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# What Drives False Memories in Psychopathology? A Case for Associative Activation

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## Abstract

In clinical and court settings, it is imperative to know whether posttraumatic stress disorder (PTSD) and depression may make people susceptible to false memories. We conducted a review of the literature on false memory effects in participants with PTSD, a history of trauma, or depression. When emotional associative material was presented to these groups, their levels of false memory were raised relative to those in relevant comparison groups. This difference did not consistently emerge when neutral or nonassociative material was presented. Our conclusion is supported by a quantitative comparison of effect sizes between studies using emotional associative or neutral, nonassociative material. Our review suggests that individuals with PTSD, a history of trauma, or depression are at risk for producing false memories when they are exposed to information that is related to their knowledge base.

## Keywords

false memory, psychopathology, PTSD, depression, trauma history, associative activation

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Memory aberrations are notable characteristics of posttraumatic stress disorder (PTSD) and depression. For example, individuals with these disorders tend to recollect emotionally negative experiences better than emotionally positive events (Mathews & MacLeod, 2005) and can exhibit overgeneral memories (Ono, Deville, & Shum, 2016). It is not surprising that many studies have focused on the links among PTSD, depression, and memory. The bulk of this research has been concerned with the amount of information that can be correctly remembered (Koriat, Goldsmith, & Pansky, 2000) thereby neglecting false memories (Hertel & Brozovich, 2010). This gap in the literature is peculiar given that the mechanisms that affect correct remembering can, in principle, also affect susceptibility to false memories. For example, the fact that people with depression are most likely to retrieve emotionally negative experiences may reflect an elaborated underlying memory network. Such a network may contain densely intertwined and well-integrated negative memories that are strongly connected and easily activated and retrieved. Spreading activation theorists would argue not only that this network will

produce true negative memories more easily than neutral or positive memories, but also that it will more readily generate false negative memories (Howe, Wimmer, Gagnon, & Plumpton, 2009).

In clinical settings, individuals with PTSD or depression may seek an explanation for their complaints and therapists might ask them to retrieve childhood memories (Patihis, Ho, Tingen, Lilienfeld, & Loftus, 2014). Such dialogues may serve as fertile ground for false memories of, for example, childhood sexual abuse (Loftus, 2005), which in turn might lead to false accusations. From a legal standpoint, the issue of false memories is especially pertinent because victims (e.g., of sexual abuse) who provide statements to the police often suffer from psychopathological disorders such as PTSD or depression (e.g., Bifulco, Brown, & Adler, 1991; Kiser, Heston, Millsap, & Pruitt, 1991). Criminal

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proceedings often boil down to the reliability of such statements because objective forensic (e.g., physical) evidence is often lacking in these cases. Hence, triers of fact are frequently being asked to base their legal decisions on the memories of alleged victims. The conundrum here is that testimony can be plagued by memory aberrations, ones that could end up in false accusations and wrongful convictions (Howe & Knott, 2015; Howe, Knott, & Conway, 2018; Otgaar, de Ruiter, Howe, Hoetmer, & Van Reekum, 2017).

Thus, what memory researchers, legal professionals, and mental health professionals need to know is whether depression or PTSD can make people more susceptible to false memories. Although empirical work in this area has been conducted, results are inconsistent. Indeed, as Bookbinder and Brainerd (2016, p. 1345) recently opined, "With respect to PTSD especially, available data do not provide a consistent picture of false memory effects."

In what follows, we show that when extant studies are viewed from an associative network perspective, their results are not as inconsistent as they might have first appeared. In this review, we have amassed the literature on psychopathology and false memory and demonstrate that the concept of associative activation can explain the discrepant findings in the extant literature. Although limited, most of the work in this domain has focused on false memories in PTSD, depression, and schizophrenia. Because memory distortions in schizophrenia are phenomenologically quite different (e.g., Aleman, Hijman, De Haan, & Kahn, 1999), we focus our review on PTSD and depression, which are both forms of psychopathology that are characterized by associative activation mechanisms. First, we provide a synopsis of the literature on the links among PTSD, depression, and (false) memory. We then show how these links can be explained using associative-activation theory and how the spreading of such activation through memory networks fuels the formation of false memories. Following this, we show how our findings relate to the impact of trauma on the production of false memory. To anticipate our findings, we demonstrate that PTSD, a history of trauma, and depression are strongly linked to false memory vulnerability. However, this link is apparent only when individuals with these problems are presented with emotional material (e.g., trauma-related stimuli) that is directly at the heart of their psychopathology.

## PTSD and Memory

A prerequisite for being diagnosed with PTSD is that a person has experienced or witnessed a traumatic event. One of the key symptoms of this disorder is that the

traumatic event is reexperienced. Indeed, people with PTSD are often haunted by recurrent, involuntary, and intrusive distressing memories of the traumatic event. This observation has led some authors to view PTSD as a disorder of memory (Brewin, 2014; McNally, 2003). Several theories have been postulated to explain the etiology of PTSD and many of them stress the importance of memory (aberrations) in the maintenance of this type of psychopathology.

For example, Ehlers and Clark (2000) proposed that PTSD symptoms mainly occur because traumatic memories are poorly elaborated and integrated, and hence PTSD patients often experience problems with intentionally recalling traumatic details. These authors stress that details in traumatic autobiographical memories are strongly associated with each other so that one external cue (e.g., a certain sound) may automatically evoke a cascade of related traumatic memories (e.g., the memory of being abused). Rubin, Berntsen, and Bohni (2008) place an even greater emphasis on the role of memory in the development of PTSD. They suggest that although the diagnosis of PTSD requires the identification of a traumatic *event*, the *memory* of that traumatic event is even more crucial than the event itself. Finally, Brewin, Dalgleish, and Joseph (1996) argue that traumatic experiences result in two types of memory representations. One type is concerned with the conscious experience of the trauma and includes verbally accessible knowledge. These memories refer to sensory characteristics of the event and might lead to intrusions about the experience. A second type of representation refers to the unconscious processing of the trauma and is called situationally accessible knowledge. This type of memory is automatically retrieved when a person is in a situation that is similar to the experienced trauma and this automatic retrieval can then result in flashbacks of the trauma (see also Meyer et al., 2013). Collectively, these theories put the role of memory at the center of PTSD.

## Depression and Memory

According to the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013), depression involves the presence of depressed mood or anhedonia in combination with a number of vital symptoms such as fatigue, sleeplessness, impaired concentration, and suicidal ideation. Besides these symptoms, depression is also regarded as a disorder that is characterized by several distinct autobiographical memory dysfunctions (Dalgleish & Werner-Seidler, 2014). The basic tenet in depression concerning memory is that depressed people remember the past differently than people without depression. Specifically,

autobiographical memory functioning is compromised in four unique and interrelated ways that impact the maintenance of depressive episodes and affect proneness to new depressive episodes. The first and most notable memory characteristic of depression concerns the biased recollection of negative experiences. Depressed people are more likely to recall negative personal memories than nondepressed people and also have more unbidden intrusive and oftentimes traumatic memories (Williams et al., 2007).

Second, the biased remembrance of negative experience goes hand in hand with impoverished retrieval of emotionally positive autobiographical memories (Gotlib & Joormann, 2010). It is important that even when such positive autobiographical memories are retrieved, they do not enhance positive mood (Joormann & Siemer, 2004). Worse still, some researchers suggest that the retrieval of positive memories might worsen a sad mood (Joormann, Siemer, & Gotlib, 2007). This difficulty in recalling positive autobiographical memories appears to point to impoverished phenomenological qualities of these memories. Depressed people experience positive memories as less vivid and less emotionally intense than nondepressed people (e.g., Werner-Seidler & Moulds, 2012).

A third memory feature of depression is that efforts to cope with painful and traumatic memories seem to be prevalent (Beblo et al., 2012; Dalgleish & Yiend, 2006; Sumner, 2012). Paradoxically, coping strategies such as suppression have been found to increase intrusive memories in depressed individuals (Wenzlaff, Wegner, & Roper, 1988). Suppression might be especially pernicious in depressed individuals as it paves the way to easy access of other upsetting memories (Dalgleish & Yiend, 2006). A related issue here is that when depressed individuals recall a visually rich positive or negative autobiographical memory, such memory is oftentimes seen from an observer perspective. Research has shown that especially for positive memories, such a perspective might not be helpful as it might reduce the emotional impact related to the memory (Nigro & Neisser, 1983).

The final and fourth core characteristic of depression is that autobiographical recollections are frequently categorical and overgeneral in nature. Thus, autobiographical memories of depressed individuals frequently contain repetitions and generalizations across personal experiences. Overgeneral processing hinders the easy retrieval of more detailed autobiographical memories, which may interfere with problem solving strategies (Williams et al., 2007). Taking these memory disturbances into account, depression, like PTSD, can be regarded as a disorder in which memory aberrations are a common occurrence.

## False Memory

What has become known as the *memory wars* revolved around the issue of whether traumatic memories can be repressed and then many years later be recovered in, for example, a therapeutic environment (Lindsay & Read, 1994; Loftus, 1994). Many mental health professionals asserted that they saw patients in their clinical practice who, after extensive treatment, remembered a traumatic childhood incident (e.g., sexual abuse). In response to these clinical observations, many memory researchers argued that psychotherapeutic interventions often involve inherently suggestive prompts, ones that might have led patients to falsely remember entire episodes of trauma. In support of this notion, some patients who initially remembered having been abused, later retracted their claims, arguing that their memories were contaminated because of the suggestive treatment that they had received (e.g., Maran, 2012; Ost, Costall, & Bull, 2002). In many of these cases, people were in therapy because they suffered from some form of psychopathology. The question then arises as to whether their psychopathology might have affected their susceptibility to false memories. Scoboria and colleagues (2017) recently argued that “people struggling with psychopathology who seek help for their symptoms may be particularly vulnerable to suggestions” (p. 160). However, a meta-analytic review looking into the evidence for or against this position has so far been lacking.

In the false memory literature, two general classes of false memory can be distinguished and different paradigms have been invented to experimentally evoke these false memories (Brainerd, Reyna, & Ceci, 2008; Otgaar, Howe, Brackmann, & Smeets, 2016). On the one hand, false memories can be induced by using suggestive pressure, which we will call *suggestion-induced false memories*. On the other hand, false memories can occur spontaneously, without any external pressure, arising predominantly because of the automatic and nonconscious operation of internal memory mechanisms such as spreading activation (see the later discussion). This latter type of false memory has been referred to as *spontaneous false memories*. Both types of false memories can arise in legal cases and in clinical settings (Brackmann, Otgaar, Sauerland, & Jelicic, 2016; Howe et al., 2018).

Several paradigms have been constructed to evoke suggestion-induced false memories. One of the most popular and well-suited techniques is the misinformation paradigm (Loftus, 2005; Otgaar, Candel, Smeets, & Merckelbach, 2010). In general, the misinformation paradigm consists of three stages. During the first stage, participants are presented with a video-taped or real-life crime situation. Following this, participants are presented

with misinformation. This may take the form of suggestive questions (e.g., “What weapon did the hijacker carry?” while actually no weapon was present) or an eyewitness account in which false details have been included. The final stage is composed of a memory test, and here a recurring finding is that participants remember the suggested elements although in reality they were not present. This is known as the misinformation effect.

Another way to induce suggestion-induced false memories is through the use of the implantation paradigm (Loftus & Pickrell, 1995; Otgaar, Scoboria, & Smeets, 2013). In this paradigm, experimenters make contact with the participants' parents. Parents are asked whether they can provide an autobiographical event that their child had truly experienced. The parents are then questioned regarding whether they can confirm that their child did not experience a manufactured false event (e.g., hot air balloon ride). After this, participants are suggestively interviewed about the truly experienced event as well as a false event (e.g., a hot air balloon ride that has never taken place). Specifically, they are told that their parents stated that they experienced both events in their childhood and the instruction is to come up with everything they can still remember about the events. A recent mega-analysis on eight false memory implantation studies showed that 30.4% of statements provided in these studies were classified as false memories (Scoboria et al., 2017). Both of these paradigms, but the implantation paradigm in particular, can be seen as an experimental analogue of how false memories for parts of an event or even an entire event can be elicited in a suggestive therapeutic setting.

The most frequently used paradigm to assess the formation of spontaneous false memories is the Deese/Roediger-McDermott (DRM) paradigm (Deese, 1959; Roediger & McDermott, 1995). In this paradigm, several lists of words that are associatively related to each other are presented to participants (e.g., *night, pillow, moon*). These associatively related words are all linked to a nonpresented critical lure word (i.e., *sleep*). A reliable finding is that participants falsely recall or recognize the critical lure with rates that are often indistinguishable from true memory rates (Roediger, Watson, McDermott, & Gallo, 2001). There are two main reasons for the popularity of the DRM paradigm. First, the DRM false memory illusion is a robust memory effect that is quite resistant against forewarnings or divided attention tasks (e.g., Otgaar, Peters, & Howe, 2012; Peters et al., 2008). Second, emotionally laden DRM memories are likely to evoke emotional reactions quite similar to the ones evoked by emotional autobiographical memories (Rubin & Talarico, 2009).

As will become clear shortly, most work regarding the link between false memory and psychopathology has concentrated on spontaneous false memory. In the

current article, we will focus predominantly on the link between (spontaneous) false memories as elicited by the DRM paradigm and psychopathology. Where possible, we will discuss work on psychopathology and suggestibility, but, as will become obvious, this area is heavily underresearched. The main focus on spontaneous false memory is imperative as research has shown that results from the spontaneous false memory field cannot easily be generalized to the field of suggestion-induced false memory. Specifically, there are studies showing that spontaneous false memories and suggestion-induced false memories are unrelated to each other (e.g., Calvillo & Parong, 2016; Ost et al., 2013; Otgaar & Candel, 2010). This suggests that different mechanisms might lead to the production of these false memories. A key mechanism underlying the production of spontaneous false memory is associative activation.

### **Associative Activation**

Associative activation plays a major role in theories related to memory. One prominent theory concerning the organization of memory is the spreading activation (or associative activation; Howe et al., 2009) account (Anderson, 1983; Balota & Duchek, 1989; Collins & Loftus, 1975; Reder, Park, & Kieffaber, 2009). According to this account, memory contains a multitude of representations or nodes that are interconnected by associative links. Whenever someone experiences an event and encodes information from that event, nodes containing information from that particular event will activate related nodes and concepts in memory networks. This activation will automatically spread to neighboring nodes, thereby triggering related memories concerning the event that may or may not have been part of the original event.

Traditionally, spreading activation accounts have been used to explain semantic priming effects in memory experiments. The semantic priming effect refers to the finding that participants are faster at responding to a certain word (e.g., *cat*) when it is preceded by an associatively related word (e.g., *dog*) relative to when it is preceded by an unrelated word (e.g., *house*; Hutchinson, 2003). The most likely explanation for this effect is that stimulus encoding will lead to spreading activation across related concepts and this occurs quickly and unintentionally outside of conscious awareness (Neely, 1977; Posner & Snyder, 1975).

### ***Associative activation and false memory***

The concept of spreading activation has also attracted the interest of false memory researchers. The activation-monitoring theory (AMT; Roediger, Balota, & Watson, 2001) posits that false memories are the product of

activation of concepts that were not part of the original event but that become activated when information from the actual event spreads through a network of interrelated nodes. For example, when the words “bed,” “night,” and “pillow” are learned, concepts (words) that are strongly related to these words (i.e., “sleep”) will be activated as well thereby giving rise to false memories. AMT stipulates that besides spreading activation, monitoring processes play an important role in editing out the retrieval of false memories. That is, AMT posits that during retrieval, memories are monitored and checked as to whether they were internally generated (e.g., through imagination) or originated from an external source. AMT would predict that false memories are more prevalent among children than adults because children have more problems with source monitoring (Lindsay, Johnson, & Kwon, 1991). However, DRM research has oftentimes found that adults are more susceptible to false memory production than children (Brainerd et al., 2008; Howe et al., 2009; Otgaar & Candel, 2010).

An alternative theory using the notion of spreading activation is associative-activation theory (AAT; Howe et al., 2009; Otgaar, Howe, Peters, Smeets, & Moritz, 2014). AAT proposes that throughout the course of development, people acquire new knowledge and learn new information. The consequence of this is that their knowledge base becomes more elaborated, interrelated, and dense. According to AAT, a knowledge base consists of interrelated nodes that contain representations of information (e.g., autobiographical memories). When someone experiences an event, these nodes will be triggered by associative activation. The links between these nodes will become stronger and associative activation will be faster and more automatic as development proceeds and new knowledge is acquired. AAT posits that during the process of associative activation, nodes will be activated that represent concepts of information that were not really experienced, thereby leading to the formation of false memories. The concepts can be quite diverse and may involve phonological, semantic, or categorical bits of information. It is important that although it has been argued that associative activation mainly has short-lived effects on memory (Reyna, Corbin, Weldon, & Brainerd, 2016), there exists considerable evidence that this mechanism actually has some very long-lasting effects on memory and memory-related tasks (e.g., problem solving; e.g., Hayne & Gross, 2017; Steyvers & Tenenbaum, 2005).

For example, recent experimentation has focused on the adaptive and long-term consequences of false memories (e.g., Howe, Garner, Charlesworth, & Knott, 2011; Otgaar et al., 2015). In this line of research, participants are first presented with the DRM paradigm and then receive a compound remote association task (CRAT)

problem. A CRAT consists of three words (e.g., *walk/beauty/over*) and the task is to come up with the word that connects these words (i.e., *sleep*). The standard finding is that CRAT problems primed by false memories for critical lures are solved more frequently than problems without priming. It is important that such priming effects seem to last after a delay of one week (Howe, Wilkinson, & Monaghan, 2012; Wang et al., 2017). The longevity of associative activation is present not only in adults, but also in young children (Hayne & Gross, 2017).

Another theory used to explain the occurrence of false memories, one that is linked to AAT, is fuzzy trace theory (FTT; Brainerd et al., 2008). According to this theory, when someone experiences an event, two opponent memory traces are stored: verbatim and gist. Verbatim traces store the exact details of an experience whereas gist traces are involved in the processing of the underlying meaning of the event. FTT states that exact details of the verbatim trace cannot be retrieved when there is a long interval between encoding and retrieval. In that case, memory will tend to rely on gist traces. When gist is relied on, false memories might occur. Although gist extraction and FTT seem to be similar to associative activation and AAT, there are important differences between these approaches. First, AAT is a single process theory that only relies on the core notion of association activation. FTT requires an additional step in that both gist and verbatim traces are thought to be important in (false) memory development. Whether a dual-process theory (FTT) or a more parsimonious single-process theory (AAT) is necessary to account for the production of false memories is still a matter for debate (Howe, 2008). Second, associative activation refers to the association between many different types of concepts (e.g., phonological, semantic, categorical), whereas gist extraction is only focused on the retrieval of semantics. Thus, whereas AAT can account for the fact that both semantically and phonologically related items can increase false memory rates, FTT cannot (see Finley, Sungkhasettee, Roediger, & Balota, 2017). Third, and something that is critical to what we are discussing in the current article, recent evidence has demonstrated that mood-congruent false memories are better explained by AAT than FTT particularly for moods that lead to increased automatic processing of information (Zhang, Gross, & Hayne, in press).

### ***Associative activation and psychopathology***

Our focus on the role of associative activation in the production of false memory also aligns well with recent insights into how psychiatric symptoms may develop over time. Increased empirical attention has been

targeted at the notion that mental disorders should be viewed as causal systems integrated in networks of interrelated symptoms (Borsboom & Cramer, 2014; Fried & Cramer, in press). According to this network model interpretation of psychopathology, symptoms are causally linked to other symptoms (e.g., insomnia leads to concentration problems). It is interesting that in network models of PTSD, memory-related symptoms such as amnesia and intrusions are included as well (McNally et al., 2015). This implies that memory-related symptoms might be related to each other, which is also the case in associative memory theories.

For the current purpose, we have specifically focused on the role of associative activation in false memories that might accompany depression and PTSD. There are good reasons for this approach. Based on the literature, it becomes clear that PTSD and depression are uniquely characterized by increased levels of spreading activation. Several theories assert that in PTSD, traumatic autobiographical memories are highly associated to each other in that traumatic memories can automatically activate other related memories (Ehlers & Clark, 2000). Likewise, one common notion is that in PTSD, intrusive memories are the result of spreading activation in which nodes of traumatic information automatically activate each other (Foa & Kozak, 1986; Hayes, Van Elzaker, & Shin, 2012). For example, PTSD is thought to contain fear networks in which nodes representing states of arousal (e.g., threats) predispose individuals to interpret events (even innocuous ones) as potential threats. Furthermore, spreading activation in this network leads to the triggering of related threat nodes that might result in the development of intrusive recollections.

Similar spreading activation mechanisms seem to be at work in depression. Specifically, the bias of retrieving negative episodes in depression might be partly due to spreading activation in a network containing strong associations between concepts containing negative memories. Furthermore, recent evidence indicates that different moods, such as a negative mood and rumination, might be the result of spreading activation mechanisms in an associative memory network (Bar, 2009; Baror & Bar, 2016; Foster et al., 2011). For example, depressed individuals often focus on specific thoughts (e.g., “Why did I make that bad remark to my friend?”) and make associations about possible consequences of these thoughts (e.g., “Will my friend hate me now?”; Bar, 2009). We now turn to the evidence on how psychopathology and false memory are linked to each other.

## Structure of the Review

To examine whether PTSD and depression are linked with false memory propensity, we conducted a literature search. We searched the Web of Science database

using the following keywords: “PTSD AND false memory.” For depression, we entered the keywords “depression AND false memory.”<sup>1</sup>

For PTSD, we identified 57 hits. We looked at the content of these articles to select those studies in which PTSD patients were tested in a false memory paradigm. It is interesting that we found that in many of these articles, participants were tested who did not have PTSD but only experienced a traumatic event (e.g., sexual abuse) and in whom false memory propensity was measured. Because a traumatic precursor is a requirement for PTSD, we will discuss these articles here as well.

We found 12 experimental studies in which PTSD patients underwent some sort of false memory procedure (see Table 1), and 7 articles were identified that examined the effect of trauma on false memory formation in traumatized participants who were not diagnosed with PTSD. Different false memory tasks were employed, but the DRM procedure was used most frequently. This means that the findings of our review are mainly centered around the link between *spontaneous* false memories and PTSD. Furthermore, in those 12 experimental studies, different populations were tested (children, adolescents, and adults).

When we concentrated our search on depression and false memory, we found 166 hits. We were specifically interested in studies that addressed false memories in (sub)clinically depressed individuals. When studies did not have this combination (elicitation of false memories *and* depressed individuals), we excluded them. Based on this criterion, six articles were identified as being useful for our review (see Table 1). The majority of these studies employed the DRM paradigm and all included adult participants. Again, this indicates that the results of our review refer mainly to the link between *spontaneous* false memories and depression. In what follows, we first provide a narrative review of false memory studies for PTSD and depression separately. We will specifically focus on the stimuli that were presented to participants and the impact of this on susceptibility to false memory. Next, we will present a meta-analysis in which effect sizes are juxtaposed against different types of material.

## PTSD, Trauma, and False Memory

A quick glance at the studies in which false memory susceptibility was investigated in PTSD patients reveals a rather inconsistent pattern of findings. That is, some studies failed to find a link between PTSD and elevated levels of false memories (e.g., Dasse, Juback, Morissette, Dolan, & Weaver, 2015), whereas other studies did obtain this link between PTSD and heightened levels of false memories (e.g., Moradi et al., 2015). Our

**Table 1.** Overview of Studies

Study	Category	Population	Journal
Bremner et al. (2000)	PTSD	Adults	<i>Psychological Science</i>
Hauschildt et al. (2012)	PTSD	Adults	<i>Consciousness and Cognition</i>
Triantafyllou et al. (2015)	PTSD	Adults	<i>Annals of Clinical Psychiatry</i>
Jelinek et al. (2009)	PTSD	Adults	<i>Journal of Behavior Therapy and Experimental Psychiatry</i>
Zoellner et al. (2000)	PTSD	Adults	<i>Journal of Abnormal Psychology</i>
McWilliams et al. (2014)	PTSD	Children and adolescents	<i>Behavioral Sciences and the Law</i>
Chae et al. (2011)	PTSD	Children and adolescents	<i>Journal of Experimental Child Psychology</i>
Moradi et al. (2015)	PTSD	Adults	<i>Journal of Abnormal Psychology</i>
Brennen et al. (2007)	PTSD	Adults	<i>Consciousness and Cognition</i>
Hayes et al. (2011)	PTSD	Adults	<i>Journal of Psychiatric Research</i>
Dasse et al. (2015)	PTSD	Adults	<i>Military Psychology</i>
Brewin et al. (2012)	PTSD	Adults	<i>Cognition</i>
Howe et al. (2004)	Trauma	Children	<i>Child Development</i>
Valentino et al. (2008)	Trauma	Children	<i>Development and Psychopathology</i>
Eisen et al. (2002)	Trauma	Children	<i>Journal of Experimental Child Psychology</i>
Howe, Toth, et al. (2011)	Trauma	Children	<i>Child Development</i>
Baugerud et al. (2016)	Trauma	Children	<i>Journal of Experimental Child Psychology</i>
Goodman et al. (2011)	Trauma	Adolescents and adults	<i>Development and Psychopathology</i>
Otgaar et al. (in press)	Trauma	Children	<i>British Journal of Developmental Psychology</i>
Moritz et al. (2005)	Depression	Adults	<i>Depression and Anxiety</i>
Moritz et al. (2008)	Depression	Adults	<i>Memory</i>
Yeh and Hua (2009)	Depression	Adults	<i>Depression and Anxiety</i>
Joormann et al. (2009)	Depression	Adults	<i>Journal of Abnormal Psychology</i>
Toffalini et al. (2014)	Depression	Adults	<i>Personality and Individual Differences</i>
Howe and Malone (2011)	Depression	Adults	<i>Memory</i>

working hypothesis was that when confronted with neutral or nonassociative material, PTSD patients should not exhibit greater susceptibility to false memories than healthy comparison participants, but when emotionally associative material is employed, they should be more prone to report false memories.

To understand why, we return to the principle of spreading activation. This principle holds that false memories are more likely to occur when incorrect associations are strong and are made quickly in a person's knowledge base; that is, they arise automatically and outside of conscious awareness (Howe et al., 2009). The knowledge base and associative networks of PTSD patients will contain many strong links between concepts referring to negative experiences. Indeed, a hallmark symptom of PTSD is the involuntary occurrence of traumatic intrusions as a result of spreading activation (Ehlers & Clark, 2000; Hayes et al., 2012). Thus, when PTSD patients are exposed to stimuli that map onto their own knowledge base, they are extremely likely to make rapid associations, producing linkages to nonpresented constructs, leading to relatively high false memory levels for psychopathology-related information. To be more specific, for PTSD this would

indicate that such stimuli should contain negatively valenced or trauma-related details that are closely linked to each other. The DRM paradigm is an excellent method to study this as previous research has made effective use of different, emotionally valenced word lists (Howe, Candel, Otgaar, Malone, & Wimmer, 2010; Otgaar et al., 2012).

### ***Different types of emotional associative material***

When examined more closely, the studies we analyzed did show that PTSD patients show elevated false memory levels for negative- or trauma-related associative material (see Table 2). For example, Brennen, Dybdahl, and Kapidzic (2007) tested participants with PTSD induced by war-related trauma and traumatized participants without PTSD. Participants were presented with trauma-related DRM lists (e.g., critical lures: blood, concentration camp, war, funeral) and non-trauma-related, neutral DRM lists (e.g., critical lures: sleep, wedding, child, music). After each list presentation, participants were asked to freely recall all the words they could still recollect. The PTSD group had significantly higher

**Table 2.** Effect Sizes of Studies Using Neutral or Non-Associative Material and Studies Using Emotional Associative Material

Studies	Category	Material	Cohen's <i>d</i>
Neutral or nonassociative material			
Bremner et al. (2000)	PTSD	Neutral DRM	0.51 (recognition)
Bremner et al. (2000)	PTSD	Neutral DRM	0.04 (recall)
Hauschildt et al. (2012)	PTSD	Visual neutral DRM	No data <sup>a</sup>
Triantafyllou et al. (2015)	PTSD	Neutral DRM	0.22
Jelinek et al. (2009)	PTSD	Visual neutral DRM	No data
Zoellner et al. (2000)	PTSD	Neutral DRM	No data
McWilliams et al. (2014)	PTSD	Video	-0.45 (incorrect recall, Exp. 1)
McWilliams et al. (2014)	PTSD	Video	0.63 (commission, Exp. 1)
McWilliams et al. (2014)	PTSD	Video	0.5 (incorrect recall, Exp. 2)
McWilliams et al. (2014)	PTSD	Video	0.33 (commission, Exp. 2)
Chae et al. (2011)	PTSD	Interactive event	-0.06 <sup>b</sup>
Chae et al. (2011)	PTSD	Interactive event	-0.12
Chae et al. (2011)	PTSD	Interactive event	0.16
Chae et al. (2011)	PTSD	Interactive event	0.21
Howe et al. (2004)	Trauma	Neutral DRM	No data
Valentino et al. (2008)	Trauma	Words	0.14 (abused group)
Valentino et al. (2008)	Trauma	Words	-0.56 (abused group)
Valentino et al. (2008)	Trauma	Words	-1.13 (abused group)
Valentino et al. (2008)	Trauma	Words	0.63 (abused group)
Valentino et al. (2008)	Trauma	Words	-0.64 (neglected group)
Valentino et al. (2008)	Trauma	Words	0.39 (neglected group)
Valentino et al. (2008)	Trauma	Words	-1.09 (neglected group)
Valentino et al. (2008)	Trauma	Words	0.25 (neglected group)
Eisen et al. (2002)	Trauma	Interactive event	-0.19 (misleading incorrect)
Eisen et al. (2002)	Trauma	Interactive event	0.22 (misleading correct)
Eisen et al. (2002)	Trauma	Interactive event	-0.06 (abuse misleading incorrect)
Eisen et al. (2002)	Trauma	Interactive event	0.09 (abuse misleading correct)
Eisen et al. (2007)	Trauma	Interactive event	-0.27 (overall error misleading)
Eisen et al. (2007)	Trauma	Interactive event	-0.26 (commission abuse-related)
Different types of emotional associative material			
Moradi et al. (2015)	PTSD	Trauma-related video	1.92
Brennen et al. (2007)	PTSD	Trauma-related DRM	0.65
Hayes et al. (2011)	PTSD	Trauma pictures	No data
Howe, Toth, et al. (2011)	Trauma	Negative DRM	No data
Baugerud et al. (2016)	Trauma	Negative DRM	0.59
Goodman et al. (2011)	Trauma	Trauma-related DRM	0.77
Goodman et al. (2011)	Trauma	Trauma-related DRM	0.57
Otgaar et al. (in press)	Trauma	Negative DRM	0.61
Moritz et al. (2005)	Depression	Depression-relevant DRM	0.56
Moritz et al. (2005)	Depression	Depression-relevant DRM	0.88
Moritz et al. (2008)	Depression	Negative DRM	No data
Yeh and Hua (2009)	Depression	Negative DRM	No data
Joormann et al. (2009)	Depression	Negative DRM	0.63
Toffalini et al. (2014)	Depression	Negative script	1.65
Howe and Malone (2011)	Depression	Depression-relevant DRM	1.10

Note: DRM = Deese/Roediger-McDermott paradigm; PTSD = posttraumatic stress disorder.

<sup>a</sup>For some studies, we could not calculate the Cohen's *d* and the original authors could not provide them. In these instances, we refer to "no data."

<sup>b</sup>See Table 4 of the original article. The effect sizes refer to the overall error of misleading questions for the comparison between different types of trauma (e.g., sexual abuse, neglect) and a control group.

trauma-related false memory levels than the comparison group, whereas the two groups did not differ statistically with respect to neutral false memories.

In another study, the authors made use of a visual version of the DRM paradigm in which participants were presented with neutral (electrician at work), positive (child's birthday), negative (surveillance), and trauma-related (real-life war) video scenes (Moradi et al., 2015). The videos contained items that were related to each other and were linked to a related but not presented item (for the trauma video: injured soldier). After the presentation of the videos, military personnel with and without PTSD and a non-trauma-exposed comparison group received a memory recognition task. PTSD participants had higher false recognition rates for the trauma video scenes than the non-PTSD groups. This effect was not seen for the other videos.

Similar results were obtained in a study that included different types of related pictures (Hayes et al., 2011). In that study, combat veterans diagnosed with PTSD and trauma-exposed comparison participants viewed trauma-related, positive, and neutral pictures. After one week, all participants were involved in a recognition test in which they were also presented with related but not presented pictures. False memory rates for trauma-related pictures were higher in the PTSD group than in the comparison group. Again, for other types of pictures, groups did not differ in terms of their false memory levels.

An exception to this pattern is a study by Dasse and colleagues (2015), who presented neutral and trauma lists to veterans with and without PTSD. False memory rates did not differ between the groups. However, as the authors noted, the reason for this is probably that false memory rates were at ceiling levels and that more neutral ( $n = 12$ ) than trauma ( $n = 4$ ) lists were included in their study, thereby decreasing the chance for trauma-related false memory formation.

Brewin, Huntley, and Whalley (2012) instructed individuals with PTSD to write an account of their traumatic event. Participants were also asked to report whether the narrative induced any flashbacks. One week later, they were presented with details of their own and another person's narrative and had to indicate whether the details were from their own or someone else's account. Furthermore, participants had to identify whether the details led to flashbacks at the time of writing. The majority of individuals with PTSD reported having a flashback for a detail that was not from their own narrative. This suggests that they erroneously linked a detail from someone else with a flashback of their own.

We now turn to false memory studies that relied on traumatized individuals, of whom it was unknown

whether PTSD was present. Like individuals with PTSD, people with traumatic experiences may have formed associative networks of trauma memories. If true, the logical prediction would be that for traumatized people, too, false memory rates are heightened when they encounter negatively laden or trauma-related material.

Most of the studies in this domain have tested children with a history of maltreatment. Furthermore, in many of these studies, children had faced serious traumatic adversity. Recent studies in this area confirm that, when traumatized, children are presented with trauma-related or negative associative stimuli, false memory rates are heightened (e.g., Goodman et al., 2011). For example, Baugerud, Howe, Magnussen, and Melinder (2016) provided maltreated and non-maltreated children with emotionally negative and neutral DRM word lists. Emotionally negative false memories were easier to induce in maltreated than in non-maltreated children. In another recent study, we also tested maltreated and non-maltreated children (Otgaar, Howe, & Muris, in press). Here, too, participants were provided with emotionally negative and neutral DRM lists. Again, the maltreated group had higher levels of emotionally negative false memories relative to the non-maltreated group. In the DRM study conducted by Goodman et al. (2011), adolescents and adults with and without documented histories of child sexual abuse were tested. These participants received negative and neutral DRM lists. Participants with a history of trauma showed higher rates of false memory, especially for the negatively valenced lists.

Of importance, in these studies, there were strong indications that the maltreated children had faced serious adversity. For example, in Baugerud et al.'s (2016) study, children were all removed from their caregivers because of serious intrafamilial transgressions. In Otgaar et al.'s (in press) study, the maltreated children were recruited from forensic abuse centers or child interrogation settings. Only children for which there were serious signs of abuse would be referred to these places. This is important because if the traumatic status of what children have experienced is uncertain, it remains difficult to know whether dense and well-integrated associative networks could have been formed. The study of Howe, Toth, and Cicchetti (2011) is a case in point: Although all children in the maltreated group had experienced some form of trauma, not all of them had experienced extreme levels of maltreatment as was the case in more recent studies (Baugerud et al., 2016; Goodman et al., 2011; Otgaar et al., in press). For example, the sample used in Howe et al. comprised 141 maltreated children, with the majority (75.2%) experiencing neglect, 53.3% emotional

maltreatment, 26.2% physical abuse, and 2.8% sexual abuse. Although 58.1% of the maltreated children had experienced more than one subtype of abuse, many in the sample were not exposed to extreme levels of abuse. Because it was not possible to analyze children's memory performance as a function of subtype, Howe et al. were unable to ascertain differences in false memory rates as a function of abuse severity. This may explain why in the Howe et al. study false memory levels did not differ statistically between the maltreated and non-maltreated groups of children.

### **Neutral material or nonassociative material**

Up until now, we have outlined experimental evidence showing that when people with PTSD or a trauma history are faced with negatively laden or traumatic associative stimuli, negative false memories are likely to be elicited. What happens when PTSD (or traumatized) individuals do not receive trauma-related associative stimuli? We will show that when such stimuli are not presented, false memory patterns in PTSD (and trauma) are inconsistent. This may have led some memory researchers to conclude that *in general* there is no link between PTSD and false memory formation.

For example, Triantafyllou, North, Zartman, and Roediger (2015) gave participants with and without PTSD *neutral* DRM lists. They found that the two groups did not differ statistically in false memory propensity. Jelinek, Hottenrott, Randjibar, Peters, and Moritz (2009) provided traumatized individuals with and without PTSD and nontraumatized participants with a visual variant of the DRM paradigm, but did not specifically present them with negative or trauma-related visual scenes (for similar results using videos, see Hauschildt, Peters, Jelinek, & Moritz, 2012). The authors found that false memory propensity was not related to PTSD status. Also, Howe, Cicchetti, Toth, and Cerrito (2004) noted that maltreatment in children was not associated with false memory susceptibility when using neutral DRM lists.

To elicit false memories, researchers have also resorted to procedures other than the DRM paradigm. For example, there is some limited work on the link between trauma and suggestibility. Eisen and colleagues (Eisen, Goodman, Qin, Davis, & Crayton, 2007; Eisen, Qin, Goodman, & Davis, 2002) asked children suggestive questions about a forensic medical examination and assessed whether children with an alleged history of maltreatment were more or less likely to fall prey to such suggestions. The researchers did not find any strong signs that abuse status was linked to accepting suggestive cues (but see Otgaar et al., *in press*). Chae,

Goodman, Eisen, and Qin (2011) also concluded that trauma and suggestibility were unrelated using a similar approach. Valentino, Cicchetti, Rogosch, and Toth (2008) used a self-schema memory task and measured true and false recall in maltreated and non-maltreated children. Specifically, children were presented with several words and were asked whether these words referred to them as a person. An unexpected incidental recall test showed that the two groups of children did not differ statistically in false recall.

Some studies relying on neutral or nonassociative material did find evidence that PTSD or trauma is linked to false memories. However, these might be spurious findings as most studies ( $k = 7$ ) using neutral material did not find any false memory effects. Furthermore, studies finding heightened false memory rates in traumatized or PTSD participants with neutral material are difficult to interpret. For example, in some of these studies (Bremner, Shobe, & Kihlstrom, 2000; Zoellner, Foa, Brigidi, & Przeworski, 2000), PTSD individuals showed higher neutral false memory rates than individuals without PTSD, but there is no theoretically convincing explanation as to why neutral false memories would be raised in these individuals. McWilliams, Harris, and Goodman (2014) exposed children and adolescents with and without a history of trauma to several videos and afterward asked several memory-related questions (free recall, direct question, suggestive questions). The trauma group was more likely to form spontaneous memory errors when being confronted with direct questions than the non-trauma-exposed group. However, this result might purely reflect a general tendency to say yes instead of an increase in false memories per se, something that is unlikely to occur with methods that rely on spreading activation such as the DRM paradigm.

### **Depression and False Memory**

The link between depression and memory has originally been examined in the context of mood congruence effects (Matt, Vazquez, & Campbell, 1992). Mood congruence refers to the phenomenon that stimuli that are similar in affective valence with one's mood are better encoded and retrieved than stimuli of different valence (Blaney, 1986). A wealth of studies has confirmed that depressed individuals remember stimuli better when they match their mood (i.e., negative or depression-related stimuli). Associative activation plays a key role in mood congruence effects that have been documented for depression. Thus, mood congruence has been ascribed to associative activation in an integrated memory network in which nodes and concepts of negative experiences are strongly connected to each

other (Foster et al., 2011). This implies that when depressed individuals are presented with associative stimuli that map onto their own associative networks, false memories are likely to be created.

Moritz, Glascher, and Brassens (2005) were among the first researchers to examine false memory creation in depression. Patients with depression and healthy comparison participants were involved in a DRM procedure in which they received depression-related (i.e., loneliness), delusion-related (i.e., betrayal), positive (holiday), or neutral (window) associative word lists. Moritz and colleagues observed a mood-congruence effect in that false memories for emotionally charged words, especially depression-relevant items, were more likely to occur in depressed patients than in healthy comparison participants. Other studies have replicated this effect.

For example, Joormann, Teachman, and Gotlib (2009) examined the impact of depression on neutral and emotionally negative DRM false memories. Here, too, depressed individuals had elevated levels of emotionally charged false memories compared with a comparison group without depression. Yeh and Hua (2009) tested patients suffering from a major depressive episode and healthy comparison individuals and presented them with neutral, negative, and positive DRM lists. They found that negative false memories were more easily evoked in patients with a depressive episode than among comparison participants. Howe and Malone (2011) also tested participants with a major depressive disorder and concluded that depression-relevant critical lures were more likely to be misremembered in those participants than in healthy comparison participants.

So far, the picture is quite clear. When associative negative or depression-related DRM material is presented to depressed patients, they are more likely to misremember related but not presented items than people with no depression. It is interesting that this effect is not just confined to DRM material but extends to other materials as long as it is associative in nature. A notable example is the study by Toffalini, Mirandola, Drabik, Melinder, and Cornoldi (2014). Their study did not rely on a clinical sample but on participants scoring high and low on depressive-anxious personality traits. Participants received pictures depicting scripts of everyday experiences (e.g., family dinner, going shopping). Each script contained a sequence of several pictures referring to typical actions that are likely to take place in that particular experience. For half of the participants, some pictures depicted a negative outcome of the experience (e.g., bike accident of a boy), whereas the other half received pictures with a neutral outcome. During a recognition task, participants were exposed to presented and nonpresented pictures. It is important

that for some of the nonpresented pictures, a cause (e.g., boy crossing the road) was depicted of the negative/neutral outcome that was shown in the picture. In the high depressive-anxious group, causal false memories for the negative outcome were more likely to be elicited than for the neutral outcome and that this was not the case in the low depressive-anxious group.

To summarize, these studies have revealed a consistent pattern. In line with the notion of associative activation, depressed individuals are particularly prone to the formation of negatively valenced or depression-related false memories. The explanation for this is that depressed individuals are biased toward the retrieval of negative experiences because of increased associative activation of that information in their knowledge base. So, when they are confronted with negative or depression-related information that is associative in nature, they are more likely to spontaneously make incorrect associations than healthy comparison individuals.

## Meta-Analysis

A stronger case could be made for the idea that associative activation underlies raised levels of false memory creation in PTSD, trauma, and depression by performing a meta-analysis on the effect sizes reported in the pertinent studies. There are several routes that can be taken here. First, one could look at the categories (PTSD, trauma, and depression) separately and examine the effect sizes per stimulus type for each distinct type of psychopathology. However, there are several limitations with this strategy. To begin with, there are only a few studies per category (i.e., PTSD, trauma, and depression). Combining them would provide a better and more general estimate of the effects of certain forms of psychopathology on false memory production. Furthermore, when performing such an analysis, it would be informative to contrast effect sizes from studies using negative or trauma-related associative material with those from studies using neutral or nonassociative material. Unfortunately, this cannot be done with studies on depression and false memory because these studies have only used associative material.

With these considerations in mind, we decided to take a second route in performing a meta-analysis (see Table 2; see also the Supplemental Material available online and <https://osf.io/dfpjp/> for all materials and analyses). To this end, we have combined studies that looked at false memory formation in PTSD, trauma, and depression. Of course, one limitation here is that studies differ on certain dimensions such as the paradigm used or which memory test was employed (recall vs. recognition). We have categorized these studies in

terms of those using associative and emotional material (e.g., negative, depression-relevant) and those using neutral or nonassociative material. We then compared effect sizes between these two categories. We have chosen Cohen's  $d$  as our effect size of interest as this effect size represented how large the effect was regarding false memory rates when certain stimuli were presented (e.g., neutral or emotional associative material) between two groups (e.g., PTSD vs. control). For some studies ( $k = 9$ ), no Cohen's  $d$  could be calculated because the relevant information was not described in the text. We emailed the authors of these studies, and from one of those, we received the relevant information. Of the remaining studies, authors did not respond ( $k = 6$ ) or did not have the data anymore ( $k = 2$ ). Furthermore, for some studies, we have added multiple effect sizes, namely, in those cases where false memories were measured in more than one way (e.g., incorrect details in free recall, spontaneous memory errors or errors of commission).

We performed an independent samples Welch  $t$  test on the effect sizes with type of stimuli as a between-subjects variable (Delacre, Lakens, & Leys, in press). As expected, when participants were presented with emotional associative stimuli, effect sizes were statistically higher ( $M = 0.90$ ,  $SD = 0.47$ ) than when neutral or nonassociative material was presented ( $M = -0.07$ ,  $SD = 0.32$ ), Welch  $t(19.2) = 5.74$ ,  $p < .001$ ,  $d = 2.07$ . When we included the Dasse et al.'s (2015) study with an effect size of zero (see earlier discussion), we still found statistically higher effect sizes for emotional associative material ( $M = 0.83$ ,  $SD = 0.52$ ) than for neutral or nonassociative material ( $M = -0.7$ ,  $SD = 0.47$ ), Welch  $t(20) = 5.09$ ,  $p < .001$ ,  $d = 1.85$ . Furthermore, a Bayesian analysis of the data (without the Dasse et al.'s study) using a prior of 0.71 revealed a Bayes factor ( $BF_{10}$ ) of 13001, indicating that our data are more in favor of the alternative hypothesis than the null (i.e., no difference) hypothesis.

Our meta-analysis supports our narrative review that PTSD, trauma history, and depression go along with an increased susceptibility to false memories but only when the stimuli map onto a psychopathology-related knowledge base. So, when individuals with depression or PTSD were presented with neutral or nonassociative stimuli, on average, they were *not* more susceptible to false memories related to that material than comparison participants. However, when emotional associative material was presented, false memories linked to that material increased particularly in individuals with PTSD (or trauma) and depression relative to comparison participants.

We also examined whether our results might suffer from publication bias. First, we contacted all first

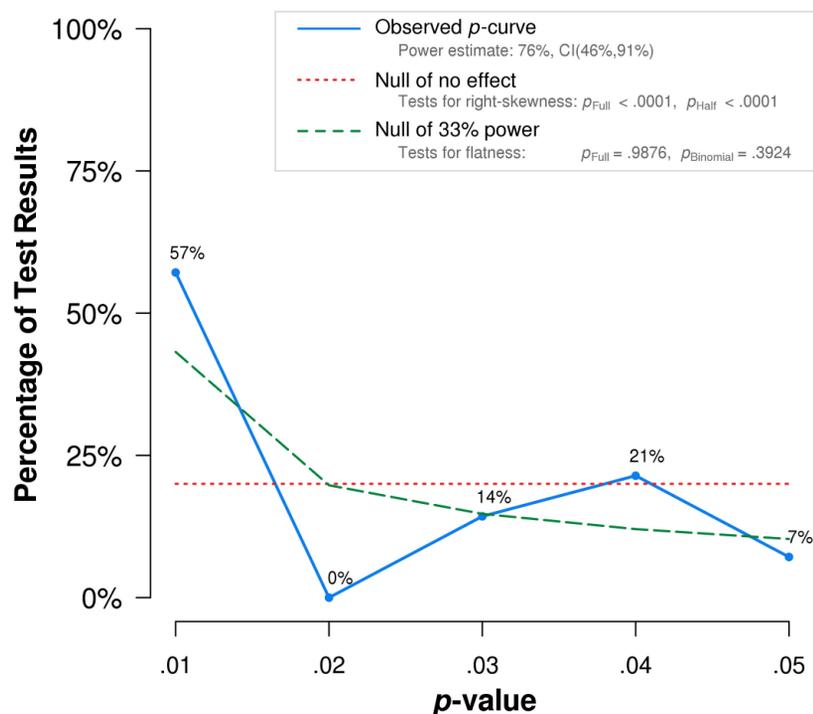
authors of the articles included in this review and asked for any unpublished work on the link between false memory and depression, PTSD, or a history of trauma. Two researchers responded but no unpublished data was present. Second, we conducted a  $p$ -curve analysis to examine the existence of publication bias and examine whether our results show any evidential value (Simonsohn, Nelson, & Simmons, 2014).  $P$ -curve refers to the distribution of statistically significant  $p$  values for a certain number of studies. A  $p$ -curve reflects evidential value when the curve is right-skewed so that there are more low (e.g., .01) than high (e.g., .045)  $p$  values. As can be seen from Figure 1, our analyzed  $p$ -curve is right-skewed implying evidential value and suggests that there are no signs of publication bias.

## Discussion and Conclusion

Associative activation is a key mechanism underlying false memory propensity and omnipresent in psychopathological conditions such as PTSD and depression. When this mechanism is taken into account in studies examining PTSD, depression, and false memories, we suggest that individuals with either PTSD or depression rapidly and automatically activate associations when they are confronted with pathology-related emotional experiences. The consequence of this automatic activation is that false memories for emotional material are more likely to be generated (also see Zhang et al., in press).

In Figure 2, we have depicted a hypothetical example of what happens when emotional associative or neutral material is presented to an individual with PTSD. Although the associative network is simple and for illustrative purposes only, it does show that it contains many nodes/concepts that are highly interconnected with each other. Furthermore, these nodes can represent previously related negative experiences or recollections (e.g., abuse), but they can also reflect feelings (e.g., anger) that are related to a negative experience. Thus, an associative network or knowledge base can contain a multitude of linkages (e.g., semantic, phonological) between different pieces of information (e.g., recollections, emotions; Bower, 1981; Foa & Kozak, 1986; Howe et al., 2010). This perspective is closely aligned with other conceptualizations of how memory and emotions are linked such as the associative network theory of memory and emotion (Bower, 1981; Pessoa, 2017). This network theory assumes that nodes can represent myriad types of information such as recollective information but also different types of emotions.

For example, when someone with PTSD is confronted with a negative related experience (in this



Note: The observed  $p$ -curve includes 14 statistically significant ( $p < .05$ ) results, of which 9 are  $p < .025$ . There were 2 additional results entered but excluded from  $p$ -curve because they were  $p > .05$ .

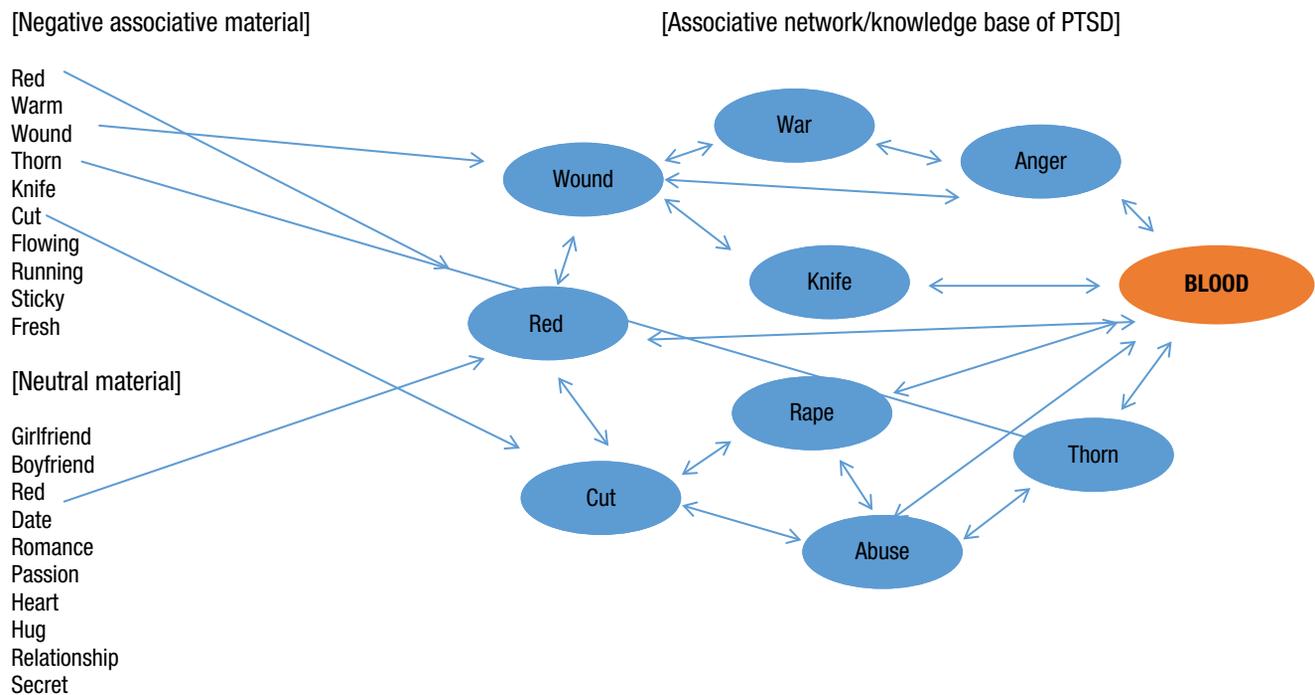
**Fig. 1.**  $P$ -curve analysis of the reported studies on the link among false memory and depression, PTSD, or a history of trauma.

example, a negative DRM list), encoding the list items will activate already existing nodes (e.g., cut) and will automatically activate related nodes as well (e.g., rape). Through this associative cascade, nodes can become activated that were not experienced and this may generate false memories (i.e., blood). As can also be seen in the figure, the creation of a false memory is unlikely to occur when neutral material is presented. Although some items of that material (e.g., red) are similar to negative-related items, other items are less likely to activate nodes in someone's knowledge base, thereby reducing the chance that associative activation will spread to neighboring nodes leading to false memories.

A similar example can be drawn for the relationship between depression or trauma and false memories. The key premise is that for individuals with PTSD (or those who have experienced a traumatic event) or depression, their basic activation levels are already geared toward processing emotionally related experiences. The net result of this memory bias is that the processing of other emotionally related experiences during encoding is facilitated. It is interesting that associative activation that facilitates remembering true emotional experiences in individuals with PTSD, a traumatic history, or depression is at the same time an important player in the

remembrance of false emotional experiences. Indeed, as Howe and Malone (2011, p. 199) argued, "This may represent a double-edged sword—the ease with which true memories of negative . . . experiences come to mind may also increase with the ease with which false recollections become confused with reality."

Our core finding concerning elevated false memories in people with PTSD, depression, or a trauma history fits well with organizational principles of how memory is constructed. Associative networks in people with trauma PTSD or depression could be organized roughly in a similar manner as those in people who have acquired a specific expertise. For example, baseball experts can recollect enormous amounts of information related to baseball (Voss, Vesonder, & Spilich, 1980), and chess experts can retrieve the precise locations of chess pieces that occurred during a chess game (Chase & Simon, 1973). However, as is the case with people who suffer from PTSD, trauma, or depression, research on expertise has demonstrated that superior skills can increase susceptibility to false memory errors. For example, Castel, McCabe, Roediger, and Heitman (2007) presented participants who had high or low knowledge of American football a list of familiar animal names (e.g., lions, broncos, bears) that were also the names of famous football teams. The authors found that the



**Fig. 2.** Hypothetical example of how false memories in PTSD are formed when emotional associative material is presented.

high knowledge group falsely recalled more football-related names (e.g., eagles) than the low knowledge group. Similar mechanisms might be at work in individuals with PTSD, a trauma history, or depression.

The notion that individuals with PTSD, a history of trauma, or depression are more likely to produce emotionally related false memories when confronted with affective rather than neutral stimuli parallels previous work on the processing of emotional stimuli in these individuals. Specifically, an abundance of studies has shown that individuals with PTSD take longer on a Stroop task to color-name trauma-related words than neutral words (e.g., Foa, Feske, Murdock, Kozak, & McCarthy, 1991; McNally, Kaspi, Riemann, & Zeitlin, 1990). This attentional bias reflects an activated associative network that prioritizes threatening information. Such attentional bias might be relevant in the context of false memory effects because it may well be the case that such effects represent false alarms associated with a semantic network that is highly activated.

Although we have emphasized the role of associative activation in this review, other memory-related mechanisms are likely to play a role in false memories as well. That is, associative activation is most likely to impact the encoding of certain experiences, but retrieval mechanisms are also relevant for false memories (Roediger, Balota, & Watson, 2001). One promising candidate that affects retrieval of false memories is cognitive control. Studies show that both PTSD and

depression are characterized by deficiencies in working memory, which is thought to undermine cognitive control (Engle & Kane, 2003; Joormann & Gotlib, 2007; Rose & Ebmeier, 2006; Schweizer & Dalglish, 2011). This implies that when associative activation has led to the activation of nonexperienced nodes, individuals with PTSD, trauma-related histories, or depression may have difficulties in inhibiting false memories because of poor cognitive control (see, e.g., Kimball & Bjork, 2002). When this activation cannot be inhibited, false memories are easily produced. This line of reasoning fits well with research showing that poor working memory is associated with increased levels of false recollections (e.g., Peters, Jelicic, Verbeek, & Merckelbach, 2007).

It is also relevant to examine whether the results of our review can be explained in terms of alternative false memory theories such as FTT. To reiterate, FTT posits that false memories are the consequences of gist extraction when verbatim traces can no longer be retrieved. If this principle is integrated in the current review, then individuals with PTSD, a history of trauma, or depression are more likely to rely on the gist or semantics when retrieving memories. It is true that such individuals have deficiencies in item-specific processing and the retrieval of verbatim traces (Hayes et al., 2011). So, gist traces are then indeed more likely to be retrieved, promoting false memory creation. However, in our view, FTT cannot explain the finding that people with

PTSD, depression, or a traumatic history have a memory bias toward a plethora of different types of memories, including autobiographical ones, and not just semantic ones. Moreover, if verbatim processes are a primary source of true memories and gist processes the primary source of false memories, how can it be that both true and false memories increase for psychopathology-congruent materials? Furthermore, traumatized or depressed individuals are often haunted with rumination and intrusive recollections and a diverse set of negative emotions. This cascade of different symptoms is difficult to explain solely in terms of gist extraction. Hence, the finding that such individuals have a diverse set of emotionally related memories is better accommodated by associative activation theories (AAT and AMT).

### **Other Forms of Psychopathology and False Memory**

In the present review, we have focused on disorders (PTSD, depression) or precursors of disorders (trauma) in which memory aberrations have been well documented. PTSD and depression are often regarded as disorders in which autobiographical memory aberrations are a key feature (Dalgleish & Werner-Seidler, 2014; Rubin et al., 2008). Arguably, associative activation as a principal factor in the development of false memories only possesses relevance for disorders in which memory aberrations are central to the symptomatology. We will now show that if we look at evidence for other memory-related disorders and false memory propensity, then our theoretical principle still holds.

One disorder in which memory deficits are of central importance is schizophrenia. The memory deficits here might be linked to reality distortion. There is an abundance of evidence showing that episodic memory is severely compromised in patients with a diagnosis of schizophrenia (Aleman et al., 1999; Heinrichs & Zakzanis, 1998). Based on this, one might expect that false memory rates are elevated in schizophrenic patients. However, the picture is much more complex concerning the impact of schizophrenia on the creation of false memories.

In line with our review, recent evidence shows that false memories can become elevated in schizophrenic patients when the method used to elicit false memories aligns with specific symptoms of schizophrenia. For example, one core symptom of schizophrenia is delusions. Delusions have been regarded as originating from a dysfunction in cognitive systems related to memory (McKenna, 1991). Bhatt, Laws, and McKenna (2010) recently advanced the idea that delusions have strong similarities with false memories in that both refer to phenomena that are experienced as true but lack

reality. In their study, they tested schizophrenic patients who were currently experiencing delusions, patients not experiencing delusions, and healthy comparison participants. The participants were provided with several DRM word lists and the authors found that false memory rates were highest in the delusion group (for similar results, see Laws & Bhatt, 2005). It is important that studies in which procedures used to elicit false memories did not relate to specific symptomatology have failed to find increased false memory levels in schizophrenic patients (e.g., Moritz, Woodward, Cuttler, Whitman, & Watson, 2004).

Another strong case for the role of associative activation in the link between psychopathology and false memory is to identify disorders with deficits in emotional processing and emotional memory retrieval. For example, a deviant processing of emotions and emotional memories is a characteristic feature of conduct disorder, antisocial personality disorder, and psychopathy (e.g., Dolan & Fullam, 2010; Glass & Newman, 2009). Individuals with these disorders display less automatic associative activation in their knowledge base regarding emotionally negative memories than individuals without these disorders. Based on this observation, one would expect that in these disorders, emotionally related false memories are less likely to occur than in disorders such as PTSD. In fact, there is evidence that points in this direction. Specifically, Thijssen, Otgaar, Howe, and de Ruiter (2013) tested children scoring high and low on callous-unemotional (CU) traits, which closely resemble the emotional detachment part of psychopathy in adult forensic populations (Frick, 2006). These children were presented with neutral and emotionally negative DRM word lists. In line with our prediction, children with high CU traits were least susceptible to the production of false memories for emotionally negative word lists.

### **Psychopathology in Practice**

A relevant issue is whether our meta-analytic findings might be informative for practical settings such as the clinical or forensic field. It is important to emphasize here that the current article was mainly focused on the link between *spontaneous* false memory as evoked by the DRM procedure and certain forms of psychopathology. Hence, we urge caution in generalizing our findings to domains outside the realm of spontaneous false memory such as issues concerning false memories elicited by external suggestive pressure.

Nonetheless, memory scholars have regularly articulated that the events (e.g., birthday party) that we experience daily consist of interrelated and meaning-based details (e.g., presents, singing, cake). Spontaneous false

memories are readily produced when people rely on the underlying meaning of such events. For more forensically related experiences, one might expect the same. Indeed, Holliday, Reyna, and Brainerd (2008) argued that “false memories induced by meaning related information embody several features of forensically relevant memories. For child witnesses of domestic violence, for example, such violence is not usually a single episode but rather a series of repeated events that are substantially similar but not exactly the same” (p. 76).

Many individuals with psychopathology are treated for their disorders and during these therapeutic interventions, such individuals often have to retrieve memories that are related to their specific problems. For example, a patient with PTSD might be asked to recall (parts of) the traumatic event after which a treatment plan is set up. Or a patient with depression might talk about his or her ruminations after which a therapist focuses on these repetitive topics.

The heated debate about recovered memories in the nineties revolved around the existence of repression and whether certain therapies might have caused clients to misremember entire episodes of sexual abuse (Lindsay & Read, 1994; Loftus, 1994). Typically, clients with recovered memories suffered from some form of psychopathological symptomatology and were motivated to know the origins of their symptoms (Ost et al., 2002). In some of these cases, clinicians *suggested* that a buried traumatic event might underlie symptoms, and clients were swayed into falsely believing and remembering that they were traumatized in their past (Loftus & Ketcham, 1994).

Inspired by the recovered memory debate, a plethora of research has been conducted on the elicitation of false memories (e.g., Frenda, Nichols, & Loftus, 2011). Although these studies have been informative (Otgaar, Merckelbach, Jelicic, & Smeets, 2017), some memory researchers have remained critical about the generalizability of their findings (e.g., Pezdek & Lam, 2007; but see Wade et al., 2007). In many of these studies, healthy participants were tested using a variety of different false memory procedures. Although this work has demonstrated the relative ease with which false memories can be generated using different manipulations (e.g., suggestive techniques), it is not always clear how these findings can be translated to clinical settings in which patients with psychopathology are at risk of being confronted with suggestive pressure, potentially leading to the formation of false memories.

More important, based on the recovered memory debate, research intensified in the area of *suggestion-induced* false memories. The focus in these studies was to examine the impact of suggestive prompts on memory. Although this work has undoubtedly revealed that

suggestion can contaminate memory and that this can be perilous in therapeutic settings, our review raises the important question of whether the potential danger of spontaneous false memory formation has been neglected in the clinical field.

Current popular treatment techniques have a specific focus on asking patients psychopathology-related questions. More specifically, many therapeutic interventions intend to change maladaptive autobiographical memories. For example, eye movement desensitization and reprocessing (EMDR; Shapiro, 1995; Van den Hout & Engelhard, 2012) is a popular intervention for dealing with trauma. During EMDR, patients have to retrieve their traumatic memories after which they receive bilateral stimulation in the form of, for example, saccadic eye movements. This procedure taxes working memory, and because of this, the emotional component of the traumatic memory loses some of its value. It is important that during EMDR, patients are exposed to a cascade of questions related to their traumatic memories. Based on our review, one prediction would be that individuals with PTSD, depression, or a trauma history may then be especially prone to the creation of false memories (Howe & Malone, 2011; Otgaar et al., in press).

Furthermore, memory scholars have recently begun to acknowledge that therapies might change memories. For example, Lane, Ryan, Nadel, and Greenberg (2015) stated that “transformation of memories happens all the time, like it or not, and the point of psychotherapy should be to harness this naturally occurring phenomenon for good purpose” (p. 45). Furthermore, Brewin (2015, p. 21) postulated that “psychotherapy creates new memories.” Meanwhile, many therapists have inadequate knowledge about concepts such as repression and false memories (Ost, Easton, Hope, French, & Wright, 2017; Patihis et al., 2014). This knowledge, however, is vital to therapists whose aim is to restructure their patients’ autobiographical memories by using EMDR (Holmes & Mathews, 2010). Therapists, cues that tap into the dominant knowledge network, and patients with certain forms of psychopathology (e.g., PTSD) can create a dangerous triad in which false memory formation is lurking at any corner in this triangle. With this in mind, it is surprising that the literature on “negative” side effects—including spontaneous false memories—of therapeutic interventions is so limited (see Jonsson, Alaie, Parling, & Arnberg, 2014; Lilienfeld, 2007).

## A Look Into the Future

Our review suggests that spontaneous false memory creation is easily evoked in PTSD, depression, and traumatized people if the stimuli being presented are associative and emotional in nature. The studies that have

been conducted regarding psychopathology and false memory frequently included the DRM paradigm. Although we have just described how the findings from these DRM studies can be relevant in practical settings, it is undeniably true that the DRM paradigm suffers from one significant limitation. That is, the DRM paradigm does not concentrate on false autobiographical memories, which are the memories that were debated during the memory wars and that are most relevant in therapeutic and forensic settings.

There is discussion about whether false memory proneness as tapped by the DRM technique is positively related to susceptibility to false autobiographical memories. Although studies show that DRM false memories can be relevant in forensic settings when spontaneous statements are at the foreground of a police investigation (Brackmann et al., 2016; Brainerd, Reyna, & Zember, 2011; Otgaar et al., 2016), there are also studies showing that DRM false memories are not that relevant in practical situations because they are not, or only weakly, related to other types of false memories (Ost et al., 2013; Otgaar & Candel, 2010; Zhu, Chen, Loftus, Lin, & Dong, 2013). This latter finding would imply that different mechanisms are at play in different variants of false memories (but see also Rubin & Talarico, 2009).

### ***Psychopathology and suggestibility***

The field of psychopathology and false memory is in urgent need of false memory studies that use procedures that are most likely to occur in practical settings such as in a treatment context (Scoboria et al., 2017). One obvious example would be paradigms using some form of external suggestion like the misinformation or implantation paradigm. These suggestions are likely to be encountered in clinical situations or other practically relevant situations (e.g., police investigations). Considering the importance of such studies, it is surprising that virtually no research base exists in which suggestion-induced false memories are examined in people with psychopathology. In our review, we only encountered limited work in which the link between trauma and suggestibility was examined (Chae et al., 2011; Eisen et al., 2002; Eisen et al., 2007). Peters, Moritz, Tekin, Jelicic, and Merckelbach (2012) examined the effects of misleading information on false memory in schizophrenic patients and found that people with schizophrenia showed a tendency to go along with misleading information. Otgaar and colleagues (in press) recently tested maltreated and non-maltreated children's ability to accept or resist external misinformation. They found that maltreated children were less prone to the misinformation effect than non-maltreated ones.

Although these studies have provided some important insights, they are limited in the sense that they have not taken into account a theoretical principle that has been the focus of the current investigation. That is, in these studies, the offered suggestions were perhaps not specifically related to the associative networks of the participants. Our prediction is that future studies that do present external suggestions that map into someone's knowledge base will show enhanced false memory rates. Based on the current review, we expect that when, for example, PTSD patients view negatively related associative stimuli such as an aversive video and are then misinformed that certain negatively related details were present, they would be more susceptible to endorsing those suggestions than healthy comparison participants. When these conditions are not met, then it is likely that PTSD patients and comparison participants will not differ in their susceptibility to suggestive pressure.

### **Concluding Remarks**

The current synopsis commenced with an often-heard claim among researchers that there is an inconsistent pattern concerning the link between psychopathology and false memory. We demonstrate, for the first time, that this pattern is quite consistent as long as we glance through the lens of associative activation. For individuals with PTSD, depression, and a history of trauma, false memory susceptibility increases when they are exposed to associative material that is linked to their knowledge base. Indeed, what this review shows is that researchers may have underestimated the occurrence of false memories in treatment settings. Whereas suggestion-induced false memories can certainly occur in these settings, spontaneous false memories are easily created for individuals with these types of psychopathologies. This surely indicates that these individuals are cursed in that the mechanisms leading to their symptomatology also lead to the occurrence of false memories, which might be harmful in legal and clinical settings.

### **Author Contributions**

H. Otgaar developed the review concept. All authors contributed to the study design. Data collection (finding articles, finding effect sizes) was performed by H. Otgaar. H. Otgaar conducted the data analysis. All authors interpreted the data. H. Otgaar drafted the article and all authors provided critical revisions. All authors approved the final version of the article for submission.

### **Declaration of Conflicting Interests**

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

## Supplemental Material

Additional supporting information may be found at <http://journals.sagepub.com/doi/suppl/10.1177/2167702617724424>

## Note

1. Instead of using the term “false memory,” we also used the search terms “false recollection,” “pseudo memory,” “memory distortion,” and “memory error” together with the search term “PTSD” or “depression.” However, no new studies were identified using these new terms.

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