SLEEP INSTITUTE
Clinical Insights by Resmed

Sleep Disorders as a Potentially Modifiable Factor in Cognitive Health

By: Sleep Institute
Published: July 14, 2026

The relationship between sleep disorders and cognitive health has become an important focus of brain health research. Sleep disorders are associated with a significantly higher risk of Alzheimer's disease and all-cause dementia, and the biological mechanisms involving the brain's own waste-clearance system are increasingly better understood.

Sleep disorders have long been viewed and managed as quality-of-life conditions. But that is changing. A growing body of evidence now links chronic sleep disruption to biological processes central to neurodegeneration, such as impaired waste clearance and neuroinflammation.[1–5]

Important scientific questions still remain, but the implication of what is known is already significant. Sleep disorders are prevalent at scale. Obstructive sleep apnea (OSA) affects an estimated 1 billion adults globally; chronic insomnia affects 10–15% of adults.[6,7] If these conditions contribute meaningfully to cognitive decline, then earlier identification and sustained treatment may represent one of the largest and most practical opportunities available in brain health today.

What Happens in the Brain: The Accumulation Phase

During deep sleep, particularly slow-wave sleep, the brain shifts into a form of maintenance mode. An important element of that is the glymphatic system, the brain waste-clearance pathway that flushes metabolic byproducts, including amyloid-β and tau proteins, through channels surrounding the brain's blood vessels.[1]

The glymphatic system is most active during slow-wave sleep and is significantly impaired by the features that define most sleep disorders, including fragmented sleep, recurrent/heightened arousals, intermittent hypoxia, and reduced time in deep sleep stages.[2,3]

Patients with OSA show abnormally low cerebrospinal fluid amyloid-β42 levels and higher amyloid burden on positron emission tomography (PET) imaging, overlapping with Alzheimer's disease (AD) biomarker pattern.[4] More, older adults with witnessed apneas have been found to carry elevated tau-PET signal in the brain.[5]

OSA-related intermittent hypoxia and oxidative stress also promote neuroinflammation and cerebrovascular injury, linking sleep-disordered breathing to both neurodegenerative and vascular pathways of cognitive decline.[5]

A recent study found that untreated OSA patients carried the highest glymphatic fluid burden of any group measured, a marker of sluggish waste clearance. In contrast, positive airway pressure (PAP)-treated OSA patients had fluid levels comparable to healthy controls.[8]

OSA is not the only sleep disorder implicated. Insomnia independently disrupts slow-wave sleep and has been

associated with elevated dementia risk through related mechanisms.[9,10]

Risk Scope: The Population-Level Signal

What makes the sleep–cognition relationship more scientifically significant is the consistency of the association across very large, independent populations studied over decades.

A 2025 meta-analysis quantified these associations:[10]

- OSA was associated with a 33% higher risk of all-cause dementia and a 45% higher risk of Alzheimer's disease.[10]
- Insomnia was associated with a 36% higher risk of all-cause dementia, a 49% higher risk of Alzheimer's disease, and a 59% higher risk of vascular dementia.[10]
- Excessive daytime sleepiness and sleep-related movement disorders showed significant independent associations with dementia risk.[10]

Another analysis of 51 studies found ten distinct sleep conditions including insomnia, sleep fragmentation, prolonged sleep latency, REM sleep behavior disorder, and excessive time in bed independently associated with higher cognitive disorder risk, surviving multivariable adjustment for age, cardiovascular disease, depression, and lifestyle factors.[11]

In short, sleep disorders like OSA and insomnia are repeatedly associated with cognitive deficits and vulnerabilities.[12]

Sleep Disorders Are Treatable, and Treatment May Matter for the Brain

The clinical importance of the sleep–cognition relationship lies primarily in modifiability. Most contributors to dementia risk, such as age, genetics, and family history, are fixed. They cannot be modified once present. Sleep disorders are different. They are identifiable and treatable, and emerging evidence strongly suggests treatment may carry meaningful cognitive benefits.[13–16]

For OSA, the most extensively studied intervention is PAP therapy. Multiple studies report improvements to varying degrees in attention, processing speed, memory, and executive function with PAP use, especially in patients with moderate-to-severe OSA or baseline cognitive impairment.[13–15]

For insomnia, cognitive behavioral therapy for insomnia (CBT-I) restores slow-wave sleep and reduces hyperarousal, both of which are directly implicated in impaired glymphatic clearance. However, direct evidence on CBT-I's effect on dementia biomarkers or long-term cognitive trajectories remains limited.[16]

For other sleep disorder presentations and when PAP is not tolerated in patients with OSA, mandibular advancement devices (oral appliances) and positional therapy offer viable alternatives, though long-term cognitive outcome data for these modalities are also not yet available.

Timing: A Variable the System is Not Built Around

One of the most consistent findings across the OSA-cognition medical literature is that treatment timing matters considerably.

PAP therapy initiated in mid-life or at the earliest stage of cognitive change appears most likely to stabilize or modestly improve cognitive trajectories.[12] On the other hand, advanced dementia patients struggle significantly to tolerate PAP therapy.[17] This makes adherence more likely to fall, compromising the cognitive benefits that may be realized from therapy.

A study found that one year of PAP adherence in patients with mild cognitive impairment (MCI) and OSA significantly improved cognitive processing speed compared to a non-adherent control group. Additional improvements were also observed for attention and everyday function.[18]

This does not mean older adults or people with cognitive impairment should be excluded from sleep treatment. It means care models should prioritize identifying sleep disorders earlier and building support around the patient and caregiver, instead of treating adherence as a strict compliance metric.

What Remains Unresolved: Current Evidence Limitations

As a whole, the evidence is convergent, not conclusive. A recent critical appraisal of thirteen years of glymphatic research found that glymphatic dysfunction is a plausible contributor to AD pathology, but its causal role in humans has not been established.[5] The evidence base relies primarily on correlative clinical data and rodent models, with no clinically validated tool yet to directly measure glymphatic exchange in the living human brain.

Epidemiology cannot fully determine whether sleep disorders are causal, contributory, early markers of neurodegeneration or some combination of these. Residual confounding may remain because people with untreated sleep disorders can differ from those without them in cardiovascular risk, body weight, depression, medication use, health care access, and socioeconomic factors. Reverse causality is also possible, especially in older adults, because disrupted sleep can be an early feature of cognitive decline.[19]

Finally, although PAP has improved selected cognitive outcomes in some studies, no completed large-scale randomized controlled trial (RCT) has demonstrated that treating a sleep disorder prevents or delays dementia.

The Trials That May Change This Conversation

Results from ongoing and recently completed multicenter studies and randomized trials will help clarify how much of the observed relationship is causal and whether treatment definitively changes neurocognitive outcomes.

- ESSENTIAL: Multicenter RCT; 400 cognitively normal adults with moderate-severe OSA. Primary outcomes: plasma AD biomarkers (Aβ42/40, p-tau181/217, NfL) and cognitive scores (PACC). *Est. completion: May 2028.*
- Memories 2: Non-randomized parallel assignment; 460 adults ≥55 with amnestic MCI and OSA. Primary outcomes: Digit Symbol-Coding Test; Clinical Dementia Rating scale. *Primary results pending; interim adherence data published.*
- CPAP RCT — Sunnybrook: RCT; 206 participants ≥55 with MCI and moderate-severe OSA. Primary outcomes: SDMT cognitive score; vascular and AD biomarkers (pTau181, GFAP); MRI parameters. *Est. completion: January 2028.*
- PRAISE: RCT comparing standard CPAP vs. personalized adherence support in Black adults aged 60–85 with OSA. Primary outcomes: CPAP adherence; AD and inflammatory biomarkers (NfL, GFAP, Tau, Aβ). *Est. completion: May 2028.*
- OSA and AD Progression: Mechanistic pilot RCT: CPAP vs. supplemental oxygen in older adults at high AD risk with OSA. Primary outcomes: AHI, neuroimaging, AD biomarkers, memory outcomes. *Results pending publication.*
- OSA and Biomarkers of Alzheimer's Disease: RCT; 100 OSA patients and 30 controls. Primary outcomes: change in Aβ-40/Aβ42 levels; P-Tau, NfL. *Results pending publication.*
- RESHAPED: Multi-site feasibility RCT assessing the effects of treating OSA on cognitive decline in older adults at risk of dementia within memory clinic settings. Designed as a precursor to a full-scale prevention trial. *Ongoing.*

Positive findings could potentially reframe PAP therapy from a symptomatic respiratory treatment to a brain health intervention. Null or mixed results would not negate existing evidence on cognitive improvements in diagnosed OSA populations but would materially temper the dementia-prevention framing. Either outcome will clarify the scientific and care policy conversation considerably.

Expanding Sleep Care Infrastructure for Long-Term Brain Health

Sleep disorders like OSA and insomnia affect hundreds of millions of people worldwide.[6,7] Both are substantially underdiagnosed. Both are independently associated with elevated neurocognitive risk across large population studies.[10,11] And both are treatable.

Yet the infrastructure supporting sleep disorder treatment has largely been built around device adherence and measurable respiratory outcomes. While those remain valuable, the sleep disorder care model must broaden.

If results from the ongoing trials confirm that treating sleep disorders modifies AD biomarkers or slows cognitive decline, clinical guidelines must be updated to reflect that. The American Academy of Sleep Medicine (AASM), the European Sleep Research Society (ESRS), and the World Sleep Society are the bodies best positioned to act on such readouts. A coordinated update establishing cognitive health as a standard dimension of sleep disorder assessment and management would represent a meaningful structural shift.

Even before those results arrive, existing evidence already supports targeted changes to how sleep disorders are identified and managed.

- **Routine sleep disorder screening:** Memory clinics, MCI assessment pathways, and primary care workflows for older adults should systematically screen for sleep disorders. Earlier identification means more opportunity to treat when patients are most able to engage with therapy and when the brain-health relevance may be greatest.
- **Connect screening to referral and treatment pathways:** Earlier identification only matters if patients can access appropriate care. In primary care and neurology settings, a positive screen should trigger a clear next step: Sleep testing, CBT-I referral, sleep services consultation, and follow-up where appropriate.
- **Redefine adherence as sustained support:** Sleep therapy adherence should be reframed as a long-term, cyclical care process involving telemonitoring, troubleshooting, caregiver support, and early continuous follow-up.

What the Science Already Makes Actionable

The question is no longer whether sleep matters for brain health. It is how much it matters and across which populations. Ongoing trials will sharpen the picture.

But the case for action does not require perfect certainty. The evidence base has reached a point where guideline bodies can reasonably act on the strong, existing associations. Sleep disorders are common, identifiable, and treatable. They are linked to the mechanisms that drive brain aging.

As populations age and dementia prevalence rises, sleep care stands as an important avenue for shaping long-term brain health. Sleep is not the magic bullet, but it is one of the few areas in the cognitive health landscape where early identification and sustained treatment are already possible today.

References

[1] Keil SA, et al. Science. 2025;389(6756):eadv8269.
[2] De la Herrán-Arita AK. Acta Neurol Belg. 2025. (Online ahead of print)
[3] Yang H, et al. Psychopharmacology (Berl). 2026. (Online ahead of print)
[4] Cui W, et al. Front Aging Neurosci. 2022;14:902408.
[5] Tsai CY, et al. Front Neurol. 2022;13:1038735.
[6] Benjafield AV, et al. Lancet Respir Med. 2019;7(8):687–698.
[7] Benjafield AV, et al. Sleep Med Rev. 2025;82:102121.
[8] Keil SA, et al. Neurobiol Dis. 2026;221:107325.
[9] Baglioni C, et al. Sleep Med Rev. 2014;18(3):195–213.
[10] Ungvari Z, et al. Geroscience. 2025;47(3):4899–4920.
[11] Xu W, et al. J Neurol Neurosurg Psychiatry. 2020;91(3):236–244.
[12] Gurbani, N., et al. Curr Treat Options Neurol. 2026;27: 26.
[13] Durtette A, et al. Sleep Med. 2024;123:7–21.
[14] Perez-Cabezas V, et al. J Clin Med. 2020;9(1):181.
[15] Rollo E., et al. Curr Treat Options Neuro.2024; 26:509–521.
[16] Sforza M, et al.Transl Psychiatry. 2026;16(1):88.
[17] Oliver C, et al. Sleep Med Rev. 2024;73:101869.
[18] Richards KC, et al. J Am Geriatr Soc. 2019;67(3):558–564.
[19] Piñol-Ripoll G, et al. Front Neurosci. 2023;16:1117265.