MOTIVATIONAL INFLUENCES ON MEMORY

Vishnu P. Murty and Kathryn C. Dickerson ☆

ABSTRACT

Motivation significantly influences learning and memory. While a long history of research has focused on simple forms of associative learning, such as Pavlovian conditioning, recent research is beginning to characterize how motivation influences episodic memory. In this chapter we synthesize findings across behavioral, cognitive, and educational neuroscience to characterize motivation's influence on memory. We provide evidence that neural systems underlying motivation, namely the mesolimbic dopamine system, interact with and facilitate activity within systems underlying episodic memory, centered on the medial temporal lobes. We focus on two mechanisms of episodic memory enhancement: encoding and consolidation. Together, the reviewed research supports an adaptive model of memory in which an individual's motivational state (i.e., learning under states of reward or punishment) shapes the nature of memory representations in service of future goals. The impact of motivation on

☆ Authors have made equal contribution to this work.

Recent Developments in Neuroscience Research on Human Motivation
Advances in Motivation and Achievement, Volume 19, 203–227

ISSN: 0749-7423/doi:10.1108/S0749-742320160000019019

learning and memory, therefore, has very clear implications for and applications to educational settings.

Keywords: Medial temporal lobe; hippocampus; ventral tegmental area; amygdala; reward; punishment

Motivation is central to human behavior: it affects the decisions we make, the experiences we seek, and consequently what we encode and remember. We often experience ease in remembering something we are motivated to do (e.g., fill out the adoption paperwork for a new puppy), but difficulty in remembering something we are unmotivated to do (e.g., fill out tedious forms at work). Although an extensive literature from behaviorists and learning theorists has studied how learning and memory can be influenced via reinforcement and feedback (Bouton, 2007), relatively little research has focused on how motivational drive influences memory. Similarly, a long history of educational psychology has investigated how feedback and performance testing influence learning (Darling-Hammond, 1994; Kluger & DeNisi, 1996), with comparatively less research investigating how leveraging students' motivational states can facilitate better learning. An emerging literature based on behavioral and cognitive neuroscience, however, has begun to explore the determinants of memory success as they relate to motivation, which has broad implications for and applications to educational settings.

In this chapter, we describe the neural circuitry involved in motivated memory and provide empirical evidence characterizing how motivation directly influences both what and how we remember. We particularly focus on how motivation influences declarative and episodic memory systems. These systems not only support our memory for events but also influence how we retrieve memories to support a variety of adaptive behaviors including reasoning, conceptual knowledge, creativity, decision-making, and future-oriented thinking (Shohamy & Turk-Browne, 2013). To better understand these systems, we integrate converging evidence across animal models and human research that supports a role for motivation in influencing multiple stages of memory, including encoding and consolidation. Furthermore, we delineate how different types of motivational states (e.g., anticipated reward vs. punishment) recruit distinct neural networks to

shape what we learn and remember. Lastly, we discuss the implications of these findings for education.

NEUROANATOMICAL SUBSTRATES OF MOTIVATED MEMORY

A substantial body of evidence converging from animal models and human literature implicates the medial temporal lobe (MTL) in memory processes. The MTL consists of the hippocampus and the surrounding parahippocampal gyrus, including perirhinal cortex, parahippocampal cortex, entorhinal cortex, and subiculum. Some of the earliest evidence supporting the role of the MTL in memory came from human patients with damage to the MTL who exhibited profound loss of episodic memories – both an inability to recall past events (retrograde amnesia) and an inability to form new memories (anterograde amnesia) (Squire, 1992). Subsequent animal studies which lesioned or temporarily inactivated parts of the MTL corroborated human patient evidence that the MTL is necessary for episodic memory encoding (Eichenbaum, Yonelinas, & Ranganath, 2007). Furthermore, functional magnetic neuroimaging (fMRI) studies investigating human memory support a role for the MTL in both episodic encoding and retrieval (Kim, 2011; Spaniol et al., 2009).

While the entirety of the MTL is known to support declarative memory, important distinctions have been made regarding the specific functions of the hippocampus proper versus the surrounding MTL cortex (Davachi, 2006; Ranganath, 2010). Namely, these discrete regions within the MTL are thought to support different types of memories. Cortical MTL, which includes perirhinal and parahippocampal cortices, supports the encoding of isolated representations of items and contexts, respectively. In contrast, the hippocampus proper supports flexible, integrative relationships between these unitized constructs and binds them into flexible, mnemonic representations. For example, whereas memory for the cover of your favorite book may only rely on your cortical MTL, memory for the exact location in which you first saw that specific book cover would be supported by the hippocampus.

The MTL does not act in isolation to support human behavior. It is richly connected with a host of brain regions, including areas closely associated with motivation, such as ventral tegmental area (VTA), ventral striatum, and ventral prefrontal cortex (vmPFC), comprising what are known

as the mesolimbic (VTA to ventral striatum) and mesocortical (VTA to vmPFC) circuits, which for simplicity we refer to in this chapter collectively as the mesolimbic pathway (Fig. 1). Critically, the interactions of the hippocampus with regions throughout the mesolimbic circuit are thought to support motivation's influence on memory. There are two predominant animal models illustrating how interactions within the mesolimbic circuit facilitate memory. Lisman and Grace (2005) proposed the VTA-hippocampal loop, which describes how newly detected information in the hippocampus is sent as a novelty signal via the subiculum, nucleus accumbens, and

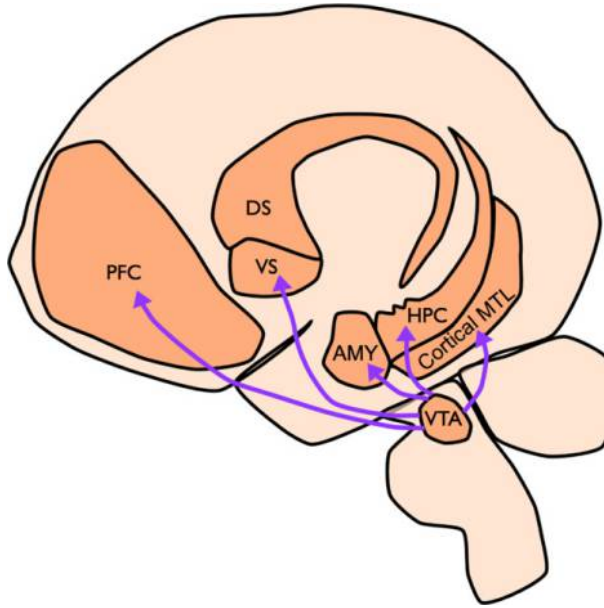

Fig. 1. Key Anatomical Regions Engaged during Motivated Memory Encoding and Consolidation. *Notes*: The ventral tegmental area (VTA) projects dopamine throughout the mesolimbic circuit (depicted here). The hippocampus (HPC) is critical for long-term memory formation and creating rich memories (binding items in a specific context). The HPC and VTA are engaged during reward-motivated memory formation. The amygdala (AMY) and cortical medial temporal lobe (MTL) are engaged during punishment motivated memory encoding (e.g., shocks as opposed to rewards). The prefrontal cortex (PFC) increases signal to noise and may distribute reward information throughout this network. The ventral striatum (VS) encodes reward valuation. The dorsal striatum (DS) is included as a reference point. Anatomical connections are adapted from Shohamy and Adcock (2010).

ventral pallidum to the VTA. Subsequently, neurons in the VTA release dopamine (as a result of novelty signals) into terminals in the hippocampus, facilitating long-term potentiation (LTP). This model therefore emphasizes the loop between the VTA and hippocampus as critical for (1) detection of novel information and (2) encoding novel information into long-term memory.

An alternative model, the hippocampal/neocortical interactions theory of memory formation proposed by Wang and Morris (2010), emphases interactions between the hippocampus and neocortex, rather than the VTA and hippocampus, as critical for memory formation. This model consists of four main components: encoding and storage, cellular consolidation, systems consolidation, and retrieval and reconsolidation. They propose that encoding occurs in the hippocampus formation (HF) and consists of enhancement of activity within cellular (neural) synapses. Cellular consolidation of memories in the HF decay rapidly, but can be augmented if encoding co-occurs with the synthesis, distribution, and capture of new proteins at synapses, which helps to stabilize synaptic changes. Systems consolidation then occurs, wherein the HF establishes memory traces with relevant regions of the cortex. Lastly, during retrieval and reconsolidation, memory traces within the HF are activated and act as a memory index, pointing to regions of the cortex where the memory is fully stored.

The mesolimbic circuit is anatomically connected via dopamine efferent projections originating in the VTA. Dopamine is a neuromodulator with several receptor types (D1−D5), which are largely classified into D1-like (D1, D5) and D2-like (D2, D3, D4). D1-like and D2-like receptors have opposing effects: broadly, they act to increase and decrease, respectively, the responsiveness of neurons they modulate (Pierce & Kumaresan, 2006). Dopamine neurons project from the VTA to receptors in regions throughout the mesolimbic circuit, including the MTL, striatum, amygdala, and prefrontal cortex (PFC) (reviewed by Shohamy & Adcock, 2010). Dopamine is critical for memory formation, as supported by evidence from both the animal and human literature. In the hippocampus, dopamine is a necessary precursor for both LTP − the well-studied cellular model of learning and memory − and the maintenance of long-term memories (Lisman, Grace, & Duzel, 2011; Wang & Morris, 2010). In humans, the effect of dopamine on memory has in part been assessed via pharmacological interventions that act on the dopamine system, including d-amphetamine, methylphenidate, L-dopa, and tolcapone (a COMT inhibitor). On the whole, these studies have shown that dopaminergic agonists improve delayed recall and recognition

(or have produced null effects) (Apud et al., 2006; Eckart, Fuentemilla, Bauch, & Bunzeck, 2014; Linssen, Vuurman, Sambeth, & Riedel, 2011; Murphy, Henry, & Weingartner, 1972; Rammsayer, Rodewald, & Groh, 2000). Taken together, converging evidence across animal models and human studies suggest that dopamine facilitates episodic memory.

More recently, human imaging studies using diffusion tensor imaging (DTI) and fMRI have examined the dopaminergic mesolimbic circuit in humans. Evidence suggests the VTA and neighboring substantia nigra (SN) can be delineated in humans using both DTI (Chowdhury, Lambert, Dolan, & Düzel, 2013; Kwon & Jang, 2014) and resting state functional connectivity (Murty et al., 2014; Tomasi & Volkow, 2014). Furthermore, intrinsic connectivity between human hippocampus, VTA, and ventral striatum has been observed during rest (Kahn & Shohamy, 2013), and, critically, intrinsic connectivity between the hippocampus, vmPFC, and large-scale networks has predicted individual differences in flexible learning in healthy, young adults (Gerraty, Davidow, Wimmer, Kahn, & Shohamy, 2014). In the next section, we will explore how motivation directly impacts this mesolimbic circuit to facilitate memory formation.

MOTIVATION INFLUENCES MEMORY ENCODING

In the past decade, scientists have been increasingly interested in understanding the effects of different types of motivational incentives on memory encoding. The majority of studies have examined the effect of monetary reward motivation on memory encoding. However, there are many other types of nonmonetary motivational incentives, including natural reinforcers (e.g., juice), emotional stimuli (e.g., smiling faces), and leveraging people's innate curiosity to learn something new. Furthermore, outside of reward motivation, there are other means of motivating individuals, including punishment. Lastly, in addition to the type of motivational incentive – reward or punishment – other factors significantly contribute to the effects of motivational incentives on encoding including (1) the physiological response elicited by the incentive and (2) the neurobiological substrates engaged during encoding. In the sections below, we describe the effects of each of these motivational incentives in detail, including their influence on both behavior and the neural substrates engaged during encoding.

Effects of Monetary Reward Motivation on Memory Performance

There is increasing evidence in the literature that monetary reward boosts memory for reward-incentivized stimuli (for review, see Miendlarzewska, Bavelier, & Schwartz, 2016). Some studies have incentivized each item to be remembered (e.g., $2 for remembering an upcoming scene), others have directly associated the items with reward (e.g., all animate objects are rewarded) and finally, some have placed items in a rewarding context (e.g., items shown within a high or low reward state). In an exemplar study, Adcock, Thangavel, Whitfield-Gabrieli, Knutson, and Gabrieli (2006) incentivized items to be remembered with either a high ($5) or low ($0.10) monetary reward, and paid participants their full earnings based on memory accuracy on a recognition test the following day. Similarly, Wolosin, Zeithamova, and Preston (2012) incentivized pairs of items with either high ($2) or low ($0.10) monetary reward and paid participants a percentage of their earnings based on memory accuracy. As expected, in both studies, memory was enhanced for high-rewarded compared with low-rewarded memoranda.

In addition to these results, similar findings have been observed when stimuli are incidentally learned (i.e., participants are unaware that they will take a memory test later), rather than intentionally learned (participants are explicitly told their memory will be tested). For example, Wittman and colleagues (2005) adapted a speeded button press task, such that each cue picture belonged to a category (animate or inanimate). The category predicted the possibility of receiving a reward, contingent on participants' successful button press within the allotted time. Participants therefore both anticipated the opportunity to earn a reward and received feedback indicating whether their response was correct or incorrect (rewarded or unrewarded). Memory in a surprise, subsequent memory test was enhanced for reward-predictive compared with non-reward-predictive items. In a subsequent study in which the stimuli were emotional images (positive, negative, or neutral), incidental memory was better for positive-rewarded images compared with the positive-unrewarded images, with no effect of reward on negative or neutral images (Wittmann, Schiltz, Boehler, & Düzel, 2008).

Mather and Schoeke (2011) observed that in studies manipulating both reward anticipation and reward outcome (Wittmann et al., 2005, 2008), the boost in memory due to reward could be caused by the effects of reward anticipation, reward outcome, or both anticipation and outcome. To clarify these findings, Mather and Schoeke (2011) embedded novel stimuli within a cued reaction time task. Participants first saw a cue that predicted the

potential to earn a monetary reward ($0.25), avoid a monetary loss (−$0.25), or obtain no outcome (+$0). They were instructed to press a button as soon as possible when a novel stimulus appeared in order to earn money, avoid monetary loss, or achieve neither monetary gain or loss (control trials). During a surprise memory test, the authors observed that memory was better for items associated with positive outcomes (both gaining $0.25 and avoiding losing $0.25) compared with negative outcomes, regardless of whether or not the positive outcome resulted in an actual monetary gain. These results suggest that in paradigms where there are both reward anticipation and behavior-contingent outcomes, the observed boost in memory may be due to positive outcomes rather than reward anticipation or reward receipt.

Lastly, the effect of embedding items within a rewarding context has been examined. In one such study, Murty and Adcock (2014) embedded novel items in a stream of repeated items under states of high ($2.00) and low reward ($0.10). In a surprise memory test, memory of the novel items was better in the high reward condition compared to both the low reward condition and compared to chance performance, whereas memory in the low reward condition was no better than chance.

In sum, monetary reward boosts encoding of declarative memory via effects of reward anticipation (Adcock et al., 2006; Wolosin et al., 2012), receipt of positive outcomes (Mather & Schoeke, 2011; Wittmann et al., 2005, 2008), and presentation in a rewarded context (Murty & Adcock, 2014). In the next section, we examine the effects of monetary reward motivation on the underlying neural circuitry that may drive these observed behavioral enhancements.

Effects of Monetary Reward Motivation on Neural Systems Underlying Memory Encoding

Results from neuroimaging studies consistently demonstrate activation of the dopaminergic midbrain: in particular, the VTA and hippocampus have been observed during monetary reward-motivated memory (for review, see Miendlarzewska et al., 2016). Interestingly, not only does the magnitude of activation within these individual regions predict reward enhancements on memory, but, critically, the strength of the correlation between VTA and hippocampus activation predicts the memory benefit due to reward (Adcock et al., 2006). These findings suggest that the interactions between the VTA and hippocampus described in animal models are preserved in humans and support reward-motivated memory.

In addition to subcortical areas, regions of the PFC have been shown to interact with the VTA and hippocampus to promote reward-related memory formation (Cohen, Rissman, Suthana, Castel, & Knowlton, 2014; Murty & Adcock, 2014). The ventrolateral PFC has been shown to be engaged during encoding of high-value words and correlated with memory selectivity for high-valued words (Cohen et al., 2014). Further, correlated activity between the VTA and PFC regions predict reward-related hippocampal signals (Murty & Adcock, 2014). Relatedly, it has been observed that reward information is communicated to subcortical areas (including the VTA and ventral striatum) via the PFC (Ballard et al., 2011), which could act as an executive signal to initiate processes enhancing memory for rewarding events.

Notably, the regions described above are within the mesolimbic network. It is hypothesized that the rewarded memory benefit is due to reward-induced increases in dopamine within the network, which facilitates LTP of memoranda. However, evidence to support this hypothesis is tangential at present and includes primarily genetic indices of individual differences in dopamine. In one study, participants were given L-dopa (the precursor to dopamine) while performing a motivated memory paradigm (Sumner, Duffy, Chen, & Adcock, 2013). The authors observed that individuals with low baseline memory performance, as well as genetic profile scores and personality/symptom questionnaires associated with low dopaminergic tone, benefited the most from L-dopa – specifically, the drug increased memory for high-rewarded scenes in these individuals. However, no memory boost was observed with L-dopa in individuals with high baseline memory performance. Similarly, administration of L-dopa has been associated with better episodic memory performance during incidental encoding (Eckart et al., 2014). These studies emphasize that the impact of dopamine on memory performance varies across individuals depending on a variety of factors including genetics and personality traits. Furthermore, these experiments lay the foundation for future work, including direct measurements of dopamine using PET imaging, to confirm the role of dopamine in motivated memory formation.

Effects of Nonmonetary Reward Motivation on Memory Encoding

While a considerable amount of research has focused on the effects of monetary reward motivation on memory performance, there are other methods of motivating individuals, including using natural reinforcers (e.g., juice), emotional stimuli (e.g., smiling faces), and leveraging people's

intrinsic curiosity in learning something new. Recent work, adapted from the paradigm developed by Adcock et al. (2006) demonstrated that natural rewards (e.g., juice) also boost memory performance. In this study, instead of receiving monetary rewards based on memory accuracy, participants were promised a liquid reward for memory performance (e.g., water, soda, juice). Memory was better for images paired with a liquid reward compared with no liquid reward (Rainey, Dickerson, & Adcock, 2014).

In addition to natural rewards, social cues can also be interpreted as rewarding stimuli. One study examined the effect of facial expression (smiling faces vs. neutral faces) on memory performance for names paired with each face. The authors observed better memory for names paired with smiling faces than neutral faces (Tsukiura & Cabeza, 2008). Activation of the PFC and hippocampus was greater for smiling than for neutral faces during successful encoding and retrieval of face-name pairs. Furthermore, activation across these regions was more strongly correlated for smiling than neutral faces during successful encoding and retrieval. These findings suggest that engagement of reward motivation via a variety of incentives may facilitate episodic memory.

Lastly, recent work has leveraged people's natural curiosity in learning new information to explore how curiosity impacts memory. In a seminal study by Gruber, Gelman, and Ranganath (2014), the authors showed participants a list of trivia questions and asked them to rank each question according to how likely there were to know the answer and how curious they were to know the answer. For each participant, the experimenters subsequently divided the questions into those associated with high curiosity and low curiosity. Participants were then shown each question inside the MRI machine and, while they were waiting for the answer, they were presented with incidental information (a trial-unique, novel face). Participants' memory was better for both the trivia questions and for the faces associated with high curiosity than low curiosity. The authors observed that activation in the midbrain and ventral striatum tracked curiosity, while the hippocampus displayed greater activation for remembered than forgotten trivia questions only during states of high curiosity. These findings suggest that the same neural architecture supporting monetary reward-motivated states is utilized during states of intrinsic motivation, such as natural curiosity.

Limitations on the Benefits of Motivation on Memory Encoding

The above literatures demonstrate that motivation, induced by a variety of incentives, can facilitate hippocampal function and memory encoding.

Motivation, however, does not always result in better memory performance. Rather motivation can either impair or enhance learning depending on the nature of a performance incentive and how an individual perceives it. Outside of the domain of memory, a large literature has documented how incentivizing performance on a task can lead to worse performance in certain contexts, a phenomenon referred to as "choking." For example, research has demonstrated that offering people high rewards, which mimicked a high-stakes situation, resulted in greater errors on a variety of both motor and cognitive tasks (Ariely, Gneezy, Loewenstein, & Mazar, 2009). These deficits were interpreted as resulting from individuals perceiving the high reward state as a stressful opportunity, which in turn yielded states of physiological arousal maladaptive to task performance.

More recently, this concept of "choking" has been demonstrated within the domain of reward-motivated memory encoding. Callan and Schweighofer (2008) investigated whether reward's influence on memory was mediated by individual's anxiety about earning the reward. The authors found that reward motivation only benefited memory performance when individual levels of anxiety were reported as low. Murty, LaBar, Hamilton, and Adcock (2011) utilized a complementary approach to investigate this phenomenon by measuring individual's physiological arousal during reward-motivated memory. Similar to the prior study, the authors found that increased physiological arousal, a putative marker of anxiety, during reward motivation negatively predicted memory performance. Furthermore, participants that showed high arousal responses to reward incentives performed worse on rewarded versus non-reward conditions. These findings suggest that the benefits of reward motivation on memory vary across individuals and may be specific to low arousal/stress contexts.

To more directly test the relationship between aversive behavioral contexts and motivated memory, researchers have begun to investigate encoding in the context of punishment. These lines of research have investigated how negative reinforcers, such as irritating shocks or mildly painful thermal probes, influence encoding. These studies have demonstrated that punishment motivation enhances very simple forms of memory, but impairs more flexible and integrative forms of memory. For example, a study tested differences in reward and punishment motivation on spatial memory in a virtual-reality environment (Murty et al., 2011). Critically, successful performance on this task required the formation of flexible memories, which could integrate disparate features of the surrounding environment. Within this

paradigm, reward motivation enhanced performance, while punishment motivation impaired performance. However, other studies have demonstrated that shock incentives enhance simple forms of memory, such as scene recognition (Murty et al., 2011; Schwarze, Bingel, & Sommer, 2012).

In an elegant study performed by Bauch, Rausch, and Bunzeck (2014), the researchers showed that painful reinforcers enhanced simple forms of memory but impaired more complex forms of memory within the same group of individuals. The authors observed that increasing threat of receiving a painful thermal probe resulted in better familiarity-based memory, which relies on simple item-based representations devoid of any contextual representations, but worse recollection-based memory, which contains details about the relationships amongst multiple features of an episode.

Neuroimaging studies have begun to isolate how different motivational incentives may result in different memory outcomes: namely, by engaging discrete targets in the MTL during encoding. As detailed above, many studies of reward motivation have revealed that memory enhancements are predicted by interactions between the VTA and hippocampus. Critically, these systems are known to support the encoding of integrative representations of memoranda (Davachi, 2006; Ranganath, 2010; Shohamy & Wagner, 2008). Interestingly, hippocampal activation is reduced during states of reward-evoked anxiety (Callan & Schweighofer, 2008) or increasing threat (Bauch et al., 2014; Forkmann et al., 2013). In states of punishment motivation, encoding-related activation has been observed in the amygdala and cortical MTL (Bauch et al., 2014; Murty, Labar, & Adcock, 2012; Schwarze et al., 2012). For example, Murty and colleagues (2012) showed that when individuals are motivated to encode scenes by the threat of shock, successful encoding is predicted by amygdala activation as well as amygdala-cortical MTL connectivity.

The neuroimaging findings dovetail well with the behavioral characterization of punishment motivation: cortical-MTL interactions are thought to support item-based representations in memory, as opposed to richer representations of both item and context supported by hippocampal activity. Thus, the resultant influence of motivation on memory may be determined by a host of factors, including the physiological response of an individual to the incentive as well as the neuromodulatory systems and MTL targets engaged during encoding. Further, negative affect during motivated encoding may shift encoding from hippocampus toward cortical-MTL pathways and lead to more sparse memory representations.

Summary: Motivation and Memory Encoding

In sum, the influence of motivational incentives on memory encoding is complex. Our present knowledge indicates that the impact of motivational incentives on memory encoding depends on three key factors: (1) incentive valence (reward or punishment), (2) the physiological response an individual has to the incentive, and (3) the neural substrates engaged during motivated encoding. Monetary reward motivation generally improves memory outcomes and engages the VTA and hippocampus during motivated encoding. However, this may change depending on an individual's response to the monetary reward incentive. If the reward causes an anxiety/stress response, then individuals tend to "choke" under pressure, perform worse under high incentives, and engage a distinct set of neural substrates – including the amygdala and cortical MTL – during encoding. This response parallels what is observed under punishment motivation, which typically enhances simple memories (e.g., familiarity), but impairs complex memories (e.g., recollection) that are thought to rely on the MTL. Going forward, differential responses to different types of motivational incentives should be accounted for and future work should explore the phenomenon of why identical incentives (e.g., money) may have dramatically distinct effects in different contexts or sub-populations of individuals.

MOTIVATION INFLUENCES MEMORY CONSOLIDATION

While the majority of studies have investigated the role of reward motivation on encoding-related processes (detailed above), motivation further supports memory by facilitating memory consolidation. Consolidation refers to the sequelae of events that occur after encoding that stabilize memory representations. These processes range in scale from cellular processes that strengthen experience-dependent plasticity in newly formed synapses, such as LTP, to broader systems-level processes that distribute memory representations throughout the cortex (Dudai, Karni, & Born, 2015). These processes result in memory representations that are resistant to the natural decay that is typically associated with forgetting. Thus, a hallmark measure of memory consolidation is that memory manipulations affect delayed, but not immediate memory tests, indicating the memory has been consolidated into long-term memory storage. Critically, animal and human research has shown that mesolimbic engagement and reward motivation can facilitate memory

consolidation. In the next sections, we review animal and human literature demonstrating that reward motivation supports the consolidation of information via engagement of the mesolimbic system.

Mesolimbic Engagement and Memory Consolidation

Early support for the facilitation of consolidation via reward motivation emerged from animal work investigating downstream consequences of mesolimbic engagement during encoding. These early studies demonstrated that manipulating mesolimbic activation via drug administration influenced learning on delayed, but not immediate, assays of memory (Wang & Morris, 2010). Early work investigating hippocampal LTP demonstrated that application of a dopamine agonist, which enhances dopaminergic activation, facilitated markers of cellular consolidation, that is, late LTP (Huang & Kandel, 1995). Critically, increased dopaminergic activation did not influence early markers of LTP, suggesting that events following encoding further support memory stabilization.

These findings were further explored in a series of studies investigating memory performance while manipulating dopaminergic modulation during encoding. These studies demonstrated that administering dopamine antagonists (which block dopamine activation) during encoding, impaired performance on delayed tests of memory, but did not influence immediate tests of memory (reviewed by Wang & Morris, 2010). The fact that increasing dopamine activity facilitated consolidation, and decreasing dopamine activity impaired consolidation, provides strong initial support that mesolimbic activation helps to solidify long-term encoded memories, a hallmark of memory consolidation. In sum, dopamine can influence consolidation prior to and after encoding. As a result, the impact of dopamine is at times evident at the level of both encoding and consolidation, solely encoding, or solely consolidation. Future research should aim to elucidate the specific circumstances in which the impact of dopamine is evident at the level of encoding, but not consolidation, and vice versa.

Effects of Reward Motivation on Memory Consolidation

While the findings described above demonstrate a prominent role for mesolimbic activation in memory consolidation, they did not directly test whether reward motivation facilitates learning by acting on consolidation

mechanisms. Rodent studies have recently demonstrated that motivation facilitates consolidation via mesolimbic activation. For example, Salvetti, Morris, and Wang (2014) tested rodents on a spatial navigation task, which was followed by either rewarding or neutral events. Critically, post-encoding introduction of reward enhanced performance on the spatial navigation task at a delayed memory test. Relatedly, previous work showed post-encoding memory enhancements by arousing stimuli (such as reward or novelty) are disrupted by dopamine blockade. These findings provide a critical link between motivation, mesolimbic activation, and facilitation of memory via consolidation.

Recent research in humans has paralleled rodent findings by manipulating reward motivation during encoding and assessing memory at immediate and delayed tests. A seminal study demonstrated that rewarding individuals for learning trivia information had no effect on immediate tests of memory (Murayama & Kuhbandner, 2011). When memory was tested at a 24-hour delay, however, motivation significantly enhanced memory, suggesting that post-encoding consolidation mechanisms support facilitation of memory by reward. Furthermore, studies have shown that rewarding events that take place after encoding enhance motivated memory on delayed, but not immediate, tests (Braun, Vail, Wimmer, & Shohamy, 2014; Murayama & Kitagami, 2014; Patil, Murty, Dunsmoor, Phelps, & Davachi, Under-revision). Finally, a recent human pharmacological study demonstrated that facilitation of memory consolidation is dependent on mesolimbic engagement during consolidation (Feld, Besedovsky, Kaida, Münte, & Born, 2014). Specifically, the authors demonstrated that the delayed memory benefits of reward motivation are abolished when individuals are administered dopamine antagonists post-encoding. These findings suggest that similar mechanisms may be guiding reward-motivated memory consolidation across species.

Effects of Reward Motivation on Neural Mechanisms of Consolidation

Results from both rodent neurophysiology studies and human neuroimaging studies have begun to unpack the mechanisms guiding reward's influence on memory consolidation. These studies have provided evidence that reward motivation promotes systems-level consolidation, a process by which events originally encoded in the hippocampus are "replayed" (meaning the neural traces are reactivated following initial activation during encoding) during post-encoding rest to distribute memories throughout the brain. In animal models, patterns of VTA activation and hippocampal activation that

occur during rewarding events "replay" more often than activation patterns that occur during non-rewarded events (Gomperts, Kloosterman, & Wilson, 2015; Singer & Frank, 2009; Valdés, McNaughton, & Fellous, 2015). The higher rates of "replay" for rewarding events are thought to enhance the consolidation for these events.

Recent human neuroimaging techniques have begun to investigate "replay"-like events by looking at shifts in connectivity or reactivation of stimuli during periods of post-encoding rest (Gruber, Ritchey, Wang, Doss, & Ranganath, 2016; Murty, Tompary, Adcock, & Davachi, 2016). Using these techniques, post-encoding shifts in connectivity between the VTA, hippocampus, and sensory cortex have been shown to predict the benefits of reward motivation on memory. Together rodent and human studies indicate that reward motivation may increase the "replay" of rewarding experience to increase their durability in long-term memory.

Summary: Motivation and Memory Consolidation

In sum, motivation may facilitate memory performance, in part by facilitating memory consolidation and stabilization. Behaviorally, reward motivation has stronger effects on memory tested at delay, with only modest or null effects on memory tested immediately. Pharmacological evidence, in both rodents and humans, has associated reward-motivated consolidation with mesolimbic activation as (1) post-encoding mesolimbic engagement can enhance consolidation and (2) blockade of dopaminergic activation during encoding impedes memory stabilization. One potential mechanism by which reward may be supporting consolidation is by reactivating memories of rewarding events, which results in distribution of memory representations throughout cortex. In future studies, the relationship between reward motivation's influence on encoding and later consolidation should be examined. For example, how do enhancements in VTA-hippocampal connectivity during encoding relate to measures of memory consolidation and/or replay of rewarding events? Finally, the effect of motivation on memory consolidation in domains outside of reward motivation remains to be tested.

MOTIVATED MEMORY: IMPLICATIONS FOR EDUCATION

Thus far, we have reviewed findings across animal and human research investigating the influence of motivation on memory. These studies provide

evidence that motivation promotes engagement of mesolimbic dopamine systems, which results in long-lasting influences on memory. We highlight two mechanisms by which motivation facilitates memory by (1) enriching encoding and (2) bolstering consolidation (see Fig. 2). Together, these literatures provide the foundation for investigations of how basic research can be translated to boost academic achievement. Below, we discuss potential implications for neuroscience-motivated memory research in educational contexts.

In regard to reward-motivated encoding, research demonstrates that reward incentives engage mesolimbic regions to support hippocampus-dependent memory encoding. The traditional focus on incentivizing memory in educational contexts has been on memory retrieval, that is, performance-based testing. The reviewed findings suggest that incentivizing encoding, as opposed to memory testing, can foster states of enriched learning. However, the majority of reviewed studies used monetary rewards to incentivize learning. The implementation of monetary rewards may not be tractable in educational contexts. Emerging research demonstrates that intrinsically rewarding incentives, such as a smiling face or curiosity, can similarly engage mesolimbic circuitry and promote enhanced learning. Capitalizing on these intrinsic incentives to improve learning in the classroom holds great promise to increase educational outcomes.

More recently, other behavioral contexts that facilitate mesolimbic engagement, such as the more active forms of learning and novelty, have also been shown to facilitate mesolimbic-hippocampal interactions during learning (Murty, DuBrow, & Davachi, 2015; Voss, Gonsalves, Federmeier, Tranel, & Cohen, 2011; Wittmann, Bunzeck, Dolan, & Düzel, 2007) and may be readily introduced into the classroom. Future pedagogical research could introduce incentive structures into curriculum to examine if these result in enrichment of student's academic experiences and performance. Particularly, these interventions need to focus on incentivizing learning as opposed to testing.

Critically, neuroscience research has demonstrated that incentivizing learning does not uniformly enhance memory. Instead, different motivational states engage discrete learning systems, which has downstream consequences on stored memories. When incentives are viewed positively, motivation facilitates VTA engagement and encoding in the hippocampus. Critically, hippocampus-dependent memories, which are highly contextualized containing details about multiple features of an experience and the relationships amongst them, may be the most ideal representations to foster in academic settings. These types of memories not only support

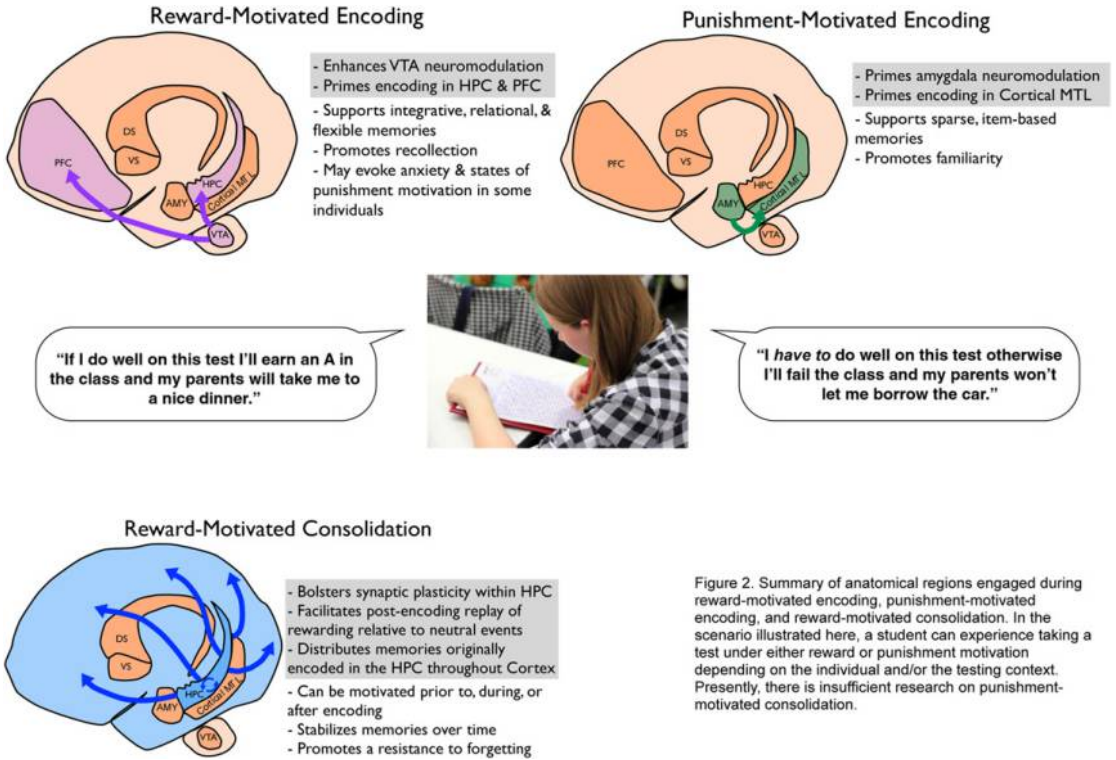

Fig. 2. Summary of Motivation's Influence on Memory Encoding and Consolidation.

vivid, associative memory retrieval (Yonelinas, 2002), but they also track future planning (Schacter, Addis, & Buckner, 2007), imagination and creativity (Buckner, 2010), emergence of conceptual knowledge (Kumaran, Summerfield, Hassabis, & Maguire, 2009), and generalization of learned information into novel domains (Kumaran & McClelland, 2012; Shohamy & Wagner, 2008; Wimmer & Shohamy, 2012).

Importantly, incentives, particularly monetary rewards, can often be viewed as aversive or stressful. In these aversive contexts, motivation fosters engagement of the amygdala and encoding within cortical MTL. These learning states support relatively sparse, inflexible memories. This motivational state may allow a student to perform well on a test of item-based knowledge, but it does not support the broader representations of knowledge that are most amenable to long-term educational goals. Thus, educational settings need to emphasize the fostering of environments by which motivation can best enhance learning: that is, ensuring motivational incentives evoke positive affect as opposed to stress. Whether an incentive induces positive affect or stress has been shown to vary across individuals, which has important implications for classroom settings. Namely, an individually titrated approach to education may be most beneficial for students. Importantly, it cannot be assumed that all incentives will have the same effect on all students. This may increase the burden on educators, but holds promise to ultimately significantly improve individual learning and educational outcomes.

Finally, motivation has been shown to support memory stability by supporting mechanisms of post-encoding consolidation. By engendering both cellular- and systems-level processes, consolidation can transform weakly encoded memories into strong, durable memories. Specifically, these consolidation processes are thought to distribute memories originally encoded in the MTL broadly throughout the cortex, which is proposed to greatly reduce susceptibility to forgetting. Fostering consolidation, thus, offers a means to ensure that information learned within a classroom persists for months to years.

One intervention that could facilitate consolidation in an academic setting is the introduction of positive or rewarding events throughout the course of a school day. In line with this, a recent study in an elementary school showed that introducing a novel learning experience, which theoretically engaged mesolimbic reward systems, resulted in better memory for unrelated information learned throughout the day (Ballarini, Martínez, Díaz Perez, Moncada, & Viola, 2013). Another intervention would be to restructure school days to include periods of rest and/or naps; indeed recent

research shows that individuals have improved performance after sleep on a variety of tasks including memory, vocabulary, and motor imagery learning (Feld & Diekelmann, 2015). These findings are consistent with animal research which shows that mechanisms of consolidation are most prominent during periods of awake rest and sleep (Dudai et al., 2015; Rasch & Born, 2013). By introducing breaks throughout the school day, there may be improved efficacy in the retention of information learned in the classroom.

Neuroscience research has demonstrated that manipulating an individual's motivational state during and after learning is a powerful tool to facilitate memory. A broad array of interventions, centered on engagement of mesolimbic dopamine systems, could be implemented to translate these findings into educational contexts. Similarly, research on motivational constructs from educational psychology could kindle new domains of research for the neuroscience community.

Together, interactive collaborations between both neuroscience and education researchers would yield fruitful avenues of research and opportunities for novel interventions to harness basic science in service of academic achievement. Indeed pioneering work merging these fields has already begun to examine a variety of topics, mainly focused on manipulating retrieval, including the following examples: erroneous information and feedback effects on learning (Dunlosky, Rawson, Marsh, Nathan, & Willingham, 2013; Marsh, Fazio, & Goswick, 2012; Marsh, Lozito, Umanath, Bjork, & Bjork, 2012), how metacognition, feedback, and errors impact learning (Koriat & Goldsmith, 1996), testing effects on learning (for review, see Roediger & Butler, 2011), goal orientation and learning (Grant & Dweck, 2003; Smiley & Dweck, 1994), and how anxiety, stereotype threat, and stress impact learning (for review, see Inzlicht & Schmader, 2012). This initial, elegant work bridging the fields of cognitive neuroscience and education makes it the ideal time to conduct studies examining how research on motivated memory can be leveraged to impact educational outcomes.

ACKNOWLEDGMENTS

We would like to thank Sonali Biswas, Nathan Clement, and Dr. Kimberly Chiew for their comments, discussion, and assistance with making figures. We would also like to thank the following funding agencies for their support: 5F32MH100764-02 to KCD.

REFERENCES

Adcock, R. A., Thangavel, A., Whitfield-Gabrieli, S., Knutson, B., & Gabrieli, J. D. E. (2006). Reward-motivated learning: Mesolimbic activation precedes memory formation. *Neuron*, *50*(3), 507–517. doi:10.1016/j.neuron.2006.03.036

Apud, J. A., Mattay, V., Chen, J., Kolachana, B. S., Callicott, J. H., Rasetti, R., … Weinberger, D. R. (2006). Tolcapone improves cognition and cortical information processing in normal human subjects. *Neuropsychopharmacology*, *32*(5), 1011–1020. doi:10.1038/sj.npp.1301227

Ariely, D., Gneezy, U., Loewenstein, G., & Mazar, N. (2009). Large stakes and big mistakes. *The Review of Economic Studies*, *76*(2), 451–469. doi:10.1111/j.1467-937X.2009.00534.x

Ballard, I. C., Murty, V. P., Carter, R. M., MacInnes, J. J., Huettel, S. A., & Adcock, R. A. (2011). Dorsolateral prefrontal cortex drives mesolimbic dopaminergic regions to initiate motivated behavior. *The Journal of Neuroscience*, *31*(28), 10340–10346.

Ballarini, F., Martínez, M. C., Díaz Perez, M., Moncada, D., & Viola, H. (2013). Memory in elementary school children is improved by an unrelated novel experience. *PLoS ONE*, *8*(6), e66875. doi:10.1371/journal.pone.0066875

Bauch, E. M., Rausch, V. H., & Bunzeck, N. (2014). Pain anticipation recruits the mesolimbic system and differentially modulates subsequent recognition memory. *Human Brain Mapping*, *35*(9), 4594–4606. doi:10.1002/hbm.22497

Bouton, M. E. (2007). *Learning and behavior: A contemporary synthesis* (Vol. 13), Sunderland, MA: Sinauer Associates.

Braun, E., Vail, B., Wimmer, G., & Shohamy, D. (2014). *Both rewards and losses retroactively enhance memory for preceding neutral events*. Annual Meeting for the Society for Neuroscience, Washington, DC.

Buckner, R. L. (2010). The role of the hippocampus in prediction and imagination. *Annual Review of Psychology*, *61*(1), 27–48. doi:10.1146/annurev.psych.60.110707.163508

Callan, D. E., & Schweighofer, N. (2008). Positive and negative modulation of word learning by reward anticipation. *Human Brain Mapping*, *29*(2), 237–249. doi:10.1002/hbm.20383

Chowdhury, R., Lambert, C., Dolan, R. J., & Düzel, E. (2013). Parcellation of the human substantia nigra based on anatomical connectivity to the striatum. *NeuroImage*, *81*, 191–198. doi:10.1016/j.neuroimage.2013.05.043

Cohen, M. S., Rissman, J., Suthana, N. A., Castel, A. D., & Knowlton, B. J. (2014). Value-based modulation of memory encoding involves strategic engagement of fronto-temporal semantic processing regions. *Cognitive, Affective & Behavioral Neuroscience*, *14*(2), 578–592. doi:10.3758/s13415-014-0275-x

Darling-Hammond, L. (1994). Performance-based assessment and educational equity. *Harvard Educational Review*, *64*(1), 5–31. doi:10.17763/haer.64.1.j57n353226536276

Davachi, L. (2006). Item, context and relational episodic encoding in humans. *Current Opinion in Neurobiology*, *16*(6), 693–700. doi:10.1016/j.conb.2006.10.012

Dudai, Y., Karni, A., & Born, J. (2015). The consolidation and transformation of memory. *Neuron*, *88*(1), 20–32. doi:10.1016/j.neuron.2015.09.004

Dunlosky, J., Rawson, K. A., Marsh, E. J., Nathan, M. J., & Willingham, D. T. (2013). Improving students' learning with effective learning techniques: Promising directions from cognitive and educational psychology. *Psychological Science in the Public Interest: A Journal of the American Psychological Society*, *14*(1), 4–58. doi:10.1177/1529100612453266

Eckart, C., Fuentemilla, L., Bauch, E. M., & Bunzeck, N. (2014). Dopaminergic stimulation facilitates working memory and differentially affects prefrontal low theta oscillations. *NeuroImage*, *94*, 185–192. doi:10.1016/j.neuroimage.2014.03.011

Eichenbaum, H., Yonelinas, A. P., & Ranganath, C. (2007). The medial temporal lobe and recognition memory. *Annual Review of Neuroscience*, *30*, 123–152. doi:10.1146/annurev.neuro.30.051606.094328

Feld, G. B., Besedovsky, L., Kaida, K., Münte, T. F., & Born, J. (2014). Dopamine D2-like receptor activation wipes out preferential consolidation of high over low reward memories during human sleep. *Journal of Cognitive Neuroscience*, *26*(10), 2310–2320. doi:10.1162/jocn_a_00629

Feld, G. B., & Diekelmann, S. (2015). Sleep smart-optimizing sleep for declarative learning and memory. *Frontiers in Psychology*, *6*, 622. doi:10.3389/fpsyg.2015.00622

Forkmann, K., Wiech, K., Ritter, C., Sommer, T., Rose, M., & Bingel, U. (2013). Pain-specific modulation of hippocampal activity and functional connectivity during visual encoding. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *33*(6), 2571–2581. doi:10.1523/JNEUROSCI.2994-12.2013

Gerraty, R. T., Davidow, J. Y., Wimmer, G. E., Kahn, I., & Shohamy, D. (2014). Transfer of learning relates to intrinsic connectivity between hippocampus, ventromedial prefrontal cortex, and large-scale networks. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *34*(34), 11297–11303. doi:10.1523/JNEUROSCI.0185-14.2014

Gomperts, S. N., Kloosterman, F., & Wilson, M. A. (2015). VTA neurons coordinate with the hippocampal reactivation of spatial experience. *eLife*, e05360. doi:10.7554/eLife.05360

Grant, H., & Dweck, C. S. (2003). Clarifying achievement goals and their impact. *Journal of Personality and Social Psychology*, *85*(3), 541–553. doi:10.1037/0022-3514.85.3.541

Gruber, M. J., Gelman, B. D., & Ranganath, C. (2014). States of curiosity modulate hippocampus-dependent learning via the dopaminergic circuit. *Neuron*, *84*(2), 486–496. doi:10.1016/j.neuron.2014.08.060

Gruber, M. J., Ritchey, M., Wang, S.-F., Doss, M. K., & Ranganath, C. (2016). Post-learning hippocampal dynamics promote preferential retention of rewarding events. *Neuron*, *89*(5), 1110–1120. doi:10.1016/j.neuron.2016.01.017

Huang, Y. Y., & Kandel, E. R. (1995). D1/D5 receptor agonists induce a protein synthesis-dependent late potentiation in the CA1 region of the hippocampus. *Proceedings of the National Academy of Sciences of the United States of America*, *92*(7), 2446–2450.

Inzlicht, M., & Schmader, T. (2012). *Stereotype threat: Theory, process, and application*. New York, NY: Oxford University Press.

Kahn, I., & Shohamy, D. (2013). Intrinsic connectivity between the hippocampus, nucleus accumbens, and ventral tegmental area in humans. *Hippocampus*, *23*(3), 187–192. doi:10.1002/hipo.22077

Kim, H. (2011). Neural activity that predicts subsequent memory and forgetting: A meta-analysis of 74 fMRI studies. *NeuroImage*, *54*(3), 2446–2461. doi:10.1016/j.neuroimage.2010.09.045

Kluger, A. N., & DeNisi, A. (1996). The effects of feedback interventions on performance: A historical review, a meta-analysis, and a preliminary feedback intervention theory. *Psychological Bulletin*, *119*(2), 254–284. doi:10.1037/0033-2909.119.2.254

Koriat, A., & Goldsmith, M. (1996). Monitoring and control processes in the strategic regulation of memory accuracy. *Psychological Review*, *103*(3), 490–517.

Kumaran, D., & McClelland, J. L. (2012). Generalization through the recurrent interaction of episodic memories: A model of the hippocampal system. *Psychological Review*, *119*(3), 573–616. doi:10.1037/a0028681

Kumaran, D., Summerfield, J. J., Hassabis, D., & Maguire, E. A. (2009). Tracking the emergence of conceptual knowledge during human decision making. *Neuron*, *63*(6), 889–901. doi:10.1016/j.neuron.2009.07.030

Kwon, H. G., & Jang, S. H. (2014). Differences in neural connectivity between the substantia nigra and ventral tegmental area in the human brain. *Frontiers in Human Neuroscience*, *8*, 41. doi:10.3389/fnhum.2014.00041

Linssen, A. M. W., Vuurman, E. F. P. M., Sambeth, A., & Riedel, W. J. (2011). Methylphenidate produces selective enhancement of declarative memory consolidation in healthy volunteers. *Psychopharmacology*, *221*(4), 611–619. doi:10.1007/s00213-011-2605-9

Lisman, J., Grace, A. A., & Duzel, E. (2011). A neoHebbian framework for episodic memory; role of dopamine-dependent late LTP. *Trends in Neurosciences*, *34*(10), 536–547. doi:10.1016/j.tins.2011.07.006

Lisman, J. E., & Grace, A. A. (2005). The hippocampal-VTA loop: Controlling the entry of information into long-term memory. *Neuron*, *46*(5), 703–713. doi:10.1016/j.neuron.2005.05.002

Marsh, E. J., Fazio, L. K., & Goswick, A. E. (2012). Memorial consequences of testing school-aged children. *Memory (Hove, England)*, *20*(8), 899–906. doi:10.1080/09658211.2012.708757

Marsh, E. J., Lozito, J. P., Umanath, S., Bjork, E. L., & Bjork, R. A. (2012). Using verification feedback to correct errors made on a multiple-choice test. *Memory (Hove, England)*, *20*(6), 645–653. doi:10.1080/09658211.2012.684882

Mather, M., & Schoeke, A. (2011). Positive outcomes enhance incidental learning for both younger and older adults. *Frontiers in Neuroscience*, *5*, 129. doi:10.3389/fnins.2011.00129

Miendlarzewska, E. A., Bavelier, D., & Schwartz, S. (2016). Influence of reward motivation on human declarative memory. *Neuroscience & Biobehavioral Reviews*, *61*, 156–176. doi:10.1016/j.neubiorev.2015.11.015

Murayama, K., & Kitagami, S. (2014). Consolidation power of extrinsic rewards: Reward cues enhance long-term memory for irrelevant past events. *Journal of Experimental Psychology: General*, *143*(1), 15–20. doi:10.1037/a0031992

Murayama, K., & Kuhbandner, C. (2011). Money enhances memory consolidation – but only for boring material. *Cognition*, *119*(1), 120–124. doi:10.1016/j.cognition.2011.01.001

Murphy, D. L., Henry, G. M., & Weingartner, H. (1972). Catecholamines and memory: Enhanced verbal learning during l-DOPA administration. *Psychopharmacologia*, *27*(4), 319–326. doi:10.1007/BF00429385

Murty, V. P., & Adcock, R. A. (2014). Enriched encoding: Reward motivation organizes cortical networks for hippocampal detection of unexpected events. *Cerebral Cortex (New York, N.Y.: 1991)*, *24*(8), 2160–2168. doi:10.1093/cercor/bht063

Murty, V. P., DuBrow, S., & Davachi, L. (2015). The simple act of choosing influences declarative memory. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *35*(16), 6255–6264. doi:10.1523/JNEUROSCI.4181-14.2015

Murty, V. P., Labar, K. S., & Adcock, R. A. (2012). Threat of punishment motivates memory encoding via amygdala, not midbrain, interactions with the medial temporal lobe. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *32*(26), 8969–8976. doi:10.1523/JNEUROSCI.0094-12.2012

Murty, V. P., LaBar, K. S., Hamilton, D. A., & Adcock, R. A. (2011). Is all motivation good for learning? Dissociable influences of approach and avoidance motivation in declarative memory. *Learning & Memory (Cold Spring Harbor, N.Y.)*, *18*(11), 712–717. doi:10.1101/lm.023549.111

Murty, V. P., Shermohammed, M., Smith, D. V., Carter, R. M., Huettel, S. A., & Adcock, R. A. (2014). Resting state networks distinguish human ventral tegmental area from substantia nigra. *Neuroimage*, *100*, 580–589.

Murty, V. P., Tompary, A., Adcock, R. A., & Davachi, L. (2016). Selectivity in post-encoding connectivity with high-level visual cortex is associated with rewardmotivated memory. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, In press.

Patil, A., Murty, V. P., Dunsmoor, J. E., Phelps, E. A., & Davachi, L. (Under-revision). Reward retroactively enhances memory consolidation for related items.

Pierce, R. C., & Kumaresan, V. (2006). The mesolimbic dopamine system: The final common pathway for the reinforcing effect of drugs of abuse? *Neuroscience & Biobehavioral Reviews*, *30*(2), 215–238. doi:10.1016/j.neubiorev.2005.04.016

Rainey, C., Dickerson, K. C., & Adcock, R. A. (2014). Interoceptive signals for encoding. Presented at the Annual Meeting for the Society for Neuroscience.

Rammsayer, T. H., Rodewald, S., & Groh, D. (2000). Dopamine-antagonistic, anticholinergic, and GABAergic effects on declarative and procedural memory functions. *Cognitive Brain Research*, *9*(1), 61–71. doi:10.1016/S0926-6410(99)00045-2

Ranganath, C. (2010). A unified framework for the functional organization of the medial temporal lobes and the phenomenology of episodic memory. *Hippocampus*, *20*(11), 1263–1290. doi:10.1002/hipo.20852

Rasch, B., & Born, J. (2013). About sleep's role in memory. *Physiological Reviews*, *93*(2), 681–766. doi:10.1152/physrev.00032.2012

Roediger, H. L., & Butler, A. C. (2011). The critical role of retrieval practice in long-term retention. *Trends in Cognitive Sciences*, *15*(1), 20–27. doi:10.1016/j.tics.2010.09.003

Salvetti, B., Morris, R. G. M., & Wang, S.-H. (2014). The role of rewarding and novel events in facilitating memory persistence in a separate spatial memory task. *Learning & Memory (Cold Spring Harbor, N.Y.)*, *21*(2), 61–72. doi:10.1101/lm.032177.113

Schacter, D. L., Addis, D. R., & Buckner, R. L. (2007). Remembering the past to imagine the future: The prospective brain. *Nature Reviews Neuroscience*, *8*(9), 657–661. doi:10.1038/nrn2213

Schwarze, U., Bingel, U., & Sommer, T. (2012). Event-related nociceptive arousal enhances memory consolidation for neutral scenes. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *32*(4), 1481–1487. doi:10.1523/JNEUROSCI.4497-11.2012

Shohamy, D., & Adcock, R. A. (2010). Dopamine and adaptive memory. *Trends in Cognitive Sciences*, *14*(10), 464–472. doi:10.1016/j.tics.2010.08.002

Shohamy, D., & Turk-Browne, N. B. (2013). Mechanisms for widespread hippocampal involvement in cognition. *Journal of Experimental Psychology. General*, *142*(4), 1159–1170. doi:10.1037/a0034461

Shohamy, D., & Wagner, A. D. (2008). Integrating memories in the human brain: Hippocampal-midbrain encoding of overlapping events. *Neuron*, *60*(2), 378–389. doi:10.1016/j.neuron.2008.09.023

Singer, A. C., & Frank, L. M. (2009). Rewarded outcomes enhance reactivation of experience in the hippocampus. *Neuron*, *64*(6), 910–921. doi:10.1016/j.neuron.2009.11.016

Smiley, P. A., & Dweck, C. S. (1994). Individual differences in achievement goals among young children. *Child Development*, *65*(6), 1723–1743.

Spaniol, J., Davidson, P. S. R., Kim, A. S. N., Han, H., Moscovitch, M., & Grady, C. L. (2009). Event-related fMRI studies of episodic encoding and retrieval: Meta-analyses using activation likelihood estimation. *Neuropsychologia*, *47*(8–9), 1765–1779. doi:10.1016/j.neuropsychologia.2009.02.028

Squire, L. R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychological Review*, *99*(2), 195–231. doi:10.1037/0033-295X.99.2.195

Sumner, E. J., Duffy, K. B., Chen, J. J., & Adcock, R. A. (2013). *Dopaminergic modulation of reward-motivated memory*. Presented at the Annual meeting of the Cognitive Neuroscience Society.

Tomasi, D., & Volkow, N. D. (2014). Functional connectivity of substantia nigra and ventral tegmental area: Maturation during adolescence and effects of ADHD. *Cerebral Cortex (New York, N.Y.: 1991)*, *24*(4), 935–944. doi:10.1093/cercor/bhs382

Tsukiura, T., & Cabeza, R. (2008). Orbitofrontal and hippocampal contributions to memory for face-name associations: The rewarding power of a smile. *Neuropsychologia*, *46*(9), 2310–2319. doi:10.1016/j.neuropsychologia.2008.03.013

Valdés, J. L., McNaughton, B. L., & Fellous, J.-M. (2015). Offline reactivation of experience-dependent neuronal firing patterns in the rat ventral tegmental area. *Journal of Neurophysiology*, *114*(2), 1183–1195. doi:10.1152/jn.00758.2014

Voss, J. L., Gonsalves, B. D., Federmeier, K. D., Tranel, D., & Cohen, N. J. (2011). Hippocampal brain-network coordination during volitional exploratory behavior enhances learning. *Nature Neuroscience*, *14*(1), 115–120. doi:10.1038/nn.2693

Wang, S.-H., & Morris, R. G. M. (2010). Hippocampal-neocortical interactions in memory formation, consolidation, and reconsolidation. *Annual Review of Psychology*, *61*, 49–79, C1–4. doi:10.1146/annurev.psych.093008.100523

Wimmer, G. E., & Shohamy, D. (2012). Preference by association: How memory mechanisms in the hippocampus bias decisions. *Science*, *338*(6104), 270–273. doi:10.1126/science.1223252

Wittmann, B. C., Bunzeck, N., Dolan, R. J., & Düzel, E. (2007). Anticipation of novelty recruits reward system and hippocampus while promoting recollection. *NeuroImage*, *38*(1), 194–202. doi:10.1016/j.neuroimage.2007.06.038

Wittmann, B. C., Schiltz, K., Boehler, C. N., & Düzel, E. (2008). Mesolimbic interaction of emotional valence and reward improves memory formation. *Neuropsychologia*, *46*(4), 1000–1008. doi:10.1016/j.neuropsychologia.2007.11.020

Wittmann, B. C., Schott, B. H., Guderian, S., Frey, J. U., Heinze, H.-J., & Düzel, E. (2005). Reward-related FMRI activation of dopaminergic midbrain is associated with enhanced hippocampus-dependent long-term memory formation. *Neuron*, *45*(3), 459–467. doi:10.1016/j.neuron.2005.01.010

Wolosin, S. M., Zeithamova, D., & Preston, A. R. (2012). Reward modulation of hippocampal subfield activation during successful associative encoding and retrieval. *Journal of Cognitive Neuroscience*, *24*(7), 1532–1547. doi:10.1162/jocn_a_00237

Yonelinas, A. P. (2002). The nature of recollection and familiarity: A review of 30 years of research. *Journal of Memory and Language*, *46*(3), 441–517. doi:10.1006/jmla.2002.2864