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The association between insomnia and cognitive decline: A scoping review

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ABSTRACT

Objective: This study aimed to investigate the association between insomnia and cognitive decline to provide insights for clinical interventions and future research.

Methods: The PubMed, Embase, Web of Science, Scopus, Cochrane Library, and ProQuest databases were systematically searched to identify studies on the association between insomnia and cognitive decline published within the last decade. The quality of the included studies was evaluated, followed by data extraction and summary analysis.

Results: A total of 36 studies were included in the review. Both subjective and objective measures were utilized across 12 indices to assess sleep status, while cognitive function was evaluated using 5 scales and 34 tests. The results revealed a significantly increased risk of cognitive decline or Alzheimer's disease among patients with insomnia, alongside notable impairments in attention, memory, visuospatial abilities, executive function, and verbal memory. Comprehensive assessments of cognitive domains were more sensitive in detecting group differences compared to assessments of specific cognitive sub-functions. Furthermore, MRI analyses showed reduced gray matter volumes in regions such as the prefrontal cortex, cingulate gyrus, temporal lobe, and hippocampus, together with reduced integrity of the white matter in patients with insomnia.

Conclusions: The findings indicate a potentially bidirectional relationship between insomnia and cognitive decline, suggesting that each may influence and exacerbate the other. Insomnia may increase the risk of cognitive decline and appears to be associated with reduced gray matter volume and compromised white matter integrity in the brain, which could potentially lead to declines in attention, memory, visuospatial abilities, executive function, and verbal memory. Conversely, cognitive decline may contribute to the onset of insomnia, further deteriorating sleep quality. However, further research is necessary to fully comprehend this intricate relationship.

1. Introduction

Insomnia and cognitive decline are major health issues in contemporary society, significantly affecting quality of life and work efficiency, and placing a substantial burden on the socioeconomic landscape [1]. Insomnia is a common sleep disorder characterized by difficulties in initiating and maintaining sleep, early-morning awakening with inability to return to sleep, as well as daytime functional impairments such as fatigue, decreased energy, and mood disturbances [2]. Moreover, these symptoms are not attributable to other sleep disorders, substance use, or medical conditions. For a diagnosis of insomnia, symptoms must occur at least three times a week for at least three months [3]. Similarly, cognitive decline refers to a spectrum of cognitive dysfunction categorized into three stages [4]: The presymptomatic stage has normal cognitive ability with no noticeable symptoms. The prodromal stage features mild cognitive impairment (MCI), where individuals experience modest decline but maintain daily independence with extra effort. The symptomatic dementia stage involves significant cognitive decline, leading to functional impairments that impact daily independence, with concerns noted by individuals and caregivers. Epidemiological surveys have indicated that approximately 50 % of individuals diagnosed with MCI will develop Alzheimer's disease (AD), within five years, and AD patients usually die within 5–12 years of diagnosis [5]. Given the overall aging of the population, addressing cognitive decline has emerged as a significant challenge in social health.

As the global population continues to age, the intersection between insomnia and cognitive decline has garnered increasing attention from both clinical and research communities [6]. The co-occurrence of these conditions is not merely coincidental. Insomnia has been implicated as a potential risk factor for the development of MCI and AD, with sleep

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disorders often preceding the clinical onset of cognitive symptoms [7]. This temporal relationship underscores the importance of early detection and intervention in sleep disorders as a preventive strategy against cognitive decline. Conversely, cognitive decline, particularly in the context of neurodegenerative diseases like AD, often co-occurs with sleep disorders, further impairing cognitive function and overall quality of life [8]. Studies have proposed that the complex interplay between sleep and cognition is mediated by multiple neurobiological mechanisms, including disruptions in the sleep-wake cycle [9] alterations in brain structure and function [10], and the accumulation of neurotoxic proteins like beta-amyloid [11], which are cleared during sleep. Moreover, the impact of insomnia on mood, attention, and executive function can compound the challenges faced by individuals with cognitive decline, leading to accelerated functional decline and increased caregiver burden [12].

Despite substantial evidence linking insomnia and cognitive decline, the literature remains fragmented due to methodological variations, differences in study populations, and inconsistencies in the cognitive domains assessed [13]. This scoping review addresses existing discrepancies by synthesizing evidence from the past decade and highlighting areas in need of further investigation. It specifically addresses the gap in studies that fail to integrate both subjective and objective measures of sleep and cognition. By focusing on the relationship between insomnia and cognitive decline, this review aims to provide new insights into how these conditions may reciprocally influence each other, an area that remains underexplored.

2. Materials and methods

The study adhered to the methodological framework of the Joanna Briggs Institute (JBI) scoping review guidelines from Australia [14]. This comprehensive process includes formulating research questions, developing a search strategy, screening of the relevant literature, extraction and analysis of data, evaluation of the data quality, and composing a review report. The review follows the reporting framework of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews (PRISMA-ScR) [15]. PRISMA-ScR comprises 27 core components, including the title, abstract, introduction, methods, results, discussion, and funding. Each component provides detailed guidance on relevant assessment points and standards to ensure a transparent, comprehensive, and standardized scoping review report. It facilitates authors in conducting literature screening, data extraction, and result reporting while aiding readers in developing a deeper understanding of pertinent terminology, core concepts, and key elements to be reported in scoping reviews. Following the methodological framework, the research process was structured as follows.

2.1. Formulation of research questions

The specific questions addressed in this study were: ① Is there an association between insomnia and cognitive decline, as well as an increased risk of Alzheimer's disease? ② How do insomnia and cognitive decline influence each other? ③ What structural changes occur in the brains of patients with both insomnia and cognitive decline?

2.2. Inclusion and exclusion criteria

The inclusion criteria were established using the Participants, Concept, and Context (PCC) framework for scoping reviews. ① Participants (P): Patients with insomnia aged 18 years or older; ② Concept (C): The research topic focused on the association between insomnia and cognitive decline, and included primary studies that evaluated this relationship using objective and subjective measures, such as the Pittsburgh Sleep Quality Index (PSQI), Insomnia Severity Index (ISI), Polysomnography (PSG), Mini-Mental State Examination (MMSE), and Montreal Cognitive Assessment (MoCA); ③ Context (C): Patients with difficulties in sleep maintenance, including difficulty falling asleep, frequent awakenings, and early waking, adversely affecting daytime functioning and memory, attention, and executive function. Exclusion criteria: ① Patients with sleep disorders other than insomnia or the presence of a coexisting serious disease; ② Reviews, animal studies, conference abstracts, or posters.

2.3. Development of a search strategy

Six databases were searched by two researchers. The databases were the Web of Science, PubMed, Scopus, Embase, Cochrane Library, and ProQuest. The search period was from December 21, 2013, to December 21, 2023. The search strategy combined free-text keywords and MeSH terms, supplemented by citation tracking and other search methods. The search terms included "Sleep Initiation and Maintenance Disorders", "Cognitive Dysfunction", and "Cognitive Aging". The PubMed search was as follows: ("Sleep Initiation and Maintenance Disorders"[MeSH]) OR (Insomnia) AND ("Cognitive Dysfunction"[MeSH]) OR (Cognitive decline) OR (Cognitive Aging) OR (Overinclusion).

2.4. Literature screening

The literature screening in this study was conducted using EndNote X9 software. After removing duplicate articles, two researchers with specialized training independently reviewed the studies and decided on inclusion based on predetermined criteria. In cases of disagreement, a third researcher was consulted to reach a consensus before data extraction.

2.5. Data extraction and analysis

The data extraction process was independently conducted by two researchers using a systematic approach with a predefined form to ensure consistency across all studies. The extracted key details included study identification (authors, country, publication year, journal), study design (type, sample size, follow-up duration), population characteristics (demographics, inclusion/exclusion criteria), insomnia assessment methods, cognitive function evaluation tools, key findings on the association between insomnia and cognitive decline, and study quality metrics such as risk of bias and robustness of findings. Any discrepancies between the researchers were addressed through discussion with a third reviewer, and if consensus could not be reached, the issue was escalated to the senior author for a final decision. To ensure accuracy and impartiality, the included articles were thoroughly reviewed multiple times to ensure that the data accurately reflected the original studies.

2.6. Evaluation of study quality

The quality of cross-sectional studies was evaluated using an adapted version of the Newcastle-Ottawa Cohort Scale [16]. The tool consists of three components: "Selection", "Comparability", and "Outcome", and evaluates seven subitems under the categories such as "representativeness of the sample", "sample size", "non-respondents", "ascertainment of exposure", "design and analysis", "assessment of outcome", and "statistical tests". A total score out of 9 points is assigned. The quality of cohort and case-control studies was evaluated using the Newcastle Ottawa Scale (NOS) [17]. The NOS is a meta-analysis scale for non-randomized studies, with a maximum score of 9 points per study. The selection and measurement of subjects and exposure are allocated a total of 4 points, with comparability among cohorts receiving 2 points and evaluation and follow-up results allocated 3 points. The quality of the study is classified as low (0–3 points), moderate (4–6 points), or high (7–9 points). The quality of the studies was independently evaluated by two researchers and any discrepancies were resolved through discussions with team members to reach a consensus.

3. Results

3.1. Literature search results

The initial search yielded a total of 663 articles, 352 of which were excluded following the removal of duplicates. After successive screening, 36 articles [18–53] were finally included. The flowchart illustrating the search and selection processes is presented in Fig. 1.

3.2. Basic characteristics of the included studies

The total number of included articles published between 2014 and 2024 was 36. Of these, 12 studies were from the USA [19,20,22,25–28,30,31,41,43,49], 9 from China [29,36,39,42,44,47,48,50,51], 3 each from Germany [40,52,53], and Italy [32,35,37], and 2 each from Mexico [21,23], and Canada [33,45]. Additionally, Norway [24], Japan [34], Russia [38], South Korea [46], and Sweden [18] contributed one article each. The study types included cross-sectional (n = 21) [18–38], case-control (n = 14) [39–53], and cohort (n = 1) [48] studies. A total of 31 studies [18–48] used both objective and subjective indicators to evaluate the reciprocal influence of insomnia and cognitive function; the basic features of these are shown in Table 1. 8 articles [19,39,46,49–53] described changes in the volume of gray matter or microstructural changes in white matter in patients with insomnia; the specific information is provided in Table 2.

3.3. Evaluation of the quality of the included studies

The overall quality of the included studies was high, with 29 studies [20–53] classified as high-quality and seven studies [18,19,21,29,32,38,45] as moderate-quality. Table 3 shows the NOS scores for each study.

3.4. Tools used for the assessment of insomnia and cognition

Insomnia assessment tools include subjective measures, such as the PSQI, AIS, ISI, BIQ, APS, RJSQ, ESS, PSAS, DISRS, PCII, as well as interviews, self-reports, and sleep diaries, along with objective sleep monitoring technologies like PSG and EEG. Cognitive function was evaluated using scales such as the MoCA, MMSE, HDS-R, WMS-II, and NPI-Q, along with 34 additional cognitive assessment tests. The evaluations primarily cover six cognitive domains: complex attention, executive function, learning and memory, language, perceptual-motor function and social cognition. Detailed descriptions of the specific assessment tools are provided in Table 4.

3.5. The impact of insomnia on cognitive function

The findings indicated that individuals who reported experiencing insomnia had a significantly increased risk of cognitive decline or Alzheimer's disease compared to those without sleep disorders. Table 1 provides an overview of 31 studies that objectively evaluated cognitive function in middle-aged and older adults with insomnia. Specifically, patients with insomnia exhibited notable declines in attention [21,33,34,45], memory [27,29,33,42] (including episodic memory [18,25,45], working memory [39,43,45,46], immediate memory [21], and delayed memory [21,33]), visual-spatial abilities [38,40,42,46], executive function [18,25,29,32,34,36,40,42,45,47], and language skills [21,27,29,36,42,43,46] on individual tests. However, differences in the research findings were observed between different tests. Several studies reported that individuals with insomnia exhibited poorer performance on tests assessing orientation [21], attention and calculation [21], abstraction [40,42], and perceptual speed [17]. However, the level of research support for these findings was limited. In comparison to the measurement of several cognitive subfunctions, measurements involving the aggregation and integration of various cognitive domains were observed to be more sensitive in detecting inter-group differences. The research findings consistently demonstrated significant declines in cognitive performance among patients with insomnia across multiple domains including attention, memory, orientation, and calculation, visual-spatial abilities, executive function, and language. This decline was also seen in reduced MMSE and MoCA scores [18–21,23,24,28,31,34,37,39–42,44,48]. Moreover, 12 studies [21,23,32,34,35,37–43] explored the link between insomnia severity and cognitive decline. Six studies [23,34,37,39,40,42] demonstrated a significant association between the severity of insomnia and overall cognitive decline, while the other six [21,32,35,38,41,43] provided evidence supporting the correlation between insomnia severity and specific cognitive domains, rather than global cognitive decline.

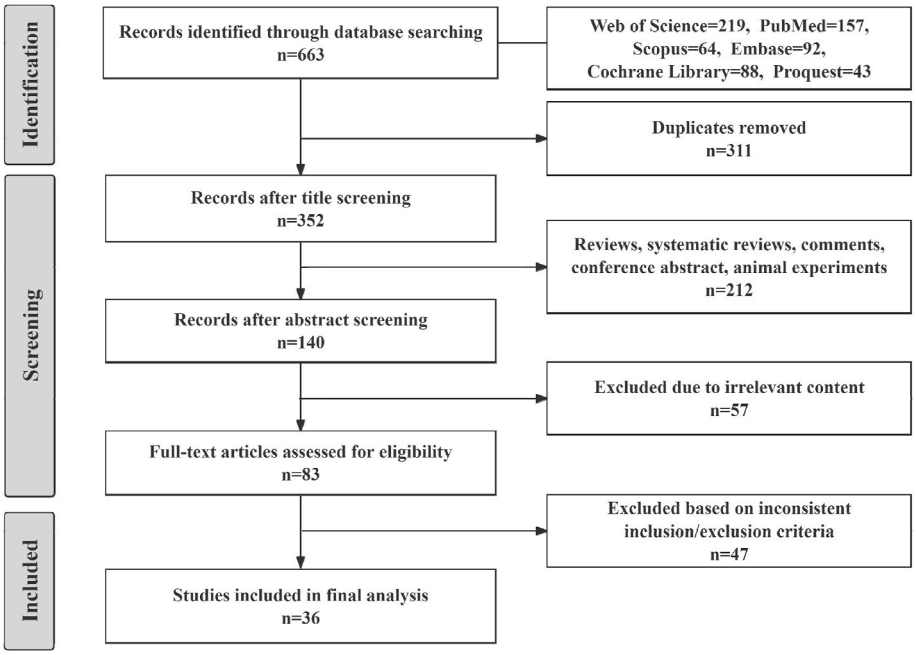

Fig. 1. Flowchart of the reviewing process.

Table 1
Included studies on the association between insomnia and cognitive function.

Author (Year/Country)	Study Type	Sample Size	Age	Age Group	Female (%)	Insomnia		Cognitive function
						Diagnostic Criteria	Evaluating Indicator	Evaluating Indicator -Assessment Content
Overton, Marieclaire et al. (2024 Sweden) [18]	Cross-sectional study	5631	74.2 ± 2.92	Older Adults	54.6 %	DSM-4	Interview Self-report	MMSE-**Episodic memory***, Verbal fluency, **Perceptual speed***, **Executive functioning***, Global cognition
Wei, Ruoqi et al. (2024 America) [19]	Cross-sectional study	PSG:632 MRI:160	I_1: 61 I_2: 67	Older Adults	PSG:55 % MRI:46 %	N/A	MRI PSG	**MMSE*** **MoCA***
Lee, Soomi et al. (2024 America) [20]	Cross-sectional study	I_1 = 1480 I_2 = 1393 C = 2949	I_1:71.94 ± 8.26 I_2:69.61 ± 10.03 C:68.57 ± 10.28	Older Adults	I_1:51.08 % I_2:50.39 % C:64.63 %	N/A	Self-report	**MoCA***
Correa-Muñoz, Elsa et al. (2023 Mexico) [21]	Cross-sectional study	107	N/A	N/A	82.3 %	DSM-5	AIS PSQI	MMSE **Orientation***, **Registration***, **Attention***, **Calculation***, **Language***, **Delayed memory***
Wong, Roger et al. (2023 America) [22]	Cross-sectional study	6284	76.28 ± 6.7 I:78.28 ± 8.0 C:73.65 ± 6.3	Older Adults	55.5 % I:59.68 % C:54.80 %	NHATS algorithm	Self-report	ADSI-Memory, Temporal orientation, Judgment, and Function IWR-Memory Date-Orientation CDT-Executive function
Sánchez-García, ergio et al. (2023 Mexico) [23]	Cross-sectional study	80	70.6 ± 6.3	Older Adults	71.2 %	N/A	AIS	**MMSE***
Selbaek-Tungevåg Selma et al. (2023 Norway) [24]	Cross-sectional study	7492	67.2 ± 6.2	Older Adults	55.5 %	DSM-5	Self-report	**MoCA*** WLMT
Zaheed, Afsara B et al. (2023 America) [25]	Cross-sectional study	2595	64.00 ± 6.76	Middle-Aged Adults	64.5 %	DSM-5	RJSQ	THCAP-**Episodic Memory*, Executive Function***, **Language**, **Visuoconstruction***, **Processing Speed**
Gallagher, Virginia T et al. (2023 America) [26]	Cross-sectional study	I_1:233 I_2: 307 C: 306	I_1:81.50 ± 6.87 I_2:85.39 ± 6.87 C:77.96 ± 5.35	Older Adults	I_1:50.21 % I_2:69.28 % C:54.07 %	DSM-3-R DSM- 4	NPI-Q	The ADAMS in-home assessment
Zhang, Yuan et al. (2023 America) [27]	Cross-sectional study	I: 7214 C: 12518	I:55.23 ± 7.46 C:46.15 ± 13.86	Middle-Aged Adults	41.2 % 39.1 %	N/A	Self-report	B-SEVLT-**Verbal episodic learning*** Recall-**Memory*** WFT- **Phonemic fluency*** DSST-**Processing speed*,** Eexecutive function
Agudelo, C.et al. (2022 America) [28]	Cross-sectional study	1391	73.5 ± 7.0	Older Adults	44.8 %	N/A	Self-report	**MMSE*** WMS-III Logical Memory
Xu, Wei et al. (2021 China) [29]	Cross-sectional study	385	73.2 ± 5.6	Older Adults	48 %	N/A	NPI	IRT- **Memory***, **Executive function***, **Language***, Visuospatial function
Resciniti, Nicholas V et al. (2021 America) [30]	Cross-sectional study	13833	66.41 ± 9.52	Older Adults	59.4 %	N/A	BIQ	Working memory, Word recall, Language, Orientation
Fernandez-Mendoza Julio et al. (2021 America) [31]	Cross-sectional study	I_1:453 I_2:172 C:899	I_1:46.1 ± 11.7 I_2:49.4 ± 9.6 C:49.8 ± 14.6	Middle-Aged Adults	53.4 % I_1:37.0 % I_2:23.9 % C:52.2 %	N/A	Questionnaire PSG	**MMSE*** SDMT-Processing speed TMT(TMT-A, TMT-B)-Executive attention BVRT-Short-term visual memory TWFT-Verbal fluency
Ballesio, Andrea et al. (2020 Italy) [32]	Cross-sectional study	80	24.03 ± 3.88	Young Adults	76.3 %	DSM-5	ISI Interview Sleep diary	TSP-**Executive functions*** PCII-**Executive functions***
Cross, Nathan E et al. (2019 Canada) [33]	Cross-sectional study	28,485	62.8 ± 10.2	Middle-Aged Adults	51.6 %	DSM-5	Self-report	MAT-**Mental flexibility***, **Processing speed**, **Memory*** RAVLT-**Learning***, **Retention*(Declarative memory)** COWAT-Executive functions (phonological fluency, knowledge) ST-**Mental speed***, **Inhibition***, **Attention***, **Mental control*** AFT-**Category fluency*** CRT test-Psychomotor speed
Hishikawa, Nozomi et al. (2017 Japan) [34]	Cross-sectional study	142	70.5 ± 9.7	Older Adults	50.0 %	N/A	AIS	**MMSE*-Attention***, **Executive function*** HDS-R MoCA-TMT,Copy cube, Draw clock, Namng, Read list digits, Read list of letters, Subtraction, Repeat, Fluency, Abstraction, Delayed recall, Orientation

(*continued on next page*)

Table 1 (*continued*)

Author (Year/Country)	Study Type	Sample Size	Age	Age Group	Female (%)	Insomnia		Cognitive function
						Diagnostic Criteria	Evaluating Indicator	Evaluating Indicator -Assessment Content
Palagini, Laura et al. (2017 Italy) [35]	Cross-sectional study	I:67 C:36	I:50.2 ± 10 C:49.7 ± 14	Middle-Aged Adults	I:61.1 % C:61.1 %	DSM-5	ISI DISRS APS PSAS	APS PSAS-**PSAS C***, **PSAS-S***
Li, Yun et al. (2016 China) [36]	Cross-sectional study	I: 36 C: 25	I: 40.4 ± 12.36 C: 39.9 ± 12.5	Middle-Aged Adults	I:43 % C:47 %	DSM-4-TR	PSQI PSG	ANT- Accuracy, Reaction Time, Alertness, Orientation, **Executive***
Palagini, Laura et al. (2016 Italy) [37]	Cross-sectional study	I:93 C:30	I:50.2 ± 15.8 C:51.1 ± 14.1	Middle-Aged Adults	I:55.9 % C:56.7 %	DSM-5	ISI PSQI DBAS- 16	**MMSE*** DBAS-16-**Beliefs*, Attitudes*, Expectations***, **Appraisals***, **Attributions***
M.Subbotina et al. (2015 Russia) [38]	Cross-sectional study	50	59.0 ± 15.9	Middle-Aged Adults	90 %	ICSD-3	ISI	Stroop: **Attention*** CVDT:Visuo-constructive abilities BVRT:**Visual perception***, **Visual memory*** VLT: **Performance of semantic memory*** TMT:Visual attention, Task switching
Yang, Lu et al. (2023 China) [39]	Case-control study	I: 25 C: 33	I:34.28 ± 11.34 C:36.06 ± 9.26	Middle-Aged Adults	N/A	ICSD-3	PSQI AIS ESS	**MoCA*** **Working memory***
Künstler, Erika C S et al. (2023 Germany) [40]	Case-control study	I:22 C:22	I:58.1 ± 12.2 C:55.3 ± 10.0	Middle-Aged Adults	I:13.6 % C:27.3 %	DSM-4	PSQI ISI PSG	**MoCA*-Visuospatial */Executive*, Verbal abstraction***, Attention, Recall, Naming, Language, Orientation
Ding, Xiaobin et al. (2023 America) [41]	Case-control study	I: 34 C: 33	I: 23.12 ± 2.74 C: 23.27 ± 2.64	Young Adults	62.7 % I:51.5 % C:73.5 %	N/A	PSQI ISI EEG	**TCOT*** **CWST***
Zhang, Li et al. (2021 China) [42]	Case-control study	I:45 C:45	I:64.07 ± 6.99 C:63.24 ± 6.97	Older Adults	I:51.11 % C:60.00 %	DSM-5	ISI PSG	**MoCA*-Spatial executive ability***, Naming, Attention, **Language fluency*, Abstract thinking***, **Memory***,Orientation
Kanady, Jennifer C et al. (2017 America) [43]	Case-control study	I:47 C:19	I:36.76 ± 11.23 C:30.74 ± 10.06	Middle-Aged Adults	I:63.0 % C:65.2 %	DSM-4-TR	ISI PSQI DSISD Sleep diary	N-Back Task, DST-**Working memory*** VLT-**Verbal learning***
Chen, Gui-Hai et al. (2016 China) [44]	Case-control study	I: 21 C: 20	I: 41.8 ± 10.4 C: 38.1 ± 10.4	Middle-Aged Adults	I:71 % C:65 %	ICSD-3	PSQI	**MoCA-C*** 9 Maze-**Object Recognition memory***, **Spatial Working Memory***,Object Working Memory, Spatial Recognition Memory, Object Recognition Memory
Fortier-Brochu et al. (2014 Canada) [45]	Case-control study	I: 25 C: 16	I: 44.0 ± 11.5 C: 42.8 ± 12.9	Middle-Aged Adults	N/A	DSM-4 ICD-10	PSG	**MMSE*** CPT-II –**Attention***(Hit Rate, Hit Rate Block Rate, Omissions, Commissions, Detectability, Perseverations) DST,PASAT, CVLT-II-Trial 1 -**Working Memory*** (Forward, Backward) CVLT-II-Trial 5-**Episodic Memory*** (Delayed Recall, Repetitions, Intrusions) Tower-**Executive Function*** (Executive, Violations) Alphabetic, Category –Verbal Fluency (% Set Loss Errors, % Repetition Errors)
Joo, Eun Yeon et al. (2014 Korea) [46]	Case-control study	I: 27 C: 30	I: 51.2 ± 9.6 C: 50.4 ± 7.1	Middle-Aged Adults	I:93 % C:93 %	ICSD-2	PSQI PSG Sleep diary	**Working Memory***, Executive Function, **Verbal Information Processing***, **Verbal Memory***, **Verbal Fluency***,**Visual Memory***
Liu, Hong et al. (2014 China) [47]	Case-control study	I: 36 C: 26	I: 42.0 ± 11.0 C: 40.5 ± 12.0	Middle-Aged Adults	I:58 % C:62 %	DSM-4	PSQI	ANT– Alertness, Orientation, **Executive***
Zhang, Ping et al. (2018 China) [48]	Cohort study	I:34 C:17	I: 41.1 ± 10.1 C: 41.4 ± 9.9	Middle-Aged Adults	I:64 % C:47 %	DSM-5	PSQI PSG	**MoCA-C*** 9 Maze–**Object Recognition emory***,**Spatial Working Memory***,**Object Working Memory***, Spatial Recognition Memory, Object Recognition Memory

Note:Significant Group Differences in Bold; *,Reduced Performance in Insomnia Group.

Abbreviation: **Diagnostic Criteria:**DSM, Diagnostic and Statistical Manual of Mental Disorders; ICD, International Classification of Disease; ICSD, International Classification of Sleep Disorders. **Group:**C, Control Group; I, Insomnia Group; N/A, Not Applicable. **Insomnia scale:**AIS,Athens Insomnia Scale; APS,Arousal Predisposition Scale; BIQ,Basic Information Questionnaire; DBAS-16,The Dysfunctional Beliefs and Attitudes About Sleep Scale; EEG,electroencephalogram; ESS,Epworth Sleepiness Scale; ISI,Insomnia Severity Index; PCII,The Pre-Sleep Cognitive Intrusions Inventory; PSAS,Pre-Sleep Arousal Scale; PSG,polysomnography; PSQI, Pittsburgh Sleep Quality Index; RJSQ, Restless Legs Syndrome Quality of Life Instrument-Japanese version. **Cognitive scale:**HDS-R,Hasegawa dementia score-revised; MoCA, Montreal Cognitive Assessment; MoCA-C, Montreal Cognitive Assessment – Chinese-Beijing; MMSE, Mini-Mental State Examination. **Cognitive test:**ADSI, AD8 Dementia Screening Interview; AFT,Animal Fluency Test; ANT, Attention Network Task; SEVLT, Brief Spanish English Verbal Learning Test; BVRT, Benton Visual Retention Test; CDT,Clock drawing test; CVDT, Cued Visual Discrimination Task; COWAT, Controlled Oral Word Association Test; CPT, Conners Continuous Performance Test; CRT test, Choice Reaction Time test; CVLT, California Verbal Learning Test; CWST,Color-Word Stroop test; DSST, Digit Symbol Substitution Test; DST, Digit Span Task; IWR,Immediate 10-word recall; IRTM-item response theory methods; MAT,the Mental Alternation Test; PASAT, Paced Auditory Serial Addition Task; RAVLT,Rey Auditory Verbal Learning Test; ST,Stroop test; SDMT-Symbol Digit Modalities Test; THCAP, tHarmonized Cognitive Assessment Protocol; TCOT,Two-choice oddball task; TMT, Trail Making Test; TWFT, Thurstone Word Fluency Test; TSP,Task Switching paradigm; VLT,Verbal Learning Task; WFT,Word Fluency test; WMS,Wechsler Memory Scale; WLMT, Word List Memory Task.

Table 2
Included studies examining the impact of insomnia on the gray and white matter microstructure.

Author (Year/Country)	Study Type	Sample Size	Age	Age Group	Female	Diagnostic Criteria	Method	Result
Wei, Ruoqi et al. (2024 America) [19]	Cross-sectional study	PSG:632 MRI:160	I_1: 61 I_2: 67	Older Adults	PSG:55 % MRI:46 %	N/A	MRI、PSG -GM	↓ anterior cortex (anterior cingulate cortex and medial orbitofrontal cortex),thalamus Significant differences in thalamus, hippocampus, amygdala, brainstem, and total ventricle volumes
Li, Yanli et al. (2024 America) [49]	Case-control study	I:41 C:43	I: 24.95 ± 13.62 C: 27.51 ± 13.52	Middle-Aged Adults	I:70.7 % C:51.2 %	DSM-5	fNIRS-GM	↓ bilateral dorsolateral prefrontal cortex and bilateral medial prefrontal cortex
Yang, Lu et al. (2023 China) [39]	Case-control study	I:25 C:33	I:34.28 ± 11.34 C:36.06 ± 9.26	Middle-Aged Adults	N/A	ICSD-3	fMRI-GM	↓Left superior frontal gyrus, bilateral posterior cingulate cortex, orbitofrontal cortex
Dai, Xi-Jian et al. (2018 China) [50]	Case-control study	I:39 C:39	I: 48.9 ± 11.4 C: 47.9 ± 9.2	Middle-Aged Adults	I: 74 % C: 67 %	DSM-4	MRI-GM: GMV, VBM	No significant differences
Li, Shumei et al. (2016 China) [51]	Case-control study	I:30 C:23	I: 41.6 ± 9.5 C: 41.1 ± 10.5	Middle-Aged Adults	I:57 % C:40 %	DSM-4	MRI-WM: TBSS	↓ 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.
Joo, Eun Yeon et al. (2014 Korea) [46]	Case-control study	I:27 C:30	I: 51.2 ± 9.6 C: 50.4 ± 7.1	Middle-Aged Adults	I:93 % C:93 %	ICSD-2	MRI-GM: ROI – Hippocampus	↓Hippocampus
Bumb, Jan M et al. (2014 Germany) [52]	Case-control study	I:23 C:27	I: 43 ± 7.4 C: 39 ± 13.1	Middle-Aged Adults	I:48 C:59	DSM-4-TR, ICSD-2	MRI-GM: ROI – Pineal Gland	↓ Pineal gland
Spiegelhalder, Kai et al. (2014 Germany) [53]	Case-control study	I:24 C:35	I: 42.7 ± 14.5 C: 40.1 ± 9.1	Middle-Aged Adults	I:58 C:57	DSM-4-TR	MRI-WM: VW	↓FA in anterior internal capsule

Note: ↓, reduced in insomnia; AxD, axial diffusivity; FA, fractional anisotropy; GM, gray matter; GMV, total gray matter volume; MD, mean diffusivity; RD, radial diffusivity; ROI, region of interest; VBM, voxel-based morphometry; VW, voxel-wise; WM, white matter.

In order to compare differences among various age groups, participants were categorized into three age groups: young adults (aged 18–35 years), middle-aged adults (aged 36–64 years), and older adults (aged 65 years and above). The results indicated that 12 studies [18–20,22–24,26,28–30,34,42] focused on the older adult group, 21 studies [25,27,31,33,35–40,43–53] on the middle-aged adult group, and two studies [32,41] on the young adult group, with one study [21] remaining uncategorized due to missing age information. The findings revealed that cognitive decline was more pronounced in older adults with insomnia compared to both young and middle-aged adults. Among the older adult group, 66.67 % of the studies [18–20,23,24,28,34,42] demonstrated a significant correlation between insomnia and scores on the MMSE and MoCA tests, while only 33.33 % of the studies [31,37,39,40,44,45,48] in the adult group showed a similar correlation. Furthermore, studies involving young and middle-aged adults were more likely to report a relationship between insomnia and specific cognitive domains or functions.

3.6. Effects of cognitive decline on insomnia

In contrast to the extensive research on insomnia's effects on cognition, only three articles [20,26,31] explicitly addressed the impact of cognitive decline on sleep disorders in patients. Soomi Lee [20] proposed that approximately 31 % of patients with cognitive decline had a diagnosable sleep disorder, and individuals with dementia are known to experience various sleep disorders and other impairments in sleep function. The findings revealed that compared to both dementia patients and healthy individuals, those with MCI exhibited a higher prevalence of sleep disorders such as insomnia. Virginia T. Gallagher's research findings [26] suggested that individuals with MCI exhibit the greatest prevalence of insomnia symptoms in comparison to their healthy counterparts. The occurrence of excessive sleepiness or symptoms of sluggishness increases gradually from cognitively normal individuals to those with MCI and subsequently to dementia patients. Moreover, the presence of insomnia symptoms increases the risk of progression from a cognitively normal state to one of MCI. Wei Xu [29] proposed that sleep-related behaviors in cognitively normal elderly subjects are associated with increased levels of amyloid-beta peptide(Aβ) in the cerebrospinal fluid, and that insomnia can modulate the relationship between Aβ and cognitive decline.

3.7. Brain structure differences of insomnia-related cognitive decline

To understand the biological processes underlying insomnia-related cognitive deficits, several studies used MRI to investigate differences in brain structures between patients with insomnia and controls. As depicted in Table 2, eight studies examined differences in the gray matter volume and white matter microstructure between the insomnia and control groups [19,39,46,49–53]. The six studies [19,39,46,49,50,52] that examined gray matter found that patients with insomnia had reduced gray matter volume in the prefrontal cortex, cingulate gyrus, temporal lobe, and hippocampus compared to controls. However, it is important to note that these results were not consistent among all studies, particularly after adjusting for confounding factors such as age and spatial considerations. In Xi-Jian Dai's study [50], although no significant correlation was found between cognitive test results and gray matter volume, combined scores for nonverbal memory, language processing ability, and verbal fluency were associated with hippocampal and dentate gyrus atrophy. Three studies [19,39,46] reported associations between gray matter volume and cognitive performance. Lu Yang [39] reported that patients with insomnia exhibited reduced MoCA scores and decreased functioning in working memory, verbal information processing, verbal memory, verbal fluency, and visual memory. These authors suggested that alterations in connection patterns within

Table 3
Quality assessment of the included studies.

Study	Study Type	Evaluation Tool	Selection	Comparability	Outcome	Score
Overton, Marieclaire 2024 [18]	Cross-sectional study	NOS for Cross-sectional study	3	0	3	6
Wei, Ruoqi 2024 [19]	Cross-sectional study	NOS for Cross-sectional study	2	0	3	5
Lee, Soomi 2024 [20]	Cross-sectional study	NOS for Cross-sectional study	2	2	3	7
Correa-Muñoz, Elsa 2023 [21]	Cross-sectional study	NOS for Cross-sectional study	3	0	3	6
Wong, Roger 2023 [22]	Cross-sectional study	NOS for Cross-sectional study	4	2	3	9
Sánchez-García 2023 [23]	Cross-sectional study	NOS for Cross-sectional study	3	2	3	8
Selbaek-Tungevåg, Selma 2023 [24]	Cross-sectional study	NOS for Cross-sectional study	4	2	3	9
Zaheed, Afsara B 2023 [25]	Cross-sectional study	NOS for Cross-sectional study	4	0	3	7
Gallagher, Virginia T 2023 [26]	Cross-sectional study	NOS for Cross-sectional study	3	2	3	8
Zhang, Yuan 2023 [27]	Cross-sectional study	NOS for Cross-sectional study	3	2	2	7
Agudelo, C. 2022 [28]	Cross-sectional study	NOS for Cross-sectional study	3	2	3	8
Xu, Wei 2021 [29]	Cross-sectional study	NOS for Cross-sectional study	2	0	3	5
Resciniti, Nicholas V 2021 [30]	Cross-sectional study	NOS for Cross-sectional study	3	2	3	8
Fernandez-Mendoza, Julio 2021 [31]	Cross-sectional study	NOS for Cross-sectional study	3	2	3	8
Ballesio, Andrea 2020 [32]	Cross-sectional study	NOS for Cross-sectional study	2	0	3	5
Cross, Nathan E 2019 [33]	Cross-sectional study	NOS for Cross-sectional study	2	2	3	7
Hishikawa, Nozomi 2017 [34]	Cross-sectional study	NOS for Cross-sectional study	3	2	3	8
Palagini, Laura 2017 [35]	Cross-sectional study	NOS for Cross-sectional study	4	2	3	9
Li, Yun 2016 [36]	Cross-sectional study	NOS for Cross-sectional study	3	2	3	8
Palagini, Laura 2016 [37]	Cross-sectional study	NOS for Cross-sectional study	4	2	3	9
M.Subbotina 2015 R [38]	Cross-sectional study	NOS for Cross-sectional study	2	0	3	5
Yang, Lu 2023 [39]	Case-control study	NOS for Case-control study	3	2	3	8
Künstler, Erika C S 2023 [40]	Case-control study	NOS for Case-control study	4	2	3	9
Ding, Xiaobin 2023 [41]	Case-control study	NOS for Case-control study	3	1	3	7
Zhang, Li 2021 [42]	Case-control study	NOS for Case-control study	3	2	3	8
Kanady, Jennifer C 2017 [43]	Case-control study	NOS for Case-control study	4	2	3	9
Chen, Gui-Hai 2016 [44]	Case-control study	NOS for Case-control study	4	1	3	8
Fortier-Brochu 2014 [45]	Case-control study	NOS for Case-control study	2	1	3	6
Joo, Eun Yeon 2014 [46]	Case-control study	NOS for Case-control study	3	2	3	8
Liu, Hong 2014 [47]	Case-control study	NOS for Case-control study	4	1	2	7
Zhang, Ping 2018 [48]	Cohort study	NOS for Cohort study	4	2	3	9
Li, Yanli 2024 [49]	Case-control study	NOS for Case-control study	3	2	2	7
Dai, Xi-Jian 2018 [50]	Case-control study	NOS for Case-control study	4	2	3	9
Li, Shumei 2016 [51]	Case-control study	NOS for Case-control study	4	2	3	9
Bumb, Jan M 2014 [52]	Case-control study	NOS for Case-control study	3	2	3	8
Spiegelhalder 2014 [53]	Case-control study	NOS for Case-control study	4	2	2	8

and between the frontal and temporal lobes may underlie cognitive decline. In addition to gray matter changes, white matter integrity was also compromised in individuals with insomnia, as evidenced by reduced fractional anisotropy in key white matter tracts, such as the anterior and posterior limbs of the internal capsule, the superior longitudinal fasciculus, and the corpus callosum. These findings suggest that insomnia affects the brain's connectivity and communication pathways, which are essential for efficient cognitive processing. Although research on white matter microstructure was limited among the included studies, two diffusion tensor imaging studies [51,53] reported consistent findings that further supported this area of investigation.

4. Discussion

4.1. Impact of insomnia on cognitive function

The study demonstrates that insomnia detrimentally impacts cognitive function, leading to lower MMSE and MoCA scores among patients with insomnia. The included studies evaluated six cognitive domains in insomnia patients, revealing significantly impaired performance in attention, memory, visuospatial and executive function, and language memory. These factors are closely associated with cognitive decline and the onset of AD [54]. However, the studies exhibited heterogeneity, with only a few reporting statistically significant differences in orientation, attention, calculation, abstract thinking, and perceptual speed. A meta-analysis by Sophie Wardle-Pinkston [55] included 48 eligible studies that categorized cognitive function into objective and subjective aspects. The analysis identified varying degrees of sleep-related effects on working memory, episodic memory, problem-solving ability, vigilance, and complex attentional processing, as well as visuospatial and executive function. These findings are consistent with the results obtained in the present study.

Moreover, insomnia is not only associated with cognitive decline but may also accelerate the process of cognitive decline and increase the risk of developing AD [56]. The severity of insomnia is inversely correlated with cognitive function, with more pronounced deficits in attention, memory, and executive function observed in those with more severe insomnia. However, inconsistencies across studies [21,32,38,41,43] regarding its impact on specific cognitive functions, such as working memory and language, may stem from variations in assessment tools, cognitive tests, sample characteristics, and study designs. These discrepancies underscore the necessity for standardized methodologies and more rigorous research designs to better elucidate this complex relationship.

Subgroup analysis by age reveals that insomnia impacts cognitive function, particularly in older adults, as 66.67 % of studies [18–20,23,24,28,34,42] showed a correlation between insomnia and MMSE or MoCA scores. This finding suggests that insomnia is a critical factor in cognitive decline in older adults, consistent with Mander's research [57] indicating increased neurological susceptibility to insomnia's effects with aging. In contrast, only 33.33 % of studies [31,37,39,40,44,45,48] in middle-aged adults found similar correlations, possibly reflecting greater neurophysiological resilience, although specific cognitive domains may still be affected. Research [58] suggested that in middle-aged adults, insomnia primarily impacts specific cognitive functions, likely due to their higher cognitive flexibility and neuroplasticity. Additionally, differences in study results may also be attributed to variations in insomnia symptoms across age groups. Shi Le [59] indicated that the type of insomnia symptom changes as a function of age, with difficulties initiating sleep being more common among young adults and problems maintaining sleep occurring more frequently among middle-age and older individuals. These findings underscore the age-related

Table 4
Assessment tools for insomnia and cognitive function.

Assessment Tool	Description
Subjective Insomnia Assessment Tools	
Pittsburgh Sleep Quality Index (PSQI)	Assess overall sleep quality
Athens Insomnia Scale (AIS)	Measures the severity of insomnia
Insomnia Severity Index (ISI)	Evaluates the severity of insomnia symptoms
Bedtime Insomnia Questionnaire (BIQ)	Assesses insomnia symptoms specifically at bedtime
Anxiety and Preoccupation about Sleep (APS)	Assesses anxiety and preoccupation related to sleep
Restorative Sleep Questionnaire (RJSQ)	Evaluates the restorative quality of sleep
Epworth Sleepiness Scale (ESS)	Measures daytime sleepiness
Pre-Sleep Arousal Scale (PSAS)	Assesses pre-sleep physiological and cognitive arousal
Dysfunctional Beliefs and Attitudes about Sleep (DISRS)	Evaluates negative beliefs and attitudes towards sleep
Perceived Control over Insomnia Inventory (PCII)	Assesses perceived control over insomnia
Sleep Monitoring Technology	
Polysomnography (PSG)	Monitors physiological activities during sleep
Electroencephalogram (EEG)	Records brain electrical activity
Cognitive Assessment Questionnaire	
Montreal Cognitive Assessment (MoCA)	Screens for mild cognitive impairment
Mini-Mental State Examination (MMSE)	Assesses overall cognitive function
Hasegawa's Dementia Scale-Revised (HDS-R)	Evaluates dementia, especially in elderly patients
Wechsler Memory Scale - Third Edition (WMS-III)	Evaluates different aspects of memory function
Neuropsychiatric Inventory Questionnaire (NPI-Q)	Assesses psychiatric and behavioral symptoms
Cognitive Test	
ANT, CPT, CRT test, DSST, DST, N-back task, PASAT, ST, TSP, SDMT	Assesses cognitive domain 1: complex attention
CRT-test, CWST, TMT, MAT, Tower, TCOT, COWAT, WFT, TWFT	Assesses cognitive domain 2: executive function
BSEVLT, BVRT, CVLT, IWR, RAVLT, Recall, WMS, WLMT, VLT	Assesses cognitive domain 3: learning and memory
AFT, COWAT, WFT, TWFT	Assesses cognitive domain 4: language
CDT, CVDT, BVRT, Tower, TCOT	Assesses cognitive domain 5: perceptual-motor function
ADSI, THCAP	Assesses cognitive domain 6: social cognition

Note: Cognitive test abbreviations are as defined in Table 1.

heterogeneity in insomnia's cognitive effects. Older adults, with diminished neurophysiological reserve, are particularly vulnerable, highlighting the need for targeted interventions to delay cognitive decline. Middle-aged and young adults may exhibit domain-specific sensitivity to insomnia, necessitating focused cognitive assessments and interventions.

4.2. Impact of cognitive decline on insomnia

The study demonstrates that people with cognitive decline commonly suffer from insomnia, and there is a complex interrelationship between insomnia and cognitive decline. On one hand, cognitive decline itself can lead to sleep disorders, such as memory problems and emotional instability, which in turn can exacerbate insomnia and impact sleep quality. These problems are all closely related to cognitive function. On the other hand, sleep disorders can worsen cognitive decline. Cognitive decline weakens stress-coping abilities, intensifying stress responses—a key factor contributing to insomnia—thus creating a vicious cycle that complicates diagnosis and management. Vermunt L [60] showed that patients with cognitive decline undergo significant sleep pattern changes. In the presymptomatic stage, despite normal cognition, there is reduced sleep efficiency, less NREM3 and REM sleep, increased NREM1, and possible subclinical epileptiform activity due to neuronal hyperexcitability. In the prodromal stage, sleep disorders worsen with further NREM3 reduction, increased REM latency, and decreased sleep spindles, all linked to cognitive decline. In symptomatic dementia stage, sleep architecture is severely disrupted with major NREM3 and REM reductions, more nocturnal awakenings, frequent daytime naps, fragmented circadian rhythms, and increased epileptiform activity associated with cognitive decline.

Throughout the review process, we observed a relative paucity of research on insomnia in patients with cognitive decline, and the existing studies show varying results. The potential reasons for this may include the following: Despite being recognized as symptoms of cognitive decline, sleep disorders like insomnia may receive less clinical research focus compared to efforts to preserve and restore core cognitive functions. Additionally, diagnosing sleep disorders in this population is challenging due to the multifaceted symptoms often accompanying cognitive decline [61], further complicating the study of insomnia and resulting in fewer related studies. Furthermore, the heterogeneity among patients with cognitive decline complicates study design, further reducing the attention given to insomnia in specific subgroups.

4.3. Brain structure differences related to insomnia and cognitive decline

This study reveals significant impacts of insomnia on both gray and white matter in the brain. Structural changes were observed in the gray matter of insomnia patients, particularly in regions such as the prefrontal cortex, cingulate gyrus, temporal lobes, and hippocampus, including reductions in volume and alterations in morphology. Changes in white matter involved abnormalities in connectivity, microstructural alterations, reductions in white matter volume, and network dysfunction [62]. Notably, there was a significant decrease in anisotropy within the subcallosal frontal tract, which may impair cognitive responses, leading to cognitive decline, attentional deficits, and abnormalities in emotional regulation. Recent research suggests a potential association between chronic insomnia and brain atrophy, particularly in the structural integrity of gray and white matter [63]. Insomnia may affect brain structure through various mechanisms, including the induction of neuroinflammation [64], disruption of waste clearance processes [65], limitation of neuronal plasticity and regeneration [66], elevation of cortisol levels, and damage to white matter fiber tracts [67]. However, despite these proposed mechanisms providing a theoretical basis for understanding the link between insomnia and brain atrophy, current research findings remain inconsistent. This study also did not observe significant structural differences in brain MRI between insomnia and non-insomnia states in patients with cognitive decline. Further research is warranted to confirm and elucidate these findings.

4.4. Methodological considerations

The study utilizes a broad range of assessment tools for evaluating insomnia and cognitive function. Insomnia assessment tools, including subjective measures (PSQI, ISI, AIS) and objective tools (PSG), exhibit

strong validity and reliability across studies, enhancing research comparability. However, the distinct focus of each tool, such as PSQI on sleep quality, ISI on insomnia severity, and PSG on physiological data, can lead to varying outcomes depending on research objectives. Cognitive assessments primarily involve MoCA and MMSE, both of which are widely validated as reliable screening tools. Notably, MoCA showing superior sensitivity in detecting MCI [68]. It is important to note that this study employs a variety of cognitive tests, particularly domain-specific assessments like the RAVLT, TMT, and Stroop Test, which are recognized for their validity and reliability in assessing specific cognitive domains. However, their comparability may vary by domain [69]. For example, while both TMT and Stroop Test assess executive function, they focus on different processes (processing speed vs. inhibitory control), potentially leading to divergent outcomes. Similarly, the BVRT and CRT Test may yield varied results depending on the cognitive functions under investigation. To enhance accuracy and comparability, this study categorizes cognitive tests into six domains. Although these tools perform well for their intended purposes [70], future research should integrate subjective and objective assessments and adopt standardized protocols to improve reliability.

4.5. Clinical implications

The bidirectional relationship between cognitive decline and insomnia underscores the critical importance of effective screening and intervention strategies in clinical practice. Comprehensive screening and targeted treatment can enhance quality of life and potentially slow cognitive decline [71]. Specifically, assessing cognitive function in insomnia patients and screening for insomnia in those with cognitive decline are vital for holistic care. Secondly, early intervention and personalized treatment strategies are essential for managing insomnia and cognitive decline. According to Yo-El S Ju [72], timely management of insomnia, including cognitive behavioral therapy, sleep environment optimization, and non-benzodiazepine medications, can positively impact both sleep and cognitive function. Finally, given the variability among cognitive decline patients, prioritizing non-pharmacological interventions and tailoring treatments to individual needs can minimize adverse cognitive effects [73].

4.6. Future research directions

To advance our understanding of the relationship between insomnia and cognitive decline, several avenues for future research are recommended. Firstly, more longitudinal studies are needed to explore the causal relationship between insomnia and cognitive decline, clarifying whether insomnia directly contributes to cognitive decline or if other mediating factors are involved. Additionally, investigating the physiological and neurobiological mechanisms underlying this association is crucial, as it could inform the development of targeted therapeutic interventions [74]. Furthermore, long-term follow-up studies should assess the prolonged impact of insomnia on cognitive function, explore how different insomnia subtypes impact cognitive performance, and elucidate the reciprocal relationship between varying degrees of cognitive decline and insomnia. Moreover, further research should prioritize the development of personalized intervention strategies tailored to individuals' specific insomnia symptoms, cognitive status, and physiological characteristics to guide clinical practice [75].

4.7. Limitations of the study

This study has several limitations. Primarily, the predominance of cross-sectional designs among the included studies poses challenges in establishing causality and increases the risk of recall bias, misinterpretation of temporal sequences, and underrepresentation of rare events [76]. Additionally, the heterogeneity in insomnia definitions across studies, ranging from subjective self-reports to DSM-5 criteria, may create inconsistencies in findings and hinder comparability between research efforts. Furthermore, the variability in cognitive assessments employed across studies results in disparate outcomes, thereby impeding the synthesis of results and limiting the generalizability of conclusions. Finally, confounding variables such as demographic factors (including age, sex, and nationality) and disease characteristics (such as duration, severity, and treatment status) may independently influence both insomnia and cognitive decline, thereby complicating efforts to isolate the specific impact of insomnia on cognitive decline [77].

5. Conclusion

The findings of this scoping review indicate a potential bidirectional relationship between insomnia and cognitive decline. Insomnia may increase the risk of cognitive decline and appears to be associated with reduced gray matter volume and compromised white matter integrity in the brain, potentially leading to declines in attention, memory, visuospatial abilities, executive function, and verbal memory. Conversely, cognitive decline may contribute to the onset of insomnia, further diminishing sleep quality. Therefore, monitoring and managing sleep in individuals with cognitive decline could improve functional outcomes and prognosis, while also enhancing overall quality of life.

CRediT authorship contribution statement

Xiaotu Zhang: Writing – original draft, Visualization, Software, Resources, Investigation, Conceptualization. **Jiawei Yin:** Writing – review & editing, Visualization, Resources, Data curation. **Xuefeng Sun:** Writing – review & editing, Visualization, Resources, Data curation. **Zihan Qu:** Validation, Investigation. **Jindan Zhang:** Validation, Investigation. **Hongshi Zhang:** Writing – review & editing, Project administration, Methodology, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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