

MEMORY CONSOLIDATION, THE DIURNAL RHYTHM OF CORTISOL, AND THE NATURE OF DREAMS: A NEW HYPOTHESIS

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I am not sure where I am, but I'm floating with a friend in what looks like a murky ocean, or perhaps it is a gigantic lake. The water is turbid but extremely deep, and there is no place to touch bottom. Waterlogged sticks and pieces of wood float on the surface, but none look strong enough to support a human's weight. We swim for what seems a frustrated eternity, until we finally find a log that will hold us. We cling to it with our arms while our torsos and legs remain underwater. The water is cleaner "out to sea" but more cluttered with debris "toward the edge." But these terms are meaningless because there is no shore, no horizon, and no sense of space other than the clarity of the water in one direction and the unsettling presence of flotsam and jetsam in the other. I am afraid to swim in the direction of the debris because I don't know what's under it. Color is almost

entirely missing, except for a grayish-blue (under us, above us) and a sickly, perpetually wet brown. Sepia tones that render the dream sinister and sad. . .empty. After days/weeks (?) of paddling, we come to a strange doorway. On the other side float another two people, strangely familiar, perhaps doppelgangers on a different log. They wear no expression, but I think one of them says, "We've been here for 3 years." Panic shoots through me because I suddenly know the place is shoreless. We join forces and start looking for land more earnestly, but there is no land. Not being able to stand up or lie down is torture, and I desperately need to stretch. As this feeling grows more intense, I realize that I will soon go mad. The terror that has been building throughout the dream finally escalates to the point of waking me up.

This dream includes many characteristic features of dreaming (Hobson, 1988), including (1) sensory fragments that get woven into cognitively bizarre themes, (2) irrational content and organization, where the unities of time, place, and person are disjointed and physical laws are disobeyed, (3) the uncritical acceptance of such themes as normal within the dream, and (4) emotion so intense that it is capable of penetrating, changing, or abruptly terminating the dream state. Another feature of dreaming, that dreams are notoriously fleeting and difficult to remember, did not apply here, perhaps because of the highly emotional content in this dream (Goodenough, 1978).

While these characteristics capture the general flavor of dreams, defining precisely what dreams are, where they come from, and what, if any, purpose they serve has proven more difficult. Although there is currently no convincing explanation for why we dream, dreaming is a universal human experience that has been studied for centuries. Until the mid-1900s, however, the study of dreams relied almost exclusively on subjective reports. Not until Aserinsky and Kleitman's (1953) discovery, that dreaming is often associated with Rapid Eye Movement (REM) sleep, did the study of dreaming become more objective. Since this discovery, researchers have been searching for the neurobiological underpinnings of the bizarre, fragmented, and often highly emotional aspects of dreams. Advances in cognitive neuroscience have furthered this goal by providing a unique window on neurocognitive processes, and recent dream studies suggest that at no other time in the diurnal cycle do such large variations in thought and imagery vary so systematically with changes in the central nervous system (Antrobus, 1993; Fosse *et al.*, 2001). It thus seems promising that dreams can be explicated in terms of underlying neurobiological mechanisms (Hobson, 2009; Payne and Nadel, 2004; Wamsley *et al.*, 2010), but the search for such mechanisms is still ongoing.

The most influential contemporary theory of dreaming undoubtedly belongs to Hobson (1988, 2009; Hobson and McCarley, 1977). Hobson's theory, known as the activation-synthesis, and, more recently, AIM (activation, input, modulation) model, is a brain-based account of REM sleep dreaming,

where “activation” refers to the automatic intensification and reduction of brain activity as it progresses through various sleep states. During REM sleep, the activated brainstem generates signals that randomly stimulate the cortex, a process that results in the uniquely bizarre nature of dreams (Hobson, 1988). “Synthesis,” the second piece of Hobson’s theory, refers to the process that weaves these bizarre and discordant dream images into a “best possible fit” narrative by the activated brain. Although Hobson has since expanded his theory (see Hobson, 2009), it has always maintained the above two pivotal ideas: activation, which is provided by the brain stem, and synthesis or integration, which is governed by the forebrain.

I. Memory Consolidation, the Diurnal Rhythm of Cortisol, and the Formal Features of Dreaming: A New Hypothesis

The purpose of this chapter is to put forward a new, brain-based dream hypothesis, one that focuses on the interrelationships among sleep, memory consolidation, and diurnal release of cortisol during late night sleep. Many have argued, as I do here, that dreams are a reflection of the memory consolidation process (e.g., Foulkes, 1985). What has not yet been considered is the influence of the neuroendocrine system and specifically stress hormone release on memories as they are consolidated during sleep (Payne and Nadel, 2004). Cortisol is a stress hormone that has widespread effects on mnemonic and emotional functioning during wakefulness (Payne and Nadel, 2004; Payne *et al.*, 2002, 2004, 2006, 2007; see de Quervain *et al.*, 2009 for recent review). I posit here that neural regions associated with the processing of memories and emotions (i.e., hippocampus, amygdala, regions of prefrontal cortex), via their unique modulation and activation by stress hormones, give rise to the nature of dreams. Specifically, I suggest that (1) memories will be processed differently depending on the diurnal release of cortisol, (2) memories formed under conditions of elevated cortisol share important similarities with dreams, and (3) nightly fluctuations in cortisol secretion not only help explain the formal features of dreams, but may also contribute to the general brain activation necessary for dreaming to reach conscious awareness. In this sense, HPA activation and cortisol secretion late in the sleep cycle may contribute, perhaps causally, not only to our experience of dreams, but also to dream production.

Before expanding upon these ideas, I will first review several lines of evidence. A critical claim of the current account is that sleep participates fundamentally in memory consolidation. Thus, *Section II* will examine the role of sleep in the consolidation of memories. It demonstrates that different types of memory are preferentially consolidated during different stages of sleep,

and argues that this difference may stem in part from nightly fluctuations in the stress hormone cortisol. *Section III* examines the literature on stress hormones and memory during wakefulness to show that, as in sleep, cortisol has disparate effects on memory depending on emotional valence of the experience. *Section IV* argues that the features of memories formed when cortisol is elevated during wakefulness are strikingly similar to the features of late night sleep dreams—a parallel that I argue is produced by cortisol's influence on memories processed during both wakefulness and sleep. Finally, *Section V* explores how these disparate lines of evidence converge on new predictions about dreaming.

II. Sleep and Memory: The Case for Consolidation

A. STAGES OF SLEEP

There are two main types of sleep. The first, rapid eye movement (REM) sleep, occurs in roughly 90-minute cycles and alternates with four additional stages (Stages 1–4) known collectively as NREM sleep, and which comprise the second type of sleep (Aserinsky and Kleitman, 1953). Slow wave sleep (SWS) is the deepest of the NREM phases, and is characterized by high-amplitude, low frequency EEG oscillations. REM sleep, on the other hand, is lighter stage of sleep characterized by eye movements, decreased muscle tone, and low-amplitude, fast electroencephalographic (EEG) oscillations. More than 80% of SWS is concentrated in the first half of the typical 8-hour night, whereas the second half of the night contains roughly twice as much REM sleep than the first half (see Fig. 1). This domination of early sleep by SWS, and of late sleep by REM sleep likely has important functional consequences, but also makes it difficult to know which distinction is critical: NREM sleep versus REM sleep or early versus late sleep.

Neurotransmitters, particularly the monoamines (serotonin, 5-HT, and norepinephrine, NE) and acetylcholine, play a critical role in switching the brain from one sleep stage to another. REM sleep occurs when activity in the aminergic system has decreased enough to allow the reticular system to escape its inhibitory influence (Hobson, 1988). The release from aminergic inhibition stimulates cholinergic reticular neurons in the brainstem and switches the sleeping brain into the highly active REM state, in which acetylcholine levels are as high as in the waking state. REM sleep is also associated with higher levels of cortisol than NREM overall, especially late in the sleep cycle (Lavie, 1996; Wagner and Born, 2008; see also Fig. 3). 5-HT and NE, on the other hand, are virtually absent

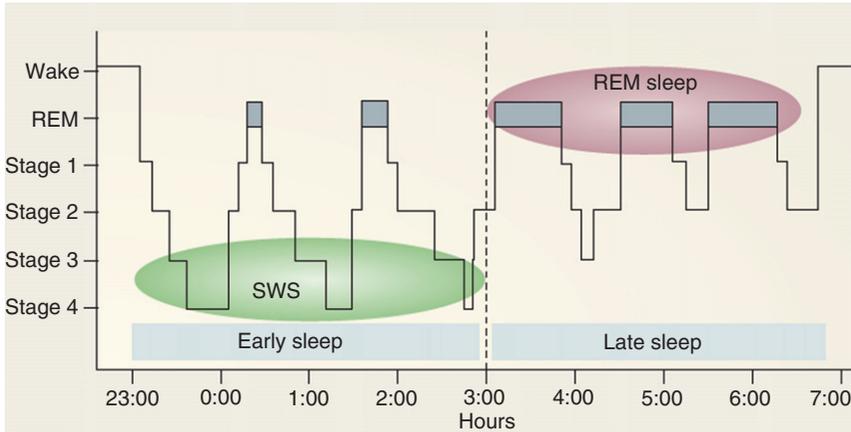


FIG. 1. Sleep histogram showing the distribution of SWS and REM sleep.

during REM. SWS, conversely, is associated with an absence of acetylcholine and low levels of cortisol but nearly normal levels of 5-HT and NE (Hobson *et al.*, 2000).

B. STAGES OF MEMORY FORMATION AND DIFFERENT MEMORY TYPES

Memory consolidation is the process by which newly acquired information, initially fragile, is integrated and stabilized into long-term memory (McGaugh, 2000). Evidence overwhelmingly suggests that sleep plays a role in the consolidation of a range of memory tasks, with the different stages of sleep selectively benefiting the consolidation of different types of memory (Diekelmann and Born, 2010; Payne *et al.*, 2008; Smith, 1995; Stickgold, 2005; Walker, 2009).

Most taxonomies break memory down into key types (Schacter and Tulving, 1994; Squire and Zola-Morgan, 1991), and several are important for our purposes here. First, there are various types of memory that we can recall explicitly, including *episodic* memories, or memories of the events in our lives, and *semantic* memories, which consist of knowledge (e.g., facts, word meanings) that has been uncoupled from place and time (Tulving, 1983). Unlike retrieving a semantic memory, retrieving an episodic memory from one's past requires access to defining contextual features of the event, such as specific details about the place of its occurrence. Because of this emphasis on space and context, *episodic*

memories and *spatial* memories are closely connected. Second, there are “how to” memories for the various skills, procedures, and habits we acquire through experience. Because these memories are not so easily made explicit and are usually only evident in behavior, they are referred to as *procedural* or *implicit* memories. Third, there are *emotional memories* for the positive and negative experiences in our lives. This class of memories is mediated by a system that is particularly concerned with learning about fearful and negative stimuli, although evidence suggests it plays a role in memory for pleasant information as well (e.g., Hamann, 2001).

Each of these memory types is subserved by distinct neural systems (Schacter and Tulving, 1994). While episodic and spatial memories are governed by the hippocampus and surrounding medial temporal areas, procedural or implicit memories are thought to be independent of the hippocampus and anatomically related regions, relying instead on various neocortical and subcortical structures (Schacter and Tulving, 1994; Squire and Zola-Morgan, 1991). The emotional memory system is centered in the amygdala, a limbic structure that is richly connected to the hippocampus.

C. SLEEP'S ROLE IN EPISODIC MEMORY CONSOLIDATION

Memories are processed during most, if not all, stages of sleep, and the neurochemical milieu of the brain has important consequences for memory consolidation. Although there is substantial evidence that sleep benefits the consolidation of procedural memories (see Smith, 1995, 2001; Walker and Stickgold, 2006 for excellent reviews), the evidence that sleep is critical for explicit episodic and emotional memories is most relevant to the current hypothesis on dreams.

There is a general consensus that NREM sleep, especially SWS, is essential for the consolidation of hippocampus-dependent episodic and spatial memories, whereas REM sleep is more important for procedural and emotional memory consolidation (see Ellenbogen *et al.*, 2006; Marshall and Born, 2007; Payne *et al.*, 2008 for review). In a landmark study, Plihal and Born (1997) capitalized on the unequal distribution of SWS and REM sleep to assess the recall of word pairs (an episodic memory task) and improvement in mirror-tracing (a procedural memory task) after retention intervals of early sleep (the first 3–4 hours of the sleep cycle), and late sleep (the last 3–4 hours of the sleep cycle).

Recall of word pairs improved significantly more after a 3-hour sleep period rich in SWS than after a 3-hour sleep period rich in REM or a 3-hour period of wakefulness. Mirror tracing, on the other hand, improved significantly more after

a 3-hour sleep period rich in REM than after 3 hours spent either in SWS or awake. These findings dovetail nicely with older studies of sleep and episodic memory, which showed that sleep rich in SWS produced less forgetting of episodic memory materials than sleep rich in REM (Barrett and Ekstrand, 1972; Fowler *et al.*, 1973; Yaroush and Sullivan, 1971).

Similarly, using a nap paradigm, Tucker *et al.* (2006) found that naps containing only NREM sleep enhanced memory for word pairs, but did not benefit mirror tracing. Following an afternoon training session, performance on these tasks was assessed after a 6 hour delay, either with or without an intervening nap. Not only did the nap subjects recall more word pairs than the subjects who remained awake, but they also showed a weak correlation between improved recall and the amount of SWS in the nap.

These results are consistent with neurophysiological evidence derived from electrophysiological studies in rodents, which demonstrate that patterns of hippocampal place cell activity first seen during waking exploration are later re-expressed during post-learning SWS (Wilson and McNaughton, 1994; Ji and Wilson, 2007 reviewed in O'Neil *et al.*, 2010). Consistent with these findings is a PET study in humans (Peigneux *et al.* 2004), which demonstrated that learning-related hippocampal activity seen while training on a virtual maze task was again expressed during post-learning SWS, and importantly, this hippocampal reactivation during sleep strongly predicted overnight improvement on the task. Similarly, Rasch *et al.* (2007) exposed human subjects to an odor while they were learning object-location pairings in a task similar to the memory game "concentration"; subjects who were re-exposed to the odor during SWS (but not REM sleep) showed enhanced hippocampal activity and enhanced memory for the memory pairings. Likewise, in some of the strongest evidence for to date, Marshall *et al.* (2006) showed that hippocampus-dependent memories are specifically enhanced when slow oscillations (slow, <1 Hz oscillatory activity during SWS) are induced during sleep by transcranial electrical stimulation. These observations suggest that learning triggers the reactivation and reorganization of memory traces during NREM SWS, a systems-level process that in turn enhances behavioral performance.

D. SLEEP'S ROLE IN EMOTIONAL MEMORY CONSOLIDATION

Emotional memories, which rely critically on the amygdala for their consolidation, appear to benefit most from REM sleep (see Payne and Kensinger, Current Directions in Psychological Science for review). Wagner *et al.* (2001) found that 3 hours of late night, REM-rich sleep (but not 3 hours of early

night slow-wave rich sleep or 3 hours of wakefulness) facilitated memory for negative arousing narratives, an effect that could still be observed years later when the subjects were re-contacted for a follow-up memory test (Wagner *et al.*, 2006). Consistent with these findings, the amygdala and hippocampus are among the most active brain regions during REM sleep, with some evidence suggesting that they are more active during REM sleep than during wakefulness (Maquet *et al.* 1996). This suggests that emotional memory processing may be a primary function of REM sleep. Moreover, several studies have correlated features of REM sleep, including oscillatory activity in the theta frequency band range (Nishida *et al.*, 2009), with enhanced emotional memory consolidation (see Walker, 2009 for review). These findings strongly suggest a role for sleep, especially REM sleep, in the processing of memory for emotional experiences.

E. SLEEP TRANSFORMS MEMORIES IN USEFUL WAYS

The findings reviewed above provide compelling evidence that sleep plays an important role solidifying experience into long-term memory in a veridical manner, more or less true to its form at initial encoding. However, it has long been known that memories *change* with the passage of time (Bartlett, 1932), suggesting that the process of consolidation does not always yield exact representations of past experiences. On the face of it, this may seem strikingly maladaptive, yet such flexibility in memory representation allows the emergence of key cognitive abilities, such as generalization and inference (Ellenbogen *et al.*, 2007), future thought (Schacter *et al.*, 2008), and the selective preservation of useful information extracted from a barrage of incoming stimulation and experience (Payne *et al.*, 2009). Consistent with these ideas, growing evidence suggests that sleep does more than simply consolidate memories in veridical form; it also transforms them in ways rendering memories less accurate in some respects, but more useful and adaptive in the long run. Sleep leads to flexible restructuring of memory traces so that insights can be made (Wagner *et al.*, 2004), inferences can be drawn (Ellenbogen *et al.*, 2007), and integration and abstraction can occur (Payne *et al.*, 2009). In each of these cases, sleep confers a flexibility to memory that may be at times more advantageous than a literal representation of experience.

As a specific example of such qualitative changes in memory representation, recent studies demonstrate that sleep transforms the emotional memory trace. Payne *et al.* (2008) examined how the different *components* of complex negative arousing memories change across periods of sleep versus wakefulness. Emotional scenes could be stored as intact units, suffering some forgetting over time but retaining the same relative vividness for all components. Alternatively, the

components of an experience could undergo differential memory processing, perhaps with a selective emphasis on what is most salient and worthy of remembering.

Participants viewed scenes depicting negative or neutral objects embedded on neutral backgrounds at 9 am or 9 pm (see Fig. 2 for e.g., stimuli). Twelve hours later, after a day spent awake or a night including at least 6 hours of sleep, they were tested on their memory for objects and backgrounds separately to examine how these individual components of emotional memories change across periods of sleep and wake. Daytime wakefulness led to forgetting of negative arousing scenes in their entirety, with both objects and backgrounds suffering forgetting at similar rates. Sleep, however, led to a selective preservation of negative objects, but not their accompanying backgrounds, suggesting that the two components undergo differential processing during sleep. This finding suggests that, rather than preserving intact representations of scenes, the sleeping brain effectively “unbinds” scenes to consolidate only their most emotionally salient, and perhaps adaptive, emotional element (see Payne *et al.*, 2009; Wagner *et al.*, 2004 for additional examples of unbinding during sleep).

Paralleling these behavioral findings, an fMRI study provided evidence that a single night of sleep is sufficient to provoke changes in the emotional memory circuitry, leading to increased activity within the amygdala and the ventromedial prefrontal cortex, and resulting in strengthened connectivity between the amygdala and both the hippocampus and the ventromedial prefrontal cortex (Payne

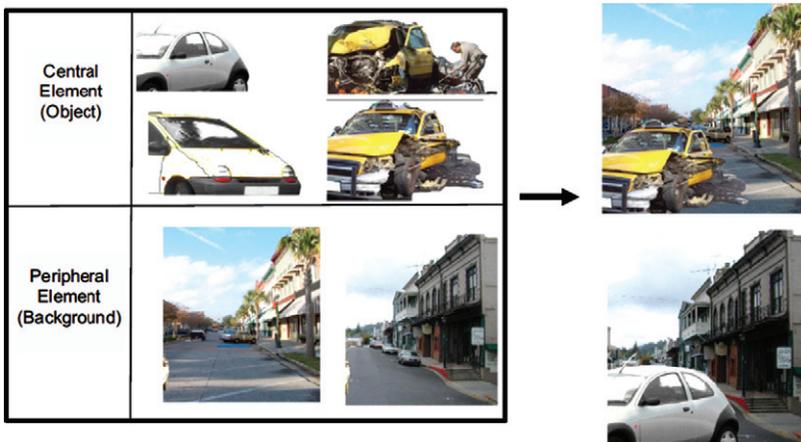


FIG. 2. Examples of the scenes presented to subjects. Eight versions of each scene were created by combining each of four similar objects (two neutral objects, two negative emotional objects) with each of two plausible neutral backgrounds. In this example, the two neutral central objects are cars, and the two negative central objects are cars badly damaged in an accident; the neutral backgrounds are street scenes. Two of the eight versions of the completed scene are shown.

and Kensinger, in press, *Journal of Cognitive Neuroscience*). These findings are consistent with a study by Sterpenich *et al.* (2009) and suggest that sleep strengthens the modulatory effect of the amygdala on other regions of the emotional memory network as memories undergo consolidation (McGaugh, 2004).

F. NEUROPHYSIOLOGICAL AND NEUROCHEMICAL EVIDENCE FOR SLEEP'S ROLE IN MEMORY CONSOLIDATION

Each of the sleep stages is characterized by a unique collection of electrophysiological, neurotransmitter, and neuroendocrine properties that tend to overlap with the different sleep stages, but are not perfectly correlated with them. For example, SWS is associated with cortical slow oscillations (slow, <1 Hz oscillatory activity during SWS), sleep spindles (faster, 11–16 Hz, bursts of coherent brain activity, and hippocampal sharp-wave “ripple” complexes (~200 Hz)—all of which have been associated with episodic memory consolidation. Indeed, the co-occurrence of these electrophysiological events may underlie the coordinated information flow back and forth between hippocampus and neocortex as memories are integrated within neocortical long-term storage sites (e.g., Buzsaki, 1996, 1998).

There is also evidence to suggest that nocturnal changes in neurotransmitter and neurohormone levels contribute to memory consolidation. Acetylcholine, norepinephrine, serotonin, and cortisol all play important roles both in modulating sleep (Hobson *et al.*, 2000) and in memory function (Cahill and McGaugh, 1998; Hasselmo, 1999; Payne *et al.*, 2004). Cortisol, for instance, follows a marked circadian rhythm where it is at its nadir during early night, slow-wave rich sleep and reaches its zenith during late night, REM-rich sleep. Indeed, the difference between the cortisol level in the blood at sleep onset and at awakening is so great that the interpretation of cortisol blood levels is meaningless without knowing exactly when the sample was taken (Lavie, 1996; Weitzman *et al.*, 1971). Moreover, the secretion of cortisol is not continuous but comprised of gradually increasing peaks that tend to coincide with REM sleep episodes (see Fig. 3). REM sleep thus tends to co-occur with cortisol elevations (Lavie, 1996; Wagner and Born, 2008).

Interestingly, the early night reduction in acetylcholine and cortisol may be necessary for hippocampus-dependent memories to undergo effective consolidation, as experimentally elevating either substance during early sleep impairs performance on episodic memory tasks. Gais and Born (2004) trained subjects on word pair task and mirror tracing tasks before 3 hours of nocturnal sleep or wakefulness during which they received a placebo or an infusion of the cholinesterase inhibitor physostigmine (which increases cholinergic tone). When tested after 3 hours of early sleep rich in SWS, recall on the paired associates task was

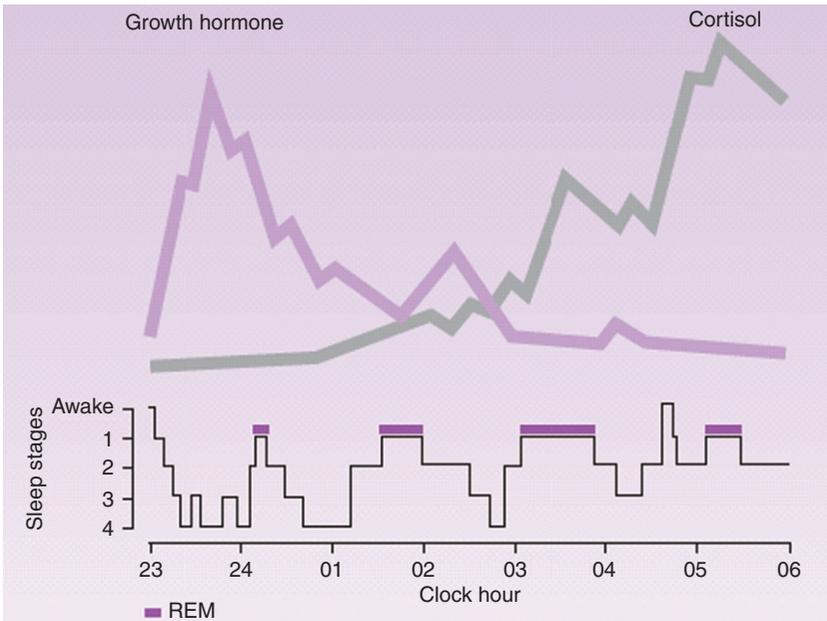


FIG. 3. The relationship between sleep-stage architecture and circulating levels of growth hormone and cortisol. Note both the linear increase in cortisol across the night and also the cortisol peaks riding on top of REM periods.

markedly impaired in the physostigmine group, while procedural memory performance was unaffected. Using a similar design, [Plihal and Born \(1999\)](#) showed that when cortisol was infused into the early, SWS-rich interval, retention of episodic information that is normally facilitated during this time was impaired. Thus, enhancing plasma cortisol concentrations during early sleep eradicated the benefit typically observed for episodic memory while leaving procedural memory unimpaired (see [Fig. 4](#)).

[Plihal and Born \(1999\)](#) concluded that as episodic, but not procedural, memory relies on hippocampal function, cortisol inhibition during early nocturnal sleep is necessary for episodic memory consolidation.¹ Thus, because cortisol release is inhibited during early night periods dense in SWS, this time window may provide the ideal physiological environment for episodic memory consolidation. REM sleep, on the other hand, is an inefficient time to consolidate episodes, due to the deleterious effect of elevated cortisol on hippocampus-dependent memory processing. Thus, the neurobiological properties of early sleep and late

¹This study also specifies glucocorticoid receptors, as opposed to mineralocorticoid receptors, as being responsible for the observed effects.

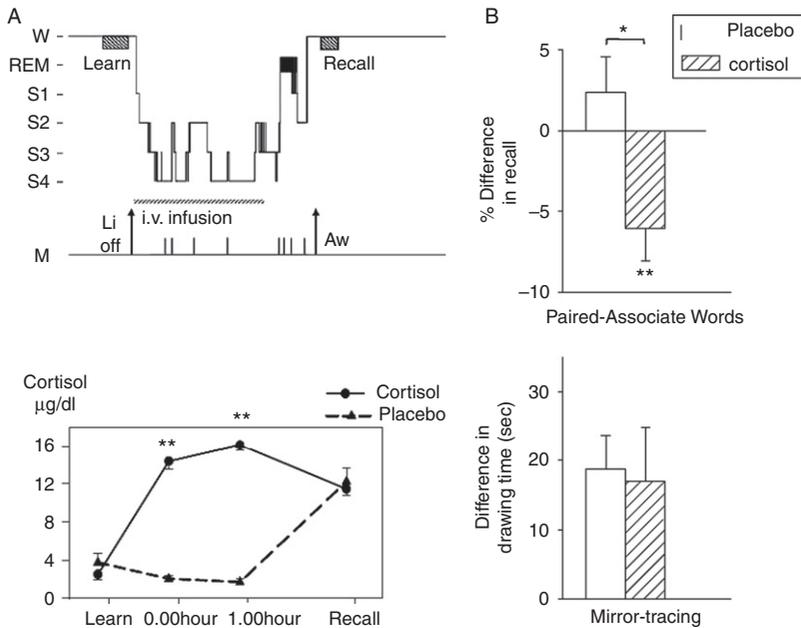


FIG. 4. (A) Top: Experimental design for examining the impact of cortisol on memory consolidation during a 3 hour period of early nocturnal sleep rich in SWS, illustrated by an individual sleep profile. Before sleep, subjects learned episodic and procedural memory tasks to a criterion. Recall was tested 15–30 minutes after awakening. Infusion of cortisol or placebo began at 11 pm and was discontinued after 2.5 hours. Bottom: Mean plasma cortisol concentrations during Placebo (dotted line) and Cortisol (solid line) conditions. (B) Cortisol infusion during sleep, compared with placebo, impaired hippocampus-dependent episodic memories for word pairs across sleep (top), but did not affect hippocampus-independent procedural memory (speed in mirror tracing, bottom). $N=14$, $*p < .05$, $**p < .01$. Data from Plihal and Born (1999).

sleep, as opposed to SWS and REM sleep *per se*, may be essential for the consolidation of different types of memory.

In line with the above findings, cortisol elevations during wakefulness can also impair performance on episodic memory (de Quervain *et al.*, 2009). Interestingly, cortisol in the Plihal and Born (1999) study was elevated just enough to mimic the late night peak of circadian cortisol activity (15.2 ± 0.68 mg/dl); this amount is proportionate to the cortisol typically released in response to a mild to moderate stressor ($\sim 10\text{--}30$ mg/dl) and is a sufficient dose to disrupt episodic memory function during wakefulness (e.g., Kirschbaum *et al.*, 1996; Wagner and Born, 2008), particularly when administered at retrieval (de Quervain *et al.*, 2000). Thus, cortisol elevations seen during late night REM may help explain both why replay of episodic memories in REM sleep dreaming is so scarce (Baylor and

Cavallero, 2001; Fosse *et al.*, 2003) and why dreams are difficult to remember (Hobson, 1988).

III. Cortisol's Impact on Memory During Wakefulness

There is a substantial literature demonstrating that stress can produce cortisol elevations capable of altering memory function in animals and humans (de Quervain *et al.*, 2009; Kim and Diamond, 2002; Lupien *et al.*, 2009; Payne *et al.*, 2004; Roozendaal *et al.*, 2009). The hippocampus, amygdala, and memory relevant regions of the prefrontal cortex all have dense concentrations of receptors for cortisol. Elevated cortisol can impair the neuronal structure and function of the hippocampus² by altering hippocampal morphology, disrupting neurogenesis, and blocking the synaptic plasticity (e.g., long-term potentiation “LTP” and primed-burst potentiation “PBP”) thought to underlie memory formation (McEwen, 2000). Inducing stress in the laboratory by exposing subjects to a brief, one-time stressor (e.g., a public speaking task, or the cold pressor task, which involves submerging the arm in cold water), or administering glucocorticoids directly, typically leads to impairments in episodic memory (e.g., Kirschbaum *et al.*, 1996; Payne *et al.*, 2002, 2006, 2007), particularly when cortisol is administered at retrieval (de Quervain *et al.*, 2000). As might be expected, it also disrupts spatial memory (Laurance *et al.*, unpublished data). Thus, cortisol elevations, including those consistent with increases seen during late night sleep, are capable of disrupting episodic memory function.

However, recent studies suggest that cortisol facilitates consolidation of emotional relative to neutral episodic memories (Payne *et al.*, 2007), and even emotional relative to neutral features within a complex episode (Payne *et al.*, 2006). This is similar to the previously discussed sleep and episodic memory finding by Wagner *et al.* (2001), showing that emotionally laden episodic memories were *facilitated* relative to neutral memories during late night, REM-rich sleep. Indeed, substantial evidence in animals and humans demonstrates that cortisol can selectively impair hippocampus-dependent neutral memories, while leaving emotional memories intact (Payne *et al.*, 2006) or even enhancing them (Buchanan and Lovallo, 2001; de Quervain *et al.*, 2009; Jelicic *et al.*, 2004; Payne *et al.*, 2007).

Paralleling these functional effects, animal research demonstrates that hippocampal structural plasticity suffers under high levels of cortisol, while amygdala

² It is important to note that extremely low levels of cortisol can disrupt hippocampal function as well. It thus appears that a moderate level of circulating glucocorticoids enhances memory whereas maximal or minimal levels disrupt it, perhaps explaining why some studies of stress and memory fail to find memory disruption.

plasticity is enhanced (Vyas *et al.*, 2002). Human neuroimaging studies likewise show that cortisol elevations impair hippocampal activity, while facilitating activity in the amygdala. For example, Pruessner *et al.* (2008) showed that acute stress induced prior to an encoding task caused significant deactivation in the hippocampus, and degree of hippocampal deactivation was significantly correlated with the cortisol stress response. van Stegeren *et al.* (2007), on the other hand, showed that elevated cortisol levels correlated with intensified amygdala activation at encoding and better memory for emotional information later on. Together, these findings provide a basis for the claim that stress hormone modulation of hippocampal activity underlies the damaging effects of stress on neutral episodic memory, while their modulation of the amygdala underlies the enhancing effects of stress on emotional events (see Payne *et al.*, 2004).

Cortisol's opposing effects on neutral and emotional memories are mainly observed in studies examining the memory consolidation phase specifically. For example, direct cortisol administration before or after encoding selectively enhances memory for emotionally arousing, but not neutral material (Buchanan and Lovallo, 2001; Cahill *et al.*, 2003; Okuda *et al.* 2004; Payne *et al.*, 2007). Moreover, cold pressor stress enhances memory for emotional slides but does not affect memory for neutral slides. Consistent with these findings, endogenous cortisol levels at encoding correlate with enhanced memory consolidation only in individuals who were emotionally aroused. Furthermore, Payne *et al.* (2007) showed that psychosocial stress (elicited by a public speaking task called the Trier Social Stress Test, or TSST), which elevates cortisol (Fig. 5, right), enhances memory for an emotionally arousing story while impairing memory for a closely matched neutral story (see Fig. 5, left). Interactions between cortisol and amygdala activity are likely key to determining this selectivity (de Quervain *et al.*, 2009).

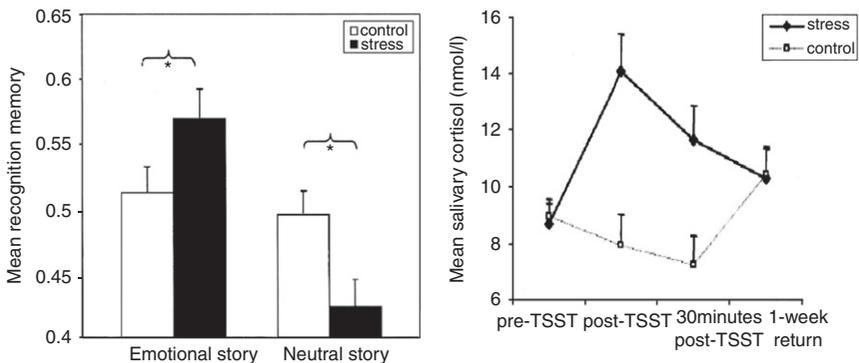


FIG. 5. Stress enhances the consolidation of negative emotional information, but disrupts the consolidation of neutral information (left). The Trier Social Stress Test (TSST), a social stressor involving public speaking, increases cortisol levels compared to a control condition (right). From Payne *et al.* (2007).

IV. A Clinical View of Memory Under Stress

Before connecting these studies of cortisol and waking memory with the realm of sleep and dreams, we will next examine the clinical characteristics of memories formed under stressful or traumatic conditions. A discussion of stress and memory from a clinical viewpoint serves a twofold purpose: It deepens our understanding of the cortisol-based memory deficits reviewed above, and serves as a bridge between the waking relationship between cortisol and memory and the relationship cortisol might have to sleep and dreaming. A close look at these clinical characteristics reveals memories that share much in common with late-sleep dreams; they are often bizarre, fragmented, lacking in spatial and temporal context, highly emotional, and difficult to remember.

A. CORTISOL, THE HIPPOCAMPUS, AND FRAGMENTED MEMORIES OF TRAUMATIC EXPERIENCE

Retrieving an episodic memory requires one to access and integrate multiple fragments of experience stored in disparate memory systems—what happened, who engaged in which actions, what it all looked like, sounded like, smelled like, etc. In addition to this episodic “content,” there is also spatial-contextual information. Because all events by definition occur *someplace*, it can be difficult if not impossible to divorce memories of episodes from memories of context, a notion that led us to propose that context serves as an organizing frame to which the various elements of an episodic memory trace are attached (Nadel and Payne, 2002).

Consider a hypothetical traumatic war experience: a memory of this experience might include smells (the jungle, unwashed bodies), sounds (gunfire and explosions), sights (the flash of a sniper’s weapon, the sight of a seriously wounded friend), tactile feelings (the humidity, pain from a wound), actions (diving for cover, returning fire), and emotions (fear, anger, guilt). Each of these independent features is stored in the relevant part of the brain, typically, but not exclusively, in the neocortex. The hippocampus and adjacent medial temporal regions are critical for binding these disparate fragments of an episode from multiple brain regions into a unified memory trace (e.g., Cohen and Eichenbaum, 1994; Schacter and Tulving, 1994). Regions of the prefrontal cortex may also share, with the hippocampus, responsibilities for binding in episodic memory (e.g., Mitchell *et al.*, 2000).

The idea that memories are disaggregated during storage and then re-aggregated during retrieval has several implications for understanding interactions

between stress and memory (Jacobs and Nadel, 1998). By disrupting the hippocampus and prefrontal cortical-based contextual representation system, stress may impair memory for time, space, and other contextual information. By disrupting the hippocampus and prefrontal cortical-based binding function, stress may leave the various aspects of a memory trace disconnected. Thus, memories formed under high levels of stress and cortisol may be disjointed, fragmented, and lacking in detail and spatial or temporal context, thus creating specific deficits in episodic memory.

Indeed, clinical reports suggest that memory for stressful experiences lack coherence, context, and episodic detail and can be highly fragmented (Bremner, 1999; Murray *et al.*, 2002; van der Kolk, 1991, 1997, 1998; van der Kolk and Fisler, 1995; Verfaellie and Vasterling, 2009). In a clinical sense, the term “fragmented” means that the various bits and pieces of experience are not related to one another as a whole, and that the various features of memory fail to be bound together to produce a “good gestalt,” or a coherent episode (Jacobs and Nadel, 1998; Payne *et al.*, 2004; van der Kolk, 1997, 1998). For example, memory fragmentation is an important feature of post-traumatic stress disorder (PTSD), in which patients describe gaps in their memories, not only of the trauma, but of other personal experiences as well (Bremner, 1999).

Rather than simply retrieving fragments and reporting them as such, some individuals make educated guesses about memory in a process we have called narrative smoothing (Jacobs and Nadel, 1998; Payne *et al.*, 2004). Constructing narratives is, to an extent, a normal function of human memory. The hippocampal system, rather than creating inflexible representations that serve as permanent records of events, stores representations from which episodic memories are recreated from the various attributes or features of the event (Cohen and Eichenbaum, 2004). In this way, memory is fundamentally reconstructive. But in the presence of high levels of stress hormones, this normal process can be taken to an extreme. In both clinical settings and the laboratory, recombining these fragments in an attempt to make sense of one’s experience can lead to inconsistencies in one’s recall or even blatantly “false” memories (e.g., Murray *et al.*, 2002; Payne *et al.*, 2002, 2004, 2006).

B. CORTISOL, THE AMYGDALA, AND ENHANCED MEMORY FOR EMOTIONAL ASPECTS OF TRAUMATIC EVENTS

The amygdala is highly active during emotionally charged, stressful experiences and the memories dependent on this region are highly resistant to forgetting (LeDoux, 2002). This “stamping in” of emotional memories is at least partly mediated by stress hormones such as cortisol (and norepinephrine—see McGaugh, 2000, 2004 for review) and is another core feature of PTSD. Hormones released during times of stress thus have opposing effects on different

neural structures, and consequently opposing effects on the types of memories subserved by these structures. This is highlighted in the following quote from a Vietnam veteran suffering from PTSD:

Parts of my memory are very vivid, especially the emotional parts. At other times, there are these weird holes. I can't make the memories make sense together like in a story. I don't know how to say it. It's like my memories [for the event] are fragments. . . puzzle pieces, but I can't figure out how to put the puzzle together.

In the absence of an intact hippocampus-based memory system, the amygdala-based system is left to store emotional information that is not bound to the spatio-temporal context within which the relevant events occurred. This results in a pool of isolated memory fragments, many of which are emotional, that have been encoded without a coherent spatio-temporal frame to organize them (Jacobs and Nadel, 1998). Thus, the stress encountered during traumatic events does not lead to a complete eradication of memory. Rather, it leads to the storage of fragments that lack a spatial/contextual framework to bind and define them as belonging to an individual episode. This binding failure leads to one of two outcomes. As in the case of the veteran above, the memory fragments surface as disconnected images, feelings, or sensations (referred to as “body memories” by Van der Kolk, 1991), or they elicit narrative smoothing, where retrieved memory fragments are cobbled together by a narrative based upon gist, inference and educated guesswork, often guided by preserved emotional information (Jacobs and Nadel, 1998; Nadel and Jacobs, 1998; Payne *et al.*, 2004). Although these narratives can be logical and similar to the real experience, more often they are bizarre and distorted. Indeed, we have previously argued that schematization of disconnected features underlies many cases of false memory reported in the media and the scientific literature (e.g., Jacobs and Nadel, 1998; Nadel and Payne, 2002b; Payne *et al.*, 2004).

The above account offers a neurobiological explanation for (1) why memories laid down under high levels of stress can be fragmented or forgotten, (2) why preserved fragments are often emotional and remembered exceptionally well, and (3) how memories can emerge as coherent, if distorted and bizarre, emotionally guided reconstructions of personal experience. If high levels of cortisol can influence memory processing in this manner during wakefulness, it follows that similar influences may be at work during sleep. I suggest that as memories undergo sleep-based consolidation, the presence of elevated cortisol during late-night sleep may contribute to the subjective characteristics of dreaming. As readers will no doubt have noted, dream features are highly similar to the features of waking memories that were formed in the presence of high levels of stress.

V. Tying It All Together: Toward a New Hypothesis of Dreaming

It is generally assumed that long-term memory consolidation involves interactions among multiple brain systems, modulated by various neurotransmitters and neurohormones. I propose that the characteristics of dreams are best understood in the context of this neuromodulatory impact on brain systems. Although a number of neurotransmitters and neurohormones are likely involved, I argue that the relationship between late night elevations in cortisol and explicit memory consolidation have important consequences for dreams: it produces fragmented dreams, gives dreams their uniquely bizarre flavor, accounts for their emotional nature, and explains not only why veridical replay of episodic memories during dreaming is rare, but also why dreams are so fleeting and difficult to remember. While many researchers have argued, as I argue here, that sleep is important for the consolidation of memories, I suggest that memory consolidation proceeds differently depending on changes in the neurochemical milieu of the sleeping brain. This, in turn, means that our experience of dreams should differ depending on when they occur.

A. CORTISOL AND THE FORMAL FEATURES OF DREAMING

1. *Dreams are Fragmented*

Neurochemical properties, rather than sleep stages *per se*, determine how memories are processed, and the degree to which they are experienced as coherent “units” as opposed to fragments. As in wakefulness, cortisol elevations during late night sleep disrupt the ability of the hippocampus and PFC to contribute contextual information to a memory and bind its elements into a coherent whole. This leads to activation of memory fragments in the absence of contribution from the structures that normally contextualize and connect them. It is these memory fragments, which are stored in dispersed neocortical regions, that compose the disconnected sounds and images and bizarre plot lines that constitute dreams. When we become conscious of this altered memory processing, we experience a typical REM sleep dream.

Although dreams can be extremely fragmented, they are not experienced as completely random sequences of associated images. Rather, they exhibit varying degrees of thematic coherence (Cipolli, 1995; Foulkes, 1985). The cortisol notion of dreaming accounts for narratization in dreams in the same way it accounts for narratization in wakefulness. When the sleeping brain is confronted with fragmented information, it automatically attempts to synthesize them into narrative themes.

The neurohormonal milieu of during late night sleep can explain why we experience memories in our dreams as far removed from the waking episodes upon which they are based, rather than re-experiencing actual episodes (Baylor and Cavellero, 2001). This idea also helps resolve a paradox in dream research—that memory function is profoundly impaired in dreams and yet memories must be processed during sleep because dreams consist of memory fragments (Hobson *et al.*, 2000). Indeed, Hobson and colleagues have argued that a deficiency of memory in dreaming goes “a long way” toward explaining the characteristics of dreaming (Hobson *et al.*, 2000). The cortisol account explains how memory processing under specific conditions gives rise to dream characteristics. Memories are processed during sleep and give rise to dreams, and are also profoundly altered in sleep periods most associated with dreaming. By the cortisol account, these things are not mutually exclusive.

Not all dreams with these features have to be REM dreams. While such dreams may be most prevalent during REM sleep, NREM dreams occur more frequently than was once believed (Nielsen, 2000). Foulkes (1985) and Solms (2000) have both argued against a simple REM sleep = dreaming perspective, and are proponents of NREM dreaming. By simply changing the question asked of awakened subjects from “Did you dream?” to “Did you experience any mental content?,” Foulkes was able to show a far higher percentage of dream reports from NREM stages than original studies had suggested (e.g., Aserinsky and Kleitman, 1953). These dream reports after NREM awakenings led Foulkes and others to conclude that the stream of consciousness never ceases during sleep and that the brain engages in cognitive activity during all sleep stages (also see Antrobus, 1991, 1993).

Some NREM dreams are similar in content to REM dreams, and the majority of these come from NREM periods occurring early in the morning (Cicogna *et al.*, 1998; Kondo *et al.*, 1989), during the peak phase of the diurnal rhythm when cortisol levels are at their zenith. Interestingly, Antrobus has demonstrated that dreaming is associated with two independent sources of neural activation. The first, as Hobson has consistently shown, is associated with the REM–NREM cycling that is governed by the brainstem. The second is associated with the rising morning phase of the diurnal rhythm. This is the period during which cortisol levels are at their highest, and one tantalizing but speculative possibility is that cortisol contributes directly to neural activation as the sleep cycle nears its end. Kondo *et al.* (1989) have shown that the two sources of activation are additive, so that late REM episodes are associated with more vividness of visual imagery, more bizarreness, and more discontinuities of person, place and time than are earlier REM episodes (Antrobus, 1991). There is also evidence that NREM dreaming not only increases but that NREM dream reports become more vivid and disjointed during the early morning hours (Antrobus, 1991; Cicogna *et al.*, 1998). In fact, most of the characteristic features of dreaming intensify across the night in both REM and NREM sleep (Wamsley *et al.*, 2007).

Note that these are precisely the qualities of dreaming we would expect to intensify as cortisol levels become elevated in the morning. I argue that a simple increase in cortical arousal alone would be unlikely to produce the bizarre and fragmented features of dreaming—if anything, an activated brain (i.e., during wakefulness) is thought to process information much more smoothly and effectively. Thus, it is much more likely that escalating cortisol, as it takes its toll on ongoing memory processes, helps shape the features of our dreams and perhaps contributes to general cortical arousal as well. [Antrobus \(1991\)](#) notes that although dream features (such as fragmentation and bizarreness) are regarded as the most salient characteristics of dreaming, they are not particularly prominent in laboratory REM reports *unless they are obtained in the late morning hours* ([Kondo et al., 1989](#)). Given this, it seems likely that the rising morning phase of the diurnal rhythm, which is associated with elevated cortisol, contributes seminally to many of the hallmark features of dreams.

2. *Sensory Dream Fragments Produce Bizarre Cognitive Themes*

Though fragmentation and bizarreness are sometimes treated as separate features of dreaming, the cortisol hypothesis sees them as intimately intertwined. The recombination of activated memory fragments into unusual narratives is what produces the experience of bizarreness. Why, however, are some dreams particularly fragmented and bizarre whereas others are less so? By the current view, memory processing that occurs when cortisol is elevated should lead to dreams that are more fragmented and/or bizarre, while memory consolidation occurring when cortisol levels are low should be less so. For this reason, waking up earlier in the night (before cortisol levels are at their peak) should result in dreams that are fairly straightforward, whereas waking up later should result in dreams that are prototypically bizarre.

In line with this, some evidence suggests that dreams during these earlier periods look more like actual episodic memories. NREM and SWS dreams contain more mentation and replay of actual episodic experience than REM dreams ([Foulkes, 1962](#)), and NREM dreams are typically less bizarre than REM dreams. Thus, early night NREM dreams are typically associated with coherent episode memories, while REM dreams are associated with a mixture of episodic, personal semantic, and semantic fragments that combine to produce bizarre plot lines (e.g., [Cavallero et al., 1992](#); [Cicogna et al., 1991](#)). Comparisons between REM and NREM dreams later in the night, however, reveal that many of these differences disappear and NREM and REM dreams begin to look similar. This finding fits with the notion that NREM dreams begin to resemble REM dreams as cortisol levels rise late in the night, becoming increasingly more fragmented and bizarre.

[Antrobus \(1991, 1993\)](#), [Foulkes \(1985\)](#), and [Hobson \(1988, 2009\)](#) have proposed related explanations of bizarreness. For example, much like the current

proposal, [Foulkes \(1985\)](#) views dreaming as a process of semantic and episodic memory activation. He believes this activation proceeds in a largely diffuse and arbitrary fashion, but that dreams nonetheless consist of predictable features derived from memory. Dream content thus loosely reflects the recent or remote past of the dreamer, not as a simple replay of past events but rather as a plausible variation on such events. Similarly, [Hobson](#) ascribes narratization to forebrain structures including the hippocampus, amygdala, and medial prefrontal cortex, and, like the current account, attributes bizarreness to the unnatural fitting together of dream fragments.

[Schwartz and Maquet \(2002\)](#) also have an interesting account of dream bizarreness, although they focus solely on two aspects of bizarre dreams. They point to similarities between dream reports and neuropsychological syndromes in which faces and places are misidentified. Fregoli syndrome, for instance, involves the delusional misidentification of faces, or hyperidentification of faces, where an unknown face is recognized as familiar, and typically develops after damage to frontal and temporal regions. [Schwartz and Maquet \(2002\)](#) suggest that Fregoli-like delusions in dreams imply neuronal processes during sleep that simultaneously engage unimodal visual regions that produce facial percepts (e.g., the fusiform gyrus), and distinct multimodal associative areas in the temporal lobe that allow retrieval of facial identity—areas that are clearly active during REM sleep. Corresponding deactivations in dorsal frontal regions would dampen supervisory control functions that would normally signal a mismatch between facial appearance and facial identity. This, they argue, produces the delusive quality of Fregoli-like representations in dreams ([Schwartz and Maquet, 2002](#)).

Misidentifications during dreams also extend to places. Familiar places in dreams often share little if anything in common with real places in waking life. For example, a dream that takes place in one's old house may look nothing like the place where one used to live. These authors attribute misidentified places to the parahippocampal cortex (which is adjacent to the fusiform face area). They note that passive viewing of spatial scenes and layouts activates this region during wake, and thus suggest that it is likely involved in the “reduplicative paramnesia” for places seen in dreams and in patients with combined damage to temporal and prefrontal areas—a disorder that likely results from faulty integration of a particular place with semantic information about place identity ([Schwartz and Maquet, 2002](#)).

Both of these phenomena can be considered binding deficits, and can be understood as the consequence of disrupted hippocampal, parahippocampal, and prefrontal (and perhaps fusiform given its location in the temporal lobe and proximity to the parahippocampal region) processing under conditions of elevated cortisol. As mentioned, these regions are needed to integrate dispersed features into a coherent memory or percept, and this binding occurs at various levels. It involves the ability to bind a particular face with a particular identity, and also the ability to bind the retrieved place or person representation with the

appropriate time, situation, and so on. Both processes are clearly altered in dreaming, as they are in amnesic patients with damage to the hippocampus and surrounding areas of the medial temporal lobe (and sometimes PFC regions as well).

As one example, amnesics make a substantial number of “memory conjunction errors,” which are produced in paradigms used to test mnemonic binding (Kroll *et al.*, 1996; Reinitz *et al.*, 1992, 1996). Memory conjunction errors occur when components of previously presented information are inappropriately recombined into episodes that never occurred, or into stimuli that were never presented. For example, subjects are shown facial stimuli, which are later recombined to make new faces, or words, which are later recombined into new words. For instance, subjects might be shown “SPANIEL” and “VARNISH” and then asked if they recognize the unrepresented conjunction word “SPANISH,” or shown two faces and then asked if they recognize a new face combining features of both.

While healthy subjects make only a moderate number of conjunction errors, they jump dramatically when such stimuli are shown to amnesic patients. Amnesics have particular difficulty conjoining features into an internal representation of the correct configuration that defines a memory trace. Thus, they falsely recognize recombinations of previously presented features, even though they can correctly discriminate them from new items that were not previously studied. This deficit is not surprising, considering that amnesics have damage to areas so critical for efficient binding (e.g., Eichenbaum and Bunsey, 1995; Reinitz *et al.*, 1996). To the extent that hippocampal processing is altered by elevations in cortisol, as in late night sleep, misidentifications and recombinations would be expected. Indeed, the fact that memory fragments are activated in the absence of hippocampus and PFC-provided context virtually insures that such misidentifications and recombinations will occur in dreams.

By the current view then, dream fragmentation and bizarreness result from intensified HPA activity, particularly late in the sleep cycle. Episodic memories become activated in an increasingly unbound or disconnected manner as cortisol levels rise. High levels of cortisol render the patterns of reactivation incomplete, which may produce the fragmented quality of dreams. As in waking studies of stress hormones and memory, the disconnected or “unbound” fragments are woven into the bizarre storylines that constitute dreams. Narrative smoothing may begin during sleep, as the brain attempts to impose meaning on fragmented memory traces activated in circuits that include the hippocampus and amygdala, but it likely intensifies upon awakening as we impose additional structure and logic upon remembered fragments and themes. This is where we find ourselves reasoning through our dreams, thinking things like, “I’m not sure where I was, but it must have been Tucson because it looked like the desert,” or “I assume I was with John although it didn’t look like him.” Our attempts to synthesize and impose narrative structure on the fragments produce the bizarreness of dreams.

Of course, on some occasions narratization fails or simply isn't complete, and in these cases we are left with severely fragmented dreams and with gaps in our morning recall. If we recall none of these fragments or rudimentary themes, we say that we did not dream.

VI. The Emotional Nature of Dreams

We are biased to consolidate emotional information during both wakefulness and sleep (Buchanan and Loyallo, 2001; Payne *et al.*, 2006, 2007, 2008; Wagner *et al.*, 2001), ensuring that we remember information and events that have adaptive value. The stress response system has evolved to ensure that we remember emotionally salient information, in some cases indefinitely (e.g., LeDoux, 2002). Stress hormones, including cortisol and norepinephrine, are thought to modulate the amygdala/hippocampus interactions that allow such memories to be stored in long-lasting format (Buchanan and Loyallo, 2001; McGaugh, 2000). Because high levels of cortisol facilitate amygdala function but impair hippocampal and PFC function, emotional memories are enhanced at the expense of other (neutral) aspects of memory (e.g., Buchanan and Loyallo, 2001; Jacobs and Nadel, 1998; LeDoux, 2002; Payne *et al.*, 2006).

Similar to the study by Payne *et al.*, 2007, (see Fig. 5), Buchanan and Loyallo (2001) have demonstrated that a low dose of cortisol (20 mg) administered during wakefulness enhances highly negative (high emotion) relative to neutral (low emotion) pictures compared to placebo (see Fig. 6).

Interestingly, these data are similar to the sleep data of Wagner *et al.* (2001), which demonstrate that emotional memory formation is selectively enhanced

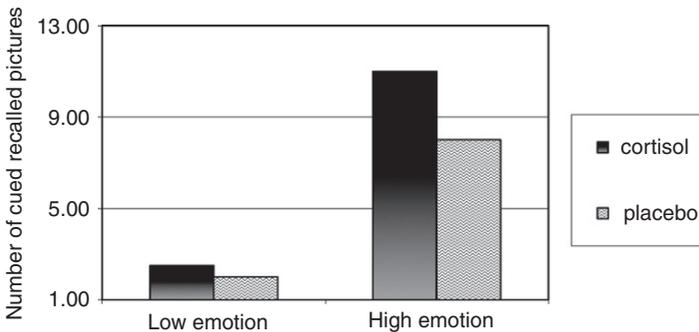


FIG. 6. Cortisol administration enhances the consolidation of highly emotional pictures, but not neutral (low-emotion) pictures. From Buchanan and Loyallo (2001).

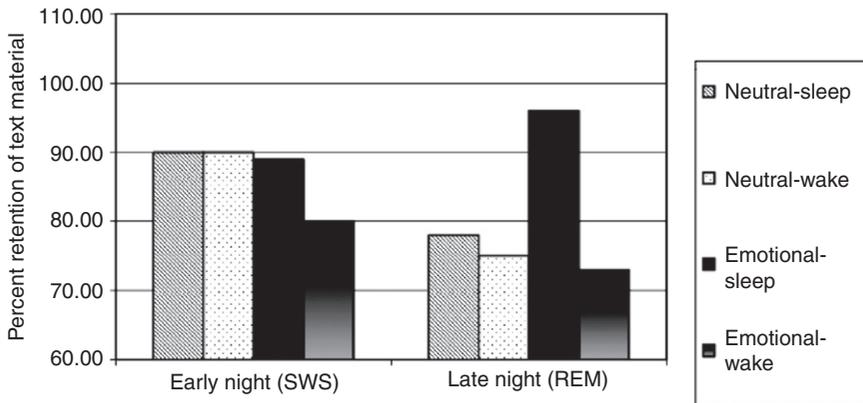


FIG. 7. Emotional information is selectively enhanced by late night, REM-rich sleep (black bar, right), but not by early night sleep rich in SWS. From Wagner *et al.* (2001).

after REM-rich sleep periods late in the night when cortisol activity is at a maximum (see Fig. 7).

It is illustrative to note the similarities between the above two figures. During both wakefulness and sleep, emotional memory is enhanced when cortisol levels are elevated. Thus, cortisol activity may directly contribute to both the emotional memory enhancement seen in late night, REM-rich sleep, and to the emotionality of REM sleep dreams (Casagrande *et al.*, 1996; Foulkes, 1962).

Many theorists have pointed out that dreams are often emotional. They are frequently biased toward negative emotions, although positive emotions, such as sexual pleasure and elation are also featured (Hartmann and Basile, 2003; Merritt *et al.*, 1994). The amygdala plays a role in all of these emotions, although research suggests that it may be preferentially involved in negative emotions like fear and anxiety (LeDoux, 2002), which may be the most common dream emotions. Dreams also incorporate instinctual programs (Revonsuo, 2000), such as the stress response system's "fight or flight" programs (McEwen, 2007), which, like emotion, can act as powerful synthesizers of dream fragments. Thus, via its effects on amygdala activation and associated selective preservation of emotional memory, cortisol might inspire an emotionally guided narratization process. Newell and Cartwright (2000) argue that emotion is a primary shaper of dream narratives. Fragments may be synthesized according to one's emotional state, and within an emotional framework provided by preserved emotional memory themes. Emotion might continue to inspire a search for meaning and personal salience upon awakening, as emotional themes connect with waking agendas to influence dream reconstruction. In this sense, dreams may well have some "meaning."

A. ACCEPTANCE OF BIZARRE IMAGES AS COMMONPLACE WITHIN THE DREAM

When dreaming, we often accept bizarre and even outlandish themes as completely normal, never questioning them until we awaken (except in the case of lucid dreaming, see LaBerge *et al.*, 1981; LaBerge, 1990). Hobson and others have speculated that selective inactivation of the frontal cortices during REM sleep may be responsible for this phenomenon (Maquet, 2001). Together with evidence that the frontal lobes contribute to reality monitoring (Johnson, 1991; Schnider, 2001) and executive functions (Smith and Jonides, 1999) during wake, these massive frontal deactivations likely contribute to the intellectual complacency exhibited in dreams. Reality monitoring refers to our ability to compare dream or other fictitious events with waking reality and deem them “not real” (Johnson *et al.*, 1984). Interestingly, damaged frontal lobes are associated with failures in reality monitoring and also bizarre confabulations that often resemble dreams (Kopelman, 1987; Schnider, 2001; Schnider and Ptak, 1999; Shallice, 1999).

The frontal lobes have received increased attention in the stress literature (e.g., Lupien and LePage, 2001). Both Type I (mineralocorticoid, “MR”) and Type II (glucocorticoid, GR) cortisol receptors are present in cortical regions, with a preferential and dense distribution in the prefrontal cortex. Hence, stress may exert effects not only on hippocampal neurons, but also on neurons critical for the normal function of the PFC. Given evidence that both hippocampus and PFC are components of a memory circuit underlying memory retrieval (e.g., Fletcher *et al.*, 1998) and the binding of memory elements (e.g., Mitchell *et al.*, 2000), this would mean that both parts of the binding system are affected by cortisol. Thus, given the high concentrations of cortisol receptors in PFC regions, late night cortisol impairment of both hippocampal and prefrontal cortical function may contribute to the binding deficits and impaired reality monitoring seen in dreams.

B. DREAMS ARE DIFFICULT TO RECALL AND EPISODIC MEMORY “REPLAY” IS RARE IN DREAMS

Dreams are notoriously difficult to recall (e.g., Goodenough, 1978). Hobson asserts that the difficulty of dream recall is a thorny issue, but one that any dream theory must explain in the face of the robust activation of memory regions in REM sleep (Hobson *et al.*, 2000). According to Hobson, dream amnesia is largely attributable to the aminergic demodulation of REM sleep (Hobson, 1988; Hobson *et al.*, 2000).

Because elevated cortisol disrupts memory retrieval (de Quervain *et al.*, 2000), it seems reasonable to assume that it plays a role in dream amnesia upon awakening,

impairing recall of dreams in the same way it impairs recall during wake (see *Section III*). This is not the only memory impairment concerning dreams, however, as there is evidence for memory deficits within dreams themselves. Although a widely held belief is that dreams incorporate events from our waking lives, this has not been borne out in the laboratory. Complete events are rarely re-experienced in dreams, with recent estimates suggesting that only 1–2% of dream reports accurately reflect waking-life experience (Fosse *et al.*, 2003). Yet in spite of this, individual features (or fragments) of events are incorporated into dreams, although these often get woven into scenarios bearing little resemblance to real waking events (e.g., Baylor and Cavallero, 2001).

Interestingly, while the phenomenological experience of episodic memory processing during dreams is only loosely related to the original experience, activation of isolated features of a memory may be enough to boost memory consolidation. Recently we examined the influence of post-learning dream content on improved performance on a spatial navigation task (Wamsley *et al.*, 2010). We found that dreaming of the maze task did indeed benefit post-sleep performance, even though the dream reports never consisted of exact, veridical replays of the original learning experience. Instead, while subjects' dream reports were unquestionably related to the maze, they primarily consisted of remote memories and themes connected to the task, or of isolated fragments and thoughts of the maze-navigation experience.

This lack of exact replay mirrors that observed in animal studies of neuronal-level reactivation (e.g., Ji and Wilson, 2007; Wilson and McNaughton, 1994) and in human brain imaging studies (e.g., Pigneux *et al.*, 2004) where the reactivation observed is not precisely identical to that seen during encoding. For example, although patterns of neural reactivation seen in rodent sleep statistically resemble the activity of these networks during prior waking task performance, the activity patterns are never identical to those observed during prior wake, more typically containing only fragments of waking experience. These findings fit with the fact that complete episodes are not typically replayed in dreams. Elevated cortisol is likely to produce precisely these unbound fragments of episodic memories in the phenomenological experience of dreams.

C. PREDICTIONS

The ideas presented above are quite testable and lead to several interesting predictions. Cortisol administration during early sleep should modify the nature of dreams via its impact on memory processing; specifically, it should (1) intensify dream fragmentation and bizarreness relative to the administration of a placebo

and (2) reduce the differences between early and late night REM sleep dreams (such that they would be equally fragmented and bizarre). The assumption here is that cortisol levels account for the differences between dreams from early versus late REM sleep episodes. As such, dreams reported from late REM sleep periods (when cortisol is at its peak) should be more bizarre and more fragmented than early REM periods (when cortisol is low). There is already evidence that REM dreams are generally more bizarre than NREM dreams (Antrobus *et al.*, 1995; Casagrande *et al.*, 1996; Foulkes, 1962), a finding that might be informed by cortisol elevations during late-night sleep that is largely composed of REM sleep. However, early and late REM episodes have not, to my knowledge, been directly compared on measures of fragmentation and bizarreness. Cortisol administration during early sleep should also (3) increase the emotionality of dreams, both during REM and NREM sleep stages and (4) if administered during early NREM sleep specifically, reduce the likelihood of thinking about or re-experiencing recent episodes.

D. CAVEATS

Several of the neurotransmitters that fluctuate across the sleep cycle are also known to affect memory function during wakefulness (e.g., acetylcholine, see Hasselmo, 1999; norepinephrine, see Cahill and McGaugh, 1998), and there has been much speculation about their influence on memory processing during sleep (Hobson *et al.*, 2000; Solmes, 2000). Moreover, these neurotransmitters likely interact with cortisol during sleep. For example, acetylcholine and cortisol may interact to modulate dreams. Also, because the PFC is important for memory function generally, its deactivation during REM sleep (Braun *et al.*, 1997; Maquet *et al.*, 1996) likely contributes directly to impaired memory processing during late night sleep. Thus, although I have suggested that diurnal elevation in cortisol may help explain the nature of dreams, I do not mean to suggest that cortisol is the only factor affecting their structure and content.

VII. Concluding Remarks

In sum, converging evidence suggests that cortisol's impact on the hippocampus, amygdala, and prefrontal cortex can account for many formal features of dreaming, including dream fragmentation and bizarreness, the emotional nature

of dreams, the lack of episodic memory replay in dreams, and the tendency for dreams to be forgotten upon awakening. Memory consolidation, a process that includes activation and reprocessing of memory traces, as well as their integration with pre-existing knowledge and experience, appears to play a critical role in dreaming: We dream when we become aware of these activated memory traces, and we experience them as fantastical as they are woven into confabulatory, bizarre, but also enormously creative, story lines during late night REM sleep.

An interesting question is whether the process of weaving unbound fragments into creative narratives is functional. Obviously, when high levels of stress impact memory during wakefulness, fragmentation and the resulting reconstruction may be truly disruptive, as when reconstruction produces memories that are flagrantly “false” (Schacter *et al.*, 2003). Many people think of such memory inaccuracies as strictly negative events that lead to false recollections at best and devastating confabulations at worst (Loftus, 1996). While it is true that accurate recall is adaptive in many cases and imperative to our survival in a few (e.g., accurately remembering that a certain mushroom is poisonous), there may be a positive side to the process that disrupts memory binding and produces fragmentation, especially during the protected state of sleep.

When the bonds attaching the components of a memory are broken, this information can be flexibly recombined. In other words, memories are “unbound,” which in turn allows novel configurations of knowledge and events. This has already been suggested in studies of sleep and memory showing that the sleeping brain goes beyond simple memory solidification to actually transform memories in ways that are useful and adaptive for future behavior. Such transformation, whether it be selectively consolidating emotional foreground information at the expense of neutral information in the background (Payne *et al.*, 2008), or suppressing a well-learned problem-solving strategy to allow insight into a novel shortcut (Wagner *et al.*, 2004), requires unbinding, as rigid representations do not allow for the flexible recombinations necessary for these effects. In wakefulness these recombinations may lead us to misremember episodes, but during sleep, they may take us down different paths to creative insights and novel ideas, and allow us to role-play and test possible future scenarios before we ever encounter them. Although each of these ideas awaits experimental confirmation, cortisol-induced fragmentation during dreams might be ultimately beneficial for human cognition.

In conclusion, I argue that dreams are a reflection of the memory consolidation process, which serves not only to strengthen the neural traces of recent events and integrate them with older memories and previously stored knowledge, but also to recombine and restructure features of experience. All new ideas are based upon previously stored information, and nearly every definition of creativity includes combining this information in novel and useful ways. Unbinding the individual features of a memory trace may be essential for these processes to

occur, and cortisol's impact on the dreaming brain may be the ideal time to safely make new connections and to test out new ideas for the future.

*Dreams are but interludes which fancy makes
When monarch reason sleeps, this mimic wakes.
Compounds a medley of disjointed things
A mob of cobblers and a court of Kängs.*

—John Dryden (1700)

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