant relationship with the vividness of intrusion not found		❖LSG: Low HR predicted less vivid intrusion ❖MSG & HSG: Significant relationship not found	
HPA axis	❖Significant relationship with the frequency of intrusion not found ❖Low cortisol level predicted greater vividness of intrusion		❖LSG: - Stronger correlation between low cortisol and high vividness of intrusion - More vivid intrusion	❖Accelerators: Higher cortisol level predicted more frequent intrusion

Note. Psy. = psychological response; ANS = autonomic nervous system; HPA axis = Hypothalamic-pituitary-adrenal Axis; sHR = startle heart rate; CDR = cardiac defence response; LSG = Low Startle Group; MSG = Medium Startle Group; HSG = High Startle Group; HR = heart rate

a. Three groups were found in the healthy sample: Low Start Group, Medium Startle Group, and High Startle Group.

b. Two groups were found in the healthy sample: Accelerators and Decelerators.

7.2 What does the heart say?

7.2.1 A calm heart under traumatic stress

In the previous PTSD literature, fight/flight responses and the associated heightened activation of the sympathetic nervous system (SNS) and increased HR, have been related to the pathology of PTSD (e.g., Blechert et al., 2007; Bryant et al., 2008; Bryant et al., 2007; Cohen et al., 1997; De Young, Kenardy, & Spence, 2007; Kraemer et al., 2008; Zatzick et al., 2005). Moreover, heightened HR has been commonly found as a reaction to reminders of traumatic events (e.g., Ehlers et al., 2010; Hetzel-Riggin, 2010). These existing studies described HR fluctuations in the aftermath of traumas, and when individuals involuntarily encountered reminders of trauma. In contrast, the current thesis adopted different study designs, and separately assessed the SNS and vagal system to examine their contributions to cardiovascular reactions during a traumatic event, as well as a therapy-like voluntary trauma memory retrieval procedure.

Adding to the existing literature, our findings of suppressed HR during exposure to trauma and traumatic memory retrieval demonstrated situations with vagal dominant, and hence calming, cardiovascular reactions to traumatic stimuli. Such alternative findings echo the diversity of stress coping mechanisms in response to circumstances involving various levels of autonomy (van der Kolk et al., 1996) and different stages of defence response (Bradley & Lang, 2000). Specifically, the findings suggest the involvement of more inhibitory and calming effects of the heart, in dealing with traumatic situations, when escaping is not appropriate, and when memory recollection is voluntarily initiated.

An interesting contrast is demonstrated through the comparison between our findings and the existing literature, particularly the comparison between the cardiovascular responses associated with different coping situations and at different memory processing phases. During

the encoding phase, a higher level of autonomic defence (e.g., fight or flight) is associated with a highly activated and dominant SNS, as well as a quieter vagal effect (e.g., Blechert et al., 2007; Bryant et al., 2008; Bryant et al., 2007; Cohen et al., 1997; De Young, Kenardy, & Spence, 2007; Kraemer et al., 2008; Zatzick et al., 2005). However, at the memory retrieval phase, the involvement of greater autonomy (e.g., voluntarily recalling a trauma instead of being involuntarily reminded) is associated with a stronger control of the vagal system and a more suppressed SNS, according to our results. Employing an evolutionary perspective, such contrasts in the biological coping mechanisms of threat may be associated with differences in the survival strategies involved in the two stages of trauma: active defending at the peri-traumatic stage vs. impact buffering and managing at the recollection or aftermath stage.

However, given the fact that 1) the suggestions related to the memory retrieval phase of trauma are based on our data among PTSD patients, and 2) the level of HR decrease at this stage was correlated with greater state dissociation, generalisation of the findings should be cautious. Specifically, it is unknown whether the dominant vagal effect that we found during the voluntary retrieval was an extension of an ordinary threat defence mechanism, or a phenomenon among PTSD patients, and especially those with a greater dissociation tendency. Similar paradigms should be applied to healthy populations in order to provide a more complete picture regarding this issue.

7.2.2 Is a calm heart peri-trauma a resilience or risk factor for the development of PTSD?

HR has been applied in studies adopting the trauma film paradigm, with its greater suppression peri-trauma suggested to be a risk factor for the development of greater memory symptoms of PTSD (e.g., Holmes, Brewin, & Hennessy, 2004). While more suppressed HR was implied to be an indicator of dissociation (Holmes, Brewin, & Hennessy, 2004), direct investigations in the current thesis suggest that this hypothesis only applies to individuals who are more prone to dissociation in response to stress (i.e., the LSG). Specifically, our studies have found three subgroups among healthy individuals. Each of the subgroups has a unique pattern of startle HR responses. When individuals were experiencing trauma, low HR was related to greater fear and dissociation only among the LSG, who have higher baseline HR to begin with, respond to sudden threat with immediate HR suppression, and tend to dissociate in general. In contrast, among those with moderate baseline HR and moderate sHR (i.e., the MSG), low HR during trauma should be read as a positive sign, which suggests a less anxious and less fearful state. These findings highlight the significance of taking account individual difference while applying HR as an indicator of certain psychological states.

Following the above, individual differences were found in the relationships between peri-traumatic HR and the vividness of intrusion. Only among the LSG, lower peri-traumatic HR was predictive of the development of less vivid intrusive memories. This and the above-mentioned findings of individual differences may have contributed to the inconsistent results in previous studies, which examined HR in the early aftermath of accidents and its correlation with the development of PTSD (Blanchard et al., 2002; Buckley et al., 2004; O'Donnell et al., 2007; Ostrowski, Christopher, & Delahanty, 2007). Moreover, multiple pathological mechanisms and threat-related physiological reactivity have been related to the development

of different subtypes of PTSD (Ginzburg et al., 2006; McTeague & Lang, 2012; van der Kolk et al., 1996). For example, PTSD patients with multiple exposures to trauma tend to present differently, such as with more dissociative symptoms and with more chronic pathology, compared to patients whose PTSD are related to a single and discrete event (McTeague & Lang, 2012; van der Kolk et al., 1996). Similarly, a subgroup of PTSD patients, whose pathology was related to prolonged traumatic exposure, has been found to have blunted startle reactivity (McTeague & Lang, 2012). Supporting this literature, the LSG found in our healthy sample and its unique symptom development mechanism provide evidence for the existence of a subgroup of PTSD patients, who tend to cope with passive defence strategies, and suffered from fewer intrusive, but more dissociative symptoms (Ginzburg et al., 2006).

On the other hand, a subtype of PTSD, which is prone to psychological and physiological hyper-reactivity, and greater suffering from intrusive and hyper-vigilant symptoms, has been proposed in the literature (McTeague & Lang, 2012). Although, we found a subgroup (i.e., the HSG) with an exaggerated sHR pattern, a significant association between peri-traumatic HR and the intrusive memory measures was not shown among these individuals. This nonsignificant finding may be related to the nature of the trauma film paradigm, which creates a traumatic situation when active defence was less appropriate. Based on this, traumatic stimuli and study paradigms that trigger more active coping strategies are needed, in order to more completely investigate the role of sHR in moderating the relationship between peri-traumatic HR and PTSD development.

7.2.3 How to read the heart in exposure therapy?

As restricted HR increases during exposure therapy have been related to limited therapeutic progress (Halligan et al., 2006), the psychological implications of cardiovascular indices are therefore of research interest. This thesis examined HR as an indicator of different psychological states that PTSD patients commonly experience during exposure-based therapies. A greater increase in HR was found to indicate an overall greater decrease in calmness, and hence an increase in emotional arousal. Moreover, an analysis of HR in relation to different psychological states during trauma recall showed an association between HR increases and the occurrence of flashbacks during the automatic retrieval of traumatic memories. Overall, the findings suggested a positive correlation between cardiovascular and emotional arousal, which in turn supported the previous study (Halligan et al., 2006), and the theory associating the therapeutic effects of exposure therapy with sufficient emotional arousal (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989).

On the other hand, it was found that a smaller increase in HR during memory retrieval may also suggest a greater feeling of fear and threat. Consistent with this, the investigations of HRV also showed that heightened vagal activities, a suppressed SNS, and a stronger dominance of the former were all signs of a more dissociative state. To sum up, in addition to the above-mentioned responses depicting highly aroused physiological and psychological states, our data supported previous literatures that suggested an association between an activated vagal system and passive defence mechanisms (Bradley & Lang, 2007; Richter, Schumann, & Zwiener, 1990).

Overall, an interesting distinction between HR and HRV as physiological indices of psychological states has been demonstrated in our data. The former is more informative of emotional arousal, whereas the latter is indicative of the involvement of consciousness and

higher cognitive functions. Both emotional arousal (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989) and the involvement of higher cognitive functions (Brewin et al., 2010) have been suggested to be crucial elements in exposure-based therapy for the recovery of posttraumatic memory symptoms. The current findings suggest the potential of utilising these measures as objective indices of the above-mentioned psychological states, which may in turn predict and enhance the treatment outcome of exposure-based therapy.

It was noteworthy that while individual differences were evident in our healthy sample, in terms of the associations between peri-traumatic cardiovascular response and various psychological outcomes, more consistent correlations were shown during the traumatic memory retrieval stage among the PTSD patients. Such a contrast may be related to a relatively more homogeneous PTSD patient population. Alternatively, these contrasting results may be a reflection of the differences between the two phases of memory processing.

7.3 Cortisol speaks two languages

7.3.1 Language of hormonal coordination

While increases in cortisol level were found among a healthy sample in response to the exposure of an analogue trauma in our study, those who had experienced a traumatic event more recently and still suffered greater negative impacts from it released a smaller amount of cortisol after the trauma film. These findings supported the previous literature that suggested a suppressive effect of previous trauma on cortisol secretion in response to a later trauma (Resnick et al., 1995), and that such effect is only visible in the presence of current psychopathology (Cohen, Zohar, & Matar, 2003; Otte et al., 2005).

Moreover, our data suggested that the lack of responsiveness of the HPA axis among those more recently and severely traumatised individuals was not related to its over-excitation at baseline. We found that, first, the difference in basal cortisol level was nonsignificant between those who have and have not experienced a trauma. Second, basal cortisol level was not significantly associated with the strength of impact from a previous trauma, or the elapsed time since a previous trauma. Third, the above-mentioned significant correlations between suppressed reactive cortisol level and trauma were established after basal cortisol level was controlled.

In terms of the impact of cortisol on PTSD pathology, we supported the previous hypothesis, which suggested that the insufficient cortisol secretion is a cause of the memory symptoms of PTSD (Yehuda & Harvey, 1997), with our finding that individuals who had lower post-traumatic cortisol levels reported more vivid intrusive memories. This finding suggested the potential of utilising the level of cortisol immediately after a trauma as a risk factor to screen and target the population with high risk of developing PTSD. However, replications of the current findings with real-life trauma and longer follow-up assessment of

intrusive memory symptoms should be performed before more solid conclusions can be drawn. This will be discussed further in 7.4.

Regarding the risk factors for PTSD, in line with the literature that suggested the association between previous traumatic experience and a later development of PTSD related to a new trauma (see the meta-analysis by Brewin, Andrews, & Valentine, 2000), our data showed that those individuals with more recent traumatic experiences, and those who sustained more severe impacts from a previous trauma tended to release less cortisol in response to the trauma film. Additionally, given the findings that: 1) the negative association between post-traumatic cortisol level and the vividness of intrusive memory was stronger among the LSG, and 2) the LSG had more vivid intrusive memory overall, it is of research interest to further examine the LSG as a vulnerable population for PTSD development. However, as explained with more details in 7.4, it is unknown whether the greater vividness shown in our study was a part of healthy recovery, or an initial sign of pathology. In order to respond to this question and to evaluate whether the effects of cortisol were clinically substantial, intrusive memory and overall PTSD symptom should be examined with studies involving real-life traumas, and following the symptoms for at least a month.

In terms of our findings on the memory retrieval phase of trauma, consistent with the suggested association between insufficient cortisol reaction and PTSD pathology (Yehuda & Harvey, 1997), our PTSD patient sample showed a lack of significant cortisol increase after recalling a traumatic memory. The voluntary recall procedure in exposure-based therapies has been used as a trigger to activate traumatic memory, in order to further process with the involvement of higher-level cognitive functions (e.g., Brewin et al., 2010), or to restructure trauma-related memory schema (e.g., Smucker, Dancu, Foa, & Niederee, 1995). In other words, voluntary retrieval of traumatic memory provides a platform for secondary memory

encoding and consolidation to occur in therapeutic ways. Consider the general suppressive cortisol response found among our PTSD sample during this voluntary memory retrieval procedure, and the above-mentioned results regarding the negative correlation between cortisol level during memory encoding and the vividness of intrusive memories. It is likely that the vividness of PTSD patients' traumatic memories were increased through therapy. To examine this hypothesis, investigations of the relationships between cortisol responses and memory outcomes associated with exposure-based therapies should be conducted (see 7.4 for details). Moreover, echoing our previous question, longitudinal studies should be performed, in order to identify the role of enhancing vividness of traumatic memory in the progress of recovery.

7.3.2 Language of physiological arousal

Agreeing with the literature (Bowirrat et al., 2010; Het, Ramlow, & Wolf, 2005; Takai et al., 2004), our data showed cortisol's role in indicating experiences of distress as a form of physiological arousal. Among our healthy sample, we found that cortisol level increased as a result of the exposure to a traumatic stressor. Although such fluctuation has been recognised as a healthy response to stress, among individuals who are more hyper vigilant in general (i.e., the CDR Accelerators), and those who were more sympathetically activated immediately after encountering a traumatic stressor (i.e., the sAA responders), a heightened cortisol level has been found to predict more frequent intrusive memories later on.

These findings suggest individual differences underlying the relationship between the experience of distress and the development of pathology. Given that significant differences in the levels of psychological distress were not found between Accelerators and Decelerators, nor between individuals with different levels of sympathetic activation, the above-mentioned findings may reflect a difference related to individual's sensitivity to the level of distress. In other words, it is not a high level of subjective negative impact that causes post-traumatic memory symptoms, but a greater sensitivity to the distress. In reference to the Dual Representation Theory (DRT; Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996), highly stressful situations and significant emotional arousal are often triggers of the development of more long lasting and salient sensation-based memories (S-memories) and their weaker associations with the contextual memories (C-memories). However, the important role of individual differences in the DRT (Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996), in terms of how different vulnerabilities moderate the relationships between stress and pathological consequences, have been highlighted with our findings.

To sum up, although the insufficient release of cortisol after a trauma has been found to be associated with vivid intrusive memories (see 7.3.1), the other extreme (i.e., heightened secretion of cortisol) should be carefully considered as a sign of risk among those who are more physiologically ready for fight/flight responses: Accelerators and sensitive sAA responders. Both extremes of cortisol reaction are worth further investigation, in order to evaluate their clinical application to PTSD risk assessment and prevention.

Additionally, our investigations of the reactions of the HPA axis during traumatic memory retrieval showed that while, overall, a significant difference was not found between cortisol levels after the neutral and trauma recalls, PTSD Accelerators and the patients who did not experience dissociation during the recall showed significantly decreased cortisol levels after the trauma recall. Similar to the descriptions in 7.3.1, the procedure of voluntary memory retrieval in exposure-based therapies is a phase for secondary memory encoding and consolidation. Given our previously summarised findings on the association between low level of cortisol during memory encoding and the development of highly vivid intrusions, it is likely that the vividness of traumatic memories of the PTSD Accelerators and of those do not tend to dissociate during this procedure may be especially strengthened through the therapy. Similar to the suggestions in 7.3.1, this hypothesis and its clinical implications require future studies to examine.

7.3.3 Carefully balance the lever

With the investigations of both characteristics of intrusive memory (i.e., vividness and frequency), our findings suggest two different kinds of effect of cortisol on intrusion. First, its insufficient secretion leads to over consolidation of traumatic memory. Second, its heightened release indicates a greater arousal brought about by the impact of trauma. To be more specific and to account for individual differences, while a low level of cortisol generally contributes to salient and vivid intrusions, for individuals who are more hyper-vigilant (i.e., Accelerators and sensitive sAA responders), a higher level of cortisol leads to more recurrent intrusions. These diversities and individual differences may be a source of inconsistency between previous studies, which examined cortisol level after motor vehicle accidents and its correlation with PTSD symptom development (Delahanty et al., 2005; Delahanty, Raimonde, & Spoonster, 2000; Kolaitis et al., 2011; McFarlane, Atchison, & Yehuda, 1997; Pervanidou et al., 2007).

Following the above, it is interesting to note that among those studies finding positive associations between posttraumatic cortisol level and PTSD development (Delahanty et al., 2005; Kolaitis et al., 2011; Pervanidou et al., 2007), the participants were mostly youngsters (i.e., aged between 7 and 18). As the PTSD symptom profile is different among children and adolescents, compared to adults (American Psychiatric Association, 1994), it is possible that the mechanism through which youngsters' PTSD develops is different from that of adults. It has been suggested that hyper-vigilance plays a more important role in children and adolescents' PTSD symptom profile (American Psychiatric Association, 1994). Given the congruence in the relationship between cortisol and PTSD between the youngster sample in the previous studies (Delahanty et al., 2005; Kolaitis et al., 2011; Pervanidou et al., 2007), and the Accelerators and sAA responders in our study, further investigations should be

performed to examine the similarity of these populations, in terms of their PTSD symptom profiles and developing mechanisms.

In the recent literature, there has been a trend to investigate the effects of cortisol injection on PTSD prevention and treatment (e.g., Bowirrat et al., 2010; Schelling et al., 2001; Schelling et al., 2004; Suris et al., 2010). Our finding of a negative association between posttraumatic cortisol level and vividness of intrusion supported such application in terms of preventing major memory symptoms. However, given our findings on the effect of cortisol on the frequency of intrusion, and the related individual differences, it is important to examine the effect of such cortisol application among individuals who are more hyper vigilant in future studies. It is likely that while artificially increasing cortisol level eliminates the vividness of intrusive memories in general, it may cause more frequent intrusions for certain populations.

On the other hand, as for the application of cortisol to treating PTSD, although a general lack of response of the HPA axis has been shown in our PTSD sample after a voluntary recollection of trauma, the reactivity level of the HPA axis was not significantly associated with the severity of PTSD. In other words, with our cross-sectional data, we did not find clear evidence suggesting a correlation between different severities of pre-existing PTSD pathology and the responsiveness of the HPA axis to the memory recall manipulation in exposure-based therapies. Longitudinal examinations should be performed to assess the relationship between reactive cortisol level during exposure-based therapy and PTSD symptom variations through-out the therapy.

7.4 Dissociation: A psychological and physiological phenomenon

7.4.1 The psychological profile of dissociation

It has been well established that dissociation is a common passive coping strategy to trauma and other forms of extreme stress (American Psychiatric Association, 2013; Bradley & Lang, 2000). In line with this, increased state dissociation was found in our healthy participants while they were exposed to the trauma film. Those with more pre-existing life adversities showed even greater increases in state dissociation during the film. Consistently, among our PTSD sample, state dissociation was significantly raised when a personal traumatic memory was voluntarily retrieved. Moreover, higher state dissociation during this procedure was significantly associated with greater number of pre-existing life adversities ($r = .42$, $p < .05$). These findings, as well as the other piece of data showing a positive correlation between the number of types of adversities and trait dissociation among our PTSD sample, support the existing literature (e.g., van der Kolk et al., 1996), which suggested an association between multiple life adversity and greater dissociative tendency.

As an alteration in mental state, which involves disruptions in the integration of consciousness (American Psychiatric Association, 2013), dissociation was suggested to be a means to escape from extreme threats by mentally detaching oneself from the external world, their own body, or their sense of self (review paper by Holmes et al., 2005). However, in contrast to previous literature which suggested an absence of apparent emotional experience, 'spaced out', or a dream-like feeling during this detached state (Griffin, Resick, & Mechanic, 1997; Sierra & Berrios, 1998), our healthy sample showed that higher state dissociation was significantly correlated with greater state anxiety ($r = .51$, $p < .001$), fear ($r = .49$, $p < .001$), and less calmness ($r = -.30$, $p < .01$) during the trauma film. Consistently, the PTSD sample also showed that greater state dissociation during trauma recall was significantly associated

with stronger fearful ($r = .71$, $p < .001$), threatened feelings ($r = .63$, $p < .001$), and less calmness ($r = -.39$, $p < .05$).

The differences between the current thesis and the previous literature (Griffin, Resick, & Mechanic, 1997; Sierra & Berrios, 1998), in terms of the type of emotional phenomena and the timing of assessment, may have contributed to the diverse findings. For example, Sierra and Berrios (1998) suggested a general lack of emotional feelings based on clinical observation. According to them, patients with strong depersonalisation symptoms tended to self report loss of interest and inability to enjoy - symptoms commonly found among individuals with depression (American Psychiatric Association, 2013). In contrast to their general and symptom-oriented assessment, the emotional feelings targeted in the current thesis were more acute and direct responses to trauma. Similarly, although an association between peri-traumatic dissociation and lower perceived threat was found in a retrospective study (Griffin, Resick, & Mechanic, 1997), the correlation between greater state dissociation and stronger negative emotions, including the feeling of threat, found in the current thesis may have demonstrated the perception of individuals of their psychological phenomena at a more acute phase of trauma. Future empirical studies are needed to further examine the relationships between dissociation and different types of emotions at different phases of the course of PTSD development and recovery.

In terms of the effect of dissociation on the development of intrusive memory, the data from our healthy sample showed a significant correlation between greater state dissociation during the trauma film and more frequent intrusive memories related to the film ($r = .30$, $p < .01$). However, a significant correlation between state dissociation and the vividness of intrusion was not found ($r = .01$, $p = .96$). Given that dissociation involves interruption in the integration of consciousness, the former finding supported the DRT

(Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996), which suggested an association between a lack of involvement of high-level cognitive function and the development of intrusion. This finding also echoes the established role of dissociation on predicting PTSD (meta-analysis by Ozer et al., 2003). Additionally, the inconsistent findings between the frequency and vividness of intrusion in the current thesis suggest diverse mechanisms underlying the two characteristics of intrusion. As suggested in 4.4.3, separate examinations on these two indices of intrusion should be performed in future studies.

7.4.2 The physiological profile of dissociation

An association between dissociation and vagal-dominance in the ANS during the exposure of traumatic stimuli has been consistently demonstrated in the current thesis. First, a significant correlation between stronger state dissociation and greater vagal dominance was found among PTSD patients while they voluntarily recalled their traumatic memories. Moreover, among our healthy participants, although a significant relationship between state dissociation and HR was not found in the overall sample, a subgroup with stronger trait dissociation (i.e., LSG) showed a significant correlation between greater state dissociation and lower HR during the trauma film.

For the first time, this thesis examined concurrent variations of state dissociation and the cardiovascular responses peri-trauma, as well as during a voluntary memory recall procedure similar to exposure-based therapies. Our findings supported a recent study (Sack, Cillien, & Hopper, 2012), which examined the cardiovascular activities during a script-driven trauma imagery, and found lower HR and a smaller decrease in vagal activation among PTSD patients who experienced greater state dissociation. Further replications of the current findings are needed. Moreover, we have found that momentary HR increases accompanied flashbacks during the voluntary retrieval of trauma. However, the number of individuals with valid ECG data and with dissociation experience during the voluntary retrieval was too few for statistical analysis in our study. Future studies with bigger sample sizes are required to investigate the interesting question: whether periodic HR may be applied to identify the dissociative periods during the voluntary retrieval of trauma.

Moreover, in the current thesis, those PTSD patients who were more dissociated during the voluntary recall of trauma were found to have a smaller reduction of cortisol level during the recall. In addition to the discussion in 6.4.1, it is interesting to note that, while the

ANS was calmer, according to the above-mentioned finding, the HPA-axis was less suppressed among the more dissociative PTSD patients. This relationship is in contrast to what we found in our PTSD sample as a whole, which suggested co-occurring arousal in both biological systems (i.e., associations between greater cortisol level, greater SNS, and weaker vagal activation). Few studies have examined the SNS and HPA-axis at the same time among PTSD patients. The current results are informative of the reactions of the two important physiological systems to the recollection of trauma. However, given the current small sample size, replications of the current findings with larger samples are emphasised.

7.5 Now and the future

7.5.1 Contributions to the existing literature

7.5.1.1 Highlighting the role of individual differences

The psychological mechanisms through which PTSD develops, and through which psychotherapy relieves symptoms have been of great research interest (e.g., Brewin et al., 2010; Ehlers & Clark, 2000; Foa & Rothbaum, 1998), not only due to the scientific value of these topics, but also because of their significant clinical relevance. While studies have been applying physiological measures as indices of the psychological factors relevant to the developmental and treatment issues of PTSD, the current thesis added to the existing literature by highlighting important sources of individual differences in interpreting these physiological indices, and hence contributing to more sophisticated models.

For example, suppressed peri-traumatic HR has been associated with dissociation (Holmes, Brewin, & Hennessy, 2004). It has therefore been considered as a sign of risk in adopting a more pathological pathway of traumatic memory encoding, which, according to the dual representation theory (DRT; Brewin et al., 2010), has less involvement of higher cognitive functions and creates more enduring sensation-based memories (S-memories), as well as weaker associations between S-memories and contextual memories (C-memories). However, adding a layer to the above-mentioned hypotheses, we found a subgroup (i.e., the LSG) with suppressed sHR and greater trait dissociation, and that only among this subgroup, a negative association between HR and dissociation was shown. In other words, our data suggested that there are multiple psychological implications of HR variations, depending on different pre-existing traits. Individual differences should be considered while applying peri-traumatic HR to predict relevant psychological outcomes.

Similarly, elevated levels of cortisol have been suggested as a sign of intense emotional arousal (Het, Ramlow, & Wolf, 2005). As an extreme emotional situation has been proposed as a trigger of maladaptive traumatic memory processing in the DRT (Brewin et al., 2010), high levels of cortisol may be predictive of the involvement of a more pathological pathway, as described above. However, our investigation of CDR and the finding that a positive correlation between peri-traumatic cortisol level and the frequency of intrusion was only significant among the Accelerators has again pointed out the significant role of individual differences. That is, only among individuals who are more ready to initiate extreme coping strategies, higher levels of cortisol during trauma exposure may indicate risk of engaging in a more pathological memory encoding process.

In sum, our findings emphasise the relevance of sHR and CDR as important cardiovascular defence response traits in the context of trauma research. Individual differences related to these measures should be taken into account in examining the relationship between different physiological indicators and memory processing pathways (Brewin et al., 2010).

7.5.1.2 Suggesting potential predictors of PTSD risk and treatment outcome

Predicting, preventing and treating PTSD with physiological measures have been significant concerns in the latest PTSD literature (e.g., Bowirrat et al., 2010). The current thesis adopted experimental paradigms to directly examine important associations between the physiological and psychological reactions to trauma and therapeutic procedures. Many of our findings have provided information to build our knowledge further around these issues.

In terms of the prediction of the development of PTSD, we have found, for example, that individuals with more recent traumatic experiences and greater impact from them tend to release less cortisol when they encounter a new trauma. Following this, lower cortisol secretion immediately after trauma tended to produce more vivid intrusive memories. A subgroup of individuals (i.e., the LSG) have shown an even stronger trend in this direction. These findings have pointed out important factors, such as recent trauma history, peri-trauma cortisol level, sHR, that may potentially be applied to predict the risk of PTSD development.

Additionally, in our investigations of the psychological and physiological reactions to a voluntary memory retrieval of trauma, we found that PTSD Accelerators had more restricted emotional arousal in response to this procedure, and individuals with more previous life adversities tended to become more dissociated. Given the crucial role of emotional involvement in exposure-based therapies (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989), these populations may represent those who benefit from therapy less.

Moreover, our data showed significant associations between HR, flashbacks, and the level of emotional arousal during memory retrieval. Associations between HRV and overall dissociation level during the memory retrieval were also found. These findings suggested the potential of adopting these cardiovascular indices to indicate the psychological and mental states occurring during exposure therapy. Such applications may not only predict the

treatment effect, but also eventually become a tool to identify and train emotional involvement during exposure therapy.

Overall, our findings have provided valuable information of clinical relevance. Follow up studies should be conducted to further investigate the potential of applying these physiological measures (e.g., cortisol level, HR, HRV) in facilitating clinical interventions on posttraumatic reactions.

7.5.2 What should be done to better examine traumatic memory encoding?

7.5.2.1 Limitations of our studies

There are a number of limitations in the studies involved in the current thesis. The main issues in our method to examine the memory encoding phase of trauma are the adaptation of an analogue stimulus, the trauma film, the inclusion of a non-clinical sample, and the reliance on a self-report diary to assess intrusive memories. The application of an analogue trauma and a non-clinical sample may relate to the non-significant findings of group differences in memory and psychological states. For example, in Chapter Three, the nonsignificant difference in intrusion frequency, fear and state dissociation between groups may be partly due to a floor effect.

However, the use of trauma film does fulfil diagnostic criteria A1 and A2 for PTSD in DSM-IV (APA, 1994) as the participants witnessed actual death and reported significantly distress after viewing it. Moreover, it has been well established that the nature, amplifiers and attenuators of intrusive memories for the trauma film are in line with those of the intrusions resulting from real traumas (see review by Holmes and Bourne, 2008). Because of these and the other advantages of trauma film paradigm, such as enabling the investigation of peri-traumatic phenomena and offering laboratory control, it has been recognized as a valid approach to study trauma and PTSD (Holmes & Bourne, 2008). Moreover, considering the fact that the average compliance rating for the intrusion diary in the current sample is satisfactory (9.3 on a 0-10 scale), this measure arguably has advantages over retrospective reports that average over longer periods such as a week.

Nevertheless, given the nature of the applied paradigm and the fact that the data were only collected up to one-week post film viewing, experimental effects should not be generalised to first-hand and different types of trauma, and should be interpreted with

caution. For example, as the instructions for the trauma film viewing asked participants to sit as still as possible and avoid conversation, passive rather than active coping strategies are more likely to be triggered. Therefore, the trauma film paradigm may not be a suitable design to investigate active defence coping mechanisms, such as fight or flight responses.

Moreover, we used the finding of higher vividness of intrusions to infer a 'higher level of memory consolidation'. However, as the assessment of the vividness of intrusion was only perfomed in the first week after viewing the trauma film, in terms of the clinical implications, it is unknown whether the 'higher-level consolidation' observed and defined in the current study is a beneficial or harmful process in the long run. To further discuss this issue in the context of the DRT (Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996), it is unclear whether this vivid memory is a product of an adaptive process, in which memories were well contextualised and integrated with existing memory structures, like the contextualised representations (C-reps). If it is, despite having more vivid intrusions in the first week, these individuals with lower cortisol secretion may suffer less from intrusive symptoms in the long run. However, it is also possible that highly vivid memories are not well contextualized. Like the sensory-bound representations in the DRT, they might be unprocessed sensory materials from the original events which lack corresponding C-reps or strong connections to C-reps. If this is the case, more severe intrusive symptoms may still be observed among these individuals in a latter assessment (Brewin et al., 2010; Brewin, Dalgleish, & Joseph, 1996).

Finally, the sample size involved in the examinations of the effects of cortisol in Chapter 4 (i.e., 59) was sufficiently smaller than what was suggested by the priori power calculation (i.e., 66 and 78). Future studies with greater power should be conducted to replicate the current findings.

7.5.2.2 Future research directions

Overall, our studies suggest the valuable potential of adopting cortisol and cardiovascular indices at early stages of the trauma response to predict the risk of PTSD development. Before making stronger suggestions, future studies should, firstly, clarify whether the increased vividness of intrusion, which we found to be associated with low level of trauma-related cortisol secretion, is a part of the recovery process, or is a negative influence on psychological well-being without clear therapeutic contributions in the long run. Secondly, it is crucial to investigate whether our findings may be replicated in studies involving victims of real-life trauma, and traumas that trigger more active coping strategies. Similarly and finally, in order to be more informative for real-life practice, future studies should examine the validity of these physiological measures in predicting intrusive memory symptoms present at least a month after the target trauma.

To examine these hypotheses, ECG data and salivary cortisol and sAA samples may be collected among trainee medical service providers (e.g., intern doctors and nurses) in the emergency room of hospitals before, during, and after providing/witnessing an emergency medical treatment. The intrusive memories related to this experience should be followed up for at least a month with the intrusion diary. Overall PTSD symptoms should be assessed with a diagnostic interview a month later. Similar to our studies, investigations of the correlations between the physiological data, memory and overall symptom measures, as well as the roles of potential moderating factors (i.e., sHR and CDR) should be conducted.

Sufficient cognitive processing and its accompanying clarity of memory in the early stage of trauma have been suggested as a resilience factor (e.g., Brewin et al., 2010; Horowitz, 1986). However, overly encoded sensory information of the trauma may create more dominant S-memories (Brewin et al., 2010) and may relate to greater emotional alarm

and hence greater hyper-vigilant symptoms (Jones and Barlow, 1990). Based on these theories, a quadratic relationship (U-shape) between the vividness of intrusion in the early aftermath of trauma, and overall PTSD symptoms a month later is predicted. Following this hypothesis, a low level of post-traumatic cortisol may only predict greater PTSD symptoms when the vividness of intrusion in the early aftermath of trauma is close to an extreme level. Additionally, as among the LSG, lower peri-traumatic HR has been found to be associated with dissociation and lower vividness of intrusion in our study, lower HR and stronger dominance of the vagal system among this population is predicted to correlate with greater PTSD symptoms, especially dissociation, in future studies.

7.5.3 What should be done to better examine traumatic memory retrieval?

7.5.3.1 Limitations of our studies

The main methodological limitation in our examinations of the memory retrieval phase of trauma (Chapter Five and Chapter Six) is the small sample sizes. Specifically, the sample sizes in Chapter Five (i.e., 22) and Chapter Six (i.e., 18) were sufficiently below the suggestions of the priori power calculations for the multiple regressions and mixed design ANOVAs conducted in the studies (i.e., 55 and 34 respectively). This has resulted in a restricted statistical power involved in these analyses. Moreover, in comparing the difference in HR variation between PTSD patients with different experiences during the trauma recall (Chapter Five), the number of individuals who experienced dissociation was too small for statistical analysis. Similarly, there were considerable diversities in terms of PTSD symptom severity, length, and cause of pathology, among our sample. These factors have introduced covariance in the statistics. For example, several characteristics (e.g., peri-traumatic dissociation, the number of previously experienced traumas) have been found to be significantly different between the patients with high (i.e., HSPG) and moderate sHR patterns (i.e., MSPG). However, due to the small sample size, we did not have sufficient statistical power to control these covariances, and to further examine sHR as a source of individual differences in the relationship between HR and psychological states (see Chapter Five). Future studies with greater statistic power should be conducted to replicate the current findings and to examine the topics that lacked a sufficient sample size to examine.

The second major limitation in this part of the thesis is the fact that we only collected cross-sectional data, without associating the physiological and psychological responses during the trauma recall with the psychological outcomes in a later period of time. Specifically, we have found an association between heightened activation of the vagal system

and greater state dissociation during the voluntary memory retrieval of trauma. Although we have inferred from our data that vagal dominance during exposure therapy may be a predictor of poor treatment outcome, as fewer cognitive resources may be involved, our data have not provided direct evidence to support this hypothesis. Similarly, we found that PTSD patients' cortisol levels were not significantly elevated due to a voluntary retrieval of their traumatic memories, and PTSD Accelerators even showed a significant cortisol decrease in response to this procedure. However, without associating these findings with symptom related measures assessed at a later time point, it is hard to confirm the clinical implications of these physiological phenomena during the memory retrieval phase. Future studies with longitudinal designs will improve the potential of applying our findings to predict therapeutic outcomes.

7.5.3.2 Directions of future studies

Following the previous section, replications of our studies with larger sample sizes are needed. Moreover, in order to have better clinical relevance, future studies should associate the cardiovascular and cortisol responses during the memory retrieval of trauma with measures of symptom variation.

For example, ECG data and salivary cortisol and sAA samples may be collected during exposure therapies in real-life clinical settings. Similar to our study, associations between these physiological measures and psychological states during memory exposure/retrieval procedures should be investigated. Moreover, it is of research interest to examine the relationship between cortisol level during memory retrieval and the quality (e.g., specificity and vividness) of the memories recalled. Furthermore, physiological and psychological reactions during the memory retrieval procedure should be associated with the level of symptom reduction after a certain number of therapy sessions.

Associations similar to our findings are predicted between cortisol, HR, HRV and psychological reactions (i.e., emotional arousal, flashback, dissociation) to the memory retrieval procedure in the future study proposed above. As we have found a significant and negative correlation between cortisol level and the vividness of intrusive memories, low levels of cortisol during the memory retrieval procedure are expected to predict greater specificity and vividness of the recalled materials. Moreover, we found that PTSD Accelerators had greater levels of cortisol reduction in response to the trauma recall. Following the above hypothesis, they are predicted to develop more vivid memories. Similar to our hypothesis regarding the relationship between the vividness of traumatic memory and overall PTSD symptoms (7.4.2.2), a U-shape correlation between the two is expected in this

study. In sum, these investigations will help to strengthen the clinical implications of our current findings.

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Appendix 1: Instructions for the intrusion diary and a sample diary sheet

CONFIDENTIAL

Participant ID

Date

IF FOUND, PLEASE RETURN TO:

Chia-Ying Chou
Research Department of Clinical,
Educational and Health Psychology, UCL,
Torrington Place, London WC1E 7HB

Email: chia-ying.chou.10@ucl.ac.uk
Mobil: 07412493009

DIARY OF INTRUSIVE MEMORIES

- **Each day over the next 7 days**, please note down in this diary any spontaneously occurring **INTRUSIONS** you have about the film you have just watched.
- By INTRUSIONS, I mean intrusive memories of the video that suddenly **pop into your mind spontaneously**. I do not mean times when you deliberately think about it or mull it over.
- Intrusions may take the form of **pictures or thoughts** of the film you have just seen.
- You may find it useful to set aside **certain time each day** when you can fill in the diary. I will also send you a text message reminder each day.
- Look at the table over the page. You are asked to record the **timing** of intrusions, and for each individual intrusion,

1) whether it was primarily **an image or a thought or both** and 2) **what** the intrusion was of.

- Also, fill in the boxes on level of **distress**, and **vividness** that accompany each intrusion by entering a number between 0 and 100 that reflects your experience.

0 = not at all **50 = moderately** **100 = extremely**

- Please use one diary sheet per day
- If you cannot fit all the intrusions for one time of day into the space provided please continue on another sheet.
- If you have no intrusions please put zero for that time of day.

TIME AND DATE OF FOLLOW UP APPOINTMENT:

Please rate how **compliant** you have been in keeping this diary over the past week.
(0 = not at all ~ 100 = completely):_____

DAY 1: _____/_____/_____( )

Time of day	approximate **Timing** of intrusions	Was the intrusion an **IMAGE (I), THOUGHT (T)** or **BOTH (IT)**?	**Content**: (Please describe briefly what each intrusion was of)	How **DISTRESSED** were you by the intrusion? (0~100)	How **VIVID** was the intrusion? (0~100)
MORNING (before lunch)					
AFTERNOON (before dinner)					
EVENING (before bed)					
NIGHT					

Appendix 2: Recognition task

ID:_____ date: ___/___/_____

Recognition Memory Test

Please choose the correct answer from each of the statements below that relate to the film you watched:

Scene 1

1) At the start of the scene, fire fighters hurry to extinguish the flames on a:
   a) Motorbike
   b) Sports car
   c) School bus
   d) Lorry

2) The accident had occurred due to:
   a) An oil spillage
   b) A drunk driver
   c) A sudden rain storm
   d) A police chase

3) The several collisions have occurred:
   a) On a local housing estate
   b) On a motorway
   c) In a busy town centre
   d) In a car park

4) A man who has a bandaged head and is badly cut is helped away from the wreckage by two men. The man is:
   a) In a wheelchair
   b) Walking
   c) On a stretcher
   d) On crutches

5) Due to the accident:
   a) A single person later died in hospital
   b) A number of people lost their lives
   c) There were some injuries but no deaths
   d) There were no serious injuries or deaths

6) A man opens a blanket to reveal an injured child. The child is wearing:
   a) A black jacket
   b) A blue jacket
   c) An orange Jacket
   d) A red jacket

Scene 2

1) A woman screams in agony and seems to lose consciousness. She is wearing:
   a) A black t-shirt
   b) A white t-shirt
   c) A red t-shirt
   d) A green t-shirt

2) A man lies on a stretcher as paramedics cut away his clothes to reveal:
   a) Cuts to his arms
   b) Cuts his legs
   c) Cuts to his back
   d) Cuts to his chest

3) Due to the remote location, it took the ambulance and fire crew a long time to reach the accident. This resulted in:
   a) The woman being permanently disabled
   b) A child losing its life
   c) No one was injured
   d) A family drowned

4) The accident has taken place in:
   a) England
   b) Germany
   c) America
   d) France

5) The man involved in the accident is:
   a) A middle aged Asian man
   b) A young white man
   c) A middle aged white man
   d) A young Asian man

6) The man crashed into the vehicle because:
   a) He had swerved to miss a pedestrian
   b) He was drunk
   c) He suffered a heart attack
   d) He was driving too fast

Scene 3

1) The accident has involved a
   a) Head on collision on a bridge
   b) Car that swerved to miss a pedestrian
   c) Multiple pile up on the motorway
   d) School bus that lost control

2) A body that lies at the side of the wreckage is covered by
   a) A striped blanket
   b) A bright red blanket
   c) A plain blue blanket
   d) A transparent plastic sheet

3) Fire fighters lift the dead body from the wreckage. The body is a :
   a) Young woman
   b) Middle aged white man
   c) A young Asian man
   d) A middle aged woman

4) The crowd that has gathered
   a) Help the injured from the wreckage
   b) Observer the accident from the side of the road
   c) Help push the smashed vehicle to the side of the road
   d) Are pushed away from the scene by armed police

5) A dead body is revealed outside a car as emergency workers lift the body. The body is wearing a blood soaked:
   a) Blue jacket
   b) White t-shirt
   c) Green sweatshirt
   d) The body has no top on

6) As the body is pulled away from the wreckage, the rescue crew:
   a) Drag away the smashed vehicle
   b) Extinguish the flames that have engulfed the vehicle
   c) Lay the body on the road and cover it with blankets
   d) Make way for the emergency helicopter to land

Scene 4

1) A woman lies motionless in a vehicle with no roof as emergency workers surround the vehicle. The vehicle is:
   a) Blue
   b) Red
   c) White
   d) Brown

2) Due to the accident:
   a) A number of people died
   b) No one was seriously injured
   c) A man was permanently disabled
   d) A cyclist was killed

3) As the emergency crew work around the vehicle, one emergency worker holds the motionless woman by her:
   a) Arms
   b) Legs
   c) Collar
   d) Hand

4) The accident involved:
   a) Two cars that had collided on a road
   b) A school bud that had hit a tree
   c) A car and a pedestrian
   d) A good vehicle that had hit a building

5) One of the dead bodies that is placed in a coffin is wearing a:
   a) Blue dress
   b) Pink jumper
   c) Orange jacket
   d) Yellow jacket

6) The accident had taken place:
   a) In the snow
   b) On a clear mild day
   c) On a rainy night
   d) On a clear night

Scene 5

1) The accident had involved:
   a) A family
   b) A single male
   c) A mother and child
   d) Two female students

2) Paramedics work frantically to:
   a) Gain access to the injured
   b) Attend to the injured girl in the ambulance
   c) Drive the ambulance to the scene of the accident
   d) Move the injured man from the wreckage

3) The paramedic bandages the individual's injured:
   a) Leg
   b) Head
   c) Arm
   d) Shoulder

4) The injured individual is worked on in:
   a) An emergency helicopter
   b) An ambulance
   c) The hospital emergency room
   d) The middle of the road

5) The paramedic attending to the injured person is wearing a:
   a) Blue uniform
   b) Green uniform
   c) Brown uniform
   d) Orange uniform

6) The injured individual was wearing:
   a) A torn t-shirt
   b) A dark jacket
   c) No top
   d) A blue sweatshirt

Appendix 3: Life stressor checklist – revised (LSC-R)

Life Stressor Checklist – Revised

This is a questionnaire about life events that are very stressful.

1. Please read the description of each event.
2. Circle "NO" if you have *never* experienced that event.
3. Circle "YES" if you have *ever* experienced that event.
4. If you circle "YES" please go on to answer questions a-c below the event.
   If you circle "NO" you may skip questions a-c below the event.

Please think about your **<u>whole lifetime</u>** when answering the questions.

Be sure to fill in the age you were when the event first happened and the age you were the last time the event happened. If the event only happened once you only need to fill in the first age.

1.	**Have you ever been in a serious disaster (for example, an earthquake, hurricane, tornado, large fire, or explosion)?**	**Yes**	**No**
a.	How many times have you experienced this?	time(s)	
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
2.	**Have you ever seen a serious accident (for example, a bad car wreck or an on-the-job accident)?**	**Yes**	**No**
a.	How many times have you experienced this?	time(s)	
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
3.	**Have you ever had a very serious accident or accident-related injury (for example, a bad car wreck or an on-the-job accident)?**	**Yes**	**No**
a.	How many times have you experienced this?	time(s)	
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
4.	**Was a close family member ever sent to jail?**	**Yes**	**No**
a.	How many times have you experienced this?	time(s)	
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No

5.	**Have you ever been sent to jail?**	**Yes**	**No**
a.	How many times have you experienced this?	_____	time(s)
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
6.	**Were you ever put in foster care or put up for adoption?**	**Yes**	**No**
a.	How many times have you experienced this?	_____	time(s)
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
7.	**Did your parents ever separate or divorce while you were living with them?**	**Yes**	**No**
a.	How many times have you experienced this?	_____	time(s)
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
8.	**Have you ever been separated or divorced?**	**Yes**	**No**
a.	How many times have you experienced this?	_____	time(s)
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
9.	**Have you ever had serious money problems (for example, not enough money for food or a place to live)?**	**Yes**	**No**
a.	How many times have you experienced this?	_____	time(s)
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
10.	**Have you ever had a very serious physical or mental illness (for example, cancer, heart attack, serious operation, felt like killing yourself, hospitalized because of nerve problems)?**	**Yes**	**No**
a.	How many times have you experienced this?	_____	time(s)
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No
11.	**Have you ever been emotionally abused or neglected (for example, being frequently shamed, embarrassed, ignored, or repeatedly told that you were "no good")?**	**Yes**	**No**
a.	How many times have you experienced this?	_____	time(s)
b.	How old were you when it first and last happened?	first:	last:
c.	At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*?	Yes	No

12. Have you ever been physically neglected (for example, not fed, not properly clothed, or left to take care of yourself when you were too young or ill)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

13. WOMEN ONLY: Have you ever had an abortion or miscarriage (lost your baby)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

14. Have you ever been separated from your child against your will (for example, the loss of custody or visitation or kidnapping)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

15. Has a baby or child of yours ever had a severe physical or mental handicap (for example, mentally retarded, birth defects, can't hear, see, walk)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

16. Have you ever been responsible for taking care of someone close to you (not your child) who had a severe physical or mental handicap (for example, cancer, stroke, Alzheimer's disease, AIDS, nerve problems, can't hear, see, walk)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

17. Has someone close to you died suddenly or unexpectedly (for example, an accident, sudden heart attack, murder or suicide)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

18. Has someone close to you died (do not include those who died suddenly or unexpectedly)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

19. When you were young (before age 16) did you ever see violence between family members (for example, hitting, kicking, slapping, punching)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

20. Have you ever seen a robbery, mugging, or attack taking place? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

21. Have you ever been robbed, mugged, or physically attacked (not sexually) by someone you did not know? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

22. *Before age 16*, were you ever abused or physically attacked (not sexually) by someone you knew (for example, a parent, boyfriend, or husband hit, slapped, choked, burned, or beat you up)? **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

***23. After age 16*, were you ever abused or physically attacked (not sexually) by someone you knew (for example, a parent, boyfriend, or husband hit, slapped, choked, burned, or beat you up)?** **Yes** **No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

24. Have you ever been bothered or harassed by sexual remarks, jokes, or demands for sexual favors by someone *at work or* **Yes** **No**

***school* (for example, a co-worker, a boss, a customer, another student, a teacher)?**

a. How many times have you experienced this? _____time(s)
d. How old were you when it first and last happened? first: last:
e. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

25. *Before age 16*, were you ever <u>touched</u> or made to <u>touch someone else</u> in a *sexual way* because he/she forced you in some way or threatened to harm you if you didn't? **Yes No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

26. *After age 16*, were you ever <u>touched</u> or made to <u>touch someone else</u> in a *sexual way* because he/she forced you in some way or threatened to harm you if you didn't? **Yes No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

27. *Before age 16*, did you ever have sex (oral, anal, genital) when you didn't want to because someone forced you in some way or threatened to harm you if you didn't? **Yes No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

28. *After age 16*, did you ever have sex (oral, anal, genital) when you didn't want to because someone forced you in some way or threatened to harm you if you didn't? **Yes No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

29. Have you ever been directly exposed to war, armed conflict, or terrorism (were there soldiers or others fighting or hurting people near where you lived)? **Yes No**

a. How many times have you experienced this? _____time(s)
b. How old were you when it first and last happened? first: last:
c. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

30. Have you ever had leave where you were living and move to another location (country, state, or city) because you could not pay for basic needs, like food clothing or shelter, or because you **Yes No**

felt unsafe?

a. How many times have you experienced this? _____time(s)

d. How old were you when it first and last happened? first: last:

e. At the time of the event did you believe that *you or a loved one* could be *killed* or seriously *harmed*? Yes No

31. Are there any events we did not include that you would like to mention? Yes No

What was the event?

Appendix 4: Posttraumatic stress diagnostic scale

Post-traumatic Stress Diagnostic Scale

(Version 1: 26/04/2012)

PART 1

Many people have lived through or witnessed a very stressful and traumatic event at some point in their lives. Indicate whether or not you have experienced or witnessed each traumatic event below by marking Y for Yes or N for No.

1.	Y N	Serious accident, fire, or explosion (for example, an industrial, farm, car, plane or boating accident)
2.	Y N	Natural disaster (for example, tornado, hurricane, flood, or major earthquake)
3.	Y N	Non-sexual assault by a family member or someone you know (for example, being mugged, physically attacked, shot, stabbed, or held at gunpoint)
4.	Y N	Non-sexual assault by a stranger (for example, being mugged, physically attacked, shot, stabbed, or held at gunpoint)
5.	Y N	Sexual assault by a family member or someone you know (for example, rape or attempted rape)
6.	Y N	Sexual assault by a stranger (for example, rape or attempted rape)
7.	Y N	Military combat or a war zone
8.	Y N	Sexual contact when you were younger than 18 with someone who was 5 or more years older than you (for example, contact with genitals, breasts)
9.	Y N	Imprisonment (for example, prison inmate, prisoner of war, hostage)
10.	Y N	Torture
11.	Y N	Life-threatening illness
12.	Y N	Other traumatic event
13.	If you answered Yes to item 12, specify traumatic event below. _____	

PART 2

14. If you marked Yes for more than one traumatic event in Part 1, indicate *which one bothers you the most.* If you marked Yes for only one traumatic event in Part 1, mark the same one below.
    1. Accident
    2. Disaster
    3. Non-sexual assault/someone you know
    4. Non-sexual assault/stranger
    5. Sexual assault/someone you know
    6. Sexual assault/stranger
    7. Combat
    8. Sexual contact under 18 with someone 5 or more years older
    9. Imprisonment
    10. Torture
    11. Life-threatening illness
    12. Other traumatic event

Below are several questions about the traumatic event you marked in Item 14.

15. How long ago did the traumatic event happen? (mark ONE)
    1. Less than 1 month
    2. 1 to 3 months
    3. 3 to 6 months
    4. 6 months to 3 years
    5. 3 to 5 years
    6. More than 5 years

For the following questions, mark Y for Yes or N for No

During the traumatic event:

16.	Y N	Were you physically injured?
17.	Y N	Was someone else physically injured?
18.	Y N	Did you think that your life was in danger?
19.	Y N	Did you think that someone else's life was in danger?
20.	Y N	Did you feel helpless?
21.	Y N	Did you feel terrified?

PART 3

Below is a list of problems that people sometimes have after experiencing a traumatic event. Read each one carefully and choose the answer (0-3) that best describes how often that problem has bothered you IN THE PAST MONTH. Rate each problem with respect to the traumatic event you marked in Item 14.

0	Not at all or only one time
1	Once a week or less/once in a while
2	2 to 4 times a week/half the time
3	5 or more times a week/almost always

22.	0 1 2 3	Having upsetting thoughts or images about the traumatic event that came into your head when you didn't want them to
23.	0 1 2 3	Having bad dreams of nightmares about the traumatic event
24.	0 1 2 3	Reliving the traumatic event, acting or feeling as if it was happening again
25.	0 1 2 3	Feeling emotionally upset when you were reminded of the traumatic event (for example, feeling scared, angry, sad, guilty, etc)
26.	0 1 2 3	Experiencing physical reactions when you were reminded of the traumatic event (for example, breaking out in a sweat, heart beating fast)
27.	0 1 2 3	Trying not to think about, talk about, or have feelings about the traumatic event
28.	0 1 2 3	Trying to avoid activities, people, or places that remind you of the traumatic event
29.	0 1 2 3	Not being able to remember an important part of the traumatic event
30.	0 1 2 3	Having much less interest or participating much less often in important activities

0	Not at all or only one time

1	Once a week or less/once in a while
2	2 to 4 times a week/half the time
3	5 or more times a week/almost always

31.	0 1 2 3	Feeling distant or cut off from people around you
32.	0 1 2 3	Feeling emotionally numb (for example, being unable to cry or unable to have loving feelings)
33.	0 1 2 3	Feeling as if your future plans or hopes will not come true (for example, you will not have a career, marriage, children, or a long life)
34.	0 1 2 3	Having trouble falling or staying asleep
35.	0 1 2 3	Feeling irritable or having fits of anger
36.	0 1 2 3	Having trouble concentrating (for example, drifting in and out of conversations, losing track of a story on television, forgetting what you read)
37.	0 1 2 3	Being overly alert (for example, checking to see who is around you, being uncomfortable with you back to a door, etc.)
38.	0 1 2 3	Being jumpy or easily startled (for example, when someone walks up behind you)
39.	How long have you experienced the problems that you reported above? (mark ONE) 1. Less that one month 2. 1 to 3 months 3. More than 3 months	
40.	How long after the traumatic event did these problems begin? (mark ONE) 1. Less than 6 months 2. 6 or more months	

PART 4

Indicate below if **the problems you rated in Part 3** have interfered with any of the following areas of your life DURING THE PAST MONTH. Mark Y for Yes or N for No.

41.	Y N	Work
42.	Y N	Household chores and duties
43.	Y N	Relationships with friends
44.	Y N	Fun and leisure activities
45.	Y N	Schoolwork
46.	Y N	Relationships with your family
47.	Y N	Sex life
48.	Y N	General satisfaction with life
49.	Y N	Overall level of functioning in all areas of your life

Appendix 5: State trait anxiety inventory (STAI)

STAI – Form Y-1

Participant ID_____ Date_____ A / B / C

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle a number to indicate how you feel ***right now,*** that is, ***at this moment***. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

1. **I feel calm**

not at all	slightly	considerably	extremely
1	2	3	4

2. **I feel secure**

not at all	slightly	considerably	extremely
1	2	3	4

3. **I am tense**

not at all	slightly	considerably	extremely
1	2	3	4

4. **I feel strained**

not at all	slightly	considerably	extremely
1	2	3	4

5. **I feel at ease**

not at all	slightly	considerably	extremely
1	2	3	4

6. **I feel upset**

not at all	slightly	considerably	extremely
1	2	3	4

7. **I am presently worrying over possible misfortunes**

not at all	slightly	considerably	extremely
1	2	3	4

8. **I feel satisfied**

not at all	slightly	considerably	extremely
1	2	3	4

9. **I feel frightened**

not at all	slightly	considerably	extremely
1	2	3	4

10. **I feel comfortable**

not at all	slightly	considerably	extremely
1	2	3	4

11. **I feel self-confident**

not at all	slightly	considerably	extremely
1	2	3	4

12. **I feel nervous**

not at all	slightly	considerably	extremely
1	2	3	4

13. **I am jittery**

not at all	slightly	considerably	extremely
1	2	3	4

14. **I feel indecisive**

not at all	slightly	considerably	extremely
1	2	3	4

15. **I am relaxed**

not at all	slightly	considerably	extremely
1	2	3	4

16. **I feel content**

not at all	slightly	considerably	extremely
1	2	3	4

17. **I am worried**

not at all	slightly	considerably	extremely
1	2	3	4

18. **I feel confused**

not at all	slightly	considerably	extremely
1	2	3	4

19. **I feel steady**

not at all	slightly	considerably	extremely
1	2	3	4

20. **I feel pleasant**

not at all	slightly	considerably	extremely
1	2	3	4

STAI – Form Y-2

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle a number indicating how you ***generally*** feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

21. **I feel pleasant**

Almost never	sometimes	often	Almost always
1	2	3	4

22. **I feel nervous and restless**

Almost never	sometimes	often	Almost always
1	2	3	4

23. **I feel satisfied with myself**

Almost never	sometimes	often	Almost always
1	2	3	4

24. **I wish I could be as happy as others** seem to be

Almost never	sometimes	often	Almost always
1	2	3	4

25. **I feel like a failure**

Almost never	sometimes	often	Almost always
1	2	3	4

26. **I feel rested**

Almost never	sometimes	often	Almost always
1	2	3	4

27. **I am** "**calm, cool, and collected**"

Almost never	sometimes	often	Almost always
1	2	3	4

28. **I feel that difficulties are piling up so that** I **cannot overcome**

Almost never	sometimes	often	Almost always
1	2	3	4

29. I **worry too much over something that really doesn't matter**

Almost never	sometimes	often	Almost always
1	2	3	4

30. **I am happy**

Almost never	sometimes	often	Almost always
1	2	3	4

31. **I have disturbing thoughts**

Almost never	sometimes	often	Almost always
1	2	3	4

32. **I lack self-confidence**

Almost never	sometimes	often	Almost always
1	2	3	4

33. **I feel secure**

Almost never	sometimes	often	Almost always
1	2	3	4

34. **I make decisions easily**

Almost never	sometimes	often	Almost always
1	2	3	4

35. **I feel inadequate**

Almost never	sometimes	often	Almost always
1	2	3	4

36. **I am content**

Almost never	sometimes	often	Almost always
1	2	3	4

37. **Some unimportant thought runs through my mind and bothers me**

Almost never	sometimes	often	Almost always
1	2	3	4

38. **I take disappointments so keenly that I can't put them out of my mind**

Almost never	sometimes	often	Almost always
1	2	3	4

39. **I am a steady person**

Almost never	sometimes	often	Almost always
1	2	3	4

40. **I get in a state of tension or turmoil as I think over my recent concerns and interests**

Almost never	sometimes	often	Almost always
1	2	3	4

Appendix 6: Dissociative experience scale (DES)

DISSOCIATIVE EXPERIENCES SCALE
(Carlson & Putnam, 1993)

Directions: This questionnaire consists of 28 questions about experiences that you may have had in your daily life. We are interested in how often you have these experiences. It is important, however, that your answers show how often these experiences happen to you when you **are not under the influence of alcohol or drugs** (prescribed or otherwise). To answer the questions, please determine to what degree the experience described in the question applies to you and circle the number to show what percentage of the time you have the experience.

Example:

0%	10	20	30	40	50	60	70	80	90	100%
(never)										(always)

Some people have the following experiences. Please circle the number to show what percentage of the time this happens to you.

1. Some people have the experience of driving or riding in a car or bus or subway and suddenly realizing that they don't remember what has happened during all or part of the trip.

0%	10	20	30	40	50	60	70	80	90	100%

2. Some people find that sometimes they are listening to someone talk and they suddenly realize that they did not hear part or all of what was said.

0%	10	20	30	40	50	60	70	80	90	100%

3. Some people have the experience of finding themselves in a place and having no idea how they got there.

0%	10	20	30	40	50	60	70	80	90	100%

4. Some people have the experience of finding themselves dressed in clothes that they don't remember putting on.

0%	10	20	30	40	50	60	70	80	90	100%

5. Some people have the experience of finding new things among their belongings that they do not remember buying.

0%	10	20	30	40	50	60	70	80	90	100%

6. Some people sometimes find that they are approached by people that they do not know who call them by another name or insist that they have met them before.

0%	10	20	30	40	50	60	70	80	90	100%

7. Some people sometimes have the experience of feeling as though they are standing next to themselves or watching themselves do something and they actually see themselves as if they were looking at another person.

0%	10	20	30	40	50	60	70	80	90	100%

8. Some people are told that they sometimes do not recognize friends or family members.

0%	10	20	30	40	50	60	70	80	90	100%

9. Some people find that they have no memory for some important events in their lives (for example, a wedding or graduation).

0%	10	20	30	40	50	60	70	80	90	100%

10. Some people have the experience of being accused of lying when they do not think that they have lied.

0%	10	20	30	40	50	60	70	80	90	100%

11. Some people have the experience of looking in a mirror and not recognizing themselves.

0%	10	20	30	40	50	60	70	80	90	100%

12. Some people have the experience of feeling that other people, objects, and the world around them are not real.

0%	10	20	30	40	50	60	70	80	90	100%

13. Some people have the experience of feeling that their body does not seem to belong to them.

0%	10	20	30	40	50	60	70	80	90	100%

14. Some people have the experience of sometimes remembering a past event so vividly that they feel as if they were reliving that event.

0%	10	20	30	40	50	60	70	80	90	100%

15. Some people have the experience of not being sure whether things that they remember happening really did happen or whether they just dreamed them.

Some people have the following experiences. Please circle the number to show what percentage

0% 10 20 30 40 50 60 70 80 90 100%

16. Some people have the experience of being in a familiar place but finding it strange and unfamiliar.

0% 10 20 30 40 50 60 70 80 90 100%

28. Some people sometimes feel as if they are looking at the world through a fog so that people and objects

17. Some people find that when they are watching television or a movie they become so absorbed in the story that they are unaware of other events happening around them.

0% 10 20 30 40 50 60 70 80 90 100%

18. Some people find that they become so involved in a fantasy or daydream that it feels as though it were really happening to them.

0% 10 20 30 40 50 60 70 80 90 100%

19. Some people find that they sometimes are able to ignore pain.

0% 10 20 30 40 50 60 70 80 90 100%

Some people have the following experiences. Please circle the number to show <u>what percentage of the time this happens to you</u>.

20. Some people find that that they sometimes sit staring off into space, thinking of nothing, and are not aware of the passage of time.

0% 10 20 30 40 50 60 70 80 90 100%

21. Some people sometimes find that when they are alone they talk out loud to themselves.

0% 10 20 30 40 50 60 70 80 90 100%

22. Some people find that in one situation they may act so differently compared with another situation that they feel almost as if they were two different people.

0% 10 20 30 40 50 60 70 80 90 100%

23. Some people sometimes find that in certain situations they are able to do things with amazing ease and spontaneity that would usually be difficult for them (for example, sports, work, social situations, etc.).

0% 10 20 30 40 50 60 70 80 90 100%

24. Some people sometimes find that they cannot remember whether they have done something or have just thought about doing it (for example, not knowing whether they have just mailed a letter or have just thought about mailing it).

0% 10 20 30 40 50 60 70 80 90 100%

25. Some people find evidence that they have done things that they do not remember doing.

0% 10 20 30 40 50 60 70 80 90 100%

26. Some people sometimes find writings, drawings, or notes among their belongings that they must have done but cannot remember doing.

0% 10 20 30 40 50 60 70 80 90 100%

27. Some people sometimes find that they hear voices inside their head that tell them to do things or comment on things that they are doing.

0% 10 20 30 40 50 60 70 80 90 100%

Appendix 7: Peritraumatic dissociative experiences questionnaire (PDEQ)

Please complete the items below by circling the choice that best describes your experiences and reactions ***during and immediately after the event that you selected in Q14 on page 1*** *of the* ***Post-traumatic Stress Diagnostic Scale.***

1. I had moments of losing track of what was going on – I "blank out", or "spaced out" or in some way felt that I was not part of what was going on.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

2. I found that I was on "automatic pilot" – I ended up doing things that I later realized I hadn't actively decided to do.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

3. My sense of time changed – things seemed to be happening in slow motion.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

4. What was happening seemed unreal to me, like I was in a dream or watching a film or play.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

5. I felt as though I were a spectator watching what was happening to me, as if I were floating above the scene or observing it as an outsider.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

6. There were moments when my sense of my own body seemed distorted or changed. I felt disconnected from my own body, or that it was unusually large or small.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

7. I felt as though things that were actually happening to others were happening to me – like I was being trapped when I really wasn't.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

8. I was surprised to find out afterwards that a lot of things had happened at the time that I was not aware of, especially things I ordinarily would have noticed.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

9. I felt confused; that is, there were moments when I had difficulty making sense of what was happening.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

10. I felt disoriented; that is, there were moments when I felt uncertain about where I was or what time it was.

1 Not at all true | 2 Slightly true | 3 Somewhat true | 4 Very true | 5 Extremely true

Appendix 8: Dissociative state scale (DSS)

Dissociative State Scale

(Version 2: 29/06/2012)

Participant ID _____ Date _____ 0 / 1 / 2 / 3 / 4

Please answer the following questions by circling a number from 1 to 5 indicating how you feel **AT THIS MOMENT IN TIME**, in this room:

1. At this moment in time: Do things seem to be moving in slow motion?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

2. At this moment in time: Do things seem unreal to you as if you are in a dream?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

3. At this moment in time: Do you have some experience that separates you from what is happening; for instance, do you feel as if you are in a film or play, or as if you are a robot?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

4. At this moment in time: Do you feel as if you are looking at things from outside your body?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

5. At this moment in time: Do you feel as if you are watching the situation as an observer or spectator?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

6. At this moment in time: Do you feel disconnected from your own body?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

7. At this moment in time: Does your sense of your own body feel changed: for instance, does your own body feel unusually large or unusually small?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

8. At this moment in time: Do people seem motionless, dead or mechanical?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

9. At this moment in time: Do objects look different that you would expect?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

10. At this moment in time: Do colours seem diminished in intensity?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

11. At this moment in time: Do you see things as if you were in a tunnel, or looking through a wide angle photographic lens?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

12. At this moment in time: Does this experience seem to take much longer than you would have expected?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

13. At this moment in time: Do things seem to be happening very quickly, as if there is a lifetime in each moment?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

14. At this moment in time: Do things happen that you cannot account for?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

15. At this moment in time: Do you space out or in some way lose track of what is going on?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

16. At this moment in time: Do sounds almost disappear or become much stronger that you would have expected?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

17. At this moment in time: Do things seem to be very real, as if there is a special sense of clarity?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

18. At this moment in time: Does it seem as if you are looking at the world through a fog, so that people or objects seem far away or unclear?

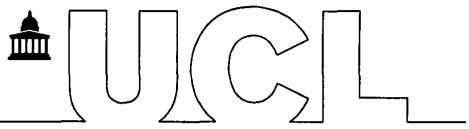
not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

19. At this moment in time: Do colours seem much brighter than you would have expected?

not at all	slightly	moderately	considerably	extremely
0	1	2	3	4

Appendix 9: Ethics approval for Chapter Three and Chapter Four

UCL RESEARCH ETHICS COMMITTEE
GRADUATE SCHOOL OFFICE



Professor Chris Brewin
Sub-Department of Clinical, Educational and Health Psychology
UCL Psychology and Language Sciences

16 March 2011

Dear Professor Brewin

Notification of Ethical Approval
Ethics Application: 3014/001: Cardiovascular responses to traumatic information

I am pleased to confirm that in my capacity as Chair of the UCL Research Ethics Committee, I have approved your study for the duration of the project (i.e. until October 2013). However, as outlined below, please ensure that you notify the committee of any adverse reactions to the film or questionnaires.

Approval is subject to the following conditions:

1. You must seek Chair's approval for proposed amendments to the research for which this approval has been given. Ethical approval is specific to this project and must not be treated as applicable to research of a similar nature. Each research project is reviewed separately and if there are significant changes to the research protocol you should seek confirmation of continued ethical approval by completing the 'Amendment Approval Request Form'.

The form identified above can be accessed by logging on to the ethics website homepage: http://www.grad.ucl.ac.uk/ethics/ and clicking on the button marked 'Key Responsibilities of the Researcher Following Approval'.

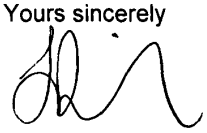
2. It is your responsibility to report to the Committee any unanticipated problems or adverse events involving risks to participants or others. Both non-serious and serious adverse events must be reported.

   Reporting Non-Serious Adverse Events
   For non-serious adverse events you will need to inform Helen Dougal, Ethics Committee Administrator (ethics@ucl.ac.uk), within ten days of an adverse incident occurring and provide a full written report that should include any amendments to the participant information sheet and study protocol. The Chair or Vice-Chair of the Ethics Committee will confirm that the incident is non-serious and report to the Committee at the next meeting. The final view of the Committee will be communicated to you.

   Reporting Serious Adverse Events
   The Ethics Committee should be notified of all serious adverse events via the Ethics Committee Administrator immediately the incident occurs. Where the adverse incident is unexpected and serious, the Chair or Vice-Chair will decide whether the study should be terminated pending the opinion of an independent expert. The adverse event will be considered at the next Committee meeting and a decision will be made on the need to change the information leaflet and/or study protocol.

On completion of the research you must submit a brief report (a maximum of two sides of A4) of your findings/concluding comments to the Committee, which includes in particular issues relating to the ethical implications of the research.

Yours sincerely



Sir John Birch
Chair of the UCL Research Ethics Committee

Cc: Chia-Ying Chou

Appendix 10: Information sheet used in Chapter Three and Chapter Four

VOLUNTEER INFORMATION SHEET

You will be given a copy of this information sheet.

Title of project: Cardiovascular responses to traumatic information

This study has been approved by the UCL Research Ethics Committee. [Project ID Number: 3014/001]

Purpose of the study:

To investigate the relationship between physiological reactions such as heart rate and the response to viewing a traumatic film.

Investigators: Chia-Ying Chou, Prof. Chris R. Brewin

We would like to invite you to participate in this research project. You should only participate if you want to; choosing not to take part will not disadvantage you in any way. Before you decide whether you want to take part, it is important for you to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or you would like more information.

……Please read the following carefully…….……..……

Background of the study:

Many physiological reactions are thought to be important in understanding the response to stress. However, studies to date have rarely investigated how these reactions are related to mental processes and their ability to predict memory function. This study aims to find out more about the relationship between physiological reactions (such as cardiac activities and the release of stress-related hormones and enzymes) and the mental processing styles during exposure to a film with distressing content.

Who can participate?

Healthy male and females, aged 18-40 years without history of cardiovascular or mental health problems.

Do I have to take part?

It is up to you to decide. We will describe the study and go through this information sheet, which we will then give to you. We will then ask you to sign a consent form to show you have agreed to take part. You are free to withdraw at any time, without giving a reason.

What will happen to me if I take part?

If you decide to take part you will be given this information sheet to keep and be asked to sign a consent form. You will need to attend two sessions at UCL; the first session will last around 1 hour, the second session will be 7 days later and will last around half an hour. For the 7 days in between, you will be asked to keep a short daily diary.

What will I have to do?
If you agree to participate, at the first session, you will be asked to fill out some questionnaires about your emotional state and will be presented with a short film containing graphic scenes of the aftermath of road traffic accidents, including seriously injured and dead victims. During the session you will be attached to a heart rate recording sensor on your chest (total duration: around 50 minutes), and have a cotton rod in your mouth to collect saliva 3 times (for 2 minutes each time).
For the following 7 days, you will keep a simple 'diary' of any spontaneous memories about the film. You will return 7 days later for a follow up session to discuss the diary and answer some questions about the film. Apart from completing a questionnaire, you will also be asked about the content of the film while being tape-recorded. You will then be debriefed and given the opportunity to ask questions about the study.

What are the possible disadvantages and risks of taking part?
The experiment involves watching a distressing video, containing graphic scenes of the aftermath of road traffic accidents, including blood, seriously injured, and dead victims. You may have an emotional reaction when watching the film, and there is a very slight chance that you may experience some physical effects (e.g. faint). After watching the video, you may spontaneously think about and may be distressed by it. Spontaneous memories may take the form of visual images, thoughts or mood changes. In previous research with this film involving over 500 participants no persistent emotional problems have been reported, but this does not mean there is no risk to you.

What are the possible benefits of taking part?
We cannot promise the study will help you but the information we get from this study may help improve the treatment of people with post-traumatic stress disorder or other anxiety disorders. You will be sent a copy of the final findings (please inform the investigator if you would like one).

Will my taking part in the study be kept confidential?
Yes. We will follow ethical and legal practice and all information about you will be handled in confidence. All data will be collected and stored in accordance with the Data Protection Act 1998. This means that only the investigators will have access to the data from the study. Your results will not be identified by your name as you will be given a participant number.

What will happen if I don't want to carry on with the study?
If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or decision not to take part, will not affect the standard of care or education you receive. You may withdraw your data from the project at any time up until it is transcribed for use in the final report

What if there is a problem?
If you have a concern about any aspect of this study, you should ask to speak to:

Chia-Ying Chou,
Research Department of Clinical,
Educational and Health Psychology, UCL,
Torrington Place, London WC1E 7HB.
Email: chia-ying.chou.10@ucl.ac.uk
Tel: 020 7679 8279

Pr Prof. Chris Brewin
Research Department of Clinical,
Educational and Health Psychology, UCL,
Gower Street, London WC1E 6BT
E-mail: c.brewin@ucl.ac.uk
Tel: 020 7679 5927

Appendix 11: Feedbacks from NRES for Chapter Five and Chapter Six

NHS
Health Research Authority
NRES Committee London - London Bridge
(Formerly Guy's REC)
Research Ethics Committee (REC) Centre Charing Cross
Room 12, 4th Floor West
Charing Cross Hospital
London
W6 8RF

Telephone: 020 3311 0107
Facsimile: 020 3311 7280

22 June 2012

Professor Chris Brewin
Research Department of Clinical, Educational and Health Psychology
University College London
4th Floor, 1-19 Torrington Place
London WC1E 7HB

Dear Professor Brewin

Study Title:	**PTSD patients' psychological and physiological reactions to the voluntary retrieval of traumatic memories - examining the effect of dissociation.**
REC reference:	**12/LO/0795**

The Research Ethics Committee reviewed the above application at the meeting held on 30 May 2012. Thank you for attending to discuss the study together with Chia-Ying Chou.

Documents reviewed

The documents reviewed at the meeting were:

Document	*Version*	*Date*
Advertisement	1	27 April 2012
Evidence of insurance or indemnity		15 August 2011
Investigator CV	Professor Chris Brewin	27 March 2012
Other: Student CV	Chia-Ying Chou	27 March 2012
Other: Letter fo support from Academic Supervisor		17 April 2012
Participant Consent Form	1	22 March 2012
Participant Information Sheet	1	22 March 2012
Protocol	2	27 March 2012
Questionnaire: Post-traumatic Stress Diagnostic Scale		
Questionnaire: Peritraumatic Dissociative Experiences		
Questionnaire: Mood Rating Scale		
Questionnaire: Dissociative State Scale		

Questionnaire: Traumatic Life Event	2	27 April 2012
REC application	Parts A - D	01 May 2012
Referees or other scientific critique report	Independent review of research proposal	20 March 2012
Summary/Synopsis	1	13 April 2012

Provisional opinion

In answer to questions from the Committee you clarified that:

- A6-2 - The samples that will be sent to Zurich will be linked anonymised, each sample will have a number so that the results can be matched with the relevant participant in the UK.

- A12 – The interview and questionnaires are widely used in the treatment and assessment of PTSD. Dissociation is not usually assessed in these patients. Patients have to be able to retrieve memories deeply. Dissociation is a symptom where memories are blocked. People with dissociation have less benefit from this kind of therapy, however this has not been empirically tested.

- This study aims to look at the quality of the memory retrieved, measuring heart rate whilst they are recalling the memory and testing saliva. You will also be assessing the difference in these between those will high and low dissociative symptoms.

- You will observe the patients behaviour and their heart rate, and would then gauge dissociation from the patient self reporting afterwards. Through this you should be able to match the period the patient had dissociation to an episode of the heart rate readings.

- The Cardiac Defensive Response test using the noise is not standard practice.

- Before patients participate they will be informed of what is going to happen, if they feel uncomfortable at any time they can stop. The researcher will be there while they are watching the video of themselves recalling the traumatic memory. They are not trying to induce dissociation. Before the participants leave the room they will be given a full debriefing and their current mental state will be assessed. If they have a strong reaction they will be given the name of someone to contact to seek further help. The researcher, Chia-Ying Chou will carry out the debriefing but under the supervision of Professor Brewin. If necessary the patient will be accompanied to see someone else or home. You will then call them the next day to ensure that they are alright.

- A22 – When the volunteers who have given their contact information are contacted they will be advised of the inclusion and exclusion criteria, for instance age and symptoms. When they come to participate in the study they will have a diagnostic interview that will be done by the researcher. The researcher will also write to their GP to ask whether there are any reasons why they should not take part and to confirm the diagnosis if it has already been made.

- You do not have the resources to offer treatment to the volunteers from outside the clinics. You can advise them of what is available and make it clear that there is no more than a single session as part of the study.

- A24 & A46 – Usually you compensate volunteers with between £7 and £8. This study is deemed to be more stressful with more tests so they will be compensated an additional £2 per hour totalling £10 per hour.

- The advert does not have the inclusion and exclusion criteria on it as some people will not know if they have the disorder or not. You also do not want to introduce a selection bias. You will then screen all volunteers in a standardised way.

- When the interested volunteers are contacted they will be asked for their GP's details. If they give them over the phone then this will be taken as consent to contact the GP. This can be put in the advertisement.

The Committee felt that if you struggle to recruit from the clinics then you should put in an amendment to recruit from the general public via advertising, however initially they should recruit solely from clinics so that the diagnosis is assured and participants have access to treatment and support after the study has finished.

The Committee would be content to give a favourable ethical opinion of the research, subject to receiving a complete response to the request for further information set out below.

The Committee delegated authority to confirm its final opinion on the application to the Chair or Vice-chair.

Further information or clarification required
Application

Please remove the volunteer group from the protocol and solely recruit from the clinics. The Committee felt that this was necessary to ensure that all participants have access to treatment and support after the study has been completed.

Please consider sending the questionnaires to the participant prior to their attendance at the clinic.

Information sheet(s) and Consent form(s)

Please simplify the language throughout so that it is easily understandable to a lay person.

Please give more information regarding the questionnaires and test and the time they will take to complete.

What will happen if I take part in the study?
Please give further clarification regarding the taking of drugs and drinking of alcohol. Presumably this means that participants must refrain from drinking alcohol before the test and refrain from taking none-prescribed drugs before the test but not after.

Please make it clear what will happen to their data if they lose capacity to consent or withdraw from the study, ensuring this is consistent with the response to A35 of the application form.

PDE Questionnaire – please correct the typo in Question 1 (“part”).

On the various Scale documents consider changing the references from “movie” to “film”.

If you would find it helpful to discuss any of the matters raised above or seek further clarification from a member of the Committee, you are welcome to contact Stephanie Hill on 020 331 10107 or stephanie.hill7@imperial.nhs.uk.

When submitting your response to the Committee, please send revised documentation using tracked changes to highlight the changes you have made and giving revised version numbers and dates.

If the committee has asked for clarification or changes to any answers given in the application form, please do not submit a revised copy of the application form; these can be addressed in a covering letter to the REC.

The Committee will confirm the final ethical opinion within a maximum of 60 days from the date of initial receipt of the application, excluding the time taken by you to respond fully to the above points. A response should be submitted by no later than 20 October 2012.

Membership of the Committee

The members of the Committee who were present at the meeting are listed on the attached sheet.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

12/LO/0795	**Please quote this number on all correspondence**

Yours sincerely

pp

Mr Brady Pohle
Vice-Chair

Email: stephanie.hill7@imperial.nhs.uk

Enclosures: *List of names and professions of members who were present at the meeting and those who submitted written comments.*

Copy to: *Mr Dave Wilson*
Miss Angela Williams, Camden And Islington NHS Foundation Trust

NRES Committee London - London Bridge

Attendance at Committee meeting on 30 May 2012

Committee Members:

Name	*Profession*	*Present*	*Notes*
Professor David Bartlett	Honorary Consultant	No	
Dr Frances Dockery	Consultant Physician	Yes	
Mr David Gallacher	Consultant Physicist	Yes	
Dr Michael Goggin	Consultant Physician	Yes	
Dr Nedim Hadzic	Consultant and Honorary Reader in Paediatric Hepatology	Yes	
Ms Christine Higgins	Lay Member	No	
Miss Tamsin Jones	Lay Member	No	
Dr Margreet Luchtenborg	Cancer Epidemiologist	Yes	
Mrs Marion Maidment	Lay Member	No	
Mr Barry Moody	Lay Member	Yes	
Mr Brady Pohle	Legal Services Advisor	Yes	Vice-Chair
Ms Karen Sanders	Senior Lecturer Nursing, Health Care Ethics & Law	Yes	
Miss Josephine Studham	Clinical Research Facilities Manager	No	
Ms Lorna Sutcliffe	Project Manager/Researcher	No	
Mr William Thornhill	Senior Pharmacist, Paediatric Renal Services	Yes	
Mr Paul Tunstell	Pharmacist	No	
Dr Ralph White	Lay Member	No	

Also in attendance:

Name	*Position (or reason for attending)*
Ms Stephanie Hill	REC Coordinator

Appendix 12: Initial ethics approval for Chapter Five and Chapter Six

NHS
Health Research Authority
NRES Committee London - London Bridge
Health Research Agency
Skipton House
80 London Road
London
SE1 6LH

Telephone: 020 7972 2582

06 September 2012

Professor Chris Brewin
Research Department of Clinical, Educational and Health Psychology
University College London
4th Floor, 1-19 Torrington Place
London WC1E 7HB

Dear Professor Brewin

Study title:	**PTSD patients' psychological and physiological reactions to the voluntary retrieval of traumatic memories - examining the effect of dissociation.**
REC reference:	**12/LO/0795**

Thank you for your letter of 04 September 2012, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Vice-Chair.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

NHS sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Non-NHS sites

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
Advertisement	1	27 April 2012
Evidence of insurance or indemnity		15 August 2011
Investigator CV	Professor Chris Brewin	27 March 2012
Other: Student CV	Chia-Ying Chou	27 March 2012
Other: Letter fo support from Academic Supervisor		17 April 2012
Participant Consent Form	3	03 September 2012
Participant Information Sheet	3	03 September 2012
Protocol	3	29 June 2012
Questionnaire: Post-traumatic Stress Diagnostic Scale		
Questionnaire: Mood Rating Scale		
Questionnaire: Traumatic Life Event	2	27 April 2012
Questionnaire: Dissociative State Scale	2	29 June 2012
Questionnaire: Peritraumatic Dissociative Experiences	2	29 June 2012
REC application	Parts A - D	01 May 2012
Referees or other scientific critique report	Independent review of research proposal	20 March 2012
Response to Request for Further Information		04 July 2012
Response to Request for Further Information		04 September 2012
Summary/Synopsis	1	13 April 2012

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

Feedback

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

12/LO/0795	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

pp

Mr Brady Pohle
Vice-Chair

Email: nrescommittee.london-londonbridge@nhs.net

Enclosures: "After ethical review – guidance for researchers" *SL-AR2*

Copy to: *Mr. Dave Wilson*
Miss Angela Williams, Camden And Islington NHS Foundation Trust
Ms Chia-Ying Chou

Appendix 13: Amendment approval for Chapter Five and Chapter Six

NHS
Health Research Authority
NRES Committee London - London Bridge
Health Research Agency
Skipton House
80 London Road
London
SE1 6LH

Tel: 020 7972 2582

23 April 2013

Professor Chris Brewin
Research Department of Clinical, Educational and Health Psychology
University College London
4th Floor, 1-19 Torrington Place
London WC1E 7HB

Dear Professor Brewin

Study title:	**PTSD patients' psychological and physiological reactions to the voluntary retrieval of traumatic memories - examining the effect of dissociation.**
REC reference:	**12/LO/0795**
Amendment number:	**AM01/1**
Amendment date:	**12 April 2013**
IRAS project ID:	**101776**

Thank you for submitting the above amendment, which was received on 16 April 2013. It is noted that this is a modification of an amendment previously rejected by the Committee (our letter of 22 March 2013 refers).

The modified amendment was reviewed by the Sub-Committee in correspondence. A list of the members who took part in the review is attached.

Ethical opinion

The sub-committee felt the you had addressed all their previous concerns regarding the recruitment of the general public, satisfactorily. They asked that the Committee name be listed correctly on the documents and you made this minor change.

I am pleased to confirm that the Committee has given a favourable ethical opinion of the modified amendment on the basis described in the notice of amendment form and supporting documentation.

Approved documents

The documents reviewed and approved are:

Document	Version	Date
Advertisement	1	27 April 2013
Participant Information Sheet: Leaflet for supporting resources	1	08 April 2013
Participant Information Sheet	4	15 April 2013

Protocol	5	10 April 2013
Modified Amendment	AM01/1	12 April 2013

R&D approval

All investigators and research collaborators in the NHS should notify the R&D office for the relevant NHS care organisation of this amendment and check whether it affects R&D approval of the research.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

We are pleased to welcome researchers and R & D staff at our NRES committee members' training days – see details at http://www.hra.nhs.uk/hra-training/

12/LO/0795:	**Please quote this number on all correspondence**

Yours sincerely

pp

Professor David Bartlett
Chair

E-mail: nrescommittee.london-londonbridge@nhs.net

Enclosures: *List of names and professions of members who took part in the review*

Copy to: *Mrs Angela Williams, North Central London Research Network*
Mr. Dave Wilson

NRES Committee London - London Bridge

Attendance at Sub-Committee of the REC meeting on 19 April 2013

Name	*Profession*	*Capacity*
Professor David Bartlett	Honorary Consultant	Expert
Mr Brady Pohle	Legal Services Advisor	Lay Plus

Part 2

How will my data be kept confidential?

All information collected *during the course of the research will be kept strictly confidential. All the data collected from you will be stored in accordance with Data Protection laws and only the main researchers will have access to your information for the purposes of this study. Information will only be shared with your GP or other health professionals involved in your care if absolutely necessary for your health and well-being.*

All information about you *will be anonymised. You will be allocated a unique number or code which we will use to record and recall your personal details should you need to be contacted in the future. You will not be identified by name in any report concerning this study.*

Your salivary samples *will be kept anonymised and be destroyed immediately after analyses. The video files and the raw data (e.g. the paper copy of the questionnaires) will be deleted and destroyed within a year after they are recorded or collected.*

Will this study be published?

We plan to publish the results of this study *so that doctors and scientists can discover more about people's reactions during the recall of traumatic memories in psychological therapies. We hope these results will improve our ability to help PTSD patients.*

Who is organizing & funding this study? Who has reviewed this study?

This study is organised by *Chia-Ying Chou and Professor Chris R. Brewin at University College London, and is funded by the Department of Clinical, Educational & Health Psychology, UCL.*

The London Bridge Local Research Ethics Committee *has approved the study.*

What will happen if I do not want to, or am unable to, carry on with the study?

You are free to withdraw *from the study at any time and can do so without giving a reason. If you choose to withdraw (or are unable to carry on), all the identifiable data (e.g. video) will be destroyed immediately. However, the unidentifiable data collected up to that point would be entered into the results of the study and stored anonymously.*

Who do I speak to if I have further questions of if there is a problem?

If you would like to have any further information *at all, please do not hesitate to contact Chia-Ying Chou, who is the primary investigator.*

Chia-Ying Chou
Research Department of Clinical, Educational and Health Psychology, UCL, Torrington Place, London WC1E 7HB.
Email: chia-ying.chou.10@ucl.ac.uk
Tel: 020 7679 8279

Should you have any complaints, *you may speak to Professor Chris Brewin, who is supervising this project. If you believe you have been harmed in any way by the project, you may be covered under the NHS indemnity scheme.*

Professor Chris Brewin
Research Department of Clinical, Educational and Health Psychology, UCL, Gower Street, London WC1E 6BT
E-mail: c.brewin@ucl.ac.uk
Tel: 020 7679 5927

Camden and Islington NHS
NHS Foundation Trust

UCL

Information Sheet

Version 3: 03/09/12

Reactions to Remembering Traumatic Events

We would like to invite you to participate in this research project. Before you decide whether you want to take part, it is important for you to read the following information carefully.

Part 1 tells you the purpose of this study and what will happen to you if you take part.
Part 2 gives you more detailed information.

Part 1

What is the purpose of the study?

Psychological therapy *is effective in the treatment of post-traumatic stress disorder (PTSD). Its effect depends on patients' remembering of their traumatic events and full emotional engagement during this process. For this reason, we are examining emotional and bodily reactions while people recall traumatic memories. Characteristics of the trauma (e.g. how long ago it happened) and PTSD symptoms, which may influence a person's reactions to the recall, will also be investigated. The results from this study may contribute to more effective psychological therapy, in which patients' characteristics may be taken into consideration.*

Why have I been invited to participate in this study?

You *have been approached to volunteer for this study, as you have been diagnosed with PTSD and are waiting for or receiving treatment.*

Do I have to take part?

It is up to you *to decide whether or not to take part. If you do decide to participate, you will be asked to sign a consent form. You are free to withdraw at any time, without giving a reason. Choosing not to take part or to withdraw will not affect your medical care or legal rights in any way.*

What will happen if I take part?

Should you wish to take part *you will be invited to attend an interview on a pre-arranged date. If you are taking regular medication, we recommend that you continue doing so. However, please refrain from using any non-prescribed drugs or alcohol during the 24 hours before your interview, as these will affect the study results. The procedures should take about two hours. To compensate your time, you will be paid £10 per hour for taking part. Your travel expenses will also be reimbursed up to £5.*

What do I have to do?

If you agree to take part, *you will receive three questionnaires to complete before we meet. They are (1) Traumatic Life Event Questionnaire, which asks whether you have experienced certain types of trauma and, if yes, their frequency; (2) Post-traumatic Stress Diagnostic Scale, which asks about the post-traumatic symptoms related to your past traumatic experiences ; and (3) Peritraumatic Dissociative Experiences Questionnaire, which asks about your mental state while you were experiencing the trauma. It takes about 25 minutes to fill in these questionnaires.*

When we meet, *you will be asked to sign a consent form. We will then ask you about your PTSD symptoms. This will take about 25 minutes.*

Next, *you will be introduced to a test examining the effect of sound on relaxation (8 minutes). In this test, you might hear a loud noise. A heart rate recording sensor will be attached to your chest. After a resting period, you will be invited to talk about your memory of a traumatic experience for 10 to 15*

Finally, *you will be shown the video taken during the recall task and asked to identify particular mental states that you experienced while remembering the traumatic event.*

What are the possible advantages & disadvantages of taking part?

We cannot promise *there will be direct benefit to you personally. However, the information we get from this study may help improve the treatment of people with post-traumatic stress disorder (PTSD) or other anxiety disorders. You will be sent a copy of the final findings if you would like one (please inform the investigator).*

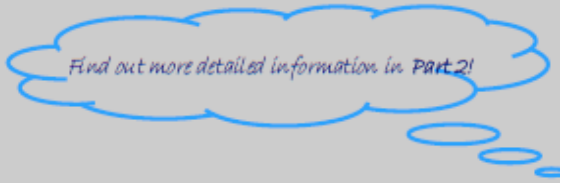
As *the procedure includes remembering traumatic memories and exposure to a loud noise, some people might find this unpleasant or distressing. You will be able to stop taking part at any point in the study if you wish to do so.*

What if there is a problem?

Any complaint *about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. The detailed information about this is given in Part 2.*

Will my participation in this study be kept confidential?

Yes. *We will follow ethical and legal practice and all information about you will be handled in confidence. The details are included in Part 2.*



Appendix 15: Consent form used in Chapter Five and Chapter Six

Consent Form (Version 3: 03/09/12)

PSYCHOLOGICAL & PHYSIOLOGICAL REACTIONS TO TRAUMATIC MEMORY RECALL

(student research project)

Investigators: Chia-Ying Chou, Prof. Chris R. Brewin

Thank you for considering taking part in this research.
Please read the following statements carefully and initial in the box at the end of each statement to give your consent.

1. I confirm that I have read and understand the information sheet dated 03/09/12 (version 3) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected. ☐

3. I agree that, for the purpose of this study, my heart rates will be recorded, salivary samples will be taken, and a part of my responses will be videotaped. ☐

4. I understand that data collected during the study (including the video) and relevant sections of my medical notes may be looked at by the researchers of this study ☐

5. I agree that if any important information about my physical or mental health arises from the study, my GP or my psychologist if appropriate will be informed. ☐

6. I understand that taking part in this study is for research and does not serve diagnostic or therapeutic purposes. ☐

7. I agree to take part in the above study. ☐

Participant:
PRINT.....Signed.....Date.....……..
Researcher*:*
PRINT.....Signed.....Date.....……..