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Review article

A review of neurobiological factors underlying the selective enhancement of memory at encoding, consolidation, and retrieval

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ABSTRACT

How is the strength of a memory determined? This review discusses three main factors that contribute to memory enhancement - 1) emotion, 2) targeted memory reactivation, and 3) neural reinstatement. Whilst the mechanisms through which memories become enhanced vary, this review demonstrates that activation of the basolateral amygdala and hippocampal formation are crucial for facilitating encoding, consolidation, and retrieval. Here we suggest methodological factors to consider in future studies, and discuss several unanswered questions that should be pursued in order to clarify selective memory enhancement.

1. Introduction

> *"Of some (experiences), no memory survives the instance of their passage. Of others, it is confined to a few moments, hours or days. Others, again… may be recalled as long as life endures. How can we explain these differences?" (James, 1890).*

Memory is conceptualised within the information processing theory of human cognition as a process whereby information is encoded from the environment, stored and consolidated within neural networks, and subsequently retrieved (Atkinson and Shiffrin, 1968; Baddeley, 2013). The capacity for memory has become increasingly adaptive for modern humans because this function underlies a variety of tasks spanning the recall of survival-related information to the development of language (Gathercole and Baddeley, 2014; Nairne et al., 2007). However, it is well established that information is not equally well remembered because the memory system is also characterised by forgetting, whereby previously encoded information cannot be recalled (Wixted, 2004). The American psychologist, William James, acknowledged this discrepancy over 100 years ago with his question noted above. Since then, researchers have attempted to identify which factors determine whether one event will be enhanced in memory compared to other events (Buchanan and Adolphs, 2002; Javadi et al., 2017; LaBar and Cabeza, 2006; LeBlanc et al., 2015; McGaugh, 2018; Oudiette and Paller, 2013; Rasch and Born, 2013; Watrous and Ekstrom, 2014). Numerous factors have since been found to enhance memory strength by inducing medial temporal lobe activations and oscillatory activity, including stimulus novelty (Courchesne et al., 1975; Kishiyama et al., 2009; Knight, 1996; Li et al., 2003; von Restorff, 1933; for reviews, Kafkas and Montaldi, 2018; see van Kesteren et al., 2012), reward (Gruber et al., 2013; Javadi et al., 2015; Murayama and Kuhbandner, 2011; for review, see Miendlarzewska et al., 2016), future relevance (Badets et al., 2006; Goschke and Kuhl, 1993; Wilhelm et al., 2011; for review, see Stickgold and Walker, 2013), and mnemonic strategies (Craik and Lockhart, 1972; Dresler et al., 2017; Fellner et al., 2016; Maguire et al., 2003; Roediger, 1980). However, these factors tend to elicit memory enhancements at a general level for all similar stimuli in a learning episode, whereas there are factors that can enhance an individual memory exclusively (or rather selectively) compared to other, even similar, stimuli encountered in the same learning episode. Therefore, this review will only consider factors that contribute to selective memory enhancements, and three factors (emotion, targeted memory reactivation, and neural reinstatement) have been chosen to demonstrate how selective memory enhancements can occur at each processing stage: encoding, consolidation, and retrieval. The goal of this review is to raise

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new questions and perspectives regarding an aspect of the memory enhancement literature that receives minimal attention: selective memory enhancements. Additionally, this review aims to consider how factors relating to memory enhancement can be embedded intrinsically within the stimulus, or modulated extrinsically by the experimenter.

2. Encoding

In line with the perspective that memory is an adaptive cognitive function, it is predicted that recall will be superior for emotionally valenced information because positive and negative events are more related to survival and reproduction than neutral events (Adolphs and Damasio, 2000; McGaugh, 2000). Crucially, one of the most persistent findings in memory literature – the emotional enhancement of memory (EEM) effect – concerns the extent to which emotional information is recalled quicker and more accurately than neutral information (Cahill and McGaugh, 1995; Ferré et al., 2015; Kensinger and Corkin, 2003; Pillemer et al., 1986; for reviews, see Buchanan and Adolphs, 2002; Hamann, 2001; LeBlanc et al., 2015). For example, Kensinger and Corkin (2003) asked participants to perform recognition tasks for neutral and negative words that had previously been encountered in a semantic judgement task. The recognition tasks required participants to indicate whether they vividly remembered previously encountered words (versus simply knowing that they were familiar) and to identify which colour the words had been presented in. Hence, the strength and contextual detail of memories were assessed. The results found that not only did all participants vividly remember more negative words than neutral words, but also 17/18 participants had greater source memory for negative compared to neutral words. Consequently, Kensinger and Corkin concluded that emotionality is an inherent stimulus property which incurs quantitative and qualitative enhancements in memory, such that negative information is remembered more robustly (quantitative enhancement) and with more detail than neutral information (qualitative enhancement). Notably, this review will refer to enhancements in the number of stimuli recalled or recognised as 'quantitative memory enhancements', whereas 'qualitative memory enhancements' will refer to enhancements in memory detail such as source memory, and generalisation.

Kensinger and Corkin (2003) proposed that the mechanism through which negative compared to neutral memories become selectively enhanced is related to heightened encoding of emotional information via autobiographical and semantic elaboration. Notably, autobiographical elaboration occurs when newly encoded information is associated with previously established autobiographical memories, whereas semantic elaboration refers to the association of newly encoded information to semantically related memories (Kensinger, 2004; Macrae et al., 2004; Rogers and Kuiper, 1977). Although Kensinger and Corkin did not provide empirical evidence to support this proposal, it is plausible that negative words would be associated with autobiographical memories more easily than neutral words, because autobiographical memories refer to personal events and thus are inherently emotive (Schulkind and Woldorf, 2005). Similarly, semantic elaboration should be greater for negative compared to neutral words because emotional stimuli are often shown to be more semantically related than neutral stimuli (Talmi and Moscovitch, 2004; White et al., 2014). Crucially, previous research has demonstrated that elaborative encoding strategies are associated with increased activity in the prefrontal cortex and medial temporal lobe (Kensinger and Corkin, 2004; Krendl et al., 2006; Savage et al., 2001; Sharp et al., 2004). Therefore, in order to provide more conclusive support for the suggestion that elaborative encoding underlies EEM, research could use multi-voxel pattern analyses to decode which encoding strategies are related to a subsequent increase in remember responses for emotional information.

Dougal and Rotello (2007) noted that participants may have different response strategies for responding to emotional versus neutral words, and recognition accuracy cannot be appropriately compared between two conditions if the conditions differ with respect to response bias (Kroll et al., 2002; Wixted, 2007). Consequently, Dougal and Rotello used receiver operating characteristic (ROC) curves and modelling analyses to investigate differences in recognition accuracy for neutral and emotional words as a function of response bias. In this study, participants learned neutral, negative, and positive words before making old-new judgements on a 6-point confidence scale and indicating whether previously encountered words were explicitly remembered versus simply feeling familiar. The results found that although participants 'remembered' negative words more than positive or neutral words, a response bias was also present such that old judgements were most likely to be made for negative words regardless of whether they had actually been encountered previously. Crucially, modelling analyses revealed that this response bias was the primary cause for the finding that negative words received more remember responses than positive or neutral words, and hence, recall of negative information was actually less accurate than EEM would suggest. Consequently, it could be argued that the increased remember responses for negative compared to neutral words in Kensinger and Corkin's (2003) study may also have been caused by a response bias for negative stimuli rather than the selective enhancement of emotional memories per se (see also, Bowen et al., 2016; Kapucu et al., 2008). Therefore, it must be considered that memory advantages for emotional stimuli may not always be reliable, and instead, could sometimes be caused by bias rather than increased salience, and this raises ambiguity towards the strength of emotion's influence on memory performance.

Despite evidence that emotionality leads to the selective enhancement of these stimuli, it is important to note that dimensional models of human emotion conceptualise emotion within a two-dimensional space of valence and arousal (Barrett and Russell, 1999; Citron et al., 2014; Robinson et al., 2004). Consequently, research has attempted to determine which component of emotion is the contributing factor to memory enhancements. Crucially, evidence suggests that arousal, rather than emotional valence per se, is the inherent stimulus property causing EEM (Bradley et al., 1992; Cahill et al., 1996; de Voogd et al., 2016; Dolcos et al., 2004; Tambini et al., 2017; for reviews, see LaBar and Cabeza, 2006; McGaugh, 2000). Dolcos et al. (2004) investigated this using the subsequent memory paradigm whereby event-related potentials are used to identify patterns of brain activity, during stimulus encoding, associated with subsequent retrieval. Specifically, participants rated the pleasantness of low-arousal and high-arousal pictures that were positively or negatively valenced during fMRI, and subsequently performed a cued recall task. Behavioural findings revealed that regardless of emotional valence, recall was greater for high-arousal compared to low-arousal pictures. Further, fMRI analysis demonstrated that during encoding of subsequently recalled stimuli, activity in the basolateral amygdala, anterior hippocampus, and the entorhinal cortex was greater for high-arousal compared to low-arousal pictures, and these brain regions co-activated consistently more for high-arousal pictures. Hence, these results suggest that a particular dimension of emotion – arousal – is the inherent stimulus property associated with selectively enhancing memories by activating neural patterns associated with successful encoding (see also Kensinger and Corkin, 2004). Notably, these results are also in line with the modulation hypothesis of EEM, which assumes that emotional memories are selectively enhanced because the associated arousal facilitates consolidation via activation of the basolateral amygdala and its interactions with the medial temporal lobe (Cahill and McGaugh, 1998; McGaugh, 2004). Therefore, it seems that emotion contributes to selective memory enhancements during consolidation as well as encoding (see also, Dunsmoor et al., 2015; for reviews, see Hermans et al., 2014; Roozendaal and Hermans, 2017).

The arousal-related enhancement of memory, both during encoding and consolidation, is hypothesised to be linked to the release of adrenal stress hormones such as epinephrine and cortisol (Cahill and Alkire, 2003; Cahill et al., 1994; Carr and Rickard, 2016; Maheu et al., 2004; for reviews, see LaLumiere et al., 2017; McGaugh, 2000, 2004, 2018).

One prediction based on this model is that if adrenal stress hormones are administered exogenously following an encoding phase, memory consolidation will be facilitated and thus recall will be enhanced. In a seminal paper, Cahill and Alkire (2003) investigated this by asking participants to freely recall neutral, pleasant, and unpleasant pictures one week after an initial encoding phase. Importantly, participants received an intravenous administration of either saline solution or epinephrine immediately after the encoding phase, and heart rate and electrodermal skin response were monitored throughout. The results found that recall of recency (final three) pictures did not differ between saline or epinephrine administration post-learning. However, recall of primacy (first three) pictures was greater if participants were administered with 80 ng/kg/min, 3 min of epinephrine compared to saline. Interestingly, it was also found that arousal, as indicated by increased heart rate and electrodermal skin response, was greater during the encoding of primacy compared to recency pictures. Consequently, Cahill and Alkire concluded that epinephrine activity, following post-learning administration, interacts with the arousal associated with a stimulus to facilitate consolidation of high-arousal memories specifically. Therefore, the modulation hypothesis seems correct in its assertion that selective memory enhancements occur for emotional information because high-arousal memory consolidation is facilitated by epinephrine. Moreover, these results indicate that the endogenous consolidation mechanism underlying EEM can be modulated exogenously via adrenal stress hormone administration.

To summarise, increasing the emotion of a stimulus results in an enhancement in memory strength. Despite potential problems with response bias, evidence suggests that emotional valence is an inherent stimulus property which attracts elaborative encoding strategies, and thus quantitative and qualitative memory enhancements may be elicited. Research has also demonstrated that arousal contributes to the selective enhancement of emotional memories by facilitating encoding (and consolidation) through activation of the basolateral amygdala and medial temporal lobe. Importantly, emotional arousal is a factor that can be manipulated exogenously, by administering adrenal stress hormones such as epinephrine, to facilitate endogenous mechanisms underlying memory processing of individual events. Henceforth, EEM may be related to factors that are generated intrinsically within stimuli and extrinsically through interventions, but ultimately both these factors selectively enhance emotional memories by activating the same neural mechanisms. See Table 1 for a summary of the empirical studies cited in this review relating to the effect of emotionality on selective memory enhancements at encoding.

3. Consolidation

The selective enhancement of memory can also be achieved using experimental interventions which directly modulate neural mechanisms underlying memory consolidation. Notably, memory consolidation is dependent on the neural phenomenon of “replay”, whereby the neural pattern of activity representing an encoded behavioural episode (or stimulus) spontaneously reactivates. Repeated “replay” is thought to not only stabilise the initial memory trace but also facilitate its redistribution into the neocortex for long-term storage (Diekelmann and Born, 2010; McClelland et al., 1995; O’Neill et al., 2010). Consequently, targeted memory reactivation (TMR) has been developed as a technique to selectively enhance memories by experimentally biasing the content of neural reactivation. More specifically, using TMR, items are associated with contextual cues during encoding which are subsequently represented during sleep in order to reactivate the memory trace for those items (Belal et al., 2018; Bendor and Wilson, 2012; Cairney et al., 2018; Cousins et al., 2016; Rasch et al., 2007; Rothschild et al., 2017; Smith and Weeden, 1990; for reviews, see Ólafsdóttir et al., 2018; Oudiette and Paller, 2013; Rasch and Born, 2013; Spiers and Bendor, 2014).

In one of the first investigations of TMR, Rasch et al. (2007) asked participants to perform a visuospatial card-pairing task and a finger-tapping task, in the presence of a rose-scented odour, before a retention interval consisting mostly of nocturnal sleep. Crucially, the same rose-scented odour or an odourless vehicle was presented in the retention interval during slow-wave sleep (SWS), rapid eye-movement (REM) sleep, or wakefulness. fMRI analysis revealed that hippocampal activity increased when the rose-scented odour was re-presented during SWS compared to wakefulness. Correspondingly, the behavioural data demonstrated that post-sleep performance on the card-pairing task was greater if the rose-scented odour, as opposed to the odourless vehicle, had been re-presented during SWS compared to REM sleep or wakefulness. In contrast, no such finding occurred for the finger-tapping task or if the rose-scented odour had not been presented during encoding. Consequently, Rasch et al. demonstrated that experimentally induced hippocampal reactivation facilitates the consolidation of hippocampus-dependent (declarative) memories but not hippocampus-independent (procedural) memories. Henceforth, there is evidence in support of the suggestion that the selective enhancement of memories can be modulated exogenously by experimentally influencing neural reactivation and thus memory consolidation of previously encoded stimuli.

Because the contextual cue (rose-scented odour) in the experiments by Rasch and colleagues, was not associated with specific stimuli (rather the entire learning phase), this alone does not fully demonstrate the ability to strengthen selective memories using TMR. However, Rudoy et al. (2009) used a variant of the spatial card-pair task, with the addition of auditory cues, to provide unique auditory-visual-spatial associations. After presenting half of the auditory cues during a post-learning sleep session, decreased error rates for positioning cued stimuli in their learned location were observed during post-sleep testing (compared to the non-cued stimuli), providing evidence for enhancement of specific memories using TMR. To examine the underlying mechanism responsible for TMR, Bendor and Wilson (2012) trained rodents to perform an auditory spatial association task, and recorded reactivation activity in the hippocampus during post-learning sleep. They observed that presenting a task-related auditory cue biased reactivations towards replaying the spatial trajectory previously associated with that auditory cue, revealing the underlying mechanism of TMR: cue-directed biasing of neural replay content towards the targeted memory. More recently, Schreiner et al. (2018) have shown similar findings in humans using TMR in a word-learning paradigm. In this study, participants learned to associate Dutch cue words with German target words before a 3-hour nap in which the cue words were represented auditorily. The results found that auditory cueing during NREM sleep biased neural replay in the same way that occurred during wakeful recall of the associated target word, and theta oscillations coordinated both these reactivation processes.

Numerous studies have now explored which type of memories can benefit from TMR. For example, Cousins et al. (2016) asked participants to learn two serial reaction time (SRT) sequences that were simultaneously presented with high pitch or low pitch auditory tones, before a retention interval in which one auditory sequence was re-presented during SWS. Importantly, during post-sleep testing of the SRT sequences, reaction times were shown to have improved significantly more for the sequence that was acoustically cued during SWS compared to the uncued sequence. Moreover, fMRI analysis revealed that post-sleep performance of the cued SRT sequence, compared to the uncued sequence, elicited greater functional activity and connectivity in brain regions responsible for motor consolidation. Consequently, Cousins et al. provided behavioural and neural evidence demonstrating that after learning multiple SRT sequences, memory consolidation can be biased towards a sequence cued using TMR (see also Antony et al., 2012). Henceforth, the implications of these findings are two-fold. Firstly, it has been further demonstrated that contextual cues can be associated with specific stimuli in order for individual memories to be selectively reactivated and thus enhanced using TMR. Secondly, TMR was demonstrated to selectively enhance another form of memory – procedural memories, in contrast to the negative finding by Rasch et al.

Table 1

Summary of the studies using selective enhancement of memory at encoding.

Citation	N	Age Mean (SD)/ Range	Task/ Paradigm Summary	Stimuli	Manipulation	Findings Summary
Bowen et al. (2016)	73	18-30	Recognition	Pictorial	Emotion	arousal ↑ memory at immediate test but ↓ memory at delayed test, ↑ memory for positive stimuli at immediate and delayed test[1]
Bradley et al. (1992)	152	N/A	Free recall, recognition	Pictorial	Emotion	↑ memory for arousing stimuli
Cahill and Alkire (2003)	42	21.9 (0.7)	Free recall	Short stories	Ephinephrine immediately post-encoding	↑ arousal response for primacy stimuli, ↑ memory for primacy stimuli
Cahill and McGaugh (1995)	18	20.9	Recognition, free recall	Short stories	Emotion	↑ memory for emotionally arousing stimuli[2]
Cahill et al. (1994)	36	27.4 (4.6)	Recognition, free recall	Short stories	Propranolol pre-encoding	↓ memory for emotionally arousing stimuli
Cahill et al. (1996)	8	21.1 (1.1)	Free recall	Film clips	Emotion	↑ memory for emotional stimuli, ↑ activity in right amygdaloid complex for recalled emotional stimuli
Carr and Rickard (2016)	37	35 (9.76)	Free recall	Pictorial	Music pre-encoding	emotional music ↑ memory for stimuli
Dolcos et al. (2004)	16	25-29.6	Cued recall	Pictorial	Emotion	↑ memory for arousing stimuli, ↑ activity in basolateral amygdala, anterior hippocampus, and entorhinal cortex for arousing stimuli
Dougal and Rotello (2007)	60	N/A	Recognition	Verbal	Emotion	↑ memory for emotional stimuli[3]
Dunsmoor et al. (2015)	119	23.4 (3.15)	Recognition	Pictorial	Fear conditioning	fear conditioning ↑ memory for stimuli
Ferré et al. (2015)	159	21.8 (4.4)	Free recall	Verbal	Emotion	↑ memory for emotional stimuli[4]
Kapucu et al. (2008)	22, 23	19.6 (0.8), 71.9 (7.5)	Recognition	Verbal	Age, emotion	↑ memory for negative stimuli in younger and older adults[5]
Kensinger and Corkin (2003)	108	18-33	Recognition	Verbal	Emotion	↑ memory for negative stimuli, ↑ source memory for negative stimuli
Kensinger and Corkin (2004)	28	Young adults	Recognition	Verbal	Emotion	↑ memory for negative stimuli, arousal ↑ activity in amygdalar–hippocampal network, valence ↑ activity in prefrontal cortex–hippocampal network
Maheu et al. (2004)	64	19-36	Free recall	Pictorial	Propranolol, metyrapone, arousal	propranolol ↓ short-term and long-term memory for emotional stimuli, metyrapone ↓ long-term memory for emotional and neutral stimuli
Pillemer et al. (1986)	137	Young adults	Auto-biographical recall	None	Emotion	emotional intensity ↑ autobiographical memory
Tambini et al. (2016)	44	19-34	Recognition	Scene	Emotion	↑ memory for emotional stimuli, ↑ memory for neutral stimuli after encoding emotional stimuli, neural activity during emotional encoding was reinstated during subsequent neutral encoding

[1] Emotional valence and emotional arousal elicited memory and response biases.

[2] The first study to use the same stimuli to create different (neutral and emotional) stories in order to overcome problems associated with having different stimuli in each condition.

[3] Modelling analyses showed a response bias for negative stimuli.

[4] Effects of emotion were modulated by encoding task.

[5] Receiver-operating characteristic (ROC) analyses revealed a response bias for negative stimuli in younger adults and a response bias for positive and negative stimuli in older adults.

(2007).

Next, Tamminen et al. (2017) investigated whether TMR can facilitate qualitative memory enhancements, such as the integration of novel words into an existing mental lexicon (lexical integration). In this study, participants learned novel words (e.g., cathedruke), which were derived from phonologically similar real words (e.g., cathedral), before a 90-minute retention interval containing sleep or wakefulness. Importantly, participants in the sleep condition were acoustically re-presented with half of the learned novel words during SWS. All participants completed test sessions immediately after learning and the retention interval, which assessed recall and recognition of the novel words as well as the speed of lexical decision judgments for phonologically similar real words. This lexical decision task measured lexical integration since reaction times to familiar words increase when there is competition from phonologically similar words in the mental lexicon (Dumay and Gaskell, 2007). Interestingly, it was found that the extent of lexical integration for cued, learned novel words correlated positively with the duration of REM sleep, whereas no such finding occurred for uncued words. These results are in line with previous behavioural and neuroimaging work demonstrating that cueing during SWS modulates the role of REM sleep in memory consolidation (Cairney et al., 2014b; Cousins et al., 2016; Oudiette et al., 2013; Hu et al., 2015). Consequently, Tamminen et al. argue, albeit tentatively, that TMR had a qualitative memory enhancement effect that was dependent on REM sleep, because words that were reactivated (and thus destabilised) during SWS were ‘tagged’ for subsequent reconsolidation and integration during REM sleep. This interpretation is in line with consolidation theories proposing that SWS and REM sleep have complementary roles, such as the sequential hypothesis which assumes that memory consolidation involves the cyclic succession of destabilised memories in SWS being reconsolidated during REM sleep (Ambrosini and Giuditta, 2001; Diekelmann and Born, 2010; Giuditta et al., 1995). Crucially, an important caveat to this interpretation of Tamminen et al.’s findings, however, is that TMR is often shown to induce memory enhancements in the absence of REM sleep correlations (Cairney et al., 2014a; Durrant et al., 2012; Lehmann et al., 2016; Rasch et al., 2007; see also Tucker et al., 2006).

In addition to procedural and semantic memory, TMR has also been studied in relation to emotional memory. He et al. (2015) investigated whether TMR can facilitate memory extinction by re-presenting a conditioned stimulus (CS) in the absence of an unconditioned stimulus (US) during SWS. Firstly, participants performed a fear-conditioning paradigm whereby a mild electric shock (US) was associated with an auditory tone (CS) to elicit fear (conditioned response; CR) as indicated by electrodermal skin response. The same auditory tone (CS), a different auditory tone, or no auditory tone was then re-presented during SWS in a four-hour retention interval containing nocturnal sleep. Following this, electrodermal skin responses to the CS were reassessed, and it was found that responses decreased significantly more post-sleep if the same auditory tone had been represented during SWS compared to a different auditory tone or no auditory tone. These results suggest that TMR may work differently with emotional memories, selectively weakening rather than strengthening them, possibly a consequence of fear conditioning and extinction relying on different neural pathways (Tovote et al., 2015). These findings have practical implications for clinical settings in which TMR could be used to modify fearful memories in psychological disorders such as anxiety and PTSD (see also Simon et al., 2018). In fact, since individuals are asleep during TMR, they are unaware of CS re-exposure, and thus this technique might be preferable to traditional exposure therapies during wakefulness whereby anxiety symptoms can be worsened (Meuret et al., 2012).

He et al.’s results are in line with previous work by Hauner et al. (2013) which demonstrated that if odours, which had been presented whilst participants viewed faces paired with electrical shocks, were re-presented during subsequent SWS, there was a post-sleep reduction in fear response to the faces. Despite this, it must be emphasised that other research has found contradictory findings. For example, Barnes and Wilson (2014) paired electrical stimulation of rats’ olfactory bulb (simulates odour perception) with foot shocks, and subsequently re-applied olfactory bulb stimulation during SWS. The results found that, in complete contrast to fear extinction, rats’ fear responses were actually strengthened post-sleep, as evidenced by increased freezing in response to olfactory stimulation. Similarly, Rolls et al. (2013) found similar results following actual odour delivery in mice. On the one hand, this discrepancy of findings in the TMR and fear conditioning literature could be due to differences between studies in conditioning procedures, such as reinforcement contingencies, and experimental protocols, such as delay between cueing and testing (for a discussion, see Diekelmann and Born, 2015). On the other hand, Barnes and Wilson (2014) did also find that if the olfactory bulb stimulation was reapplied during wakefulness, subsequent fear extinction was elicited. Therefore, the possibility remains that TMR may work differently with emotional memories by inducing inhibitory learning (i.e. extinction) as indicated by He et al. (2015), but perhaps the effect is more complex than our current understanding, and is somehow modulated by other factors, such as sleep versus wake states in rats.

To summarise, the consolidation and resulting selective enhancement of memory can be exogenously modulated by experimentally inducing neural reactivation using TMR. Although the neural mechanism (biased replay) governing TMR was observed in the hippocampus (Bendor and Wilson, 2012), this technique is still effective in enhancing a wide range of memory types, which are not exclusively hippocampally dependent. Table 2 displays a summary of studies referenced in this review relating to the role of TMR in selectively enhancing memories during consolidation.

4. Retrieval

Whilst evidence suggests that emotion and TMR contribute to quantitative and qualitative selective memory enhancements by facilitating encoding and consolidation processes, there is also a factor – neural reinstatement – which enhances the last stage of the memory process: retrieval. Neural reinstatement is related to the encoding specificity principle of context-dependent memory whereby memory performance is found to be optimal when conditions that were present during stimulus encoding are also present during retrieval (Tulving and Thomson, 1973). More specifically, neural reinstatement is the assumption that neural activity associated with stimulus encoding should reoccur during retrieval in order for memory recall to be facilitated (Marr, 1971; McClelland et al., 1995; Norman and O’Reilly, 2003; Teyler and Rudy, 2007). Notably, neural reinstatement depends on the hippocampus because CA3 pyramidal cells have extensive synaptic connections which enable previous patterns of neural activity to be easily reinstated during retrieval (Marr, 1971; Norman and O’Reilly, 2003; Rolls, 2016).

To investigate the role of neural reinstatement in selective memory enhancement, initial studies used a procedure in which fMRI data is analysed, using multivoxel pattern analysis, to assess memory performance as a function of the similarity (measured using correlational analyses) between neural activity during encoding and retrieval (Kuhl et al., 2012; Johnson et al., 2009; Johnson and Rugg, 2007; Polyn et al., 2005; Staresina et al., 2012). Using this paradigm, Staresina et al. (2012) asked participants to learn a series of words associated with specific scenes, and to subsequently perform a cued recognition task in which they indicated whether each word had previously been encountered and recalled its corresponding scene. The results found that not only was the specific neural activity pattern associated with stimulus encoding reinstated in the parahippocampal cortex (PHC) during retrieval, but also the extent of neural reinstatement was greater when participants successfully, compared to unsuccessfully, recalled the corresponding scene for a word. Moreover, Staresina et al. support the role of the hippocampus in neural reinstatement because there was a

Table 2

Summary of the studies using selective enhancement of memory at consolidation.

Citation	N	Age Mean (SD)/Range	Task/ Paradigm Summary	Stimuli	Manipulation	Cue	Phase	Findings Summary
Barnes and Wilson (2014)	90	Rats	Fear conditioning	None	Shock	Olfactory bulb stimulation	SWS, awake	cue during SWS ↑ fear response, cue during wake ↓ fear response[1]
Bendor and Wilson (2012)	4	Rats	Navigation task	None	None	Auditory tone	NREM, awake	↑ memory for auditory-spatial associations cued during sleep[1]
Cairney et al. (2014a)	15	20.4 (3.07)	Cued recall	Picture, location	Emotion	Auditory stimuli	SWS	↑ memory for cued negative picture-location associations
Cairney et al. (2018)	46	19.70 (1.51)	Cued recall	Picture, words	None	Auditory words	SWS, wake	cues during sleep ↑ fast spindles and ↑ memory, picture-word association decoding during cue-induced fast spindles predicted TMR benefit
Cousins et al. (2016)	22	23.5 (4.3)	Serial reaction time task	Number sequences	None	Auditory tones	SWS	↓ RT for cued sequence, ↑ activity in bilateral caudate nucleus, hippocampus, cerebellum, and motor cortex for cued sequence[2]
Crowley and Javadi (in submission)	82	19.98 (2.16)	Free recall	Picture, words	tACS frequency	tACS	SWS, REM	congruent tACS frequency between encoding and SWS ↑ memory
Hauner et al. (2013)	15	24.5 (3.2)	Fear conditioning	Faces	Shock	Odour	SWS	cue during SWS ↓ fear response
He et al. (2015)	96	24.0 (2.4)	Fear conditioning	Auditory tone	Shock	Auditory tone	SWS	cue during SWS ↓ fear response
Lehmann et al. (2016)	62	22.1 (0.5)	Cued recall	Picture, words	Emotion	Auditory words	NREM, REM, wake	↑ memory for emotional picture-word associations cured during NREM sleep, ↑ theta and spindle oscillations for cued emotional stimuli
Rasch et al. (2007)	70	20-30	Visuospatial memory, finger-tapping task	Picture, location, number sequences	None	Odour	SWS, REM, wake	↑ memory for picture-location associations cued during SWS, ↑ hippocampal activity during SWS cueing
Rolls et al. (2013)	6	Mice	Fear conditionng	None	Shock	Odour	NREM	cue during SWS ↑ fear response[1]
Rudoy et al. (2009)	24	19-24	Visuospatial memory	Picture, location	None	Auditory tone	SWS, wake	↑ memory for stimuli cued during sleep
Schreiner et al. (2018)	17	22.45 (2.39)	Cued recall	Words	None	Auditory words	NREM	cueing during NREM sleep biases neural replay in the same way as during wakefull recall, theta oscillations coordinated both reactivation processes
Smith and Weeden (1990)	20	Young adults	Logic task	Verbal	None	Auditory clicks	REM	↑ memory performance for cued participants
Tamminen et al. (2017)	40	19.3	Free recall, recognition, lexical competition	Verbal	None	Auditory words	SWS	extent of lexical integration for cued words correlated positively REM sleep duration

[1] Findings based on research with rodents.

[2] Activity in bilateral caudate nucleus and hippocampus was associated with time in SWS, activity in cerebellum and motor cortex was associated with time in REM sleep.

positive correlation between the magnitude of hippocampal activity during retrieval and the degree of PHC neural reinstatement. Consequently, it seems that memory strength is related to the extent to which hippocampal-mediated neural reinstatement occurs during retrieval. Henceforth, this evidence suggests that neural reinstatement is a factor which selectively enhances memories by facilitating memory retrieval. An important point, however, is that improved recall via neural reinstatement does not necessarily indicate a stronger memory trace in the same way that emotion and TMR selectively enhance memories, but rather this form of enhancement could alternatively be explained solely by a stronger recall mechanism.

Although Staresina et al.'s (2012) findings support the assumption that the hippocampus is implicated in neural reinstatement; the temporal resolution of fMRI is limited because hemodynamic responses are used to assess neural activity (Huettel et al., 2004). Consequently, more recent studies have employed neuroimaging techniques with better temporal resolution to determine which specific oscillatory mechanisms underlie the role of the hippocampus in neural reinstatement (Jafarpour et al., 2014; Kerren et al., 2018; Lohnas et al., 2018; Parish et al., 2018; Staresina et al., 2016; Yaffe et al., 2014). For example, Staresina et al. (2016) recorded intracranial EEG (iEEG) activity in the hippocampus of pre-surgical epilepsy patients whilst they performed a cued recognition task for word-scene pairs (based on Staresina et al., 2012). Notably, time-frequency analyses were used to compare frequency-specific oscillatory activity during encoding and retrieval. The results furthered Staresina et al.'s (2012) findings by demonstrating that when participants successfully recalled the corresponding scene for a word, greater hippocampal neural reinstatement was elicited during periods of high gamma activity (˜50–90 Hz) and low alpha activity (˜8–12 Hz). In contrast, no such finding occurred when the corresponding scene was not recalled. Crucially, increased gamma activity has previously been implicated in synchronising CA3 pyramidal cell firing rates, whereas decreased alpha activity reflects an increase in available mnemonic information during retrieval (Bartos et al., 2007; Hanslmayr et al., 2016). Thus, these results indicate that synchronising CA3 activity plays an important role in neural reinstatement and the resulting selective memory enhancement.

Having said this, there are neurobiological memory models, such as the spectro-contextual encoding and retrieval theory (SCERT), which argue that oscillatory activity in any frequency band, rather than specifically gamma band activity, can underlie the selective enhancement of memories (Canavier, 2015; Hanslmayr and Staudigl, 2014; Siegel et al., 2012; Sutterer et al., 2018; Watrous and Ekstrom, 2014; Watrous et al., 2015, 2018). Specifically, SCERT emphasises that oscillatory activity occurs at different frequencies between different encoding events, and it is the reinstatement of this frequency-specific oscillatory activity during retrieval which underlies neural reinstatement (hereinafter referred to as oscillatory reinstatement) and thus selective memory enhancement. Frequency-specific oscillatory activity is assumed to underlie neural reinstatement because such activity coordinates neural mechanisms (phase synchronisation and cross-frequency coupling) related to neural communication and plasticity (for reviews, see Canolty and Knight, 2010; Fell and Axmacher, 2011; Fries, 2005; Jutras and Buffalo, 2010; Womelsdorf et al., 2007). Hence, SCERT assumes that selective memory enhancement is not elicited by the occurrence of specific oscillatory activity per se, but rather selective memory enhancement is elicited when the frequency of oscillatory activity during retrieval is congruent with that which occurred during encoding. This is known as the oscillatory reinstatement hypothesis (Javadi et al., 2017).

In attempt to provide the first causal evidence for the oscillatory reinstatement hypothesis, Javadi et al. (2017) used transcranial alternating current stimulation (tACS) to experimentally induce implicit neural contexts during encoding and retrieval. Notably, tACS is a non-invasive electrical brain stimulation technique which has the capacity for neuronal entrainment whereby neural oscillations synchronise to the specific frequency of stimulation (Antal and Paulus, 2013; Helfrich et al., 2014; Strüber et al., 2014). In Javadi et al.'s study, participants performed a word recognition task, and tACS was administered to the left dorsolateral prefrontal cortex (DLPFC) at either the same or different gamma frequency during encoding and retrieval. Compared to a sham stimulation condition, memory accuracy was greater when participants received the same frequency of stimulation during encoding and retrieval (60 Hz & 60 Hz or 90 Hz & 90 Hz). In contrast, no memory enhancement occurred between sham and active stimulation conditions if participants received different stimulation frequencies during encoding and retrieval (60 Hz & 90 Hz or 90 Hz & 60 Hz). Consequently, these results demonstrate that memory retrieval is not enhanced in the presence of gamma activity per se, but rather retrieval is enhanced when there is congruency between the frequency of oscillatory activity that occurred during encoding and retrieval (see also Crowley and Javadi, in submission). Moreover, these results demonstrate that, similarly to emotion and TMR, the effect of oscillatory reinstatement on selective memory enhancement can be modulated exogenously using electrical brain stimulation.

Despite this, it seems that the congruency of frequency-specific oscillatory activity during encoding and retrieval does not always lead to the selective enhancement of memories. The reason being that evidence indicates that oscillatory reinstatement effects may be dependent on congruency between other contextual features during encoding and retrieval (Staudigl and Hanslmayr, 2018; based on Staudigl et al., 2015). In this study, participants learned a series of words presented visually or acoustically, and performed a subsequent recognition memory task in which cue words were also presented visually or acoustically. The behavioural data demonstrated that when words were initially presented acoustically, recognition memory performance was greater if the cue words were also presented acoustically (match condition) compared to visually (mismatch condition). Interestingly, time-frequency analysis of magnetoencephalography recordings revealed that although reinstatement of theta (6–8 Hz) activity occurred in both match and mismatch conditions, the extent of oscillatory reinstatement was greater for remembered words in the match condition, whereas it was greater for forgotten words in the mismatch condition. Therefore, these results suggest that oscillatory reinstatement enhanced memory retrieval when sensory modalities were congruent between encoding and retrieval, whereas memory retrieval was impaired by oscillatory reinstatement when sensory modalities were incongruent. Henceforth, it seems that the selective enhancement of memories by theta activity reinstatement may be limited to conditions in which there is also congruency between other contextual features at encoding and retrieval.

Crucially, though, it is important to highlight, here, that a growing body of evidence indicates that the 6–8 Hz (theta) oscillation may be a unique case in that it has a role in coding learned information that does not seem to rely on reinstatement (for review, see Schreiner and Rasch, 2017). In fact, these effects have been found to be independent of sensory modality (Michelmann et al., 2016), memory stage (Fuentemilla et al., 2010; Michelmann et al., 2018), and sleep state (Schreiner et al., 2018, 2015). Hence, the consistent evidence that the phase of theta has a specific memory function may be seen as a challenge to the assumption that frequency-specific activity must be reinstated between encoding and retrieval to exert selective memory enhancements.

To summarise, neural, or oscillatory, reinstatement is a factor selectively enhancing memory retrieval. Whilst early research implicated CA3 pyramidal cells as having a functional role in this enhancement process, later research has indicated that oscillatory mechanisms are also fundamental. Moreover, evidence highlights that the extent of the role of oscillatory mechanisms in this process is related to the extent to which there is congruency between frequency-specific oscillatory activity during encoding and retrieval, rather than the mere presence of oscillatory activity. Although the effect of oscillatory reinstatement on

Table 3
Summary of the studies using selective enhancement of memory at retrieval.

Citation	N	Age Mean (SD)/ Range	Task/Paradigm Summary	Stimuli	Method	Findings Summary
Jafarpour et al. (2014)	11	23.0 (2.0)	Cued recall	Picture, word	MEG	retrieval cues triggered neural reinstatement
Javadi et al. (2017)	70	22.12 (2.16)	Free recall	Picture, word	tACS	congruent tACS frequency between encoding and retrieval ↑ memory[3]
Johnson and Rugg (2007)	26	18-35	Recognition	Picture, word	fMRI	scene recollections ↑ activity in regions associated with scene encoding (left occipital cortex and anterior fusiform gyrus), sentence recollections ↑ activity in regions associated with sentence encoding
Johnson et al. (2009)	16	18-31	Recognition	Verbal	fMRI	neural reinstatement elicited during recollection and familiarity judgements
Kerren et al. (2018)	24	22.1 (4.7)	Cued recall	Picture, word	EEG	neural reinstatement is modulated by theta phase
Kuhl et al. (2012)	18	18-27	Cued recall	Picture, word	fMRI	temporal lobe and prefrontal cortex activity during encoding indicate picture category, classifier estimates of picture category correlate positively with memory
Lohnas et al. (2018)	5	19-42	Recognition	Picture	iEEG[1], ECoG[2]	neural reinstatement in hippocampus and occipitotemporal cortex, extent of neural reinstatement correlates positively with hippocampal encoding
Parish et al. (2018)	Simulation	N/A	None	None	None	successful memory encoding and retrieval rely on desynchronisation of neocortical alpha and synchronisation of hippocampal theta
Polyn et al. (2005)	9	18-27	Free recall	Picture	fMRI	encoding-related activity for picture category was reinstated during category recall
Sederberg et al. (2007)	52	8-53	Free recall	Verbal	iEEG	reinstatement of gamma activity in hippocampus, prefrontal cortex, and left-temporal lobe ↑ memory
Staresina et al. (2012)	20	20-35	Cued recall	Picture, word	fMRI	neural reinstatement in parahippocampal cortex, extent of neural reinstatement ↑ memory, extent of neural reinstatement correlates positively with hippocampal activity
Staresina et al. (2016)	11	23-51	Cued recall	Picture, word	iEEG	periods of high gamma activity and low alpha activity ↑ neural reinstatement in hippocampus
Staudigl and Hanslmayr (2018)	24	19-26	Recognition	Verbal	MEG	reinstatement of theta activity ↑ memory when stimulus modality was congruent between encoding and retrieval and ↓ memory when stimulus modality was incongruent
Sutterer et al. (2018)	51	18-35	Visuospatial memory	Picture	EEG	neural reinstatement in the alpha-band, alpha reinstatement is related to accuracy and speed
Wimber et al. (2012)	16	20-28	Recognition	Verbal	EEG	neural reinstatement in the frequency of background flicker that occurred during encoding
Yaffe et al. (2014)	32	33.5 (2.2)	Cued recall	Verbal	iEEG	neural reinstatement is mediated by high-gamma activity that precedes theta activity in the temporal lobe, timing of theta and gamma activity changes between encoding and retrieval

[1] iEEG: intracranial EEG.

[2] ECoG: electrocortigography.

[3] This study used transcranial alternating current stimulation (tACS), rather than neuroimaging, to investigate neural reinstatement because it was the first study attempting to provide causal evidence that reinstatement of neural oscillations facilitates memory.

selective memory enhancement is undermined by findings that retrieval is impaired given certain contextual conditions and that theta activity may have a unique role that is independent of reinstatement, the evidence overall implicates oscillatory reinstatement as another factor that is produced endogenously as well as exogenously, and which selectively enhances memory processing. See Table 3 for further information relating to the studies discussed in this section regarding neural, or oscillatory, reinstatement and memory enhancement during the retrieval phase.

5. Future directions

Since it has been shown that the utility of oscillatory reinstatement for selectively enhancing memories may be dependent on congruency between other contextual features, future research should determine whether there are conditions under which the other hitherto mentioned factors are unable to exert selective memory enhancements. For example, adrenal stress hormone administration has a dose-dependent inverted-U effect on emotional memory consolidation such that memory is impaired at high doses (Roozendaal, 2000). Importantly, the endogenous release of adrenal stress hormones fluctuates according to circadian rhythms (Leliavski et al., 2015). Therefore, perhaps post-learning administration of adrenal stress hormones would impair memory performance during periods of the day when endogenous levels are already high, such as the morning. Additionally, the successful enhancement of memory following auditory cueing during sleep has only been demonstrated when auditory cues are paired with encoding stimuli in controlled laboratory settings. However, auditory stimuli are experienced in most environmental contexts (Heittola et al., 2013). Therefore, auditory cues may not be an effective tool for enhancing memories using TMR in the real world since attempts to pair auditory cues with encoding stimuli may be less successful when there is competition from similar environmental stimuli. Additionally, future research should ask; which mechanisms determine whether emotional memories are selectively weakened versus enhanced by TMR? Do neural and oscillatory reinstatement enhance memory strength or simply the ability to retrieve a memory? Do these factors have the same selective memory enhancement effects for patient groups and healthy populations? Can free recall, cued recall, and recognition be selectively enhanced by the same mechanisms? And, can these mechanisms be combined for a greater effect or will this lead to a reduction in efficacy?

6. Conclusion

To conclude, although there is mixed evidence regarding the role of each factor, this review has demonstrated ample theoretical and empirical evidence to suggest that each stage of memory processing is selectively enhanced by factors that are not only inherent within stimuli, but also those that constitute experimentally induced interventions. Firstly, emotion, or specifically arousal, is an inherent stimulus factor which selectively enhances memories quantitatively as well as qualitatively by facilitating encoding and consolidation, and this mechanism can be modulated exogenously via adrenal stress hormone administration. Targeted memory reactivation (TMR) is an experimentally induced intervention that selectively enhances memory consolidation by biasing the endogenous mechanism of neural replay. Finally, neural, or oscillatory, reinstatement is another factor produced endogenously, but can be modulated exogenously, which contributes to selective memory enhancement by facilitating the retrieval process. Therefore, in answer to William James' question, the selective enhancement of memories can be explained by the effects of both stimulus-inherent and experimentally induced factors at each stage of memory processing. Crucially, future research must examine the scope of these factors, whether their effects are constrained by other conditions, and whether these factors can be combined and used to enhance memory in educational and occupational settings that rely on optimal memory performance.

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Appendix A The Peer Review Overview and Supplementary data

The Peer Review Overview and Supplementary data associated with this article can be found in the online version, at doi: https://doi.org/10.1016/j.pneurobio.2019.04.004.

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