

# **Affect without Recollection in Posttraumatic Stress Disorder**

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## **Thesis Declaration form**

I 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.

Signature



Name: Elizabeth Woodward

Date: 17.07.2020

## Overview

Re-experiencing symptoms in posttraumatic stress disorder (PTSD) include an affective or physiological response to a reminder of the trauma, not identifiable by the patient as a trauma memory. This has been termed ‘Affect without recollection’ (AWR; Ehlers & Clark, 2000). AWR has been anecdotally reported, but never formally defined or investigated. **Part 1** of the thesis provides a conceptual introduction to AWR in PTSD. Theoretical frameworks of reexperiencing in PTSD are reviewed, including cognitive (Ehlers & Clark, 2000) and neurobiological (Brewin, Dalgleish, & Joseph, 1996; Brewin, Gregory, Lipton, & Burgess, 2010) models of PTSD. From this, a possible definition of AWR and its theoretical underpinnings are proposed. **Part 2** of the thesis presents a novel empirical study of AWR, designed to investigate four key research questions: 1) Does affect without recollection exist in PTSD?; 2) What is the nature (phenomenology) of affect without recollection? 3) What predicts affect without recollection in PTSD? 4) Is affect without recollection improved with PTSD treatment? The results showed that AWR exists and is associated with cognitive behavioural responses. Cognitive processes such as dissociation, unhelpful response to intrusions, and negative appraisals predicted AWR, which was improved with PTSD treatment. The results have clinical and theoretical implications for understanding and treating reexperiencing symptoms in PTSD.

## **Impact Statement**

The results of this thesis could be of use both academically and clinically. There has been a call from researchers in the field for more studies focused on understanding reexperiencing symptoms in posttraumatic stress disorder (PTSD), which have been termed the hallmark of the disorder. Reexperiencing symptoms are still relatively poorly understood, and yet have a significant impact on the lives of people with PTSD, and have been shown to predict and maintain PTSD symptoms (Michael, Ehlers, Halligan, & Clark, 2005). This thesis explored a previously proposed, but never formally defined or investigated, form of reexperiencing termed affect without recollection (Ehlers & Clark, 2000). Affect without recollection is the experience of an emotional or physiological reaction from a traumatic event that is not recognised by the individual as a trauma memory and can be a very confusing and frightening experience. Although reported anecdotally by patients, prior to this thesis it had not been measured and therefore it was unknown whether it existed, what its consequences were, and whether current PTSD treatments are effective in resolving this symptom. The empirical study conducted as part of this thesis provided the first evidence of the existence of AWR, its predictive factors, its consequences for the individual, and the effects of PTSD treatment. The results have the potential for significant clinical impact, including helping clinicians to identify, understand and treat this symptom. It may also have implications for individual sufferers to understand their experiences after a traumatic event. Academically, it contributes to understanding the impact of trauma and PTSD on memory. Methodologically, it adds to the body of literature on the usefulness of ambulatory self-report measures on understanding the daily experiences of people suffering from mental health conditions. It demonstrated the use of smartphone applications for assessing PTSD symptoms over time, which was found to be highly

acceptable by participants (reflected in the high completion rates). Finally, it presents modelling techniques which may be useful for the analysis of ecological momentary assessment data in a clinically meaningful way. In sum, it is hoped that the results of this thesis will have implications both clinically and academically.

## Table of Contents

<b>Thesis Declaration form</b> .....	<b>2</b>
<b>Overview</b> .....	<b>3</b>
<b>Impact Statement</b> .....	<b>4</b>
<b>Table of Contents</b> .....	<b>6</b>
<b>Table of Figures</b> .....	<b>8</b>
<b>Table of Tables</b> .....	<b>8</b>
<b>Acknowledgments</b> .....	<b>9</b>
<b>Part 1: Conceptual Introduction</b> .....	<b>10</b>
<b>Abstract</b> .....	<b>11</b>
<b>Introduction</b> .....	<b>12</b>
<b>Posttraumatic Stress Disorder</b> .....	<b>13</b>
Defining Trauma .....	13
Posttraumatic stress disorder: definition and epidemiology .....	14
Cognitive model of PTSD.....	16
<b>Memory in PTSD</b> .....	<b>18</b>
Memory in PTSD .....	19
<b>Reexperiencing symptoms</b> .....	<b>21</b>
Defining reexperiencing.....	21
Specificity of reexperiencing to PTSD .....	23
Mechanisms underpinning reexperiencing in PTSD .....	26
Cognitive models of reexperiencing in PTSD .....	27
Neurobiological models of reexperiencing in PTSD .....	35
<b>Affect without recollection</b> .....	<b>39</b>
Defining affect without recollection .....	40
Evidence of affect without recollection .....	41
Possible mechanisms underpinning affect without recollection .....	42
Disjointed from the autobiographical memory base .....	42
Priming and associative learning .....	43
Appraisals and coping strategies.....	44
Dissociation.....	45
<b>Summary and aims of the thesis</b> .....	<b>46</b>
Summary .....	46
Theoretical predictions.....	47

Aims of the thesis.....	48
<b>References</b> .....	<b>50</b>
<b>Part 2: Empirical Paper</b> .....	<b>57</b>
<b>Abstract</b> .....	<b>58</b>
<b>Introduction</b> .....	<b>59</b>
<b>Method</b> .....	<b>63</b>
<b>Results</b> .....	<b>75</b>
<b>Discussion</b> .....	<b>90</b>
<b>References</b> .....	<b>104</b>
<b>Part 3: Critical appraisal</b> .....	<b>109</b>
Introduction.....	110
Reflections on selection of subject area.....	110
Reflections on conceptual introduction .....	111
Establishing research questions .....	113
Reflections on methodology .....	114
Reflections on analysis process .....	116
Reflections on limitations and changes.....	118
Conclusion .....	119
<b>References</b> .....	<b>120</b>
<b>Appendices</b> .....	<b>121</b>
<b>Appendix A</b> .....	<b>121</b>
Life event checklist.....	121
<b>Appendix B</b> .....	<b>122</b>
Affect without Recollection smart-phone application .....	122
<b>Appendix C</b> .....	<b>123</b>
Participant information sheet .....	123

## Table of Figures

<b>Figure 1</b> Recruitment flow-chart for PTSD patients from referral into the study to post-treatment assessment (n = 23).....	64
<b>Figure 2</b> Cluster dendrogram of emotions and clusters in the PTSD sample (n = 28); extracted through agglomerative hierarchical cluster analysis. Red = Cluster 1; Blue = Cluster 2 (see Table 6). Height = cophenetic distance (similarity/dissimilarity between objects/clusters).....	71
<b>Figure 3</b> Mean emotions ratings and sum scores (PTCI, TDQ, RIQ) for participants in cluster 1 (n = 3) and cluster 2 (n = 25).....	84
<b>Figure 4</b> Mean emotional intensity for PTSD participants reporting at least one AWR at pre- (n = 28) and post-treatment (n =8). .....	88

## Table of Tables

<b>Table 1</b> Demographic information and PTSD and depression symptom severity for each group, and statistical comparison of groups on distributions ( $\chi^2$ ) and mean scores (F or t). .....	76
<b>Table 2</b> Baseline demographic information and test-statistics (Mann Whitney) comparing participants who attended both sessions, and those who dropped out from the study after Session 1. ....	77
<b>Table 3</b> Descriptive and test-statistics for each group, for participants who attended both Session 1 and Session 2. ....	78
<b>Table 4</b> Frequency of trigger type by group (N = number of times this was identified as a trigger. % = valid percent of overall known triggers), for participants who experienced AWR at Session 1.....	81
<b>Table 5</b> Mean (SD) of emotional intensity (0-100) reported over 7 days (for those reporting AWR) at Session 1. ....	82
<b>Table 6</b> Emotion clusters at Session 1 for PTSD group (n = 28). ....	83
<b>Table 7</b> Results of linear-mixed models predicting cognitive behavioural-responses from emotion clusters. ....	85

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# **Part 1: Conceptual Introduction**

## **Understanding Affect without Recollection in Posttraumatic Stress Disorder**

## **Abstract**

It has been suggested that re-experiencing symptoms may take the form of an affective (emotional) or physiological response to a reminder of the trauma, which is not identifiable by the patient as a trauma memory, termed ‘affect without recollection’ (Ehlers & Clark, 2000). This concept has been proposed but not yet clearly defined or investigated. Researchers have developed detailed theoretical frameworks of the mechanisms underpinning reexperiencing in PTSD (Brewin et al., 1996; 2010; Ehlers & Clark, 2000). This conceptual introduction will review these frameworks and apply them to AWR. Drawing on clinical (Ehlers & Clark, 2000) and neurobiological models (Brewin et al., 1996; 2010) of PTSD, it is suggested that AWR could represent a predominantly perceptual memory that is highly disjointed from the autobiographical memory base, to the extent that emotions from the trauma are experienced in the absence of a trauma memory. The role of perceptual priming, associative learning and dissociation is considered in the development and triggering of AWR, and theoretical predictions and research questions are proposed.

## Introduction

People with posttraumatic stress disorder (PTSD) experience the trauma as if it is happening again. This has been termed a flashback memory, a re-experiencing symptom of PTSD. It is considered one of the hallmark symptoms of PTSD and is a defining feature distinguishing PTSD from other psychiatric conditions, including those with intrusions (Brewin, 2014; Bryant, O'Donnell, Creamer, McFarlane, & Silove, 2011). A number of researchers have provided detailed theoretical frameworks to understand the occurrence and nature of flashbacks in PTSD. However, the quality of trauma memories is still poorly understood due to limited empirical research. Research has shown that flashbacks and their nature (such as a sense of 'nowness' i.e. that the event is happening again) maintain PTSD symptoms, predict the course of the disorder (Michael et al., 2005), and decrease with successful PTSD treatment (Hackmann, Ehlers, Speckens, & Clark, 2004; Nijdam, Baas, Olf, & Gersons, 2013; Speckens, Hackmann, Ehlers, & Cuthbert, 2007), and therefore have significant clinical impact. This emphasises the need to better understand flashbacks in PTSD, which could help to identify treatment targets and in turn have enormous clinical benefits. Given that it is problematic to ask participants retrospectively about flashback experiences, a handful of studies have attempted to better understand re-experiencing in PTSD through daily diary studies (Kleim, Graham, Bryant, & Ehlers, 2013), which have provided useful information on the daily experiences of reliving in PTSD, free from recall bias. However, there is still more to be done, particularly on anecdotally reported but understudied aspects of re-experiencing, such as affect without recollection (Ehlers & Clark, 2000). This refers to symptoms such as a sudden emotional (affect) or physiological experience, such as a feeling of terror, *without* a recollection of the event. This has been reported anecdotally by patients with PTSD, but has not yet been clearly

defined, investigated or measured. Nor have the possible theoretical underpinnings been described or fully explored.

This introduction will explore the concept of affect without recollection and its possible theoretical underpinnings. It will begin by describing PTSD and associated memory characteristics, followed by a description of re-experiencing symptoms in PTSD and proposed underlying mechanisms. Finally, affect without recollection (AWR) will be described and theoretical underpinnings will be proposed, in line with the previously outlined literature.

## **Posttraumatic Stress Disorder**

### **Defining Trauma**

Posttraumatic stress disorder (PTSD) is mental disorder that may develop following exposure to a traumatic event. A traumatic event is strictly defined in the diagnostic and statistical manual of mental disorders (5<sup>th</sup> edition; DSM-5, American Psychiatric Association, 2013). It defines trauma as ‘exposure to actual or threatened death, serious injury, or sexual violence’ and specifies that this may occur through direct experience, witnessing it happening to another, learning about it happening to another person (for death it specifies that the cause must have been violent and accidental), or by experiencing repeated exposure to details of traumatic events, for example through work (but not through television exposure) (APA, 2013). Trauma under the DSM-5 definition is referred to as a Criterion A event. Examples of traumatic events include, rape/sexual assault, road traffic accidents, and natural disasters. In a recent survey of 2,953 US adults, 90% were estimated to have been exposed to at least one traumatic

event in their lifetime, and most had experienced more than one event, with three events estimated as the most common number of traumatic events experienced (Kilpatrick et al., 2013). A recent survey conducted in the UK in 2014 has yielded lower estimates, where a third (31%) of UK adults reported having experienced at least one Criterion A traumatic event in their lifetime, and prevalence was similar for women (31%) and men (32%) (Fear, Bridges, Hatch, Hawkins, Wessely, 2016).

### **Posttraumatic stress disorder: definition and epidemiology**

Following a traumatic event, a minority of people may go on to develop PTSD. PTSD is characterised by four clusters of symptoms; re-experiencing symptoms associated with the trauma (Criterion B); avoidance of stimuli associated with the trauma (Criterion C); negative alterations in cognitions and mood (Criterion D); and alterations in arousal (Criterion E). To qualify for a diagnosis of PTSD, the trauma must meet the definition for a Criterion A event, symptom(s) in each cluster must be endorsed, must have persisted for more than one month since the traumatic event, and must be associated with clinically significant distress and impairment.

Specifically, the symptom clusters specified in the DSM-5 (APA, 2013) include the following symptoms:

**Criterion B:** Re-experiencing (endorsement of at least one symptom, occurring at least twice a month):

- B1. Recurrent, involuntary distressing memories
- B2. Recurrent, distressing dreams with content/affect related to the trauma

- B3. Dissociative reactions or flashbacks where the trauma feels like it is happening again
- B4. Intense and prolonged distress in response to external or internal cues associated with the traumatic event
- B5. Physiological reactions to cues associated with the traumatic event

**Criterion C:** Avoidance (endorsement of at least one symptom):

- C1. Avoidance of distressing memories, thoughts or feelings associated with the trauma, at least twice a month.
- C2. Avoidance of people or places or things associated with the trauma, at least twice a month.

**Criterion D:** Negative alterations in cognition and mood (endorsement of at least two symptoms, beginning or getting worse with the trauma)

- D1. Inability to remember important aspects of the trauma (not due to drugs, alcohol, medication or injury)
- D2. Persistent, exaggerated negative beliefs about oneself, the world or others
- D3. Persistent, distorted cognitions about the cause of the traumatic event, leading to misplaced blame (self/others)
- D4. Negative emotional state (fear, horror, anger, guilt, or shame)
- D5. Diminished interest in significant activities
- D6. Feeling detached from others
- D7. Difficulty experiencing positive emotions, like love or happiness

**Criterion E:** Altered arousal (endorsement of at least 2 symptoms, beginning or getting worse with the trauma)

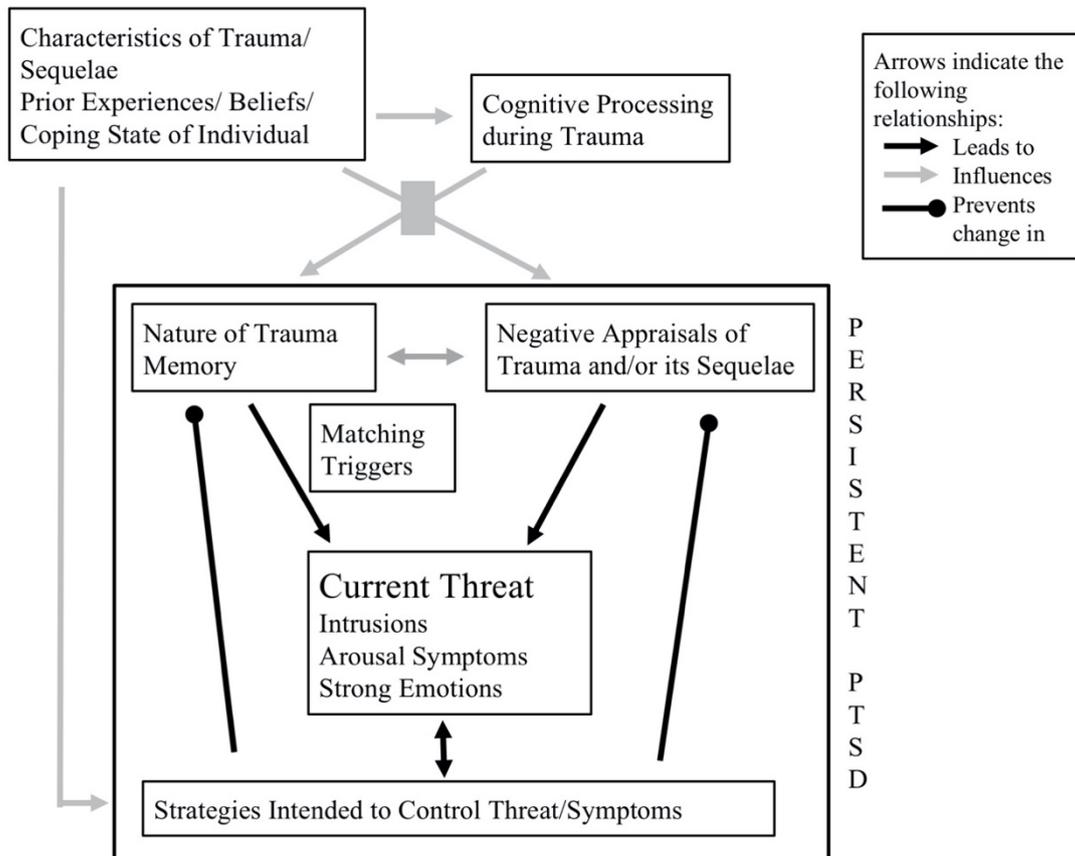
- E1. Irritable behaviour, including anger, without much provocation
- E2. Reckless or destructive risk-taking behaviour, with risk of being harmed
- E3. Hypervigilance, i.e., alert, watchful, or on guard
- E4. Exaggerated startle response
- E5. Difficulty concentrating
- E6. Problems falling or staying asleep, at least twice a month with at least 30 minutes sleep loss

A recent epidemiological study of American adults found that following a traumatic event 8% developed PTSD within their lifetime (related to that event). PTSD prevalence was higher in women (11%) than in men (5%). The type of traumatic event affected the likelihood of developing PTSD, which was most likely after interpersonal violence, and combat. Likelihood of developing PTSD increased with the number of traumatic events (Kilpatrick et al., 2013). The development of other psychiatric conditions was also found to be associated with the experience of a traumatic event, such as major depression, anxiety disorders and substance abuse (Ehring, Ehlers, & Glucksman, 2008; Kessler, Chiu, Demler, & Walters, 2005).

### **Cognitive model of PTSD**

Ehlers and Clark (2000) proposed a cognitive model to account for the development and persistence of PTSD (Figure 1). They proposed that unhelpful appraisals (e.g., ‘I have to be on guard all the time’ or ‘The trauma showed that I am inadequate’) induce a sense of current threat that maintains PTSD symptomatology such as hyperarousal

and hypervigilance, and motivates maladaptive coping strategies (cognitive and behavioural) that in turn prevents a change in appraisals and nature of the trauma memory (Ehlers & Clark, 2000; Ehlers, Clark, Hackmann, McManus, & Fennell, 2005).



**Figure 1.** Ehlers and Clark's (2000) cognitive model of PTSD.

A second source of the current threat, of particular significance for this thesis, is thought to be the disjointed nature of trauma memories that leads to easy triggering of intrusive memories with a flashback-quality, i.e. the experience that the memories are happening in the present rather than the past (Ehlers & Clark, 2000). These are also proposed to maintain PTSD symptoms as they contribute to a continuing hyperarousal and

hypervigilance (as the trauma memories intrude unexpectedly, and induce a feeling that the trauma is constantly happening again), as well as motivating maladaptive coping strategies to manage the memories such as thought suppression and avoidance of triggers. These prevent the trauma memory from being processed, and so flashbacks continue (see section ‘Cognitive models of reexperiencing in PTSD’, page 27), and also prevent change in appraisals, such as ‘I would go mad if I thought about the trauma’ (Ehlers & Clark, 2000). In their model, Ehlers and Clark (2000) coined the term ‘Affect without Recollection’ (p. 324) and classified it as a re-experiencing symptom (Criterion B). Therefore, the model proposes that Affect without Recollection (AWR) would contribute to a sense of current threat, which maintains other PTSD symptomatology (hyperarousal, hypervigilance and maladaptive coping strategies).

Other models of PTSD have been proposed, however Ehlers and Clark’s (2000) cognitive model of PTSD is most relevant to the thesis as AWR was first described in this model, and so other clinical models of PTSD will not be reviewed here. Brewin, Dagleish and Joseph’s (1996) neurobiological model of re-experiencing symptoms in PTSD will be described in the section on mechanisms underlying re-experiencing symptoms in PTSD (p. 35).

### **Memory in PTSD**

PTSD has long been described as a disorder of memory (Brewin, 2003; McNally, 2003; van der Kolk, 2007). This is due to the co-existence in PTSD of 1) involuntary memories of the traumatic event that are re-experienced as if the event is happening again (acting or feeling the same way), and 2) difficulties intentionally retrieving ordinary episodic memories of the trauma. Clinical theories of PTSD have drawn in

part on existing memory theory (Brown & Kulik, 1977) to account for these memory difficulties. For example, most clinical theories of PTSD share the assumption that re-experiencing symptoms can be understood by general memory processes described in cognitive psychology and neuroscience (e.g., Brewin et al., 1996; 2010; Ehlers & Clark, 2000). The literature on memory in PTSD is vast and reviewing all of it would be beyond the scope of this thesis. Therefore, this section will provide only a brief overview of some memory phenomena in PTSD that are significant to this thesis, as a background to understanding possible memory mechanisms underpinning reexperiencing.

## **Memory in PTSD**

### *Disorganisation and disjointedness*

Studies have shown difficulties in the intentional recall of trauma memories in PTSD, such as gaps in the memory, a lack of coherence and difficulty remembering the order of events (Foa, Riggs, Dancu, & Rothbaum, 1993; Halligan, Michael, Clark, & Ehlers, 2003) which has been termed ‘disorganisation’ (Ehlers, Hackmann, & Michael, 2004). Studies using methods such as subjective self-report (Halligan et al., 2003) and objective ratings of participants’ written trauma narrative (Jones, Harvey, & Brewin, 2007) have shown more disorganised trauma memories in trauma survivors with PTSD compared to those without. It is under debate whether disorganisation is specific to trauma memories (Ehring, 2004; Halligan et al., 2003), or whether people with PTSD have more disorganised memories more generally (Jelinek, Randjbar, Seifert, Kellner, & Moritz, 2009).

The worst moments of a trauma have been found to be disjointed from other information in autobiographical memory (Ehlers et al., 2004). For example, people may remember the moment that they thought they were going to die in the absence of the later information that they survived. This is supported by studies showing that the moments from the trauma that are reexperienced may be more disorganised than other moments from the trauma memory (Evans, Ehlers, Mezey, & Clark, 2007b; Jelinek et al., 2010). Recent experimental studies have shown that exposure to analogue trauma leads to memory-disjointedness of the worst moments, suggesting a causal effect of trauma (Sachschaal, Woodward, Wichelmann, Haag, & Ehlers, 2019). It is proposed that disjointedness of trauma memories adds to understanding the triggering and content of reexperiencing symptoms in PTSD (Ehlers, 2015).

#### *Non-trauma memories*

People with PTSD have also shown difficulties remembering specific non-trauma autobiographical events (see Moore & Zoellner, 2007 for review), unrelated to differences in general intelligence (Schönfeld & Ehlers, 2006). This has been termed ‘over-general memory’ (Williams et al., 2007) and has been evidenced in trauma survivors with PTSD but not those without (Bryant, Sutherland, & Guthrie, 2007; Schönfeld, Ehlers, Böllinghaus, & Rief, 2007), and shown to predict subsequent PTSD development, partly mediated by perceived permanent change (Kleim & Ehlers, 2008). While over-general memory is found in depression (Dalgleish et al., 2007), which is highly comorbid with PTSD, it has also been specifically linked to trauma history in studies showing over-general memory in depressed women with but not without a history of childhood abuse (Kuyken & Brewin, 1995).

### *Summary*

In sum, there is some evidence that people with PTSD have difficulties intentionally retrieving memories of their trauma, however not all studies have found this. Research also points to difficulties with autobiographical memory more generally in PTSD. Difficulties appear to be most pronounced for the worst moments of the trauma, which are relevant for understanding reexperiencing symptoms.

## **Reexperiencing symptoms**

### **Defining reexperiencing**

Re-experiencing symptoms are one of the hallmark symptoms of PTSD. They have been conceptualised as spontaneous, emotionally-charged, intrusive memories of the trauma in the form of visual or sensory flashbacks, which are separate from other contextual information in the autobiographical memory system (Ehlers et al., 2004; 2002). Reexperiencing symptoms have recently been formally defined in the DSM 5 (APA, 2013) as intrusive memories of a traumatic event (or moments from the trauma) that are relived as if they are happening again in the present (flashbacks). This element of reliving is now seen as existing on a continuum from losing all contact with the present surroundings (most extreme, now termed a dissociative flashback) to including some element of reliving the event in the present, even if only briefly. While they are memories of an autobiographical experience, reexperiencing in PTSD appears to lack a key feature of autobiographical memories, termed “autonoetic awareness” (Tulving, 2002), that is, the awareness that a memory of the self is happening in the past.

Research has investigated the contents of reexperiencing. It has shown that reexperiencing can include a range of different experiences. It often consists of a small number of scenes from the trauma that are repeatedly experienced (Brewin et al., 2010). They are typically vivid, perceptually detailed (Ehlers & Steil, 1995; van der Kolk & Fisler, 1995) distressing, sensory impressions of moments from the trauma. They are often multi-sensory and are usually visual (Ehlers et al., 2002, Ehlers & Steil, 1995, Hackman et al., 2004), but not always. For example, a car accident survivor might reexperience the flash of headlights before they were hit, or an assault survivor might reexperience the attackers face before the assault. Studies have shown that involuntary memories often relate to the most emotional parts of a trauma (Berntsen & Rubin, 2008; Holmes, Grey, & Young, 2005). However, it is important to note that these ‘worst moments’ are subjectively defined. For example, studies have shown that reexperiencing often includes moments that signal the onset of the trauma rather than the moment the traumatic outcome happened, such as the sound of footsteps before a guard entered the cell, rather than the torture itself. This has been conceptualised as a ‘warning signal’ (see Ehlers, 2015). This is consistent with associative learning theory that would suggest that sensory information in the trauma hold the information value of a conditioned stimulus (CS) which predict the unconditioned stimulus (UCS) i.e. the trauma (Rescorla, 1988). Studies with PTSD patients have shown that the CS is more often reexperienced than the UCS after trauma, as it has functional significance that *predicted* the worst moment of the trauma (Evans, Ehlers, Mezey, & Clark, 2007a; Hackmann et al., 2004). Therefore, while they are linked to the most emotional parts of the trauma, they are often of the perceptions just before the traumatic outcome, that indicated something terrible was about to happen, i.e. acting as a ‘warning signal’ (see Ehlers, 2015).

Importantly for this thesis, reexperiencing can also include phenomena that are not recognised as a memory. This can include reexperiencing emotions or physiological reactions from the trauma, such as an intense feeling of fear without knowing why. This has been termed ‘affect without recollection’ (AWR; Ehlers & Clark, 2000), which will be described in more detail in section (‘Affect without recollection’, page 38).

Researchers have investigated the specificity of re-experiencing symptoms to PTSD and developed theories of the development and maintenance of flashbacks (Brewin et al., 2010; Ehlers & Clark, 2000). The following section will review this literature.

### **Specificity of reexperiencing to PTSD**

Intrusive/unwanted memories themselves are not specific to PTSD (Brewin, 2014; Brewin et al., 2010) and have been found in other psychological disorders such as depression, panic, and Obsessive Compulsive Disorder (Speckens et al., 2007). There has also been some debate over whether flashbacks are normal autobiographical memories (Rubin, Berntsen, & Bohni, 2008a). However, clinical observations and studies disagree (see Brewin, 2015; Ehlers, 2010). There is arguably now consistent evidence that flashbacks are distinct from autobiographical memories for the same event (Hellawell & Brewin, 2002). For example, Hellawell and Brewin (2002) found that participants struggled to write a narrative of a section of a trauma memory that they had identified as a flashback and showed involuntary motor movements while doing so. Studies have also shown that sections of trauma narratives that are of a flashback are rated as more negative and arousing (Brewin, Huntley, & Whalley, 2012), accompanied by heart rate increase (La Marca, Steptoe & Brewin, 2018) and contain

more core PTSD emotions such as fear, helplessness and horror (Hellawell & Brewin, 2004) compared to narratives of sections that are not experienced as a flashback.

Consistent with this, systematic comparisons of *intrusive trauma memories* in people with and without PTSD have shown important differences which distinguish the intrusive memories in PTSD as ‘flashbacks’ rather than just an intrusive trauma memory. These distinguishing features are described by Ehlers (2010) and are as follows:

**1) a sense of ‘nowness’**, i.e. a sense that the trauma is happening again in the here and now, without the knowledge that it is in the past (Ehlers et al., 2004). People with PTSD describe experiencing their intrusive memories of the trauma with a sense of nowness that is not reported by trauma-survivors without PTSD (Michael et al., 2005). Further they report AWR (Ehlers & Clark, 2000) which are emotions and behaviours from the trauma without a conscious memory of the trauma (e.g., suddenly feeling terrified without knowing why). Therefore, people with PTSD report memories without auto-noetic awareness, a distinct feature of regular episodic memories (Tulving, 2002).

**2) lack of context:** intrusive memories in PTSD lack contextual information and appear disjointed from other autobiographical memories that contain relevant information, such as that the person survived the trauma (Ehlers et al., 2004; Michael et al., 2005). This relevant information is necessary to update the original impressions from the trauma (e.g., changing the impression from ‘I’m going to die’ to ‘I thought I was going to die but I know now that I survived’). Difficulties with contextualising autobiographical trauma memories is supported by neuroimaging research. For

example, context in autobiographical memories appears to be hippocampally-mediated, and hippocampal volume reductions are one of the most replicated structural findings in people with PTSD (Hull, 2002; Woon & Hedges, 2008). Smaller hippocampal volume has also been found in twins of veterans with PTSD, suggesting this may represent a genetic vulnerability (Gilbertson et al., 2002; Pitman, 2006).

**3) ease of triggering by matching cues:** in PTSD intrusive memories are easily triggered by a wide range of situations and stimuli, some of which are not identified by the individual as triggers, and some without an obvious connection with the trauma. However, on closer examination these triggers typically share *sensory* similarities with stimuli in the trauma (e.g., colour) which were temporally related to the traumatic event (see Ehlers et al., 2002). Research has shown that whether or not a memory is reexperienced will depend on the available environmental cues which are uniquely associated with the memory (Berntsen, Staugaard, & Sørensen, 2013). Experimental studies have demonstrated that perceptual cues (particularly negative ones) are more likely to lead to involuntary perceptual memories than verbal cues (Brewin & Langley, 2019).

**4) distress:** people with PTSD report more distress in response to their intrusive memories compared to trauma-survivors without PTSD (e.g., Michael et al., 2005).

In line with evidence distinguishing intrusive trauma memories in PTSD, there has been a call for more consistent terminology which discriminates between ‘involuntary autobiographical memories’ (an everyday memory phenomena); ‘intrusive memories’ (involuntary memories with distressing content) and flashbacks (involuntary memories

involving re-experiencing distressing events in the present, thought to be specific to PTSD) (Kvavilashvili, 2014). The terms flashbacks and reexperiencing symptoms will be used throughout this thesis.

### **Mechanisms underpinning reexperiencing in PTSD**

Flashbacks immediately after a trauma are not in themselves considered an indicator of PTSD. In the initial weeks after a traumatic experience, flashbacks are common, and in normal recovery will decrease over time. However, in PTSD, flashbacks persist for more than a month (APA, 2013) and models propose they contribute to the maintenance of PTSD (Ehlers & Clark, 2000). Consistent with this, flashbacks have also been found to predict the course of PTSD over and above initial PTSD symptoms (Kleim, Ehlers, & Glucksman, 2007; Michael et al., 2005).

Complimentary cognitive (Ehlers and Clark, 2000) and neurobiological (Brewin et al., 1996; 2010) models of mechanisms underpinning flashbacks in PTSD have been proposed. Both models suggest that trauma memories in PTSD can be understood by generic memory processes that have been previously studied and identified in cognitive psychology and neuroscience, as well as systematic differences in the encoding of the trauma, between trauma-survivors with and without PTSD. Both models propose that the combination of these explain reexperiencing symptoms. Other theories exist that account for the involuntary retrieval of memory content that is recognised as a trauma memory but does not account for the aspects of reexperiencing that are not recognised as a memory such as AWR (Berntsen, 2009; Rubin, Boals, & Berntsen, 2008b), and excludes involuntary auditory content and visual imagery (Bernsten, 2009). These aspects are accounted for by Ehlers & Clark's (2000) and Brewin's (1996; 2010)

models which aim to explain the range of reexperiencing in PTSD, including those aspects not recognised by the individual as a trauma memory (such as AWR). Ehlers and Clark (2000) and Brewin's (1996; 2010) models will be reviewed in the following section.

### *Cognitive models of reexperiencing in PTSD*

Ehlers and Clark's (2000) model of PTSD proposed that intrusive reexperiencing is maintained by **1) memory processes responsible for the easy triggering of intrusive memories, 2) the individual's interpretation of their trauma memories, and 3) their cognitive behavioural responses to trauma memories.** Within memory processes, three basic memory processes are proposed that could account for reexperiencing and the ease with which it is triggered in PTSD. These are: **a) strong perceptual priming; b) strong associative learning, and c) poor memory elaboration** (binding with other information in autobiographical memory) (see also Ehlers, 2010). The model in particular emphasises priming and associative learning, as these are memory processes that facilitate cue-driven retrieval of the trauma or moments from the trauma. It is proposed that poor memory elaboration leads to a failure to inhibit cue-driven retrieval. These mechanisms are proposed to be general memory mechanisms that are not specific to trauma memories and aim to account for the full phenomenology of reexperiencing in PTSD, such as the involuntary nature of this memory, and experiences not recognised as trauma memories (AWR). Experimental evidence supports the role of these processes in reexperiencing.

### *Memory processes: Perceptual priming*

Perceptual priming is a form of implicit memory where processing of a stimulus (or related stimulus) is facilitated because it has been processed before (Bowers & Turner, 2003). Priming has been shown to be stable for up to 17 years (Mitchell, 2006) and can transfer to other contexts (McKone & French, 2001). Ehlers and Clark (2000) propose that people with PTSD may have heightened priming for trauma-related stimuli, and that enhanced perceptual priming for stimuli during (and in the aftermath of) a trauma may increase risk of the development of PTSD. Heightened perceptual priming for stimuli present during the trauma would result in a processing advantage for these and similar stimuli, with the consequence that these stimuli are more likely to be noticed in the environment and trigger trauma memories through unintentional, cue-driven retrieval (Ehlers & Clark, 2000; Ehlers, Ehring, & Kleim, 2012a). This is supported by prospective studies showing enhanced perceptual priming in trauma-survivors for trauma-related words predicted the later development of PTSD symptoms (Ehring & Ehlers, 2011; Kleim, Ehring, & Ehlers, 2011; Michael et al., 2005). Cross-sectional studies also offer support, for example Lyttle et al., (2010) showed that people with PTSD showed greater perceptual priming (word-stem completion test) than conceptual priming (word-cue association task) compared to trauma-survivors without PTSD. This is also supported by several experimental studies which have induced perceptual priming by pairing stimuli with traumatic pictures and showed stronger perceptual priming than those paired with neutral pictures (Ehlers, Ehring, & Kleim, 2012a; Ehlers, Mauchnik, & Handley, 2012b; Ehlers, Michael, Chen, Payne, & Shan, 2006; Michael & Ehlers, 2007) which predicted more involuntary intrusions over the following months (Arntz, de Groot, & Kindt, 2005; Ehlers et al., 2006; Ehlers, Mauchnik, & Handley, 2012b; Michael & Ehlers, 2007; Sündermann, Hauschildt, &

Ehlers, 2013). Finally, some (Lyttle, Dorahy, Hanna, & Huntjens, 2010; Michael & Ehlers, 2007) but not all (Sündermann et al., 2013) studies have shown greater state dissociation (the feeling that the world is unreal, alterations in experience of self) during exposure to traumatic stories was associated with stronger perceptual priming. Together these findings demonstrate how perceptual priming can account for triggering of reexperiencing symptoms by a range of stimuli (directly or indirectly related) in the environment (see Ehlers, Ehring & Kleim, 2012).

*Memory processes: Associative learning*

Associative learning is emphasised in many information-processing models of PTSD to explain the triggering of emotional and physiological responses people with PTSD exhibit in response to trauma reminders (Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989). From Pavlovian conditioning theory it is proposed that a trauma (unconditioned stimulus; US) triggers an unconditioned emotional response (UR; such as fear) which becomes paired with stimuli in the environment such as smells, sounds and sights (conditioned stimuli; CS) that are present at the same time (temporally associated). As a result of this pairing, the cues (CS) can then trigger the same emotional/physiological response (conditioned response; CR) in the absence of the US (trauma). Therefore, reexperiencing can be understood as CRs which persist for a long time after trauma. Associative learning models of PTSD also suggest that the fear response becomes generalised more broadly to other stimuli that were not present at the time of the trauma, but share perceptual features with stimuli that were present (e.g., colour), which accounts for why a wide range of stimuli can trigger reexperiencing symptoms in the form of emotional and physiological responses, and the persistence of reexperiencing symptoms after a trauma. It is also proposed that failure to extinguish

the original fear response due to avoidance of the stimuli or impairment in extinction learning, may play a role in maintaining reexperiencing in PTSD (Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007), and may serve as a vulnerability to the development of PTSD (Lommen, Engelhard, Sijbrandij, van den Hout, & Hermans, 2013). There is a large body of evidence to support the role of associative learning in reexperiencing in PTSD. For example, associative learning theories would predict that people with PTSD show enhanced physiological responses to trauma reminders, compared to trauma-survivors without PTSD. This is supported by a meta-analysis of cross-sectional studies showing that physiological responses (such as heart rate, skin conductance, EMG and blood pressure) to idiographic and standardised trauma cues could identify PTSD participants with up to a 77% specificity and 91% sensitivity (Pole, 2007). Several prospective studies have found that enhanced physiological responses to trauma reminders shortly after a trauma predicts PTSD three months later (Ehlers et al., 2010; Elsesser, Sartory, & Tackenberg, 2004; 2005; Sundermann, Ehlers, Boellinghaus, Gamer, & Glucksman, 2010). These studies demonstrate strong learned fear responses to trauma reminders and suggest that this physiological responsiveness is not merely a symptom of PTSD but may also play a role in the development of persistent PTSD. Consistent with this, there is evidence that pre-trauma ease of acquisition of fear conditioning, or 'enhanced conditionability' (Wegerer, Blechert, Kerschbaum, & Wilhelm, 2013) and reduced fear extinction learning predicts the later development of PTSD (Blechert et al., 2007; Guthrie & Bryant, 2006; Lommen et al., 2013). These may indicate susceptibility factors to developing PTSD. Some studies have also shown a relationship between greater generalised fear response to stimuli not present at the time of the trauma and later PTSD development (Ehlers et al., 2010; Sundermann et al., 2010). In line with this, experimental studies have shown that

participants with PTSD show greater conditioned responses to neutral stimuli paired with trauma reminders and slower fear extinction than trauma-survivors without PTSD, suggesting that second-order conditioning such as this may act as a mechanism for stimulus generalisation (Wessa & Flor, 2007). This has also been shown to occur even if the individual is not aware of the link between the neutral stimulus and trauma reminders (Michael et al., 2010). This could account for the broad triggering of reexperiencing in PTSD by reminders that are unrelated/not obviously related to the trauma. There is also evidence that people with PTSD show reduced contingency awareness (i.e. awareness that the CS was paired with the US) compared to trauma-exposed controls. However, it is unclear whether this is due to dissociation and anxiety during learning, or due to impairments in memory in PTSD (Blechert et al., 2007). Functional neuroimaging studies also support the involvement of both priming and associative learning in PTSD, showing activation in areas involved in priming and associative learning (retrosplenial cortex) in response to trauma-related stimuli, in participants with PTSD (Sartory et al., 2013). Similarly, studies have also shown increased hypersensitivity in brain areas thought to assess salience and respond to threat, such as the amygdala (Liberzon, 2006), and a dysfunctionality in higher cortical structures (such as the medial pre-frontal cortex) that may regulate emotional responses and extinction of fear conditioning. Associative learning can also account for the ‘warning signal hypothesis’ (Ehlers et al., 2002) of the content of reexperiencing in PTSD (Evans et al., 2007; Hackmann et al., 2004), as introduced on page 22. In this hypothesis Ehlers et al., (2002) suggested that the CS has *predictive* informational value which triggers sensory impressions that indicate the onset of the worst moments of the trauma (unconditioned stimulus). Therefore, the CS (e.g., headlights) is reexperienced, rather than the UCS (e.g., impact of car), which is supported in clinical studies (Evans

et al., 2007). In sum, there is good evidence that associative learning plays a role in the development and persistence of reexperiencing symptoms in PTSD.

*Memory processes: Poor memory elaboration*

Finally, poor memory elaboration is also proposed by Ehlers and Clark (2000) to account for reexperiencing in PTSD. This is also suggested in neurobiological information-processing models such as Brewin et al.'s (1996; 2010) dual representation theory (DRT) of PTSD. **Firstly**, Ehlers and Clark (2000) drew on Roediger's (1990) transfer-appropriate processing account, which suggests that information is encoded into memory via conceptual (processing meaning of the situation and placing it in context) and data-driven processing (processing sensory impressions). From this, Ehlers and Clark (2000) proposed that the nature of the trauma memory will depend on how information is processed and encoded during the trauma. Engaging in *predominantly data-driven processing* during a trauma is hypothesised to lead to strong encoding of perceptual information (such as taste, smell, colour) and weak encoding of contextual information (such as time and place). The resulting memory trace would contain predominantly sensory information and will be easily triggered by associated stimuli (cue-driven retrieval) due to strong perceptual priming and associative learning (between triggers and emotional responses) during the trauma, thereby leading to the development of reexperiencing symptoms. **Secondly**, with regards to the poor elaboration of the memory in the autobiographical memory base, Ehlers and Clark (2000) proposed that due to predominantly data-driven processing, the trauma memory is *not incorporated into the autobiographical memory base* (e.g., time and place context). Autobiographical memories are typically organised by themes and time periods (Conway, 1997; Conway & Pleydell-Pearce, 2000; Markowitsch, 1995), and

processed in this way so that they can be retrieved intentionally using higher-order meaning-based strategies, and activate both specific event information and connected general information about the lifetime period the event took place in. In this sense, in PTSD trauma memories are proposed to be poorly elaborated (not integrated in time and place context), and are more likely to consist of sensory impressions that are unintentionally triggered (by physically similar stimuli) and have a here and now quality (due to lack of integration) (Ehlers & Clark, 2000). This hypothesis is supported by clinical prospective studies showing that self-reported data-driven processing assessed after a trauma predicts PTSD development (Halligan et al., 2003). Consistent with this, self-reported dissociation (which may overlap with data-driven processing) during a trauma has also been found to predict PTSD (see Ozer, Best, Lipsey, & Weiss, 2003 for a review). Experimental studies investigating information processing during analogue trauma (e.g., a trauma film) have shown that participants who scored higher on data-driven processing showed more frequent reexperiencing symptoms in the following week (Halligan, Clark, & Ehlers, 2002). And studies that have manipulated processing by delivering a task that interferes with conceptual processing during a trauma film, have shown that this predicted a greater frequency of unintentional retrieval (Bourne, Frasquilho, Roth, & Holmes, 2010). Possible neurobiological underpinnings of deficient autobiographical memory encoding during trauma is offered by studies showing that high stress modulates hippocampal function, via inhibition of glucocorticoid receptors (see also Brewin et al., 2010; Metcalfe & Jacobs, 1998). This theory (poor memory elaboration) also accounts for the finding that people with PTSD simultaneously display both reexperiencing symptoms and difficulty intentionally retrieving the trauma memory (Brewin et al., 1996; Halligan et al., 2003), as well as disjointed memories of the trauma (Rubin, 2011), which can be accounted for by

reduced conceptual processing. Reduced conceptual processing (poor elaboration) is thought to contribute to PTSD in two ways. Firstly, it leads to poor links between the sensory information in the trauma memory and other information in autobiographical memory, which therefore impairs inhibition of cue-driven retrieval. Secondly, it contributes to problematic appraisals of the trauma (e.g., 'It's all my fault') due to poor links between the worst moments of the trauma and other relevant information stored later in the autobiographical memory (e.g., 'someone told me I had done everything that I could') that would disconfirm these appraisals (see Ehlers & Clark, 2000; Ehlers et al., 2004; Ehlers, Ehling & Kleim, 2012). Experimental support for this hypothesis comes from studies with PTSD patients showing that disjointedness of the worst moments is associated with difficulties accessing other information in memory that would put the meaning of these moments into perspective (see Ehlers, 2010). Studies have shown that both memory disjointedness/disorganisation and change in appraisals after a trauma predict PTSD (Dunmore, Clark, & Ehlers, 2001; Ehlers & Clark, 2000; Halligan et al., 2003). In sum, poorly elaborated and disjointed trauma memories may play a role in the development and maintenance of reexperiencing symptoms in PTSD.

#### *Appraisals and cognitive-behavioural responses*

Ehlers and Clark (2000) also propose that the individual's interpretation of their trauma memories, and their cognitive behavioural responses to trauma memories maintain reexperiencing in PTSD. They suggest that unhelpful interpretations of the trauma memories (theirnowness, and disorganised nature), such as 'my reactions since the event show that I am going crazy' (Ehlers & Steil, 1995) or 'I have permanently changed' will lead an individual to engage in maladaptive coping strategies such as trying to suppress the memory, ruminating on the trauma, or engaging in safety

behaviours such as avoiding triggers. Ehlers and Clark (2000) propose that this maintains reexperiencing by preventing the trauma memory from being processed, updated and integrated into the autobiographical memory base (i.e. elaborated). This is supported by prospective studies showing that negative appraisals of PTSD symptoms, rumination, thought suppression and safety behaviours predict the development of chronic PTSD over and above what can be predicted by initial PTSD symptom severity (Dunmore et al., 2001; Ehring et al., 2008; Halligan et al., 2003; Kleim et al., 2007; 2011). Negative appraisals of memory disorganisation have also been shown to predict PTSD symptoms even after controlling for memory characteristics (Halligan et al., 2003). Experimental studies have also shown that thought suppression and rumination may play a causal role in PTSD symptom maintenance (see Ehlers et al., 2012 for review). Finally, change in appraisals with PTSD treatment has been shown to predict PTSD symptom improvement (Lommen et al., 2013, Woodward, Lommen & Ehlers, in prep), including reexperiencing. In sum, more negative appraisals and maladaptive cognitive behavioural responses may be relevant for the development and maintenance of reexperiencing in PTSD.

### *Neurobiological models of reexperiencing in PTSD*

Brewin proposed a model of the neurobiological basis of flashbacks in PTSD (Brewin et al., 1996; 2010): the Dual Representation Theory (DRT). This model relates clinical observations to neural pathways. A vast body of evidence supports the idea that flashbacks are dependent on the involvement of the *perceptual memory system*, which is distinct from the ordinary *episodic memory system* (Brewin, 2014). Normal episodic memory is proposed to depend on the conscious focus of attention onto objects and

scenes that are bound together due to sharing the same location in space. This creates a stable, contextualised representation that can be retrieved or inhabited at will (Treisman & Gelade, 1980). Brewin proposed that during a trauma high levels of stress lead to a difference in memory encoding. Attention is narrowed onto the main source of threat, and so perceptual information from the wider scene is less effectively bound together. This results in a memory trace that is poorly contextualised and hard to control (triggered involuntarily). The DRT proposes that flashbacks are a product of activity in two memory systems: excessive stress-related activity in the dorsal stream (responsible for creating images of the environment), and a reduction in activity in the ventral visual stream in the medial temporal lobe (where elements of objects are bound together in an abstract form that allows them to be related to past experience). The resulting memory is of fragmented images that are experienced with a 'here and now' quality, due to poor integration into the autobiographical memory base. These have been termed 'sensory representations' (S-reps) produced via detailed, rapid, non-conscious processing. They are automatically triggered when a person re-enters a context (internal or external), and consist of sensory information from the trauma, such as visual, auditory and olfactory information. These are proposed to be poorly integrated with spatial and contextual information processed in the dorsal stream, termed 'contextual representations' (C-reps). Brewin et al., (2010) proposed that C-reps can be consciously processed, intentionally retrieved, require greater hippocampal encoding due to their spatial and temporal context (Brewin, 2001), and are easily communicated verbally. According to this theory, flashbacks are an adaptive part of the recovery process. In normal recovery, they are automatically triggered by sensory cues (trauma reminders) which prompts the allocation of attention, leading to re-encoding of perceptual information (S-reps) in episodic memory, where it is assigned spatial and temporal context (C-reps). The DRT

suggests that in normal autobiographical memory C-reps and S-reps are integrated, therefore the fear contained in the S-rep is inhibited by the simultaneous activation of the C-rep which includes the spatial and temporal context (e.g., the knowledge that it happened in the past). It is proposed that in PTSD, C-reps and S-reps are poorly integrated, therefore the S-rep is activated without the C-rep, resulting in a flashback memory that feels like it is happening again. This theory accounts for difficulties observed in PTSD such as problems intentionally recalling the trauma memory and disorganised trauma narratives (Foa & Riggs, 1993; Halligan et al., 2003). This theory is supported by studies showing that acute stress impairs performance on spatial learning tasks that are dependent on medial temporal lobe processing in healthy participants (Meyer et al., 2013) and those with PTSD (Smith, Burgess, Brewin, & King, 2015). Studies have also shown that in adults with PTSD, sections of flashbacks relating to narratives include greater perceptual detail and participants showed greater autonomic and motor behaviours (Hellowell & Brewin, 2002; 2004). This can be interpreted as evidence for the distinctiveness of S-reps. Further, as noted above, experimental studies using trauma-film paradigms have shown that tasks that interfere with C-rep encoding (e.g., verbal interference task) are associated with more frequent flashbacks in the following week. Tasks that interfere with S-rep encoding (e.g., visuo-spatial tapping) were associated with reduced flashbacks (Holmes, Brewin, & Hennessy, 2004). This lends support to the DRT's hypothesis that flashbacks are a result of enhanced activity supporting S-rep encoding and reduced activity associated with C-rep encoding.

#### *Methodological considerations*

When critically appraising the literature, it must be noted that while theories of PTSD and flashback maintenance and development are developed from clinical observations, the impact of trauma can be difficult to study experimentally – a method often used to isolate possible mechanisms and test theories. Experimental paradigms using analogue traumas (such as film clips) can provide useful information but must be caveated with the limitation that it cannot replicate the full impact of trauma. Therefore, conclusions may be limited by this, including about the possible mechanisms underpinning flashbacks. Similarly, in clinical populations, studying reexperiencing symptoms in the laboratory (for example using imaging) can also be challenging due to the uncontrollable nature of their occurrence.

### *Summary*

In sum, both the cognitive (Ehlers & Clark, 2000) and neurobiological (Brewin et al., 2010) models are complimentary accounts of the mechanisms underpinning reexperiencing in PTSD. They integrate evidence from conditioning (associative learning theory), autobiographical memory theory, clinical observations and imaging studies. Together they explain the development of reexperiencing symptoms, the ease of triggering by a wide-range of stimuli, reexperiencing content, and the vivid, ‘here and now’ quality of the memories. Therefore, the models are fully complimentary as the cognitive model (Ehlers & Clark, 2000) predicts how the nature of the trauma memory maintains PTSD symptoms and relates to an ongoing sense of current threat, and the DRT (Brewin et al., 2010) proposes the neurobiological underpinnings of the formation of the trauma memory and flashbacks. The cognitive model also includes the relationship between appraisals, behaviours and the nature of the trauma memory in maintaining PTSD (Ehlers & Clark, 2000). The clinical implications of both models

are that the trauma memory requires conscious attention to process it and integrate it into the autobiographical memory base. Therefore, they also explain why cognitive-behavioural coping strategies such as avoiding memory triggers and suppressing/avoiding the trauma memory prevent change in the trauma memory – because they prevent conscious attention being directed at the memory (Ehlers & Clark, 2000), which prevents integration of the C-reps and S-reps (Brewin et al., 2010), and therefore flashbacks persist (Brewin et al. 2010; Ehlers & Clark, 2000). Ehlers et al., (2005) outline a method for processing the trauma memory in trauma-focused cognitive therapy for PTSD (Ehlers et al., 2005) by imaginal reliving of the trauma with particular attention to the worst moments. This helps individuals to access information that can update the worst moments (e.g., ‘I survived’) and integrate this into the trauma memory (Ehlers & Clark, 2000). In considering the neurobiological underpinnings of this process, the DRT would propose that the process of reliving the trauma (in therapy) focuses attention on the trauma memory and leads to integration of the S-reps (e.g., fear) with relevant information in the C-reps (e.g., that it happened in the past), so subsequent activation of the S-rep is accompanied by automatic activation of the C-rep which would inhibit fear and a sense of nowness.

### **Affect without recollection**

As described previously, Ehlers and Clark (2000) proposed that reexperiencing may take the form of an affective (emotional) or physiological response to a trauma reminder, without awareness by the individual that this stems from a memory of the trauma. An example could be suddenly feeling very frightened or feeling a strong urge to leave a situation without knowing what caused it and without an associated memory of the trauma. This has been termed Affect without recollection (AWR). It has been

reported anecdotally by patients with PTSD but has never been formally investigated nor formally defined. Ehlers (2010) describes it as a form of reexperiencing that lacks auto-noetic awareness, which is the awareness of the self in the past and the knowledge of when in time a memory happened (i.e. the past). This awareness is a distinct feature of episodic memory (Tulving, 2002; 2016), and sets AWR apart from other involuntary autobiographical memories. The following section will describe a definition of AWR and possible mechanisms that could account for this phenomenon of an experience without a memory or awareness of triggers.

### **Defining affect without recollection**

Based on Ehlers and Clark (2000) a definition of AWR is proposed here that will be used throughout this thesis. AWR could be defined as the involuntarily experience of an emotional (e.g., fear) or physiological reaction (e.g., fast heart-beat) that was experienced during the trauma. The experience of this emotion/physiological reaction would be unrelated to, or stronger than expected given the situation the person is in. It would feel similar to how they felt during the trauma but would not be recognised as a memory, nor simultaneously accompanied by a recognisable trauma memory, such as an image. An example would suddenly feel very frightened, without knowing why. It could motivate the use of maladaptive coping strategies, a proposed consequence of reexperiencing (Ehlers & Clark, 2000). It is possible that once the reaction (AWR) is highlighted, that an individual could then work out retrospectively what triggered the feeling.

### **Evidence of affect without recollection**

There is a small amount of evidence in the literature of re-experiencing emotions and physiological sensations, rather than visual representations of a trauma. For example, studies have shown that some people report reexperiencing of pain, as though it were happening again in the present (Whalley, Farmer, & Brewin, 2007), demonstrating that physiological symptoms can be reexperienced. There is also evidence from a single case report that an individual with PTSD and organic amnesia from a head injury continued to reexperience emotional and physiological reactions in response to specific trauma reminders. It was conceptualised that associative learning during the trauma had been retained in the form of implicit memory. This meant that trauma reminders (a similar car) continued to trigger an emotional response and avoidance behaviours (such as swerving off the road) in the absence of recollection of the event (King, 2001). These studies provide preliminary evidence that AWR exists. Studies have also shown that people with schizophrenia show impairment in auto-noetic awareness and source memory (awareness of where, when and how it was learned). While they can recognise a source (familiarity) they cannot remember (auto-noetic awareness) the source's role in learning. Similar to PTSD, this is proposed to be due to ineffective links (relational binding) between content and source information due to difficulties during encoding (Danion, Rizzo, & Bruant, 1999). While the cause and consequence of encoding difficulties is different between Schizophrenia and PTSD, the result of poor relational binding in episodic memory is similar. Therefore, these studies provide evidence of memory in the absence of source information in other areas of psychopathology, suggesting that this may also be possible in PTSD in the form of AWR, perhaps as a result of poor relational binding as proposed in cognitive (Ehlers & Clark, 2000) and neurobiological models of PTSD (Brewin et al., 1996; 2010)

### **Possible mechanisms underpinning affect without recollection**

AWR could be understood using both the cognitive (Ehlers & Clark, 2000) and neurobiological models (Brewin et al., 1996; 2010) of PTSD that have been outlined previously in this introduction.

#### *Disjointed from the autobiographical memory base*

AWR may represent the extreme of the proposed functional independence between perceptual (S-reps) and episodic (C-reps) memory systems (Brewin, 2014; Brewin et al., 2010). According to Brewin et al.'s model (1996; 2010), the S-rep contains a sensory memory of the emotional and physical reactions from the trauma, which could be triggered without the accompanying C-rep due to poor integration. Brewin et al., (1996; 2010) also proposed that this poorly integrated memory is a result of enhanced perceptual processing during a trauma. Ehlers and Clark (2000) similarly proposed that due to *predominantly data-driven processing*, sensory information from the trauma is strongly encoded, and not integrated into the autobiographical memory base (poorly elaborated). The result could be a predominantly sensory memory (S-rep) that is disjointed from the autobiographical memory base (Ehlers et al., 2004; Michael et al., 2005) and is automatically triggered without the accompanying context information (C-reps). The result would be reexperiencing an emotional/physiological experience encoded during the trauma that feels like it is happening again and is *not recognised by the individual as a trauma memory* (AWR). This latter aspect, that it is not recognised by the individual as stemming from a trauma memory, is proposed to be the crucial difference discriminating AWR from other forms of reexperiencing such as flashbacks. Flashbacks (which could be visual, auditory, sensory), while experienced with a degree

of nowness which could extend to full dissociation, are still be recognised by the individual as stemming from a trauma memory. Therefore, AWR may reflect greater separation between the S-reps and C-reps, resulting in the triggering of the S-reps and perceptual memory representation only. People with AWR may have experienced higher levels of data-driven processing during the trauma, resulting in sensory dominated memory that is highly disjointed (to a greater degree than other reexperiencing symptoms) to the extent that it is experienced in the absence of any other autobiographical information.

Experimental research suggests that hormonal activity during high stress such as an acute trauma, diminishes neural activity in anatomical structures that support conscious processing and enhance activity in structures responsible for perception (Jacobs & Nadel, 1985; Metcalfe & Jacobs, 1998). Higher stress traumas could lead to weaker integration between C-reps and S-reps (Brewin et al., 2010). Therefore, one possibility is that AWR might be more likely after certain traumas, such as those that have a longer duration, resulting in more prolonged stress, or those that have been found to be more likely to predict PTSD, such as interpersonal traumas (Breslau et al., 2007).

### *Priming and associative learning*

The triggering of AWR can be understood as resulting from strong perceptual priming and associative learning between triggers and emotional responses during the trauma (Ehlers & Clark, 2000). Using associative learning theory, AWR can be understood as the emotional/physiological response (UR) to a trauma (US) which became paired with stimuli in the environment (CS). The same emotional/physiological reaction (CR) is later triggered by these stimuli (or stimuli sharing perceptual features) in the absence

of the US. Evidence from experimental studies has shown that participants can form these associations without awareness of learning, and later without awareness of the triggers (CS) (Bleichert:2007gc Grillon & Morgan, 1999) which has been termed lack of contingency awareness. This can explain the triggering of emotional responses from the trauma, in the absence of an accompanying memory and possibly without awareness of the trigger.

Ehlers (2015) also propose that strong perceptual priming may facilitate associations between emotional reactions and isolated perceptual features, which could account for the easy triggering of emotional reactions in response to stimuli sharing perceptual features with those present during the trauma (e.g., colour), which may be harder for individuals to recognise in the environment or make the link with the trauma.

#### *Appraisals and coping strategies*

Ehlers and Clark (2000) propose that reexperiencing symptoms motivate maladaptive coping strategies including avoidance of triggers and suppression of trauma memories, which prevent change in the nature of the trauma memory, as the attention required to process and integrate the S-reps and C-reps is not focused on the memory (Brewin et al., 1996; 2010). Interpretation of the trauma and trauma memory is another mechanism proposed to maintain reexperiencing symptoms (Ehlers & Clark, 2000). As AWR theoretically involves reexperiencing an emotion/physiological sensation that is not recognised as a trauma memory, it may be more frightening and confusing than other reexperiencing symptoms, and more likely to lead to appraisals such as 'I am going mad' or 'my brain is permanently damaged'. It could also make it harder to identify the trigger and may perhaps have consequences such as more widespread avoidance (such

as finding it hard to leave the house), as would be expected if the cause is unknown. Prospective studies have shown that frequency of, and distress at, reexperiencing predict PTSD development (Michael et al., 2005), as do negative appraisals of reexperiencing symptoms (Halligan et al., 2003). Based upon this, one hypothesis is that experiencing AWR after a trauma may make PTSD development more likely, more persistent, and may mean that AWR is harder to treat than other reexperiencing symptoms.

### *Dissociation*

Studies have consistently shown that self-reported dissociation during a trauma (Ozer et al., 2003) and persistent dissociation after a trauma predict PTSD (Murray, Ehlers, & Mayou, 2002; Ozer et al., 2003), and memory fragmentation and disorganisation (Halligan et al., 2003; Harvey & Bryant, 1998; Kindt, Van den Hout, & Buck, 2005; Murray, Ehlers, & Mayou, 2002; Ozer et al., 2003). Experimental studies have also shown greater state dissociation during analogue trauma predicts stronger perceptual priming (Lyttle et al., 2010; Michael & Ehlers, 2007). It has been proposed that dissociation during a trauma may lead to more data-driven processing and encoding deficits (Ehlers et al., 2004), and therefore makes the development of reexperiencing symptoms more likely. This is supported by studies showing that peritraumatic dissociation is highly correlated with data-driven processing (Halligan et al., 2003). Further it is suggested that persistent dissociation after a trauma while recalling trauma memories may prevent emotional processing of the event (Foa & Hearst-Ikeda, 1996), thereby maintain reexperiencing, which is supported by clinical studies (Murray, Ehlers & Mayou, 2002). If AWR is conceptualised as a highly disjointed trauma memory

(Ehlers et al., 2004), then it is possible that AWR may be more likely in people who experienced more severe dissociation during or after the trauma.

### **Summary and aims of the thesis**

#### **Summary**

In sum, AWR has been defined as reexperiencing an emotional/physiological reaction from the trauma, in the absence of a trauma memory (Ehlers and Clark, 2000). It may represent a trauma memory that is sensory as a result of predominantly data-driven processing of perceptual information (Ehlers & Clark, 2000), and that is highly disjointed from the autobiographical memory base (Ehlers et al., 2004) to the extent that emotions are reexperienced without a memory. It could be considered the extreme example of a functional independence between the perceptual and episodic memory systems, reflecting a poor integration between S-reps and C-reps (Brewin et al., 1996; 2010; Brewin, 2014). The content and easy triggering can be explained using associative learning and perceptual priming theories. Studies on conditioning in PTSD and memory in schizophrenia provide evidence of memory without contingency (Blechert et al., 2007) or source (Danion et al., 1999) awareness. Reexperiencing symptoms are maintained by negative appraisals of the trauma and of the trauma memory (Ehlers & Clark, 2000; Halligan et al., 2003) and are thought to motivate maladaptive coping strategies that maintain PTSD (Ehlers & Clark, 2000). Therefore, AWR is likely to be associated with both. Finally, dissociation has been associated with trauma memory fragmentation, disorganisation and predicts PTSD development and severity (Halligan et al., 2003; Ozer et al., 2003).

## **Theoretical predictions**

From the literature reviewed in this chapter, the following theoretical predictions were developed about AWR:

1. AWR may be associated with more data-driven processing during the trauma
2. Individuals with AWR may have experienced more peritraumatic dissociation
3. Individuals with AWR may also have more severe persistent dissociation
4. AWR may be predicted by more negative appraisals of the trauma and trauma memory
5. AWR should predict engagement with maladaptive cognitive-behavioural coping strategies
6. AWR may be maintained by maladaptive coping strategies such as response to intrusions, which prevent processing the trauma memory
7. Individuals with AWR could show more severe memory deficits (e.g., more susceptible to fear conditioning, worse contingency awareness, worse source recognition).
8. AWR may be associated with worse intentional recall of trauma memories, due to the possibility of reduced conceptual processing during the trauma.
9. AWR may be more likely after certain types or after longer duration of traumas.

As no studies of AWR currently exist, these predictions remain to be tested. Some will be tested in the study presented in this thesis, and some are suggestions for future research.

## **Aims of the thesis**

This introduction provided a possible definition of AWR however it remains unknown whether AWR exists, what the nature of AWR is, who experiences it, what predicts it and whether it is effectively treated using current PTSD treatments such as trauma-focused cognitive therapy (Ehlers et al., 2005).

The overall aim of this thesis was to investigate AWR for the first time, to determine its existence in PTSD, the nature of this symptom (phenomenology) and whether it is improved with PTSD treatment. The following key research questions were investigated:

1. Does affect without recollection exist in PTSD?
2. What is the nature (phenomenology) of affect without recollection?
3. What predicts affect without recollection in PTSD?
4. Is affect without recollection improved with PTSD treatment?

To investigate these research questions, the following approach was taken.

**Research question 1:** trauma survivors with and without PTSD were compared on the frequency and intensity of AWR. AWR was measured using a method called ecological momentary assessment (EMA), which required participants to self-report their AWR experiences and responses at multiple time points throughout the day. This enables the study of the symptom ‘in the moment’ and without retrospective recall bias. Measuring a symptom that may not be recognised as a trauma memory presented a challenge, and therefore EMA was particularly well-suited to studying this phenomenon, as

participants could simply be asked ‘did you experience a strong emotion or physiological sensation in the last few hours?’. EMA is described in more detail in Chapter 2.

**Research question 2:** The phenomenology of AWR was investigated by exploring the type, frequency and intensity of emotions reexperienced, as well as trigger types (when known) and whether AWR predicted the use of certain types of cognitive behavioural coping strategies.

**Research question 3:** To address predictors of AWR, in line with clinical models of PTSD (Ehlers & Clark, 200) it was investigated whether negative appraisals and responses to intrusions (such as suppression) predicted the occurrence and nature of AWR. Based upon the theoretical predictions described above, it was also investigated whether trauma type and dissociation predicted the occurrence and nature of AWR.

**Research question 4:** To investigate treatment effects, AWR was compared before and after Cognitive therapy for PTSD (Ehlers et al., 2005).

A better understanding of AWR could have important theoretical and clinical implications. These include advancing understanding of the nature and maintenance of reexperiencing symptoms in PTSD and helping clinicians to identify and treat AWR in PTSD treatment, which could enhance PTSD treatment outcomes.

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## **Part 2: Empirical Paper**

### **Daily experiences of Affect without Recollection in Posttraumatic Stress Disorder**

## Abstract

Re-experiencing symptoms in posttraumatic stress disorder include an affective or physiological response to a reminder of the trauma, not identifiable by the patient as a trauma memory. This has been termed 'Affect without recollection' (AWR; Ehlers & Clark, 2000). AWR has been anecdotally reported, but never investigated. **Aim** The phenomenology of AWR and the effect of cognitive therapy for PTSD (CT-PTSD; Ehlers et al., 2005) was investigated. **Method** Ecological momentary assessment (EMA) was used. 115 participants (42 PTSD, 43 trauma-exposed controls, 30 non-trauma-exposed controls) reported daily experiences of AWR four times a day for seven days before and after trauma-focused CBT, or an equivalent wait (trauma-exposed controls). Response to intrusions, dissociation and posttraumatic cognitions were also assessed. Modelling techniques developed for the analysis of EMA data were used to analyse patterns of AWR responses in PTSD. **Results** People with PTSD reported a significantly higher frequency and intensity of AWR compared to trauma-exposed controls. Re-experienced emotions fell into two cluster which were associated with different cognitive behavioural responses. More severe dissociation predicted higher likelihood of AWR, and negative appraisals and unhelpful responses to intrusions predicted the type and intensity of reexperienced emotions. AWR was improved with CT-PTSD. **Conclusion** The study provided the first evidence of the existence of AWR and its consequences.

## Introduction

### *Affect without recollection*

Re-experiencing symptoms (spontaneous, emotion-laden intrusions) are one of the hallmark symptoms of PTSD. These can be experienced as intrusive memories of the trauma (or moments from the trauma) in the form of visual or sensory flashbacks (Ehlers, Hackmann, & Michael, 2004a; Ehlers et al., 2002). Re-experiencing symptoms can be conceptualised as emotionally charged memories, separate from other contextual information in the autobiographical memory system. They are experienced with a sense of ‘nowness’, i.e. a sense that the trauma is happening again in the here and now, without the awareness that it is in the past (Brewin, 2014; Ehlers & Clark, 2000). However, re-experiencing symptoms may take the form of an affective (emotional) or physiological response to a reminder of the trauma, as defined in the diagnostic criteria for PTSD (5<sup>th</sup> edition; DSM-5, American Psychiatric Association, 2013). Ehlers and Clark (2000) point out that people with PTSD may have such emotional and physical responses without any recollection of the trauma, i.e., without realising that they are experiencing a trauma memory. An example could be the sudden feeling of terror without knowing why and without an associated recollection of the trauma in any other form. This has been termed *Affect Without Recollection* (AWR) (Ehlers & Clark, 2000). AWR and its possible theoretical underpinnings were described in detail in the Conceptual Introduction (Part 1), therefore only a brief overview will be given here. AWR has been reported anecdotally by patients with PTSD but has not yet been investigated or measured. It is therefore unknown how frequently trauma survivors experience this symptom, whether people with PTSD experience it more intensely than trauma survivors without PTSD, and how they respond to the symptom when it is experienced. For example, cognitive-behavioural responses to re-experiencing symptoms in PTSD such as rumination and suppression of

involuntary memories have been found to maintain PTSD (Ehring, Ehlers, & Glucksman, 2008). It would therefore be important to investigate not only the frequency and nature of the experience of AWR, but also the cognitive-behavioural responses. Furthermore, studies have found differences in re-experiencing symptoms between trauma survivors with and without PTSD. For example, increased sense of ‘nowness’, higher distress, and lack of memory context are greater in PTSD than trauma survivors (Kleim, Graham, Bryant, & Ehlers, 2013a), and are predictive of later PTSD (Michael, Ehlers, Halligan, & Clark, 2005a). Therefore, to fully understand the nature of AWR, it would be worthwhile not only studying AWR in PTSD, but also to compare experiences in trauma survivors with and without PTSD. Finally, there is evidence that negative appraisals, persistent dissociation (Halligan, Michael, Clark, & Ehlers, 2003a), and response to intrusions (suppression, avoidance, rumination) are involved in the maintenance of PTSD (Ehring et al., 2008). Therefore, the role of these cognitive processes in predicting AWR should also be considered.

### *EMA approaches*

Previous studies investigating symptom phenomenology have used daily diary approaches, such as ecological momentary assessment (EMA) (Kleim, Graham, Bryant, & Ehlers, 2013b; Kleindienst et al., 2017). EMA approaches involve repeated sampling (e.g., multiple times a day and week) of participant’s ‘in the moment’ experiences, in their naturalistic environment, in real time (Kleim, Graham, Bryant, & Ehlers, 2013a; Shiffman, Stone, & Hufford, 2008). This therefore increases ecological validity and a more in depth and detailed understanding of a symptom. It also avoids the disadvantages of retrospective, summarised accounts of symptoms (e.g., questionnaire assessment of symptoms over the previous two weeks) including recall bias and error, and failure to capture symptom or behaviour change over time and context (Sato & Kawahara, 2011).

It has been used to investigate stress, depression, schizophrenia and many other psychopathological symptoms (Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001; Oorschot, Kwapil, Delespaul, & Myin-Germeys, 2009). A recent study applied an EMA approach to study intrusive re-experiencing in PTSD (Kleim, Graham, Bryant, & Ehlers, 2013a). This approach enabled the researchers to investigate ‘in the moment’ behavioural, emotional and cognitive responses and possible triggers of re-experiencing symptoms. It provided valuable information that trauma survivors with and without PTSD experienced a similar frequency of intrusions, but that PTSD patients reported a greater sense of ‘nowness’. Given the advantages of EMA for studying phenomenology, this approach was used in the present study to investigate daily experiences of AWR, including emotional, behavioural and cognitive responses, and possible triggers.

#### *PTSD treatment*

Trauma-focused cognitive therapy for PTSD (CT-PTSD; Ehlers & Clark, 2000; Ehlers, Clark, Hackmann, McManus, & Fennell, 2005) was developed from Ehlers and Clark's (2000) model of PTSD. The treatment aims to reduce an individual's sense of current threat and in doing so decrease intrusive re-experiencing symptoms, by reducing problematic appraisals, updating the worst moments of the trauma memory, and reducing problematic behaviours (such as avoidance), and cognitive strategies (such as rumination), that also inadvertently maintain the appraisals and memory features. CT-PTSD has been found to be a highly effective treatment for PTSD (Ehlers et al., 2005). However, the effect of CT-PTSD on AWR has not been measured and remains unknown.

The present study investigated the phenomenology of AWR, by analysing quantitative EMA data on daily experiences of AWR. It further investigated the effect of CT-PTSD for PTSD on AWR symptoms.

## **Research questions**

Specifically investigated were 1) whether frequency and intensity of AWR experiences differed between trauma survivors with and without PTSD; 2) the phenomenology of AWR in PTSD, 3) does trauma type or baseline dissociation, response to intrusions and appraisals predict AWR; and 4) whether CT-PTSD improved AWR in patients with PTSD.

Given the exploratory nature of the research, no specific hypothesis was derived for the phenomenology of AWR (question 2). However, given previous findings that intrusive re-experiencing symptom phenomenology differs between trauma survivors with and without PTSD (Kleim et al., 2013) it was predicted that people with PTSD would differ in frequency and intensity of AWR experiences (question 1). Given evidence that negative appraisals, persistent dissociation (Halligan, Michael, Clark, & Ehlers, 2003a) and unhelpful response to intrusions (Ehring et al., 2008) play a role in PTSD maintenance, it was predicted that these cognitive processes would also predict a greater likelihood of experiencing AWR. Based upon evidence that PTSD (Kilpatrick et al., 2013) and more intense emotional reactions are more likely after interpersonal compared to non-interpersonal trauma (Amstadter & Vernon, 2008), it was predicted that interpersonal trauma would lead to a greater likelihood of experiencing AWR (question 3). Finally, it was predicted that AWR frequency and intensity would reduce with CT-PTSD, based on previous findings that CT-PTSD improves re-experiencing symptoms in PTSD (Ehlers et al., 2005; 2014) (question 4).

## Method

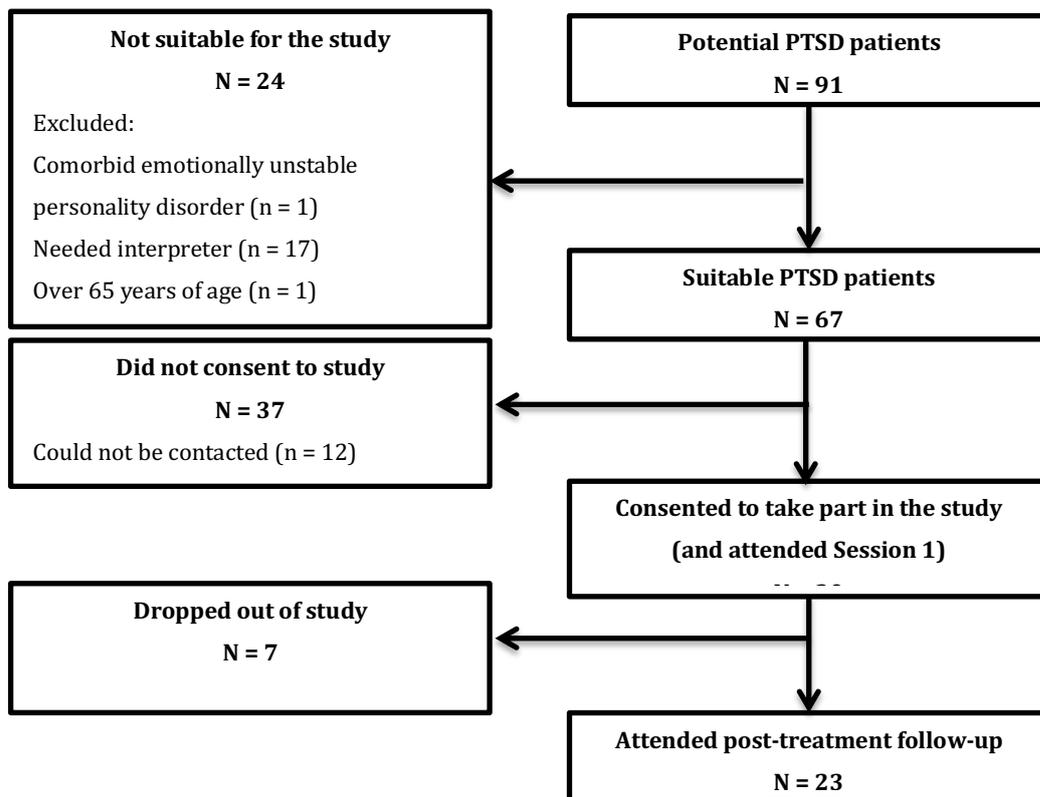
### Participants

A total sample of 115 participants (80 women) took part in the study (Mean age: 32.73, SD: 11.73); 42 participants with PTSD (comprised of 30 treatment-seeking PTSD patients recruited from NHS services, and 12 recruited from the community), 43 trauma-exposed participants without PTSD and 30 non-exposed control participants. Participants were recruited via adverts, and from participating NHS clinics in London and Oxford. Figure 1 shows the recruitment flow-chart for PTSD patients. Participants were aged between 18 and 65, could read and write in English, had no history of or current bipolar or psychosis, no current substance or alcohol dependence, and if they were taking psychotropic medication had been on a stable dose for at least 1 month. The PTSD group met diagnostic criteria for a DSM-5 diagnosis of PTSD, as assessed with the *Clinician-administered PTSD Scale for DSM-5* (Weathers et al., 2013). The trauma-exposed control group had experienced a Criterion A traumatic event as defined in DSM-5 but did not meet criteria for PTSD. The non-exposed control group had never experienced a Criterion A traumatic event and reported no current mental health problems. Table 1 shows demographic characteristics and test-statistics for the groups. The study received NHS ethical approval (Ref 14/SC/0198) and complied with the ethical principles of research with human participants.

Of the treatment-seeking PTSD patients, 23 were assessed again three months later, after a course of PTSD therapy. Seven patients were not assessed as they could no longer be contacted or had dropped out of treatment. As a comparison, 29 trauma-exposed control participants were assessed three months later (after no intervention) to check for effects

of re-assessment. Therefore, the final follow-up sample sizes were 23 people with PTSD and 29 trauma controls ( $n = 52$ ).

As no previous studies exist, the effect size of AWR was unclear therefore post-hoc power analyses (also called sensitivity analysis) only were calculated. This revealed that the recruited sample of 76 (trauma-exposed and PTSD at session 1) yielded a power of 76% to detect medium effect size (0.7) in between group differences. For the post-treatment analysis, the recruited sample size ( $n = 52$ ) provided 61% power to detect medium effect sizes in pre to post-treatment changes. For modelling analyses, approaches were used that did not include assumptions of sample size.



**Figure 1** Recruitment flow-chart for PTSD patients from referral into the study to post-treatment assessment ( $n = 23$ ).

## **Measures**

### ***Assessment of Trauma and PTSD***

*Life Events Checklist (LEC)*. The LEC (Gray, Litz, Hsu, & Lombardo, 2016) is a self-report questionnaire containing a list of stressful and traumatic (according to DSM-5 criteria) life events. Respondents indicate whether they have experienced each event in their lifetime. The LEC was administered to determine whether participants had experienced a Criterion A traumatic event, according to DSM-5 criteria (APA, 2013), and to identify a negative life event experienced by non-traumatised control participants (see Appendix A for scale).

*Clinician-administered PTSD Scale for DSM-5 (CAPS)*. The CAPS (Weathers et al., 2013) is a clinician-administered measure of the severity of PTSD symptoms, according to DSM-5 criteria. The CAPS was administered by the author of this thesis, if participants reported a traumatic event on the LEC.

### ***PTSD symptom measure***

*PTSD Checklist for DSM-5 (PCL-5)*. The PCL-5 (Weathers et al., 2013) is a validated self-report measure assessing the severity of DSM-5 specified PTSD symptoms over the previous week.

### ***Cognitive process measures***

*Posttraumatic Cognitions Inventory (PTCI)*. The 20-item short version of the PTCI (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999b), was used to assess excessively negative appraisals of the trauma and its consequences. Respondents rate their agreement with each appraisal from 1 (*totally disagree*) to 7 (*totally agree*). Items are summed to produce a total score (range: 1–140), with higher scores indicating more maladaptive appraisals.

The measure has been found to have good internal consistency (Cronbach's alpha = .97) and psychometric properties (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999a). The measure has three sub-scales: 1) Negative thoughts about the self (*e.g., my reactions mean I am going crazy*); Self-blame (*e.g., I disgraced myself during the event*); 2) Overgeneralised danger (*I have to be on guard all the time*) and 3) Preoccupation with unfairness (*e.g., If I stop being angry, the people who caused the event will get away with it*).

*Response to Intrusions Questionnaire (RIQ)*. The RIQ (Clohessy & Ehlers, 1999) is a 19-item questionnaire assessing response to intrusions in PTSD. Respondents rate how often they have responded on a scale from Never, Sometimes, Often, Always. Items are scored from 0 (Never) to 3 (Always). It comprises three subscales: Thought suppression (*e.g., I try to push them out of my mind*); Rumination (*e.g., I think about why the event happened to me*); and Numbing (*e.g., I detach myself from the memories*).

*Trait dissociation questionnaire-short version (TDQ-s)*. The TDQ-s (Murray, Ehlers, & Mayou, 2002) is a 10 item version of an original 38-item questionnaire assessing pre-trauma disposition for dissociative experiences. It measures seven different aspects of dissociation: detachment from others and the world, sense of split-self, lability of mood and impulsivity, inattention and memory lapses, emotional numbing, confusion and altered sense of time and amnesia for important life events. The scale has good internal consistency and test-retest reliability (Cronbach's alpha = 0.95), and has been shown to predict PTSD symptom severity after a trauma (Murray et al., 2002).

## ***Ecological Momentary Assessment***

### *Affect Without Recollection Application*

A smartphone app was developed by the author and used to collect real-time and detailed assessment of AWR. Using an alarm, the app prompted participants to record occurrences and details of AWR experiences four times a day, at set times and intervals, for seven days. Participants recorded any strong emotional or physiological sensations they experienced in the last four hours, the strength/intensity of the sensation rated from 0 (not at all) to 100 (extremely), cognitive-behavioural responses (e.g., dwelling, distraction, alcohol use) and the extent to which they were used (rated from 0-100), noticeable triggers, if it made sense in their current context, if it was similar to how they felt during the trauma, and whether the emotion was followed by a trauma memory (see Appendix B for application).

According to the previously laid out definition of AWR (see Conceptual Introduction; ‘Defining affect without recollection,’ page 38), a report in the app was classified as AWR if a participant reported a strong feeling/bodily sensation/urge to leave a situation (‘Yes’ to Q1, see Appendix B), that was either unrelated to what they were doing at the time, or, stronger than expected given the situation (Q2 – either option selected, Appendix B) and was similar to how they felt during the trauma (‘Yes’ to Q3, Appendix B).

### **Procedure**

Participants who met inclusion criteria for the study were contacted via phone about the research and received a full information sheet in advance of the session. Full informed consent was completed at the research session. Upon arrival participants completed a general demographic information sheet. The LEC was then completed to assess for trauma exposure, and trauma-exposed participants were assessed for PTSD diagnosis

using the CAPS (Weathers et al., 2013). Following this, participants received a copy of the AWR application either on their own smart phone (if they owned one) or were given this on a loaned iPod device with the app already installed. The app included standardized instructions (including a description of the symptom being investigated, see Appendix B), which were read aloud to participants, and understanding was checked. Participants were shown how to work the app and asked to demonstrate understanding by completing a practice entry. They were asked to complete the app four times a day for seven days, by responding to prompts from the app (an alarm with a reminder) every four hours at set times (10am, 2pm, 6pm, 10pm), to capture a 12-hour period. The app also included the option to complete a voluntary entry if they experienced this symptom between set times. Control participants were asked to complete the app in relation to the most distressing negative event they had identified on the LEC.

At the end of the session, participants completed a questionnaire pack, to assess self-reported PTSD symptoms (PCL-5) and cognitive processes: appraisals, response to intrusions and dissociation (PTCI; RIQ; TDQ). Trauma-exposed participants were asked to answer these measures (PCL-5; PTCI; RIQ; TDQ) in relation to the traumatic event, and control participants answered them in relation to the most distressing negative event they had identified on the LEC. PTSD patients awaiting treatment were invited back to a second session three months later, after receiving Cognitive Therapy for PTSD (CT-PTSD; Ehlers et al., 2005), which follows Ehlers and Clark's (2000) cognitive model of PTSD. Trauma controls were invited back after an equivalent time period (three months) after no intervention. At the second session, the CAPS was repeated to assess PTSD diagnosis, followed by the same procedure and measurement week as in Session 1.

## **Data Analysis**

Analyses were conducted in IBM SPSS statistics (version 26) and R (3.6.1 and RStudio version 1.2.5019) with R packages tidyverse, nlme, Imeresampler, and boot (Davison & Hinkley, 1997; Lo & Steele, 2020; Canty & Ripley, 2019; Pinheiro, Bates, DebRoy, Sarkar, R Core Team, 2019; R Core Team, 2019; Rstudio Team, 2019; Wickham et al., 2019).

Data was checked for assumptions of normality and homogeneity, and appropriate tests were used.

It was first examined whether groups differed on PTSD symptom severity (PCL-5), posttraumatic appraisals (PTCI), response to intrusions (RIQ) and dissociation (TDQ), groups (PTSD, trauma-exposed, non-exposed controls), using ANOVAs, Kruskal-Wallis or Mann-Whitney tests, and planned contrasts.

The following questions were then investigated: 1) whether frequency and intensity of AWR experiences differs in trauma survivors with and without PTSD; 2) what the phenomenology of AWR is; 3) whether trauma type or baseline dissociation, response to intrusions and appraisals predict AWR, and 4) whether CT-PTSD improves AWR in patients with PTSD.

### ***Research Questions 1 and 2: Group differences and phenomenology of Affect without Recollection***

To investigate whether the frequency and intensity of AWR experiences differ between trauma survivors with and without PTSD (Question 1), the data was first explored using simple descriptive statistics, and groups were compared on the frequency of AWR and

intensity ratings of emotions using independent t-tests. An AWR experience was defined according to the criteria set out in the description of the AWR app (see ‘Measures,’ page. 64).

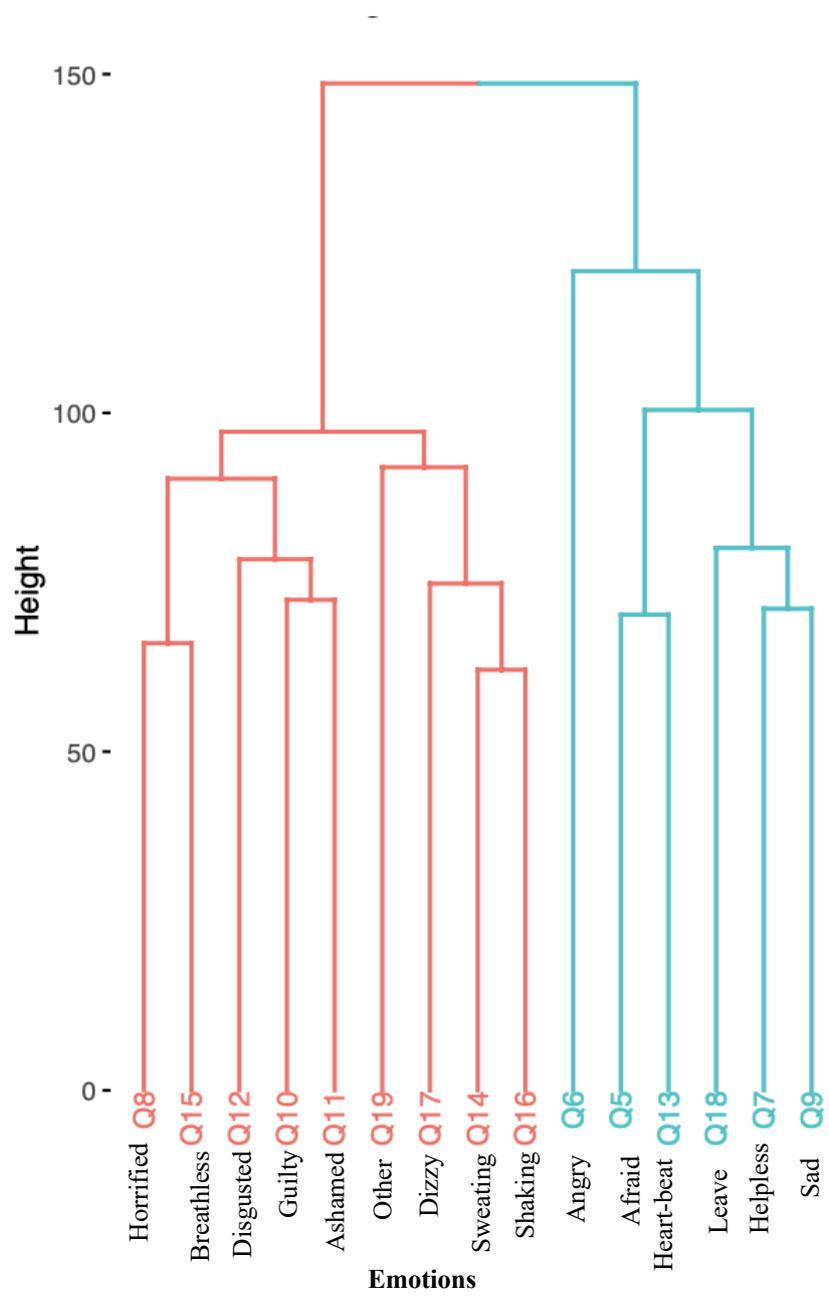
To investigate the phenomenology of AWR in PTSD (Question 2), responses from PTSD participants reporting defined AWR were analysed. Trauma-survivors reporting AWR were not included in this analysis, as re-experiencing symptoms between PTSD and trauma-survivors have been shown to differ (Kleim, Graham, Bryant, & Ehlers, 2013a). In PTSD patients reporting AWR it was investigated whether a) patterns of re-experienced emotions co-occurred together over time, and b) re-experienced emotional patterns were associated with particular cognitive-behavioural responses.

The following data analysis strategy was used:

a) *Do patterns of re-experienced emotions co-occur over time?*

An agglomerative hierarchical cluster analysis was used (Nielsen, 2016), which falls into the broader category of an unsupervised machine learning approach. This approach is well-suited to dealing with small sample sizes. It involves identifying patterns in the data (with the absence of pre-existing labels) by calculating the similarity/dissimilarity between each element (in this case each of the 15 emotion variables) over time i.e. the ‘Euclidean distance’. Agglomerative clustering is then done using a ‘bottom-up’ approach, which initially considers each element (emotion) as a single cluster. At each step of the algorithm similar elements are combined into larger clusters, until all elements are combined into one cluster. This clustering method is done using a ‘linkage function’ which uses the distance information. The clustering steps create a dendrogram (a graph of the hierarchical tree; Figure 2), which is used to decide where to cut the tree into clusters, to achieve the best solution for the number of clusters. The goal is to identify clusters of

datapoints, that are as close to each other as possible / maximally similar, and as distant as possible from other clusters / maximally dissimilar. The chosen cluster solution is verified by correlating linkages in the cluster tree (distance between cluster means) with the distances in the original matrix (distance between observations). Correlations should be above 0.75 (a higher correlation indicates greater more representativeness of the original data), otherwise the method of linkage should be changed.



**Figure 2** Cluster dendrogram of emotions and clusters in the PTSD sample (n = 28); extracted through agglomerative hierarchical cluster analysis. Red = Cluster 1; Blue = Cluster 2 (see Table 6). Height = cophenetic distance (similarity/dissimilarity between objects/clusters).

*b) Are patterns of re-experienced emotions associated with particular cognitive-behavioural responses?*

Linear mixed-models were used, which take into account the nested structure of the data (nested time points within participants). This approach does not assume independence of data points, and is therefore appropriate for data where there is the possibility of inter-dependence between items (Bosker & Snijders, 1999). For example, intensity ratings on each day are likely to be related and inter-dependent, and therefore these observations are not fully independent. Finally, MLM can handle varying time intervals between entries and missing data (Bosker & Snijders, 1999), and is appropriate for longitudinal data (Singer & Willett, 2003).

To analyse the data, the mean scores for each cluster of emotions (defined in question a) was used for each available time point. These cluster means were considered time-varying predictors. The model consisted of time (level 1) nested within participants (level 2). The covariance structure matrix was unstructured, intercepts were random, time was fixed, and the estimator used was Maximum Likelihood estimation (MLE). Due to the small sample size, standard errors for fixed effects (cluster scores) were bootstrapped (1000 times), using the normal distribution as the sampling distribution. Significance tests were corrected (95% confidence intervals using these corrected standard errors - a confidence interval that does not include zero indicates a significant effect). Bonferroni correction for multiple correlated outcomes was not used due to the explorative nature of the analysis.

***Research question 3: do trauma type and baseline dissociation, response to intrusions and appraisals predict AWR?***

*c) Do trauma type, baseline dissociation, response to intrusions and appraisals predict patterns of emotions?*

Multiple linear regressions were used to examine whether sum scores on baseline predictors (PTCI, TDQ and RIQ) predicted intra-individual means over time for each emotion (cluster means). The intraindividual mean over time for each emotion was calculated, then the intraindividual mean score for each cluster of emotions was calculated. Trauma type (interpersonal/ non-interpersonal) was used in a linear regression as a dichotomous predictor of patterns of emotions over time (intra-individual cluster means).

*d) Do trauma type, baseline dissociation, response to intrusions and appraisals predict presence/absence of AWR?*

Trauma type was used as a dichotomous predictor (interpersonal/non-interpersonal) in a logistic regression to predict presence (defined as at least one AWR experience over time) or absence of AWR. The dependent variable was presence of AWR in PTSD participants versus absence in all other participants not reporting AWR (remaining PTSD, trauma-exposed and non-exposed controls;  $n = 75$ ). Similarly, a logistic regression was used to examine whether sum scores on baseline predictors (PTCI, TDQ and RIQ) predicted the presence/absence of AWR, using the same method. Zero-order point-biserial correlations were used to examine the relationship between sub-scales scores on baseline measures (PTCI, TDQ and RIQ) and the presence and absence of AWR. Beta values were statistically compared for significant difference using a z-test to compare unstandardized regression weights.

***Research question 4: does AWR improve with CT-PTSD?***

To check whether treatment led to improvements in PTSD symptoms and related cognitive processes, groups with participants who attended both sessions (PTSD:  $n = 23$ ; Trauma  $n = 29$ ) were compared on measures of PTSD symptom severity (PCL-5), appraisals (PTCI), response to intrusions (RIQ) and dissociation (TDQ) at Session 1 and Session 2. The PTSD sample included those recovered ( $n = 16$ ) and not-recovered from PTSD at post-treatment ( $n = 7$ ) according to the CAPS. A mixed repeated-measures ANOVA was conducted with the between-subjects factor 'group' (trauma, PTSD), and within-subjects factor 'time' (Session 1, Session 2), followed by planned paired t-tests.

To investigate whether AWR improved with CT-PTSD, paired t-tests were conducted to explore changes in the frequency and intensity of AWR from Session 1 to Session 2.

## Results

### Demographic statistics

Demographic and test statistics are shown in Table 1. The groups did not differ on age and gender distribution and medication use at baseline and medication use did not change from Session 1 to Session 2 in either group ( $p > .05$ ). There was no difference in trauma type (interpersonal versus non-interpersonal) between PTSD and trauma-exposed controls (Table 1). Groups differed on distribution of ethnicity. This showed no significant effect on between group differences or Session 1 to 2 changes in PTSD symptom severity (PCL-5) when entered as a covariate in the analysis,  $p$ 's  $< .05$ , and so was not included in further analyses.

People with PTSD had significantly higher scores on all baseline measures (PCL-5; PTCI; RIQ; TDQ) compared to trauma-exposed controls, who did not differ from non-exposed controls (see Table 3).

**Table 1** Demographic information and PTSD and depression symptom severity for each group, and statistical comparison of groups on distributions ( $\chi^2$ ) and mean scores (F or t).

	PTSD (N = 42)	NON-EXPOSED CONTROLS (N = 30)	TRAUMA- EXPOSED CONTROL (N = 43)	STATISTICAL SIGNIFICANCE
		M (SD)		
<b>AGE (years)</b>	32.51 (9.93)	31.17 (10.38)	34.02 (14.01)	$F(2, 109) = .53, p = .59$
<b>SYMPTOM MEASURES</b>				
PCL-5	35.10 (11.45)		8.4 (6.78)	$t = -12.57, p < .001$
PTCI	134.83 (43.78)		72.21 (36.63)	$t = -6.92, p < .001$
TDQ	16.18 (12.14)		3.53 (3.789)	$t = -6.50, p < .001$
RIQ	33.26 (11.49)		11.58 (8.40)	$t = -9.581, p < .001$
<b>AFFECT WITHOUT RECOLLECTION</b>				
Frequency	5.12 (4.67)	0.33 (1.54)	0.89 (1.86)	$H = 48.13, p < .001$
	N (Valid %)			
<b>GENDER</b>				
Women	26 (61.9%)	23 (76.7%)	31 (72.1%)	
Men	16 (38.1%)	7 (23.3%)	12 (27.9%)	$\chi^2(2) = 2.1, p = .37$
<b>Education (years)</b>	15.63 (3.54)	17.43 (2.85)	16.40 (2.65)	$F(2, 107) = 2.10, p = .06$
<b>ETHNIC BACKGROUND</b>				
	N (%)			
Caucasian	28 (66.6%)	29 (96.7%)	35 (81.4%)	
Ethnic minority	14 (33.4%)	1 (3.3%)	8 (18.6%)	$\chi^2(2) = 9.93, p = .007$
<b>MEDICATION USE</b>				
	N (%)			
No medication	22 (52.4%)	18 (60%)	30 (69.8%)	
Non-psychotropic medication	10 (23.8%)	10 (33.3%)	10 (23.3%)	
Use of psychotropic medication (all)	10 (23.8%)	2 (6.7%)	3 (7.0%)	<sup>3</sup> Med use: $\chi^2(4) = 7.76, p = .10$
<b>TRAUMA TYPE (trauma survivors; n = 85)<sup>1</sup></b>				
Interpersonal	20 (47.62%)		14 (32.56%)	
Non-interpersonal	22 (52.39%)		29 (67.44%)	<i>Interpersonal vs. non-interpersonal trauma: <math>\chi^2(1) = 1.59, p = .26</math></i>
<b>TRAUMA CHARACTERISTICS (trauma survivors; n = 85)<sup>1</sup></b>				
Time since Trauma (years)	8.23 (9.71)		10.12 (10.18)	$t(78) = .85, p = .39$

Note <sup>1</sup>Trauma type distribution was compared by collapsing groups into interpersonal and non-interpersonal trauma types and comparing these. <sup>2</sup>Due to missing data, the sample sizes were: Time since trauma was calculated as the time (years) from trauma to study participation date. <sup>3</sup>Med use = comparison of groups on any medication (psychotropic, non-psychotropic, no medication).

People with PTSD recruited from the community and treatment-seeking patients did not differ on PTSD symptom severity  $t(39) = 0.51, p = .62$ . PTSD patients who did and did not drop out of treatment were comparable in age and PTSD symptom severity (Table 2).

**Table 2** Baseline demographic information and test-statistics (Mann Whitney) comparing participants who attended both sessions, and those who dropped out from the study after Session 1.

	<u>Attended both sessions</u>	<u>Dropped out</u>	<u>Test statistic</u>
	M (SD)	M (SD)	Mann-Whitney U, <i>p</i>
<b>PTSD patients (<i>n</i>)</b>	<i>n</i> = 23	<i>n</i> = 7	
Age (years)	33.00 (9.33)	37.71 (13.19)	41.00, .33
PCL-5	34.00 (10.49)	29.00 (13.34)	74.00, .30
<b>Trauma controls (<i>n</i>)</b>	<i>n</i> = 29	<i>n</i> = 14	
Age (years)	31.82 (11.20)	33.38 (15.72)	231.50, .46
PCL-5	8.55 (7.31)	8.15 (6.04)	180.00, .55

PCL-5 = PTSD symptom severity

**Table 3** Descriptive and test-statistics for each group, for participants who attended both Session 1 and Session 2.

	<b>TRAUMA (n = 29)</b>		<b>PTSD (n = 23)</b>		<b>TEST STATISTICS</b>			<b>TRAUMA</b>	<b>PTSD</b>
	SESSION 1	SESSION 2	SESSION 1	SESSION 2	TIME (F, p)	GROUP (F, p)	INTERACTION (F, p)	Paired T-TEST t, p, (d)	Paired T-TEST t, p (d)
	Mean (SD)								
<b>PCL-5</b>	8.48 (7.49)	5.81 (8.28)	35.5 (11.01)	19.35 (12.67)	51.17, p < .001	52.47, p < .001	27.27, p < .001	2.44, .03	6.87, p < .001 (1.51)
<b>PTCI</b>	72.14 (33.62)	63.71 (34.08)	127.42 (35.65)	79.11 (26.64)	28.42, < .001	20.01, < .001	14.40, < .001	1.44, .16 (.25)	4.99, < .001 (1.54)
<b>RIQ</b>	12.31 (8.80)	7.07 (8.35)	31.72 (10.84)	16.32 (12.44)	69.71, < .001	33.64, < .001	16.88, < .001	4.22, < .001 (0.61)	6.97, < .001 (1.32)
<b>TDQ</b>	2.89 (3.38)	3.18 (5.04)	13.08 (9.63)	5.96 (7.27)	14.45, < .001	61.72, < .001	16.96, < .001	-61, .55 (.07)	3.31, < .01 (.83)
<b>AWR frequency</b>	0.76 (1.33)	0.45 (1.02)	6.86 (5.76)	2.09 (2.68)	27.25, < .001	28.80, < .001	20.99, < .001	2.07, .05 (.26)	4.22, < .001 (1.06)

*Note.* Test statistics (F/independent t-test) and significance levels (p) are shown for main effect of group (Trauma, PTSD), time (session 1, session 2) and paired t-test compare session 1 to 2 within each group. (d) = effect size, where a small effect size = .20, medium = .50, and large = .80, according to Cohen (1992). Includes those who did and did not report AWR.

## **Research Questions 1 and 2: Group differences and phenomenology of Affect without Recollection in PTSD.**

### *Affect without recollection: Descriptive statistics (Session 1)*

#### *Application completion rates*

A total of 111 participants completed the app at session 1. There was no data for five participants at either session due to a technical failure (PTSD = 2; Control = 3). On average participants responded to 98% (SD = 15.12) of app prompts at session 1 (PTSD: 98%, SD = 12.11; Trauma: 95%, SD = 21.07; Control: 99, SD = 0.69) and 87% (SD = 33.57) at session 2 (PTSD: 81%, SD = 40.13; Trauma: 93%, SD = 25.74).

#### *Reported AWR*

A total of 36 people reported at least one incidence of AWR over seven days at session 1 (28 PTSD (67%); 6 trauma-exposed controls (14%); 2 non-exposed controls (7%)). In the PTSD group, 14 people (33%) did not report any AWR, and two did not complete the app. There were 107 reports of AWR in total across all three groups over seven days; 93 in the PTSD group, 11 from trauma-exposed controls, and three in the control group (see Table 1). A Kruskal-Wallis test showed a significant difference between the groups for mean number of AWR experiences,  $H(2) = 48.13, p < .001$ . The PTSD group reported more AWR experiences ( $M = 5.12$ ;  $SD = 4.67$ , range 0-19) than the trauma-exposed ( $M = 0.89$ ;  $SD = 1.86$ , range = 0 -8) and non-exposed controls ( $M = 0.33$ ,  $SD = 1.54$ , range =0-8 )  $p$ 's  $< .001$ , who did not differ ( $p = 0.59$ ). Due to the small number of reported experiences (three experiences reported by two people) in the control group, only the PTSD and trauma-exposed controls were subsequently analysed. The results

indicate that the frequency of AWR differs between trauma survivors with and without PTSD (Research Question 1).

### *Triggers*

In the PTSD group the trigger was known for 54% of reported AWR experiences (Q5, Appendix B), and 30% in the trauma-exposed group. Percentage of triggers known (vs. unknown) did not significantly differ between these groups,  $t(43) = -0.31, p = .76$ . Participants who knew the trigger were asked to describe it. Triggers descriptions were classified into 9 categories. Initial category classifications were based on categories used in a previous study (Kleim et al., 2013). Four categories were added to capture triggers that did not fit into these pre-defined categories (Things that make you jump/Crowds/Nightmares/General stressors). Category assignment was done by the author and reviewed (un-blinded) with the supervisor (A. Ehlers). The most commonly reported trigger in the PTSD group was a perceptually similar trigger, and in the trauma-exposed group it was related conversations, physiological symptoms, or nightmares (see Table 4). This suggests that type of AWR triggers differ between trauma survivors with and without PTSD (Research Question 1) and in PTSD it is more commonly triggered by perceptually similar stimuli (to stimuli present during the trauma), which adds to the understanding of AWR phenomenology (Research Question 2).

### *Trauma memory*

A memory of the trauma was reported to follow for 29% of AWR experiences in the PTSD group (17% did not know) and 50% for trauma-exposed controls (10% did not know). This shows that trauma survivors with and without PTSD report different AWR

experiences (Research Question 1) and that in PTSD participants, two-thirds of AWR experiences occur in the absence of a subsequent trauma memory (Research Question 2).

**Table 4** Frequency of trigger type by group (N = number of times this was identified as a trigger. % = valid percent of overall known triggers), for participants who experienced AWR at Session 1.

Trigger type	PTSD	Trauma
	N (% of known triggers)	
Trauma-related conversation/reading	13 (22.81%)	1 (33%)
Physiological (incl. unwell, pain, tight chest, got up too quickly?)	3 (5.26%)	1 (33%)
Flashback/unwanted memory	3 (5.26%)	0
Thoughts about the trauma	2 (3.51%)	0
Perceptually similar trigger, situation, person	18 (31.58%)	0
Things that make you jump (something running out, people walking too closely behind)	5 (8.77%)	0
Crowds/confined space	2 (3.51%)	0
Nightmares	4 (7.02%)	1 (33%)
General stressors	7 (12.28%)	0

*Note. General stressors = argument, anger at UK politics, someone talking too quickly, the day ahead, bad news, dissatisfied with life, work stress; Perceptually similar triggers include walking same route home, seeing someone similar, driving, darkness, car horn, hearing screaming.*

### *Emotions*

Mean scores were calculated for each emotion (reported as part of an AWR experience) over seven days (Table 5). Group comparisons (independent t-tests) showed that people with PTSD rated re-experienced emotions of anger, horror, shame, disgust, heart beating fast, sweating and feeling short of breath significantly higher (more intensely) than trauma-exposed controls. Sadness was the most strongly rated emotion in both groups, followed by helplessness (see Table 5). The results show differences in AWR intensity (Research Question 1) between trauma survivors with and without PTSD.

**Table 5** Mean (SD) of emotional intensity (0-100) reported over 7 days (for those reporting AWR) at Session 1.

Reexperienced emotion	PTSD Session 1 <i>n</i> = 28	Trauma Session 1 <i>n</i> = 6	Group comparison <i>t</i> , <i>p</i>
Afraid	42.37 (24.09)	37.67 (27.96)	-.42, >.05
<b>Angry</b>	42.53 (25.22)	16.67 (23.26)	-.23, .03
Helpless	57.63 (26.79)	38.61 (29.07)	-1.55, >.05
<b>Horrified</b>	34.94 (25.60)	7.33 (8.64)	-4.56, <.001
Sad	58.97 (30.93)	41.72 (27.01)	-1.26, >.05
Guilty	28.88 (28.17)	11.5 (15.67)	-1.45, >.05
<b>Ashamed</b>	26.83 (27.85)	5.61 (10.50)	-3.09, .005**
<b>Disgusted</b>	25.52 (26.12)	0.5 (1.0)	-4.37, <.001
<b>Heart beating fast</b>	52.81 (20.46)	24.67 (20.75)	-3.04, .005
<b>Sweating</b>	31.64 (23.65)	9.11 (14.22)	-2.23, .03
<b>Feeling short of breath</b>	34.06 (24.28)	10.06 (15.71)	-2.30, .03
Shaking	25.73 (20.61)	14.61 (18.45)	-1.22, >.05
Feeling dizzy or faint	20.07 (20.84)	8.89 (15.59)	-1.23, >.05
An urge to leave the situation	53.83 (29.35)	32.42 (31.91)	-1.32, >.05
Other (please state)	29.23 (21.63)	22.61 (17.65)	-.70, >.05

*Note.* Bold text indicates significant difference between groups.

### **Modelling results**

#### a) *Do patterns of re-experienced emotions (AWR) co-occur over time?*

Patterns of emotions reported for AWR were analysed for the PTSD participants reporting AWR (*n* = 28). There were not sufficient data to analyse trauma and non-exposed controls. Agglomerative hierarchical cluster analysis with dimensions ‘time’ and ‘emotions’ revealed a two-cluster solution. The cluster tree was verified with a

correlation of 0.81 (greater than 0.75 is considered good). A three-cluster solution would have revealed a third cluster that consisted only of one emotion ‘angry’, therefore a two-cluster solution was chosen as the most informative (see Table 6).

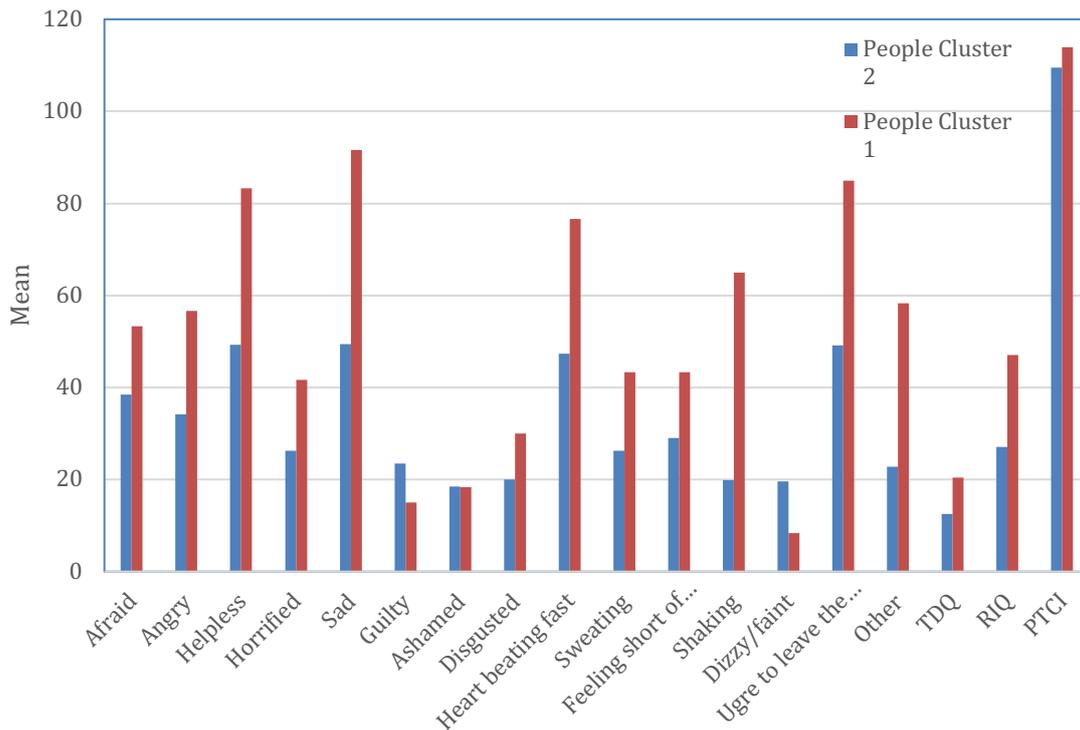
**Table 6** Emotion clusters at Session 1 for PTSD group (n = 28).

	<b>Cluster 1</b>	<b>Cluster 2</b>
<b>Emotions</b>	<ul style="list-style-type: none"> <li>• Horrified</li> <li>• Guilty</li> <li>• Ashamed</li> <li>• Disgusted</li> <li>• Sweating</li> <li>• Short of breath</li> <li>• Dizzy/faint</li> <li>• ‘Other’</li> </ul>	<ul style="list-style-type: none"> <li>• Afraid</li> <li>• Angry</li> <li>• Helpless</li> <li>• Sad</li> <li>• Heartbeat</li> <li>• Urge to leave</li> </ul>
<b>‘Other’ emotions</b>	“Alert, anxious, exhausted, unease/dread, hot, irritable, jittery, chest pain, on edge, panic, overwhelmed.”	
<b>‘Other’ behaviours</b> <b>Re-labelled: Redirection of attention and cognitive avoidance</b>	“Avoided looking at it, focused on breathing, listened to loud music”	

*Note.* ‘Other’ = free descriptive responses entered by participants.

As a second exploratory step, a model with the dimensions ‘people’ and ‘emotions’ (excluding time) explored whether participants differed in the patterns of emotions they reported (between-person variance). The model revealed that three people clustered together (Cluster 1), and 25 people clustered together (Cluster 2), suggesting that three people reported a different intensity or type of emotion (frequency of AWR did not differ). However, verification of the cluster tree showed that the solution was not as good when ‘people’ were included in the model ( $r = 0.69$ ), and the small sample size in on cluster 1 ( $n = 3$ ) limits the conclusions that can be drawn. Preliminary exploration of the means showed that the three people in cluster 1 rated the intensity of re-experienced emotions as higher than the individuals in cluster 2 and had higher scores on all baseline measures (see Figure 3). However, as this analysis was for exploratory

purposes, and cluster 1 included only three people, the results must be interpreted with caution. Together these results suggest that certain emotions are more commonly reexperienced together (co-occur) over time, and that there may be individual differences in the intensity of re-experiencing in participants with PTSD and AWR (Research Question 2).



**Figure 3** Mean emotions ratings and sum scores (PTCI, TDQ, RIQ) for participants in cluster 1 (n = 3) and cluster 2 (n = 25).

*b) Are patterns of re-experienced emotions associated with particular cognitive-behavioural responses to AWR?*

Linear mixed-models (using cluster mean scores as time-varying predictors) with bootstrapping (of standard errors) showed that cluster two was significantly associated with dwelling, suppression, distraction, and leaving the situation. Cluster 1 was significantly associated with ‘other’ behaviours, which comprised a range of strategies of redirecting attention. Neither cluster predicted drug/alcohol use (see Table 7).

**Table 7** Results of linear-mixed models predicting cognitive behavioural-responses from emotion clusters.

Cognitive-behavioural responses	Emotion clusters					
	Cluster 1			Cluster 2		
	$\beta$	SE	<i>P</i>	$\beta$	SE	<i>P</i>
Dwelling	-0.01	0.14	0.92	0.85	0.11	<.001
Suppression	-0.12	0.17	0.46	0.74	0.15	<.001
Distraction	0.01	0.18	0.96	0.39,	0.15	0.01
Leaving situation	0.19	0.18	0.29	0.72	0.16	<.001
Drug/alcohol use	0.05	0.09	0.59	0.12	0.08	0.16
Other (redirecting attention)	0.41	0.12	<.001	0.21	0.11	0.06

**Note.** ‘Other’ behaviours is defined in Table 6. SE = standard error.

As a second exploratory step the ‘people’ cluster was added as a predictor of cognitive-behavioural responses into the model. Results showed that the three people (who clustered together) scored significantly higher on distraction ( $\beta = 23.53$ , SE = 11.32,  $p = 0.047$ ), marginally higher on suppression ( $\beta = 23.18$ , SE = 11.60,  $p = .056$ ), and no significant difference on dwelling ( $\beta = 9.48$ , SE = 12.22,  $p = 0.44$ ), leaving the situation ( $\beta = 19.00$ , SE = 13.14,  $p = 0.16$ ), or ‘other’ ( $\beta = 6.44$ , SE = 8.55,  $p = 0.46$ ). Results must be interpreted with caution due to the small sample of three people who clustered separately. Together these results show that re-experienced emotions in cluster 1 and 2 are associated with different cognitive-behavioural responses, and there may be individual differences in the extent to which responses are used (Research Question 2).

**Research question 3: does trauma type and baseline dissociation, response to intrusions and appraisals predict AWR?**

a) *Do baseline dissociation, response to intrusions and appraisals at baseline predict patterns of emotions (AWR)?*

Multiple linear regressions showed that cluster 1 of emotions during AWR was significantly predicted by greater baseline PTCI scores ( $\beta = 0.24$ ,  $SE = 0.06$ ,  $p < 0.01$ ), but not by RIQ ( $\beta = 0.26$ ,  $SE = 0.25$ ,  $p = 1.04$ ) or TDQ scores ( $\beta = 0.07$ ,  $SE = 0.24$ ,  $p = 0.31$ ). The overall model was significant  $F(3, 21) = 11.27$ ,  $p < 0.01$ ,  $R^2 = 0.62$ . Cluster 2 was significantly predicted by greater RIQ sum scores ( $\beta = 0.68$ ,  $SE = 0.33$ ,  $p = 0.04$ ), but not by PTCI ( $\beta = 0.04$ ,  $SE = 0.07$ ,  $p = 0.36$ ) or TDQ scores ( $\beta = 0.22$ ,  $SE = 0.32$ ,  $p = 0.50$ ). The overall model was significant  $F(3, 21) = 4.41$ ,  $p = 0.01$ ,  $R^2 = 0.39$ . This suggests that more negative appraisals (PTCI) predict greater intensity of reexperiencing cluster 1 emotions (horror, guilt, shame, disgust, sweating, short of breath, dizzy), and more maladaptive responses to intrusions (RIQ) predict greater intensity of reexperiencing cluster 2 emotions (afraid, angry, helpless, sad, heartbeat, urge to leave).

b) *Do trauma type, dissociation, response to intrusions and appraisals at baseline predict presence/absence of AWR?*

Logistic regressions showed that trauma type (interpersonal/non-interpersonal) did not significantly predict the occurrence of AWR (yes/no) in the PTSD group versus all groups (participants reporting no AWR in PTSD, trauma and health control groups),  $\beta = 0.66$ ,  $SE = 0.48$ ,  $p = 0.17$ . This suggests trauma type is not related to experiencing AWR. A multiple logistic regression including all three baseline cognitive process measures (TDQ, RIQ, PTCI) showed that the TDQ score uniquely predicted the occurrence of AWR ( $\beta = 0.10$ ,  $SE = 0.05$ ,  $p = .004$ ) but the PTCI ( $\beta = 0.01$ ,  $SE = 0.01$ ,  $p = 0.14$ ) and RIQ ( $\beta = 0.06$ ,  $SE = 0.04$ ,  $p = 0.09$ ) did not. Statistical comparison of the

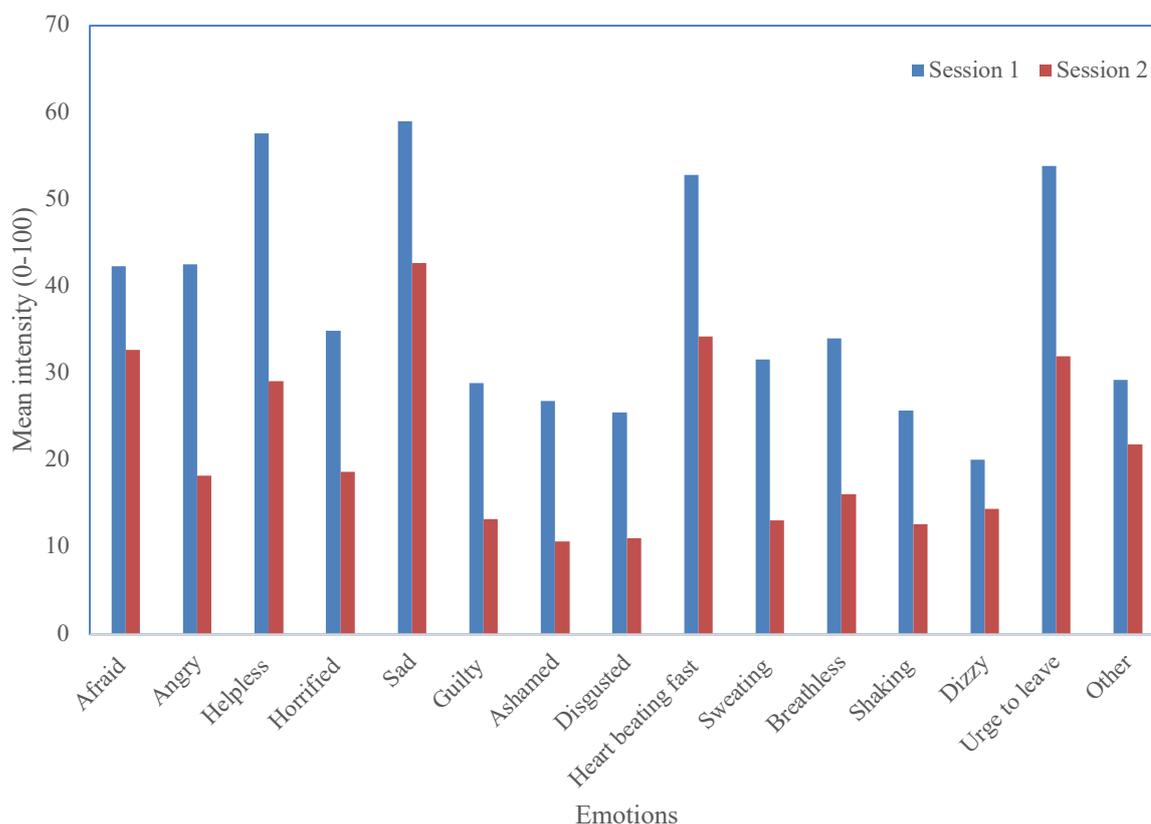
Beta values (z-test for comparison of unstandardized regression weights) for RIQ, TDQ and PTCI predicting presence/absence of AWR showed that TDQ was a significantly stronger predictor for the presence/absence of AWR than PTCI,  $z = 1.79, p = .036$ , and did not differ from RIQ  $z = 1.22, p = 0.11$ , and which also did not differ from PTCI  $z = 1.33, p = .09$ . This suggests that more severe dissociation predicts a higher likelihood of AWR, over and above negative appraisals or unhelpful responses to intrusions. Zero-order point-biserial correlations showed that all PTCI subscales were significantly, positively correlated with occurrence of AWR (yes/no): ‘negative thoughts about self’ ( $r = 0.55, p < .001$ ), ‘overgeneralisation of danger’ ( $r = 0.55, p < .001$ ), and ‘preoccupation with unfairness’ ( $r = 0.51, p < .001$ ) and ‘self-blame’ ( $r = 0.37, p = .001$ ). All three sub-scales of the RIQ were also significantly, positively correlated with the occurrence of AWR: suppression ( $r = 0.38, p < .001$ ), rumination ( $r = 0.62, p < .001$ ), numbing ( $r = 0.46, p < .001$ ). This shows that RIQ and PTCI likely predict similar and over-lapping variance. These results add to understanding AWR phenomenology by developing a profile of who may be more or less likely to experience AWR.

#### **Research question 4: Does AWR improve with CT-PTSD?**

##### ***Symptom and appraisal change with treatment***

Descriptive and full test statistics are shown in Table 3. Mixed ANOVAS showed a significant main effect of time, group, and group x time interaction for all measures (PCL-5; PTCI; RIQ; TDQ), indicating greater change in the PTSD group. Paired t-tests showed that all measures significantly decreased from pre- to post-treatment for PTSD patients, and RIQ and PCL-5 decreased from Session 1 to 2 for trauma-exposed controls.

Post-treatment (Session 2) CAPS assessment showed that 16 out of 23 (69.57%) PTSD patients no longer met criteria for PTSD diagnosis after treatment, and none of the trauma-exposed controls met criteria for PTSD diagnosis.



**Figure 4** Mean emotional intensity for PTSD participants reporting at least one AWR at pre- (n = 28) and post-treatment (n = 8).

***Affect without recollection: Descriptive statistics (Session 2)***

At Session 2, 21 PTSD patients and 29 trauma-exposed controls completed the app. Of these, 8 (38.10%) PTSD patients and three trauma-exposed controls (10.34%) reported at least one experience of AWR (PTSD range: 0–6; Trauma range: 0-3) (see Table 3). Three of the PTSD patients reporting AWR had not recovered from PTSD at post-treatment. In total there were 25 reports of AWR across both groups (PTSD: 18; Trauma: 7).

### *Frequency of AWR*

A mixed ANOVA showed a significant main effect of group (PTSD/Trauma), time (Session1/Session 2), and a significant group x time interaction (Table 3). PTSD patients reported a higher frequency of AWR than trauma-exposed controls at pre- and post-treatment. Planned paired t-tests showed that PTSD patients (all:  $n = 21$ ) reported a significant reduction in AWR frequency from pre to post-treatment, and trauma-exposed controls reported no change (see Table 3). Of the PTSD patients who reported at least one incidence of AWR at *both* Sessions 1 and 2 ( $n = 8$ ) there was a significant reduction in AWR frequency with treatment  $t(7) = 2.50, p = .04$  (Session 1: Mean = 10.63, SD = 5.80; Session 2: Mean = 4.88, SD = 2.36). This shows that AWR frequency is reduced with PTSD treatment, suggesting CT-PTSD improves AWR (Research Question 3).

### *Emotional intensity*

Mean emotional intensity ratings for PTSD patients reporting AWR at pre- ( $n = 28$ ) and post-treatment ( $n = 8$ ) are shown in figure 4. Paired t-tests showed that PTSD patients who reported at least one experience of AWR at both session 1 and 2 ( $n = 8$ ) had a significant reduction from pre to post-treatment in the rated intensity of fear,  $t(7) = 2.58, p = .04$ , helplessness  $t(7) = 2.81, p = .03$ , and the physiological sensations of heart beating fast  $t(7) = 3.59, p = .01$ , sweating,  $t(7) = 2.31, p = .05$  and feeling short of breath  $t(7) = 2.94, p = .02$  and the urge to leave the situation,  $t(7) = 3.39, p = .013$ . There was no change in the intensity of other emotions,  $p > .05$ . This shows that AWR intensity is reduced with PTSD treatment, suggesting CT-PTSD improves AWR (Research Question 3).

## Discussion

### Summary

A novel investigation was conducted into the previously defined (Ehlers & Clark, 2000) but not yet investigated symptom ‘Affect without recollection’ (AWR), using ecological momentary assessment (EMA) to capture this symptom and cognitive-behavioural responses in participants everyday life. Specifically, the study investigated 1) AWR differences between trauma-survivors with and without PTSD; 2) the phenomenology of AWR in PTSD; 3) predictions of AWR, and 4) whether AWR improved with PTSD treatment (CT-PTSD). Hypotheses were derived for research questions one, three and four, but not for question two due to its exploratory nature. The results confirmed the hypotheses. A summary of the results is as follows: 1) Frequency and intensity of AWR differed between trauma survivors with and without PTSD. Compared to trauma-exposed controls, people with PTSD reported a greater prevalence of and more frequent AWR (over seven days), higher intensity of emotional reexperiencing (on some, but not all emotions), triggers that were more commonly perceptually similar (to the trauma) and a lower likelihood of a subsequent trauma memory; 2) Exploration of the phenomenology of AWR revealed that two-thirds of PTSD patients reported AWR. In AWR in PTSD sadness and helplessness were the most strongly rated emotions, and certain re-experienced emotions were more commonly experienced together and fell into two distinct clusters. The emotions in cluster 2 (fear, anger, helplessness, sadness, heartbeat, urge to leave) were associated with active cognitive behavioural responses of dwelling, suppression, distraction and leaving the situation), whereas cluster 1 (horrified, guilty, ashamed, disgusted, dizzy, faint) was associated with other responses indicating a redirection of attention; 3)

Occurrence of AWR was predicted by more severe dissociation. The pattern of reexperienced emotions (cluster) were predicted by more negative trauma-related appraisals (higher PCTI scores) and unhelpful response to intrusions (higher RIQ scores), and finally 4) AWR improved with PTSD treatment, with fewer PTSD patients reporting AWR after treatment, and reduced AWR frequency (over seven days) and intensity (in predominantly cluster two emotions) in those still reporting AWR at post-treatment.

### **Group differences**

The results showed that nearly three quarters of participants with PTSD reported AWR, with an average of five (maximum 19 by individual participants) incidences over the week. This is smaller than previous studies using EMA, where an average of seven intrusions per week (maximum 41) have been reported (Kleim et al., 2013). This suggests that AWR may occur more rarely than reexperiencing symptoms that are identified as a trauma memory. Both the proportion of PTSD participants reporting AWR, and the frequency of AWR reported in the PTSD group was higher than trauma-exposed (14%) and non-exposed (7%) control groups, who did not significantly differ in the proportion or frequency of AWR (once in a week). These results reflected group differences in PTSD symptom severity, appraisals, and response to intrusions. In contrast, Kleim et al., (2013) found that trauma survivors with and without PTSD did not differ on the frequency of re-experiencing symptoms. However, Kleim and colleagues investigated reexperiencing symptoms that were recognised by participants as a trauma memory, and study inclusion criteria required that each participant was reporting at least one intrusion a week. This was not required in the present study therefore the results are likely to be more reflective of the true prevalence of AWR in

trauma survivors without PTSD. Reports of AWR in this group could be explained by sub-clinical PTSD symptoms. The results also suggest that AWR can be experienced in response to a negative but non-traumatic event, which could highlight the impact of strong emotion on memory. Studies have shown that in conditions of high emotion, perceptual memory is strengthened over other types of memory such as episodic (Arntz, de Groot, & Kindt, 2005; Kensinger, Addis, & Atapattu, 2011) and emotional memory, and receives prolonged processing (compared to neutral memories) in sensory memory (Kuhbandner, Spitzer, & Pekrun, 2011). Therefore, although it would not be classed as a reexperiencing symptom in response to a trauma, the results suggest that reexperiencing in the form of AWR may occur to highly emotive negative life events, perhaps via enhanced perceptual and sensory processing of emotional memory.

Sadness and helplessness were the most strongly rated reexperienced emotions in both groups. Trauma survivors with PTSD reported greater intensity of re-experienced anger, horror, shame, disgust, fast heartbeat, sweating and feeling short of breath compared to trauma survivors without PTSD. The groups did not differ on the intensity of fear, helplessness, sadness, guilt, shaking, feeling dizzy, or an urge to leave the situation. This is consistent with previous studies showing that reexperiencing in PTSD is associated with more intense emotional responses (including anger and shame) compared to trauma-survivors without PTSD (Kleim, Graham, Bryant, & Ehlers, 2013a; Michael, Ehlers, Halligan, & Clark, 2005b). The results differ from studies showing that participants with PTSD experienced more fear and helplessness in response to intrusions compared to trauma survivors without PTSD (Hellawell & Brewin, 2002; Kleim, Graham, Bryant, & Ehlers, 2013a). However, previous studies have measured emotional *responses* to reexperiencing, whereas the present study

measured *reexperienced emotions* as the content of reexperiencing. This could account for differences between these studies and suggests that trauma survivors with and without PTSD reexperience helplessness and sadness (among other emotions) with a similar intensity during AWR, but participants with PTSD reexperience more intense anger, horror, shame and disgust. These findings are also in line with studies proposing dominant emotions other than fear in PTSD (Andrews, Brewin, Rose, & Kirk, 2000; Ehlers & Clark, 2000; Hathaway, Boals, & Banks, 2010; Holmes, Grey, & Young, 2005).

Of those reporting AWR, trauma-survivors with and without PTSD did not differ on the proportion of known triggers but differed on trigger types. Triggers were known for up to half of AWR experiences which is lower than previous studies of ‘typical’ reexperiencing where participants reported knowing up to 65% of intrusion triggers (Kleim et al., 2013). In the PTSD group perceptually similar (to the trauma) stimuli was the most frequent trigger, whereas in the trauma-exposed controls it was equally related conversations, physiological symptoms, or nightmares. This is in line with the previous findings that perceptual trauma reminders (such as hearing a siren) are most commonly identified intrusive memory triggers by trauma-survivors (Kleim et al., 2013), and suggests that this may also apply to reexperiencing in the form of AWR. This is typically explained using associative learning theory (see ‘Conceptual introduction’, page 41) that associations form during a trauma between previously neutral stimuli and fear responses, which then form the basis of conditioned responses to these stimuli (Pitman, 1988). As associations often form via strong perceptual priming, individuals are often unaware of the triggers of the emotional response (Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007), consistent with the findings in this study. Previous studies

show that many triggers do not have to have meaningful associations with the trauma but are more likely to share perceptual features and be temporally associated (Ehlers et al., 2002; Ehlers, Hackmann, & Michael, 2004b). This may also apply to AWR, however conclusions cannot be drawn from this study about the mechanisms of triggering AWR and future work is needed to directly investigate this.

Trauma survivors with and without PTSD did not differ on whether a trauma memory followed an AWR experience. A trauma memory followed for 30% (PTSD) to 50% (trauma-controls) of participants, suggesting that AWR may as a trigger a subsequent trauma memory. However, for more than half, affect was reexperienced in the full absence of recollection of the trauma. This absence of a trauma memory supports the proposed definition of AWR (see 'Conceptual Introduction' page 37, and Ehlers & Clark, 2000) and is consistent with the posited functional independence of perceptual and episodic memory systems (see Brewin, 2014 for review), where the perceptual memory systems can '*store information about the person's bodily response to the trauma*' (Brewin 2003, p.110). The present study suggests that these bodily responses are reexperienced in AWR in the absence of information stored in episodic memory (Brewin, Dalgleish, & Joseph, 1996; Brewin, Gregory, Lipton, & Burgess, 2010) and without auto-noetic awareness (Tulving, 2002). Furthermore, Ehlers, (2015) emphasised important characteristics of the trauma memory suggested to maintain PTSD (Ehlers & Clark, 2000; Ehlers et al., 2004), such as 'disjointedness' of the worst moments of the trauma from other relevant autobiographical information (such as 'I survived'), and 'poor elaboration' (integration) of these worst moments with this information. AWR could represent the extreme of this; a trauma memory that is highly disjointed and poorly elaborated, to the extent that the emotion from the trauma is reexperienced in

the absence of any other autobiographical information (such as other aspects of the trauma memory), as shown by the finding that half of AWR experiences occurred without a preceding or subsequent trauma memory. In cases where a trauma memory followed AWR, the emotional experience may represent an internal trigger for a trauma memory (see Ehlers & Clark, 2000).

In sum, consistent with previous studies, reexperiencing in the form of AWR showed phenomenological differences between trauma survivors with and without PTSD. AWR in PTSD was reported as more frequent, and more emotionally intense than AWR in trauma-exposed controls, and more often occurred in the absence of a subsequent trauma memory or trigger awareness.

### **Exploration of AWR phenomenology**

In PTSD participants with AWR, exploration of the patterns of reexperienced emotions showed revealed that certain emotions co-occurred over time (seven days) and fell into two clusters. Horror, guilt, shame, disgust and physiological sensations sweating, short of breath, dizzy/faint, and ‘other’ were found to co-occur in one cluster (cluster 1). Fear, anger, helplessness, sadness, the urge to leave a situation and physiological sensation of fast heart-beat co-occurred in another cluster (cluster 2). ‘Other’ emotions consisted of free-text descriptions that appeared to consist of hyperarousal symptoms that were not listed in the app (e.g., alert, anxious, irritable, jittery, on edge). The separate clusters are consistent with EMA studies showing a negative correlation between shame and fear (more shame in response to an intrusion co-occurred with less fear) (Kleim et al., 2013). Emotion theories (Siemer, Mauss, & Gross, 2007) and cognitive models (Ehlers & Clark, 2000) have shown that different appraisals are associated with shame and fear.

For example, appraising a situation as life-threatening may be more likely to lead to fear, whereas thinking that you are to blame may lead to shame (see Siemer, Mauss & Gross, 2007). One possibility is that emotions associated with specific appraisals from the trauma clustered together, such as emotions related to self-blame (cluster 1) and life-threat (cluster 2). However, subsequent analyses showed that more negative appraisals predicted cluster one emotions only, whereas unhelpful responses to intrusions predicted cluster two.

A preliminary analysis also showed individual differences in the intensity of AWR; three participants reported more intense emotions and more severe baseline symptoms. This could simply suggest more severe PTSD, or another possibility is that strong emotional responses may predict more severe PTSD symptoms, as shown by previous experimental studies (Bub & Lommen, 2017). The results warrant further investigation as they cannot be properly interpreted due to the small sample size.

The results also showed that the pattern of emotions reexperienced predicted the type of cognitive-behavioural response. This is consistent with previous suggestions that perceptual intrusions may contribute to control processes (Brewin, 2014). The majority of cognitive behavioural responses (dwelling, suppression, distraction and leaving the situation) were associated with emotions in cluster two (fear, anger, helplessness, sadness, heartbeat, urge to leave). Cluster one emotions were associated with 'other' behaviours only, which was a free text option that responses indicating redirection of attention and cognitive avoidance. Similarly, EMA studies of PTSD participants' emotion regulation strategies in response to stressors showed that avoidance (pushing away negative emotions) was a commonly used maladaptive strategy which maintained

PTSD symptoms (Short, Boffa, Clancy, & Schmidt, 2018). One possibility is that cluster two emotions were more likely to lead to unhelpful coping strategies known to maintain PTSD (Ehlers & Clark, 2000). Whereas cluster one emotions, such as shame, could be more likely to lead to freeze or avoidance responses.

The results are inconsistent with a previous EMA study which did not find an association between emotional responses to intrusions and cognitive-behavioural strategies. However, in that study strategy-use was measured as a categorical assessment (yes/no) (Kleim et al., 2013). The present study measured degree of engagement (0-100) in strategies which provided a more nuanced measure and could explain the discrepancy in the results between these studies. The results are in line other diary studies which have found that trauma memories led to greater dwelling than non-trauma memories in participants with compared to without PTSD (Schönfeld & Ehlers, 2017). The cognitive model of PTSD (Ehlers & Clark, 2000) suggests that unhelpful coping strategies prevent change in appraisals and the nature of the trauma memory (Ehlers et al., 2005; Ehlers & Clark, 2000), thereby maintaining PTSD symptoms including reexperiencing symptoms, which encompasses AWR (Ehlers & Clark, 2000). Therefore, the cognitive-behavioural responses reported by patients in the present study could indicate unhelpful coping strategies that may be maintaining AWR. However, due to question wording, this was not explored in the present study. Future research could re-word the question and examine if there is a bi-directional relationship between coping strategies and AWR.

### **Predicting AWR**

More severe dissociation predicted a greater likelihood of AWR over and above negative appraisals or unhelpful responses to intrusions. It has been suggested that dissociation reflects an inability to process a traumatic event (Brewin et al., 1996; Ehlers & Clark, 2000; Foa & Hearst-Ikeda, 1996). Incomplete processing may lead to deficits in the trauma memory including memory fragmentation (Amir, Stafford, Freshman, & Foa, 1998) and disorganisation (Harvey & Bryant, 1998). This is consistent with evidence that fragmentation and disorganisation of trauma memories are predicted by dissociative reactions during and after the trauma (Halligan, Michael, Clark, & Ehlers, 2003a). Therefore, in the present study one explanation is that dissociation could lead to increased fragmentation and disorganisation of trauma memories which could be more likely to result in AWR. Future research would need to investigate this as a possible pathway.

Trauma type did not predict who had AWR. This is inconsistent with studies which have shown that survivors of more interpersonal trauma show more intense emotional reactions compared to those surviving non-interpersonal trauma (Amstadter & Vernon, 2008), but consistent with Kleim et al.'s (2013) findings that emotional response to reexperiencing was not predicted by trauma type.

Appraisals and response to intrusions predicted the nature (emotions reexperienced) but not the presence of AWR. More negative appraisals of the trauma and its consequences predicted reexperiencing of cluster one emotions (horror, guilt, shame, disgust, sweating, short of breath, dizzy), whereas more unhelpful responses to intrusions predicted reexperiencing cluster two emotions (afraid, angry, helpless, sad, heartbeat, urge to leave). This consistent with the cognitive model of PTSD which proposes that

negative appraisals maintain PTSD symptomatology, and maladaptive responses to intrusions (such as suppression) prevent change in the trauma memory (Ehlers & Clark, 2000), thereby accounting for continuing reexperiencing symptoms. This is supported by studies showing that appraisals predict the development (Ehlers & Steil, 1995; Halligan, Michael, Clark, & Ehlers, 2003b; Michael, Halligan, Clark, & Ehlers, 2007) and maintenance of PTSD (Kleim, Grey, Wild, Nussbeck, Stott, Hackmann, Clark, & Ehlers, 2013c), and studies showing that responses to intrusions such as rumination and suppression maintain PTSD (Ehring et al., 2008). The results of the present study suggest that appraisals and response to intrusions are also relevant to AWR, however further research is needed to determine their role in its development and maintenance.

These results add to understanding AWR phenomenology by developing a profile of who may be more or less likely to experience AWR. Consistent with cognitive models (Ehlers & Clark, 2000), the results suggest that it is the appraisals and response to the traumatic event (rather than the type of event itself) that predicted the present and type of reexperiencing symptoms.

### **PTSD treatment effects**

CT-PTSD treatment improved both the frequency and intensity of AWR. This is consistent with previous studies which showed that reexperiencing in PTSD decreases with successful treatment (Hackmann, Ehlers, Speckens, & Clark, 2004; Speckens, Ehlers, Hackmann, Ruths, & Clark, 2007), and suggests that CT-PTSD is also effective for treating AWR. In those still reporting AWR at post-treatment, the intensity of fear, helplessness, heart-beat, sweating, breathing, and urge to leave a situation was reduced, but intensity of cluster one emotions (such as shame) did not change. There is some

evidence that shame may be less responsive to exposure-based PTSD treatments than fear (Lee et al., 2001), however there is limited evidence that adding interventions that target shame enhance CBT for PTSD (Beaumont, Galpin, & Jenkins, 2012). Cognitive models of PTSD (Ehlers & Clark, 2000) suggest that appraisals related to shame (e.g., ‘it’s my fault’) should be targeted and modified in PTSD treatment (Ehlers et al., 2005). Specifically addressing flashbacks in therapy has also been shown to contribute to better outcomes (Nijdam, Baas, Olf, & Gersons, 2013). Therefore, specifically addressing AWR alongside modifying appraisals related to shame, may be useful for enhancing PTSD treatment outcomes.

### **Limitations and future directions**

The present study had a number of limitations. First, emotions were only rated if an individual confirmed that they had experienced a strong emotion in the last four hours. This was problematic for post-treatment analyses, as it meant that only data from individuals still reporting AWR could be analysed. Future studies should include emotion ratings for all participants, so changes in intensity of emotions with PTSD treatment can be explored, including in those no longer reporting AWR. Second, interpretation of the results is limited by the small sample size, despite using an analysis strategy that was specifically developed for small sample sizes. Larger PTSD sample sizes would be of benefit, particularly for post-treatment analyses. Third,nowness, vividness and distress have been found to differ between trauma survivors with and without PTSD (Ehlers, Hackmann, & Michael, 2004b; Kleim, Graham, Bryant, & Ehlers, 2013b; Michael, Ehlers, Halligan, & Clark, 2005a) but were not assessed in the present study. It would be informative to add this in future research. Fourth, adaptive cognitive behavioural responses were not measured. Adaptive responses such as

reappraisal have been linked to less severe PTSD symptoms (Boden, Bonn-Miller, Kashdan, Alvarez, & Gross, 2012). It is unknown whether some participants may have also engaged in adaptive responses to AWR and would be of interest to measure this in future studies. It would also be of clinical interest to see if reduction in AWR is accompanied by an increase in adaptive coping responses. Fifth, participants free text response to 'other' emotions indicated that there were emotions and physiological symptoms that were not included in the app and could be classified as 'hyperarousal' cluster of symptoms. The app should be expanded to include these in future research. Sixth, the number of times a participant experienced AWR in the previous four hours was not included in the app. Event-based (recording when reexperiencing is identified) versus time-based (responding to time prompts) have been found to yield different estimates of frequency of reexperiencing symptoms in PTSD (Kleindienst et al., 2017). Including a frequency estimate of AWR and comparing event-based and time-based measures would be of benefit in future work. Sixth, studies have shown that monitoring PTSD symptoms can have a therapeutic effect in the absence of intervention (Dewey et al., 2015). It is unlikely that this accounted for treatment effects in the present study as trauma-exposed controls showed no change in AWR symptoms. However, future research should investigate whether AWR symptoms improve with PTSD treatment in the absence of EMA monitoring, to exclude the possibility of a therapeutic effect of monitoring on AWR. Finally, in order to fully conclude that the reduction in AWR was due to treatment rather than time or repeated assessments, the study would have benefitted from a PTSD waitlist group as a control, rather than a trauma-exposed control group.

Future directions could also include developing the diary measure by including a camera to capture the triggering environment, which may be helpful for clinical work and for further understanding of AWR triggers, particularly when the trigger is unknown. It would also be of interest to explore participants' appraisals of AWR specifically, as it is possible that the nature of AWR as a surge of emotion in the absence of a memory could be more likely to result in appraisals such as 'I'm going mad' which could further maintain PTSD. Finally, prospective studies could explore whether factors identified in this study (dissociation, appraisals and response to intrusions) are also related to the development of AWR.

### **Clinical implications**

Despite limitations, the results of this study have a number of important clinical implications. It has provided evidence of the existence of the formerly anecdotal symptom of AWR in PTSD (Ehlers & Clark, 2000). The results provide insight into the everyday experience of AWR in PTSD using a method with high ecological validity and showed that AWR motivates the use of maladaptive coping strategies that are known to maintain PTSD (Ehlers & Clark, 2000). It has responded to an urgent call to understand reexperiencing symptoms in PTSD better, due to their clinical impact on clients (Brewin, 2015) and their potential to bring about huge therapeutic gains when targeted in PTSD treatment (Nijdam et al., 2013). A greater understanding of AWR will hopefully add to theory-guided treatment procedures for PTSD (Ehlers et al., 2005). Knowing that AWR exists and may play a role in PTSD maintenance emphasises that clinicians should also ask clients about AWR, rather than just known trauma memories. The experience of AWR could be more likely to lead to negative interpretations of memory characteristics such as gaps (e.g., 'I must have brain damage') which have

been shown to maintain PTSD (Dunmore, Clark, & Ehlers, 2001; Halligan, Michael, Clark, & Ehlers, 2003b), and change in appraisals has been shown to be a mechanism of change in PTSD treatment (Kleim, Grey, Wild, Nussbeck, Stott, Hackmann, Clark, & Ehlers, 2013c). Therefore, asking about AWR in therapy and providing psychoeducation could help to modify these appraisals and improve PTSD. The finding that reexperienced emotions such as fear were more likely to reduce than shame after treatment highlights shame as an important target in therapy. The information that negative appraisals predict AWR suggests that helping clients to understand AWR may also be useful to modify unhelpful appraisals such as 'I am going mad' which could maintain PTSD (Ehlers & Clark, 2000). Finally, an EMA approach could be useful in monitoring changes in AWR over treatment and help both patients and clinicians better understand this symptom and its triggers, which could be crucial information to bring into therapy.

## **Conclusion**

The present study provided novel evidence of the existence of a form reexperiencing called AWR (Ehlers & Clark, 2000), and the experience and consequences of this in everyday life in participants with PTSD. The results add to understanding of what AWR is, who gets it, what predicts it, and what the cognitive and behavioural consequences are. The results showed that the likelihood of AWR is increased with dissociation, and the type of emotions reexperienced are associated with negative appraisals and unhelpful responses to intrusions. The consequences of AWR include engaging in maladaptive coping strategies, and it is improved with PTSD treatment.

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## **Part 3: Critical appraisal**

## **Introduction**

This critical appraisal is a reflection on my experience of conducting the literature review described in Chapter 1 and the study described in Chapter 2, and the challenges I encountered in this process. Firstly, it includes my reflections on selecting this topic, and factors that may have influenced my theoretical stance in the conceptual introduction; Secondly, I discuss the process of developing the research questions in Chapter 2; Thirdly, I reflect on the methodology I used, including choosing a daily measurement technique and developing a smartphone application; Fourthly, I reflect on the analysis process – specifically developing a modelling strategy, and finally, I discuss which aspects I would change if I were to repeat the study.

## **Reflections on selection of subject area**

Posttraumatic stress disorder (PTSD) has been a long-standing interest of mine. PTSD and sleep disturbances were the topic of my PhD and post-doctoral research. This interest evolved with my clinical work on the D Clin Psy, particularly my specialist placement at Freedom from Torture in my final year. As a result of my clinical practice, my research interests grew beyond just sleep disturbances in PTSD as I began to witness first-hand some of the reexperiencing symptoms that I previously only read about, such as affect without recollection (AWR) – the topic of this thesis. My PhD research supervisor and external DClinPsy supervisor (Anke Ehlers) had defined this symptom in her cognitive model of PTSD (Ehlers & Clark, 2000), but no study had been conducted to determine its existence. I personally became particularly interested in exploring the data on this further when I began to witness this symptom in my patients at Freedom from Torture. At this point the definition set forward by Ehlers & Clark

(2000) of the possibility of a reexperiencing symptom whereby emotions/physiological reactions were reexperienced without an accompanying trauma memory, suddenly made sense to me. I watched during reliving as my patients clutched their stomachs without noticing, until I asked if they felt pain there? They would look confused and then look down and only then realise what they were doing but would not be able to say why. It was only during the reliving that it would become clear that they had been stabbed there, and I would ask if they could feel pain there like they did at the time? Which would lead to us making the link between this detached physical pain that they reexperienced and an event that happened during the trauma, that had until now been experienced in detachment of a trauma memory or even a link between the two. From my clinical work I observed that AWR appeared to be highly prevalent this population of torture survivors, who had experienced severe, prolonged torture and now suffered PTSD typically with high levels of accompanying dissociation. This drove my interest in researching AWR formally, in an attempt to define AWR and to provide the first evidence of its existence in clinical populations. It seemed clear to me, as it is to most trauma clinicians, that this symptom existed, however it remained anecdotal until this research was conducted. Until analysing the results I remained unsure as to whether I would find evidence of this symptom.

### **Reflections on conceptual introduction**

In the conceptual introduction I attempted to review the literature on reexperiencing symptoms in PTSD more generally, as this is the only available literature that could be applied to AWR. This was relevant as AWR is defined as a reexperiencing symptom. I hoped that in reviewing this literature it would lead me to being able to propose a more formal definition and some possible theoretical underpinnings of AWR. My initial

review of the literature was influenced by the cognitive model of PTSD developed by Ehlers and Clark (2000), which is the model I have worked most closely with academically and clinically. I was also heavily influenced by the dual representation theory developed by Brewin, Dalgleish, & Joseph, (1996; see also Brewin, Gregory, Lipton, & Burgess, 2010). These models and their predictions no doubt influenced the lens with which I reviewed the literature, and the theoretical underpinnings I proposed for AWR which was drawn from these. I am aware of some controversies in the study of reexperiencing and memory in PTSD, particularly disagreements about which memory mechanisms are responsible for some of the reexperiencing phenomena evidenced in PTSD (see Ehlers, 2010 for review). However, I felt this was beyond the scope of the conceptual introduction and did not describe this in much detail as a result. The results of this thesis may contribute to this debate, and so it may be something to explore in greater detail in future research. My clinical work perhaps also played a role in my reading around possible mechanisms of AWR. In particular, witnessing high levels of dissociation in the client group I was working with who also reported high levels of AWR (anecdotally) led me to read more on the role of dissociation in memory in PTSD. Although other authors have investigated these links previously (Halligan, Michael, Clark, & Ehlers, 2003; Ozer, Best, Lipsey, & Weiss, 2003). My clinical work with torture survivors likely also influenced my suggestion that AWR may be more likely after more prolonged or high stress traumas. The field of memory research generally and memory research in PTSD is broad, and there are areas that warrant further discussion and exploration that might also be related to AWR. For example, I did not discuss possible neurobiological underpinnings of AWR, which have been described by Brewin and colleagues (1996; 2010) in relation to memory in PTSD. I also did not discuss in depth the literature on memory disorganisation and

disjointedness in PTSD, which is an emerging field of research (Sachschaal, Woodward, Wichelmann, Haag, & Ehlers, 2019) that may have relevance. These areas could be explored in future explorations of literature relevant to AWR. Finally, I developed a definition of AWR in collaboration with Anke Ehlers. We discussed clinical examples and anecdotal reports from PTSD patients of experiences that could be considered AWR. We developed a definition drawn from Ehlers & Clark's (2000) original description of AWR, expanding it to make it more testable for an empirical quantitative research project (see Reflections on Methodology, below). This was of great use for developing more specific research questions that could be investigated in the empirical paper.

### **Establishing research questions**

I set out with some simple aims: to determine whether AWR as defined by Ehlers & Clark (2000) existed, and what its characteristics were and whether it was improved with treatment. The nature of my research was exploratory, as nothing yet was known about AWR other than a possible definition. My literature review on reexperiencing symptoms in PTSD led me to develop some specific research questions, still with an exploratory, curious stance. I held in mind that I did not yet know whether AWR existed, and so it was difficult to propose highly specific research questions without having established this. However, working from the literature and guided by theoretical assumptions, I developed a number of research questions to guide my exploration of the AWR data. For example, informed by the research that reexperiencing differs between trauma survivors with and without PTSD, I asked whether this was also true of AWR. Based on the theoretical predictions I had developed I asked whether AWR was predicted by dissociation, trauma type, and cognitive behavioural coping strategies

such as response to intrusions. As noted previously, this was no doubt influenced by my clinical work, where I observed high levels of dissociation in clients with severe traumas (torture) accompanying AWR. Finally, I wanted to know whether PTSD treatment improved AWR, and so I asked this question quite simply. This was derived from both academic and clinical interest, and with a view to providing a study with clinical implications.

### **Reflections on methodology**

Investigating AWR presented a challenge – how do you ask participants about something that you (the researcher) have conceptualised as a reexperienced trauma memory, but by definition you expect that participants do not identify it as a trauma memory? To do this, we developed a description of AWR that asked only if participants had experienced a ‘strong emotion or bodily sensation or urge to leave a situation in the last 4 hours.’ This avoided the difficulty of asking about a memory that was not identifiable by participants as a memory. If the answer to this general question was ‘yes’, then further questions could be asked to discover whether it felt similar to how they felt in the trauma, and what they did in response to this. Given the difficulty of asking about something that by its nature and definition was going to be difficult for participants to identify, I was reluctant to use a retrospective questionnaire as it would be difficult for participants to remember, or to understand what was being asked. Therefore, I chose to use an ‘in the moment’ measurement technique that would help me to ask regularly about this symptom. This felt well-suited to a study that was an initial exploration of a phenomenon that had not yet been studied. I aimed to gather regular and detailed data on the experience of this symptom in participants daily lives. Ambulatory assessment such as this is becoming more widely used, as it is recognised

that it avoids difficulties such as retrospective recall bias and forgetting and has high ecological validity. It produces more data than a retrospective questionnaire which requires participants to average over a week, and instead provides data that is likely more accurate, can be analysed in more detail, and can be summarised to an average that is more reflective of the 'ground truth', if the researcher chooses to do so. Therefore, inspired by a previous study investigating daily experiences of intrusions in PTSD (Kleim, Graham, Bryant, & Ehlers, 2013), I chose to use an ecological momentary assessment (EMA) approach in a similar format to this study. However, while the study by Kleim and colleagues (2013) used specific equipment for participants to record their intrusions on, I wanted to find a way to measure this that was as easy as possible for participants to incorporate into their daily lives, in an attempt to make it as ecologically valid as possible and to reduce participant burden. Therefore, I developed an application for smartphones that participants could download and use and provided iPod touches to participants who did not have a smartphone, to avoid biasing the study towards those who could afford their own. I was relieved to see that the completion rates suggested this was very acceptable to participants. One difficulty with this approach, which I discussed in the limitations section of Chapter 2, is that it remains unknown whether this measurement technique may have biased participant's reporting. This is of particular interest when attempting to measure something like AWR, as asking questions such as 'was it similar to how you felt in the trauma' may have created this link for participants and influenced later experiences or reporting of AWR. Unfortunately, this is something that was hard to avoid, and could be investigated in future research by comparing AWR rates using different measurement techniques.

## **Reflections on analysis process**

EMA time-series data is well suited to a more complex modelling approach. As this study was an exploration of a new phenomenon, I was keen to use an analysis technique that made the most of the data and did not involve simply averaging the results. I also wanted to use a technique that applied very few assumptions to the data, in order to engage in an unbiased exploration that retained the possibility of discovering things in the data that I had not thought of asking or hypothesising. Therefore, after establishing the existence of AWR using a simple frequency measure, where one or more experience in the week counted as having the symptom, I engaged in a more advanced modelling process. In collaboration with a statistician, Esther Beierl, we explored the data using a machine learning technique that is well-suited for exploring data sets such as this. As I had no prior information about whether AWR existed or the likely frequency of it if it did, it was difficult to know what size sample was required to produce enough data for modelling. Therefore, I was somewhat disappointed to realise that the number of data points counted as ‘small’ for modelling purposes. However, this was solved by using a modelling technique that was specifically developed for the analysis of EMA data with a small sample size, and so was perfect for this dataset. The modelling process itself took many months. It produced results I had not expected (such as the clustering of emotions) and required a constant return to theory to understand the results produced and to decide on the next step of the modelling process. We went through a number of analysis phases, including decisions such as whether to explore AWR in a combined group of trauma-survivors with and without PTSD, or in PTSD alone. Eventually I took the decision to analyse AWR in PTSD participants only, guided by the evidence base that differences exist in reexperiencing between trauma survivors with and without PTSD. I also took this decision as I wanted the results to be as useful as possible to

clinicians working to treat PTSD. I had hoped to separately model the AWR data from the trauma-survivors without PTSD, however the available data was too small to reliably interpret. As there were (and are still) many different ways this data could have been analysed, it was a challenge to focus the analysis on areas that were deemed most important for this initial exploration of AWR. I settled on exploring whether emotions co-occurred together over time, and whether they predicted cognitive behavioural responses to these emotions. I am aware that cognitive-behavioural strategies can also maintain reexperiencing symptoms, and therefore could also maintain AWR (Ehlers & Clark, 2000). However, the wording I used in the app asked what participants did '*in response to*' the emotion/physical sensation. Therefore, it did not seem appropriate to model whether coping strategies predicted emotional reexperiencing intensity, which is a question that remains to be explored for AWR. Finally, I struggled with the decision of whether to analyse and report AWR data from non-trauma-exposed controls. I had included them as a comparison against the trauma-exposed controls with and without PTSD, expecting that they would not report AWR. I was surprised to find that some did. I chose to include this data as, similar to literature showing that intrusions occur outside of PTSD, it suggests that strong emotions can be reexperienced after a negative but non-traumatic event, which may be of interest for literature on emotional memory. Finally, the potential for circular reasoning in the empirical study must be noted. Asking participants to recollect something that by definition is unlinked from a trauma memory, may cause them to link it to the trauma memory. In addition, taking part in a study that has posttraumatic stress disorder in the title (See Appendix C for the participant information sheet) may also have had an impact on participants, including social desirability bias – perhaps to provide responses, or to make links between their experiences and trauma exposure, which they may not have done before. This could be

addressed in future studies through less explicit ways of measuring reexperiencing symptoms, such as ambulatory measurement, which is discussed below.

### **Reflections on limitations and changes**

As discussed in Chapter 2, this study had a number of limitations. These will not be repeated in detail here, but there are a number of things I would do differently were I to conduct this study again. Firstly, I would include a comparison group to determine what impact measurement was having on participants reports of AWR, in case the act of reflecting on and reporting the symptom was biasing the results. I would also gather a larger sample of PTSD participants to produce enough data to study the symptom in more detail. Secondly, I would also consider recruiting a sample of trauma-exposed participants who were reporting at least one experience of AWR a week, similar to the approach taken by Kleim et al., (2013). This would enable a comparison of AWR in trauma-survivors with and without PTSD, which could be informative. Thirdly, I may also change the wording of question ‘what did you do in response to the feeling’ to ‘what did you do while you were feeling this’, to explore bi-directional relationships between coping strategies and AWR. Fourthly, I would possibly also remove the first question of whether they ‘had a strong emotion/bodily sensation in the last 4 hours’ and replace it with emotion intensity ratings. This would provide data on emotional intensity throughout the day, which could serve as emotional baseline data as a point of comparison and would include those who have not identified that they had a strong emotion in the last four hours. Finally, I would consider using ambulatory measures of physiological arousal and activity, which could provide objective indicators of sudden strong emotions or physiological sensations. It would be of interest to see if these aligned with the frequency of participant reports, as has been done in previous studies

comparing subjective and objective measures of symptoms in PTSD (Tsanas, Woodward, & Ehlers, 2020). As the study was the first to explore the existence of AWR, it was important to try not to over-stretch the conclusions that could be drawn from this initial study. However, the results provided an interesting insight into this symptom, and opened up lots of avenues for future research.

## **Conclusion**

In conclusion, undertaking this research required a balance between a curious, open-minded approach required for exploratory research, with a theoretically driven approach to developing research questions and deciding on analysis approach. This was at times challenging, due to the paucity of literature on AWR specifically. This was my first experience of conducting a study into something that has not been researched before. This challenged me to accept the inevitable limitations and learning that comes with exploration of a new phenomenon, such as a long list of unanswered questions and possible future directions. Overall, I found this to be a thought-provoking process, which deepened my interest in memory and PTSD.

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# Appendices

## Appendix A

### Life event checklist

Used in 'Part 2: Empirical Paper'. Combines traumatic and non-traumatic life events.

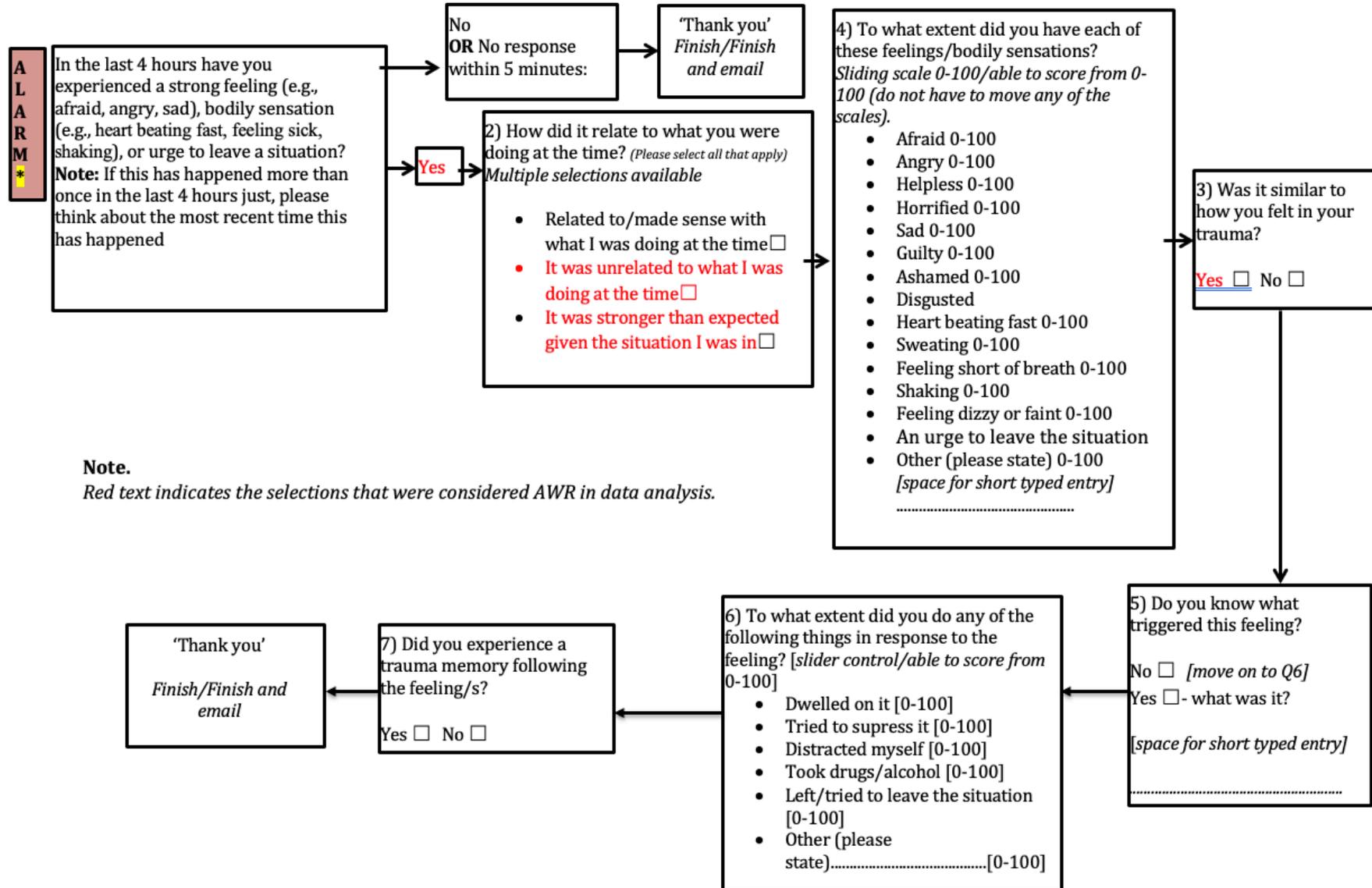
Many people have lived through or witnessed a very stressful event at some point in their lives. Indicate whether or not you have experienced each event listed below by circling **Y** for Yes or **N** for No.

1. Break-up of relationship	Y	N
2. Major argument with partner	Y	N
3. Parents separating	Y	N
4. Major argument with family or friends	Y	N
5. Betrayal by someone important to you	Y	N
6. Being excluded from something important to you	Y	N
7. Death of someone close to you	Y	N
8. Sudden, traumatic death of someone close to you	Y	N
9. Negative experience at work or school (e.g., failed exam, lost job)	Y	N
10. Serious problem with living situation	Y	N
11. Losing something important to you (e.g., phone, passport, sentimental item)	Y	N
12. Scary situation when travelling (e.g., when swimming, hiking)	Y	N
13. Dangerous encounter with animals	Y	N
14. Non-violent crime (e.g., victim of fraud or theft)	Y	N
15. Natural disaster (e.g., tornado, hurricane, flood, major earthquake)	Y	N
16. Serious traffic accident, (e.g., car, bike, train, or boating accident)	Y	N
17. Serious other accident, fire, or explosion (e.g., accident at work, fire at home)	Y	N
18. Serious but not life threatening illness	Y	N
19. Injury (e.g., breaking a bone)	Y	N
20. Life threatening illness	Y	N
21. Non-sexual assault (e.g., being mugged, shot, physical attacked, stabbed or held at gunpoint)	Y	N
22. Sexual assault (for example, rape or attempted rape)	Y	N
23. Abuse or neglect in childhood	Y	N
24. Military combat or a war zone <i>Please indicate whether you were: civilian / military personnel</i>	Y	N
25. Other violence inflicted by other people (e.g., abduction, bombing, torture)	Y	N
26. Witnessing others die / being seriously hurt	Y	N
27. Other stressful event <i>Please specify:</i>	Y	N

**PLEASE CIRCLE THE TWO EVENTS THAT YOU FOUND MOST UPSETTING**

## Appendix B

### Affect without Recollection smart-phone application



**Note.**  
 Red text indicates the selections that were considered AWR in data analysis.

\*(3 times a day every 4 hours. Set times 10am, 2pm, 6pm, 10pm) Set alarms with patient and label them. Set automatic text reminder too.

## Appendix C.

### Participant information sheet

*Note.* '[ ]' indicates text not in the participant information sheet. Researchers deleted as appropriate.

#### Participant Information Sheet Sleep, Affect and Memory in Posttraumatic Stress Disorder (PTSD)

**We would like to invite you to take part in a research study looking at the relationships between memory, sleep and trauma.** Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take the time to read the following information carefully. You can ask the researcher who has given you this information sheet any questions you may have about this study. If you are happy to participate, you will have the opportunity to ask further questions, and sign a consent form to join the study.

#### **What is the purpose of the study?**

Experiencing a trauma can sometimes lead to unwanted memories of the event, and difficulties sleeping. This study aims to better understand how trauma affects memory and sleep, and to do this we are recruiting 35 individuals who have experienced a trauma but who do not have PTSD and 35 individuals who have not experienced a trauma, to compare to a group of 35 participants who do have PTSD. The data from this study will contribute to two doctoral theses, and the results may help future refinement of treatment for PTSD and enable a better understanding of the effects of trauma.

#### **Why have I been invited?** *[researcher deleted as appropriate]*

You have been invited because you have experienced a traumatic event, but do not have a diagnosis of PTSD. *[trauma-exposed group]*

**[OR]**

You have been invited because you have received a diagnosis of PTSD, which you are waiting or receiving treatment for. *[PTSD group]*

**[OR]**

You have been invited because you have not experienced a traumatic event and do not have a diagnosis of PTSD. *[control group]*

**Do I have to take part?** You do not have to take part in this study, and may choose not to take part without providing a reason. *[trauma-exposed/control group]*

**[OR]**

You do not have to take part in this study, and you may choose not to take part without providing a reason, and without it affecting your right to NHS treatment. *[PTSD group]*

#### **What will happen to me if I take part?**

A researcher from the team will contact you to discuss this information sheet and any questions you may have, and also to ask you a few short questions to see if you might be suitable to take part in the study. If you are suitable and choose to participate, you will then also arrange to come to your first research session at the **Henry Wellcome Building, Institute of Psychiatry** or the **Oxford Centre for Anxiety Disorders and Trauma, University of Oxford**.

Before coming to your session you will be posted some questionnaires about your sleep and how you feel in general to complete at home and bring with you. These will take between 20 and 30 minutes to complete. At your first research session you will complete some additional questionnaires and 4 short tasks looking at memory. This session will last approximately **up to 2 hours** in total.

**Over the week following your first research session**, we will measure how well you sleep. You will have a short sleep diary to complete each morning and evening. Each will take about 5 minutes to complete, so 10 minutes in total per day. You will also have a small lightweight wristband to wear called an ‘actigraphy watch’, which will record your sleep and wake. This looks like a watch and can be worn on your non-dominant hand. During that week you will also be asked 4 times a day (at 10am, 2pm, 6pm and 10pm) to answer a few short questions about your mood, which you can record on an app on your phone (or on paper if more convenient). For the first day you will also have a small, lightweight heart rate monitor to wear, which goes under your clothes on your chest, and you will take 3 samples of your saliva (to measure cortisol, a stress hormone) once in the morning, once in the afternoon and once in the evening, full instructions will be given on how to do this.

You will then meet very briefly with the researcher to return the equipment and to arrange your second research session **3 months later**. This will be identical to the first research session, and will also last about 2 hours, therefore the study will last for a total of 7 weeks.

### **Expenses and payments**

You will be reimbursed a fixed total amount of **£120** for your time and travel expenses. You will receive **£60** when you return the equipment after the first week of monitoring your sleep, and **£60** when you return the equipment for the second time after a second week of monitoring.

### **Will my taking part in this study be kept confidential?**

**All information you provide will be strictly confidential.** Your name will be removed from your questionnaires and the anonymised research data will be stored on computers at the Department of Experimental Psychology, University of Oxford. The data will be given a unique code that will be used in all subsequent data analysis and does not contain your name or any other personal information. Cortisol samples will also have your name removed from them, and identified only by a unique code. Anonymised cortisol samples will be stored in locked freezers at the Institute of Psychiatry, before being analysed by a research group in the School of Biomedical and Molecular Sciences at the University of Surrey.

Responsible members of the University of Oxford, Oxford Health Foundation Trust or South London and Maudsley NHS trust may be given access to data for monitoring and/or audit of the study to ensure we are complying with regulations.

The results from this study may be published within the next 7 years. You will not be personally identified in any literature. You can obtain a copy of any publications from the contact numbers below and have the option on the consent form to select to be contacted about the results of the study. If you do consent to this then your contact details will be retained for this purpose.

You will also have the option to consent to being contacted about future research projects. If you consent to this then your contact details will be stored so that we may contact you about upcoming research. If we do contact you, you will be under no obligation to participate.

**What will happen if I don't want to carry on?**

You have the right to withdraw from the study at any time, without having to give a reason. If you withdraw, any data and samples already collected will, with your consent, be retained and used in the study and analysis, however no further data will be collected.

**Who is organising and funding the research?**

The study is a collaboration between the Oxford Centre for Anxiety Disorders and Trauma (which is part of the University of Oxford), the Centre for Anxiety Disorders and Trauma in London (which is part of the NHS) and the Institute of Psychiatry (which is part of King's College London). It is funded by the Wellcome Trust, a medical charity, the Medical Research Council, and the German National Academic Foundation.

**Who has reviewed the study?**

All research in the NHS is looked at by an independent group of people, called a Research Ethics Committee, to protect your interests. This study has been reviewed and given a favourable opinion by the South-Central Oxford C Research Ethics Committee.

**What are the possible benefits of taking part?**

We hope that the information we get from this study will help us inform understanding of how trauma affects memory and sleep, and to better understand how treatment works and to improve treatment effects on areas such as sleep for people with PTSD.

**What if there is a problem?**

Given the nature of this study, it is highly unlikely that you will suffer harm by taking part. However, the University of Oxford, as Sponsor, has appropriate insurance in place in the unlikely event that you suffer any harm as a direct consequence of your participation in this trial.

If you wish to complain about any aspect of the way in which you have been approached or treated during the course of this study, you should contact Lizzie Woodward or Juliane Sachschal Tel. **01865 618 608/6** or Professor Anke Ehlers (**01865 618602**) or you may contact the Research Ethics Office ([rec@kcl.ac.uk](mailto:rec@kcl.ac.uk)) who will re-direct your complaint as appropriate, or you may contact the University of Oxford Clinical Trials and Research Governance (CTRG) office on 01865 572224 or the head of CTRG, email [ctrig@admin.ox.ac.uk](mailto:ctrig@admin.ox.ac.uk)

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