Dementia as a failure of nightly closure: a hypothesis derived from a theory of loops, with exploratory tests on open EEG and sleep data

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Highlights

- Dementia is framed as a failure of the nightly closure of the day's open loops.
- Closure is derived exactly in a ternary lattice; about 31 % of points never close.
- In open EEG data the alpha peak is absent in 11 of 36 Alzheimer patients.
- Sleep cycles without deep sleep rise from 15 % below 40 to 47 % above 80.
- Stimulation that follows the brain's own rhythm fits better than a fixed tone.

Abstract

Late-onset dementia is proposed to be a slow failure of nightly closure. The idea comes from a theory of loops in which a loop persists only if it closes, and what does not close is carried forward. Closure is first derived exactly in a simple number system on the triangular lattice. There about 31 % of points never close, and their share swings with each threefold change of scale. In the brain, waking activity opens loops and deep sleep closes them. When deep sleep thins, a remainder is carried into the next day. Over years the remainder becomes the disease. The primary cause is therefore lack of deep sleep. Other risk factors act through one chain of input, rhythm and removal. Published evidence fits this view. Amyloid-β is made at a normal rate in late-onset Alzheimer's disease but cleared about 30 % more slowly. Clearance runs mainly in deep sleep. Loss of sleep raises amyloid and predicts dementia. Exploratory tests on open data point the same way. The alpha peak was absent in 11 of 36 people with Alzheimer's disease against 1 of 29 controls. In healthy sleepers the share of cycles without deep sleep rose from 15 % below age 40 to 47 % above 80. The hypothesis predicts that stimulation which follows the brain's own slow rhythm helps more than a fixed imposed frequency. Five tests are proposed.

Keywords: dementia; Alzheimer's disease; slow-wave sleep; glymphatic clearance; closure; balanced ternary; phase-locked stimulation

1. Introduction

Dementia affects tens of millions of people, and their number grows each year. The 2024 Lancet Commission lists fourteen modifiable risk factors. Together they would account for about 45 % of cases [1]. The list runs from little schooling in childhood to hearing loss, high LDL cholesterol, untreated vision loss and social isolation in later life.

The list is useful, but it is a list. It does not say why such different factors lead to one disease. It also leaves something out. Sleep is not among the fourteen, although a large body of work links it to the removal of the proteins that build up in Alzheimer's disease [5,6,10].

This article proposes one mechanism behind the list. It starts from a theory of loops and derives its central idea, closure, in a system where closure can be computed exactly (Section 2). It then states the hypothesis (Section 3), sets it against published evidence (Section 4), describes the methods of two exploratory tests on open data (Section 5), reports their results, including tests that failed (Section 6), reads recent trials as natural tests (Section 7) and lists predictions that can prove the hypothesis wrong (Section 8). No earlier knowledge of the theory is assumed.

2. Closure: from a theory of loops to an exact computation

2.1 Starting point

The theory used here starts from movement, not from objects [2]. Nothing is assumed except a single strand that moves. Each step of the strand can go forward, stand still or go back. These three options are written +1, 0 and −1, a *trit*. A sequence of trits is a path. A path that returns to where it started is a *loop*.

Five rules fix how the strand behaves [2]. They are stated here as the theory states them.

- **N1.** There is exactly one strand.
- **N2 (closure).** A loop persists only if it closes. What does not close passes on as tension.
- **N3.** The first passage through a point fixes a direction.
- **N4.** The response of the medium depends on its state.
- **N5.** Coarse blocks of the strand obey the same rules again.

Rule N2 is the one this article needs. It says that there are two kinds of outcome. A loop either closes and stays, or it does not close and its remainder is carried on. Rule N5 says that the same holds at every scale: in a molecule, a cell, a rhythm, a night and a life. Without a concrete system, however, N2 is only a sentence. The next three subsections build such a system from trits alone and compute exactly which loops close.

2.2 A system in which closure can be computed

Let ω be a cube root of one other than 1:

$$\omega = (-1 + i\sqrt{3})/2, \quad \text{so that} \quad \omega^2 + \omega + 1 = 0.$$

The numbers $z = a + b\omega$, with a and b whole numbers, form a triangular lattice in the plane. The squared distance of z from 0 is its norm, $N(z) = a^2 - ab + b^2$. Take as base $\beta = 1 - \omega$. Its length is $\sqrt{3}$. Multiplying by β stretches the plane by $\sqrt{3}$ and turns it by −30°. One also has $3 = -\omega^2\beta^2$. So one step of the ladder ×3 equals two steps of β, up to a turn.

Now let the strand unwind one trit at a time. For a point $z = a + b\omega$, choose the trit d in {−1, 0, +1} with $d \equiv a + b \pmod 3$. Then $z - d$ is divisible by β, and the step is

$$T(z) = (z - d)/\beta. \quad \text{In coordinates, with } x = a - d \text{ and } y = b: \quad T(z) = ((2x - y) + (x + y)\omega)/3.$$

Each step removes one trit and brings the point closer to 0. A point *closes* when repeated steps reach 0: all its trits have been used up and the path has returned. A point that never reaches 0 does not close.

An example shows both outcomes. The point 2 has digit −1, because $2 \equiv -1 \pmod 3$. So $T(2) = 3/\beta = 2 + \omega$. This point has digit 0, and $T(2 + \omega) = 1 + \omega$. Then $1 + \omega$ has digit −1, and T(1 +

ω) = 1 + ω again. The orbit is caught. It turns forever. By contrast T(1) = 0 at once: the point 1 closes in one step.

2.3 Proposition 1: every point ends in one of three places

Proposition 1. Every orbit of T ends either in 0 or in one of the two fixed points +(1 + ω) and −(1 + ω). The two kinds of non-closing points are equal in number.

Proof. Since $|d| \le 1$ and $|\beta| = \sqrt{3}$, each step gives $|T(z)| \le (|z| + 1)/\sqrt{3}$. This is smaller than $|z|$ whenever $|z| > 1/(\sqrt{3} - 1) \approx 1.37$. So every orbit enters the disc of radius 1.37 and cannot leave it. The only lattice points in that disc are 0 and the six units $\pm 1, \pm\omega, \pm\omega^2$. Direct computation gives $T(\pm 1) = 0$, $T(\omega) = -1$ and $T(-\omega) = 1$, and hence 0 one step later, and $T(\pm(1 + \omega)) = \pm(1 + \omega)$. Note that $\omega^2 = -(1 + \omega)$, so the units $\pm\omega^2$ are the two fixed points. Finally, replacing z by −z changes the sign of every digit and of every step. This maps the orbits ending in +(1 + ω) one to one onto those ending in −(1 + ω). This completes the proof.

The proof uses nothing but the step rule. It was also checked by computer for all lattice points of norm up to 22,500 [21].

2.4 Proposition 2: the share that does not close never settles

Let h(N) be the share of lattice points with norm at most N that never close.

Proposition 2. As N grows, h(N) has no limit. It swings between about 29.6 % and 32.0 %, and its course repeats exactly each time N grows by a factor 3. Its mean is 30.87 %.

Outline of the proof. The set of points that close is built from 0 by the rule 'multiply by β and add a trit'. So this set, enlarged by β, is again the same set. Its share of a large disc therefore depends only on the radius modulo a factor $\sqrt{3}$, that is, on the norm modulo a factor 3. A share with this property is either constant or periodic in the logarithm of the scale. Exact counts at two radii, with certified error bounds, show that it is not constant. The full proof is given in [3].

For this article the values were recomputed independently from the step rule alone [21]. Over two full periods, from norm 3^7 to 3^9, the share ranged from 29.6 % to 32.0 % with a mean of 30.9 %. At norms 3^5, 3^6, 3^7 and 3^8 it took the same phase value each time within 0.3 percentage points, as the proposition requires. Fig. 1 shows the two outcomes in the plane and the swing of the share.

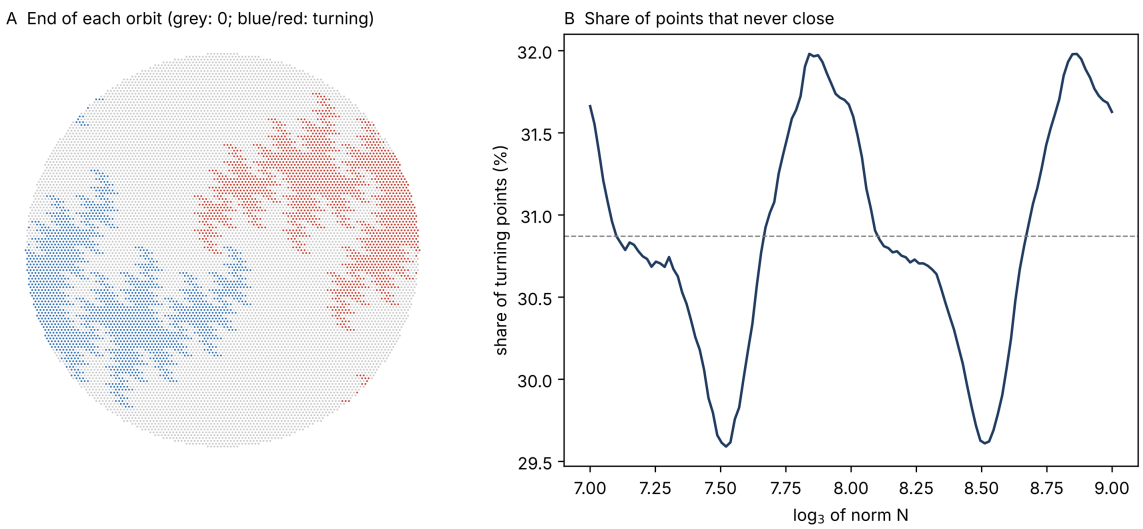


Fig. 1. Closure in the triangular lattice. (A) Lattice points of norm up to 3,600, coloured by the end of their orbit under T: grey points close (reach 0); blue and red points turn forever around $+(1 + \omega)$ and $-(1 + \omega)$. (B) Share of points that never close, h(N), against $\log_3 N$ over two periods. The dashed line marks the mean, 30.87 %.

2.5 What carries over to the body

Propositions 1 and 2 are mathematics. What follows is a reading. It is offered as a frame and must earn its place through the predictions in Section 8. It does not claim that the lattice exists in the brain.

Three lessons carry over. First, even a perfectly ordered system leaves a fixed part that never closes. The relevant question is not whether a remainder exists but whether something lets it close after all. Second, the remainder is not lost. It is carried on, exactly as N2 states. Third, the amount that fails to close is not a constant. It changes with scale.

A recent essay asks the question directly: do the points that never close have a 'sleep', a rule that lets them close [20]? This article proposes that in the body the answer is literal. Sleep is that rule.

Three terms then have a physical meaning. A **loop** is any process that must return to its start to be complete: a metabolic cycle, an exchange of fluid, the consolidation of a memory, a rhythm. **Closure** is the moment the loop returns. The **remainder** is what is left when a loop does not return in time. In the brain the remainder has a material form. Amyloid-β is a normal product of active neurons. When it is not removed it stays and builds up.

3. The hypothesis

Late-onset dementia is a slow failure of nightly closure. Its primary cause is lack of deep sleep.

Waking activity opens loops. Neurons fire, make proteins and leave waste. Deep sleep closes these loops. In slow-wave sleep the slow brain rhythm drives waves of blood and fluid that wash the brain [6]. When deep sleep is enough, each day is closed by the following night. When it is not, part of the day remains. The next day adds its own load. Over years the remainder becomes plaques, tangles and lost function.

The other risk factors act through one chain of three links.

1. **Input.** The senses keep the brain's loops driven and timed. Hearing and vision loss weaken this drive. The Lancet Commission assigns them 7 % and 2 % of cases [1].
2. **Rhythm.** The brain's rhythms are its loops. Their bands form an even ladder on a logarithmic scale [13]. A rhythm that no longer closes first weakens and then vanishes.
3. **Removal.** Clearance runs mainly in deep sleep. High blood pressure and high LDL cholesterol weaken the vascular pulsing that drives the fluid. Depression, alcohol and isolation disturb sleep.

Three consequences follow.

- **C1.** Disease should show as rhythms that disappear, not only as rhythms that shift.
- **C2.** The share of sleep cycles that do not reach deep sleep should rise with age and with risk.
- **C3.** Help should work best when it follows the brain's own rhythm and lets the loop close. A rhythm imposed from outside should work less well.

The hypothesis concerns the common late-onset form. Rare genetic forms with overproduction of amyloid lie outside it.

4. Evaluation against published evidence

4.1 The remainder is a removal problem

The most direct evidence comes from metabolic labelling in people. In late-onset Alzheimer's disease amyloid-β is produced at the normal rate, and its clearance is about 30 % slower [4]. The authors estimate that this deficit builds the observed load in roughly ten years. In the terms of Section 2, the loop opens normally but does not close in time.

4.2 Removal runs in sleep

In mice, sleep enlarges the space between brain cells by about 60 %. Fluid then flows through the tissue and amyloid-β is removed faster [5]. In people, the slow waves of deep sleep are followed by oscillations of blood volume, which are coupled to waves of fluid flow [6]. The slow rhythm and the washing are one process. The effect is fast. One night without sleep raised amyloid in the hippocampus and thalamus of healthy adults [7].

4.3 Lack of sleep predicts dementia

In the Whitehall II cohort, sleeping six hours or less at age 50 and 60 went with a higher risk of dementia decades later [8]. In the Framingham Heart Study, each percent of deep sleep lost per year after age 60 went with a 27 % higher risk [9].

4.4 The direction of cause

Sleep and Alzheimer pathology feed each other [10]. Amyloid disturbs sleep, and poor sleep raises amyloid. The association alone cannot show which comes first. Two facts favour sleep as the starting point for the common form. Short sleep predicts dementia more than 25 years ahead, long before pathology is expected [8]. One night of lost sleep raises amyloid in healthy people [7]. Once the cycle runs, each turn makes the next worse. This is what N2 describes: what does not close is carried on.

5. Methods of the exploratory tests

5.1 Data

Two public datasets were used. The first, OpenNeuro ds004504, holds eyes-closed resting EEG from 19 scalp electrodes at 500 Hz of 36 people with Alzheimer's disease, 23 with frontotemporal dementia and 29 healthy controls, each with a Mini-Mental State Examination score [11]. The preprocessed recordings provided by the dataset authors were used without further cleaning.

The second, the Sleep Cassette part of Sleep-EDF Expanded on PhysioNet, holds full-night recordings of 78 healthy people aged 25 to 101 [12]. The EEG channel Fpz–Cz, sampled at 100 Hz, was used. Sleep stages had been scored by experts in 30-second epochs. Stages 3 and 4 were merged as deep sleep (N3). For each person the first available night was analysed. A second night was used only to check stability.

5.2 Alpha rhythm

For each person the power spectrum of the five posterior electrodes (O1, O2, P3, P4, Pz) was estimated with Welch's method, using 4-second windows with 50 % overlap, and averaged. A straight line was fitted to log power against log frequency between 1 and 45 Hz, leaving out 4–14 Hz so that a peak would not bend the line. The residual above this line is the periodic part. The alpha peak was taken as the highest local maximum of the residual between 4 and 14 Hz that exceeded 0.1 $\log_{10}$ units. If no such maximum existed, the person was recorded as having no alpha peak. Groups were compared with a one-sided Fisher exact test (presence of a peak), a Mann–Whitney test (peak frequency) and Levene's test (spread).

5.3 Closure of sleep cycles

Within the sleep period, from the first to the last sleep epoch, the onsets of REM sleep were located. REM epochs less than 15 minutes apart were merged into one REM period. A sleep cycle was taken as the stretch from sleep onset to the first REM onset, and from each REM onset to the next. A cycle was called **non-closing** when it contained no epoch of deep sleep. For each person the share of non-closing cycles was computed and related to age with Spearman's rank correlation. As a check on the definition, the merge interval was set to 10 and 20 minutes.

The slow-oscillation peak was found in the same way as the alpha peak, from all deep-sleep epochs joined, with 8-second Welch windows, a background fit between 0.2 and 6 Hz leaving out 0.3–2 Hz, and a threshold of 0.05 $\log_{10}$ units. People with fewer than 20 deep-sleep epochs were recorded as having no measurable peak.

5.4 Procedure

Every hypothesis was written into the analysis script before the script was run. Tests that failed are reported in Section 6.3. Analyses used Python 3 with MNE-Python [22] and SciPy. Scripts and results are available [21].

6. Results

6.1 Resting EEG in Alzheimer's disease

Measure	Alzheimer	Control	Test
No alpha peak	11 of 36	1 of 29	Fisher, one-sided, p = 0.005
Mean alpha frequency, where present	no difference	—	Mann–Whitney, p = 0.23
Spread of alpha frequency (SD)	2.07 Hz	0.92 Hz	Levene, p = 0.0007
No alpha peak, frontotemporal dementia	5 of 23	—	—

Table 1. Alpha rhythm in resting EEG (OpenNeuro ds004504).

In almost a third of the patients the rhythm had not slowed. It had gone. Where it remained, it wandered in both directions rather than slowing as a group. The mean spectra show the same: the alpha peak of the patients is less than a third of that of the controls (Fig. 2). This fits consequence C1.

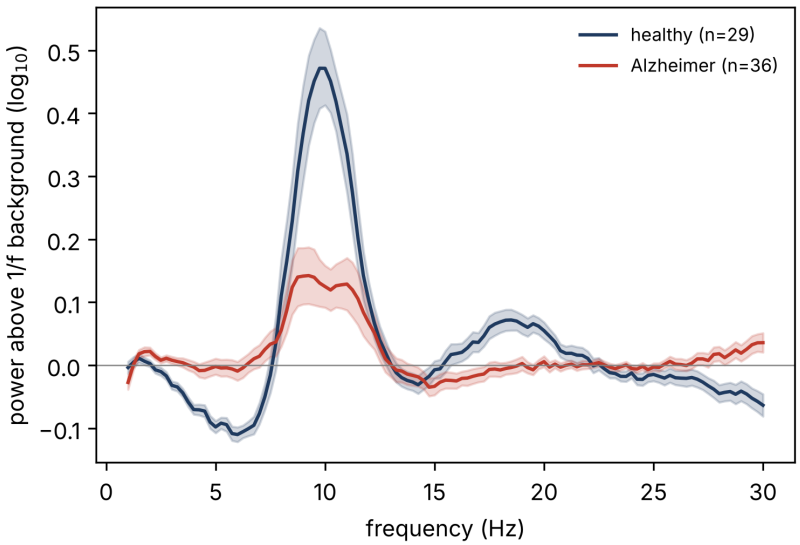


Fig. 2. Mean periodic spectrum (power above the 1/f background) of the posterior electrodes, with standard error. Healthy controls (n = 29) and people with Alzheimer's disease (n = 36).

6.2 Sleep in healthy ageing

Measure	Result	Test
Non-closing cycles, all ages	mean 31 %, median 25 %	—
Non-closing cycles, age 25–39	15 %	—
Non-closing cycles, age 80 and over	47 %	rise with age: Spearman $\rho = 0.25$, $p = 0.028$
Minutes of deep sleep	fall with age	$\rho = -0.43$, $p < 0.001$
No measurable slow-oscillation peak	27 of 78, mostly older	—
Slow-oscillation frequency, where present	falls with age	$\rho = -0.44$, $p = 0.001$

Table 2. Closure of sleep cycles in healthy ageing (Sleep-EDF Expanded, Sleep Cassette, n = 78).

The share of non-closing cycles about triples between the youngest and the oldest group (Fig. 3). This fits consequence C2. With merge intervals of 10 and 20 minutes the overall mean was 0.30 and 0.33. Its closeness to the 30.87 % of Proposition 2 is a coincidence of numbers: the sleep share depends on age and on the definition of a cycle, so it is not a constant.

The pattern of Section 6.1 returns. In a third of the sleepers, mostly older ones, the slow-oscillation peak was not measurable. The rhythm that drives removal had not only slowed. It was absent.

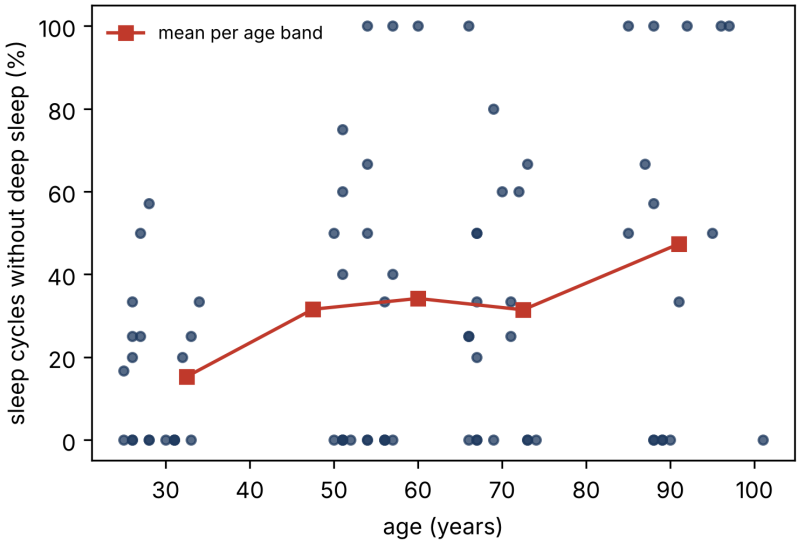


Fig. 3. Share of sleep cycles without deep sleep against age, one point per person. Squares give the mean per age band (25–39, 40–54, 55–64, 65–79, 80 and over).

6.3 What was not confirmed

An earlier proposal placed body rhythms on fixed rings of a ladder with factor three [19]. This was tested in five ways: the alpha peak, all spectral peaks together, the spectrum folded on the ladder, the slow oscillation across age, and two nights per person. The rings did not perform better than ladders with random ratios, nor better than the plain distance from the healthy average. Rhythms proved to be stable personal fingerprints across two nights: their variation within a person was two to four times smaller than between persons. But their position on the

ladder did not carry the effect. What carried it was whether the rhythm was present at all. This is why the hypothesis is phrased in terms of closure and not of position.

7. Interventions as natural tests

Consequence C3 can be checked against recent trials, which differ in exactly the way the hypothesis cares about (Table 3).

Intervention	How it works	Outcome
Fixed 40 Hz light and sound (HOPE, phase 3) [17]	Imposes one frequency from outside	Primary endpoint not met; pre-specified mild subgroup better
Phase-locked sound in deep sleep [14]	Follows the brain's own slow waves	Slow waves and memory enhanced; out of phase no effect
Phase-locked sound in cognitive impairment and Alzheimer's disease [15,16]	As above, at home	More deep sleep, better amyloid dynamics; small studies
Hearing aids (ACHIEVE) [18]	Restores input	No effect overall; about half the decline in the higher-risk group

Table 3. Interventions read as tests of consequence C3.

The fixed tone and the phase-locked tone use the same sense, hearing. The difference is timing. A fixed tone ignores where the brain's own loop stands. A phase-locked tone arrives when the loop is ready to close. In the original study, stimulation in phase strengthened the slow rhythm, and stimulation out of phase did nothing [14]. That is closure in its plainest form.

The hearing trial adds the input link. Restoring input helped those at higher risk and not those who were already well [18]. This is expected if input supports a closure that is already under strain.

8. Predictions

The hypothesis is wrong if the following fail.

- **P1. Non-closing cycles predict dementia.** In cohorts with sleep recordings and long follow-up, the share of cycles without deep sleep predicts incident dementia and adds to the total minutes of deep sleep. Suitable data exist in Framingham, MESA and other cohorts of the National Sleep Research Resource.
- **P2. Following beats imposing.** In a head-to-head trial, phase-locked stimulation during deep sleep outperforms fixed-frequency stimulation of equal dose on clearance markers and cognition.
- **P3. Closure moves the markers.** Within the same person, nights with more closed cycles are followed by lower blood or fluid markers such as p-tau217 and a better amyloid-β42/40 ratio.
- **P4. Input supports closure.** Correcting hearing loss increases deep sleep in people with poor sleep, before any cognitive effect appears.
- **P5. Disappearance precedes slowing.** In repeated EEG over years, loss of the alpha or slow-oscillation peak comes before, or independently of, a shift in its frequency.

9. Limitations

- The human evidence for the link between sleep and dementia is observational, and the relation runs both ways [10].
- The two datasets are small and were not designed for this question. No open dataset combines sleep recordings with a diagnosis of Alzheimer's disease.
- The definition of a non-closing cycle is simple and depends on visual sleep scoring.
- The results of the HOPE trial are so far known only from a conference presentation and a company announcement [17].
- The risk percentages of the Lancet Commission are model estimates, not measured effects of each factor [1].
- Section 2 supplies a frame. Its transfer to the brain is a reading and is tested only through the predictions.

10. Conclusion

In a system built from trits alone, a fixed part of all points never closes, and the size of that part swings with scale. The brain faces the same problem each day. Deep sleep is where the day is closed. When the night no longer closes the day, a remainder is carried forward. Amyloid is made at a normal rate but removed too slowly. Rhythms do not only shift; they vanish. Sleep cycles that never reach deep sleep become more common with age. Help that follows the brain's own rhythm works better than help that imposes one.

The proposal is simple. Treat lack of deep sleep as the primary cause of late-onset dementia. Measure closure night by night. Support it by restoring input and by stimulation that follows the brain. Then test whether dementia follows.

Declarations

Ethics statement. This study used only public, anonymised datasets released for reuse. No new data were collected from people, and no ethical approval was required.

Data availability. OpenNeuro ds004504 (https://openneuro.org/datasets/ds004504) and Sleep-EDF Expanded on PhysioNet (https://physionet.org/content/sleep-edfx/). Analysis scripts are available from the author [21].

Author contributions. J. Konstapel conceived the hypothesis, directed the analysis and wrote the manuscript.

Declaration of generative AI in the writing process. The author used Claude (Anthropic) for literature search, data analysis and drafting. The author reviewed and edited the content and takes full responsibility for it.

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Declaration of competing interest. The author declares no competing interests.

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Supplementary material: annotated reference list

Each entry repeats the reference and adds a short note on what it contributes and where to read further.

[1] Livingston G, Huntley J, Liu KY, Costafreda SG, Selbæk G, Alladi S, et al. Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. Lancet 2024;404:572–628. https://doi.org/10.1016/S0140-6736(24)01296-0.

The source of the fourteen modifiable risk factors and the 45 % estimate. The methods section explains how the percentages are modelled.

[2] Konstapel J. Vacuum.Net Theory, Ninth Edition: Foundations 9.2 and Physics 9.0. Leiden: Constable Research; 2026.

The full statement of the theory whose axioms are used in Section 2. Contains the proofs on the ladders ×2 and ×3.

[3] Konstapel J. The Trit Tile — a reference work, version 1.1. Leiden: Constable Research; 2026.

Gives the full proof of Proposition 2, with certified bounds, and links the tile to self-affine tilings and log-periodic signals.

[4] Mawuenyega KG, Sigurdson W, Ovod V, Munsell L, Kasten T, Morris JC, et al. Decreased clearance of CNS amyloid-β in Alzheimer's disease. Science 2010;330:1774. https://doi.org/10.1126/science.1197623.

Twelve patients and twelve controls. Amyloid-β was produced at a normal rate and cleared about 30 % more slowly. The key fact behind the hypothesis.

[5] Xie L, Kang H, Xu Q, Chen MJ, Liao Y, Thiyagarajan M, et al. Sleep drives metabolite clearance from the adult brain. Science 2013;342:373–7. https://doi.org/10.1126/science.1241224.

Mouse study. Sleep enlarges the interstitial space by about 60 % and speeds removal of amyloid-β. The origin of the glymphatic sleep link.

[6] Fultz NE, Bonmassar G, Setsompop K, Stickgold RA, Rosen BR, Polimeni JR, et al. Coupled electrophysiological, hemodynamic, and cerebrospinal fluid oscillations in human sleep. Science 2019;366:628–31. https://doi.org/10.1126/science.aax5440.

Human data. Slow waves are followed by oscillations of blood volume, which are coupled to waves of fluid flow.

[7] Shokri-Kojori E, Wang GJ, Wiers CE, Demiral SB, Guo M, Kim SW, et al. β-Amyloid accumulation in the human brain after one night of sleep deprivation. Proc Natl Acad Sci USA 2018;115:4483–8. https://doi.org/10.1073/pnas.1721694115.

Twenty healthy adults, PET imaging. One night without sleep raised amyloid in hippocampus and thalamus.

[8] Sabia S, Fayosse A, Dumurgier J, van Hees VT, Paquet C, Sommerlad A, et al. Association of sleep duration in middle and old age with incidence of dementia. Nat Commun 2021;12:2289. https://doi.org/10.1038/s41467-021-22354-2.

Whitehall II, 7,959 people, about 25 years of follow-up. Six hours of sleep or less at age 50 and 60 went with a higher dementia risk.

[9] Himali JJ, Baril AA, Cavuoto MG, Yiallourou S, Wiedner CD, Himali D, et al. Association between slow-wave sleep loss and incident dementia. JAMA Neurol 2023;80:1326–33. https://doi.org/10.1001/jamaneurol.2023.3889.

Framingham, 346 people over 60 with two sleep recordings. Each percent of deep sleep lost per year went with 27 % more dementia risk.

[10] Ju YS, Lucey BP, Holtzman DM. Sleep and Alzheimer disease pathology — a bidirectional relationship. Nat Rev Neurol 2014;10:115–9. https://doi.org/10.1038/nrneurol.2013.269.

The standard statement that sleep and Alzheimer pathology feed each other. Needed to judge the direction of cause.

[11] Miltiadous A, Tzimourta KD, Afrantou T, Ioannidis P, Grigoriadis N, Tsalikakis DG, et al. A dataset of scalp EEG recordings of Alzheimer's disease, frontotemporal dementia and healthy subjects from routine EEG. Data 2023;8:95. https://doi.org/10.3390/data8060095.

The open resting EEG data used here (OpenNeuro ds004504): 36 Alzheimer, 23 frontotemporal dementia, 29 healthy.

[12] Kemp B, Zwinderman AH, Tuk B, Kamphuisen HAC, Oberyé JJL. Analysis of a sleep-dependent neuronal feedback loop: the slow-wave microcontinuity of the EEG. IEEE Trans Biomed Eng 2000;47:1185–94. https://doi.org/10.1109/10.867928.

Source of the open sleep data used here (Sleep-EDF Expanded, PhysioNet): 78 healthy people aged 25 to 101.

[13] Penttonen M, Buzsáki G. Natural logarithmic relationship between brain oscillators. Thalamus Relat Syst 2003;2:145–52.

Shows that the bands of brain rhythm form an even ladder on a logarithmic scale.

[14] Ngo HVV, Martinetz T, Born J, Mölle M. Auditory closed-loop stimulation of the sleep slow oscillation enhances memory. Neuron 2013;78:545–53. https://doi.org/10.1016/j.neuron.2013.03.006.

Sound in phase with the brain's own slow oscillation strengthened it and improved memory. Out of phase it did nothing. Central to prediction P2.

[15] Van den Bulcke L, Peeters AM, Heremans E, Davidoff H, Borzée P, De Vos M, et al. Acoustic stimulation as a promising technique to enhance slow-wave sleep in Alzheimer's disease: results of a pilot study. J Clin Sleep Med 2023;19:2107–12. https://doi.org/10.5664/jcsm.10778.

First pilot of phase-locked sound in Alzheimer's disease. Small, but it shows the method can be used at home.

[16] Zeller CJ, Wunderlin M, Wicki K, Teunissen CE, Nissen C, Züst MA, et al. Multi-night acoustic stimulation is associated with better sleep, amyloid dynamics, and memory in older adults with cognitive impairment. GeroScience 2024;46:6157–72. https://doi.org/10.1007/s11357-024-01195-z.

Older adults with cognitive impairment, three stimulation nights. Better deep sleep and amyloid dynamics. Short and uncontrolled over the long term.

[17] Cognito Therapeutics. Phase 3 HOPE study of Spectris AD. Presented at: Clinical Trials on Alzheimer's Disease (CTAD); 17 November 2026. Announcement by the Alzheimer's Drug Discovery Foundation.

A fixed 40 Hz light-and-sound stimulus. The primary endpoint was not met; a pre-specified mild subgroup did better. Company-sponsored; no full paper yet.

[18] Lin FR, Pike JR, Albert MS, Arnold M, Burgard S, Chisolm T, et al. Hearing intervention versus health education control to reduce cognitive decline in older adults with hearing loss in the USA (ACHIEVE): a multicentre, randomised controlled trial. Lancet 2023;402:786–97. https://doi.org/10.1016/S0140-6736(23)01406-X.

Hearing aids did not slow decline overall. In the higher-risk subgroup decline slowed by about half.

[19] Konstapel J. Rhythms out of tune: the frequencies of bodily and mental imbalance and the frequencies that treat them. constable.blog; 8 November 2026.

Proposed fixed rings of the ladder ×3 as reference points for body rhythms. Section 6.3 reports that this placement was not confirmed.

[20] Konstapel J. The Border: an essay on the threshold in science. constable.blog; 24 November 2026.

Asks whether points that never close have a 'sleep' that lets them close after all. This article proposes an answer.

[21] Konstapel J. Dementia and nightly closure: analysis scripts and results, version 0.5. Leiden: Constable Research; 2026 (available on request).

All scripts, pre-registered hypotheses and results, including the tests that failed and the recomputation of the tile.

[22] Gramfort A, Luessi M, Larson E, Engemann DA, Strohmeier D, Brodbeck C, et al. MEG and EEG data analysis with MNE-Python. Front Neurosci 2013;7:267. https://doi.org/10.3389/fnins.2013.00267.

The software used to read and process the EEG files.