A THEORETICAL PERSPECTIVE ON THE FUNCTION OF REM SLEEP.

ABSTRACT

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The paper explores the possibility of a connection between neurogenesis and REM sleep. Current knowledge about the impact of depression, stress and antidepressant medication on REM sleep and the rate of neurogenesis is examined. Research has shown that neurogenesis occurs in the adult human brain throughout life.[1,2] The hippocampus is one of the sites of neurogenesis[3] and it is this area of the brain that is involved in learning new information.[4] The rate of hippocampal neurogenesis decreases with age[5] and it is known that the time spent in REM sleep also decreases with age. During depression, REM latency is shortened,[6] the time spent in REM is increased[7] and neurogenesis is inhibited;[8] probably due to elevated levels of the glucocorticoid, cortisol.[9] It has also been reported that depression, as well as inhibiting neurogenesis, causes significant loss (up to 20%) of hippocampal volume[10] but that long term use of antidepressants can prevent this loss.[11] In the past it has been suggested that antidepressant medication is effective because it reduces the amount of time spent in REM sleep;[12] thus causing some medics to conclude that REM sleep can worsen or even cause depression. However, more recent research has suggested that neurogenesis inhibition may be a significant factor in the cause of depression.[13, 14]

In light of this information, the author suggests that neurogenesis occurs during periods of REM sleep. A homeostatic model is presented in which REM sleep is

described as a compensatory mechanism to help maintain a homeostatically controlled level of neurogenesis during the developmental and reproductive years. The model, as well as providing an explanation for the variation in the amount of time spent in REM, may also explain the phenomenon of REM rebound following prolonged REM deprivation.

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