Sleep through the Lifecycle:
Reducing Risk of Cognitive Decline

"Innocent sleep. Sleep that soothes away all our worries. Sleep that puts each day to rest. Sleep that relieves the weary labourer and heals hurt minds. Sleep, the main course in life's feast, and the most nourishing"—William Shakespeare, Macbeth (2.2.50–52).

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LOUISIANA DEPARTMENT OF HEALTH

Speaker

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Dr. Foundas is a Behavioral Neurologist with over 30 years of experience caring for people with cognitive decline & dementia. She is the Executive Director of the Brain Institute of Louisiana, and her clinical practice is NOLA Brain & Behavior.

Disclosure

In the past 24 months, I have NOT had any financial relationships with any ineligible companies.

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Objectives

- To understand how the duration & timing of sleep varies across the life cycle
- To learn about the regulatory mechanisms of sleep in healthy adults and what conditions predispose to sleep disorders
- To understand the bidirectional process that drives how sleep disorders can accelerate dementia and how sleep disorders may be the result of cognitive decline.

The Sleep Continuum – Changes Across the Life Cycle

- **Sleep changes dramatically across the life cycle in duration, architecture, & timing**
  - Early in life these changes are driven by the maturation of *two regulatory systems*
    - Circadian Rhythm
    - Homeostatic Sleep Pressure
  - Later in life sleep changes by aging-related decline in *sleep depth* and *continuity*.

- **Changes in *sleep duration* & *timing* are accompanied by changes in sleep structure (sleep architecture)**

Sleep Health

Table 1. Sleep Health Components.

Sleep Health Component	Definitions and Additional Details
Sufficient sleep duration	• Time spent sleeping in a 24-hour period • There are individual differences in sleep need • Sleep duration recommendations vary across the lifespan*
Continuous and efficient	• Sleep efficiency is defined as the time a person spends sleeping as a proportion of the overall time in bed • Higher efficiency scores indicate that sleep is continuous and not fragmented (e.g., by difficulty falling asleep or maintaining sleep)
Restorative	• Self-reported reviews by the sleeper that the prior sleep allowed the sleeper to wake refreshed and restored • Sleep quality can also be measured directly by asking participants to rate their sleep on Likert scales
Appropriately timed	• Sleep times (onset/bedtime and offset/wake time) are consistent across days in a typical week • Ideally, sleep times are aligned with patterns of light and darkness in the external environment

* The recommendations for sleep duration range by age.[22] Infants (4 to 12 months of age) should sleep 12 to 16 hours per 24 hours (including naps), children 1 to 2 years of age should sleep 11 to 14 hours per 24 hours (including naps), and children 3 to 5 years of age should sleep 10 to 13 hours per 24 hours; those 6 to 12 years of age should sleep 9 to 12 hours and those 13 to 18 years of age should sleep 8 to 10 hours per 24 hours on a regular basis.[22] For adults, the recommended duration range is 7 to 9 hours.[9]

REFERENCE: Robbins R, Quam S. (2024) Sleep health. NEJM.

Recommended sleep duration by age

Consensus recommendations from the American Academy of Sleep Medicine (AASM) and National Sleep Foundation:

Age group	Recommended sleep per 24 hours
Newborns (0–3 months)	14–17 hours (wide normal variation; no formal AASM recommendation <4 months)
Infants (4–12 months)	12–16 hours (including naps)
Toddlers (1–2 years)	11–14 hours (including naps)
Preschool (3–5 years)	10–13 hours (including naps)
School-age (6–12 years)	9–12 hours
Teens (13–18 years)	8–10 hours
Adults (18–64 years)	7–9 hours
Older adults (≥65 years)	7–8 hours

>50% Sleep per 24 hours

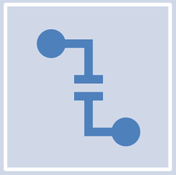
1/3 Sleep in 24 hours

Martin JL, Patel SR, Quan SF, Tasali E. Recommended Amount of Sleep for a Healthy Adult: A Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society. Sleep. 2015 Jun 1;38(6):843-4.

Regulatory Mechanisms



Two processes govern sleep across the lifespan: the *circadian rhythm* (~24 h, mediated chiefly by light via melatonin as the primary driver) and *homeostatic sleep pressure* (accumulating with wakefulness).



Developmental shifts in these *two systems*—rather than a single mechanism—account for the adolescent phase delay and the advance and fragmentation of sleep in later life.

REFERENCE: Skeldon AC, Derks G, Dijk DJ. Modelling changes in sleep timing and duration across the lifespan: Changes in circadian rhythmicity or sleep homeostasis? Sleep Med Rev. 2016 Aug;28:96-107.

Sleep Structure (Phases)

Sleep is divided into two fundamental states

1. **Non-REM (NREM) sleep** — **~75-80% of total sleep time in adults**
2. **REM (rapid eye movement) sleep** — **~20-25% of total sleep time in adults**

Alternate cyclically throughout the night in roughly 90–120 minute cycles

- 4–6 cycles per night.

Sleep Structure (Phases)

Non-REM Sleep is divided into three stages

[American Academy of Sleep Medicine]

- **N1 (light sleep / transition):** The transition from wakefulness to sleep - slow rolling eye movements and a shift to theta activity; the lightest stage with the lowest arousal threshold. **~5% of sleep.**
- **N2 (light sleep):** Characterized by sleep spindles and K-complexes on EEG. This is the largest single component, occupying **~50% of total sleep time.**
- **N3 (deep / slow-wave sleep):** The deepest stage, dominated by high-amplitude delta (slow-wave) activity. Important for cerebral restoration, memory consolidation, and metabolic regulation. **~20% of sleep in healthy young adults**

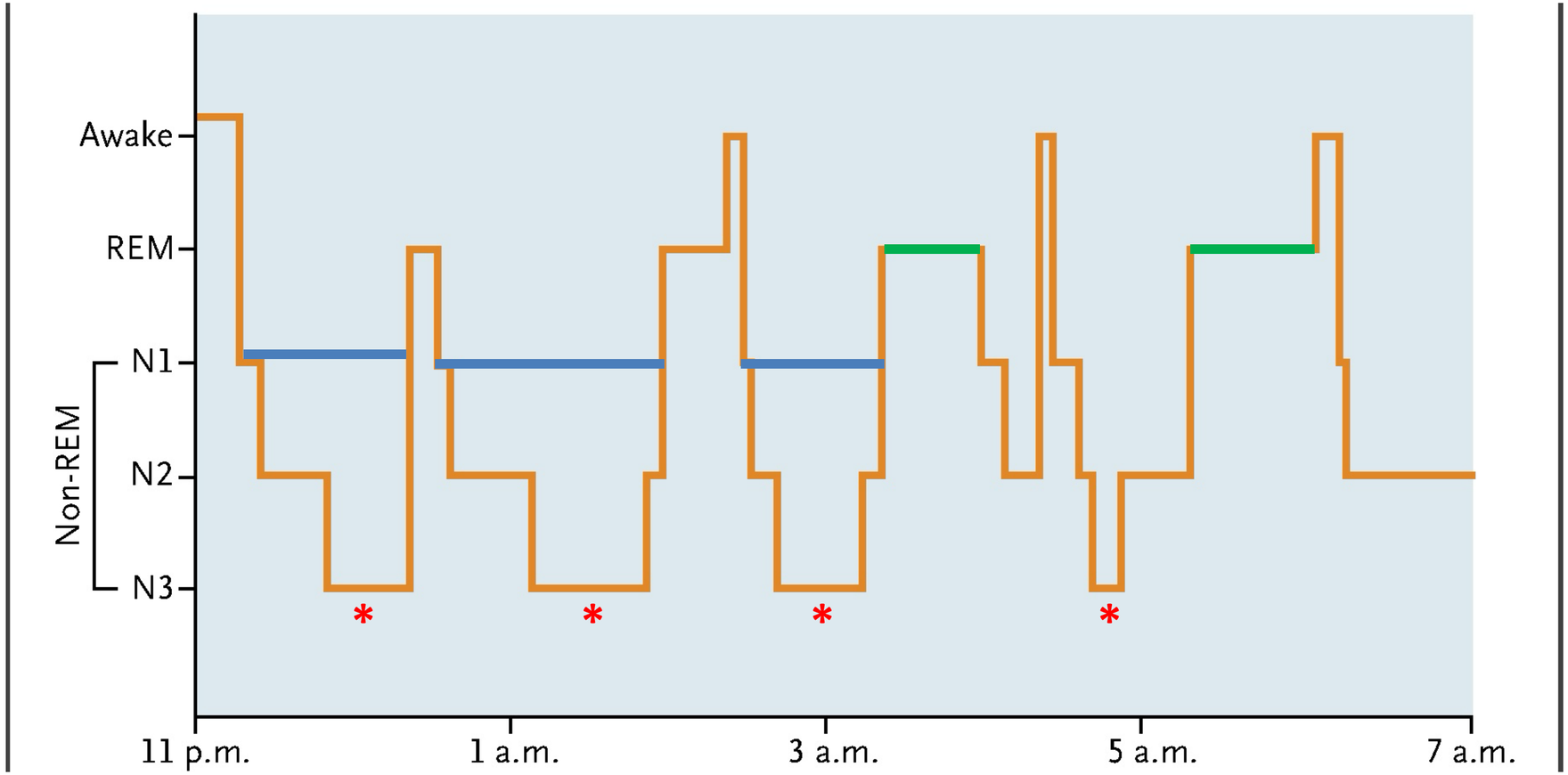
(*Older Rechtschaffen & Kales criteria split this into stages 3 and 4, which the AASM merged into N3.)*

REM (rapid eye movement) sleep (stage R)

- Features desynchronized, wake-like brain activity with theta waves, skeletal muscle atonia, bursts of rapid eye movements, and autonomic activation (increased heart rate, respiration, and blood pressure).
- This is when most structured, story-like dreaming occurs.

Key architectural features:

- **Temporal distribution:** N3 (deep sleep – slow wave sleep) predominates in the first third to half of the night, whereas REM periods lengthen and become more frequent toward morning.
- **Life-span changes:** Neonates spend a much larger proportion of sleep in REM (or its analog, "active sleep"); with aging, sleep becomes more fragmented with reductions in N3 and REM.



The hypnogram illustrates how a healthy young adult cycles through these stages across a single night, showing the early dominance of deep N3 sleep and the lengthening REM periods toward morning.

Sleep architecture undergoes profound, predictable changes from infancy to old age, with a progressive *decline in REM sleep proportion during early development* and a *steady, lifelong erosion of N3 (slow-wave, deep) sleep*

The most dramatic slow-wave losses occurring between young adulthood and midlife.

The classic pattern:

- REM proportion falls sharply from neonatal life through childhood
- Slow-wave sleep (N3) peaks in childhood and then declines continuously
- In later life there is lighter N1/N2 sleep & increased fragmentation.

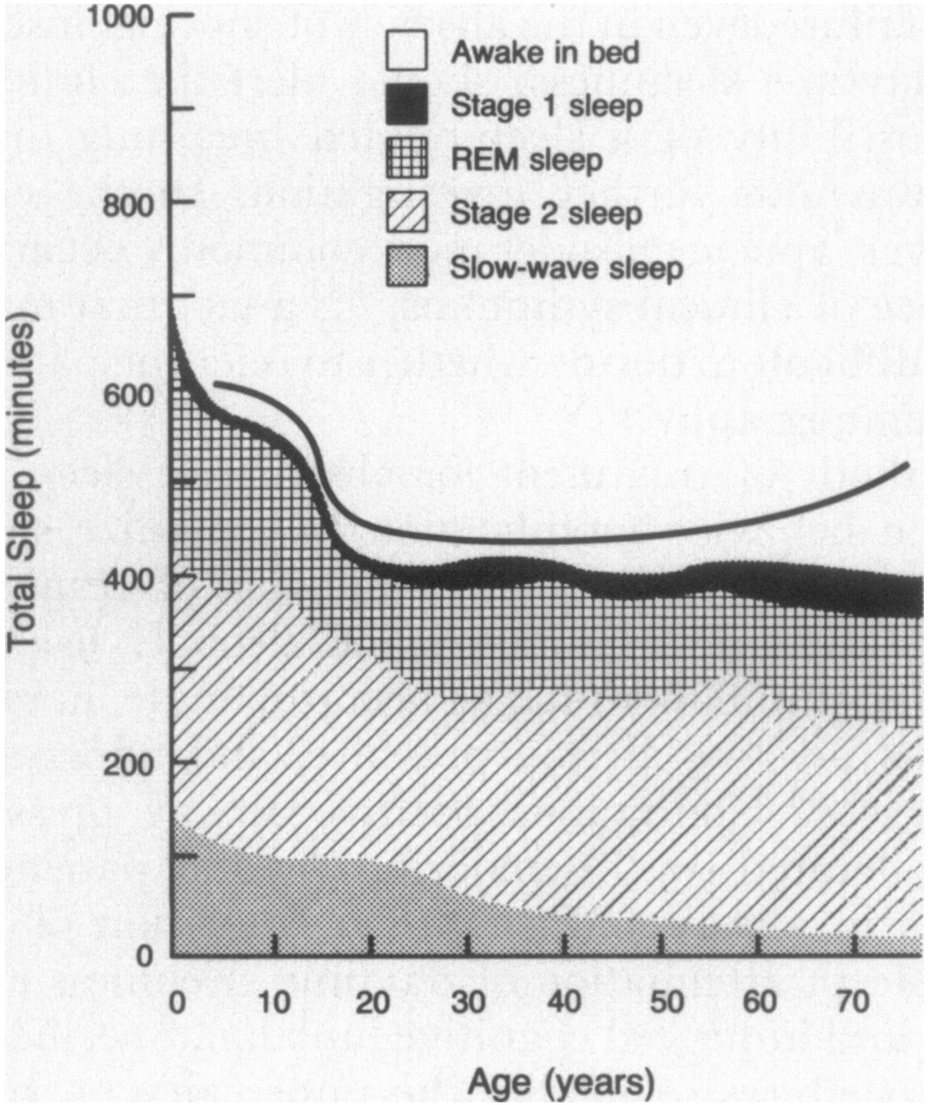


FIGURE: spanning birth to old age, integrates these trajectories—showing the steep early fall in REM and slow-wave sleep and the widening band of nighttime wakefulness in older adults.

Sleep in Older adults

A second phase of deterioration emerges after ~age 50, dominated by fragmentation: *increased wake-time after sleep onset, more nighttime awakenings, lighter sleep, earlier sleep timing, and reduced sleep efficiency*. REM declines more modestly. Sleep efficiency continues to decline significantly after age 60; other parameters largely plateau.

N3 (deep – *slow wave* - sleep) diminishes steadily and may be nearly absent in the very old. There is *compensatory increase in N2 (light sleep)*.

In a community cohort of adults aged 60–87, *slow-wave sleep decreased* by ~0.57% per year, with N1 and N2 increasing slightly. REM percentage remained relatively stable.

Reduced slow-wave (N3) activity (lower slow-wave density, amplitude, and frequency) reflects a *genuine age-related decline* in the capacity to generate slow waves, not merely reduced EEG amplitude.

Sleep Architecture changes with Age

- **Reduced slow-wave sleep (N3) and delta power** — the most striking lifespan change. Declines are already detectable in middle age and become prominent in older adults. Loss of sleep spindles and slow waves is linked to cortical thinning and white-matter degeneration.
- **Decreased REM sleep & shorter REM latency** reductions occur with age, though some meta-analytic data show the REM decrease to be non-significant once confounders are controlled.
- **Increased Stage 1 & 2 (lighter) sleep**, with a corresponding shift away from restorative deep sleep.
- **Lower sleep efficiency & reduced total sleep time**, with increased wake after sleep onset (WASO) — objectively confirmed on polysomnography, where total sleep time and sleep efficiency fall while WASO and sleep onset latency rise with age. WASO (fragmentation) rather than difficulty initiating sleep is the dominant driver of poor efficiency, as sleep latency changes only minimally in healthy aging.

Circadian & Sleep-wake changes with Age



Phase advance: a tendency to fall asleep and wake earlier, progressing roughly 15–20 minutes earlier per decade after age 20.



Reduced circadian amplitude (dampened rhythms in core temperature, melatonin, cortisol) and **decreased tolerance for phase shifts** (e.g., jet lag, shift work)



Weakening of both the homeostatic sleep drive and the central clock, with suprachiasmatic nucleus dysfunction, reduced retinal light transmission (lens pigmentation, retinal ganglion cell loss), and neurotransmitter dysregulation (galanin, orexin, serotonin, adenosine).



Increased daytime napping and sleepiness: daytime naps rise from ~10% of adults aged 55–64 to ~25% of those aged 75–84, often unplanned and reflecting fragmented nocturnal sleep.

Mechanism of Sleep-Wake changes with Age

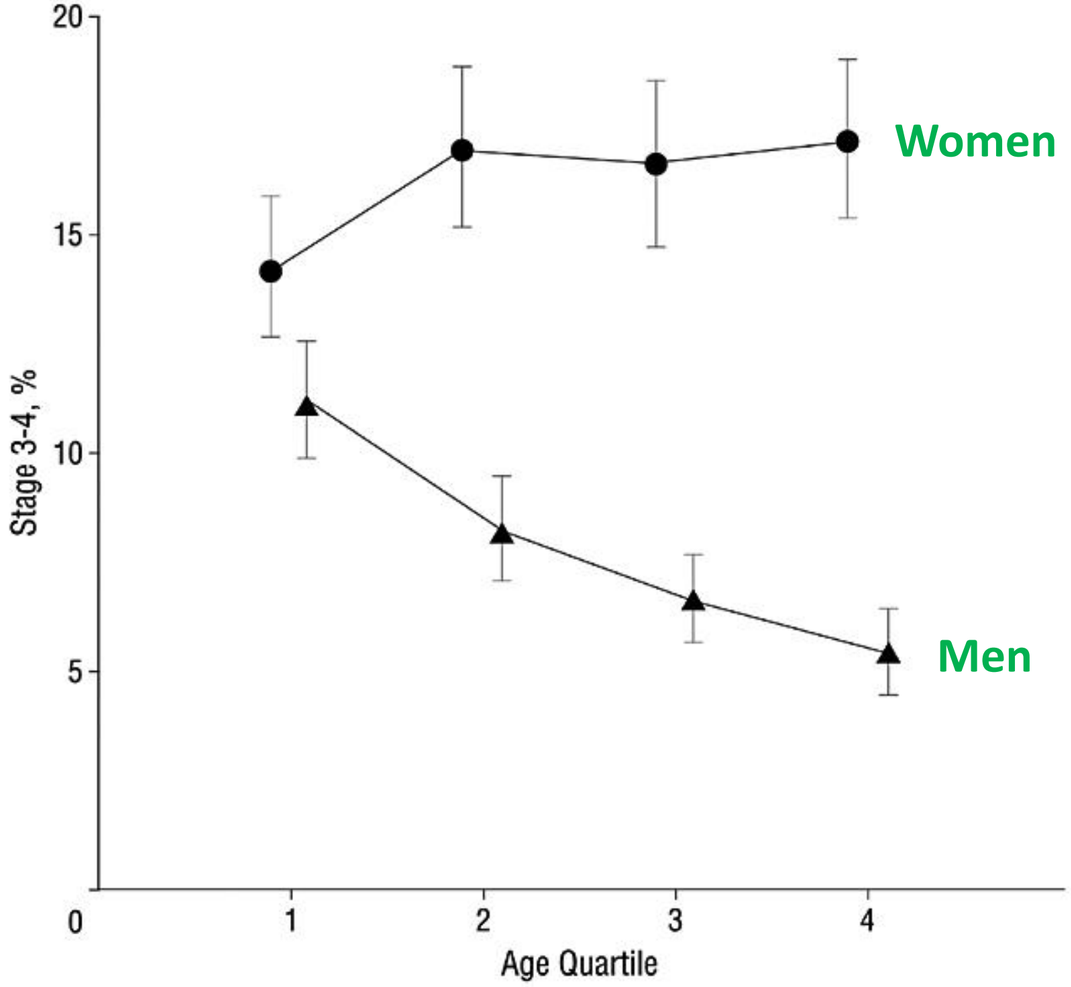
- **Mechanisms** underlying these changes include
  - cortical thinning from neurotoxin deposition
  - white-matter degeneration from vascular change
  - neurotransmitter dysregulation
  - circadian disorganization
- **There is a bidirectional relationship between aging & sleep disruption.**
- **Important caveat:** normal age-related changes must be distinguished from pathological sleep disorders, which are common in older adults
  - Insomnia (20–40%), obstructive sleep apnea (>30%), restless legs syndrome, and REM sleep behavior disorder
  - Sleep disorders carry elevated risks of cognitive decline, cardiovascular disease, falls, frailty, and mortality.

REFERENCE: Zhong HH, Yu B, Luo D, Yang LY, Zhang J, Jiang SS, Hu SJ, Luo YY, Yang MW, Hong FF, Yang SL. Roles of aging in sleep. Neurosci Biobehav Rev. 2019 Mar;98:177-184.

Sex Differences in Sleep

A notable modifier is sex: the age-related decline in slow-wave sleep (N3) is substantially more pronounced in men.

- In one large cohort, women maintained relatively stable slow-wave sleep (~14–17%) across the adult life span
- In contrast men declined from ~11% to ~5%, with women averaging ~106% more slow-wave sleep than men overall.



Association between percentage stage 3-4 and age in men & women. Adjusted (least squares mean) levels of percentage stage 3-4 at each age quartile (54, 54 to 61, 61 to 70, and 70 years) for men (triangles) and women (circles).

Redline S, Kirchner HL, Quan SF, Gottlieb DJ, Kapur V, Newman A. The Effects of Age, Sex, Ethnicity, and Sleep-Disordered Breathing on Sleep Architecture. *Arch Intern Med.* 2004;164(4):406–418.

Sleep and Alzheimer's Disease

Research suggests sleep and dementia may share a bidirectional relationship.

Damaged brain cells can cause chronic inflammation and result in poor or restless sleep

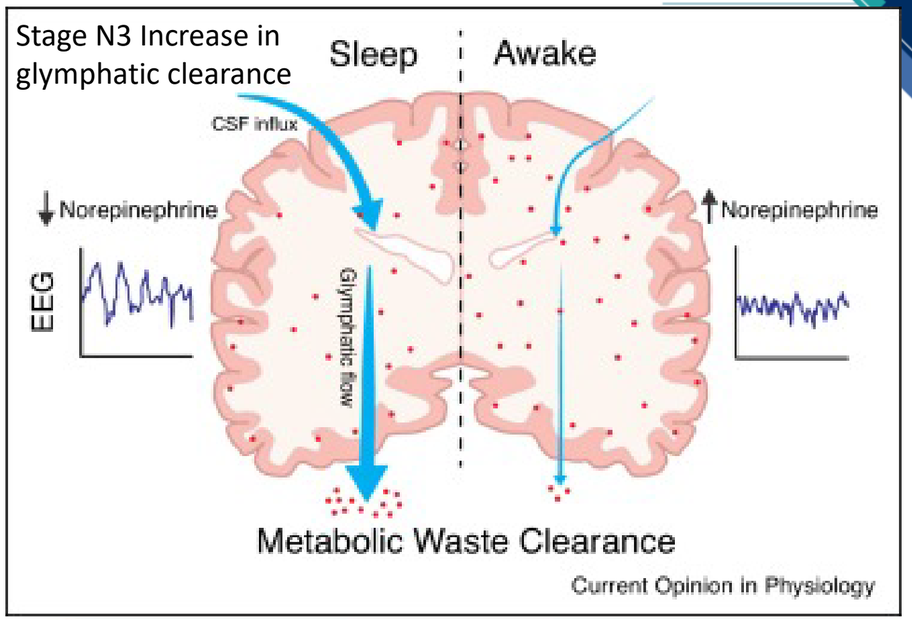
Alzheimer's disease causes excess beta-amyloid proteins to clump and build up plaque in the brain

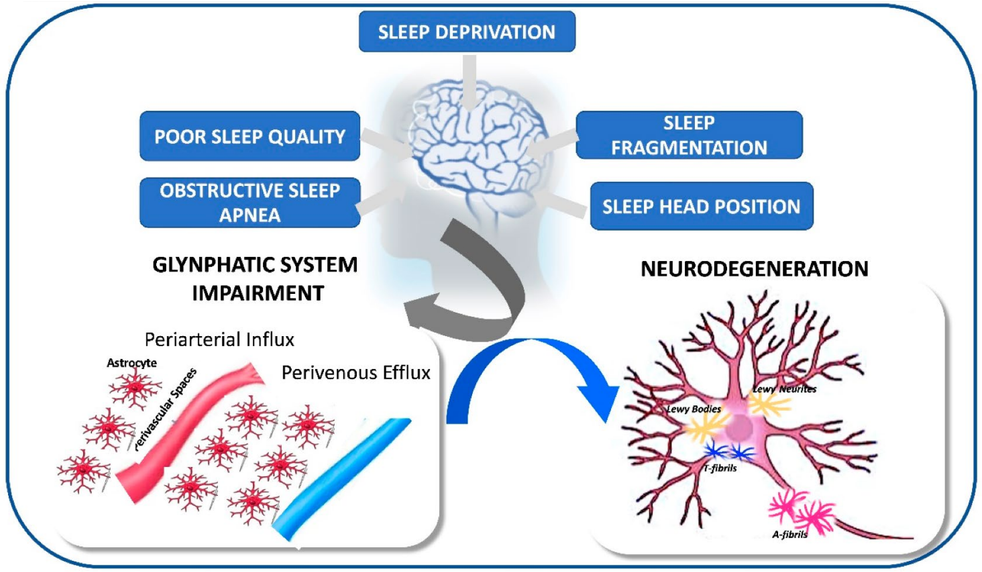
Sleep deprivation prevents the cleanup of toxic proteins that would normally be removed during sleep

Plaque damages the neurons involved in memory formation leading to problems with speech, behavior, and logic

Sleep deprivation can increase the risk of Alzheimer's disease, the most common type of dementia. People with Alzheimer's experience changes to their sleep-wake cycle and spend less time in deep sleep, worsening their symptoms the next day.

Glymphatic system is a "pseudo-lymphatic" perivascular network responsible for replenishing & cleansing the brain





Schematic of the mechanisms linking sleep disorders, glymphatic system activity & neurodegeneration.

REFERENCES: Ferini-Strambi L, Salsone M. "Glymphatic" Neurodegeneration: Is Sleep the Missing Key? *Clinical and Translational Neuroscience*. 2024; 8(2):23
Hauglund NT, Pavan C, Nedergaard M. (2020) Cleaning the sleeping brain – the potential restorative function of the glymphatic system. *Current Opinion in Physiology. 15: 1-6.*

Siow TY, Toh CH, Hsu JL, et. al. (2022) Association of Sleep, Neuropsychological Performance, and Gray Matter Volume With Glymphatic Function in Community-Dwelling Older Adults. *Neurology*. 98:e829-e838.

Abstract

Background and objectives: The glymphatic system, which is robustly enabled during some stages of sleep, is a fluid-transport pathway that clears cerebral waste products. Most contemporary knowledge regarding the glymphatic system is inferred from rodent experiments and human research is limited. Our objective is to explore the associations between human glymphatic function, sleep, neuropsychological performance, and cerebral gray matter volumes.

Methods: This cross-sectional study included individuals 60 years or older who had participated in the Integrating Systemic Data of Geriatric Medicine to Explore the Solution for Health Aging study between September 2019 and October 2020. Community-dwelling older adults were enrolled at 2 different sites. Participants with dementia, major depressive disorders, and other major organ system abnormalities were excluded. Sleep **profile** was accessed using questionnaires and polysomnography. Administered neuropsychological test batteries included Everyday Cognition (ECog) and the Consortium to Establish a Registry for Alzheimer's Disease Neuropsychological Battery (CERAD-NB). Gray matter volumes were estimated based on MRI. Diffusion tensor imaging analysis along the perivascular space (DTI-ALPS) index was used as the MRI marker of glymphatic function.

Results: A total of 84 participants (mean [SD] age 73.3 [7.1] years, 47 [56.0%] women) were analyzed. Multivariate linear regression model determined that **age** (unstandardized β, -0.0025 [SE 0.0001]; $p = 0.02$), **N2 sleep duration** (unstandardized β, 0.0002 [SE 0.0001]; $p = 0.04$), and the **apnea-hypopnea index** (unstandardized β, -0.0011 [SE 0.0005]; $p = 0.03$) were **independently associated with DTI-ALPS**. Higher DTI-ALPS was associated with better ECog language scores (unstandardized β, -0.59 [SE 0.28]; $p = 0.04$) and better CERAD-NB word list learning delayed recall subtest scores (unstandardized β, 6.17 [SE 2.31]; $p = 0.009$) after covarying for age and education. Higher DTI-ALPS was also associated with higher gray matter volume (unstandardized β, 107.00 [SE 43.65]; $p = 0.02$) after controlling for age, sex, and total intracranial volume.

Discussion: **Significant associations were identified between glymphatic function and sleep, stressing the importance of sleep for brain health**. This study also revealed associations between DTI-ALPS, neuropsychological performance, and cerebral gray matter volumes, suggesting the potential of DTI-ALPS as a biomarker for cognitive disorders.

Changes in Sleep in People with Cognitive Decline

- **Dementia often changes sleep patterns, impacting both individuals with dementia and their caregivers.**
- **People with dementia may experience increased daytime sleepiness.**
- **Loss of brain function disrupts the regulation of sleep-wake cycles.**
- **Untreated sleep disorders are strongly associated with accelerated cognitive decline.**

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Review

The Sleeping Brain: Harnessing the Power of the Glymphatic System through Lifestyle Choices

Oliver Cameron Reddy and Ysbrand D. van der Werf *

- **A complex cascade of neurotransmitters & hormones are involved in sleep regulation.**
  - These neurochemicals are secreted & synthesized by structures in the brainstem and hypothalamus.
  - These brain regions & networks are disrupted in Alzheimer's disease
- **Impaired glymphatic clearance has been linked to neurodegeneration – at least in part due to the *slowed clearance of amyloid-beta & tau proteins*.**
  - Glymphatic system may modulate and/or be protective against Alzheimer's disease.
  - Sleep is the primary driver of the glymphatic system.
    - Glymphatic clearance occurs mostly during Slow Wave Sleep(N3 – deep sleep) which occurs earlier in the night.
    - The amount of slow wave sleep decreases with age.
- **CSF produced daily (~500 cc) – deeper in the brain CSF movement facilitated by aquaporin 4 (AQP4)**

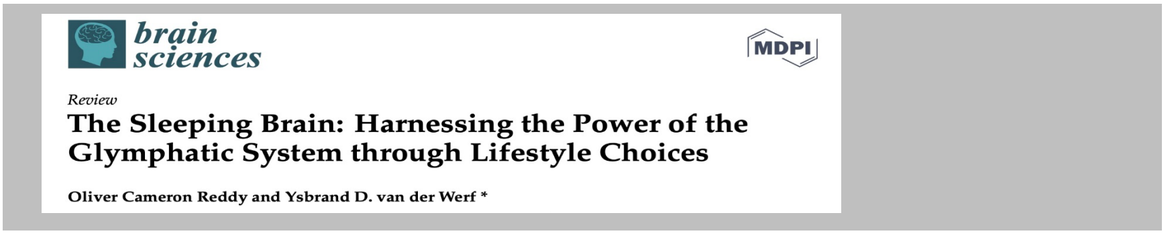


Review

The Sleeping Brain: Harnessing the Power of the Glymphatic System through Lifestyle Choices

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- **APOE4** – associated with sleep apnea, progression of sleep disturbances, cognitive performance and sleep deterioration (Dauvilliers, Y. Insomnia in patients with neurodegenerative conditions. Sleep Med. 2007, 8, S27–S34)
  - APOE4 is a genetic link between sleep & neurodegeneration
- **Slow Wave Sleep & Age:** Gradual decrease in slow wave sleep (N3) with age is associated with reduced glymphatic clearance and increased risk of toxic proteins (amyloid-beta, tau) --- neurodegeneration.
- **Aquaporin 4 (AQP4):** Glymphatic clearance is impaired in a rodent animal model of Alzheimer's disease, due to changes in the number of AQP4 water channels responsible for the movement of CSF and ISF, expressed on astrocytic endfeet [Rasmussen, M.K.; Mestre, H.; Nedergaard, M. The glymphatic pathway in neurological disorders. Lancet Neurol. 2018, 17, 1016–1024).



Lifestyle Choices & Sleep

- **Omega-3 Consumption**
  - Marine-based fish oils known as omega-3 polyunsaturated fatty acids (n3-PUFAs) h
  - The central nervous system is highly enriched with n3-PUFAs that exhibit potent anti-inflammatory properties
  - Dietary intake of n3-PUFAs improved cognitive decline in mild Alzheimer's disease by improving glymphatic transport thus reducing the chance of amyloid-related injury
- **Intermittent fasting:** downregulates the expression of AQP4-M1, decreasing the AQP4-M1/AQP4-M23 ratio, thus increasing AQP4 polarization along the paravenous space to boost glymphatic clearance
- **Sleeping position:** gravity affects movement of blood & CSF through the brain. Glymphatic clearance is most efficient in the right lateral sleeping position.

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The Sleeping Brain: Harnessing the Power of the Glymphatic System through Lifestyle Choices

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Lifestyle Choices & Sleep

- **Alcohol consumption**
  - Alcohol (ETOH) can boost or hinder glymphatic clearance depending on the dosage and whether consumption is chronic or acute.
  - Prolonged excessive ETOH consumption has adverse effects on the CNS including increased risk of developing Alzheimer's disease and other dementias.
  - Intermediate & heavy use of ETOH can decrease glymphatic clearance - however, after 24-hours of sobriety glymphatic function can be restored.
  - Acute or chronic low dose of ETOH can increase glymphatic clearance, reducing the risk of AD.
- **Exercise:** glymphatic flow increases with exercise; AQP4 expression is increased with exercise
- **Chronic Stress**
  - Chronic stress accelerates the accumulation and deposition of amyloid-beta
  - Mice exposed to stress exhibited decreased glymphatic influx and efflux, loss of AQP4 polarization and a reduction in AQP4-bearing astrocytes; Stress can increase cortisol that downregulates AQP4 channels

Common Sleep Disorders in People with Cognitive Decline

- **Restless legs syndrome (RLS)**: RLS is characterized by an overwhelming desire to move the legs, especially at night. RLS is common in people with Lewy body dementia.
- **Periodic limb movement disorder (PLMD)**: PLMD causes uncontrollable movements of the arms and/or legs at night. Many patients with PLMD also have RLS.
- **Obstructive Sleep Apnea (OSA)** is a condition marked by nighttime airway collapse leading to brief lapses in breathing (apnea). OSA is particularly common with Alzheimer's disease, occurring in 40% of patients. Having OSA also increases one's risk of developing dementia.
- **REM sleep behavior disorder**: REM sleep behavior disorder causes individuals to act out their dreams, sometimes in dangerous ways. It is most often found in individuals with Lewy body dementia. This sleep disorder can be the first symptom and may develop before cognitive symptoms.
- **Depression**: Depression is commonly associated with insomnia and other sleep disturbances. Depression is common in people with cognitive decline. There is considerable evidence that many people with preclinical and early phase Alzheimer's disease are depressed.

SUMMARY - Sleep through the Lifecycle: Reducing Risk of Cognitive Decline

- **Duration, quality, and sleep phases change with age:** Most notably older adults are more likely to have fragmented sleep with reduced deep (slow-wave) N3 sleep.
- **Glymphatic clearance:** occurs during slow wave sleep – so there is a natural risk for reduced glymphatic clearance with age. Glymphatic clearance is critical to filter out toxin proteins that accumulate during wakefulness.
- **There is a bidirectional relationship between sleep & dementia.** Multiple factors impact sleep in people with cognitive decline.
- **Lifestyle choices can impact sleep.** Modifications including diet, exercise and limiting alcohol use can positively impact sleep quality with age.
- **Sleep disorders/disruption is/are common with age.** Underlying causes of disorders sleep should be diagnosed and treated.

Resource Links

- **National Sleep Foundation** https://www.thensf.org/

  Access sleep duration recommendations, public awareness campaigns, and research updates.

- **American Academy of Sleep Medicine (AASM)**: Find patient guides, a sleep center directory, and tools like a bedtime calculator and sleep diary. https://aasm.org/

- **NIH Guide to Healthy Sleep** https://www.nhlbi.nih.gov/resources/your-guide-healthy-sleep

- **REFERENCES**

  - Robbins R, Quan S. Sleep Health. NEJM Evid. 2024 Aug;3(8):EVIDra2300269. doi: 10.1056/EVIDra2300269. Epub 2024 Jul 23. PMID: 39041870.
  - Reddy OC, van der Werf YD. The Sleeping Brain: Harnessing the Power of the Glymphatic System through Lifestyle Choices. *Brain Sci.* 2020 Nov 17;10(11):868. doi: 10.3390/brainsci10110868. PMID: 33212927; PMCID: PMC7698404.

Questions?

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