

Posttraumatic Stress Disorder

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### **Abstract**

The chapter begins by describing how PTSD is diagnosed, contrasting the ‘broad’, inclusive DSM formulations with the ‘narrow’ formulation focusing on core symptoms introduced in ICD-11. The ICD-11 distinction between PTSD and Complex PTSD is also described. We go on to consider why PTSD has been regarded as a disorder of memory, and the two signature changes consisting of vivid re-experiencing of the traumatic event in the present coupled with impaired voluntary recall of the event. Other aspects of memory affected in PTSD that are unrelated to the traumatic event include a general bias toward recalling negative rather than positive stimuli, verbal memory deficits, and difficulties in retrieving specific memories and suppressing unwanted memories. We discuss three prominent controversies, whether traumatic memories are ‘special’, whether traumatic events can be forgotten, and whether there is evidence for delayed onset PTSD. Contemporary theories of PTSD are then described including fear conditioning, neo-conditioning theories, the Ehlers and Clark cognitive model, and dual representation theory, all of which identify deficits in memory for context as central to the disorder. In the final section we discuss how traumatic memory changes with successful treatment and the likely mechanisms involved in psychological therapy for PTSD: Habituation/extinction, updating/reconsolidation, and metacognitive change.

## **What is PTSD and how to Diagnose it**

Posttraumatic stress disorder (PTSD) is a psychiatric condition arising mainly, but not wholly, from exposure to an extremely stressful event or events that generally involve actual or threatened death, serious injury, or sexual violation. It was first introduced into the Diagnostic and Statistical Manual (American Psychiatric Association, 1980) because of the need to recognize that exposure to traumatic stress could have severe long-term as well short-term psychological consequences. For a number of years PTSD was defined in terms of the presence of three symptom clusters, re-experiencing of the traumatic event, avoidance and numbing, and hyperarousal. In the most recent iteration, DSM-5 (American Psychiatric Association, 2013), PTSD is the most complex diagnosis, consisting of 20 symptoms divided into four symptom clusters (intrusion, avoidance, negative alterations in cognition and mood, and alterations in arousal and reactivity). As many symptoms are common immediately after the trauma, improving naturally, the criteria have to be met for at least one month and cause significant distress or functional impairment. A similar condition, acute stress disorder, can be diagnosed within the first month.

Although the introduction of the diagnosis has been enormously influential and led to a great deal of research, several problems have been recognized. Firstly, the symptoms are not unique to traumatic events but often occur following other sorts of stressor (Larsen & Pacella, 2016). Intrusive memories, for example, are commonly found in most psychiatric disorders (Brewin, Gregory, Lipton, & Burgess, 2010). Secondly, over half a million different combinations of symptoms can result in the diagnosis (Galatzer-Levy & Bryant, 2013). Thirdly, there are high levels of comorbidity with other diagnoses, particularly anxiety disorders and depression (Brady, Killeen, Brewerton, & Lucerini, 2000).

In 2019 the World Health Organization introduced the 11<sup>th</sup> edition of the International Classification of Diseases (ICD-11), one of the aims of which has been to simplify diagnoses by focusing on the symptoms that best distinguish the disorder from others. In ICD-11 PTSD is a condition that is diagnosed from the presence of three core elements, re-experiencing the traumatic event(s) in the present, deliberate avoidance, and a continuing sense of threat. At least one out of two symptoms indicating the presence of each element is required, yielding 27 different combinations of symptoms (along with evidence of functional impairment) that can produce a diagnosis. ICD-11 recognizes a separate disorder, Complex PTSD, which requires that in addition to these three core elements the person also reports evidence of disturbances in affect regulation, negative self-concept, and difficulties in relationships, along with functional impairment.

Studies on four continents have supported the distinction between PTSD and Complex PTSD as defined in ICD-11 and there is evidence that ICD-11 PTSD has lower comorbidity with depression than DSM-IV PTSD (Brewin et al., 2017). Although most of the evidence base to date relies on PTSD as diagnosed according to various versions of the DSM, in future it is likely that ICD-11 PTSD and Complex PTSD will feature more prominently. The greater specificity they offer may be advantageous in the search for biological markers of PTSD, which to date tend to overlap with the markers for other psychiatric disorders such as depression.

The remainder of the chapter is divided into sections including why PTSD has been regarded as a disorder of memory, the association between PTSD and aspects of memory unrelated to the traumatic event, and controversies touching on memory and PTSD. We describe contemporary theories of PTSD relevant to memory issues and make links with more general areas of the literature such as the distinction between item and context memory. Finally we discuss how memory is involved in the psychological treatment of PTSD.

### **Why is PTSD a Disorder of Memory?**

Since its inception memory phenomena have been central in the diagnosis of PTSD. Symptoms have referenced the enhanced re-experiencing of the traumatic events(s), with the recent clarification in DSM-5 that these primarily involve sensory memories (i.e., images and physical sensations) rather than verbal memories or thoughts. Both DSM-5 and ICD-11 also include another memory symptom, traumatic flashbacks. A third symptom in DSM-5 references an element of amnesia or difficulty in recalling all the important details of the events in an orderly fashion. Research has confirmed this combination of an excess of involuntary remembering coupled with impairments in voluntary recall (Brewin, 2011, 2014; Ehlers, Hackmann, & Michael, 2004), consistent with a more general dissociation between the voluntary and involuntary expressions of a memory (Lau-Zhu, Henson, & Holmes, 2019).

#### **Enhanced Re-experiencing**

Although most biological studies of PTSD have simply compared diagnosed samples with controls, a few have investigated how biological markers are associated with specific symptom clusters. For example, in one study there were no reductions in brain volume associated with scores on avoidance/numbing or hyperarousal, or to overall symptom severity, but greater re-experiencing scores uniquely predicted reduced volumes in middle temporal and inferior occipital cortex (Kroes, Whalley, Rugg, & Brewin, 2011). Similarly, in a study of gene expression in PTSD 1,040 differentially expressed genes were identified in participants with high versus low intrusion symptoms, but none were identified in participants high versus low in avoidance/numbing or hyperarousal (Rusch et al., 2019). These findings suggest that it is the re-experiencing symptoms of PTSD that provide the core of the disorder, with the others perhaps representing secondary reactions.

Re-experiencing in PTSD has been characterized by the presence of intrusive, distressing memories that repeatedly came unbidden to mind. These are usually brief and perceptually detailed, with visual details often predominating, and may consist of a series of scenes that are experienced as like a videotape (Ehlers & Steil, 1995; van der Kolk & Fisler, 1995). As noted above, most if not all psychiatric disorders are frequently accompanied by unwanted memories and images of distressing events (Brewin, Gregory, et al., 2010). Nevertheless, the intrusive memories of people with PTSD appear to contain significantly more sensory elements than those of people who are depressed (Ashbaugh, Marinos, & Bujaki, 2018; Parry & O'Kearney, 2014).

There has also been a realization that intrusive trauma memories in PTSD, compared to non-trauma memories or the trauma memories of people without PTSD, have the special feature of being re-experienced in the present or here-and-now (Brewin, 2015; Kleim, Graham, Bryant, & Ehlers, 2013; Schönfeld & Ehlers, 2017). This distortion in the sense of time clearly demarcates much re-experiencing in PTSD from the reliving associated with normal memory, in which past events that come to mind nevertheless feel as though they belong to the past. Importantly, re-experiencing trauma memories in the present is not just a clinical feature but appears to have functional significance: It is predictive of the course of the disorder over and above the effects of initial symptom levels (Kleim, Ehlers, & Glucksman, 2007; Michael, Ehlers, Halligan, & Clark, 2005).

This form of re-experiencing has traditionally been acknowledged within the DSM in the form of a PTSD 'flashback'. Although held by trauma clinicians to be, along with nightmares, among the most distinctive symptoms of PTSD (Keane, Taylor, & Penk, 1997), flashbacks have not been formally defined until recently. Now in DSM-5 and ICD-11 they are stated to encompass a spectrum of re-experiencing from a very brief sense that the events are happening again the here-and-now to a total absorption in the traumatic memory in which the person loses

all contact with their current environment, being temporarily unable to see or hear people in their immediate vicinity. Researchers have compared the nature of re-experiencing in PTSD versus other disorders that can occur after traumatic events (Bryant, O'Donnell, Creamer, McFarlane, & Silove, 2011). Only flashbacks and dissociative amnesia, another memory symptom, were specific to PTSD. In the structural MRI study by Kroes et al. (2011) described above, increased reports of flashback were the only individual re-experiencing symptom to predict reduced volume in the insula/parietal operculum and in the inferior temporal gyrus.

Re-experiencing in PTSD additionally includes both experiences that are recognized by the individual as memories of parts of the trauma (and thus can be compared with other autobiographical memories) and emotional and behavioral responses that replicate the individual's responses during the trauma, but are perceived as reactions to the present situation. Both groups of re-experiencing symptoms need to be explained by theories of PTSD and addressed in treatment.

### **Impaired Voluntary Recall**

Trauma clinicians are familiar with PTSD patients reporting puzzling gaps in their memories for the index event(s) or difficulty in constructing a coherent, ordered narrative. It is not uncommon for highly significant details to be recalled, apparently for the first time, during the course of psychological therapy (Ehlers, Hackmann & Michael, 2004). Studies of fragmentation and disorganization in traumatic memory have elicited written accounts of traumatic events and then applied one of two different types of analysis (Brewin, 2014). The most widely used method involves narratives being segmented into utterance units and the units judged for the presence of these qualities. Six studies using these methods, applied to samples of children or adults with PTSD or acute stress disorder, all found significantly more disorganization in the trauma narratives of PTSD patients than controls. The average effect size was substantial (Cohen's  $d = .72$ ). Two studies that compared trauma and non-trauma

memories within PTSD samples reported that trauma memories were more disorganized. A second method reviewed by Brewin (2014) involves having participants provide self-ratings of their narratives, and here the results have been much more mixed. No study, however, has shown significant effects in the non-predicted direction.

The conclusion that voluntary memories are impaired in PTSD has been challenged (Rubin et al., 2016). These authors contrasted the trauma narratives of 30 individuals with and 30 without PTSD, using individual single-item participant self-ratings, independent judge ratings of various aspects of narrative coherence and of overall coherence, and two sets of measures scored by computer algorithms assessing such constructs as word concreteness, referential cohesion, temporal connectives, and mentions of cognitive mechanisms and insight. No group differences were found.

It seems apparent that some methods reliably demonstrate evidence of impairment while others do not. One set of studies has analysed narratives at the level of utterance units – in addition, these studies all elicited very detailed narratives in which participants were urged to describe everything moment-by-moment in chronological order, including the worst moments and including their thoughts and feelings. Such methods are used in the clinic and typically trigger distressing involuntary memories and images. The other set of studies has used more general ratings applied to the narrative as a whole and has been less specific in their instructions. It is likely, therefore, that the first method has generated an estimate of local coherence, concerned with neighboring clauses in a text, and the second with global coherence, concerned with an entire text (Habermas & Bluck, 2000).

The contrasting studies can be reconciled by proposing that when considering the narrative as a whole, especially a general, well-rehearsed narrative that focuses on the outline of the trauma story, trauma and non-trauma memories are essentially similar in their levels of coherence or disorganization. At the level of individual utterance units, however, amnesic



gaps, other types of fragmentation, and evidence of disorganized thoughts will be present when a highly detailed narrative is elicited that includes a focus on the most frightening moments (Brewin, 2016; Ehlers et al., 2004). Studies have found that a substantial proportion of participants omit the moments that are re-experienced from their trauma narratives (Ehring, 2004; Evans, Ehlers, Mezey, & Clark, 2007), in line with the hypothesis that these have not been well integrated into the overall trauma memory. Recent analogue research with trauma films supports the hypothesis that it is disorganization or disjointedness during specific moments, rather than overall poorer memory, that is related to the development of intrusive memories (Sachschaal, Woodward, Wichelmann, Haag, & Ehlers, 2019).

Importantly, like the phenomenon of re-experiencing in the present, impairments in voluntary trauma memory have been found consistently to predict the course of PTSD (Brewin, 2014). One explanation is that higher levels of fragmentation and disorganization in trauma narratives are related to the occurrence of dissociative responses during the trauma (Foa & Rothbaum, 1998). These responses, including depersonalization, derealisation, and emotional numbing, are common during traumatic events and are themselves predictive of the risk of later PTSD (Ozer, Best, Lipsey, & Weiss, 2003). Research has consistently found that higher levels of fragmentation in trauma narratives are related to self-reported dissociation either during or after the traumatic event (Brewin, 2014).

### **Summary**

Re-experiencing in the present, either in the daytime in the form of flashbacks and intrusive memories, or at night in the form of traumatic nightmares, is now one of the core features that distinguish PTSD according to ICD-11. In DSM-5, however, several other kinds of re-experiencing, including emotional or physiological arousal to reminders of the event, count towards a PTSD diagnosis despite their lack of specificity. The evidence for impaired voluntary recall of some aspects of the traumatic event(s) has always been noted clinically.

Research findings suggest that it may be most evident when individuals with PTSD are giving a very detailed account of their trauma that focuses on the worst moments, as occurs during psychological therapy. This observation is likely related to laboratory evidence that enhanced memory for emotional items goes together with decrements in memory for adjacent items, both effects being attributable to amygdala-dependent beta-adrenergic modulation of episodic encoding (Strange, Hurlemann, & Dolan, 2003). The more general point, and a limitation of much of the research in this area, is that there is never a single trauma narrative – depending on how extended the event is in time, there will be a family of narratives that are constructed for different audiences and contain differing amounts of detail.

### **Other Effects of PTSD on Memory**

#### **General Memory Performance**

It is not just memory for the traumatic events that is affected in PTSD. Meta-analyses indicate that PTSD is associated with moderate deficits in performance on tests of verbal memory for neutral materials, and in working memory, with smaller deficits on visual memory tests (Brewin, Kleiner, Vasterling, & Field, 2007; Scott et al., 2015). Similar effects are apparent in trauma-exposed children, with greater deficits in those who additionally meet criteria for PTSD (Malarbi, Abu-Rayya, Muscara, & Stargatt, 2017). These effects appear to be independent of type of traumatic event, and not accounted for by mere exposure to trauma. It has not been established, however, that these deficits amount to actual impairment, as opposed to differences in performance that are nevertheless within the normal range. However, two studies have now demonstrated a functional significance of verbal memory deficits as they appear to prevent patients from benefiting from psychological therapy (Nijdam, de Vries, Gersons, & Olf, 2015; Wild & Gur, 2008).

#### **Overgeneral Memory**

Depression is associated with a deficit in autobiographical memory, such that in response to a variety of pre-determined positive and negative cue words depressed participants struggle to retrieve specific memories as instructed and instead tend to retrieve memories that are overgeneral in that they refer to repeated or long-lasting events. One explanation for this finding is that depressed people may be trying to avoid the activation of specific distressing memories from their past (Williams et al., 2007), an issue that would be equally relevant to people with PTSD. A narrative review of 24 studies concluded that overgenerality in autobiographical recall was associated with the presence of PTSD but not with mere exposure to traumatic events (Moore & Zoellner, 2007). In contrast, more recent meta-analytic studies (Barry, Lenaert, Hermans, Raes, & Griffith, 2018; Ono, Devilly, & Shum, 2016) have reported that trauma exposure alone increased overgenerality and reduced specificity. The two meta-analyses differ in the importance they attribute to the additional presence of PTSD symptoms, one finding this to amplify the effect of exposure and one finding a marginal effect.

There are a number of studies that have found reduced specificity/increased overgenerality of autobiographical memory to predict the risk of later PTSD (Bryant, Sutherland, & Guthrie, 2007; Kleim & Ehlers, 2008), and in addition there is evidence that memory becomes more specific with recovery from PTSD (Sutherland & Bryant, 2007). This indicates that memory specificity may play a functional role in PTSD, and could constitute a pre-existing risk factor.

### **Memory Suppression**

The greater self-reported use of thought suppression has been found to predict more severe PTSD symptoms in a number of longitudinal studies (Ehlers, Mayou, & Bryant, 1998; Joseph et al., 1996; Mayou, Ehlers, & Bryant, 2002). Much of this thought suppression is likely to have involved specific attempts to banish traumatic memories from consciousness.

These attempts are only ever partially successful in people with active PTSD and experiments show that they often backfire in that memories become more intrusive rather than less (Shipherd & Beck, 2005).

In other experimental studies PTSD patients have been taught to associate aversive scenes with naturalistic reminders and then to practise voluntarily suppressing the scenes when cued with the reminders (Catarino, Kuepper, Werner-Seidler, Dalgleish, & Anderson, 2015). This task assesses inhibitory control of memory retrieval, a skill extremely relevant to PTSD patients. The results indicated that retrieval suppression was compromised significantly in PTSD patients and that those with the largest deficits in suppression-induced forgetting were also those with the most severe symptoms. The authors suggested that the difficulties patients have in controlling their intrusive memories arise partly from deficits in engaging inhibitory control to suppress retrieval.

### **Summary**

Along with a general bias toward recalling negative rather than positive stimuli (Brewin, 2011; Durand, Isaac, & Januel, 2019), PTSD affects many aspects of memory apart from the representation of the traumatic event itself. There is evidence that some of these effects, primarily verbal and overgeneral memory deficits and thought suppression, are of functional significance for the maintenance of the disorder or for response to treatment. It is possible that some of them may also constitute a risk factor for the development of PTSD – evidence for a pre-existing decrement in allocentric spatial memory is described in the section on Dual Representation Theory below.

### **Controversies Connected to PTSD**

The diagnosis of PTSD has attracted a number of controversies, some of which appear to reflect beliefs and assumptions about trauma and memory held by those who are less familiar with the clinical presentation of this condition.

### **Are Traumatic Memories ‘Special’?**

In 1997 an influential article claimed that, according to some clinicians, memories of trauma were encoded using ‘special mechanisms’ like repression or dissociation that could lead to amnesia (Shobe & Kihlstrom, 1997). The authors argued that such mechanisms had no laboratory support and that, contrary to what they termed this ‘trauma-memory argument’, the evidence suggested traumatic memories were actually remembered better, not worse. The context in which this article was written was the controversy over recovered memories of child sexual abuse, but the ‘trauma-memory argument’ has come to be applied to any kind of traumatic memory, as illustrated by this account of what it consists of (Hembrooke & Ceci, 1995): “(i) Events that are traumatic, of which childhood sexual abuse is the choice du jour, are claimed to often be repressed; (ii) repressed memories are somehow "special", in that they are allowed to enter some special status in memory which renders them impervious to the normal forces which act upon other, nontraumatic memories, such as decay and interference; (iii) repressed memories are often expressed later in life through various and sundry behavioral manifestations that may, or may not, bear a resemblance to the original experience...; (iv) repressed memories can be exhumed using a variety of methods which include, but are not limited to, such techniques as visually guided imagery, hypnosis, dream analysis, journalling, and the interpretation of "body memories" (p. 77).

As these two articles demonstrate, in the 1990s some authors feared that if traumatic memories were acknowledged to be different in any way from normal memory, or prone to being more disorganized or fragmented, this would open the door to unwanted consequences such as the wholesale acceptance of any memory recovered in therapy as veridical. Despite the general acceptance that memories recovered in therapy from amnesia must be treated with great caution, and any attempt to exhume supposedly repressed memories being firmly rejected by all professional psychotherapy bodies, a minority of authors today still pursue the

idea that traumatic memories differ quantitatively but not qualitatively in terms of patterns in intentional and unintentional retrieval from other memories (Rubin et al., 2016; Rubin, Dennis, & Beckham, 2011).

An alternative possibility is that qualitative differences can be produced by normal mechanisms that behave differently under extreme stress. There is a great deal of evidence that high levels of stress have opposite effects on different brain structures important for memory, the amygdala and the hippocampus (Diamond, Campbell, Park, Halonen, & Zoladz, 2007; Nadel & Jacobs, 1998). As Nadel and Jacobs noted in response to Shobe and Kihlstrom (1997): “When stress is high enough to impair the function of the hippocampus, resulting memories will be different from those formed under more ordinary circumstances. These empirical data suggest that memories of trauma may be available as isolated fragments rather than as coherently bound episodes” (p. 156).

The original rejection of ‘special mechanisms’, motivated by a real concern over inappropriate therapeutic practice which had the potential to be very damaging, has led to widespread misunderstanding. Clinicians did not, as claimed, propose that memories of traumatic memories were all processed differently from normal events. They were concerned with observations of certain patients who often reported fluctuating memory for the existence or details of traumatic episodes, usually occurring in childhood. Their writings were an attempt to make sense of the experiences of this specific group, not to make broad claims about trauma in general. Repression and dissociation represented clinical hypotheses about the processes underlying the observed phenomenon, which was the forgetting of traumatic incidents. Whether or not the hypotheses were correctly formulated, and whether the mechanisms are ‘special’ or related to everyday memory processing, does not impact on the observations of the forgetting of trauma that originally motivated them.

Evidence of further misunderstanding has occurred recently with claims that any theory that suggests the memories reported by PTSD patients are somehow different from normal memories must be invoking such ‘special mechanisms’ (Malaktaris & Lynn, 2019). As outlined below in more detail, the leading clinical theories attempt instead to relate the different properties of PTSD memories to normal mechanisms that behave differently under extreme stress. The ‘special mechanisms’ argument has not been consistent or contained substantive content, and has acted as an unfortunate distraction to the field.

### **Can Traumatic Events be Forgotten?**

Critics of hypothetical mechanisms such as repression and dissociation often claim that traumatic events are invariably better recalled than non-traumatic events, citing research showing enhanced recall of stimuli that are emotional as opposed to neutral (McNally, 2003; Rubin et al., 2011; Shobe & Kihlstrom, 1997). However, other large bodies of research have found that negative events are forgotten more readily than positive events (Walker, Skowronski, & Thompson, 2003) and that, as predicted by Nadel and Jacobs (1998), high levels of stress in the person doing the remembering is associated with worse, not better recall (Deffenbacher, Bornstein, Penrod, & McGorty, 2004; Metcalfe, Brezler, McNamara, Maletta, & Vuorre, 2019). Consistent with this, several studies have reported significant levels of forgetting of documented traumatic events in adulthood (Means & Loftus, 1991; Raphael, Cloitre, & Dohrenwend, 1991; Schraedley, Turner, & Gotlib, 2002), and a substantial proportion of those reporting childhood sexual abuse have also described periods when they say they had completely forgotten the abuse had occurred (DePrince et al., 2012).

Although it may appear intuitively plausible to believe that traumatic events cannot be forgotten, there are at least two important considerations that are often ignored in the literature. The first is that for an event to be remembered it must be assimilated to people’s autobiographical knowledge about themselves. For someone with PTSD, the traumatic events

*are* often regarded as a turning point and as an important aspect of identity. But for someone without PTSD an event conventionally classified as traumatic may not have the same long-term implications and quickly cease coming to mind. Discussions concerning people's knowledge of their own history also frequently assume that there is a single (remembering) self. But the idea of a unitary self has been rejected in favor of the idea that we have multiple selves that are elicited in different contexts. Children exposed to trauma often have great difficulty in developing a coherent sense of themselves, experiencing internal conflict between multiple selves and to disruptions to the continuity of the self over time (Harter, 1998). Consistent with this, they often demonstrate long-term impairments to semantic memory for their childhood (Brewin, 2012). At least in part, this may arise because of changes in family members, parent figures, and accommodation that create an environment in which important retrieval cues are no longer present.

A second factor is that there is now substantial evidence for inhibitory processes in memory whereby people are able to deliberately suppress unwanted thoughts and memories (Anderson & Hanslmayr, 2014). Survivors of childhood sexual abuse who report having managed to forget it for a period frequently attribute this to active attempts to banish the memories from their minds (Ghetti et al., 2006).

### **Does Delayed-Onset PTSD Exist?**

Ever since clinical observations of delayed-onset (i.e., PTSD that develops at least six months after the traumatic event) were recognized in 1980 there has been skepticism expressed about the condition. Commentators have wondered whether people could accurately report their symptoms retrospectively, and whether what was occurring was delayed presentation rather than delayed onset (Gray, Bolton, & Litz, 2004; Hunt, Wessely, Jones, Rona, & Greenberg, 2014). Subsequent reviews have shown that delayed-onset cases make up approximately one quarter of PTSD onsets (Andrews, Brewin, Philpott, & Stewart,



2007; Galatzer-Levy, Huang, & Bonanno, 2018; Smid, Mooren, van der Mast, Gersons, & Kleber, 2009)).

These findings challenge the common assumption that people with PTSD invariably develop their symptoms immediately following an overwhelming event. In most cases of delayed onset there are some initial symptoms, but these do not reach the threshold required for a diagnosis of PTSD until after six months post-trauma. Rarely, however, reports indicate no or very minimal symptoms post-trauma, with a sudden onset of acute PTSD that in some cases occurs unexpectedly decades after the traumatic exposure (Andrews et al., 2007).

PTSD theories have yet to satisfactorily explain these different trajectories, partly due to the paucity of relevant data. One study of war veterans with delayed onset PTSD found that emotional and dissociative reactions during the traumatic event were indistinguishable from those reported by soldiers who never developed PTSD, and significantly less than in those who had an immediate onset (Andrews, Brewin, Stewart, Philpott, & Hejdenberg, 2009). Predictors of delayed-onset PTSD after discharge from the military include a poor disciplinary record (Brewin, Andrews, Hejdenberg, & Stewart, 2012), and in motor vehicle accident survivors include factors such as rumination, injury severity, and prior emotional problems (Ehlers et al., 1998).

## **Summary**

Concerns that first came to light in the 1990s about the existence of false memories of abuse, along with assumptions about trauma, have shaped many of the debates about trauma and memory. Such concerns have sometimes inhibited a dispassionate consideration of the evidence on both sides. This indicates that although traumatic memories are by definition prominent in PTSD, this is not necessarily the case for trauma survivors without PTSD, who may sometimes forget entirely that the events have occurred. Contrary to what

has sometimes been implied, accepting the reality of dissociative amnesia (Harrison et al., 2017; Staniloiu & Markowitsch, 2014) or of inhibitory processes in memory (Anderson & Hanslmayr, 2014) does not carry with it any implication that memories recovered in therapy should be accepted without question.

Repeated clinical observations have led to flashbacks, memory impairment, and delayed onsets being included among the symptoms of PTSD in the DSM, and there is no doubt that some patients exhibit unusual memory phenomena that are not generally observed in other contexts such as the laboratory (such as intrusive memories that lack awareness of the self in the past, flashbacks where all contact with current reality is lost, and physiological reactions or behaviors not recognized as originating with the trauma).

The study of PTSD therefore sets a number of challenges to conventional theories of memory. Whereas clinical models of PTSD were developed with the aim of explaining the full range of intrusive re-experiencing symptoms, such experiences are not addressed by models that claim “basic mechanisms” studied by cognitive psychologists are sufficient to explain memory in PTSD (Berntsen, 2009; Rubin, Boals, & Berntsen, 2008). The wholesale rejection of clinical observations, or the assertion that clinical theories should be discarded because they are proposing ‘special mechanisms’, have not been helpful in this context. It is likely to be more fruitful to explore how everyday mechanisms, such as the trade-off between item and context memory, may operate when the brain is responding to extreme stress.

### **Contemporary Theories of PTSD**

#### **Fear Conditioning**

Conditioning theories have been very influential in other anxiety disorders and have been applied to PTSD despite having little to say about enhanced re-experiencing or impaired voluntary recall. The basic idea behind all these applications is that an initial phase of fear acquisition through classical conditioning results in neutral stimuli present in the traumatic

situation acquiring fear-eliciting properties. Through a process of stimulus generalization, similar stimuli encountered in other contexts act as reminders and lead to the return of the fear memory. Under normal conditions these responses extinguish as the person learns that in other contexts they are safe and the reminders are not followed by a repeat of the original traumatic event. In PTSD, however, repeated exposure to the trauma memories triggered in everyday life fails to result in these associations being extinguished, largely because the person attempts to distract themselves or block out the memories, rendering the new contextual learning incomplete. These avoidance behaviors are reinforced by a reduction in fear, leading to the maintenance of PTSD.

Fear conditioning procedures have lent themselves to animal and neurobiological research, resulting in a neurocircuitry model of PTSD implicating three key brain areas involved in fear processing (Rauch, Shin, & Phelps, 2006). The model proposes that a hyper-responsive amygdala accounts for the exaggerated fear responses and the persistence of traumatic memories. Hypo-responsive ventral areas in the medial prefrontal cortex fail to inhibit the amygdala, preventing efficient extinction of the fear response and a withdrawal of attention from reminders of trauma. Finally, abnormal hippocampal function accounts for the memory impairments and difficulty in identifying safe contexts that characterizes PTSD. Importantly, however, the patterns of hyper-responsivity and hypo-responsivity that have been identified occur in other tasks and do not appear to be specific to fear conditioning paradigms (VanElzaker, Dahlgren, Davis, Dubois, & Shin, 2014).

Conditioning theories have addressed the origin of these functional abnormalities, attempting to explain why only a minority of individuals develop PTSD following trauma exposure. One observation is that reduced hippocampal volume is present in the unexposed co-twins of military personnel who developed PTSD, suggesting that it could be an inherited

risk factor (Gilbertson et al., 2002). In contrast, the hypo-responsivity of ventral prefrontal areas to trauma stimuli appears to be an acquired characteristic (Dahlgren et al., 2018).

Another proposal is that those vulnerable to PTSD have a constitutionally increased facility to acquire such conditioned associations or a difficulty in subsequently extinguishing them. There is some evidence supporting this: During fear acquisition people with PTSD often have stronger physiological responses to aversive rather than neutral cues, as well as stronger conditioned responses during extinction and recall of extinction (VanElzakker et al., 2014). There is also evidence for greater second-order conditioning in PTSD compared to traumatized controls, which may lead to further generalization of fear responses (Wessa & Flor, 2007). A longitudinal study of firefighters (Guthrie & Bryant, 2006) found that impaired extinction learning while they were training was associated with reporting more PTSD symptoms after they had been deployed and exposed to traumatic events. More specifically, it has been proposed that in PTSD there is a particular difficulty in learning to differentiate safe from threatening contexts (Jovanovic, Kazama, Bachevalier, & Davis, 2012; Rougemont-Buecking et al., 2011). The enhanced generalization of conditioned fear responses observed in PTSD has been found to be associated with greater generalization of activation in the right anterior insula and the left ventral hippocampus (Kaczurkin et al., 2017).

It is problematic, however, that with a few exceptions there is relatively little evidence to show that vulnerability factors for disorders such as PTSD are associated with greater sensitivity or proneness to fear conditioning (Beckers, Krypotos, Boddez, Effting, & Kindt, 2013). Basic fear conditioning provides a powerful explanation of many important aspects of PTSD, such as the wide range of potential trauma reminders, physiological and emotional arousal elicited by these reminders, and the central role of avoidance. However, as a generic theory designed to account for the acquisition of fear it does not clearly distinguish the etiology of PTSD from that of other anxiety disorders. The theory also has obvious

limitations in that it was not designed to account for specific features of PTSD such as the way the event is consciously remembered and the existence of delayed onsets. Nor can it account for the prominence in PTSD, not just of the central reaction of horror, but of other emotions such as anger, guilt, and shame, which have assumed a more important place within the DSM-5 conceptualization of PTSD.

### **Neo-Conditioning Theories**

A number of these limitations have been addressed in elaborations of the basic concept of fear conditioning. For example, in explaining why only some people develop PTSD post-trauma it has been pointed out that the outcome of conditioning depends crucially on prior experience with similar situations. Experience of the successful exercise of control in an aversive situation reduces subsequent vulnerability whereas experiencing the loss of control increases vulnerability to acquiring a fear response (Mineka & Zinbarg, 2006).

Mineka and Zinbarg pointed out that many features of PTSD, such as feelings of helplessness and emotional numbing, are similar to effects such as passivity and analgesia found in animals exposed to uncontrollable and unpredictable shock. Consistent with this, passive responding in the guise of 'mental defeat', the complete giving up of control and resignation to injury or death, is an important risk factor for PTSD (Ehlers, Maercker, & Boos, 2000). Such reactions are more likely to be found following repeated and chronic trauma, or trauma that was overwhelming because it occurred during childhood. This does not however rule out the possibility that other types of exposure increase readiness and resilience.

Other explanations have been put forward for the gradual development of PTSD over time. For example, the occurrence of subsequent unrelated aversive events can strengthen (Mineka & Zinbarg, 2006) or reinstate (Hermans et al., 2005) a conditioned fear response. Unrelated life stressors have been observed to predict vulnerability to both the immediate and delayed onset forms of PTSD (Andrews et al., 2009; Brewin, Andrews, & Valentine, 2000).

Another explanation involves sensitization (Lanius, Frewen, Vermetten, & Yehuda, 2010), a non-associative process whereby repeated exposure to trauma reminders, or to intrusive memories, leads to a gradual increase in arousal. The addition of reinstatement and sensitization to the fear conditioning model may provide an explanation of the hyperarousal symptoms that are a central part of PTSD and in several studies predict the development of the disorder (Brewin, 2011). Delayed-onset PTSD is often prefigured by such a gradual build-up of symptoms (Andrews et al., 2009). There may be an interaction between associative and non-associative processes, with sensitization being exacerbated by the difficulty in extinguishing conditioned responses noted earlier.

Mental rehearsal of an aversive event may result in more persistent conditioned fear, and interact with anxiety to increase physiological arousal to reminders (Davey & Matchett, 1994). This is consistent with evidence that rumination is a risk factor for PTSD and predicts the delayed onset for of the disorder (Ehlers et al., 1998; Michael, Halligan, Clark, & Ehlers, 2007). Finally, reevaluation of an aversive event can occur when it is reappraised in the light of new information (Davey, 1989). Thus, an assault victim may become much more symptomatic or experience a delayed onset of PTSD after they learn that the person who attacked them has gone on to commit murder.

Another cognitive reformulation of fear conditioning proposed that frightening events are represented within memory as interconnections between nodes in an associative network (Lang, 1979). A specific fear memory would consist of stimulus information, for example about the sights and sounds of the aversive event, response information about the person's emotional and physiological reactions, and meaning information, primarily about the degree of threat. When fear memories are elicited by reminders, the person experiences similar reactions to those that were present during the event. These ideas have been applied to PTSD, where it has been suggested that traumatic events lead to fear memories in which the

elements are exceptionally strongly associated with one another (Foa, Steketee, & Rothbaum, 1989). Exposure to trauma reminders leads to PTSD symptoms such as hypervigilance and intrusive sensory memories. Foa et al. argued that the overturning of basic assumptions about safety results in a fear memory that is very easily activated by a wide range of environmental cues, and is poorly integrated with other memories.

These ideas were further elaborated to include a role for pre-trauma and post-trauma beliefs (Foa & Rothbaum, 1998). Strongly-held pre-existing beliefs were considered to be a vulnerability factor for PTSD, not just concerning the lack of personal safety but also lack of competence. For example, individuals already holding rigid negative views about the self as being extremely incompetent would have these confirmed by negative appraisals post-trauma of responses and behaviors. These appraisals could involve events that took place at the time of the trauma, symptoms that developed afterwards, disruption in daily activities, and the responses of others.

Theories that have attempted to explain the clinical phenomena associated with PTSD have introduced a variety of new ideas to overcome the limitations of a basic fear conditioning approach. These include non-associative processes such as sensitization and a variety of higher-order cognitive processes such as expectancies and appraisals. This raises the question of the kind of cognitive architecture that is necessary to accommodate them, as neither conditioned associations nor associative networks are designed to explain both fast, automatic responding and more deliberate, higher-order appraisals (Dagleish, 2004).

### **The Ehlers and Clark Model**

Ehlers and Clark (2000) drew attention to the sense of continuing threat that PTSD patients experience, despite knowing that the traumatic event lies in the past. They proposed that this sense of current threat can be explained by two mechanisms, negative appraisals of the trauma or its sequelae and the nature of the trauma memory itself. Behavioral and

cognitive strategies that individuals use to control the sense of current threat such as thought suppression, rumination, and safety behaviors are thought to maintain the problem.

A very considerable body of research supports the ubiquity of negative appraisals of the self, of other people, and of the future in PTSD, and shows that these appraisals predict the course of the disorder, often over and above initial symptoms (Ehring, Ehlers, & Glucksman, 2008; Halligan, Michael, Clark, & Ehlers, 2003; Kleim et al., 2007). Further, they drive symptom change in cognitive behavioral treatments of PTSD (L. A. Brown, Belli, Asnaani, & Foa, 2019; Kleim, Grey, et al., 2013).

The second source of current threat in PTSD according to the model is that the memory of the traumatic event is poorly elaborated, not given a complete context in time and place, and inadequately integrated with other autobiographical knowledge and memories. Especially relevant for the explanation of re-experiencing symptoms is the hypothesis that the worst moments of the trauma are disjointed from preceding and subsequent information in memory that is important for the meaning of the event (Ehlers et al., 2004). These moments (e.g., when they thought they would die or felt ashamed for complying with the perpetrator's requests) need to be updated (e.g., with the information that they did not die or that they complied with the perpetrator because he threatened to kill them). Until the update is integrated with the memory of this particular moment, the original threatening meaning will be retrieved and the moment will appear to happen in the here and now.

In line with this hypothesis, some patients with PTSD report having intrusions of different moments from the trauma that have contradictory meanings (Ehlers & Clark, 2000) and people with PTSD take longer than traumatized controls to access autobiographical information when visualizing the worst moment of their trauma compared to the worst moments of other negative events (Kleim, Wallott, & Ehlers, 2008). The worst moments of the trauma are more disorganized than other parts of the trauma narrative (Evans et al., 2007).



Memory elaboration after trauma exposure reduced re-experiencing in analogue studies (Ehlers, Mauchnik, & Handley, 2012; Michael & Ehlers, 2007).

The resulting absence of clearly specified retrieval routes accounts for the difficulties in intentional recall (disorganized recall) and poor inhibition of cue-driven retrieval of unwanted memories, the absence of a context accounts for re-experiencing in the present, and the lack of connection with other relevant information accounts for the unchanged threatening meaning of the moments that are re-experienced. The easy triggering by a wide range of cues is explained by strong associations involving sensory elements from the traumatic situation, consistent with conditioning accounts, and enhanced perceptual priming, defined as a reduced perceptual threshold for trauma-related sensory stimuli.

Ehlers and Clark's model also proposes that the nature of the person's response during the trauma affects encoding and the nature of the trauma memory. Drawing on a distinction made by cognitive psychologists (Roediger & McDermott, 1993) Ehlers and Clark predicted that data-driven processing (an increased focus on sensory impressions during the traumatic event) would enhance perceptual priming whereas conceptual processing (an increased focus on the meaning of the situation and placing it in context) would facilitate integration of the trauma memory with the autobiographical database. Other responses during the trauma which would increase the risk of later PTSD were an inability to establish a self-referential perspective while experiencing the trauma, dissociation, mental defeat, emotional numbing, and lack of cognitive capacity to evaluate aspects of the event accurately.

Subsequent research by Ehlers and Clark and their colleagues has confirmed that trauma memories are frequently disorganized and disjointed, and that this disorganization is related to processes that occur during the traumatic event such as higher levels of dissociation and data-driven processing, and less self-referent processing. Both these processes, and the disorganization of trauma memories, predict concurrent and subsequent PTSD symptoms in

line with the theory (Beierl, Böllinghaus, Clark, Glucksman, & Ehlers, 2019; Ehlers, Mayou, & Bryant, 2003; Evans et al., 2007; Halligan et al., 2003; Murray, Ehlers, & Mayou, 2002), and similar results have been found with children and adolescents (Meiser-Stedman, Dalgleish, Smith, Yule, & Glucksman, 2007; Meiser-Stedman et al., 2019). In contrast measures of conceptual processing have not reliably predicted PTSD.

As predicted by Ehlers and Clark's cognitive theory, PTSD patients have demonstrated greater perceptual priming for trauma-related words on a word-stem completion task than have healthy controls (Michael, Ehlers, & Halligan, 2005). The degree of priming at two weeks post-trauma has also been found to predict the course of PTSD (Michael, Ehlers, & Halligan, 2005), including when levels of initial symptoms and priming for other words were controlled (Ehring & Ehlers, 2011). The role of perceptual priming has been explored further in analogue studies where it has been possible to investigate priming for neutral stimuli that are embedded in experimental materials. This has confirmed that priming is enhanced when stimuli are embedded in traumatic versus neutral materials, and that the degree of priming is related to the subsequent development of intrusive memories of the trauma scenes (Michael & Ehlers, 2007; Sündermann, Hauschildt, & Ehlers, 2013). Similarly, trauma survivors with PTSD were more likely to identify degraded trauma-related pictures than those without PTSD, but not general threat or neutral pictures (Kleim, Ehring, & Ehlers, 2012). In contrast, no role has been established for conceptual priming in PTSD.

### **Dual Representation Theory (DRT)**

The first version of DRT (Brewin, Dalgleish, & Joseph, 1996) was based on clinical observations that intrusive trauma memories in PTSD were extremely high in perceptual details, often appeared to occur automatically and were hard to control, and were experienced in the present. At that time most cognitive psychologists recognized a form of automatic perceptual memory but it was thought that its products were unavailable to conscious

experience (Tulving & Schacter, 1990). In order to accommodate the very conscious experience of flashbacks, Brewin et al. drew on alternative theoretical suggestions made by clinicians, autobiographical memory researchers, and neuroscientists with a particular interest in stress and trauma. These authors (R. Brown & Kulik, 1977; Jacobs & Nadel, 1985; Janet, 1904; Pillemer, Desrochers, & Ebanks, 1998) had proposed that a separate, partly image-based memory system operated in parallel with ordinary episodic memory. The image-based system was present prior to the maturation of the hippocampus, and provided a simpler associative mechanism that was able to learn but unable to contextualize how and when the information was acquired.

Applying these ideas to PTSD, Brewin et al. postulated a 'situationally accessible memory system' which during a traumatic event recorded detailed perceptual images of events that were perceived too fleetingly to allow full conscious encoding. The images in this more primitive system could not be directly retrieved but were automatically re-experienced in a stereotyped way when triggered by reminders (i.e., they were situationally accessible). They were experienced as occurring in the here-and-now because the system was unable to encode aspects of the temporal context. In contrast a 'verbally accessible memory system' recorded aspects of the event that had been consciously attended to, located these in a full temporal and spatial context, and provided accessible memories that could be used flexibly to reflect on, appraise, and communicate about the event.

Brewin et al. argued that these two systems provided the minimum cognitive architecture necessary to account for the parallel existence of repetitive and largely uncontrollable re-experiencing coupled with verbal memories, often including amnesic gaps, used to generate narrative accounts of the traumatic event. The value of such a system was that a large amount of potentially important trauma-related information could be recorded at a time of crisis when attention was narrowed onto the immediate threat and little higher-order

reflection was possible. This information was later re-experienced in the form of flashbacks. For most people, paying deliberate attention to the flashbacks and their content would result in the recoding of the information within the verbally accessible memory system. The threat would be contextualized as belonging to the past, levels of arousal would gradually decrease, and flashbacks would decrease in frequency.

In addition to normal recovery, two other trajectories were described. Rather than paying attention to flashbacks individuals could choose to suppress them, distract themselves, and reduce their exposure to trauma reminders. Information would therefore remain unaltered in the situationally accessible memory system. If successful, this strategy would result in 'prematurely-inhibited processing' - individuals would not be bothered by intrusions but would remain vulnerable to a delayed onset of PTSD if reminders became too powerful to be ignored. If unsuccessful, symptoms of PTSD such as ongoing flashbacks and sustained high levels of arousal would result.

Initial investigations of DRT confirmed that people with PTSD (but not those without PTSD) write trauma narratives in which they can readily identify passages that they experience as re-occurring in the present (flashbacks) and passages that are experienced as belonging to the past. The distribution of these passages is highly variable and idiosyncratic, emphasizing that trauma memories are complex and multi-faceted. Flashback passages, compared to episodic memory passages, have been found to be characterized by a wide range of autonomic and motor responses, including heart rate increases (Chou, La Marca, Steptoe, & Brewin, 2018), as well as by more mentions of death, more perceptual detail, and more use of the present tense (Hellowell & Brewin, 2002, 2004). Flashbacks are also accompanied by increased activation in sensory and motor areas including the insula, precentral gyrus, supplementary motor area, and mid-occipital cortex, and by decreased activation in the

midbrain, parahippocampal gyrus, and precuneus/posterior cingulate cortex (Whalley et al., 2013).

Dual representation theory has since been updated in light of neurobiological evidence for the likely substrate of the two memory systems. In the case of visual material, more recent evidence has confirmed that briefly fixated information appears to be encoded automatically and to form a relatively stable representation in long-term memory that then supports both priming and conscious forms of memory such as recognition (Brewin, 2014). There are striking parallels between the description of the hypothetical memory systems in DRT and the operation of the dorsal and ventral visual streams (Brewin, 2014; Brewin, Gregory, et al., 2010). Briefly, the dorsal stream is able to represent visual information from an egocentric perspective and provides a rapid pathway for its transmission of visual information to the motor cortex. This low-level system (termed 'sensation-based memory' within the revised DRT) operates quickly and automatically with relatively unprocessed information that is close to the sensory input. In contrast the 'contextualized memory' system operates on information that is much more highly processed within the ventral visual stream and medial temporal lobe, leading to the creation of more flexible allocentric representations and an accompanying spatial and temporal context. According to the revised DRT, extreme stress results in these normally integrated systems becoming functionally disconnected. Thus at some moments of great fear or horror, for example when the person is faced with death, sensation-based memories are created that have no linked contextualized memories, leading to specific moments being re-experienced as flashbacks.

As suggested by the theory, people with PTSD appear to have a more general difficulty in forming allocentric spatial memories (Smith, Burgess, Brewin, & King, 2015). The greater this difficulty, the more intensely they experience intrusive memories when these are provoked by reminders of their traumatic event (Sierk et al., 2019). There is some

evidence that this difficulty may be a pre-existing risk factor for the development of PTSD. Using a configural learning task to test allocentric memory in twins discordant for combat trauma and PTSD, Gilbertson et al. (2007) found allocentric memory reductions in both trauma-exposed PTSD patients and their unexposed twin. These memory decrements were associated with smaller hippocampal volume, raising the possibility that they were present before the traumatic episode.

Subsequent research has identified a general pattern whereby, even in healthy people, negative material induces an up-regulation of the amygdala, leading to improved item memory, accompanied by a down-regulation of the hippocampus such that items are bound less to their context (Bisby & Burgess, 2017; Bisby, Horner, Bush, & Burgess, 2018). Further, the better they encode contextual information the less likely people are to experience subsequent intrusive memories of a traumatic film (Meyer, Krans, van Ast, & Smeets, 2017). According to the revised DRT this is the same process that is illustrated, in a much more extreme form, in people with PTSD and, as proposed by Nadel and Jacobs (1998), explains why traumatic memories are at least in part fragmented and incoherent. The lack of context also accounts for the fact that traumatic memories are easily triggered by reminders leading to frequent intrusions.

## **Summary**

The basic fear conditioning model rooted in a behavioristic framework has been very popular because of its familiarity, its simplicity, and the fact that it can be readily tested in animal experiments or neuroimaging studies. Despite the elegant way in which it accounts for many aspects of fear acquisition and extinction, applying it to the specific symptoms of PTSD has required many additions, particularly to account for the subjective experience of PTSD. Neo-conditioning approaches have updated the explanation of fear acquisition to incorporate the role of past experience, non-associative processes such as sensitization, and

cognitive elements such as expectancies and evaluations. At this point it becomes relevant to ask what kind of cognitive architecture is required to accommodate both associative processes such as priming and automatic, cue-driven retrieval, and higher-order processes such as appraisals leading to complex value judgements and emotions such as shame and guilt. Both the cognitive model of Ehlers and Clark and dual representation theory adopt a dual-process account which explicitly recognizes the parallel existence of very different types of trauma memory.

### **Treatment and Memory Mechanisms**

#### **What Changes are Seen in Trauma Memories?**

The most obvious changes with successful treatment, as reflected in the symptoms of PTSD, involve a reduction in intrusive memories, flashbacks, and memory avoidance. More specifically, intrusive memories of the traumatic event become less vivid, less distressing, and have less of a sense of being relived in the present (Hackmann et al. 2004, Speckens et al. 2006). In contrast, the evidence that memories become more organized or less fragmented is much more equivocal (Bedard-Gilligan, Zoellner, & Feeny, 2017; Foa, Molnar, & Cashman, 1995; van Minnen, Wessel, Dijkstra, & Roelofs, 2002), consistent with naturalistic follow-up studies that did not find any corresponding improvement in these aspects of the trauma memory (Halligan et al., 2003; Jones, Harvey, & Brewin, 2007). One possible implication is that, although disorganization reflects the disruptive influence of “worst moments”, recovery is more strongly related to changes in the way these moments are processed than to general changes in the trauma narrative. Emerging evidence suggests that with recovery there are decreases in neural activity in the amygdala and insula, coupled with increased activity in the dorsal anterior cingulate cortex, pre-frontal cortex, and hippocampus (Malejko, Abler, Plener, & Straub, 2017). Many upregulated genes associated with intrusion symptoms were downregulated following recovery (Rusch et al., 2019).

## **Habituation and Extinction**

To date the most effective psychological treatments for PTSD, achieving on average a large effect size relative to waitlist control or usual care (Bisson, Roberts, Andrew, Cooper, & Lewis, 2013), include engagement with, and activation of, the trauma memory (Olf et al., 2020). Exposure therapies for PTSD draw on classic behavioral therapy applied to a range of anxiety disorders, particularly phobias. Exposure therapy for phobias involves confronting the person with their feared situation whereas in PTSD confrontation is primarily with the traumatic memory, in addition to trauma reminders. Patients are asked to recall and describe all aspects of the event in great detail, revisiting their emotional responses at the time to the degree that they can without becoming overwhelmed. Afterwards, the exposure, new details of the memory that have come to mind, and the perspective they provide are discussed. A focus on addressing the most intense or painful moments is likely to be particularly helpful (Nijdam, Baas, Olf, & Gersons, 2013). The first programmes (Foa, Hembree, & Rothbaum, 2007) consisted mainly of extended exposure sessions both in the clinic and at home that were repeated until the levels of negative emotion associated with the traumatic memory declined and it intruded much less frequently.

From the perspective of traditional memory theory it may appear puzzling that procedures which appear likely to strengthen the memory by repeatedly rehearsing it are actually therapeutic. According to habituation-based models (Foa & Kozak, 1986), a crucial element is to remain in the feared situation or hold the memory in mind until arousal naturally declines. This information is then incorporated in the original memory. Similarly, reductions in arousal that occur between therapy sessions can result in positive changes in the fear memory. Although the theory has been extremely influential, many of its predictions have not been borne out in practice (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014; Rupp, Doebler, Ehring, & Vossbeck-Elsebusch, 2017).



In any case the idea that memories, once created, can be altered is controversial. The alternative is that therapeutic experiences create new memories that then compete with the original traumatic ones for control of behavior (Brewin, 2006). This is the current understanding of the processes underlying extinction, a process thought to be influential in accounting for the effectiveness of exposure therapy. Extinction procedures expose the person to trauma reminders in alternative contexts, such as a therapy room, where they can be experienced with a reduced level of fear. This is held to produce alternative memories that can inhibit the original memories of the fear being acquired. Critically, the original memories are unaltered, and may be elicited in other contexts or when the person is exposed to other stressful events (Bouton, 2004).

Among other evidence-based treatments for PTSD, cognitive therapy (Ehlers & Clark, 2000) and the original version of cognitive processing therapy (Resick & Schnicke, 1993) also ask patients to recall the trauma memory in detail in imagination or by writing a detailed narrative, but focus on changing the negative cognitive appraisals connected with the memories that are a major feature of PTSD. Cognitive processing therapy conducted without narrative writing, Eye movement desensitization and reprocessing (EMDR) and imagery rescripting (Arntz, Sofi, & van Breukelen, 2013; Bisson et al., 2007; Raabe, Ehring, Marquenie, Olf, & Kindt, 2015) involve relatively brief exposures with an emphasis on incorporating new elements into images of the worst moments of the event or imagining those moments from a different perspective.

Overall, the success of these interventions suggests that a critical mechanism is the contextualization of the traumatic memory by having the person deliberately focus attention upon it in a safe environment or introduce safe or empowering elements or new meanings into the image or narrative. This may include methods that are known to increase hippocampal activity such as imagining the scene from an alternative perspective (Brewin,

Gregory, et al., 2010; Kaur, Murphy, & Smith, 2016). The recontextualized memory is then able to act as an inhibitory version of the original traumatic memory.

### **Updating/Reconsolidation**

Experiments have demonstrated that if animals were first taught an association and then, following a reminder of the prior learning administered a protein synthesis blocker, the memory was no longer expressed and in some cases appeared to have been abolished (Nader, Schafe, & Le Doux, 2000; see also chapter on reconsolidation in this volume). A popular interpretation of these findings has been that each time memories are brought to mind they need to be reconsolidated, and that this provides an opportunity for the memory to be permanently altered by the incorporation of new information. Parallel human experiments have typically paired an aversive event such as shock with a neutral stimulus to create a conditioned emotional response, and then manipulated whether or not the participant received a reminder of the stimulus prior to an intervention such as an anxiolytic drug or an extinction procedure. Such procedures have sometimes successfully decreased fear expression, as indexed by a startle response, while leaving the declarative memory of the learning event intact (Kindt, Soeter, & Vervliet, 2009; Soeter & Kindt, 2010), but other results have been negative (Klucken et al., 2016; Schroyens, Beckers, & Kindt, 2017).

It has emerged from this research that the use of reminders is a critical factor needed to establish empirically that reconsolidation is the most likely mechanism. Reminders do not routinely prompt reconsolidation – they must not only lead to retrieval but must successfully destabilize the memory and then update it with incompatible information (Lee, Nader, & Schiller, 2017). This generally requires that reminders signal an event that is in some way surprising or unpredicted. This appears to be more difficult with stronger and more remote memories, or with more anxious participants (Kindt, 2018), both factors that might limit the applicability of these procedures to patients with PTSD.

Despite considerable uncertainty over whether reconsolidation has been conclusively demonstrated in humans (Brunet et al., 2011; Elsey, Van Ast, & Kindt, 2018; Schiller & Phelps, 2011), interventions based on the theory have been attempted for several different clinical populations. Overall, when applied to phobia or trauma pharmacological interventions have shown a medium-sized effect and behavioral interventions a relatively small effect (Walsh, Das, Saladin, & Kamboj, 2018).

The best researched intervention involves administering propranolol, a beta-adrenergic receptor antagonist commonly used in the treatment of hypertension, to PTSD patients before they write about and recall their trauma in detail. The effect is that the trauma is remembered with reduced emotion and physiological arousal. In a recent clinical trial six weekly sessions employing propranolol brought about a substantial improvement in symptoms relative to a group given a placebo (Brunet et al., 2018). Another recent approach involves having patients monitor their trauma memories and then each week write an account of a particularly distressing memory followed by 25 minutes playing Tetris (Kessler et al., 2018). Tetris is a computer game requiring a high visuospatial memory load that has been found to reduce intrusive memories of a trauma film in analogue studies (James et al., 2016). Kessler et al. found preliminary evidence that this procedure produce a substantial reduction in targeted memories compared to non-targeted memories in their group of inpatients with PTSD.

If the aim of presenting patients with trauma reminders is to produce memory updating and reconsolidation, this should be brief and occur only once (Kindt, 2018). In Brunet et al.'s (2018) study, however, there was extended exposure to the traumatic memories on multiple occasions, raising the possibility that the procedures were effective because they led to enhanced extinction rather than to memory reconsolidation (Giustino, Fitzgerald, & Maren, 2016). Kessler et al.'s (2018) procedures did use briefer exposures that

targeted specific episodes within the overall traumatic memory, but it is unclear how these exposures would have generated prediction errors. Demonstrating reconsolidation is particularly problematic in PTSD patients because patients will often already be experiencing intrusive memories, making it impractical to manipulate the 'reactivation' of the trauma.

### **Meta-Cognition**

Although it has received relatively little attention relative to habituation, extinction, or reconsolidation, it is important to consider changes in people's understanding of their memory symptoms. As previously discussed in connection with Ehlers and Clark's (2000) model of PTSD, negative appraisals of intrusive memories are an important predictor of the course of the disorder. It is common for avoidance to be fuelled by the prediction that the person will be unable to tolerate recalling the most upsetting details of their trauma, or will be overwhelmed with shame at sharing details with another person. One important effect of successful therapy will be the correction of these negative predictions, which in itself is likely to lead to reduced arousal, reduced avoidance, reduced negative emotions, and increased self-confidence. A positive change in beliefs about one's trauma memories is therefore a factor likely to be present in all therapies that succeed in having the person with PTSD confront their symptoms.

### **Conclusions**

The study of memory under extreme stress reveals a spectrum of phenomena including, at the extreme end, unique observations of intense re-experiencing that occur while the person is temporarily dissociated from reality. As has been pointed out for many decades, extreme stress does not have an overall strengthening effect on all aspects of memory but creates a trade-off whereby central, survival-related elements of a scene are often prioritized over the surrounding context although amnesic gaps may also be present. This leads to the

pattern characteristic of PTSD in which the repetitive re-experiencing of a small number of emotional scenes occurs in the context of an overall difficulty in producing a complete and coherent moment-by-moment verbal account of events.

What is not always been appreciated is that the memory of a real-life traumatic event is frequently complex and dynamic, with some moments being clearly recalled and belonging to the past, some moments being re-experienced in the present in the form of intrusive images or ‘video clips’, and other moments remaining vague or disorganized. Importantly for researchers, survivors may produce different accounts of the event for different audiences and so the procedures used to elicit the memory are likely to be critical to any results. Moreover, the accessibility of memories of traumatic events can vary dramatically. This is true both of spontaneous images, which may start to intrude directly after the event or only after a delay, and of semantic knowledge about whether the event occurred at all. Factors such as age when the event occurred, the current relevance of the event, the availability of reminders, and the use of strategies to inhibit recall, may result in periods when knowledge of the event is lost.

The study of PTSD therefore reveals multiple memory phenomena that require explanation by theories that can address the interaction between lower-order processes, both associative and non-associative, and higher-order cognitive processes including appraisals and coping strategies. Theories also need to address the way in which sensations in all modalities (but particularly visual images) can be encoded during states of dissociation or mental defeat brought about by traumatic events. Reliance on concepts and methods developed for healthy samples are only likely to provide a partial account of how brain and body respond during moments of extreme stress.

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