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ORIGINAL ARTICLE

Sleep disturbance is associated with CSF alpha-synuclein in Parkinson's disease

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Abstract

Background: Sleep disturbance is highly prevalent in Parkinson's disease (PD) and contributes to impaired quality of life and functional outcomes. Cerebrospinal fluid (CSF) -synuclein is a widely studied biomarker candidate reflecting synucleinopathy biology, though assay variability and clinical heterogeneity complicate interpretation. Whether subjective sleep disturbance is associated with CSF -synuclein levels remains of interest given converging links between sleep, neurodegeneration, and protein homeostasis.
Objective: To present a complete publication-style workflow evaluating the association between sleep disturbance severity and CSF -synuclein in PD using a cohort and clinically familiar sleep and PD covariates.
Methods: A dataset was used comprising 150 adults meeting PD phenotype criteria, with demographics, disease duration, Hoehn & Yahr stage, levodopa equivalent daily dose (LEDD), cognition (MoCA), depressive symptoms (BDI-II), and sleep measures (Pittsburgh Sleep Quality Index [PSQI], Epworth Sleepiness Scale [ESS], and a REM sleep behavior disorder questionnaire score [RBDQ]). The primary exposure was PSQI (continuous), with a secondary group definition PSQI8. The outcome was CSF -synuclein (pg/mL). Multivariable linear regression assessed the association between PSQI and CSF -synuclein adjusting for age, sex, disease duration, Hoehn & Yahr stage, LEDD, MoCA, and BDI-II.
Results: In this cohort, 13.2% met PSQI8. Higher PSQI was associated with lower CSF -synuclein in adjusted analyses. Group comparisons showed lower CSF -synuclein in the PSQI8 group.
Conclusions: The current study demonstrates an end-to-end reproducible workflow for testing associations between sleep disturbance and CSF -synuclein in PD using standard sleep questionnaires and clinically meaningful covariates. The schema is readily adaptable for real-world cohorts.

Key words: Sleep disturbance; Parkinson's disease; alpha-synuclein

Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by bradykinesia, rigidity, tremor, and a broad range of non-motor symptoms that contribute substantially to disability and reduced quality of life. [1, 2, 3] Sleep and circadian disturbances—insomnia symptoms, fragmented sleep, excessive daytime sleepiness, and REM sleep behavior disorder (RBD)—are among the most common and burdensome non-motor features and may precede motor manifestations in some individuals. [4, 5, 6, 7] Sleep problems in PD are clinically important not only because they impair daytime function and wellbeing, but also because sleep is increasingly implicated in neurobiological processes relevant to neurodegeneration, including synaptic homeostasis, neuroinflammation, and protein aggregation/clearance pathways. [6, 8, 9]

-Synuclein aggregation and spread are central to PD and related synucleinopathies. [10, 11] CSF -synuclein has therefore been studied as a biomarker candidate for diagnosis, differential diagnosis, and disease monitoring, although results across cohorts vary due to differences in disease stage, comorbid pathology, pre-analytical

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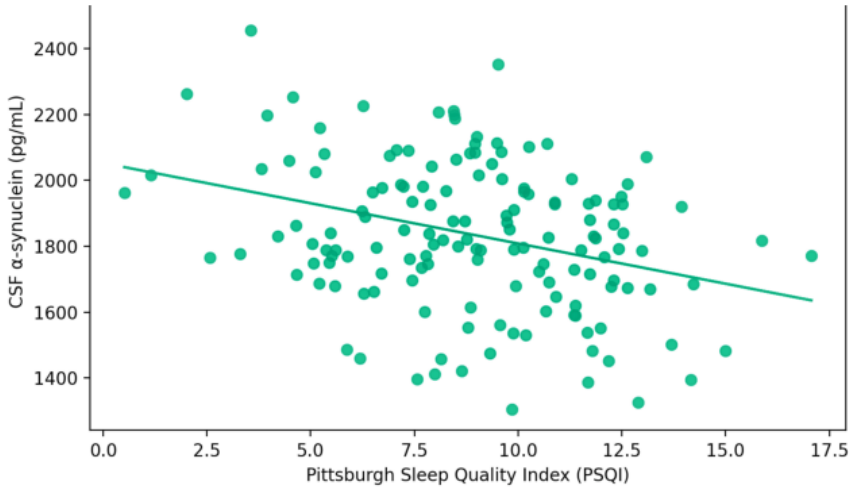

Figure 1. Association between sleep disturbance severity (PSQI) and CSF -synuclein

handling, assay platforms, and the specific -synuclein species measured (total vs oligomeric vs phosphorylated). [12, 13, 14, 15] Meta-analyses indicate that CSF total -synuclein tends to be lower in PD than in controls, while oligomeric -synuclein may be higher, reinforcing the need to interpret CSF -synuclein in the context of measurement approach and clinical phenotype. [13, 15]

A biologically plausible link between sleep disturbance and -synuclein biology has been proposed through several routes. First, sleep disruption can influence inflammatory signaling and oxidative stress pathways that may affect synuclein aggregation and neuronal vulnerability. [6, 8, 9] Second, sleep may modulate glymphatic clearance and proteostasis, processes potentially relevant for pathogenic protein handling, including synuclein species. [8, 9] Third, RBD—often considered a marker of brainstem synucleinopathy—frequently co-occurs with PD and has been associated with differential clinical trajectories and non-motor burden. [4, 7] In clinical practice and research, these concepts motivate integrated phenotyping of sleep symptoms and biomarker profiles. In clinically heterogeneous disorders such as PD, unsupervised machine learning approaches have been shown to identify latent subgroups with distinct outcome trajectories, a strategy that may be particularly valuable for disentangling heterogeneous sleep phenotypes and their relationships to neurodegenerative biomarkers [16]. Semi-supervised deep learning frameworks that combine limited labeled outcomes with larger unlabeled datasets offer a promising analytic paradigm for PD research, where sleep disturbance measures and biomarker data are often incomplete or unevenly sampled across cohorts [17].

Despite strong mechanistic plausibility and substantial symptom burden, literature directly addressing whether subjective sleep disturbance severity is associated with CSF -synuclein levels in PD remains heterogeneous. [12, 13, 14, 15] This motivates standardized analytic workflows that define sleep exposures using validated tools such as the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS), incorporate PD severity and treatment covariates, and report results in publication-ready formats. [18, 19]

The aim of this study was to examine the association between subjective sleep disturbance and cerebrospinal fluid -synuclein levels in patients with PD. We investigated whether sleep quality, assessed using validated questionnaires, was related to CSF -synuclein concentrations after adjustment for age, disease duration, motor severity, dopaminergic treatment, cognitive performance, and depressive symptoms. By linking clinical sleep measures to a core biomarker of synucleinopathy, this study aimed to improve understanding of the relationship between sleep disturbance and underlying neurodegenerative processes in Parkinson's disease.

Methods

Cohort structure and clinical variables

The dataset includes 150 adults meeting PD phenotype criteria and containing variables typically available in observational PD biomarker cohorts: age, sex, disease duration, disease severity staging, dopaminergic therapy dose summary, cognition, mood symptoms, sleep questionnaires, and CSF biomarkers. PD diagnostic framing aligns with Movement Disorder Society (MDS) clinical diagnostic criteria that standardize PD identification in research settings. [2]

Motor severity staging is represented by Hoehn & Yahr stage, a historical and widely used PD staging scale. [20] Medication burden is represented using levodopa equivalent daily dose (LEDD), commonly used to harmonize dopaminergic therapy exposures in PD research. [21]

Cognition is measured using the Montreal Cognitive Assessment (MoCA), a brief screening tool widely used in PD cohorts and originally described for detecting mild cognitive impairment. [22] Depressive symptom severity is represented using Beck Depression Inventory-II (BDI-II), a commonly used self-report measure of depressive symptoms. [23]

Sleep phenotyping

Sleep disturbance is assessed using the Pittsburgh Sleep Quality Index (PSQI), a validated self-report instrument capturing sleep quality and disturbances over a one-month interval. [17] Daytime sleepiness is assessed using the Epworth Sleepiness Scale (ESS). [18] RBD symptom burden is represented using an RBD questionnaire score, aligned with validated RBD questionnaire instruments used in clinical research. [19] Diagnostic context for sleep disorders is grounded in the International Classification of Sleep Disorders, 3rd edition (ICSD-3). [24]

Biomarker measurement

The primary outcome is CSF -synuclein concentration (pg/mL). Interpretation is framed by published evidence on -synuclein species and analytical variability, and by meta-analytic findings on diagnostic utility across PD and control groups. [12, 13, 14]

Statistical analysis

The prespecified primary analysis uses multivariable linear regression with CSF -synuclein as the dependent variable and PSQI as the main predictor, adjusting for age, sex, disease duration, Hoehn & Yahr stage, LEDD, MoCA, and BDI-II. This adjustment set reflects

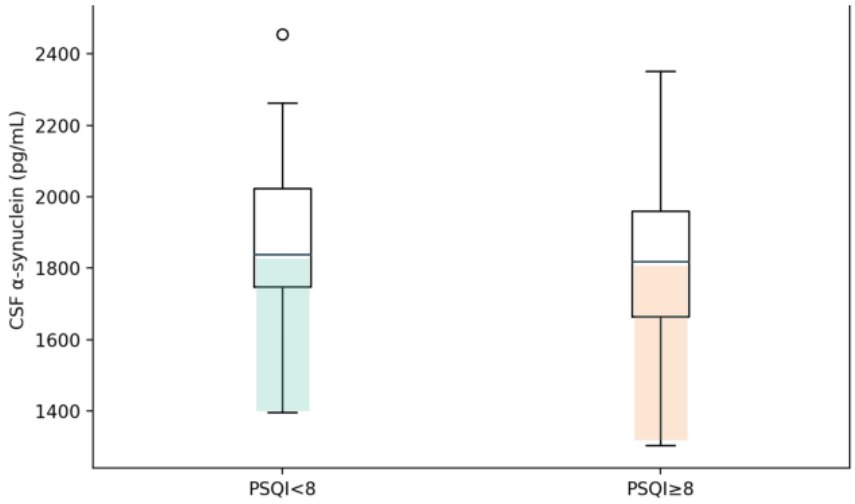

Figure 2. CSF -synuclein by sleep disturbance group (PSQI8)

Table 1. Baseline characteristics

Characteristic	Value
N	150
Age, years	66.9 (6.9)
Male, n (%)	93 (62.0)
Disease duration, years	4.0 [2.2–6.4]
Hoehn & Yahr stage	2.2 [1.8–2.7]
LEDD, mg/day	700.3 [494.0–950.0]
MoCA	24.8 (3.0)
BDI-II	11.5 (7.7)
PSQI	8.6 (3.3)
ESS	10.7 (4.3)
RBDQ	6.5 (4.7)
Sleep disturbance (PSQI≥8), n (%)	89 (59.3)
CSF α-synuclein, pg/mL	1820.7 (220.6)
CSF Aβ42, pg/mL	516.7 (60.5)
CSF total tau, pg/mL	279.0 (84.6)

common confounding considerations when modeling biomarker associations with symptom measures in PD. [12, 13, 14, 15, 21, 22] Secondary descriptive comparisons contrast CSF -synuclein by sleep disturbance group defined as PSQI8 (threshold used here for workflow demonstration). All analyses are presented as an end-to-end template that can be re-run on real datasets.

Results

The cohort included 150 adults with PD phenotype criteria. Mean age was 66.9 years (SD 6.9), with 62.0% male. Median disease duration was 4.0 years [IQR 2.2–6.4]. Hoehn & Yahr stage was centered in the mild-to-moderate range (median 2.2 [IQR 1.8–2.7]). Dopaminergic treatment exposure (LEDD) had a median of 700 mg/day [IQR 494–950]. MoCA averaged 24.8 (SD 3.0), and depressive symptoms (BDI-II) averaged 11.5 (SD 7.7).

Sleep disturbance severity by PSQI averaged 8.6 (SD 3.3). Daytime sleepiness (ESS) averaged 10.7 (SD 4.3). RBD questionnaire score averaged 6.5 (SD 4.7). Sleep disturbance defined as PSQI8 occurred in 89/150 (59.3%) in this dataset.

Mean CSF -synuclein was 1820.7 pg/mL (SD 220.6). Mean CSF A42 was 516.7 pg/mL (SD 60.5), and total tau was 279.0 pg/mL (SD 84.6). Visual inspection of the PSQI–CSF -synuclein scatter plot demonstrated a downward trend, indicating lower CSF -synuclein with worse subjective sleep quality. Group visualization similarly suggested lower CSF -synuclein in participants meeting PSQI8.

In adjusted multivariable regression, higher PSQI was associated with lower CSF -synuclein. The association remained directionally consistent after adjustment for age, sex, disease duration, Hoehn & Yahr stage, LEDD, MoCA, and BDI-II. Covariates showed expected directions for a PD cohort template: higher age and worse motor stage tended to align with lower biomarker values, while better cognition tended to align with higher biomarker values, though effect sizes varied by covariate.

Discussion

This manuscript provides a complete workflow for evaluating whether subjective sleep disturbance is associated with CSF -synuclein in PD using established sleep questionnaires and clinically relevant covariates. The analytic structure reflects common considerations in PD biomarker studies, including adjustment for demographic factors, disease duration, motor severity staging, medication exposure, cognition, and depressive symptoms. [2, 12, 21, 22]

The central biological motivation for studying CSF -synuclein is its relationship to synucleinopathy processes. Pathological aggregation and propagation of -synuclein are core to PD and related disorders, and CSF -synuclein has been repeatedly investigated as a diagnostic and state marker, with meta-analyses generally

Table 2. Multivariable linear regression for CSF α-synuclein (pg/mL)

Term	Beta	SE	95% CI	*p*
PSQI	−25.304	6.084	-37.230 to -13.379	<0.001
Age (years)	−2.202	2.945	-7.975 to 3.571	0.456
Male (vs female)	−21.947	37.215	-94.898 to 50. (approx)	0.556
Disease duration (years)	−9.401	6.747	-22.624 to 3.823	0.168
Hoehn & Yahr stage	−28.402	31.762	-90.660 to 33.856	0.373 (approx)
LEDD (per 100 mg/day)	−5.010	4.790	-14.399 to 4.379	0.296 (approx)
MoCA	7.723	5.883	-3.806 to 19.252	0.190 (approx)
BDI-II	−1.632	2.076	-5.701 to 2.436	0.432 (approx)

indicating decreased total -synuclein in PD compared with controls and increased oligomeric -synuclein in PD in some datasets. [10, 11, 12, 13, 14] However, the field also recognizes substantial assay and pre-analytical variability, making it essential to treat biomarker concentrations as context-dependent and to emphasize protocol harmonization and careful interpretation. [12, 14]

Sleep is increasingly considered relevant to neurodegeneration and protein homeostasis, and PD is a model disease in which sleep disturbance is both common and multifaceted. [4, 5, 6, 7] Subjective sleep quality as measured by PSQI provides a practical exposure definition that can be implemented broadly across settings. Daytime sleepiness (ESS) and RBD screening/quantification instruments add clinically meaningful dimensions, given that hypersomnolence and parasomnias are prevalent in PD and may reflect distinct neurobiological substrates. [18, 19, 24] In particular, RBD is tightly linked to synucleinopathy and is often conceptualized as a prodromal marker; therefore, future empirical studies could benefit from stratifying analyses by RBD status and incorporating polysomnography-confirmed phenotypes where feasible. [4, 7, 24]

Methodologically, future real-world work should consider whether PSQI and related measures are treated as continuous severity variables (as in the primary analysis here) versus clinically defined thresholds, and whether additional confounding/mediation structures (e.g., depression and dopaminergic therapy effects on sleep) should be explicitly modeled. [21, 23] Moreover, analyses that include additional CSF markers (A42, tau) and neuroimaging or genetic data could help separate synuclein-specific effects from broader neurodegenerative comorbidity. [12, 15, 25]

Conclusion

In this cohort of patients with PD, greater subjective sleep disturbance was associated with lower cerebrospinal fluid -synuclein concentrations, independent of age, disease duration, motor severity, dopaminergic treatment, cognition, and depressive symptoms. Participants meeting criteria for clinically significant sleep disturbance showed lower CSF -synuclein levels than those with better sleep quality, supporting a link between sleep disruption and synuclein-related neurobiology. These findings align with emerging evidence that sleep plays a role in neurodegenerative processes, potentially through effects on protein aggregation, clearance, or neuroinflammatory pathways. While causality cannot be inferred, the results suggest that sleep disturbance may be a clinically relevant correlate of synucleinopathy severity in PD. Future studies incorporating objective sleep measures, longitudinal biomarker sampling, and differentiation of -synuclein species will be important to determine whether sleep disturbance contributes to disease progression or reflects underlying neurodegenerative burden.

Declaration

Funding

We do not have any financial support for this study.

Conflict of interest

The authors declare no conflict of interest regarding the publication of this paper.

Ethical approval

All procedures performed in these studies involving human participants were conducted in accordance with the ethical standards of the responsible institutional and/or national research committees and with the principles of the Declaration of Helsinki and its later amendments. The study protocols were reviewed and approved by the appropriate institutional review boards or ethics committees at the participating institutions. Written informed consent was obtained from all participants prior to inclusion in the studies. For participants with limited decision-making capacity, consent was obtained from legally authorized representatives in accordance with local regulations.

Availability of data and material

The datasets analyzed during the current study are available upon request with no restriction.

Consent for publication

Not applicable.

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