COMMENTARY

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Associative Activation as a Mechanism Underlying False Memory Formation

Henry Otgaar[1,2], Mark L. Howe[1,2], Peter Muris[1], & Harald Merckelbach[1]

[1]Maastricht University, The Netherlands

[2]City, University of London, UK

Correspondence should be sent to: Henry Otgaar, Henry.Otgaar@maastrichtuniversity.nl, Faculty of Psychology and Neuroscience, section Forensic Psychology, Maastricht University.

IN PRESS: *Clinical Psychological Science*

(Accepted August 9, 2018)

Abstract

We recently made the case that associative activation is a viable mechanism underlying false memory formation and hence, also false memory formation in psychopathology (Otgaar, Muris, Howe, & Merckelbach, 2017). Tryon (in press) argued that our description of associative activation did not meet the criteria of causation and explanatory value in order to quality as a mechanism. In this commentary, we explain why we disagree with Tryon. Many studies focused on associative activation and false memory creation. We believe that these studies provide good arguments for associative activation as a likely causal antecedent in the production of false memories.

Keywords: Associative Activation; False Memory; Mechanism, Psychopathology

Associative Activation as a Mechanism underlying False Memory Formation

Which mechanism drives the formation of false memories? This issue has been much debated and is the focus of extensive empirical work (e.g., Brainerd, Reyna, & Ceci, 2008; Howe, 2008). This research domain is not only important for theory building, it also possesses practical momentum as false memories may surface in legal cases or in therapeutic contexts. Understanding the *mechanisms* that generate false memories is critical to informing legal professionals and mental health professionals about memory illusions in general as well as in specific cases.

In a recent paper, Tryon (in press) provided a detailed account of the defining criteria of a *mechanism*. Tryon is to be applauded for making a thorough and well-thought attempt to provide conceptual clarification of a term that is much used in psychological research. Tryon also critically evaluated and advanced the proposition that associative activation is a psychological mechanism. He referred to one of our recent papers (Otgaar, Muris, Howe, & Merckelbach, 2017) in which we argued that associative activation is a mechanism underlying false memory propensity and that it may help to understand why people with certain forms of psychopathology produce false memories. According to Tryon our reference to associative activation as a mechanism does not meet the criteria for a mechanism as no mention was made of causation and explanation. Below, we will explain why we think that Tryon is too pessimistic. Specifically, we will demonstrate that a number of studies imply that associative activation is causally related to the production of false memory and that this corpus of knowledge does possess explanatory value.

What is Associative Activation?

In our earlier paper, associative activation was invoked to explain the formation of false memories and possibly false memory susceptibility in psychopathology (Otgaar et al., 2017). Associative activation has its origins in spreading activation models and refers to the phenomenon that the processing of one concept (e.g., a word such as *bed*) leads to an immediate spread of activation to related concept nodes in one's knowledge base (e.g., *night, moon, blanket*; Anderson, 1983; Anderson & Pirolli, 1984; Collins & Loftus, 1975; Landauer & Dumais, 1997). Specifically, the activation of one concept will activate other related concepts and these "incorrectly" activated concepts (e.g., *sleep*; inasmuch as they were not presented) may be falsely remembered as information that was actually seen or heard (i.e., a false memory).

False memories are more likely to occur when relations between concepts become stronger and associative activation through one's knowledge base spreads faster and becomes more and more automatic. Such increases in associative strength, automaticity, and speed are assumed to be caused by the accrual of experience and concomitant changes that occur in one's knowledge base (e.g., the addition of new concept nodes, restructuring of information in memory) due to both formal (e.g., schooling) and informal (e.g., everyday life) experiences. Several theories, such as associative activation theory (Howe, Wimmer, Gagnon, & Plumpton, 2009) and activation monitoring theory (Roediger, Balota, & Watson, 2001), incorporate the notion of associative activation to explain how false memories arise.

In the past years different research lines have tested whether associative activation underlies false memory creation. We will briefly summarize the main findings. Our objective here is not to provide an exhaustive review of all studies that have been conducted in this area

but to provide a succinct overview showing that there are good arguments to believe that associative activation underpins false memory creation. Importantly, Tryon referred to the work by Kazdin (2007, 2008, 2009) postulating that mechanisms need (1) statistical evidence of mediation and then (2) experimental evidence of causality followed by a (3) theoretical explanation regarding why and how the mechanism works exactly. We agree with these criteria. However, Tryon suggested that associative activation fails the criteria of causation and explanation. We don't agree.

Increasing Associative Activation and False Memory Creation: Causation

If associative activation affects the production of false memories, then increasing the strength of these relations should enhance false memory vulnerability. If true, this would imply a causal role of associative activation in the production of false memories. Several studies have found support for this prediction. In general, these studies have made use of the so-called Deese/Roediger-McDermott (DRM) paradigm (Deese, 1959; Roediger & McDermott, 1995). In this paradigm, participants are presented with lists containing associatively-related words (e.g., *web, insect, bug, fright*) that are not only associated with each other, but also with a non-presented word called the critical lure (i.e., *spider*). Recall and recognition tests show that many participants (sometimes 70% or more) falsely recollect the critical lure as having been presented in the list.

One attractive feature of the DRM paradigm is that the associative strength between list items and the critical lure (referring to backward associative strength (BAS)) and vice versa (referring to forward associative strength (FAS)) can be varied and manipulated. Studies have shown that both BAS and FAS significantly contribute to the formation of false memories (e.g., Arndt, 2012; Brainerd & Wright, 2005; Gallo & Roediger, 2002; Roediger, Watson, McDermott,

& Gallo, 2001). For example, Roediger and colleagues (2001) showed that word lists characterized by higher BAS were more likely to give rise to false memories than lists with lower BAS (see also Arndt, 2012). These researchers also found that BAS explained most variance in false memory production than other factors (e.g., word length). Furthermore, research indicates that it is BAS and not, for example, the interconnectivity between list items (also called semantic density), that accounts for increases in false memory proneness (Howe et al., 2009).

Decreasing Associative Activation and False Memory Creation: Causation

An alternative way to examine whether associative activation causally affects the formation of false memory creation is by lowering the chances of incorrect relations in one's knowledge base. One way in which researchers have examined this is by investigating the impact of divided attention on the susceptibility to false memory. Of relevance here is the notion that as we develop (from children to adults), associative activation becomes more automatic. This means that divided attention would lead to lower false memory rates in children than in adults, because in adults false memories are automatically generated and hence less affected by manipulations such as divided attention.

Research results indeed points in this direction. Otgaar, Peters, and Howe (2012) presented 7- and 11-year-olds as well as adults with several DRM word lists. Importantly, half of the participants received a divided attention task during the encoding of the word lists. In line with our idea, children's false memory rates were considerably lowered when their attention was divided. The most likely explanation for this is that in children (but not adults), associative activation is impeded when, during encoding, they also have to perform another attention-demanding task. Thus, under this condition, associative activation is less likely to spread to related words that were not part of the presented lists.

As expected for adults, divided attention did not decrease false memory rates. On the contrary, we found the reverse pattern in that false memory rates were enhanced in the divided attention group. A possible explanation could be that divided attention might have made associative activation more likely to spread to the underlying theme nodes, something that facilitates false memory formation (see also Dewhurst, Barry, Swannell, Holmes, & Bathurst, 2007, Experiment 1; Pérez-Mata, Read, & Diges, 2002; Peters et al., 2008).

Associative Activation and Development of False Memory Creation: Mediation and Explanation

According to spreading activation theories, children's associative networks are less dense and more poorly integrated than those of adults. The consequence is that for children, associative activation spreads less automatically and associative relations are weaker than for adults (Howe et al., 2009). Thus, the logical prediction is that false memories should increase with age because the activation of related, but not-presented, concepts is more likely to occur in a well-integrated knowledge base. Recent research has confirmed that the DRM false memory illusion is more likely to be elicited in adults than in children, a phenomenon dubbed the developmental reversal effect (for a review, see Brainerd et al., 2008). So, this implies that associative activation leads to false memories but is also mediated by developmental differences in the strength and speed of associations in one's knowledge base.

Such developmental reversal effects are not just confined to semantic memories as measured in the DRM paradigm. Recent experimentation has revealed that episodic memories are also more likely to be distorted in adults than in children (for a review, see Otgaar, Howe, Merckelbach, & Muris, in press). For example, in a series of four experiments, children (4/6-, 6/9-, and 10/12-year-olds) and adults were shown a video (e.g., bank robbery) consisting of

associatively-related details (e.g., robber, vault; Otgaar, Howe, Brackmann, & Smeets, 2016). To create these videos, we conducted a pilot study in which adult participants were presented with several cue words (e.g., pistol, money). Their task was to come up with response items to these words. The response items were then used to develop the video in which the critical cue words were excluded.

In the experiments, participants received an eyewitness account falsely suggesting that a related, but not-presented detail was present during the crime (e.g., gun; Experiments 1, 2). In the last two experiments, half of the children also received the same false information but now an interviewer provided the false suggestions. Finally, participants were presented with a memory test. In all experiments, developmental reversal effects were detected. That is, in the first experiment, adults and 11-year-olds had higher false memory rates than 6/7-year-old children and in the third experiment, adults were more prone to false memory production than 7/8-year old children. In the second experiment, 10/12-year-olds formed more false memories than the 7/9- and 4/6-year-olds. Similar findings were shown in the fourth experiment in which 11/12-year-olds were more likely to give rise false memories than 7/8-year-olds and 4/5-year-olds. Collectively, these results suggest that false memories, regardless of whether they are simply suggested or induced using the DRM paradigm, follow an age-related increase from childhood through to adulthood *because* of increases in the speed and automaticity of associative activation (also see, Otgaar, Howe, Brackmann, & van Helvoort, 2017).

Associative Activation and Consequences of False Memories: Causation and Explanation

If associative activation underpins the creation of false memories, one might argue that such spread of activation will impact and have a causal effect on tasks that also rely on memory

associations, such as priming tasks. One of the first studies in this context was conducted by Howe, Garner, Charlesworth, and Knott (2011). Children and adults were presented with several DRM lists and were then asked to solve compound remote association tasks (CRATs). CRATs involve the presentation of three words (e.g., *apple, family, house*) that are all connected to a single word (i.e., *tree*). For half of the CRATs, their solution was also the critical lure on the DRM lists. When the critical lure was falsely remembered, CRATs were more frequently and more quickly solved than CRATs that were not primed by DRM lists (also see Howe, Wilkinson, Garner, & Ball, 2016). Similar findings have been reported when children and adults attempt to solve analogical reasoning problems (e.g., Howe, Threadgold, Norbury, Garner, & Ball, 2013). These findings fit well with the idea that associative activation (including the emergence of false memories) have positive and adaptive consequences when one is attempting to solve higher level (non-memory) cognitive tasks.

Concluding Remarks

Kazdin (2007, p. 2) noted that a "[m]echanism is the weather of psychotherapy research" thereby referring Mark Twain's (1835-1910) statements that "everybody talks about the weather but nobody does anything about it." We – as cognitive scientists – are cognizant of the importance of mechanisms (e.g., Howe, Rabinowitz, & Grant, 1993). Hence, in this commentary, we have provided evidence that associative activation is the vehicle behind false memories.

Of course, some of the findings that we discussed in this commentary provides a stronger case for calling associative activation a mechanism than others. Specifically, experimental studies in which, for example, BAS or divided attention was manipulated, were conducted to establish a causal relation between associative activation and false memory creation (Otgaar et al., 2012; Roediger et al., 2001). Other studies in which, for example, false memory propensity

was studied in children and adults or in which false memories impacted other memory-related tasks (e.g., compound remote association task), provided explanatory value to the proposition that associative activation is a mechanism causing false memories (Brainerd et al., 2008; Howe et al., 2009).

The search for mechanisms is sometimes referred to as a "chess game" (Kazdin, p. 11) thereby implying that different moves or research perspectives need to be taken before calling something a mechanism. That is the reason why, besides experimental work, memory researchers study false memory susceptibility in special populations such as maltreated children (e.g., Baugerud, Howe, Magnussen, & Melinder, 2016; Howe, Toth, & Cicchetti, 2011; Otgaar, Howe, & Muris, 2017) or people with some form of psychopathology (Howe & Malone, 2011; Otgaar et al., 2017). The rationale behind this research perspective is that these populations have different associative networks and that these differences affect the formation of false memories. Indeed, in our recent paper on false memories and psychopathology, we have demonstrated that DRM word lists (e.g., negatively-related) that are linked to one's knowledge base (e.g., in people who have a trauma history) are more likely to lead to false memories than word lists that do not bear such a connection (Otgaar et al., 2017). As we explain in our paper, associative activation not only provides an appropriate explanation of these findings, but did, in fact, predict these outcomes.

The quest for calling something a mechanism might be seen as problematic in the creation of new theoretical models. That is, in its strictest form, Tryon's (in press) paper suggests that new theoretical models including proposed mechanisms can only be developed when there is sufficient evidence for claiming something a mechanism. This, of course, can be challenging in certain areas where it is difficult to study something directly (e.g., associative activation).

Therefore, we agree with Tryon that in the quest for finding mechanisms it is important to team up with other disciplines (e.g., connectionism). By doing so, new testable hypotheses could be formed in how, for example, associative activation affects the formation of false memories.

Thus, we agree with Tryon (in press) that it is vital to establish whether something can be designated as a mechanism. We do not agree with Tryon when he writes that associative association does not meet the criteria for a mechanism. In the current commentary, we have demonstrated that considerable (experimental) work has focused on associative activation as a causal mechanism, work that has established associative activation as a viable mechanism that leads to the production of false memories. Of course, we do not contend that associative activation is *the sole* mechanism behind false memory formation. What we can say, though, with confidence is that associative activation is *a* mechanism in false memory formation. This knowledge is imperative as it might elucidate how false memories can occur in legal cases and in therapy.

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