

SURUNKALI UYQUSIZLIK VA DEMENSIYA RIVOJLANISH XAVFI: KOGNITIV PASAYISH BILAN BOG'LIQLIGI

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Annotatsiya. Ushbu maqolada surunkali uyqusizlikning inson miya faoliyati va kognitiv salomatlikka ta'siri yoritilgan. Mayo Clinic tomonidan olib borilgan yirik tadqiqot natijalariga ko'ra, uzoq muddatli uyqu buzilishlari demensiya va yengil kognitiv buzilishlar rivojlanish xavfini sezilarli darajada oshiradi. Tadqiqot davomida surunkali uyqusizlikka chalingan shaxslarning miya tuzilmasida amyloid blyashkalar va oq modda shikastlanishlari ko'proq aniqlangan.

Uyquning yetishmasligi miyaning biologik qarish jarayonini tezlashtirishi mumkinligi ilmiy asoslar bilan isbotlangan. Maqolada uyquning miya salomatligini saqlashdagi ahamiyati, xavf omillari va profilaktik choralar tahlil qilinadi.

Kalit so'zlar: Uyqusizlik, demensiya, kognitiv buzilish, miya qarishi, Alzheimer kasalligi, uyqu gigiyenasi

ХРОНИЧЕСКАЯ БЕССОННИЦА И РИСК РАЗВИТИЯ ДЕМЕНЦИИ: СВЯЗЬ С КОГНИТИВНЫМ СНИЖЕНИЕМ

Аннотация. В данной статье рассматривается влияние хронической бессонницы на когнитивное здоровье и риск развития деменции. Согласно исследованию клиники Майо, лица с длительными нарушениями сна имеют на 40% более высокий риск развития деменции или умеренных когнитивных нарушений. Результаты нейровизуализации показали наличие амилоидных бляшек и изменений белого вещества мозга, характерных для болезни Альцгеймера. Недостаток сна способствует ускоренному старению мозга и снижению памяти и мышления. Подчеркивается важность своевременного выявления и лечения бессонницы как меры профилактики нейродегенеративных заболеваний.

Ключевые слова: Бессонница, деменция, когнитивные нарушения, старение мозга, болезнь Альцгеймера, сон

CHRONIC INSOMNIA AND THE RISK OF DEMENTIA: ASSOCIATION WITH COGNITIVE DECLINE

Annotation. This article examines the relationship between chronic insomnia and the increased risk of dementia and cognitive decline. A large-scale study conducted by the Mayo Clinic revealed that individuals with long-term sleep disturbances are 40% more likely to develop dementia or mild cognitive impairment. Brain imaging findings demonstrated increased amyloid plaque accumulation and white matter abnormalities associated with Alzheimer's disease. Chronic sleep deprivation may accelerate brain aging and negatively impact memory and executive functions. The article highlights the importance of sleep as a protective factor for brain health and emphasizes early intervention strategies.

Keywords: Insomnia, dementia, cognitive impairment, brain aging, Alzheimer's disease, sleep health

INTRODUCTION

Sleep is a fundamental biological process essential for maintaining physical health, emotional stability, and cognitive functioning. Adequate and high-quality sleep plays a critical role in memory consolidation, neural plasticity, metabolic regulation, and clearance of neurotoxic waste products from the brain. In recent decades, however, sleep disorders—particularly chronic insomnia—have become increasingly prevalent, especially among older adults. Chronic insomnia is characterized by persistent difficulty initiating or maintaining sleep at least three nights per week for a minimum duration of three months and is often accompanied by daytime fatigue, impaired concentration, and reduced quality of life.

Beyond its immediate effects on daily functioning, growing evidence suggests that chronic insomnia may have long-term consequences for brain health. Epidemiological and clinical studies increasingly link sleep disturbances with accelerated cognitive aging and an elevated risk of neurodegenerative disorders, including dementia and Alzheimer's disease. Dementia represents a major global health challenge, with rising prevalence due to population aging, and identifying modifiable risk factors has become a priority in preventive neurology.

Recent longitudinal research conducted by the Mayo Clinic and published in *Neurology* has provided compelling evidence that chronic insomnia is associated with a significantly increased risk of mild cognitive impairment and dementia. Individuals with long-term sleep disturbances were shown to exhibit faster cognitive decline and structural brain changes detectable through neuroimaging. These findings highlight insomnia not merely as a symptom of aging, but as a potential contributor to neurodegenerative processes.

The purpose of this article is to examine the association between chronic insomnia and cognitive decline by analyzing clinical, cognitive, and neuroimaging evidence. Particular attention is given to the role of sleep duration, brain structural changes such as white matter abnormalities and amyloid plaque accumulation, and genetic vulnerability factors. Understanding these relationships may contribute to improved prevention strategies and early interventions aimed at preserving cognitive health in aging populations.

MAIN PART

Chronic Insomnia and Cognitive Decline

Chronic insomnia has been increasingly recognized as a condition with significant neurological implications. Longitudinal studies demonstrate that individuals experiencing persistent sleep disturbances are at a higher risk of developing cognitive impairment compared to those with normal sleep patterns. In the Mayo Clinic study involving over 2,700 cognitively healthy older adults with an average age of 70 years, participants with chronic insomnia showed a 40% higher risk of developing mild cognitive impairment or dementia during the follow-up period.

Cognitive assessments conducted annually revealed faster declines in memory, attention, and executive functioning among individuals with insomnia. These declines were equivalent to several additional years of cognitive aging, suggesting that insufficient or disrupted sleep may accelerate age-related cognitive deterioration. Importantly, these associations remained significant even after adjusting for confounding factors such as hypertension, sleep apnea, and the use of sleep medications.

Brain Structural Changes Associated with Insomnia

Neuroimaging findings provide further insight into the mechanisms linking insomnia and cognitive decline. Participants with chronic insomnia exhibited increased white matter hyperintensities, which are indicative of small vessel disease and reduced cerebral blood flow.

These structural abnormalities are commonly associated with impaired cognitive performance and increased dementia risk.

Additionally, amyloid positron emission tomography (PET) imaging revealed greater amyloid plaque accumulation in individuals reporting reduced sleep duration. Amyloid plaques are a hallmark pathological feature of Alzheimer's disease, and their presence in cognitively normal individuals suggests an early stage of neurodegeneration. The accumulation of amyloid may be partially explained by impaired glymphatic clearance during sleep, a process essential for removing neurotoxic proteins from the brain

Impact of Sleep Duration and Perceived Sleep Changes

The study further differentiated between individuals with insomnia who perceived reduced sleep and those who reported sleeping more than usual. Participants experiencing reduced sleep demonstrated lower baseline cognitive scores and more pronounced brain abnormalities, including higher amyloid burden and white matter damage. In contrast, those reporting increased sleep duration showed fewer white matter changes, suggesting a potential protective effect of adequate sleep.

These findings emphasize that not only the presence of insomnia, but also changes in sleep duration, may differentially influence brain health. Reduced sleep appears to be particularly detrimental, reinforcing the importance of maintaining sufficient sleep quantity and quality.

Genetic Vulnerability and Increased Risk

Genetic factors further modulate the relationship between insomnia and cognitive decline.

Individuals carrying the APOE $\epsilon 4$ allele—a well-established genetic risk factor for Alzheimer's disease—exhibited steeper declines in cognitive function when chronic insomnia was present. This suggests a synergistic interaction between genetic susceptibility and sleep disturbances, leading to accelerated neurodegeneration.

The presence of such vulnerability underscores the importance of personalized risk assessment and targeted interventions, particularly in populations at higher genetic risk.

Clinical and Preventive Implications

The evidence reviewed in this article suggests that chronic insomnia should be regarded as a clinically significant risk factor for cognitive decline rather than a benign or secondary complaint.

Early identification and effective management of sleep disorders may represent a crucial strategy in dementia prevention. Interventions aimed at improving sleep quality—such as cognitive behavioral therapy for insomnia, sleep hygiene education, and treatment of comorbid sleep disorders—may help preserve cognitive function and slow brain aging.

The 'glymphatic system' — the flow of cerebrospinal fluid during sleep to flush out toxins and waste materials from the brain — plays an essential role in keeping the brain healthy.

A new study, using MRI scans, found that people with cardiovascular risk factors that impaired the function of the glymphatic system had an increased risk of dementia.

The researchers suggest that improving sleep patterns to enhance glymphatic system function and treating cardiovascular risks could both help reduce dementia risk.

The glymphatic system is a recently discovered waste clearance system, most active during sleep, that removes toxins and waste materials, including those associated with dementia, from the central nervous system ^{Trusted Source}.

A new study has found that people with an impaired glymphatic system have a higher risk of developing dementia.

The study, published in *Alzheimer's & Dementia: The Journal of the Alzheimer's Association* ^{Trusted Source}, suggests that improving glymphatic function could be a powerful tool in reducing the risk of dementia.

“These findings are largely expected, building on a growing body of research that implicates impaired cerebrospinal fluid ^{Trusted Source} (CSF) dynamics, often referred to as the ‘glymphatic system’, in dementia. Animal studies have long shown that disrupted CSF flow hampers the clearance of toxic proteins such as amyloid beta and tau, which are central to Alzheimer’s disease pathology. What makes this study significant is that it provides large-scale, human-based evidence from over 45,000 participants in the UK Biobank, confirming that MRI markers of CSF dysfunction [...] are associated with higher dementia risk.”

— Dr Steve Alder, consultant neurologist at Re:Cognition Health, who was not involved in the study.

Testing 3 biomarkers

In their study, researchers used machine-learning algorithms, developed by Yutong Chen, one of the paper’s authors, while a medical student at the University of Cambridge. These algorithms can assess glymphatic functions from multiple MRI (magnetic resonance imaging) scans.

The researchers applied the algorithms to MRI scans from around 45,000 adults. They identified 3 biomarkers, linked to impaired glymphatic function, that predicted dementia risk over the following decade.

The three biomarkers, all of which provide insights into cerebrospinal fluid ^{Trusted Source} (CSF) dynamics, were:

BOLD-CSF coupling — blood oxygen level-dependent cerebrospinal fluid coupling, which tracks the connection between global brain activity and CSF flow.

DTI-ALPS, a measure of the diffusion of water molecules along the perivascular spaces ^{Trusted Source} — passageways along which CSF travels, that surround blood vessels in the brain.

Additionally, they found that several cardiovascular risk factors, such as hypertension, diabetes, smoking and alcohol, all impaired glymphatic function and increased dementia risk.

Glymphatic activity is greatly increased during sleep, and suppressed during waking hours.

While you are sleeping, the system clears waste products, including potentially neurotoxic products, such as β -amyloid ^{Trusted Source} which forms plaques that interfere with nerve transmission in Alzheimer’s disease, from the brain.

“There are emerging strategies to support or improve glymphatic clearance. Good cardiovascular health is key — maintaining optimal blood pressure, managing diabetes and

exercising regularly all enhance vascular pulsatility and CSF flow. Quality sleep is also critical, as glymphatic clearance is most active during deep sleep. Avoiding excessive alcohol, staying hydrated and possibly using interventions that improve arterial elasticity (e.g., aerobic fitness, dietary omega-3s) may further help preserve waste clearance efficiency.” — Steve Allder, MD

So getting a good night’s sleep could be one way to help improve your brain clearance.

Experts advise several ways to improve sleep, including exercising daily, maintaining a regular sleep schedule, avoiding large meals, beverages and alcohol near bedtime, and taking time to wind down before bed in the evening.

Potential for earlier dementia diagnosis

Allder said that with further validation, the findings could help lead to earlier diagnosis and preventive measures for dementia:

“These findings suggest that MRI-based markers of CSF dynamics — particularly DTI-ALPS, BOLD-CSF coupling, and choroid plexus volume — could become early, noninvasive indicators of dementia risk before cognitive symptoms appear. If validated across diverse populations, these measures could be integrated into routine brain imaging protocols for at-risk individuals, especially those with cardiovascular or metabolic conditions.”

“This would enable earlier identification of people with impaired brain clearance, and open opportunities for preventive interventions targeting vascular health, sleep quality and lifestyle modification. Additionally, these imaging markers could serve as biomarkers in clinical trials, helping to evaluate whether therapies aimed at improving glymphatic flow (such as sleep optimization or vascular therapies) reduce dementia risk or progression,” he told MNT.

Medical myths: How much sleep do we need?

In this Special Feature, we hack into some of the myths that surround sleep duration.

Among other questions, we ask whether anyone can truly get by on 5 hours of sleep each night. We also uncover whether sleep deprivation can be fatal.

Although we all know sleep is vital to maintain good health, there are still many unanswered questions. And, over the millennia, a variety of myths and half-truths have developed and stuck.

This feature is the second and final part of our series that tackles sleep-related myths. Find the first part [here](#).

This time, we focus on myths that surround how much sleep the average person needs. We also discuss naps, the effects of sleeping too little or too long, and sleep in the animal kingdom.

Article highlights:

1. Everyone needs 8 hours

As with many aspects of human biology, there is no one-size-fits-all approach to sleep.

Overall, research suggests that for healthy young adults and adults with normal sleep, 7–9 hours is an appropriate amount.

The story gets a little more complicated, though. The amount of sleep we need each day varies throughout our lives:

newborns need 14–17 hours

infants need 12–15 hours

toddlers need 11–14 hours

preschoolers need 10–13 hours
school-aged children need 9–11 hours
teenagers need 8–10 hours
adults need 7–9 hours
older adults need 7–8 hours

You can train your body to need less sleep

There is a widely shared rumor that you can train your body to need fewer than 7–9 hours’ sleep. Sadly, this is a myth.

According to experts, it is rare for anyone to need fewer than 6 hours’ sleep to function.

Although some people might claim to feel fine with limited sleep, scientists think it is more likely that they are used to the negative effects of reduced sleep.

People who sleep for 6 hours or fewer each night become accustomed to the effects of sleep deprivation, but this does not mean that their body needs any less sleep. Cynthia LaJambe, a sleep expert at the Pennsylvania Transportation Institute in Wingate, explains:

“Some people think they are adapting to being awake more, but are actually performing at a lower level. They don’t realize it because the functional decline happens so gradually.”

“In the end, there is no denying the effects of sleep deprivation. And training the body to sleep less is not a viable option.”

– Cynthia LaJambe

However, it is worth noting that some rare individuals do seem to function fine with fewer than 6.5 hours’ sleep each night. There is evidenceTrusted Source that this might be due to a rare genetic mutation, so it is probably not something that someone can train themselves to achieve.

2. Daytime naps are unhealthy

Generally, experts recommend people avoid naps to ensure a better night’s sleep. However, if someone has missed out on sleep during previous nights, a tactical nap can help repay some of the accrued sleep debt.

Around 20 minutes is a good nap length. This gives the body ample time to recharge.

People who sleep much longer than this could mean they descend into a deep sleep, and once awake, they feel groggy.

Daytime napping is relatively common in the United States, but taking a “siesta” is the norm in some countries. Naturally, our bodies tend to dip in energy during the early afternoon, so perhaps napping around that time is more natural than avoiding sleep until nighttime.

After all, the vast majority of mammals are polyphasic sleepers, which means they sleep for short periods throughout the day.

In a large reviewTrusted Source of the effects of napping, the authors explain afternoon naps in people who are not sleep deprived can lead to “subjective and behavioral improvements” and improvements in “mood and subjective levels of sleepiness and fatigue.” They found people who nap experience improved performance in tasks, such as “addition, logical reasoning, reaction time, and symbol recognition.”

Not all naps are equal, however. There is a great deal of variation, such as the time of day, duration, and frequency of naps. One author explains:

“Epidemiological studies suggest a decrease in the risk of cardiovascular and cognitive dysfunction by the practice of taking short naps several times a week.”

The author also acknowledges that much more research is needed to understand how factors associated with napping influence health outcomes. Medical News Today recently examined the relationship between napping and cardiovascular disease in a Special Feature.

It is also important to note if an individual experiences severe tiredness during the day, this might be a sign of a sleep disorder, such as sleep apnea.

Scientists will need to conduct more research before they can finally put all the napping myths and mysteries to bed.

3. All animals sleep

Because humans sleep, and our companion animals appear to sleep, many people assume all animals do the same. This is not true. The authors of a paper entitled “Do all animals sleep?” explain:

“Some animals never exhibit a state that meets the behavioral definition of sleep. Others suspend or greatly reduce ‘sleep’ behavior for many weeks during the postpartum period or during seasonal migrations without any consequent ‘sleep debt.’”

They also explain that some marine animals, reptiles, fish, and insects do not appear to enter REM sleep.

Because sleep is not simply a lack of consciousness, but a rhythmic cycle of distinct neural patterns, it is a challenge to distinguish whether an animal sleeps or takes a rest.

“[F]ewer than 50 of the nearly 60,000 vertebrate species have been tested for all of the criteria that define sleep,” the authors explain. “Of those, some do not meet the criteria for sleep at any time of their lives, and others appear able to greatly reduce or go without sleep for long periods of time.”

4. More sleep is always better

Although many people struggle to get the amount of sleep they need to feel refreshed, some regularly sleep longer than their body needs. One might think this could endow these individuals with superpowers.

However, researchers identify a link between longer sleep durations and poorer health. For instance, one study Trusted Source, which followed 276 adults for 6 years, concluded:

“The risk of developing obesity was elevated for short and long duration sleepers, compared to average-duration sleepers, with 27% and 21% increases in risk, respectively.”

This finding held even when the scientists controlled the analysis for age, sex, and baseline body mass index. Sleep duration might also impact mortality, according to some researchers.

A meta-analysis, which appears in the journal Sleep, concludes “Both short and long duration of sleep are significant predictors of death in prospective population studies.”

5. Sleep deprivation can be lethal

There is no record of anyone dying from sleep deprivation. In theory, it may be possible, but as far as scientists can ascertain, it is improbable.

It is understandable why this myth may have taken root, though. Sleep deprivation, as many people can attest, can feel horrendous. However, the case of Randy Gardner demonstrates that extreme sleep deprivation is not fatal.

In 1965, when Gardner was just 16, he was part of a sleep deprivation experiment. In total, he stayed awake for 11 days and 24 minutes, which equates to 264.4 hours.

During this time, he was monitored closely by fellow students and sleep scientists. As the days rolled on, sleep deprivation symptoms worsened, but he survived. So why has this myth persisted?

In their experiments, the researchers placed rats on a disc suspended above water. They continuously measured their brain activity. Whenever the animal fell asleep, the disc would automatically move, and the rat would need to act to avoid falling in the water.

However, he did not die and apparently, did not experience any long-term health issues.

Another reason why the myth that sleep deprivation can be fatal persists might be due to a condition called fatal familial insomnia. People with this rare genetic disorder become unable to sleep. However, when individuals with this disease die, it is due to the accompanying neurodegeneration rather than lack of sleep.

Although sleep deprivation will probably not kill you directly, it is worth adding a note of caution: being overtired does increase the risk of accidents. According to the National Highway Traffic Safety Administration, “Drowsy driving kills — it claimed 795 lives in 2017.”

Similarly, a review Trusted Source published in 2013 concludes, “[a]pproximately 13% of work injuries could be attributed to sleep problems.” So, although sleep deprivation is not deadly in a direct sense, it can have fatal consequences.

Additionally, if we consistently deprive our bodies of sleep for months or years, it increases the risk Trusted Source of developing several conditions, including cardiovascular disease, hypertension, obesity, type 2 diabetes, and some forms of cancer.

Conclusion

Chronic insomnia is increasingly recognized as a significant risk factor for cognitive decline and the development of dementia. Evidence from longitudinal and neuroimaging studies indicates that persistent sleep disturbances are associated with accelerated brain aging, impaired cognitive performance, and structural brain changes, including white matter abnormalities and amyloid plaque accumulation. Individuals with reduced sleep duration demonstrate particularly pronounced cognitive deficits, while genetic susceptibility further amplifies these effects.

These findings emphasize the importance of early identification and effective management of chronic insomnia as a potentially modifiable risk factor for neurodegenerative diseases.

Improving sleep quality through clinical and behavioral interventions may contribute to preserving cognitive function and reducing dementia risk in aging populations. Future research should focus on clarifying causal mechanisms and evaluating targeted sleep-based prevention strategies to support long-term brain health.

REFERENCES:

1. Carvalho, D. Z., Kolla, B. P., McCarter, S. J., St. Louis, E. K., Machulda, M. M., Przybelski, S. A., et al. (2025). Associations of chronic insomnia, longitudinal cognitive

- outcomes, amyloid-PET, and white matter changes in cognitively normal older adults. *Neurology*, 105(7). <https://doi.org/10.1212/WNL.00000000000214155>
2. Petersen, R. C. (2016). Mild cognitive impairment. *Continuum*, 22(2), 404–418.
 3. Jack, C. R., et al. (2018). NIA-AA research framework: Toward a biological definition of Alzheimer's disease. *Alzheimer's & Dementia*, 14(4), 535–562.
 4. Xie, L., et al. (2013). Sleep drives metabolite clearance from the adult brain. *Science*, 342(6156), 373–377.
 5. Lim, A. S. P., et al. (2013). Sleep fragmentation and cognitive decline. *Neurology*, 81(5), 400–407.
 6. Spira, A. P., et al. (2014). Self-reported sleep and cognitive decline. *Sleep*, 37(12), 1937–1946.
 7. Musiek, E. S., & Holtzman, D. M. (2016). Mechanisms linking circadian clocks, sleep, and neurodegeneration. *Science*, 354(6315), 1004–1008.
 8. Walker, M. (2017). *Why we sleep: Unlocking the power of sleep and dreams*. Scribner.
 9. World Health Organization. (2023). *Dementia*.
 10. National Institute on Aging. (2024). *Sleep and Alzheimer's disease*.
 11. Buysse, D. J. (2013). Insomnia. *JAMA*, 309(7), 706–716.
 12. Scullin, M. K., & Bliwise, D. L. (2015). Sleep, cognition, and normal aging. *Sleep Medicine Clinics*, 10(1), 17–27.
 13. Lim, D. C., & Pack, A. I. (2017). Obstructive sleep apnea and cognitive impairment. *Sleep Medicine Reviews*, 33, 45–54.
 14. Vemuri, P., et al. (2018). White matter hyperintensities and cognition. *Brain*, 141(2), 558–572.
 15. Somers, V. K., et al. (2008). Sleep apnea and cardiovascular disease. *Circulation*, 118(10), 1080–1111.
 16. Kryger, M. H., Roth, T., & Dement, W. C. (2017). *Principles and practice of sleep medicine*. Elsevier.
 17. Alzheimer's Association. (2024). *Alzheimer's disease facts and figures*.
 18. NIH. (2023). *Sleep deprivation and brain health*.
 19. Varga, A. W., et al. (2016). Sleep disruption and Alzheimer's pathology. *Neurology*, 87(15), 1473–1481.
 20. Scarmeas, N., et al. (2018). Lifestyle factors and dementia risk. *JAMA Neurology*, 75(8), 955–964.
 21. Irwin, M. R. (2015). Why sleep is important for health. *Psychiatric Clinics of North America*, 38(4), 553–566.