

# Sleep disturbance linked to amyloid in brain areas affected by Alzheimer's disease

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Healthy, elderly research participants who report being more sleepy and less rested have higher levels of amyloid deposition in regions of the brain that are affected in Alzheimer's disease, according to a report presented today at the American College of Neuropsychopharmacology annual meeting in Phoenix (Arizona). If sleep disturbance is a cause of amyloid accumulation, it may be an early target for intervention to prevent the progression of cognitive deficits in late life.

Numerous studies have shown the importance of sleep and the effect that sleep deprivation can have on our brains. Sleep apnea is associated with cognitive dysfunction across the lifespan and both untreated apnea and sleep disturbance are associated with increased cognitive dysfunction in patients with Alzheimer's disease. Evaluating physical changes in the brain and how they are related to sleep quality can help determine the extent to which sleep can provide a window on brain function and pathology.

Using positron emission tomography (PET) scanning with a tracer that visualizes deposits of amyloid, a protein that is elevated in the brains of Alzheimer's disease patients, Ruth Benca and her colleagues at the University of Wisconsin-Madison studied the relationship between [sleep quality](#) and brain amyloid levels in a group of 98 cognitively healthy volunteers, 50-73 years of age. The subjects, who were participants in the Wisconsin Registry for Alzheimer's Prevention, completed questionnaires about their sleep and problems related to it. Those who reported greater sleepiness showed greater amyloid accumulation in areas

of the cerebral cortex that are heavily affected in Alzheimer's disease — the supramarginal and frontal medial orbital areas. Higher amyloid in these regions was also linked to less [restful sleep](#) and more [sleep problems](#).

Although it is tantalizing to speculate that the link between [sleep disturbances](#) and [amyloid deposition](#) in the brain may help us identify an early, modifiable marker for Alzheimer's disease, it is too soon to draw that conclusion. "We still need to determine whether sleep disturbance promotes amyloid deposition in the brain, or if a neurodegenerative process produces disordered sleep", commented Dr. Benca. Future work is needed to answer that question and to determine whether interventions that improve sleep can prevent brain changes that lead to Alzheimer's disease.

Provided by American College of Neuropsychopharmacology

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