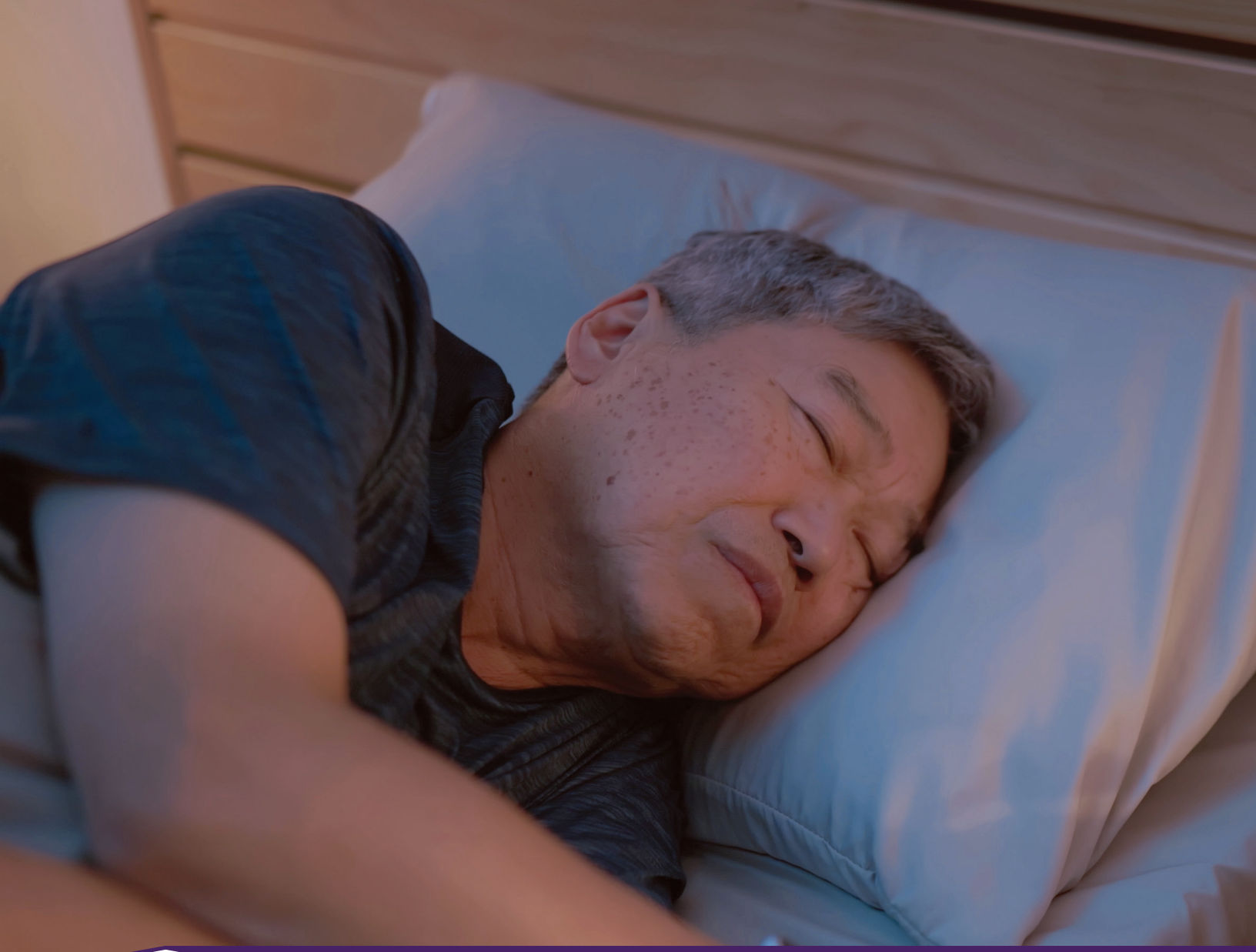

PUBLIC HEALTH CENTER OF EXCELLENCE

ALZHEIMER'S ASSOCIATION

DEMENTIA RISK REDUCTION

SLEEP AND COGNITION:

SLEEP IS IMPORTANT FOR A HEALTHY BRAIN

The Public Health Center of Excellence on Dementia Risk Reduction coordinates risk reduction efforts and helps public health agencies share best practices. The Center translates the latest science on dementia risk reduction into actionable tools, materials and messaging that public health agencies can use to reduce dementia risk for all people — including those in diverse, underserved and higher-risk communities.

Find the summaries of science and additional tools, resources and data at: alz.org/riskreduction

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SLEEP AND COGNITION

WHAT IS ALREADY KNOWN

As people age, sleep disturbances increase and sleep quality decreases. Disruptions in either sleep quality or circadian rhythm can have significant health implications. Just over half (54%) of older adults report that they sometimes or most of the time wake too early without being able to fall back to sleep, and just under half (44%) report that they rarely or never sleep through the night without waking for more than a few minutes. People with advanced dementia experience increased sundowning, fragmented nighttime sleep, changes in the sleep cycle and REM sleep, increased napping, and a high prevalence of sleep apnea. The relationship between such sleep disturbances and cognitive decline is now understood to be bidirectional, with disturbed sleep having a causal relationship with neurodegenerative disease and neurodegeneration leading to an increase in sleep disturbances.

BACKGROUND AND EVIDENCE BASE

A bidirectional association between disturbed sleep and cognitive decline is supported by evidence from several studies. In a study of women with normal cognitive function, cognitive decline over a 15-year period was associated with subsequent poor sleep quality (measured as poor sleep efficiency, taking longer to fall asleep, and waking up after falling asleep). Another study reported an association between current poor sleep quality and subsequent clinically significant cognitive decline, after adjustment for potential confounders (e.g., demographics, lifestyle factors, comorbidities, and medication use). Less efficient sleep and taking longer to fall asleep were associated with an increased risk of mild cognitive impairment and dementia. A study of adults without current dementia but at genetic risk for developing Alzheimer's disease showed that sleep duration was significantly reduced (by 1.9 hours) in people with a genetic predisposition to Alzheimer's disease, suggesting that sleep duration might be useful as a marker for future cognitive decline in this group. Another study found that sleep duration of six hours or less at age 50 or 60 was more strongly associated with the subsequent development of dementia than was sleep of six hours or less at age 70. Short sleep at age 60 was the most strongly linked to subsequent dementia risk, suggesting it may be particularly harmful to have short sleep duration around age 60.

Findings from animal studies suggest a potential mechanism for explaining the relationship between sleep and cognitive function. Two studies in mice suggested that sleep helps prevent the accumulation of harmful substances (such as amyloid-β, a peptide found in the brains of people with Alzheimer's disease). Sleep deprivation resulted in significant accumulation of amyloid-β in one study, and another study showed that sleeping mice cleared twice as much amyloid-β from their brains as conscious mice did through the *glymphatic system*, a brain waste-removal system that is believed to be most active during slow-wave (deep) sleep. Together, findings suggest that when sleep is poor, there is either greater production or reduced clearance of the toxins associated with Alzheimer's disease.

Studies in humans have linked shorter sleep duration and poorer sleep quality to greater amyloid-β deposition. Alterations in sleep architecture, such as fragmented slow-wave (deep) sleep and reductions in sleep spindles also have been linked to Alzheimer's disease pathology and poorer cognitive performance. In addition, idiopathic rapid eye movement sleep behavior disorder (iREM-SBD), a condition in which people commonly act out their dreams while asleep, is a prodromal marker of Lewy body dementia and Parkinson's disease, highlighting the involvement of sleep in the context of neurodegenerative conditions beyond Alzheimer's disease.

In addition to impaired sleep at night, napping during the day is also common in older adults. The relationship between napping and cognitive function is complex. Overall, older people who reported more frequent napping and taking naps longer than two hours in duration demonstrated a significantly greater risk of cognitive decline compared with those who napped less frequently. However, when napping was considered in the context of recent sleep quality at night (measured by sleep duration and sleep efficiency), it was found that napping during the day after a person experienced a poor night's sleep was not associated with this increased risk of cognitive decline; after adjusting for possible confounding variables, this risk appeared to be greater only for those who napped during the day after experiencing good sleep quality at night.

Changes in circadian rhythm (mental and physical behaviors over a 24-hour period associated with the light-dark cycle) have also been found to be related to cognitive decline and dementia risk. Sleep-disordered breathing (e.g., sleep apnea) increases with age and is present in about 25% of older adults by 75 years of age. It is associated with an increased risk of dementia, possibly by reducing the brain's oxygen supply. Interestingly, it has also been associated with a reduced clearance of amyloid-β in cerebrospinal fluid over a two-year period.

Some studies have explored whether treating sleep disruptions can reduce the risk of cognitive impairment. A study that evaluated the effect of continuous positive airway pressure (CPAP) therapy on cognitive function found no cognitive benefit after six months of CPAP therapy compared with a sham treatment, while another reported cognitive improvements after just three weeks of CPAP use. A small study also showed that CPAP therapy reduced amyloid-β accumulation. Meanwhile, certain insomnia medications, such as benzodiazepines and "Z drugs," have been linked to increased risk of cognitive decline. Zolpidem, a commonly used sleep aid, may interfere with glymphatic clearance during sleep. In contrast, a small study in healthy mostly middle-aged adults found that suvorexant, a dual orexin receptor antagonist, acutely reduced Alzheimer's-related proteins in cerebrospinal fluid. More research studies are needed to better understand how different types of sleep medications affect the brain, both in the short- and long-term.

Sleep disturbances are also associated with an increased risk of developing multiple chronic diseases that affect brain health. For example, longitudinal data from the Swedish National study of Aging and Care in more than 4,000 Swedish individuals aged 60+, showed that moderate to severe sleep disturbances were associated with more rapid development of chronic morbidities, particularly neuropsychiatric and musculoskeletal conditions. Cardiovascular health protects against cerebrovascular disease and stroke, with implications for dementia prevention. Obtaining sleep duration of seven to nine hours is now recognized as a key contributor to cardiovascular health, and increasing attention is being paid to other aspects of sleep (e.g., sleep quality, regularity, timing) in cardiovascular health and disease.

Cognitive-behavioral therapy for insomnia (CBT-i) is a non-pharmacological treatment for insomnia. It is effective across adulthood, and in 2016 the American College of Physicians recommended CBT-i as the first-line treatment for chronic insomnia. To date, however, there is not enough research evidence to evaluate the extent to which CBT-i improves cognitive or brain health outcomes. In general, it is not yet known whether targeting sleep is an effective method for reducing a person's risk of developing cognitive impairment and/or dementia.

IMPLICATIONS FOR PUBLIC HEALTH

It is known that improving sleep has beneficial effects on other health outcomes (including mortality, cardiovascular disease, inflammation, obesity, and others), which can improve overall quality of life. Standard sleep hygiene practices can be recommended for any person wanting to improve his or her sleep. Such practices include day-time exercise; avoiding afternoon caffeine intake and fluid, food, nicotine, and alcohol intake before bed; keeping the bedroom dark and cool; and avoiding using electronics in the bedroom. Additionally, low-cost mobile health technology that is focused on behavioral interventions that can assist people wanting to improve sleep quality is increasingly available. Such technology can monitor sleep remotely and may be included in public health initiatives in the coming years.

The role of social determinants of health

Public health interventions that address root causes of sleep disturbances – such as late-night neighborhood noise levels and economic conditions that require individuals to work multiple jobs – may improve an individual's ability to achieve adequate sleep. Sleep health disparities, driven by social, structural, and environmental inequities, play a critical and often underappreciated role in shaping global dementia risk. These disparities are intersectional, emerging across sex/gender, race/ethnicity, and socioeconomic position. Women experience greater sleep fragmentation and insomnia compared with men, particularly during midlife and the menopausal transition — a critical period also associated with rising risk for Alzheimer's disease and related dementias. In the United States, Black and Hispanic/Latino adults tend to report shorter, poorer quality sleep, and have a higher prevalence of undiagnosed and untreated sleep disorders than non-Hispanic Whites. These differences persist beyond individual behavioral or health profiles, suggesting the impact of broader structural determinants, including limited access to health care, cumulative exposure to psychosocial stressors, and residence in disadvantaged neighborhoods. Ecological factors such as housing quality, urban noise, excessive heat, light pollution, and neighborhood crime may further degrade sleep and disproportionately affect low-income populations.

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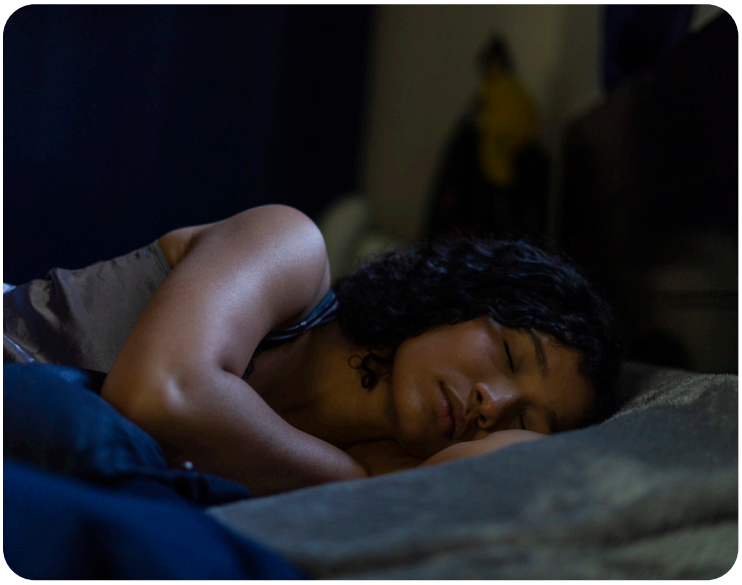
DISCUSSION

While the evidence is clear that sleep and cognitive function are interrelated, it is less clear whether treating disordered sleep can reduce a person's risk of cognitive decline. Accurate measurement of sleep quality and quantity requires objective measurement of sleep and cannot rely solely on self-reports. Today, widely available new technology-based tools permit more precise measurement of sleep quality outside of the laboratory setting and may become helpful for identifying individuals who may benefit from specific treatments to improve sleep. Additionally, research addressing underlying problems that contribute to poor sleep (such as sleep apnea, diabetes, and cardiovascular disease) is in its early stages. Furthermore, this research is examining whether improved treatments for the sleep-related disorders associated with these conditions may secondarily reduce the risk for dementia.



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Glossary

Sleep refers to a natural, reversible state of reduced responsiveness and activity.

Sleep quality encompasses factors such as sleep duration, continuity, depth, and how restorative the sleep is.

Circadian rhythm is the body's internal biological clock that regulates the timing of sleep and wakefulness over a 24-hour cycle.

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For the full list of references, please contact us at CenterOfExcellence@alz.org.