New Portents of Alzheimer Disease
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Study 1. Poor Sleep Linked to Alzheimer Disease

Poor Sleep Is Linked to Cerebrospinal Fluid (CSF) Biomarkers of Amyloid Pathology in Cognitively Normal Adults: In this study, 101 participants (mean age, 63 years) in the Wisconsin Registry for Alzheimer’s Prevention had undergone lumbar punctures to provide CSF samples and had completed sleep questionnaires. The participants were all cognitively normal and self-assessed their sleep quality.

More sleep problems and daytime somnolence were associated with greater Alzheimer disease pathology, indicated by lower CSF and higher tau levels.[1]

The researchers point out that the participants were younger than those in many previous studies and did not have dementia yet. Also, they looked at novel markers in the CSF.
Clinical Implications for Study 1: “This study shows that poor sleep is not only associated with evidence of increased amyloid deposition, which is associated with risk for Alzheimer’s disease, but also with markers of tau pathology and neurodegeneration/neuroinflammation, which are next steps in progression of the disease. These finding thus provide additional mechanistic evidence for the observed association between disturbed sleep and Alzheimer’s disease progression,” said co-author Ruth M. Benca, MD, PhD.

With effective strategies available to improve sleep, better sleep health may be a potential target for early intervention to attenuate Alzheimer disease pathogenesis. “There is increasing evidence that sleep disturbance and sleep disorders are possible risk factors for neurodegenerative processes, raising the question as to whether there is a clinical opportunity to delay or diminish progression of Alzheimer’s disease and related neurodegenerative disorders by improving sleep and/or treating sleep disorders,” said Dr. Benca.
Study 2. Epilepsy Drug Normalizes Brain Activity in Alzheimer Disease

The Epilepsy Drug Levetiracetam May Provide a Potential New Approach to Treat Cognitive Impairment in Alzheimer Disease: Twelve patients with mild Alzheimer disease received a low dose or a high dose of levetiracetam or a placebo in a double-blind, within-subject protocol. Electroencephalograms (EEGs) were taken at rest and before and after patients received the anti-seizure medication. Subsequently, they underwent MRI scanning to measure blood flow to various regions in the brain.

Only high-dose levetiracetam had a beneficial effect on abnormalities in brain activity. A single intravenous infusion of high-dose levetiracetam “normalized” abnormal brain activity characteristic of Alzheimer disease. There was no effect on cognitive function; however, the researchers suggest that multiple doses of the drug may be necessary to achieve such an effect.[2]
Clinical Implications for Study 2: “To date, levetiracetam has been tested in those with mild cognitive impairment. To our knowledge, this is the first study to test the drug in patients with mild Alzheimer’s disease. Also, we looked at EEG as a measure of neurophysiology, whereas other studies used functional MRI as an endpoint. This study adds to a growing literature that Alzheimer’s disease not only leads to neuronal suppression but hyperexcitability, and that treating that hyperexcitability is an attractive option in the goal of developing new therapeutic agents for Alzheimer’s disease,” said senior author, Daniel Press, MD.
Study 3. Proton Pump Inhibitors and Risk of Cognitive Decline

No Association Found Between PPI Use and Alzheimer Disease: A longitudinal study included 10,486 persons aged 50 years or older with a baseline diagnosis of normal cognition or mild cognitive impairment who underwent detailed annual neuropsychological evaluations. Some 884 individuals were taking PPIs at every visit, 1925 took PPIs intermittently, and 7677 reported never taking PPIs.

Those who used PPIs continually or intermittently had a lower risk of decline in cognitive function and a lower risk of conversion to mild cognitive impairment or Alzheimer disease.[3] Two previous studies had suggested a detrimental effect of PPIs, although another recent study found no link between PPI use and dementia.
Clinical Implications for Study 3: “The current findings do not support that PPIs are associated with greater risk of dementia despite mechanisms proposed as to why they should be,” the researchers stated. They caution clinicians not to speculate about the effect of PPIs on brain functioning until a randomized, prospective clinical trial reveals the effect of PPIs on cognition.
REFERENCES

