October 23, 2019

Field

Sleep or die: how good sleep decreases the risk of Alzheimer's disease

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This Break was edited by Carlos Rivera Rivera, *Managing Editor* - TheScienceBreaker

ABSTRACT

Poor-quality sleep changes a pattern of brain activity and increases the risk of Alzheimer's disease. Sufficient sleeping may serve as a cheap and powerful "treatment" to protect your brain from aging and this incurable disorder.

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Various temptations surround our modern life. If you like digital gadgets, you may quickly lose track of time by watching series on Netflix or chatting with friends on Facebook. Then, you may continue using your phone in bed and fall asleep later than expected. But, sleeping less is not how you "earn" time! The value of a good night's sleep should not be underestimated, especially concerning health. Recent studies have shown that sufficient sleep boosts mental wellbeing, immunity, and metabolism. This is why the risk of various disorders, including diabetes and heart disease, is increased by poor-quality sleep. Alzheimer's disease is such a disorder.

Alzheimer's disease is a brain disorder that causes problems with memory, thinking, and behavior. The majorities of patients are older than 65, and the symptoms worsen over time. Despite worldwide efforts, there is still no cure for this complex disease. A better understanding of the link between sleep and the development of Alzheimer's disease may help us to figure out the mechanism of this disorder and even to develop a new treatment.

The so-called tau protein is known as a hallmark of Alzheimer's disease. Tau is abundant in neurons, the primary type of cell that constitutes a brain, where it helps to form microtubules. Microtubules are protein filaments within a cell, and they are essential for the transportation of nutrients within neurons. In Alzheimer's disorder, tau proteins collapse into aggregates (they 'clump'), which causes the collapse of microtubules and the loss of nutrients transport,

eventually killing the cell. This neuronal cell death is essential since neurons, unlike most cells, are unable to reproduce, and as such, we lose these neurons forever. Neurons are connected and can transfer substances and information via this connection. In Alzheimer's, the tau aggregates (and the resulting cell death) spread from one neuron to another. This spreading of cell death eventually leads to the defectiveness of brain areas and the development of Alzheimer's disease.

Neurons release the tau protein into the extracellular fluid, a body fluid that fills all the space in between cells. The release of the tau protein occurs in particular when the neurons are excited. Because the neuronal activity is higher during time awake than during sleep, the authors of the study asked whether the amount of tau protein in the brain extracellular fluid varied with the sleep-wake cycle. To answer this question, the authors used mice as a model organism. The mouse genome is very similar to the human genome. Therefore, it can be applied to the investigation of human diseases, including Alzheimer's disease. First of all, the authors found that the tau protein level in the mouse brain extracellular fluid increases during waking times compared to sleeping times.

Interestingly, if mice were sleep-deprived, the tau protein level in the fluid sharply increased irrespective of the time of measurement. Moreover, they tested humans and found similar changes. These results suggest that sufficient sleep may help to avoid the release of tau protein from neurons and keep its level low in the mouse brain extracellular fluid.

Physical sleep deprivation increases wakefulness, and it also influences other physiological processes. The authors wanted to accurately analyze the activity of neurons in a brain region that controls wakefulness. To do so, they created genetically engineered mice. This genetic modification allowed the researchers to keep the neurons active and mice awake by injecting a chemical into mice. After the injection, the tau protein level in the brain extracellular fluid rapidly increased. This spike suggests that wakefulness is directly associated with the tau release by neurons.

To sum up, insufficient sleep and wakefulness associated with overexcited neurons, increase the release of the tau protein into the extracellular fluid. As these abundant tau proteins tend to aggregate, they spread to adjacent neurons. The spreading of the tau aggregates leads to the death of cell populations in the brain, which eventually results in Alzheimer's disease.

Here, the authors discovered how sufficient sleep protects brain cells from aging and Alzheimer's disease. While the mechanism of this complex disorder is still not entirely understood, this discovery might be a milestone for future advanced study. Furthermore, optimization of the sleep-wake cycle could be an excellent target to develop a new treatment.