Biological functions of dreaming

Stefan Lemke

Cellular and Molecular Neuroscience

St. Olaf College, Northfield, MN

April 2011
Research on dreaming, has shown light upon a subject which remains mostly unknown. In fact, there is not a universally accepted biological definition of dreaming. Recently, several researchers have attempted to form a link between dreams to memory, suggesting that dreaming is a method for the brain to consolidate memories.

This relationship was explored in a study performed by Cipolli, Fagioli, Mazzetti, and Tuozzi. Dream's effect on memory consolidation was tested by examining whether memory consolidation would be enhanced for characteristics of an experience which are repeatedly processed and transformed into identical or very similar contents of distinct dreams developed over the same night. Subjects were awakened during their first four periods of REM sleep (determined by an electropolygraph), and asked to report their pre-awakening dream, then asked to re-report the same dreams upon awakening. The results showed that content which was present in multiple dreams was recalled nearly twice as frequently as content which was in simply one dream. These results seem to support the idea that sleep, and dreaming, serve a biological function of memory consolidation, for at least declarative memories. Declarative memories refer to episodic (specific personal experiences) and semantic memories (factual information) (Cipolli, Fagioli, Mazzetti, & Tuozzi, 2005). This study did not explore dreaming’s effect on procedural memory, the other type of memory.

One of the main structures involved in memory storage and also believed to contribute to the creation of dream images is the hippocampus. Research has brought forth evidence of this involvement, including one study showing that hippocampus function is higher during REM sleep, than during both Non-REM sleep or waking (Nielsen & Stenstrom, 2005). While this increased hippocampal function does not represent a casual relationship between its function and the creation of dream images in REM sleep, there are several lines of evidence that support the idea that the hippocampus is involved in dream image production.

Firstly, several specific hippocampus neurons, specifically CA1, CA3 and the presubiculum region, are known to be involved in the processing of temporal and spacial events, two characteristics of episodic memories. Dreams often have the illusion of a first person experience, and are most always narrative and story like, implying a spacial and temporal binding controls dreams, much like waking experiences. The hippocampus could create this perception. Secondly, there is a link between the timetable of dream image creation, and a biological shift of memories from the hippocampus to the neocortical cortex. Episodic memories are recreated in a decreasing frequency from the day of the experience, lasting for three days, and then these images reappear in increasing frequency from day five to seven since the experience. This U-shaped pattern of dream “memory” coincides with the biological “drift” of memories from the hippocampus to neocortical structures. Dependence of new memories on the hippocampus decreases over time and their dependence on neocortical structures, such as the medial prefrontal cortex, increases. The duration of the transition may last up to several years, but the majority of the transition takes place over the period of about one week. Thirdly, emotional patterns are preserved in dreams, and present themselves much as they would in waking. This is hypothesized to be due to amygdala activity, which works with the hippocampus. Amygdala activity is higher during REM sleep than during wakefulness, and it maintains a reciprocal communication with the hippocampus in the encoding and storage of memories. These three links show support for the hypothesis that the hippocampus is directly involved in the creation of dream images, and in turn, memory consolidation (Nielsen & Stenstrom, 2005). This hypothesis bases its reasoning on the fact that dreams occur during REM sleep, however, it has been shown that dreaming occurs also during non-REM sleep (with 50 percent recall, compared to REM sleep's 82 percent recall) (Payne & Nadel, 2004).
Hippocampus activity is elevated in REM sleep, but not in non-REM sleep, which also creates dream images, therefore it seems that this could be evidence to support that the hippocampus is in fact not directly responsible for creating dream images, as dream images are created in non-REM sleep in the absence of high hippocampus activity.

Nonetheless, there is considerable evidence that the hippocampus is involved in the creation of dream images, which in turn would imply that hippocampus function is related to memory consolidation. The stress hormone cortisol gives us a unique opportunity to test this relationship, exploring whether an increased concentration of cortisol in the brain, known to disrupt hippocampus function, affects memory consolidation. Before we delve into the study, these four points outline the hypothesized connection between cortisol and memory.

1) Different sleep stages are known to occur throughout the night, a specific non-REM stage called slow wave sleep (SWS) is often linked to early night sleep, as more than 80% of SWS is in the first half of the night, while the second half of the night contains twice as much REM sleep as does the first half (in a typical eight hour night).

2) The types of dreams in SWS sleep, in contrast to REM dreams are much different. In SWS sleep, complete episodic memories are common. During REM sleep, complete episodic memories are rare, and instead fragmented, bizarre episodic memories are often retrieved.

3) Similar to the difference in dream content, there is also a difference in the effectiveness of memory consolidation during sleep. Specifically, procedural memory benefits from both REM/late sleep and NREM/early sleep, but declarative memory only benefits from NREM/early sleep.

4) This difference in dream types and dream consolidation has been attributed to the phenomenon of cortisol “build up” during a night of sleep. Early night sleep is characterized by an inhibited adrenal release of cortisol (Payne & Nadel, 2004). Cortisol is known to disrupt hippocampus function and declarative memory is known to rely on hippocampus function.

In a study performed by Phihal and Born, it was shown that administering Cortisol an hour after a word association learning learning task and maintaining an increased Cortisol concentration during early night sleep caused subjects to recall less word associations upon awakening than subjects who were administered a placebo. Increasing plasma glucorticoid concentrations during early sleep by administering cortisol showed a negative affect on declarative memory consolidation, known to rely on the hippocampus. Another aspect of the study involved teaching a mirror-tracing task prior to sleep and administration of cortisol or placebo, however mirror-tracing performance was not affected by cortisol enhancement, showing that cortisol does not affect procedural memory consolidation.

In this study the cortisol-memory consolidation seems to be fairly straightforward: Cortisol negatively affects memory consolidation. However, many studies have revealed that cortisol, in low concentrations, actually increases memory consolidation.

Dreaming is an vastly unknown brain phenomenon that has recently shown some very interesting biological purposes. As technology improves and we are more able to study dreams, research has shown some very interesting functions. From increased

