

## Sleepless nights show ties to Alzheimer's risk

April 10 2018, by Dennis Thompson, Healthday Reporter



Even one night of lost sleep may cause the brain to fill with protein chunks that have long been linked to the development of Alzheimer's disease, a new study warns.



People deprived of sleep for one <u>night</u> experience an immediate and significant increase in beta amyloid, a substance that clumps together between neurons to form plaques that hamper the <u>brain</u>'s ability to function, researchers found.

"We certainly show that even one night of <u>sleep deprivation</u> can increase the levels of these harmful beta amyloid compounds," said study author Ehsan Shokri-Kojori, a research fellow with the U.S. National Institute on Alcohol Abuse and Alcoholism.

"That's a very logical assumption, I would say, and it's consistent with prior research," he said.

Previous mouse and human studies have found potential links between too little sleep and an accumulation of beta amyloid in the brain, researchers said in background notes. However, many of the human studies have relied on self-reports of sleep quality.

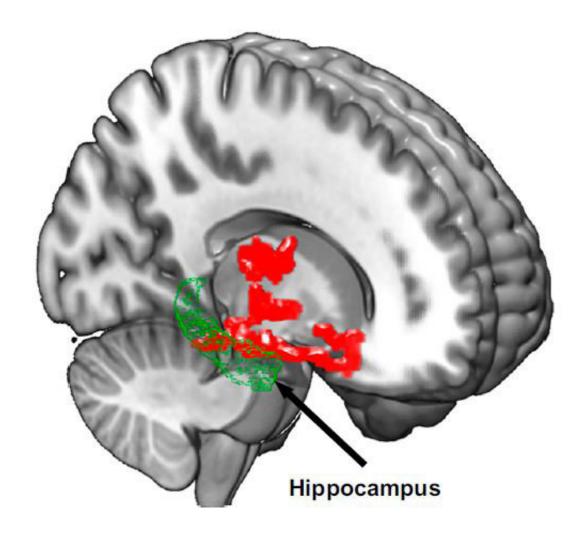
So Shokri-Kojori and his team decided to create an experiment that would more precisely test the effect of sleep deprivation on beta amyloid levels in humans.

They recruited 20 healthy people with no history of brain disorders, and had them spend two nights in the lab—one in which they were allowed to get a good night's rest, and another in which they didn't sleep a wink.

The morning after both nights, the participants underwent brain scans to assess their levels of beta amyloid.

The researchers found that sleep deprivation was associated with a significant increase in beta amyloid in the brain, when compared with a good night's sleep.





Brain imaging after one night of sleep deprivation revealed beta-amyloid accumulation in the hippocampus and thalamus, regions affected by Alzheimer's disease Credit: *Proceedings of the National Academy of Sciences* 

Further, the beta amyloid increases were observed in regions of the brain important to memory and thought, Shokri-Kojori said. These included the hippocampus, which has been tied to memory, and the thalamus, which is an important hub for relaying sensory information to the brain.

Experts suspect that every time a neuron fires, it contributes to the



production of beta amyloid in the brain, explained Dr. Andrew Varga, an assistant professor of <u>sleep medicine</u> with Mount Sinai Health System in New York City. He was not connected to the study.

When people don't sleep, their neurons continue to fire, potentially leading to a buildup of beta amyloid, Varga said.

It also is possible that sleep aids the removal of waste products from the brain, which include beta amyloid, Varga and Shokri-Kojori said.

During sleep, neurons shrink in size, creating space between the cells that allows waste products to be more readily cleared from the brain, Varga said.

However, more study needs to be done to directly link lack of sleep with risk of Alzheimer's disease, the experts said.

For example, it's not known whether a good night's sleep can wipe out the accumulation of beta amyloid from a short bout of insomnia, Shokri-Kojori said.

One future experiment might entail participants undergoing a night of sleep deprivation followed by a night of sound sleep, with brain scans detailing whether good sleep can restore brain health, he said.

Researchers also need to show that continued high levels of beta amyloid in the brain actually increase the chances of clumps forming between neurons, Varga said.

"It makes intuitive sense that if you have chronically high levels of beta <u>amyloid</u> they would congregate together and form plaques, but that piece of it is not fully fleshed out," Varga said.



The new study was published April 9 in the *Proceedings of the National Academies of Sciences*.

**More information:** Ehsan Shokri-Kojori et al. β-Amyloid accumulation in the human brain after one night of sleep deprivation, *Proceedings of the National Academy of Sciences* (2018). DOI: 10.1073/pnas.1721694115

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