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## Authors
Sexton, Claire E
Sykara, Konstantina
Karageorgiou, Elissaios
et al.

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REVIEW

# Connections Between Insomnia and Cognitive Aging

**Claire E. Sexton[1,2] · Konstantina Sykara[3] · Elissaios Karageorgiou[1,3] · Jenny Zitser[1] · Talita Rosa[1] · Kristine Yaffe[4] · Yue Leng[1,4]**

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**Abstract** Insomnia is a common sleep disorder among older adults, and a risk factor for poor physical and mental health. However, the relationship between insomnia and cognitive health is not well understood. Here, we review observational studies that have investigated whether insomnia is associated with deficits in objective cognitive performance and an increased risk of dementia, magnetic resonance imaging studies that have assessed grey matter volumes and white matter microstructure, and interventional studies that have explored whether the treatment of insomnia can improve cognitive outcomes. There are inconsistent findings regarding impaired performance in objective cognitive tests and reduced grey matter volumes, and limited, emerging, evidence that suggests that insomnia is associated with an increased risk of dementia and reduced white matter integrity. Although the interventional literature is still in its infancy, there is some indication that treatment may have an impact on vigilance. Well-powered studies examining sources of heterogeneity are warranted.

**Keywords** Insomnia · Sleep · Dementia · Cognition

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✉ Yue Leng
yue.leng@ucsf.edu

[1] Global Brain Health Institute, Memory and Aging Center, Department of Neurology, University of California, San Francisco, CA, USA

[2] Department of Psychiatry, University of Oxford, Oxford, UK

[3] Neurological Institute of Athens, Athens, Greece

[4] Department of Psychiatry, Neurology and Epidemiology, University of California, San Francisco, CA, USA

## Introduction

Insomnia is a common sleep disorder among older adults, up to 50% of whom report symptoms of insomnia and, depending on the diagnostic guidelines used, up to 20% meet the criteria for insomnia disorder [1]. Symptoms of insomnia include difficulty in initiating sleep, maintaining sleep continuity, or waking up earlier than desired, despite adequate opportunity for sleep. For a clinical diagnosis of chronic insomnia disorder to be made according to the most recent guidelines, symptoms must be present at least three times a week, for at least three months, and be associated with daytime consequences [2, 3]. Insomnia is thought to stem from graded contributions of cognitive-behavioral and neurophysiologic processes, and a range of models have been proposed to explain its etiology (Table S1). Treatments span pharmacological (e.g. benzodiazepines and benzodiazepine-receptor agonists) and non-pharmacological [e.g. cognitive behavioral therapy for insomnia (CBT-I)] approaches, with evidence-based guidelines concluding that CBT-I is superior to hypnotic treatment in terms of therapeutic efficiency in both the short and long term [4]. Insomnia is an established risk factor for poor physical and mental health [5], and has also been proposed as a risk factor for poor cognitive health [6, 7]. Indeed, over the past decade, insomnia has variably been associated with deficits in objective cognitive functioning, increased risk of dementia, and reductions in grey matter volume and white matter integrity in networks essential for cognitive functioning. The possibility that successful treatment of insomnia could impact on cognitive markers is a tantalizing prospect.

Enthusiasm, however, regarding the extent to which insomnia represents a noteworthy modifiable risk factor for cognitive health in aging has been dampened by frequent

null findings. To provide an update on the research landscape, we review observational and interventional research published over the past 10 years that has explored the relationship between insomnia and cognitive health in mid- and late-life (>40 years of age), from three different perspectives. First, we summarize observational studies that have examined whether insomnia is associated with deficits in objective cognitive performance and an increased risk of dementia. Second, we review magnetic resonance imaging (MRI) studies that have assessed grey matter volumes and white matter microstructure in insomnia. Third, we present interventional studies that have investigated whether treatment of insomnia can improve cognitive outcomes. Through considering all three approaches, we aim to review whether the results are consistent within each approach, before considering whether the findings are complimentary across approaches.

## Review of Evidence

Given that heterogeneity between studies may, in part, result from variations in the classification of insomnia (i.e. from the symptom to the disorder level) and small sample sizes, our review focuses on studies that used diagnostic criteria for insomnia, did not list a specific co-morbidity as an inclusion criterion, and included at least 20 participants with insomnia.

### Objective Cognitive Functioning and Risk of Dementia

Table 1 provides an overview of studies of insomnia in mid- and late-life that have examined objective cognitive functioning. With regard to general cognitive function, null findings [8–10] outnumber studies that report significant deficits in insomnia [11]. With regard to specific cognitive domains, reduced performance in insomnia has been reported in individual tests spanning attention [10, 12], episodic memory [8, 10], working memory [11, 13], executive function [10], and language [10]. However, the number of null findings for individual tests is substantial [9, 14, 15]. Interestingly, while significant findings from individual cognitive tests are sporadic, pooling of measures by domain by calculating composite measures may be more sensitive in detecting group differences. Reduced performance with insomnia has been reported for composite scores for attention and episodic memory [8],as well as for working memory, verbal information processing, verbal memory, verbal fluency, and visual memory [16].

Although insomnia has typically not been included in reviews of modifiable risk factors for dementia [23, 24], an increasing number of epidemiological studies have examined the effects of insomnia on cognition in older adults. However, three systematic reviews and meta-analyses have been inconsistent in their findings [25–27]. To the best of our knowledge, only two studies have used diagnostic criteria to assess insomnia. One study of 179,738 male veterans (aged 55 years and older) from the Department of Veterans Affairs National Patient Care Database suggested that those with insomnia were 26% more likely to develop Alzheimer's disease over 8 years of follow-up, but were not significantly more likely to develop vascular dementia or Lewy body dementia [28]. Another study using Taiwan's National Health Insurance Research Database found that patients (aged 50 years and older) with insomnia and long-term use of hypnotics had more than double the risk of dementia over 3 years [29]. While these two studies both supported the idea that insomnia could be a risk factor for dementia, further confirmatory longitudinal studies from different population settings are needed.

### Grey Matter Volume and White Matter Microstructure

In order to understand the biological processes that may underlie cognitive deficits in insomnia, a growing number of studies have used MRI techniques to examine differences in grey matter volumes and white matter microstructure between insomnia and control groups (Table 2). Even though certain studies indicate reduced volume of the hippocampus [16], frontal cortex [30], and pineal gland [31] in participants with insomnia compared to controls, findings are not consistent across studies, especially after full correction for multiple comparisons across space [10, 32, 33]. Two papers, which used overlapping samples, related grey matter volumes to cognitive performance. Cognitive tests scores were not significantly associated with grey matter in voxel-based morphometry analyses [10], but reduced composite measures of verbal memory, processing, and fluency scores were associated with atrophy of the dentate gyrus and the CA2-4 subfields of the hippocampus [16].

While fewer studies have examined white matter microstructure, the results appear to be more consistent, with two diffusion tensor imaging studies reporting significant reductions in fractional anisotropy in insomnia within fronto-subcortical tracts, indicating a decline in white matter integrity [34, 35]. Interestingly, such regions have also been implicated in studies of insomnia disorder in younger age groups [36] and studies of poor sleep quality in older adults [37].

**Table 1** Clinical studies assessing objective cognitive functioning.

| Study (Country) | Demographics | | | Insomnia | | | Methods & Results |
|---|---|---|---|---|---|---|---|
| | N | Age (years) | % Female | Diagnostic Criteria | Duration (years) | PSQI | Cognitive Measures – Significant Group Differences in Bold *Reduced Performance in Insomnia Group |
| Zhang *et al.* 2018 [11] (China) | I: 34 C: 17 | I: 41.1 ± 10.1 C: 41.4 ± 9.9 | I: 64 C: 47 | DSM-V | ≥ 0.5 | 14.0^ [13.0, 16.0] | **MoCA-C***, *9-Box Maze* – **ORcM***, **SWM***, **OWM***, SRM, ORM |
| Chen *et al.* 2016 [13] (China) | I: 21 C: 20 | I: 41.8 ± 10.4 C: 38.1 ± 10.4 | I: 71 C: 65 | ICSD-3 | ≥ 0.5 | 17.0^ [14.0, 19.0]* | *9-Box Maze* – **ORcM***, **SWM***, OWM, SRM, ORM |
| Li *et al.* 2016 [17] (China) | I: 36 C: 25 | I: 40.4 ± 12.36 C: 39.9 ± 12.5 | I: 43 C: 47 | DSM-IV-TR | ≥ 0.5 | 14.3 ± 2.9 | *ANT* - Accuracy, RT, Alertness, Orientation, **Executive*** |
| Fortier-Brochu *et al.* 2014 [8] (Canada) | I: 25 C: 16 | I: 44.0 ± 11.5 C: 42.8 ± 12.9 | I: 56 C: 50 | DSM-IV / ICD-10 | 17.3 ± 13.1 | N/A | MMSE<br>**Attention Composite***: *CPT-II* – Hit Rate, Hit Rate Block Rate, Omissions, Commissions, Detectability, Perseverations<br>Working Memory Composite: *Digit Span* - Forward, Backward; PASAT, *CVLT-II* – Trial 1<br>**Episodic Memory Composite***: *CVLT-II* – Trial 5, Delayed Recall, Repetitions, Intrusions*<br>Executive Function Composite: *Tower* - Executive, Violations; Verbal *Fluency* – Alphabetic, Category, % Set Loss Errors, % Repetition Errors |
| Liu *et al.* 2014 [18] (China) | I: 36 C: 26 | I: 42.0 ± 11.0 C: 40.5 ± 12.0 | I: 58 C: 62 | DSM-IV | 6.5 ± 6.0 | 13.6 ± 3.4 | *ANT* – Alertness, Orientation, **Executive*** |
| Lovato *et al.* 2013 [15] (Australia) | I: 49 C: 49 | I: 70.0 ± 9.3 C: 69.4 ± 4.8 | I: 55 C: 55 | Not specified | ≥ 0.5 | 11.7 ± 2.6 | *Double Span Memory* – Objects, Locations, Double |
| Sivertsen *et al.* 2013 [9] (Norway) | I: 30 C: 91 | T: 64.0 ± 7.6 | T: 69 | DSM-IV-TR | N/A | N/A | MMSE<br>Processing Speed: *CWIT* – Reading, Naming; Verbal Fluency – FAS, Category; TMT A<br>Executive Function: *CWIT* – Inhibition, Inhibition / Switching; FAS Category Switching; Letter Number; TMT B; Coding<br>Memory: *CVLT* – Learning, Short Delay, Long Delay, Recognition; *RCF* – Immediate, Delay, Recognition<br>Visual Cognition: WASI Matrix Reasoning, *Rey* – Copy<br>*CDT* – Accuracy, RT, Valid RT, Invalid RT, Neutral RT, Nocue RT |
| Joo *et al.* 2014 [16] (A) (South Korea) | I: 27 C: 30 | I: 51.2 ± 9.6 C: 50.4 ± 7.1 | I: 93 C: 93 | ICSD-2 | 8.4 ± 9.1 [≥ 1] | 14.9 ± 4.6 | **Working Memory Composite***; Executive Function Composite; **Verbal Information Processing Composite***; **Verbal Memory Composite***; **Verbal Fluency Composite***; **Visual Memory Composite*** |
| Joo *et al.* 2013 [10] (A) (South Korea) | I: 27 C: 27 | I: 52.3 ± 7.8 C: 51.7 ± 5.4 | I: 93 C: 85 | ICSD-2 | 7.6 ± 6.1 [≥ 1] | 19.1 ± 4.3 | MMSE<br>Attention: *Digit Span* – Forward, Backward; *Corsi* – Forward, Backward; *TMT* – A, B; **Digit Symbol***<br>Visuospatial Function: RCF<br>Memory: *RCF* – **Immediate Recall***, **Delayed Recall***, **Recognition***; *Korean-CVLT* – Total, Short Delay, Long Delay, Recognition<br>Language: **Korean Boston Naming Test***<br>Executive: *COWAT* – Animal, Supermarket, Phonemic; *Stroop* – Word, **Color*** |
| Nissen *et al.* 2011 [14] (Germany) | I: 33 C: 53 | I: 46.2 ± 5.1 C: 46.7 ± 4.7 | I: 58 C: 60 | DSM-IV | N/A | 11.7 ± 6.6 | *Mirror Tracing* – Draw Time, Error Count, Error Time; Verbal Memory; Visual Memory; Short-Term Memory; Psychomotor Speed; Alertness; Divided Attention |

**Table 1** continued

| Study (Country) | Demographics | | | Insomnia | | | Methods & Results |
|---|---|---|---|---|---|---|---|
| | N | Age (years) | % Female | Diagnostic Criteria | Duration (years) | PSQI | Cognitive Measures – Significant Group Differences in Bold *Reduced Performance in Insomnia Group |
| Altena *et al.* 2008 [12] (Netherlands) | I: 25 C: 13 | I: 60.6 ± 6.0 C: 60.1 ± 8.3 | I: 72 C: 69 | DSM-IV | N/A | 12.3 ± 3.1 | *Simple Vigilance* – Lapses; *Complex Vigilance* - Lapses, False Positives; **Complex / Simple Reaction Time Ratio*** |

Unless otherwise indicated, values are the mean ± SD. ^ denotes P50 [P25, P75] for non-normally distributed variables. (A) indicates overlapping samples.

ANT: Attention Network Task; C: Control Group; CDT: Cued Visual Discrimination Task; COWAT: Controlled Oral Word Association Test; CPT: Conners Continuous Performance Test; CVLT: California Verbal Learning Test; CWIT: Color-Word Interference Test; DSM: Diagnostic and Statistical Manual of Mental Disorders [3, 19, 20]; I: Insomnia Group; ICD: International Classification of Disease [21]; ICSD: International Classification of Sleep Disorders [2, 22]; MoCA-C: Montreal Cognitive Assessment – Chinese-Beijing; MMSE: Mini-Mental State Examination; N/A: Not Applicable; ORcM: Object Recognition Memory; ORM: Object Recognition Memory; OWM: Object Working Memory; PASAT: Paced Auditory Serial Addition Task; RCF: Rey Complex Figure; RT: Reaction Time; SRM: Spatial Recognition Memory; SWM: Spatial Working Memory; T: Total; TMT: Trail Making Test.

### Interventional Studies

Although, to the best of our knowledge, there have been no interventional studies examining the effect of treatments for insomnia on the risk of dementia or structural MRI outcomes, a growing literature is exploring whether successful treatment of insomnia has an impact on objective cognitive functioning.

In a study of 77 adults over the age of 60 who met DSM-IV-TR and ICSD-2 criteria for insomnia, Wilckens *et al.* [38] found that a 4-week brief behavioral treatment of insomnia did not improve performance in tests of recall, working memory, or reasoning compared with the provision of sleep information as a control condition. This was despite significant decreases in waking after sleep onset in the treatment group compared with the control group.

In a study of 25 adults meeting the DSM-IV criteria for primary insomnia, a six-week multi-component intervention (including sleep restriction, cognitive behavioral therapy, bright-light therapy, physical activity, and body temperature manipulations) restored reaction time in a test of psychomotor vigilance to a level comparable to that displayed by a control group without insomnia (reducing reaction time in a simple vigilance task, and increasing reaction time in a complex vigilance task) [12].

Finally, in a study of 46 adults over the age of 55 who met the DSM-IV criteria for insomnia, participants were randomized into a 6-week program of CBT-I, a hypnotic treatment group (Zopiclone), or a placebo control group [39]. Across CBT-I and Zopiclone groups, reaction time performance in a vigilance task improved both at post-treatment and at 6-month follow-up assessments. There were no significant group-by-time interactions in reaction time or number of correct responses. Performance on the number of correct responses in the vigilance task worsened significantly in the CBT-I group post-treatment, but not at follow-up. This final finding likely reflects CBT-I implementing time-in-bed restriction in the early stages of treatment. Indeed, it is consistent with a study of younger adults who met the research criteria for insomnia, in which 4 weeks of sleep restriction therapy was shown to be associated with increases in psychomotor vigilance test lapses within the acute treatment phase, returning to baseline levels by 3-month follow-up [40].

## Discussion

We reviewed a range of observational and interventional studies that have investigated the relationship between insomnia and measures of cognitive health in ageing. From the observational studies, the evidence is mixed regarding impaired performance in objective cognitive tests and reduced grey matter volumes. There is limited, emerging evidence suggesting that insomnia is associated with an increased risk of dementia and reduced white matter integrity. From the interventional studies, although the literature is still in its infancy, there is some indication that treatment of insomnia may have an impact on reaction time in vigilance tasks.

Given the variation in results for objective cognitive functioning and grey matter volumes, and the limited number of studies on the risk of dementia or white matter microstructure, it is no surprise that it is challenging to draw strong conclusions on the concordance between different observational approaches. For example, it is not the case that cognitive studies consistently highlight a particular cognitive domain, and MRI studies consistently report structural deficits in networks essential for that cognitive domain. To shed further light on this area, it is encouraging that a growing number of studies are investigating the anatomical substrates of cognitive deficits in

**Table 2** Case-control MRI studies examining grey matter volume or white matter microstructure.

| | Demographics | | | Insomnia | | | Method | Group Differences |
|---|---|---|---|---|---|---|---|---|
| | N | Age (years) | % Female | Diagnostic Criteria | Duration (years) | PSQI | | |
| Dai *et al.* 2018 [32] (China) | I: 39<br>C: 39 | I: 48.9 ± 11.4<br>C: 47.9 ± 9.2 | I: 74<br>C: 67 | ICSD-2 | 6.5 ± 5.7 [>1] | 15.1 ± 2.2 | GM: GMV, VBM | No significant differences |
| Li *et al.* 2016 [35] (China) | C: 23<br>I: 30 | C: 41.1 ± 10.5<br>I: 41.6 ± 9.5 | I: 57<br>C: 40 | DSM-IV | 1.5 ± 1.7 | 13.4 ± 3.2 | WM: Tract Based Spatial Statistics | ↓ FA in anterior and posterior limb of internal capsule, anterior and superior corona radiata, superior longitudinal fasciculus, corpus callosum. No significant differences in AxD, RD or MD. |
| Bumb *et* al. 2014 [31] (Germany) | I: 23<br>C: 27 | I: 43 ± 7.4<br>C: 39 ± 13.1 | I: 48<br>C: 59 | DSM-IV-TR, ICSD-2 | 8.6 ± 7.3 [0.5 – 30] | N/A | GM: ROI – Pineal Gland | ↓ Pineal gland |
| Joo *et al.* 2014 [16] (A) (South Korea) ↓ Hippocampus | I: 27<br>C: 30 | I: 51.2 ± 9.6<br>C: 50.4 ± 7.1 | I: 93<br>C: 93 | ICSD-2 | 8.4 ± 9.1 [≥ 1] | 14.9 ± 4.6 | GM: ROI – | Hippocampus |
| Spiegelhalder *et al.* 2014 [34] (B) (Germany) | I: 24<br>C: 35 | I: 42.7 ± 14.5<br>C: 40.1 ± 9.1 | I: 58<br>C: 57 | DSM-IV-TR | 12.0 ± 10.9 | 11.2 ± 2.8 | WM: VW | ↓ FA in anterior internal capsule |
| Joo *et al.* 2013 [10] (A) (South Korea) | I: 27<br>C: 27 | I: 52.3 ± 7.8<br>C: 51.7 ± 5.4 | I: 93<br>C: 85 | ICSD-2 | 7.6 ± 6.1 [≥ 1] | 19.1 ± 4.3 | GM: VBM | No significant differences |
| Spiegelhalder *et al.* 2013 [33] (B) (Germany) | I: 28<br>C: 38 | I: 43.7 ± 14.2<br>C: 39.6 ± 8.9 | I: 64<br>C: 55 | DSM-IV-TR | 12.1 ± 11 | 10.9 ± 3.0 | GM: ROI – Freesurfer Cortical Volumes; VBM | No significant differences |
| Altena *et al.* 2010 [30] (Netherlands) | I: 24<br>C: 13 | I: 60.3 ± 6.0<br>C: 60.2 ± 8.4 | I: 71<br>C: 69 | DSM-IV | 17.7 ± 15.8 [2.5 – 50] | 12.1 ± 3.0 | GM: VBM | ↓ OFC |

Only results after correction for multiple comparisons are presented. Values are the mean ± SD. (A, B) indicates overlapping samples.

↓, reduced in insomnia; ↑, increased in insomnia; AxD, axial diffusivity; C, control group; DLPFC, dorsolateral prefrontal cortex; DSM, Diagnostic and Statistical Manual of Mental Disorders [3, 19, 20]; FA, fractional anisotropy; GM, grey matter; GMV, total grey matter volume; I, insomnia group; ICD, International Classification of Disease [21]; ICSD, International Classification of Sleep Disorders [2, 22]; MD, mean diffusivity; N/A, not applicable; OFC, orbitofrontal cortex; rACC, rostral anterior cingulate cortex; RD, radial diffusivity; ROI, region of interest; VBM, voxel-based morphometry; VW, voxel-wise; WM, white matter.

insomnia within their samples, although the results remain mixed. Clearly, further studies are needed before a consensus can be reached.

Variability in results between studies may not only stem from differences in approach, but also from differences in the demographics of participants (age, gender, and nationality), illness characteristics (diagnosis, duration, severity, and treatment status) and methods (cognitive test or MRI analysis tool). Since a full consideration of every factor that may have influenced the results is outside the scope of this review, we instead highlight the possible role of diagnostic criteria and the duration of insomnia.

While we limited our review to studies that used recognized diagnostic criteria for insomnia (DSM-IV, DSM-IV-TR, DSM-V, ICD-9, ICD-10, ICSD-2, or ICSD-3), the criteria differ in quantitative thresholds, meaning that insomnia samples are still heterogeneous in nature. Such heterogeneity was illustrated in the results of the America Insomnia Survey, in which insomnia prevalence estimates varied from 4% when based on ICD-10 criteria to

22% when based on DSM-IV-TR criteria [41]. While limiting diagnoses to the most severe cases may be more sensitive in detecting relationships with cognitive outcomes, relevant cases may be missed. Well-powered studies examining different diagnoses and the exact phenotyping of insomnia features (sleep-maintenance *versus* sleep-onset *vs* early-awakening) are warranted. In addition, to reduce sources of heterogeneity, studies that listed a specific co-morbidity as an inclusion criterion were considered outside the scope of this review. However, it is important to note that insomnia is often co-morbid with psychiatric disorders, medical conditions, and/or other sleep disorders [42]. The extent to which such co-morbidities have been considered by research studies varies between approaches. For example, in a recent review that evaluated the impact of CBT-I on cognitive outcomes, insomnia was co-morbid with a physical or mental health condition in the majority of included studies [43]. This is in contrast with the MRI literature, where specific co-morbidities have rarely been examined. Given that the relationship between insomnia and cognitive health may differ depending on the presence and nature of co-morbidities, this is an important area for future research.

Duration of illness may also influence results. While the duration of insomnia was variably reported by case-control studies of cognition, it was consistently included in MRI studies. Across studies assessing grey matter volume, the mean duration of insomnia ranged between 1.5 and 17.7 years, with the shortest duration 0.5 years and longest 50 years. If insomnia causes reductions in grey matter volume, then it follows that increased duration could have cumulative effects on brain structure, and be associated with greater reductions in volume. Certain studies indicate that increased duration of insomnia is associated with reduced hippocampal volume [44], however, others have not found associations between duration and MRI measures after correction for multiple comparisons [10, 16, 30–32]. It is important to note, though, that duration is often poorly defined. For example, in most cases it is unclear whether duration is calculated from symptom onset or first diagnosis, and whether it is based on self-reports or corroborated by others and/or objective measurements. Going forward, a careful consideration of the duration of insomnia and its relation to outcome measures, by both observational and interventional studies, has the potential to yield important insights for the field.

The reviewed literature builds on a substantial literature that suggests that sleep plays a critical role in maintaining cognitive health. Transcriptomic studies have highlighted the protective role of sleep on oligodendrocyte function and myelination [45]. Animal studies indicate that prolonged restriction or disruption of sleep has cumulative effects on the brain, for example leading to reduced hippocampal cell proliferation, cell survival, and neurogenesis [46]. Meanwhile, in humans, experimental studies have shown that sleep deprivation is associated with significantly reduced performance on tests of attention, working and short-term memory [47], and widespread changes in white matter microstructure [48], It follows that the reduced quality or quantity of sleep in insomnia may impact on cognitive health.

Symptoms of insomnia in older populations may also be a direct consequence of age- or early neurodegeneration-related changes in networks essential for sleep onset and maintenance, consistent with a neurobiological model of insomnia (Table S1). Indeed, there has been a particular focus on the relationship between sleep and Alzheimer's pathology. For example, pathology studies indicate that the IPA/VLPO neuronal loss in Alzheimer's disease is not significant in age-matched controls [49], and amyloid-precursor protein/amyloid β overproduction causes disrupted sleep in animal models [50]. The relationship between sleep and Alzheimer's pathology is proposed to be bi-directional, though, with sleep disruption also linked to increased production and decrease clearance of amyloid β [50]. In addition, modelling sleep-wake dysregulation in Alzheimer's disease allows for linking its two core pathologies, tauopathy and amyloidopathy [51], in a *bi-directional* relationship between sleep and pathology. Specifically, the presence of early tau pathology in sleep-wake regulating nuclei (Braak stages I/II) [52–54], leads to decreased attenuation of cortical activity during sleep, which in turn promotes subsequent cortical amyloid pathology *via* activity-dependent amyloid deposition [55, 56].

## Conclusion

In conclusion, there is mixed evidence for impaired performance in objective cognitive tests and reduced grey matter volumes, and emerging evidence of an increased risk of dementia and reduced white matter integrity. Overall, the heterogeneity could be attributed to different demographics of the participants, illness characteristics, and methods used. Further longitudinal studies are needed to determine whether insomnia is a true risk factor for dementia, and the inclusion of biomarkers and MRI measures would help to understand the underlying mechanisms. Ultimately, intervention studies that assess the effects of insomnia treatment on cognition might help open up new opportunities for the prevention of cognitive decline and dementia in the long run.

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