Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Insomnia symptoms, daytime sleepiness, and quality of life in people with Alzheimer's disease: a secondary cross-sectional analysis

Magdalena Wójcik-Pędziwiatr

mpedziwiatr@neurologia.org.pl

Andrzej Frycz Modrzewski Krakow University

Agnieszka Tomaszewska

Andrzej Frycz Modrzewski Krakow University

Grażyna Dębska

Andrzej Frycz Modrzewski Krakow University

Monika Rudzińska-Bar

Andrzej Frycz Modrzewski Krakow University

Research Article

Keywords: Alzheimer's disease, insomnia, daytime sleepiness, quality of life, WHOQOL-BREF, sleep disturbances, cognitive impairment

Posted Date: September 2nd, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10705206/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Sleep disturbances are common in Alzheimer's disease (AD) and may adversely affect daytime functioning and well-being. However, insomnia symptoms and daytime sleepiness represent distinct but interrelated dimensions of sleep–wake disturbance, and their independent associations with specific domains of quality of life (QoL) in people with AD remain insufficiently characterized. This study examined the associations of insomnia symptoms and daytime sleepiness with individual domains of QoL in people with AD.

Methods

This secondary cross-sectional analysis included 70 participants aged 60–94 years with clinically diagnosed AD recruited from nursing homes and community-based support services in Kraków, Poland. Insomnia symptoms were assessed using the Athens Insomnia Scale (AIS), daytime sleepiness using the Epworth Sleepiness Scale (ESS), and QoL using the World Health Organization Quality of Life–Brief Version (WHOQOL-BREF). Cognitive impairment was assessed using an abbreviated version of the Blessed Dementia Scale (BDS). Associations between sleep measures and the Physical Health, Psychological, Social Relationships, and Environment domains of the WHOQOL-BREF were examined using Spearman correlations and multivariable linear regression. AIS and ESS were entered simultaneously into the regression models, which were adjusted for age, sex, continuous abbreviated BDS score, depressive symptoms, functional status, and care setting. HC3 robust standard errors were used, and the Benjamini–Hochberg false discovery rate (FDR) procedure was applied across eight prespecified sleep–QoL associations.

Results

Participants were aged 60–94 years, with a mean age of 78.5 ± 7.9 years, and 45.7% were women. The mean AIS score was 9.31 ± 4.01, with 80.0% of participants meeting the AIS threshold for clinically relevant insomnia symptoms. The mean ESS score was 8.91 ± 2.83, and 30.0% had excessive daytime sleepiness. AIS and ESS scores were positively correlated (Spearman's $\rho = 0.622$, $p < 0.001$). In multivariable analyses, greater daytime sleepiness was nominally associated with poorer Physical Health (B = − 1.045, 95% CI − 1.973 to − 0.117; p = 0.027; FDR q = 0.128), whereas greater insomnia symptom severity was nominally associated with poorer Psychological QoL (B = − 0.512, 95% CI − 0.980 to − 0.044; p = 0.032; FDR q = 0.128). Neither association remained statistically significant after FDR correction. No independent associations of AIS or ESS with the Social Relationships or Environment domains were observed. The direction of the two principal associations was maintained in expanded and influence sensitivity analyses.

Conclusions

Insomnia symptoms and daytime sleepiness showed distinct domain-specific patterns of association with QoL in people with AD. Daytime sleepiness showed a suggestive association with poorer Physical Health, whereas insomnia symptoms showed a suggestive association with poorer Psychological QoL. However, neither association remained statistically significant after correction for multiple testing. These findings should therefore be considered exploratory and hypothesis-generating. Larger longitudinal studies are needed to determine whether insomnia symptoms and daytime sleepiness independently contribute to specific dimensions of QoL in AD.

INTRODUCTION

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized not only by cognitive decline but also by a broad spectrum of behavioral, psychological, functional, and somatic disturbances that may substantially affect patients' everyday well-being. As the disease progresses, maintaining quality of life (QoL) becomes an increasingly important therapeutic goal, particularly because cognitive and functional deterioration may progressively limit independence and participation in everyday activities. Quality of life in AD is multidimensional and may be influenced by disease severity, functional dependence, neuropsychiatric and depressive symptoms, comorbidity, social circumstances, and care setting [1–4]. Identifying potentially modifiable factors associated with poorer QoL may therefore have considerable clinical relevance for comprehensive dementia care.

Sleep disturbances are common non-cognitive manifestations of AD and encompass a heterogeneous spectrum of nocturnal and daytime symptoms. Patients may experience difficulties initiating or maintaining sleep, fragmented nocturnal sleep, altered sleep architecture, changes in sleep–wake timing and circadian organization, sleep-disordered breathing, and excessive daytime sleepiness (EDS) [5–8]. Objective sleep studies have demonstrated alterations in sleep architecture in AD, while accumulating evidence suggests that sleep and circadian disturbances may interact with neurodegenerative processes throughout the course of the disease [5, 7, 9]. The relationship is likely bidirectional: AD-related neurodegeneration may disrupt neural systems involved in sleep and circadian regulation, whereas persistent sleep disruption has been implicated in processes associated with amyloid-β and tau pathology and subsequent cognitive decline [5, 7, 9, 10].

Importantly, sleep disturbance in AD should not be considered a single clinical construct. Insomnia symptoms and daytime sleepiness represent related but distinct dimensions of the sleep–wake cycle. Insomnia primarily reflects difficulties with sleep initiation or maintenance and inadequate or non-restorative nocturnal sleep, whereas EDS reflects an increased propensity to fall asleep or difficulty maintaining wakefulness during the day. Although these manifestations may coexist, daytime sleepiness may also be influenced by sleep fragmentation, circadian dysregulation, sleep-disordered breathing, medication effects, reduced daytime stimulation, comorbidity, or neurodegenerative changes themselves [5, 6, 8]. Conversely, clinically relevant insomnia symptoms may occur without pronounced daytime

sleepiness. Evaluating these dimensions separately may therefore provide more clinically meaningful information than treating sleep disturbance as a single global exposure.

The potential consequences of disturbed sleep in AD extend beyond sleep itself. Impaired nocturnal sleep and reduced daytime alertness may affect physical functioning, mood, motivation, participation in daily activities, and social engagement and may also contribute to caregiver burden [11]. Nevertheless, much of the existing AD literature has focused on sleep disturbance in relation to cognitive decline, AD pathology, behavioral symptoms, disease progression, or caregiver outcomes [5, 7, 9–11]. Comparatively less attention has been paid to the relationship between specific sleep-related symptoms and the patients' own multidimensional QoL. Furthermore, associations between sleep and QoL may be substantially confounded or mediated by depressive symptoms, functional dependence, disease severity, and living environment, all of which may themselves influence perceived QoL in AD [1–4].

A domain-specific assessment of QoL may help clarify these relationships. The World Health Organization Quality of Life–Brief Version (WHOQOL-BREF) conceptualizes QoL across four domains: Physical Health, Psychological, Social Relationships, and Environment [12]. These domains capture different dimensions of everyday well-being and may therefore show different relationships with specific sleep-related symptoms. For example, impaired daytime alertness may be particularly relevant to physical functioning, energy, and participation in daily activities, whereas insomnia symptoms may be more closely related to psychological well-being. Conversely, social and environmental aspects of QoL may depend more strongly on contextual factors such as functional independence, care setting, and social support. Examining individual QoL domains rather than relying solely on a global QoL measure may therefore reveal clinically relevant patterns that would otherwise remain obscured.

Accordingly, the present study aimed to investigate the associations of insomnia symptoms and daytime sleepiness with individual WHOQOL-BREF domains in people with Alzheimer's disease. Insomnia symptoms were assessed using the Athens Insomnia Scale (AIS) [13], whereas daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS) [14]. Both instruments are established symptom-based measures designed to quantify insomnia symptoms and daytime sleepiness, respectively [13, 14]. We specifically examined whether AIS and ESS scores were independently associated with the Physical Health, Psychological, Social Relationships, and Environment domains when both sleep measures were considered simultaneously, and relevant demographic and clinical characteristics were accounted for. We hypothesized that greater severity of sleep-related symptoms would be associated with poorer QoL and that the pattern and magnitude of these associations might differ between insomnia symptoms and daytime sleepiness and across individual QoL domains.

METHODS

Study design and participants

This study was a secondary cross-sectional analysis of data collected within an observational study of individuals with Alzheimer's disease (AD) conducted between December 2021 and March 2022 in Kraków, Poland. Participants were consecutively recruited from three specialized care facilities: the St. Francis Home for Sick Priests of the Archdiocese of Kraków, the Residential Home for Older Adults, and the Community Support Centre for People with Alzheimer's Disease. The first two institutions provided long-term residential care, whereas the Community Support Centre provided multidisciplinary daytime support for community-dwelling individuals with AD.

A total of 70 participants aged 60–94 years with clinically diagnosed AD were included in the present analysis. Of these, 51 (72.9%) were nursing home residents and 19 (27.1%) were community-dwelling individuals attending the Community Support Centre.

The diagnosis of AD had been established by an experienced neurologist according to the National Institute on Aging–Alzheimer's Association (NIA-AA) diagnostic criteria for probable Alzheimer's disease [15] and was verified through review of the available medical documentation. The diagnostic work-up included neurological examination, cognitive assessment, and structural brain imaging, as documented in the participants' medical records.

Eligible participants were adults with a confirmed diagnosis of AD whose clinical condition permitted participation in the study procedures. Individuals with other neurodegenerative dementias or severe medical conditions that could substantially interfere with study participation were excluded. Participants were recruited consecutively during the study period.

The present secondary analysis addressed a research question distinct from the primary objectives of the source study by specifically examining the associations of insomnia symptoms and daytime sleepiness with individual domains of quality of life.

Data collection and clinical assessment

Sociodemographic and clinical information was obtained using a structured questionnaire developed specifically for the source study and a review of available medical records. The questionnaire had not been published previously. Variables included age, sex, duration of AD, care setting, comorbid conditions, cognitive impairment, depressive symptoms, and functional status. The complete English-language version of the study-specific questionnaire is provided as Supplementary File 1.

Whenever possible, study questionnaires were completed directly by the participants. When cognitive impairment limited independent completion, individual questions were read aloud by the investigator and the participant's responses were recorded without interpretation or modification. Supplementary factual information concerning medical history and everyday functioning was obtained, where necessary, from medical records, family members, or healthcare professionals directly involved in the participant's care.

Functional independence in activities of daily living was assessed using the Barthel Index [16], with higher scores indicating greater functional independence.

Cognitive assessment and dementia severity

Cognitive impairment was assessed using an abbreviated version of the Blessed Dementia Scale (BDS) [17, 18]. The abbreviated measure used in the source study yields a total score ranging from 0 to 28 points, with higher scores indicating greater cognitive impairment. The BDS-based assessment was not used to establish the diagnosis of AD, which had been determined independently on clinical grounds, but to quantify cognitive impairment within the study population.

For descriptive characterization of the cohort, the abbreviated BDS score was additionally categorized into three study-specific severity groups: **mild dementia (≤ 16 points), moderate dementia (17–19 points), and severe dementia (≥ 20 points)**. These categories were used to provide a clinically interpretable description of the distribution of cognitive impairment within the sample. Because universally accepted cut-off values distinguishing mild, moderate, and severe dementia are not established for all abbreviated versions of the Blessed instruments, these thresholds were treated as **study-specific descriptive categories rather than validated diagnostic cut-offs**.

For the primary multivariable analyses, the abbreviated BDS score was entered as a **continuous variable ranging from 0 to 28 points**, rather than using the three-category classification. Treating the BDS score continuously preserved the information contained in the scale, avoided loss of statistical information associated with categorization, and reduced dependence of the regression estimates on study-specific thresholds.

Visuospatial and executive abilities were additionally assessed using the Sunderland Clock Drawing Test [19].

Assessment of depressive symptoms

Depressive symptoms were assessed using the 30-item Geriatric Depression Scale (GDS-30) [20]. The instrument consists of 30 dichotomous yes/no items, each scored 0 or 1, yielding a total score ranging from 0 to 30 points, with higher scores indicating greater depressive symptom burden.

For descriptive characterization, scores of 0–9 were classified as indicating no depression, scores of 10–19 as mild depressive symptoms, and scores of ≥ 20 as severe depressive symptoms. In the primary multivariable analyses, the GDS-30 score was entered as a continuous variable to retain information on depressive symptom severity and avoid loss of information associated with categorization.

Assessment of insomnia symptoms

Insomnia symptoms were assessed using the Athens Insomnia Scale (AIS) [13]. The AIS consists of eight items assessing sleep induction, awakenings during the night, final awakening, total sleep duration, overall sleep quality, daytime well-being, daytime functioning, and daytime sleepiness. Each item is scored from 0 to 3, yielding a total score ranging from 0 to 24 points, with higher scores indicating greater insomnia symptom severity.

For descriptive purposes, an AIS score ≥ 6 was considered indicative of clinically relevant insomnia symptoms. In all primary inferential analyses, AIS was treated as a continuous variable to preserve information on symptom severity and avoid loss of statistical information associated with dichotomization.

Assessment of daytime sleepiness

Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS) [14]. The ESS evaluates the likelihood of dozing or falling asleep in eight common daytime situations. Each item is scored from 0 to 3, resulting in a total score ranging from 0 to 24 points, with higher scores indicating greater daytime sleepiness.

For descriptive purposes, an ESS score > 10 was considered indicative of excessive daytime sleepiness. In all primary inferential analyses, ESS was treated as a continuous variable.

Assessment of quality of life

Quality of life was assessed using the World Health Organization Quality of Life–Brief Version (WHOQOL-BREF) [12]. The instrument comprises 26 items and provides scores for four domains: **Physical Health, Psychological, Social Relationships, and Environment**.

The Physical Health domain encompasses activities of daily living, dependence on medicinal substances and medical aids, energy and fatigue, mobility, pain and discomfort, sleep and rest, and work capacity. The Psychological domain includes positive and negative feelings, thinking, learning, memory and concentration, self-esteem, body image and appearance, and spirituality/religion/personal beliefs. The Social Relationships domain evaluates personal relationships, social support, and sexual activity. The Environment domain includes physical safety and security, home environment, financial resources, access to health and social care, opportunities for acquiring information and skills, recreation and leisure, physical environment, and transport.

Domain scores were calculated according to the WHOQOL-BREF scoring procedure and linearly transformed to a **0–100 scale**, with higher scores indicating better quality of life. The four domains were analyzed separately because the primary objective was to determine whether insomnia symptoms and daytime sleepiness showed different patterns of association with specific dimensions of quality of life.

Statistical analysis

All statistical analyses were based on the complete analytical sample of 70 participants.

Continuous variables were summarized using means and standard deviations (SDs) and, where appropriate, medians and interquartile ranges (IQRs). Categorical variables were summarized as numbers and percentages. Distributional characteristics of continuous variables were evaluated by graphical inspection and the Shapiro–Wilk test.

The relationship between insomnia symptom severity and daytime sleepiness was initially assessed using Spearman's rank correlation coefficient. Unadjusted associations of AIS and ESS scores with each of the four WHOQOL-BREF domains were subsequently examined using Spearman's rank correlations.

The primary inferential analyses consisted of four separate multivariable ordinary least squares (OLS) regression models, with the Physical Health, Psychological, Social Relationships, and Environment WHOQOL-BREF domain scores entered as dependent variables. AIS and ESS scores were entered simultaneously into each model, allowing the association of each sleep-related dimension with quality of life to be estimated while accounting for their co-occurrence.

Each primary model was adjusted for age, sex, continuous abbreviated BDS score, GDS-30 score, Barthel Index, and care setting, in addition to AIS and ESS. These covariates were selected a priori on clinical grounds because of their plausible relationships with sleep disturbances and/or quality of life. Care setting distinguished nursing home residents from community-dwelling participants.

Unstandardized regression coefficients (B), 95% confidence intervals (CIs), and standardized regression coefficients (β) were calculated for the sleep-related predictors. Model fit was summarized using R^2 and adjusted R^2. Heteroskedasticity-consistent HC3 robust standard errors were used for all primary regression models [21].

Multicollinearity among predictors was assessed using variance inflation factors (VIFs). Model assumptions were evaluated through inspection of regression residuals. Heteroskedasticity was assessed using the Breusch–Pagan test, and potentially influential observations were evaluated using Cook's distance, with values exceeding 4/N used as a screening threshold. All statistical tests were two-sided. Statistical analyses were performed using Python version 3.13.5, with multivariable regression analyses implemented using the statsmodels package (version 0.14.6).

Multiple-comparison correction

The prespecified primary family of sleep-related hypotheses comprised **eight regression coefficients**, corresponding to two sleep exposures (AIS and ESS) evaluated across four WHOQOL-BREF domains.

To account for multiple testing, the **Benjamini–Hochberg false discovery rate (FDR) procedure** was applied across these eight coefficients [22]. Nominal p values < 0.05 were reported, but sleep-related associations were considered statistically significant after correction for multiple comparisons only

when the corresponding FDR-adjusted q value was < 0.05. Associations with nominal p < 0.05 that did not remain significant after FDR correction were interpreted as **suggestive rather than confirmatory**.

The Benjamini–Hochberg procedure was also applied separately to the eight unadjusted correlations between AIS or ESS and the four WHOQOL-BREF domains.

Sensitivity analyses

Two sets of sensitivity analyses were conducted to evaluate the robustness of the primary findings.

First, expanded multivariable models additionally adjusted for **duration of AD and the number of comorbid conditions**, while retaining AIS, ESS, age, sex, continuous abbreviated BDS score, GDS-30 score, Barthel Index, and care setting. These analyses evaluated whether additional adjustment for disease history and multimorbidity materially altered the estimated associations between sleep measures and quality of life.

Second, influence sensitivity analyses were performed by repeating the primary regression models after excluding observations with Cook's distance values exceeding the **4/N** screening threshold. This approach was used to assess whether the principal sleep-related estimates were disproportionately influenced by a small number of observations.

Categorical combinations of insomnia and excessive daytime sleepiness were not used as primary inferential exposures because subgroup sizes were insufficient to support reliable multivariable comparisons.

Given the secondary, cross-sectional nature of the study and the moderate sample size, interpretation emphasized effect estimates, 95% confidence intervals, FDR-adjusted results, and consistency across sensitivity analyses rather than nominal statistical significance alone.

Ethical considerations

The study protocol was approved by the Bioethics Committee of Andrzej Frycz Modrzewski Krakow University, Kraków, Poland (**approval No. KBKA/75/O/2021; 23 November 2021**). All study procedures were conducted in accordance with the principles of the Declaration of Helsinki [23].

Written informed consent was obtained from all participants or, where appropriate, from their legally authorized representatives before enrolment. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations for cross-sectional studies [24].

RESULTS

Participant characteristics

A total of 70 participants with Alzheimer's disease were included in the analysis. Participants were aged 60–94 years, with a mean age of 78.5 ± 7.9 years; 32 participants (45.7%) were women. Fifty-one participants (72.9%) were nursing home residents, whereas 19 (27.1%) were community-dwelling individuals attending the Community Support Centre.

The mean abbreviated BDS score was **15.97 ± 7.85 points**. Using the study-specific descriptive classification, 32 participants (**45.7%**) were categorized as having mild dementia (BDS ≤ 16), 12 (**17.1%**) as having moderate dementia (BDS 17–19), and 26 (**37.1%**) as having severe dementia (BDS ≥ 20). These categories were used for descriptive purposes only; the continuous BDS score was used in the multivariable analyses.

The mean AIS score was **9.31 ± 4.01**, and 56 participants (**80.0%**) had an AIS score ≥ 6, indicating clinically relevant insomnia symptoms. The mean ESS score was **8.91 ± 2.83**, and 21 participants (**30.0%**) had an ESS score > 10, indicating excessive daytime sleepiness.

Detailed demographic, clinical, cognitive, functional, sleep-related, and quality-of-life characteristics of the study population are presented in Table 1.

Table 1
Characteristics of the study population (N = 70)

Characteristic	Value
Age, years, mean ± SD (range)	78.51 ± 7.90 (60–94)
Female sex	32 (45.7%)
Male sex	38 (54.3%)
AD duration, years	4.59 ± 2.27
Nursing home residence	51 (72.9%)
Community dwelling	19 (27.1%)
Abbreviated BDS score (0–28)	15.97 ± 7.85
BDS-based descriptive severity: mild (≤ 16)	32 (45.7%)
BDS-based descriptive severity: moderate (17–19)	12 (17.1%)
BDS-based descriptive severity: severe (≥ 20)	26 (37.1%)
GDS-30 score	13.59 ± 4.77
No depression (GDS-30 0–9)	18 (25.7%)
Mild depression (GDS-30 10–19)	41 (58.6%)
Severe depression (GDS-30 ≥ 20)	11 (15.7%)
Barthel Index	64.14 ± 29.87
AIS score	9.31 ± 4.01
Clinically relevant insomnia (AIS ≥ 6)	56 (80.0%)
ESS score	8.91 ± 2.83
Excessive daytime sleepiness (ESS > 10)	21 (30.0%)
WHOQOL-BREF Physical Health	44.59 ± 12.34
WHOQOL-BREF Psychological	55.00 ± 9.32
WHOQOL-BREF Social Relationships	59.17 ± 13.84
WHOQOL-BREF Environment	60.76 ± 8.75

Data are mean ± SD or n (%), as appropriate. BDS categories (≤ 16, 17–19, ≥ 20) are study-specific descriptive categories; the continuous abbreviated BDS score was used in multivariable analyses. Higher BDS scores indicate greater cognitive impairment. WHOQOL-BREF domain scores are presented on a 0–100 scale.

Relationship between insomnia symptoms and daytime sleepiness

AIS and ESS scores were moderately to strongly positively correlated (**Spearman's ρ = 0.622, p < 0.001**), indicating substantial co-occurrence of insomnia symptoms and daytime sleepiness. Nevertheless, the correlation was not sufficiently high to suggest that the two measures represented an identical sleep-related construct. Both variables were therefore retained simultaneously in the multivariable regression models.

Unadjusted associations between sleep measures and quality of life

In unadjusted analyses, greater insomnia symptom severity was associated with poorer quality of life across several WHOQOL-BREF domains. Higher AIS scores were associated with lower **Physical Health** scores (ρ = −0.501), lower **Psychological** scores (ρ = −0.455), and lower **Social Relationships** scores (ρ = −0.310).

Daytime sleepiness showed broader unadjusted associations with quality of life. Higher ESS scores were associated with poorer scores across all four WHOQOL-BREF domains, with Spearman correlation coefficients ranging from **− 0.283 to − 0.637**. The strongest unadjusted association was observed between daytime sleepiness and the Physical Health domain.

Overall, the unadjusted analyses indicated that greater sleep-related symptom burden was associated with poorer quality of life, although the magnitude of these associations differed across sleep measures and QoL domains.

Multivariable associations with WHOQOL-BREF domains

Four multivariable linear regression models were fitted to examine the independent associations of insomnia symptoms and daytime sleepiness with each WHOQOL-BREF domain. AIS and ESS were entered simultaneously, and all models were adjusted for **age, sex, continuous abbreviated BDS score, GDS-30 score, Barthel Index, and care setting**. HC3 robust standard errors were used.

Physical Health

After multivariable adjustment, greater daytime sleepiness was nominally associated with poorer Physical Health. Each one-point increase in ESS score was associated with an approximately one-point decrease in the WHOQOL-BREF Physical Health score (**B = − 1.045, 95% CI − 1.973 to − 0.117; p = 0.027**).

After correction for the eight prespecified sleep–QoL comparisons, however, this association did not remain statistically significant (**FDR q = 0.128**).

AIS score was not independently associated with Physical Health after simultaneous adjustment for ESS and the remaining covariates.

Psychological domain

Greater insomnia symptom severity was nominally associated with poorer Psychological quality of life. Each one-point increase in AIS score was associated with a **0.512-point lower Psychological domain score** (**B = − 0.512, 95% CI − 0.980 to − 0.044; p = 0.032**).

This association did not remain statistically significant after correction for multiple testing (**FDR q = 0.128**).

ESS was not independently associated with the Psychological domain after adjustment for AIS and the other covariates.

Social Relationships

Neither AIS nor ESS showed evidence of an independent association with the Social Relationships domain after multivariable adjustment. The unadjusted associations observed for sleep-related symptoms were therefore attenuated after accounting for demographic and clinical characteristics, including cognitive impairment, depressive symptom burden, functional status, and care setting.

Environment

No independent association of AIS or ESS with the Environment domain was observed in the fully adjusted model. Thus, after accounting for the prespecified covariates, neither insomnia symptom severity nor daytime sleepiness appeared to be independently related to environmental quality of life.

The complete results of the four primary multivariable regression models are presented in Table 2.

Table 2
Primary mutually adjusted associations of AIS and ESS with WHOQOL-BREF domains

Domain	Exposure	B	HC3 SE	95% CI	p	FDR q	Std. β	Adj. R^2
Physical Health	AIS	-0.197	0.358	-0.899 to 0.505	0.582	0.864	-0.064	0.726
Physical Health	ESS	-1.045	0.474	-1.973 to -0.117	0.027	0.128	-0.239	0.726
Psychological	AIS	-0.512	0.239	-0.980 to -0.044	0.032	0.128	-0.221	0.691
Psychological	ESS	0.099	0.385	-0.656 to 0.853	0.798	0.864	0.030	0.691
Social Relationships	AIS	0.111	0.419	-0.711 to 0.933	0.791	0.864	0.032	0.534
Social Relationships	ESS	-0.114	0.667	-1.421 to 1.192	0.864	0.864	-0.023	0.534
Environment	AIS	0.132	0.481	-0.811 to 1.075	0.784	0.864	0.060	0.228
Environment	ESS	-0.260	0.558	-1.353 to 0.833	0.641	0.864	-0.084	0.228

Models included AIS and ESS simultaneously and were adjusted for age, sex, continuous abbreviated BDS score, GDS-30 score, Barthel Index, and care setting. HC3 robust standard errors are reported. FDR correction was applied across the eight prespecified sleep–QoL coefficients. None remained significant at $q < 0.05$.

Multiple-comparison correction

Across the eight prespecified adjusted sleep–QoL coefficients, two associations met the conventional nominal significance threshold of $p < 0.05$: **ESS with Physical Health** and **AIS with Psychological quality of life**.

Neither association remained statistically significant after Benjamini–Hochberg correction for multiple comparisons, with both yielding **FDR-adjusted q = 0.128**.

Accordingly, these findings were interpreted as **suggestive rather than confirmatory associations**. No sleep-related association met the prespecified FDR-adjusted significance threshold of $q < 0.05$.

Sensitivity analyses

Sensitivity analyses were performed to assess the robustness of the two principal nominal associations.

In expanded models additionally adjusting for **duration of Alzheimer's disease and number of comorbid conditions**, the overall direction of the associations between sleep-related symptoms and quality of life remained consistent with the primary analyses.

Influence sensitivity analyses were also performed after excluding observations with Cook's distance values exceeding the prespecified **4/N** screening threshold.

Following exclusion of potentially influential observations, the association between **ESS and Physical Health remained nominally significant** (**B = − 0.829, p = 0.035**). Similarly, the association between **AIS and Psychological quality of life remained nominally significant** (**B = − 0.477, p = 0.015**).

Thus, the direction and approximate magnitude of the two principal sleep-related associations were not explained solely by a small number of influential observations. Nevertheless, given that the primary associations did not survive FDR correction, the sensitivity analyses were interpreted as supporting the **robustness of the observed pattern rather than establishing confirmatory statistical significance**.

Detailed sensitivity analyses are presented in Table 3.

Table 3
Sensitivity analyses for the two nominal primary signals

Domain	Exposure	Sensitivity analysis	N	B	95% CI	p
Physical Health	ESS	Expanded adjustment	70	-1.085	-2.030 to -0.139	0.025
Physical Health	ESS	Exclude Cook's D > 4/N	67	-0.829	-1.599 to -0.059	0.035
Psychological	AIS	Expanded adjustment	70	-0.500	-0.976 to -0.023	0.040
Psychological	AIS	Exclude Cook's D > 4/N	65	-0.477	-0.860 to -0.094	0.015

Expanded models additionally adjusted for AD duration and comorbidity count. Influence analyses excluded observations with Cook's D > 4/N.

Regression diagnostics

Regression diagnostics were examined for all four primary models. Heteroskedasticity and potentially influential observations were evaluated using the Breusch–Pagan test and Cook's distance, respectively. HC3 robust standard errors were retained for inference to reduce sensitivity to potential heteroskedasticity and influential observations.

The diagnostic findings did not indicate that the principal conclusions were driven by a major violation of the regression assumptions. Results of the regression diagnostics are summarized in Table 4.

Table 4
Regression diagnostics for primary models

Domain	Breusch–Pagan p	Shapiro residual p	Max Cook's D	n > 4/N
Physical Health	0.293	0.930	0.150	3
Psychological	0.376	0.759	0.163	5
Social Relationships	0.219	0.893	0.162	6
Environment	0.687	0.012	0.142	5
HC3 robust standard errors were used for all primary regression models.				

DISCUSSION

The present study examined the relationships of two related but distinct dimensions of sleep–wake disturbance—insomnia symptoms and daytime sleepiness—with individual domains of quality of life (QoL) in people with Alzheimer's disease (AD). Both sleep measures showed inverse associations with several WHOQOL-BREF domains in unadjusted analyses, but most of these associations were attenuated after demographic and clinical characteristics were taken into account. In the primary multivariable models, greater daytime sleepiness was nominally associated with poorer Physical Health, whereas greater insomnia symptom severity was nominally associated with poorer Psychological QoL. Neither association remained statistically significant after false discovery rate (FDR) correction. However, both associations retained the same direction and remained nominally significant after exclusion of potentially influential observations. These findings therefore suggest potentially distinct domain-specific patterns linking sleep-related symptoms with QoL in AD, but they should be interpreted as exploratory and hypothesis-generating rather than confirmatory.

Sleep disturbances and multidimensional quality of life in Alzheimer's disease

The broad inverse associations observed in the unadjusted analyses are consistent with previous evidence indicating that sleep disturbance may be related to multiple dimensions of well-being in people living with dementia. Petrovsky et al. reported in a state-of-the-art review that sleep disruption was negatively associated with physical, emotional, social/behavioral, and cognitive aspects of QoL in people with dementia, although the available evidence was heterogeneous and based on a relatively small number of studies [25]. Other observational evidence has similarly suggested that sleep disruption may coexist with poorer QoL and impaired daytime functioning in people with dementia [26]. More broadly, insomnia and other sleep disturbances represent a clinically relevant component of the symptom burden associated with AD [27].

The present study extends this literature by examining insomnia symptoms and daytime sleepiness as separate exposures and by including both measures simultaneously in the multivariable models. AIS and ESS scores were positively correlated (Spearman's ρ = 0.622), demonstrating substantial overlap between nocturnal insomnia symptoms and impaired daytime alertness. Nevertheless, the correlation was not sufficiently strong to suggest that they represented the same construct, and their adjusted associations with QoL differed by domain. This pattern supports the conceptual distinction between insomnia symptoms and daytime sleepiness and suggests that considering them separately may provide clinically useful information.

The attenuation of several associations after multivariable adjustment is also important. Sleep disturbance in AD occurs within a complex network of cognitive, affective, functional, medical, and environmental factors. Sleep and circadian abnormalities have been linked to neurodegenerative processes, neuropsychiatric symptoms, functional deterioration, and disease progression [5–10]. Accordingly, some of the crude associations between sleep symptoms and QoL may reflect shared relationships with depressive symptoms, cognitive impairment, functional dependence, or care environment rather than a direct relationship between sleep disturbance and QoL.

Daytime sleepiness and Physical Health

One of the principal findings was a nominal association between greater daytime sleepiness and poorer WHOQOL-BREF Physical Health after adjustment for insomnia symptoms, cognitive impairment, depressive symptoms, functional status, demographic characteristics, and care setting. Each one-point increase in ESS score was associated with an approximately one-point decrease in the Physical Health domain score (B = − 1.045, 95% CI − 1.973 to − 0.117; p = 0.027). However, this association did not remain statistically significant after correction for multiple testing (q = 0.128).

Several mechanisms may plausibly underlie this pattern. Daytime sleepiness may reduce alertness, energy, mobility, motivation, and engagement in everyday activities. In people with AD, whose functional reserve may already be compromised, reduced daytime alertness could further restrict participation in rehabilitation, recreational activities, and routine daily tasks. Conversely, poorer physical health, multimorbidity, lower levels of daytime activity, sedating medications, sleep-disordered breathing, circadian disruption, and reduced environmental stimulation may themselves contribute to daytime sleepiness. The cross-sectional design of the present study therefore precludes determination of the direction of this relationship.

The composition of the WHOQOL-BREF Physical Health domain also warrants consideration. This domain encompasses energy and fatigue, mobility, activities of daily living, pain and discomfort, dependence on medical treatment, work capacity, and sleep and rest [12]. Daytime sleepiness could plausibly relate to several of these components. At the same time, the inclusion of sleep and rest within the Physical Health domain introduces some conceptual overlap between the sleep-related exposure and the outcome and may have contributed to the observed association.

Importantly, the ESS–Physical Health association was not eliminated after excluding observations exceeding the Cook's distance screening threshold. The estimate remained in the same direction and was nominally significant (B = − 0.829, p = 0.035). In the expanded model additionally adjusted for AD duration and number of comorbid conditions, the association also remained nominally significant (B = − 1.085, p = 0.025). Thus, the direction of the association was relatively consistent across sensitivity analyses.

Nevertheless, because the primary ESS–Physical Health association did not remain statistically significant after FDR correction, these sensitivity analyses should not be interpreted as establishing a confirmed independent relationship. Rather, they indicate that the observed signal was not attributable solely to the primary covariate specification or to a small number of influential observations and therefore warrants replication in larger prospective studies.

Insomnia symptoms and Psychological quality of life

A different domain-specific pattern was observed for insomnia symptoms. Greater AIS scores were nominally associated with poorer Psychological QoL after simultaneous adjustment for daytime sleepiness and the other prespecified covariates (B = − 0.512, 95% CI − 0.980 to − 0.044; p = 0.032). As with the ESS–Physical Health association, this finding did not remain statistically significant after FDR correction (q = 0.128).

The relationship between insomnia symptoms and psychological well-being is clinically plausible. Disturbed sleep may coexist with negative affect, anxiety, impaired concentration, reduced emotional resilience, and poorer subjective well-being. Conversely, psychological distress and depressive symptoms may contribute to difficulties initiating or maintaining sleep. The relationship is therefore likely to be bidirectional and embedded within the broader interaction between sleep, mood, cognition, and neurodegeneration.

Of particular interest, the association between AIS and Psychological QoL was observed despite adjustment for GDS-30 score. This suggests that measured depressive symptom burden did not fully account for the observed relationship. However, this finding should not be interpreted as demonstrating an effect of insomnia that is independent of depression. The GDS-30, subjective insomnia symptoms, and the WHOQOL-BREF Psychological domain capture partially overlapping aspects of emotional and subjective well-being, and residual confounding or construct overlap cannot be excluded.

The sensitivity analyses provided some support for the consistency of this pattern. In the expanded model additionally adjusted for AD duration and comorbidity burden, the AIS–Psychological association remained in the same direction and was nominally significant (B = − 0.500, p = 0.040). After exclusion of potentially influential observations, the association also remained nominally significant (B = − 0.477, p = 0.015). As for the Physical Health finding, these analyses strengthen confidence in the stability of the observed direction but do not overcome the absence of statistical significance after correction for multiple testing.

Accordingly, the AIS–Psychological association should be viewed as hypothesis-generating. Replication using longitudinal designs and complementary objective and informant-based sleep assessments would help clarify whether insomnia symptoms have a specific relationship with psychological QoL beyond the effects of depression and other neuropsychiatric symptoms.

Depressive symptoms and quality of life

Although sleep-related measures constituted the primary exposures of interest, depressive symptom burden emerged as an important correlate of poorer QoL across the WHOQOL-BREF domains. This observation is consistent with previous studies indicating that affective and neuropsychiatric symptoms are important determinants of perceived QoL in people with AD and other dementias [1–4].

The relationship between depressive symptoms, sleep disturbance, and QoL is likely complex and bidirectional. Insomnia may contribute to poorer mood and daytime functioning, while depressive symptoms may worsen subjective sleep quality, decrease daytime activity, and negatively influence perceptions of health and well-being. Consequently, analyses that do not account for depressive symptom burden may overestimate the relationship between sleep disturbance and QoL.

The persistence of nominal sleep-related associations after adjustment for GDS-30 is nevertheless noteworthy. It suggests that the observed relationships between ESS and Physical Health and between AIS and Psychological QoL cannot be explained solely by measured depressive symptom burden. However, given the cross-sectional design and potential residual confounding, these findings should not be interpreted causally.

Clinically, these observations support a multidimensional assessment of people with AD who present with sleep complaints. Evaluation should extend beyond sleep symptoms to include mood, functional independence, cognitive impairment, medication use, physical comorbidity, daytime activity, and environmental circumstances. Such an approach may be particularly relevant because potentially modifiable contributors to insomnia and daytime sleepiness frequently coexist.

Social Relationships and Environment domains

Neither insomnia symptoms nor daytime sleepiness showed an independent association with the Social Relationships or Environment domains after multivariable adjustment. This contrasts with the broader pattern observed in the unadjusted analyses and suggests that relationships with these QoL dimensions may be more strongly explained by clinical and contextual characteristics.

The WHOQOL-BREF Social Relationships domain reflects personal relationships, social support, and sexual activity, whereas the Environment domain includes physical safety, home environment, financial resources, access to healthcare, opportunities for recreation and information, and transport [12]. Many of these characteristics are strongly influenced by social circumstances, care arrangements, functional independence, and disease severity and may therefore be less directly related to sleep symptoms.

The inclusion of both nursing home residents and community-dwelling participants is particularly relevant in this context. Care setting may capture substantial differences in functional status, social support, daily routines, environmental stimulation, and access to care. However, any relationship between care setting and QoL should be interpreted cautiously because institutionalization is not a randomly assigned exposure and is closely related to disease severity, disability, multimorbidity, and availability of informal care.

These findings may also indicate that sleep-related symptoms are more closely linked to individual physical and psychological experiences than to broader social and environmental aspects of QoL. This interpretation remains tentative, however, because the modest sample size may have limited the ability to detect weaker associations in the Social Relationships and Environment domains.

Clinical implications

The present findings suggest that sleep complaints in people with AD may benefit from characterization beyond the broad label of “sleep disturbance.” Nocturnal insomnia symptoms and daytime sleepiness may represent different clinical problems and may relate to different dimensions of patients’ well-being.

When prominent daytime sleepiness is reported, clinical assessment may reasonably include nocturnal sleep fragmentation, sleep-disordered breathing, sedating medications, circadian disruption, medical comorbidity, and the amount of daytime physical and social activity. Similarly, clinically relevant insomnia symptoms should prompt consideration of depressive and anxiety symptoms, pain, nighttime behavioral symptoms, medication effects, environmental factors, and sleep–wake routines.

The present study does not establish that treatment of insomnia symptoms or daytime sleepiness will improve QoL. Nevertheless, sleep–wake disturbances represent potentially modifiable clinical features, and pharmacological and non-pharmacological approaches to sleep problems in people with cognitive impairment continue to be investigated [27, 28]. The current findings support the clinical relevance of distinguishing nocturnal insomnia symptoms from daytime sleepiness when evaluating sleep–wake disturbances in AD.

Future intervention studies should determine whether targeting these different symptom dimensions translates into improvements in specific domains of QoL. Domain-specific QoL measures may be particularly informative in such studies because an intervention that improves daytime alertness, for example, may primarily affect physical functioning, whereas improvement in insomnia symptoms may have a stronger relationship with psychological well-being.

Strengths and limitations

Several strengths of the present study should be highlighted. First, insomnia symptoms and daytime sleepiness were evaluated as separate continuous exposures and entered simultaneously into the multivariable models, allowing their mutually adjusted associations with QoL to be examined. This is particularly relevant given the substantial correlation between AIS and ESS scores.

Second, QoL was evaluated across four WHOQOL-BREF domains rather than reduced to a single global score, enabling identification of potentially different domain-specific patterns. Third, the analyses accounted for several clinically relevant characteristics, including depressive symptoms, functional status, cognitive impairment measured using the continuous abbreviated BDS score, and care setting.

Fourth, the statistical approach incorporated HC3 robust standard errors, correction for multiple testing, assessment of potentially influential observations, and prespecified sensitivity analyses. The direction of the two principal nominal associations was maintained after additional adjustment for AD duration and comorbidity burden and after exclusion of potentially influential observations.

Several limitations should also be considered. First, the cross-sectional design precludes conclusions regarding temporality or causality. Sleep symptoms may contribute to poorer QoL, but poorer physical or psychological health may also exacerbate sleep disturbance and daytime sleepiness.

Second, the sample size was modest. This limited statistical power, precision of some estimates, and the feasibility of subgroup analyses. Importantly, none of the eight primary sleep–QoL associations remained statistically significant after FDR correction. The observed ESS–Physical Health and AIS–Psychological associations should therefore be considered exploratory signals requiring independent replication rather than confirmed associations.

Third, sleep was assessed using questionnaire-based instruments rather than objective measures such as actigraphy or polysomnography. AIS and ESS quantify clinically relevant symptom dimensions but cannot determine the underlying causes of sleep disturbance or identify specific disorders such as obstructive sleep apnea, circadian rhythm disorders, periodic limb movements, or medication-related somnolence. Cognitive impairment may also affect the reliability of self-reported sleep symptoms, although investigator assistance was provided when necessary.

Fourth, the WHOQOL-BREF was developed as a generic QoL instrument rather than specifically for people with dementia. Cognitive impairment may influence the reliability and interpretation of self-reported QoL, particularly in more advanced disease. Nevertheless, use of a multidomain measure enabled assessment of potentially distinct physical, psychological, social, and environmental patterns.

Fifth, the WHOQOL-BREF Physical Health domain includes sleep and rest. Some degree of conceptual overlap between the sleep-related exposures and this outcome is therefore unavoidable and may have contributed to the observed association between daytime sleepiness and Physical Health.

Sixth, the cohort included both nursing home residents and community-dwelling participants. Although this increased clinical heterogeneity and allowed care setting to be incorporated into the models, the relatively small community subgroup limited meaningful stratified analyses.

Finally, residual confounding remains possible. Detailed information on psychotropic and sleep-related medications, sleep-disordered breathing, pain severity, physical activity, light exposure, nighttime

behavioral disturbances, and caregiver characteristics was not sufficiently comprehensive for inclusion in the final models.

Conclusions

In this secondary cross-sectional analysis of people with Alzheimer's disease, insomnia symptoms and daytime sleepiness showed broad inverse associations with QoL in unadjusted analyses, but most associations were attenuated after demographic and clinical characteristics were taken into account. Greater daytime sleepiness showed a suggestive association with poorer Physical Health, whereas greater insomnia symptom severity showed a suggestive association with poorer Psychological QoL. Neither association remained statistically significant after FDR correction, although both retained their direction and remained nominally significant in influence sensitivity analyses.

These findings suggest that insomnia symptoms and daytime sleepiness may capture different dimensions of sleep–wake disturbance and may show distinct domain-specific relationships with well-being in AD. They also emphasize the importance of considering sleep symptoms within the broader clinical context of depressive symptoms, cognitive impairment, functional status, and care environment. Larger longitudinal studies incorporating objective sleep assessment are needed to determine whether these domain-specific patterns are reproducible and clinically meaningful and whether targeted management of sleep–wake disturbances can improve quality of life in people with AD.

Declarations

Human Ethics and Consent to Participate declarations

This study was approved by the Institutional Bioethics Committee at Andrzej Frycz Modrzewski Cracow University (approval number: KBKA/75/O/2021). All procedures were conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was obtained from all participants or their legal guardians.

Consent for publication

Not applicable.

Clinical trial number

Not applicable.

Availability of data and materials

The datasets generated and/or analyzed during the current study are available from the corresponding author upon **reasonable request**.

Competing interests

The authors declare that they **have no competing interests**.

Funding

This research received **no external funding**.

Authors' contributions

MW-P and AT conceptualized and designed the study.

AT collected the data and performed the statistical analysis.

MW-P and AT interpreted the results and drafted the manuscript.

MW-P prepared the final version of the manuscript and approved the final submission.

GD supervised the study and approved the plan of the study.

MR-B supervised the study and critically revised the manuscript.

All authors critically revised the manuscript, read, and approved the final version.

Acknowledgements

The authors would like to thank the staff of the **nursing homes and the Community Support Centre for Patients with Alzheimer's Disease in Kraków** for their assistance in participant recruitment and data collection. The authors also thank all participants and their caregivers for their valuable contribution to this study.

References

1. Froelich L, Lladó A, Khandker RK, et al. Quality of life and caregiver burden of Alzheimer's disease among community dwelling patients in Europe: variation by disease severity and progression. J Alzheimer's Disease Rep. 2021. https://doi.org/10.3233/ADR-210025.
2. Conde-Sala JL, Turró-Garriga O, et al. Self-perceived quality of life among patients with Alzheimer's disease: two longitudinal models of analysis. J Alzheimer's Disease. 2016. https://doi.org/10.3233/JAD-160040.
3. Heßmann P, Seeberg G, Reese JP, et al. Health-related quality of life in patients with Alzheimer's disease in different German health care settings. J Alzheimer's Disease. 2016. https://doi.org/10.3233/JAD-150835.
4. Cubero-Plazas L, Sancho-Cantus D, et al. Impact of family function on the quality of life and health perception of non-institutionalized dementia patients. J Alzheimer's Disease. 2023. https://doi.org/10.3233/JAD-230324.

5. Musiek ES, Xiong DD, Holtzman DM. Sleep, circadian rhythms, and the pathogenesis of Alzheimer disease. Exp Mol Med. 2015;47:e148. https://www.nature.com/articles/emm2014121.
6. Borges CR, Poyares D, Piovezan R, Nitrini R, Brucki S. (2019). Alzheimer's disease and sleep disturbances: a review. Arquivos de Neuro-Psiquiatria. https://www.scielo.br/j/anp/a/5jKK7JSN5M8SD5wmFgQfcGb/.
7. Zhang Y, Ren R, Yang L, Zhang H, Shi Y, Sanford LD, Tang X. (2022). Sleep in Alzheimer's disease: a systematic review and meta-analysis of polysomnographic findings. Translational Psychiatry, 12. https://www.nature.com/articles/s41398-022-01897-y
8. Guarnieri B, Maestri M, Cucchiara F, et al. Multicenter study on sleep and circadian alterations as objective markers of mild cognitive impairment and Alzheimer's disease reveals sex differences. J Alzheimer's Disease. 2020. https://doi.org/10.3233/JAD-200632.
9. Carpi M, Fernandes M, Mercuri NB, et al. Sleep biomarkers for predicting cognitive decline and Alzheimer's disease: a systematic review of longitudinal studies. J Alzheimer's Disease. 2024. https://doi.org/10.3233/JAD-230933.
10. Wang S, Zheng X, Huang J, Liu J, Li C, Shang H. Sleep characteristics and risk of Alzheimer's disease: a systematic review and meta-analysis of longitudinal studies. J Neurol. 2024. 10.1007/s00415-024-12380-7. https://link.springer.com/article/.
11. Gehrman P, Gooneratne NS, Brewster GS et al. (2018). Impact of Alzheimer disease patients' sleep disturbances on their caregivers. Geriatr Nurs. https://www.sciencedirect.com/science/article/pii/S0197457217301568
12. The WHOQOL Group. Development of the World Health Organization WHOQOL-BREF quality of life assessment. Psychol Med. 1998;28(3):551–8. https://doi.org/10.1017/S0033291798006667.
13. Soldatos CR, Dikeos DG, Paparrigopoulos TJ. Athens Insomnia Scale: validation of an instrument based on ICD-10 criteria. J Psychosom Res. 2000;48(6):555–60. https://doi.org/10.1016/S0022-3999(00)00095-7.
14. Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep. 1991;14(6):540–5. https://doi.org/10.1093/sleep/14.6.540.
15. McKhann GM, Knopman DS, Chertkow H, et al. The diagnosis of dementia due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimer's Dement. 2011;7(3):263–9. https://doi.org/10.1016/j.jalz.2011.03.005.
16. Mahoney FI, Barthel DW. Functional evaluation: the Barthel Index. Maryland State Med J. 1965;14:61–5.
17. Blessed G, Tomlinson BE, Roth M. The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. Br J Psychiatry. 1968;114(512):797–811. https://doi.org/10.1192/bjp.114.512.797.
18. Davis PB, Morris JC, Grant E. Brief screening tests versus clinical staging in senile dementia of the Alzheimer type. J Am Geriatr Soc. 1990;38(2):129–35. https://doi.org/10.1111/j.1532-

5415.1990.tb03473.x.

19. Sunderland T, Hill JL, Mellow AM, et al. Clock drawing in Alzheimer's disease: a novel measure of dementia severity. J Am Geriatr Soc. 1989;37(8):725–9. https://doi.org/10.1111/j.1532-5415.1989.tb02233.x.
20. Yesavage JA, Brink TL, Rose TL et al. Development and validation of a geriatric depression screening scale: a preliminary report. *Journal of Psychiatric Research.* 1982–1983;17(1):37–49. https://doi.org/10.1016/0022-3956(82)90033-4
21. Long JS, Ervin LH. Using heteroscedasticity consistent standard errors in the linear regression model. Am Stat. 2000;54(3):217–24. https://doi.org/10.1080/00031305.2000.10474549.
22. Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. J Roy Stat Soc B. 1995;57(1):289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x.
23. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191–4. https://doi.org/10.1001/jama.2013.281053.
24. von Elm E, Altman DG, Egger M, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–7. https://doi.org/10.1016/S0140-6736(07)61602-X.
25. Petrovsky DV, McPhillips MV, Li J, Brody A, Caffeé L, Hodgson NA. Sleep disruption and quality of life in persons with dementia: A state-of-the-art review. Geriatr Nurs. 2018. https://doi.org/10.1016/j.gerinurse.2018.04.014.
26. Hodgson NA, Gitlin LN, Huang J. The influence of sleep disruption and pain perception on indicators of quality of life in individuals living with dementia at home. *Geriatric Nursing.* 2014;35(5):394–398. https://doi.org/10.1016/j.gerinurse.2014.08.*005*.
27. Benca R, Herring WJ, Khandker R, et al. Burden of insomnia and sleep disturbances and the impact of sleep treatments in patients with probable or possible Alzheimer's disease: a structured literature review. J Alzheimer's Disease. 2022. https://doi.org/10.3233/JAD-215324.
28. Gibson R, Dowell A, Jones L, Gander P. Non-pharmacological interventions a feasible option for addressing dementia-related sleep problems in the context of family care. *Pilot Feasibility Stud*. 2021;7(1):114. Published 2021 May 26. 10.1186/s40814-021-00851-x

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryFile1StudySpecificQuestionnaire.docx