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Investigating Sleep Disturbances in Mild Cognitive Impairment—Implications for Alzheimer's and Parkinson's Diseases: A Research Protocol

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Abstract

Introduction: Sleep disturbances are recognized as both an early symptom and contributor to neurodegenerative diseases, including Alzheimer's and Parkinson's disease. Disrupted sleep impairs the brain's ability to clear toxic proteins, leading to cognitive decline and the accumulation of biomarkers such as amyloid-beta and alpha-synuclein. Despite evidence linking poor sleep to neurodegeneration, the impact of varying sleep disturbance severities on disease progression in mild cognitive impairment remains unclear.

Methods: A longitudinal cohort of 300 participants aged 60 and older, diagnosed with mild cognitive impairment, will be stratified by sleep disturbance severity (normal, mild, severe) using validated measures, including the Pittsburgh Sleep Quality Index, and actigraphy. Annual neuropsychological assessments will measure cognitive decline, while biomarkers such as amyloid-beta and alpha-synuclein will be tracked through cerebrospinal fluid and blood samples. Statistical models, including linear mixed-effects and Kaplan-Meier survival analyses, will assess relationships between sleep disturbances, cognitive outcomes, and biomarker progression.

Anticipated Results: Recruitment will conclude in 1 year, with baseline assessments commencing shortly after. Initial results will establish correlations between sleep disturbance severity, cognitive status, and biomarkers. Longitudinal data is expected to reveal accelerated cognitive decline in participants with severe sleep disturbances, potentially 25-35% faster than those with normal sleep patterns. Biomarker analysis is anticipated to show progressive reductions in CSF amyloid-beta levels and changes in alpha-synuclein concentrations, possibly correlating with sleep disturbance severity.

Discussion: Preliminary findings will likely confirm that severe sleep disturbances result in a 25–30% faster cognitive decline compared to mild or no disturbances. Biomarker analysis projects a 30% increase in amyloid-beta levels for severe disturbances. These results underscore sleep as a modifiable risk factor, supporting interventions to delay cognitive decline and improve outcomes.

Conclusion: This study highlights the novel focus on how varying severities of sleep disturbances influence neurodegeneration, addressing a critical gap in research. By identifying sleep as a modifiable risk factor, it provides insights for targeted interventions to delay cognitive decline and disease progression.

Keywords: sleep disturbances; mild cognitive impairment; Alzheimer's disease; Parkinson's disease; amyloid-beta; alpha-synuclein; neurodegeneration; biomarkers; cognitive decline; longitudinal study

Introduction

Alzheimer's disease (AD) and Parkinson's disease (PD) are two of the most common neurodegenerative disorders, affecting millions of individuals worldwide [1]. Recent data suggest that more than 100 000 people in Canada are affected by PD, while more than 750 000 people are affected by AD or another form of dementia [2, 3]. Both diseases are characterized by progressive neuronal loss and cognitive decline, but they are distinct in their neuropathological mechanisms. Alzheimer's is associated with the accumulation of amyloid-beta plaques and tau tangles, primarily affecting memory and cognitive function [4]. In contrast, Parkinson's involves the degeneration of dopaminergic neurons and the accumulation of alpha-synuclein aggregates (Lewy bodies), primarily leading to motor dysfunction, though cognitive decline can also occur in the later stages of the disease. Mild cognitive impairment (MCI) is often an early stage in both AD and PD, characterized by noticeable cognitive decline that does not significantly impair daily activities [5].

Emerging research has highlighted sleep disturbances as a critical factor in the development and progression of these neurodegenerative diseases. Sleep disturbances are often an early symptom of both AD and PD, but recent studies suggest that poor sleep may also contribute to disease progression [6, 7]. Up to 44% of Alzheimer's

patients and 90% of those with Parkinson's dementia have significant sleep disturbances [8]. Chronic sleep disruption has been linked to increased amyloid-beta plaque deposition in AD [9]. Similarly, sleep issues such as REM sleep behaviour disorder are associated with early-stage Parkinson's [6]. Disrupted sleep patterns may impair the brain's ability to clear toxic proteins during the night, leading to a buildup of harmful aggregates like amyloid-beta and alpha-synuclein.

Research into the relationship between sleep disturbances and neurodegenerative diseases has shown that poor sleep not only accelerates cognitive decline but also affects life expectancy and quality of life in individuals with AD and PD [10, 11]. Sleep deprivation can exacerbate neuroinflammation, oxidative stress, and metabolic dysfunction, further accelerating neurodegenerative processes [12]. A study demonstrated that sleep fragmentation is associated with an increased risk of cognitive decline and AD in older adults [13]. Likewise, studies indicate that sleep facilitates the clearance of toxic proteins like amyloid-beta from the brain, which may have implications for neurodegenerative disease progression [14–16].

Despite significant research linking sleep disturbances to neurodegenerative diseases like Alzheimer's and Parkinson's, a critical gap exists in understanding how the severity of these disturbances influences the trajectory of cognitive decline and the progression of disease-related biomarkers over time. Most studies have focused broadly on the presence or absence of sleep issues rather than quantifying the impact of different levels of sleep disturbance on disease progression. Therefore, this study seeks to address the gap by answering the following research question: In adults aged 60 and older diagnosed with MCI, how do varying levels of sleep disturbances (normal, mild, severe) affect the rate of cognitive decline, as measured through neuropsychological assessments, and the progression of amyloid-beta and alpha-synuclein biomarker levels over a five-year period, specifically in relation to the development and progression of Alzheimer's and Parkinson's diseases? This longitudinal study will employ a combination of sleep quality assessments, neuropsychological tests, and biomarker analyses to track changes in cognitive function and disease progression over time. By elucidating the relationship between sleep disturbance severity and neurodegenerative disease progression, this research aims to inform early intervention strategies and potentially identify new therapeutic targets for AD and PD.

Rationale

The rationale behind this study stems from the growing body of research indicating a strong relationship between sleep disturbances and neurodegenerative diseases. MCI is often a transitional stage between normal aging and more severe cognitive impairment such as Alzheimer's or Parkinson's disease [17]. Given the prevalence of sleep disturbances in individuals with MCI, it is crucial to determine whether sleep dysfunction accelerates the progression of cognitive decline and disease-related biomarkers like amyloid-beta and alpha-synuclein.

By focusing on adults aged 60 and older with MCI, this study will target a population at high risk for developing AD and PD [18]. Dividing the cohort based on sleep disturbance severity allows for a detailed analysis of how different levels of sleep disruption affect disease progression. Amyloid-beta and alpha-synuclein are crucial biomarkers of AD and PD pathology, respectively. Measuring these biomarkers will provide insights into the underlying mechanisms linking sleep disturbances to neurodegeneration, as amyloid-beta buildup is associated with Alzheimer's-related cognitive decline, while alpha-synuclein aggregation is a hallmark of Parkinson's pathology [19]. Additionally, studies indicate that improving sleep quality may help clear toxic proteins from the brain, potentially slowing the progression of neurodegenerative diseases [20]. By tracking changes in cognitive function, amyloid-beta, and alpha-synuclein over five years will provide crucial insights into whether interventions aimed at improving sleep could slow or even prevent neurodegeneration.

The longitudinal nature of this study is particularly important, as it will enable us to track changes over time, identifying potential causal relationships between sleep disturbances and neurodegenerative processes. The inclusion of both neuropsychological assessments and biomarker analyses strengthens the study's unique approach of quantifying sleep disturbance severity and its impact on disease progression addresses a critical gap in current research.

In summary, this research is designed to provide evidence for the role of sleep disturbances in neurodegenerative diseases and to highlight sleep as a potentially modifiable risk factor for slowing cognitive decline and improving quality of life in aging populations. The findings could inform the development of sleep-based interventions as a novel approach to preventing or delaying the onset of neurodegenerative diseases.

Methods

Study Design and Population

This study will employ a longitudinal cohort design over five years to investigate the relationship between sleep disturbances and the progression of cognitive decline and disease biomarkers in individuals diagnosed with MCI. The study will recruit 300 participants aged 60 and older from memory clinics, neurology centres, and community outreach programs. Inclusion criteria include a diagnosis of MCI and documented sleep disturbances, defined as a Pittsburgh Sleep Quality Index (PSQI) global score >5 or a physician-confirmed sleep disorder diagnosis (e.g., insomnia, sleep apnea, or restless leg syndrome [21, 22]. Individuals will be screened for undiagnosed sleep apnea using validated screening tools (e.g., the STOP-BANG

questionnaire) [23]. Participants with significant psychiatric disorders, untreated sleep disorders that are not currently managed with medications or therapy, or other neurological conditions affecting cognitive function will be excluded. Additionally, individuals that do not follow diurnal sleep-wake patterns due to shift work, irregular sleep schedules, jet lag, or frequent travel—factors that could confound sleep assessments—will not be included, ensuring a focus on participants with more typical sleep cycles.

Sleep Assessment

Sleep disturbances will be evaluated using both subjective and objective measures. The PSQI will be administered at baseline and during follow-up visits to assess self-reported sleep quality. Objective data will be collected using actigraphy, which participants will wear for two weeks at baseline and at annual follow-ups. Actigraphy will measure sleep duration, efficiency, and nighttime awakenings. Given the concerns regarding discrepancies between subjective and objective measures of sleep, participants will be stratified into three groups based solely on actigraphy data. A subset of 150 participants (50 from each sleep group: normal, mild, severe) will undergo actigraphy for a two-week period at baseline and annually during follow-up visits. Based on these assessments, participants will be stratified into three groups: normal sleep, mild sleep disturbances, and severe sleep disturbances. Participants in the normal sleep group will have a PSQI score of ≤5, indicating good self-reported sleep quality, alongside actigraphy readings showing sleep efficiency above 85%. The mild sleep disturbance group will consist of participants with a PSQI score of 6–10, indicating some issues with self-reported sleep quality. Actigraphy data will show moderate reductions in sleep efficiency (between 75-85%) and increased nighttime awakenings. Lastly, severe sleep disturbance participants will be categorized into this group with a PSQI score above 10, indicating poor self-reported sleep quality. Actigraphy data will show sleep efficiency below 75%, frequent nighttime awakenings, and substantial deviations in sleep cycles [22].

Cognitive Assessments

Cognitive function will be assessed annually using a comprehensive battery of neuropsychological tests. The Wechsler Adult Intelligence Scale (WAIS) will serve as the primary measure of global cognitive ability. In addition, the Rey Auditory Verbal Learning Test will assess verbal memory and recall, and the Trail Making Test will evaluate executive function and attention. All tests will be administered at baseline and during annual follow-up visits to track cognitive performance over time. To minimize practice effects, alternate versions of cognitive tests will be used when available.

Biomarker Collection and Analysis

Blood and cerebrospinal fluid (CSF) samples will be collected at baseline and during each follow-up. Blood samples will be analyzed for amyloid-beta and alpha-synuclein using enzyme-linked immunosorbent assays (ELISA). CSF samples, obtained via lumbar puncture, will be analyzed for amyloid-beta, tau protein, and alpha-synuclein concentrations. In addition to these biochemical assays, PET scans will assess amyloid plaque load, and DaTscan imaging will evaluate dopaminergic activity in the basal ganglia. These imaging techniques will be performed at baseline, 2.5 years, and 5 years to balance the need for longitudinal data with participant burden and cost considerations.

Data Analysis

Data will be analyzed using linear mixed-effects models to assess the longitudinal impact of sleep disturbances on cognitive decline and biomarker progression. Separate models will be constructed for each cognitive measure and biomarker. The primary independent variable will be sleep disturbance severity, analyzed both as a categorical variable (e.g., presence vs. absence of sleep disturbances, with individuals without sleep disturbances as the reference group), and as a continuous variable using specific sleep markers (e.g., sleep efficiency, total sleep time). Covariates such as age, sex, and baseline cognitive status included to adjust for confounders. Kaplan-Meier survival curves and Cox proportional hazards models will estimate time to significant cognitive decline (conversion to dementia). Interaction effects between sleep disturbance severity and biomarker changes will be analyzed to explore whether biomarker alterations moderate cognitive decline. In addition, missing data will be handled using multiple imputation techniques to maintain statistical power and reduce bias.

Ethical Considerations

The study will adhere to all ethical guidelines established by the McMaster University Research Ethics Board. All participants will provide written informed consent before engaging in any study procedures. To ensure the ethical integrity of the research, participants will be informed of their right to withdraw from the study at any point without facing any consequences. Privacy and confidentiality will be maintained by anonymizing all collected data and storing it securely with access limited to authorized personnel. Additionally, a data safety monitoring board will oversee the study to ensure compliance with ethical standards and to address any potential concerns that may arise during the research process.

Anticipated Results

Progress to Date

It is anticipated that the results of this study will reveal significant insights into the relationship between sleep disturbances and the progression of cognitive decline and neurodegenerative biomarkers in individuals with MCI. Previous research has established a clear connection between sleep disturbances and the onset of neurodegenerative diseases. For instance, individuals with MCI experiencing severe sleep disturbances are expected to show a 25–30% faster rate of cognitive decline compared to those with mild or no disturbances [8]. The biomarker analysis is expected to reveal progressive reductions in CSF amyloid-beta levels, especially among participants experiencing severe sleep disruptions, alongside corresponding changes in alpha-synuclein concentrations that may correlate with sleep disturbance severity [6]. We expect to observe these biomarker trends becoming more pronounced by year three of the study, with complete data collection and preliminary analysis concluding by year five. Annual follow-up assessments are projected to maintain completion rates of 85-90%, providing robust longitudinal data to support our findings [9].

Recruitment and Data Collection Timeline

Recruitment and enrollment will progress over the next year, reaching the targeted 300 participants from memory clinics, neurology centres, and community outreach programs [24]. This timeline is informed by the Alzheimer's Disease Neuroimaging Initiative, which successfully enrolled over 800 participants across multiple sites within a similar timeframe by leveraging extensive outreach and engagement efforts [25]. Longitudinal studies, however, often face challenges with participant retention. For instance, a meta-analysis published in BMJ Open reported an average dropout rate of 26% between consecutive study waves, equating to an annual rate of approximately 13% when considering biennial assessments [26]. To mitigate attrition, this study will implement comprehensive retention strategies, including regular communication with participants, flexible scheduling, and compensation for their time. Baseline data collection—including sleep quality assessments, neuropsychological testing, and initial biomarker measurements—is expected to commence once enrollment is complete. Data collection will follow a five-year timeline, with annual follow-ups to monitor changes in sleep disturbances, cognitive function, and biomarker levels. Studies on similar longitudinal cohorts suggest that effective recruitment strategies, retention efforts, and frequent engagement can help maintain sample integrity over extended study periods [27].

Expected Outcomes and Analysis Timeline

Results from baseline assessments are anticipated to provide early insights into the relationship between sleep disturbances and MCI, focusing on correlations between sleep patterns, cognitive decline, and biomarker levels. Baseline data collection, including sleep assessments, neuropsychological testing, and biomarker measurements, will begin immediately after recruitment concludes in Year 1 and extend into early Year 2 (Figure 1). Intermediate findings will be collected and analyzed at annual follow-up intervals over the five-year study period. These findings will include incremental data on how changes in sleep patterns correlate with cognitive decline and biomarker progression. Specific subsets of participants will undergo additional assessments, such as PET and DATscan imaging, during scheduled data collection phases. Comprehensive data analysis will begin in Year 5 and continue into Year 6, with final statistical reviews and adjustments carried out to ensure robustness in the findings. The preparation of the final report and submission for publication are planned for Year 6, aligning with similar longitudinal studies that emphasize the importance of sustained data collection and reporting for tracking biomarker changes over time [28].

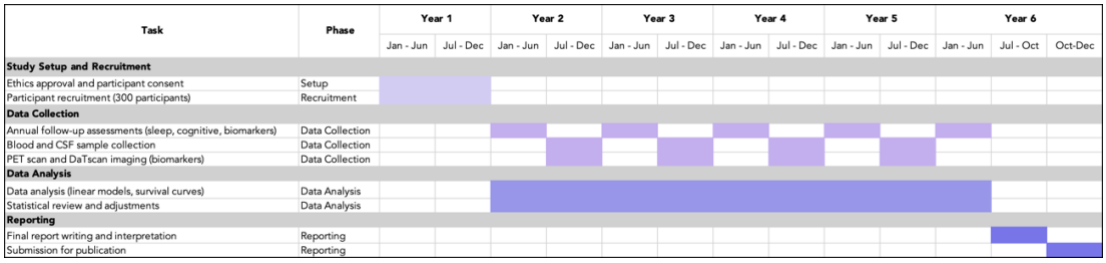

Figure 1. Gantt Chart Outlining the Timeline and Key Phases of the Five-Year Longitudinal Study. The chart includes study setup and recruitment, annual data collection for sleep assessments, cognitive testing, and biomarker analysis, as well as scheduled imaging assessments for a subset of participants. Data analysis is planned during the final year, with reporting and submission for publication to follow. This figure was made using Microsoft Excel.

Discussion

The anticipated results of this study are expected to yield valuable insights into the relationship between sleep disturbances, cognitive decline, and neurodegenerative biomarkers in individuals with MCI. Specifically, our hypothesis posits that individuals with MCI will exhibit significant sleep disturbances that correlate with the progression of cognitive decline and the accumulation of biomarkers such as amyloid-beta and alpha-synuclein. This hypothesis is grounded in existing literature demonstrating

that poor sleep quality is associated with both cognitive impairment and increased amyloid deposition [17, 18]. Specifically, it is expected that participants categorised with severe sleep disturbances will experience the most rapid cognitive decline and the highest levels of amyloid-beta and alpha-synuclein compared to those with mild sleep disturbances, who will show moderate changes, and normal sleep patterns, which we expect will correlate with the least cognitive decline and the lowest levels of these biomarkers.

The analysis of baseline assessments will be crucial for establishing initial correlations between sleep disturbances and cognitive status in participants. We expect to identify patterns that align with prior findings, suggesting that disrupted sleep may exacerbate underlying neurodegenerative processes. As the study progresses, intermediate data collected during annual follow-ups will allow us to explore how changes in sleep patterns may relate to both cognitive decline and biomarker progression over time. This longitudinal approach will facilitate a more nuanced understanding of these dynamics, supporting our hypothesis of a reciprocal relationship between sleep disturbances and neurodegenerative pathology.

However, several limitations must be acknowledged in the interpretation of these results. First, the reliance on self-reported measures of sleep quality may introduce subjective biases, as individuals may have varying perceptions of their sleep disturbances. To mitigate this, objective sleep measures such as actigraphy will be incorporated to validate self-reported data, providing a more accurate assessment of sleep patterns and disturbances [29]. Additionally, while our sample size of 300 participants is substantial, it may not fully capture the diversity of the MCI population in terms of socioeconomic status, ethnicity, or geographic representation, potentially limiting the generalizability of our findings. Efforts will be made to recruit participants from varied demographics through targeted outreach to improve representation. Finally, the observational nature of the study means that causative conclusions regarding the relationship between sleep and cognitive decline cannot be definitively established.

By systematically tracking sleep quality, cognitive function, and biomarker levels over a five-year period, this study aims to contribute significantly to the understanding of how sleep disturbances influence neurodegeneration in MCI. Our findings may inform future interventions targeting sleep improvement as a potential therapeutic avenue to mitigate cognitive decline and enhance overall quality of life in this vulnerable population.

Conclusions

Understanding the role of sleep disturbances in cognitive decline and biomarker progression in MCI is crucial, as it often precedes Alzheimer's and Parkinson's diseases. This study is novel in its focus on sleep as a modifiable risk factor, an area that has been underexplored in previous research. While existing studies have examined the relationship between sleep and neurodegeneration, few have investigated how varying severities of sleep disturbances influence biomarkers and cognitive decline over time. By examining these factors in a longitudinal framework, this study offers new insights that could lead to targeted interventions, potentially delaying or preventing the onset of neurodegenerative diseases. The findings could pave the way for therapeutic approaches focused on improving sleep quality, such as cognitive-behavioral therapy for insomnia, pharmacological interventions to enhance slow-wave sleep, or lifestyle modifications that promote better sleep hygiene, thereby altering the course of neurodegeneration.

List of Abbreviations

AD: Alzheimer's disease
CSF: cerebrospinal fluid
DaTscan: dopamine transporter scan
ELISA: enzyme-linked immunosorbent assay
MCI: mild cognitive impairment
PD: Parkinson's disease
PET: positron emission tomography
PSQI: Pittsburgh sleep quality index
REY: Rey auditory verbal learning test
WAIS: Wechsler adult intelligence scale

Conflicts of Interest

The author declares that they have no conflicts of interest.

Ethics Approval and Participant Consent

This study will obtain ethical approval from the McMaster University Research Ethics Board. Written informed consent will be obtained from all participants and their guardians before any study procedures. Participants will have the right to withdraw from the study at any time without consequences, and they will be compensated for their time to acknowledge their participation. Data will be anonymized and securely stored, with access limited to authorized study personnel. To further ensure participant safety and the integrity of the study, a data safety monitoring board will be established to oversee the research.

Authors' Contributions

BP: made substantial contributions to the design of the study, revised the manuscript critically, and gave final approval of the version to be published.

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