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One Sleepless Night Increases Dopamine In The Human Brain

ScienceDaily (Aug. 21, 2008) — Just one night without sleep can increase the amount of the chemical dopamine in the human brain, according to new imaging research in the August 20 issue of The Journal of Neuroscience. Because drugs that increase dopamine, like amphetamines, promote wakefulness, the findings offer a potential mechanism explaining how the brain helps people stay awake despite the urge to sleep.

However, the study also shows that the increase in dopamine cannot compensate for the cognitive deficits caused by sleep deprivation.

"This is the first time that a study provides evidence that in the human brain, dopamine is involved in the adaptations that result from sleep deprivation," said Nora Volkow, MD, director of the National Institute on Drug Abuse, who led the study.

Volkow and colleagues found that in healthy participants, sleep deprivation increased dopamine in two brain structures: the striatum, which is involved in motivation and reward, and the thalamus, which is involved in alertness. The researchers also found that the amount of dopamine in the brain correlated with feelings of fatigue and impaired performance on cognitive tasks.

"These findings suggest dopamine may increase after sleep deprivation as a compensatory response to the effects of increased sleep drive in the brain," said David Dinges, PhD, at the University of Pennsylvania School of Medicine, an expert unaffiliated with the study. "The extent to which this occurs may differentiate how vulnerable people are to the neurobehavioral effects of sleep loss," Dinges said.

The researchers studied 15 healthy participants who were either kept awake all night or allowed a good night's sleep. Researchers tested the same participants in both conditions. On the morning of the study, participants rated how tired they were and did cognitive tasks testing visual attention and working memory.

The researchers used the imaging technique positron emission tomography to study the changes in the dopamine system that occur with sleep deprivation. Compared to well-rested participants, sleep-deprived participants showed reduced binding of a radiolabeled compound ([11C]raclopride) that binds to dopamine receptors in the striatum and thalamus. Because raclopride competes with dopamine for the same receptors, decreased raclopride binding indicates increased levels of dopamine, according to the study authors.

Although decreases in raclopride binding could also indicate a reduction in the number of dopamine receptors, these findings are consistent with prior research implicating increased dopamine levels in wakefulness. For example, some stimulants that prevent sleep, like amphetamines, increase dopamine in the brain, and sleepiness is common in people with Parkinson's disease, which kills dopamine neurons.

The rise in dopamine following sleep deprivation may promote wakefulness to compensate for sleep loss. "However, the concurrent decline in cognitive performance, which is associated with the dopamine increases, suggests that the adaptation is not sufficient to overcome the cognitive deterioration induced by sleep deprivation and may even contribute to it," said study author Volkow. Future research will examine the long-term effects of chronic sleep disturbances on dopamine brain circuits.

The study was supported by the National Institute of Health Intramural Research Program and the U.S. Department of Energy.

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