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Finding the roots of memory impairment from sleep deprivation

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Anyone who has pulled an all-nighter knows there is a price to be paid the next day: trouble focusing, a fuzzy memory and other cognitive impairments. For students, these impairments might just result in a bad grade. But for professionals in high-risk industries that require long hours of concentration, such as emergency medicine or aviation, sleep deprivation can have life-and-death consequences.

A research team led by Ted Abel, a professor of biology in Penn's School of Arts and Sciences and director of the University's interdisciplinary Biological Basis of Behavior program, has now found the part of the brain and the neurochemical basis for sleep deprivation's effects on memory. Specifically, the team aimed to better understand the role of the nucleoside adenosine in the hippocampus, the part of the brain associated with memory function.

"For a long time, researchers have known that sleep deprivation results in increased levels of adenosine in the brain, and has this effect from fruit flies to mice to humans," Abel says. "There is accumulating evidence that this adenosine is really the source of a number of the deficits and impact of sleep deprivation, including memory loss and attention deficits. One thing that underscores that evidence is that caffeine blocks the effects of adenosine, so we sometimes refer to this as 'the Starbucks experiment.'"

Abel's research involved two parallel experiments on sleep-deprived mice, designed to test adenosine's involvement in memory impairment in

different ways.

One experiment was done with genetically engineered mice. These mice were missing a gene involved in the production of gliotransmitters, chemical signals that originate from glia, the brain cells that support the function of neurons. Without these gliotransmitters, the engineered mice could not produce the adenosine the researchers believed might cause the cognitive effects associated with sleep deprivation.

The other experiment took a pharmacological approach. The researchers grafted a pump into the brains of mice that hadn't been genetically engineered; the pump delivered a drug that blocked a particular adenosine receptor in the hippocampus. If the receptor was, indeed, involved in memory impairment, sleep-deprived mice would behave as if the additional adenosine in their brains was not there.

To see whether these mice showed the effects of sleep deprivation, the researchers used an object recognition test. On the first day, mice were placed in a box with two objects and were allowed to explore them while being videotaped. That night, the researchers woke some of the mice halfway through their normal 12-hour sleep schedule. On the second day, the mice were placed back in the box, where one of the two objects had been moved, and were videotaped as they reacted to the change.

"Mice would normally explore that moved object more than other objects, but, with sleep deprivation, they don't," Abel says.

Both sets of treated mice explored the moved object as if they had received a full night's sleep. "These mice don't realize they're sleep-deprived," Abel says.

Abel and his colleagues also examined the hippocampi of the mice, using an electrical current to measure their synaptic plasticity, or how

strong and resilient their memory-forming synapses were. The pharmacologically and genetically protected mice showed greater synaptic plasticity after being sleep deprived than the untreated group.

The knowledge that interrupting the pathway at either end results in mice that show no memory impairments is a major step forward in understanding how to manage those impairments in humans.

“To be able to reverse a particular aspect of sleep-deprivation, such as its effect on memory storage, we really want to understand the molecular pathways and targets,” Abel says.

Such treatments would be especially enticing, given how sensitive the brain is to sleep deprivation’s effects.

“Our sleep deprivation experiments are the equivalent of losing half of a night’s sleep for a single night,” Abel says. “Most of us would think that’s pretty minor, but it shows just how critical the need for sleep is for things like cognition.”

Provided by University of Pennsylvania

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