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Molecular Events during Dream

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Introduction

According to the legendary psychoanalyst Dr. Sigmund Freud, dreams are 'subjective consciousness achieved during sleep'. Dreams represent a subconscious state of brain in which a series of molecular events leads to a shunt pathway of neuronal circuitry where rapid outbursts of neuronal firing occurs that ultimately helps to bypass the memory overload (attained in the conscious state) through the mechanism of dreaming.

No biological phenomenon can be considered without having an evolutionary significance and dreams are no exception to that rule. The evolutionary theory behind dreaming suggests that, slow-wave (SW-) sleep evolved from mere rest in early reptiles as a prolonged and quiescent offline state able to promote calcium dependent memory consolidation. This cognitive role is performed by increased reverberation of waking pattern of neuronal activity during SW sleep. Rapid-Eye-Movement (REM-) sleep [1], a second offline state characterized by high cerebral activity and maximum sensory disconnection, evolved in early birds and mammals as a post-SW-sleep mini-state, lasting merely for transient periods. Despite short duration, REM-sleep is capable of boosting memory consolidation by activating genes [a gene called zif268 (zinc finger binding protein clone 268) [2] is important in this respect] linked to synaptic and neuronal plasticity. At some point, mammals evolved extended REM episodes, prolonging the non-stationary neuronal reverberation that characterizes REM-sleep to promote memory restructuring rather than memory stabilization. Dreams as vivid narratives that unfold in time arose as a by-product of neuronal reverberation during extended REM-sleep.

Due to such non-stationary reverberation, dreams are hyper-associative strings of fragmented memories that stimulate past events and future expectations, enacting possible solutions for cognitive challenges facing the dreamer. Though probabilistic, dream simulations can at times yield accurate predictions of future events. Under increased brain metabolism, it is quite possible to experience an enhanced REM-sleep state in which dream events are under partial or total voluntary control. The cognitive potential of such 'lucid' dreams remains uncharted territory for science [3].

Sleep and Dream

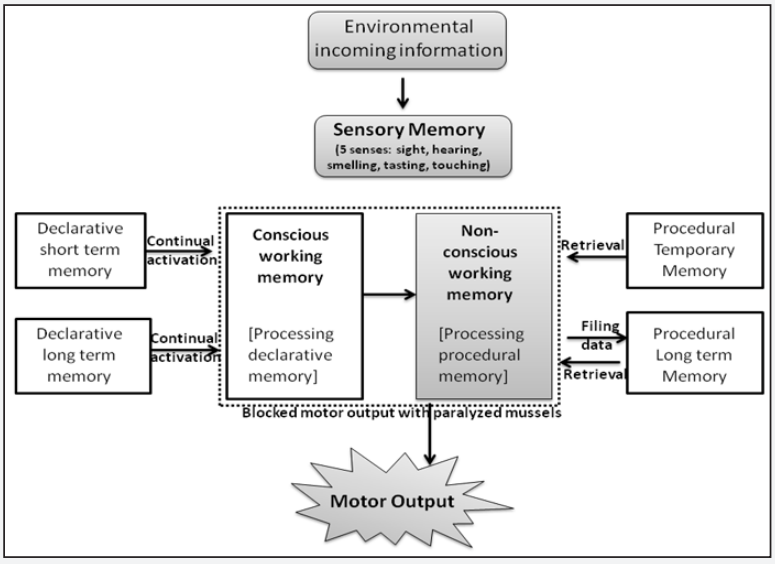

Figure 1: Memory model for the sleeping brain.

It is known to all that, for dreaming sleep is necessary. Sleep is essentially a state of rest in which the subject is cut off from the sensory world. One do not hear, feel, taste, or smell, and would not see if the eyes are pulled open. Everyone has different thresholds during sleep [4], though enough of any stimulus can awake them up. How does the brain manage to cut off sensory input, yet still let in the really important (or insistent) stimuli. The answer lies in the thalamus. No sensory information can get up to the cerebral cortex without first passing through the thalamus. If the gate of the thalamus is closed, then the cortex can shut out the world and go into sleep-mode (Figure 1).

Discussion & Conclusion

In view of our goal, stated in the introduction, we have reviewed contemporary perspectives primarily from research psychology, neuropsychology, neurobiology & molecular biology. The revised Activation-Synthesis theory of dreaming, summarized below, constitute current and necessarily approximate synthesis of these data which may stimulate many future hypo-thesis-testing experiments. With regard to the areas not covered here, we refer to published works (and we eagerly await future reviews) on dreaming and consciousness from scientists and scholars with specific expertise in clinical psychology, philosophy, literature, neural networks, artificial intelligence, as well as functional evolutionary and molecular biology perspectives on sleep and dreaming. We have shown that phenomenological differences between waking, NREM and REM sleep are measurable. In our view, these differences are so great that they represent qualitative differences.

In the case of the major stages of sleep, it may be more useful to envisage psycho-physiological continua, manifested at levels of both the brain and the mind, whose various combinations define not only commonly experienced states of the brain-mind but uncommon ones as well. This is the strategy adopted by the AIM model with the dimensions activation, input source & neuro-modulation representing three such continua. Rather than fixed conditions which must always show similar characteristics in order for brain-mind-body isomorphisms to be valid, behavioral states can be seen as relatively stable sets of values for these continua which have evolved as a result of adaptive benefit to the organism. Such multidimensional combinations can be influenced both at the level of the brain (as when we take a sleeping pill) and at that of the mind (as when we count sheep). Along the dimension of Activation, neuro-imaging studies strongly support an updated view of brain arousal in REM sleep as resulting from ascending influences from the brainstem and sub-cortex. The limbic sub-cortex and related cortex play a major part in the translation of this activation to associative & perhaps even to sensori-motor areas of the cortex. Along the dimension of Input Source, newer research reinforces earlier findings on maximal sensorimotor blockade in REM. Along the dimension of modulation, recent research has confirmed the neuro-modulation of conscious states by the interplay of cholinergic and aminergic influences arising from brain-stem nuclei. This interplay is mediated and modulated by a diversity of cell populations and their neuro-modulators in both the brain stem and the subcortical forebrain [5]. In a revised version of our Activation-Synthesis theory, the distinctive form of dream cognition may be explained at the level of the brain as follows:

a. The intense and vivid visual hallucinosis is due to auto-activation of the visual brain by pontine activation processes impinging, initially, at the level of unimodal visual association cortex.

b. The intense emotions, especially anxiety, elation, and anger are due to the auto-activation of the amygdala, and more medial limbic structures. The emotional salience of dream imagery is due to the activation of the paralimbic cortices by the amygdala.

c. The delusional belief that we are awake, the lack of directed thought, the loss of self-reflective awareness, and the lack of insight about illogical and impossible dream experience are due to the combined and possibly related effects of aminergic demodulation and the selective inactivation of the frontal cortices.

d. The bizarre cognition of dreaming, characterized by incongruities and discontinuities of dream characters, loci, and actions, is due to an orientational instability caused by the chaotic nature of the pontine auto-activation process, its sporadic engagement of association cortices, the absence of frontal cortical monitoring and episodic memory deficits that are, in part, due to, failures of aminergic neuromodulation. We present a schematic explanation for the generation of these cognitive dream features which combines the above findings on state-dependent regional activation with the reciprocal interaction model for the neuromodulation of conscious states.

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