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ORIGINAL RESEARCH

Functional Impairment and Post-Stroke Depression: Potential Roles of Sleep Quality and Fatigue in a Cross-Sectional Structural Equation Analysis

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Objective: To examine the association between functional impairment and depressive symptoms in hospitalized patients undergoing post-stroke rehabilitation, and to explore whether sleep quality and fatigue were statistically linked to this association within a structural equation modeling framework. We also compared the relative contributions of the Barthel Index (BI) and modified Rankin Scale (mRS).

Methods: In this single-center retrospective cross-sectional study, 137 patients with stroke admitted for rehabilitation were included. Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9), sleep quality using the Pittsburgh Sleep Quality Index (PSQI), and fatigue using the Fatigue Assessment Scale (FAS). Functional status was evaluated with the BI (reverse-transformed to Barthel_rev) and mRS. Structural equation modeling with maximum likelihood estimation and bootstrap resampling (2,000 samples) was used to estimate direct and indirect associations.

Results: In the primary latent-variable model, greater functional impairment was associated with poorer sleep quality (β=0.407, p=0.012) and greater fatigue (β=0.692, p=0.010). Poorer sleep quality was associated with higher PHQ-9 scores (β=0.296, p=0.003), whereas fatigue was not. The indirect association through sleep quality was significant (b=0.068, 95% CI 0.018–0.131), but the indirect association through fatigue was not. In a supplementary mutually adjusted model, mRS showed significant indirect associations with depressive symptoms through both sleep quality and fatigue, whereas Barthel_rev was linked primarily through fatigue. Because the supplementary model showed suboptimal fit, these findings should be interpreted cautiously.

Conclusion: These findings suggest that sleep quality may represent a more consistent statistical pathway linking functional impairment and depressive symptoms in post-stroke rehabilitation, whereas the role of fatigue appears to vary by functional measure and model specification. Early screening of sleep disturbance and fatigue may still be clinically relevant, but causal inferences cannot be drawn from this cross-sectional study.

Keywords: stroke, depressive symptoms, functional impairment, sleep quality, fatigue, structural equation modeling

Background

Stroke remains one of the leading causes of mortality and long-term disability worldwide. In 2021, an estimated 93.8 million individuals were living with stroke, and approximately 11.9 million new stroke events occurred globally.[1] Traditional outcome assessments post-stroke have primarily focused on motor function and activities of daily living (ADL). However, non-motor dysfunctions, including depression, sleep disturbances, and fatigue, are also highly

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prevalent following stroke and are frequently underrecognized. A systematic review encompassing 279 prospective cohort studies reported that most non-motor outcomes showed little improvement over time.[2]

Post-stroke depression (PSD) represents one of the most common neuropsychiatric complications after stroke. The cumulative incidence of depression within one year after stroke has been reported to reach approximately 38%, with nearly 71% of depressive episodes occurring within the first three months after stroke.[3] Depression during the early post-stroke period is strongly associated with unfavorable functional outcomes. Early identification and management of depressive symptoms during the acute phase of stroke have been demonstrated to contribute to improved functional recovery.[4] Therefore, clarification of the mechanisms linking functional impairment to depressive symptoms is essential for optimizing comprehensive post-stroke care.

Individuals with PSD demonstrate poorer sleep quality, reduced ADL performance, and greater levels of functional disability compared with those without PSD.[5] In addition, fatigue represents a highly prevalent and debilitating symptom among individuals with neurological disorders. The prevalence of post-stroke fatigue (PSF) varies substantially across studies, depending on factors such as stroke subtype and timing of assessment, but remains consistently high. PSF frequently overlaps with depressive symptoms and may interact with depression, thereby constituting a complex clinical condition.[6,7]

Rather than assuming a fixed one-way sequence, sleep disturbance, fatigue, and depressive symptoms after stroke may be closely interrelated and mutually reinforcing. In the present study, functional status was comprehensively evaluated using the Barthel Index (BI) to reflect ADL performance and the modified Rankin Scale (mRS) to assess overall disability. Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9), while sleep quality and fatigue were measured using the Pittsburgh Sleep Quality Index (PSQI) and the Fatigue Assessment Scale (FAS), respectively.[5,8] Within this context, structural equation modeling was used in the present study as a theory-informed analytic approach to examine whether the observed data were consistent with an exploratory pattern in which functional impairment was statistically linked to depressive symptoms through sleep quality and fatigue. We further compared the Barthel Index and the modified Rankin Scale because these measures reflect complementary dimensions of post-stroke functioning, namely activities of daily living and global disability. Accordingly, the aim of this study was not to establish causal or temporal mediation, but to identify clinically relevant association patterns that may help refine post-stroke symptom assessment and hypothesis generation. These findings may provide clinically relevant evidence to support early risk identification and the development of modifiable intervention targets for PSD.

Materials and Methods

Study Design and Participants

This study employed a single-center, retrospective, cross-sectional observational design. Data were extracted from inpatient medical records and routine rehabilitation assessment records in the Department of Rehabilitation Medicine at the Affiliated Hospital of Hebei University from October 2023 to March 2025. Patients with stroke admitted for inpatient rehabilitation during the study period were screened consecutively. To ensure a relatively uniform assessment window, only patients who remained hospitalized and had complete routine evaluations available at approximately 1 month after stroke onset were included; patients discharged before that time point were excluded. The study examined the association between functional impairment and depressive symptoms, with sleep quality (PSQI) and fatigue (FAS) analyzed as theory-informed intermediate variables in structural equation modeling (SEM). Because all variables were assessed within the same approximately 1-month post-stroke window, SEM was used to test whether the observed data were consistent with hypothesized association patterns rather than to infer temporal or causal mediation. A supplementary mutually adjusted model was further used to compare the relative contributions of the Barthel Index (BI) and the modified Rankin Scale (mRS).

Inclusion and Exclusion Criteria

Inclusion Criteria

Participants were eligible for inclusion if all of the following criteria were met: age ≥ 18 years; a first-ever diagnosis of stroke confirmed by the Department of Neurology or Neurosurgery prior to admission, with a clearly defined stroke

subtype, including ischemic stroke or hemorrhagic stroke; assessment conducted at one month post-stroke onset, at which time the patient was hospitalized in the Department of Rehabilitation Medicine for rehabilitation treatment; and availability of complete scale assessments and key clinical data during hospitalization. Required data included scores from the PHQ-9, PSQI, FAS, BI, and mRS, as well as baseline characteristics such as age, sex, educational level, occupational status, body mass index (BMI), National Institutes of Health Stroke Scale (NIHSS) score, stroke subtype, and past medical history.[8]

Exclusion Criteria

Participants were excluded if any of the following conditions were present: transient ischemic attack (TIA) or non–stroke-related brain injury, including traumatic brain injury or brain tumors; severe disturbance of consciousness, severe aphasia or agnosia, severe cognitive impairment, or pronounced psychiatric or behavioral abnormalities that could substantially compromise the validity of scale assessments, resulting in an inability to complete evaluations or unreliable questionnaire data; or absence of key variables or major scale data.[8]

Data Collection and Variable Definitions

Data were extracted from the inpatient medical record system and routine assessment records during the index rehabilitation hospitalization. Collected information included demographic characteristics, lifestyle factors, clinical features, past medical history, and standardized scale scores. Demographic and clinical background variables were obtained from admission records or the earliest available documentation during the same hospitalization, whereas PHQ-9, PSQI, FAS, BI, and mRS scores were taken from the first complete routine assessment available at approximately 1 month after stroke onset.Because this was a retrospective study based on routinely collected clinical data, only variables that were consistently available in the medical records were included in the present analysis; lesion location, medication use, and anxiety symptoms were not entered as covariates because these data were not recorded in a sufficiently standardized manner across all patients. Variable definitions were specified as follows:

Depressive symptoms: Depressive symptoms were evaluated using the PHQ-9, with total scores ranging from 0 to 27; higher scores reflect greater severity of depressive symptoms. Based on the established clinical cutoff (PHQ-9 $\geq$ 10), participants were classified into the PSD group or the non-PSD group.[8,9]

Baseline characteristics: Referring to previous population-based studies on sleep issues,[10] we considered a number of baseline factors. Age (years) and BMI (kg/m^2) were recorded as continuous variables. Sex was categorized as male or female. Smoking status was classified as never smoker, current smoker, or former smoker. Alcohol consumption was categorized as never drinker, current drinker, or former drinker.

Clinical characteristics: Past medical history was categorized according to inpatient records as none, hypertension, coronary heart disease, diabetes mellitus, or two or more comorbidities. Stroke subtype was classified as ischemic stroke or hemorrhagic stroke. Stroke severity was assessed using the NIHSS, which evaluates the degree of neurological deficit, with higher scores indicating greater severity.

Outcome measures: Sleep quality was assessed using the PSQI, with higher total scores indicating poorer sleep quality. Fatigue severity was measured using the FAS, with higher scores indicating more severe fatigue. Functional status and disability were assessed using two instruments: ① BI: Total scores range from 0 to 100, with higher original scores indicating better ADL performance. To ensure consistency in analytical direction, such that higher values reflect poorer functional status, BI scores were reverse-transformed as follows: Barthel_rev = 100 − BI. ② mRS: Higher scores indicate greater levels of disability.

Statistical Analysis

After exclusion of patients with missing data, a total of 137 participants were included in the final analysis. The minimum required sample size was satisfied based on the applicable sample size calculation formula $n = \left(\frac{Z_{1-\alpha/2} \times \sqrt{\bar{p}(1-\bar{p})}}{E} \right)^2$. Based on the background literature, the cumulative incidence of depression within 1 year after stroke is approximately 38%, and about 71% of depressive episodes occur within the first 3 months;[3] accordingly, the proportion relevant to the early post-

stroke window was approximated as 0.38×0.71 = 0.27. With a two-sided confidence level of 99% (Z = 2.576) and an allowable error of 0.10, the minimum required sample size was 131. Because this was a retrospective study based on consecutive eligible admissions during a prespecified study period, data collection was not continued until a prospectively defined sample size was reached. The final sample of 137 complete cases therefore exceeded the minimum requirement. Participants were classified into a non-PSD group (n = 68) and a PSD group (n = 69) according to PHQ-9 scores, with no missing values across study variables. Data management, descriptive analyses, and between-group comparisons were conducted using SPSS version 27.0. Distributional characteristics of continuous variables were visualized using violin plots generated with jamovi version 2.6.44. SEM was performed using the semlj module in jamovi. Normality of continuous variables was assessed using the Shapiro–Wilk test in SPSS. As most continuous variables did not conform to a normal distribution ($p < 0.05$), data were summarized as median values with interquartile ranges (IQR), and between-group comparisons were performed using the Mann–Whitney *U*-test. Categorical variables were expressed as percentages and compared using the χ^2-test or Fisher's exact test, as appropriate. All statistical tests were two-sided, and a p value < 0.05 was considered statistically significant.

To investigate the association between functional impairment and depressive symptoms, as well as the potential intermediary roles of sleep quality and fatigue, two SEMs were specified and evaluated. In both models, parameters were estimated using maximum likelihood (ML) estimation. Unstandardized path coefficients (b) and standardized path coefficients (β) were reported. Model fit was assessed using the χ^2 statistic with degrees of freedom and corresponding p values, the Standardized Root Mean Square Residual (SRMR), and the Root Mean Square Error of Approximation (RMSEA) with 95% confidence intervals.

Indirect associations were evaluated using a nonparametric bootstrap procedure with 2,000 resamples to estimate 95% confidence intervals (CI) for specific indirect effects, total indirect effects, and total effects. An indirect effect was considered statistically significant if the corresponding CI did not include zero. Given the cross-sectional design, these indirect effects were interpreted as statistical associations consistent with the hypothesized model structure rather than as evidence of temporal or causal mediation. When discrepancies were observed between p values and bootstrap CI, the bootstrap results were preferentially reported.

Model 1: The reverse-transformed Barthel Index (Barthel_rev) and the mRS were used as indicators to construct a latent variable representing functional impairment (D). Paths were specified from D to the PSQI and the FAS, from the PSQI and the FAS to the PHQ-9, and a direct path from D to the PHQ-9 was retained. This model was used to evaluate the parallel mediating effects of sleep quality and fatigue, as well as the total effect of functional impairment on depressive symptoms.

Model 2: Barthel_rev and mRS were simultaneously entered as two parallel, mutually adjusted independent variables. Both variables were specified to predict the PSQI and the FAS, which subsequently predicted the PHQ-9. Direct paths from Barthel_rev to the PHQ-9 and from mRS to the PHQ-9 were retained. Bootstrap procedures were applied to estimate the indirect effects, total indirect effects, and total effects of Barthel_rev and mRS via the PSQI and the FAS, enabling comparison of the relative contributions of the two functional measures to depressive symptoms and exploration of their corresponding indirect association patterns in this supplementary mutually adjusted model.

Structural Equation Path Diagrams Were Generated Using Boardmix Software

Results

No significant differences were observed between the PSD and non-PSD groups with respect to age, BMI, sex distribution, educational level, occupational status, or smoking and alcohol consumption status (all $p > 0.05$), indicating comparable baseline demographic and lifestyle characteristics and minimizing the potential impact of these variables as confounders. Detailed baseline characteristics are presented in Table 1.

Similarly, no significant differences were identified between the two groups in stroke subtype or stratification of past medical history ($p > 0.05$), indicating comparable disease categories and overall comorbidity burden. In contrast, neurological deficit severity differed markedly between groups. NIHSS scores were significantly higher in the PSD group than in the non-PSD group ($p < 0.001$), indicating more severe neurological impairment at admission among patients with PSD. Detailed clinical characteristics are presented in Table 1.

Table 1 Baseline Characteristics, Clinical Features, and Between-Group Comparisons by PSD Status

Variable		Non-PSD Median (IQR)	PSD Median (IQR)	*p*
PHQ-9		6 (3–7)	12 (11–15)	<0.001
Age		56 (47–62)	59 (48–68)	0.133
BMI		25.2 (23.8–27.7)	26.7 (24.5–27.7)	0.086
Variable	**Category**	**Non-PSD n (%)**	**PSD n (%)**	***p***
Gender	Female	16 (23.5%)	22 (31.9%)	0.275
	Male	52 (76.5%)	47 (68.1%)	
Education (edu)	Primary	33 (48.5%)	37 (53.6%)	0.239
	Secondary	14 (20.6%)	19 (27.5%)	
	Tertiary	21 (30.9%)	13 (18.8%)	
Occupation (job)	Freelance	37 (54.4%)	39 (56.5%)	0.362
	Employed	20 (29.4%)	14 (20.3%)	
	Retired	11 (16.2%)	16 (23.2%)	
Alcohol (drink)	Never	37 (54.4%)	34 (49.3%)	0.815
	Current	16 (23.5%)	19 (27.5%)	
	Former	15 (22.1%)	16 (23.2%)	
Smoking (smoke)	Never	31 (45.6%)	37 (53.6%)	0.467
	Current	22 (32.4%)	22 (31.9%)	
	Former	15 (22.1%)	10 (14.5%)	
Stroke type	Ischemic stroke	55 (80.9%)	57 (82.6%)	0.794
	Hemorrhagic stroke	13 (19.1%)	12 (17.4%)	
Past medical history	Healthy	15 (22.1%)	8 (11.6%)	0.230
	Hypertension	25 (36.8%)	29 (42.0%)	
	Coronary heart disease	1 (1.5%)	2 (2.9%)	
	Diabetes	2 (2.9%)	7 (10.1%)	
	≥2 comorbidities	25 (36.8%)	23 (33.3%)	
Variable		**Non-PSD median (IQR)**	**PSD median (IQR)**	***p***
NIHSS		1 (0–3)	7 (3–9)	<0.001
Barthel		100 (97–100)	95 (86–98)	<0.001
Rankin		2 (1–2)	2 (1–3)	0.010
PSQI		3 (2–7)	7 (3–11)	<0.001
FAS		23 (22–25)	27 (25–31)	<0.001

Abbreviations: PHQ-9, Patient Health Questionnaire-9; BMI, Body Mass Index; NIHSS, National Institutes of Health Stroke Scale; PSQI, Pittsburgh Sleep Quality Index; FAS, Fatigue Assessment Scale; p, p-value.

With regard to outcome measures, the PSD group demonstrated significantly poorer sleep quality and greater fatigue severity than the non-PSD group, as reflected by higher PSQI and FAS scores (both $p < 0.001$). Consistent with the NIHSS findings, these results suggest that greater neurological impairment is associated with increased sleep disturbances and fatigue burden. In terms of functional outcomes, patients in the PSD group exhibited more pronounced functional limitations, with significantly lower BI scores ($p < 0.001$) and higher mRS scores ($p = 0.010$). These findings indicate reduced ADL performance, greater disability severity, and less favorable functional status among patients with PSD. Detailed results are presented in Table 1, and distributions of key variables are presented in Figure 1.

The overall fit of Model 1 was satisfactory ($\chi^2(3) = 3.24$, $p = 0.356$; CFI = 0.998; TLI = 0.995; RMSEA = 0.024, 95% CI 0.000–0.148; SRMR = 0.023). Within the measurement model, mRS demonstrated a standardized factor loading of 0.526 ($p < 0.001$) on the latent functional impairment variable (D), with Barthel_rev serving as the reference indicator (standardized loading = 0.604). In the structural model, D was significantly associated with PSQI ($b = 0.214$, $\beta = 0.407$, $p = 0.012$) and FAS ($b = 0.325$, $\beta = 0.692$, $p = 0.010$), indicating that greater functional impairment was related to poorer sleep quality and greater fatigue severity. Regarding depressive symptoms, PSQI was a significant positive predictor of

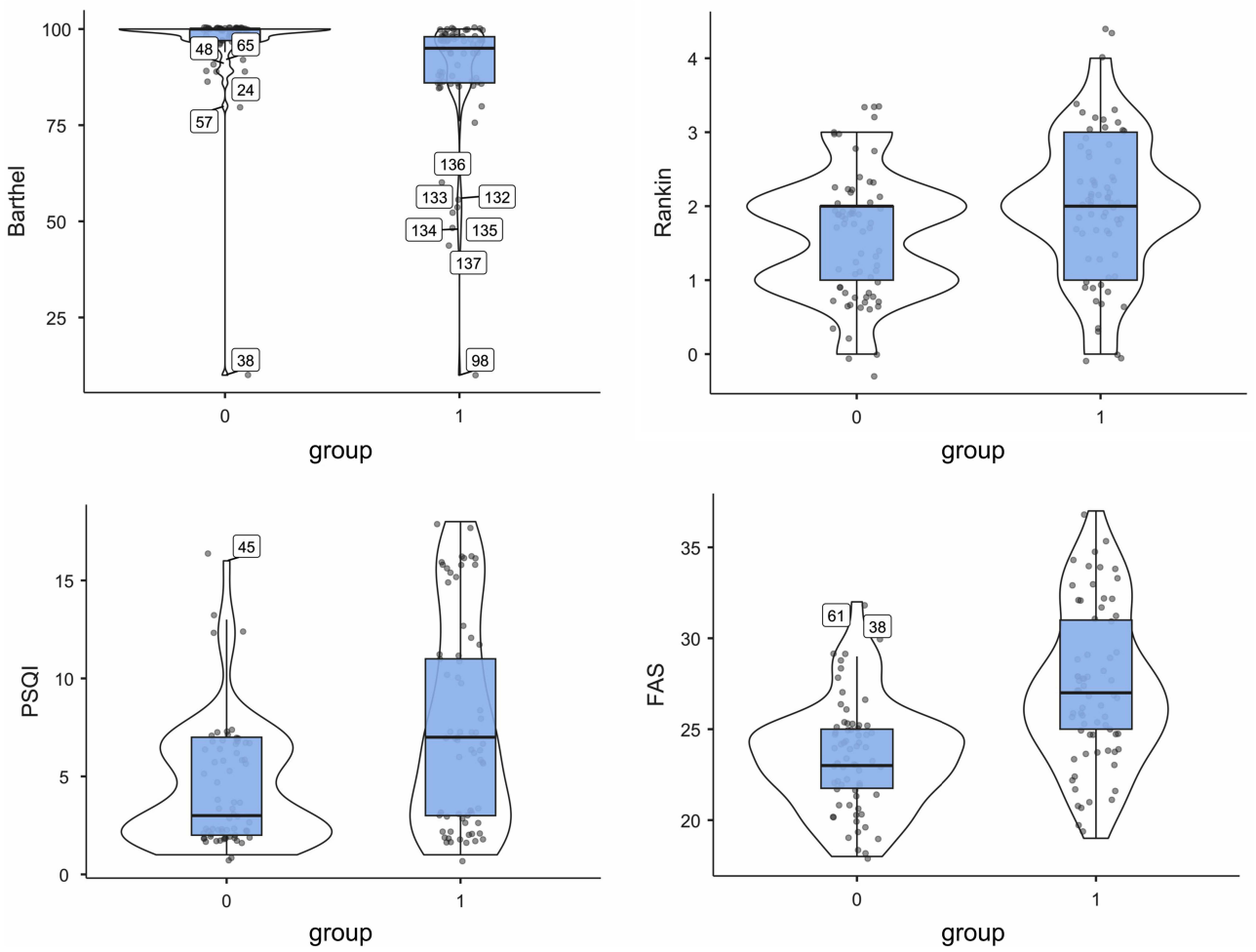

Figure 1 Distribution of clinical and functional measures by PSD status shown using violin plots (0 = non-PSD, 1 = PSD).

PHQ-9 scores ($b = 0.317$, $\beta = 0.296$, $p = 0.003$). In contrast, the association between FAS and PHQ-9 was not statistically significant ($b = 0.324$, $\beta = 0.270$, $p = 0.837$). The direct path from D to PHQ-9 was also not statistically significant ($b = 0.196$, $\beta = 0.348$, $p = 0.839$). Accordingly, the interpretation of Model 1 was based primarily on the overall model fit and the pattern of specific path estimates, rather than on a statistically significant direct association from D to PHQ-9.

Bootstrap mediation analysis indicated that the indirect effect of D on PHQ-9 through PSQI was statistically significant ($b = 0.068$, $\beta = 0.121$, 95% CI 0.018–0.131, $p = 0.020$). In contrast, the indirect effect mediated by FAS was not statistically significant ($b = 0.105$, $\beta = 0.187$, 95% CI −0.516–0.236, $p = 0.911$). Accordingly, the total indirect effect combining both mediating pathways was not significant ($b = 0.173$, $\beta = 0.308$, 95% CI −0.451–0.332, $p = 0.855$). The total effect of D on PHQ-9 was positive and statistically significant ($b = 0.368$, $\beta = 0.656$, 95% CI 0.207–0.710, $p = 0.005$). Taken together, Model 1 suggested a selective indirect association pattern in which the PSQI-related pathway, but not the FAS-related pathway, reached statistical significance. The model accounted for 16.6% of the variance in PSQI, 47.9% of the variance in FAS, and 54.1% of the variance in PHQ-9. Detailed results are presented in Table 2, and the corresponding path diagram is presented in Figure 2.

In supplementary Model 2, when Barthel_rev and mRS were entered simultaneously and mutually adjusted, both PSQI and FAS emerged as significant positive predictors of PHQ-9 (PSQI → PHQ-9: $\beta = 0.366$, $p < 0.001$; FAS → PHQ-9: $\beta = 0.430$, $p < 0.001$). With respect to upstream associations, mRS was significantly associated with both PSQI and

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Table 2 SEM-1 Path Coefficients and Mediation Effects with Latent Functional Impairment (Bootstrap 95% CI)

Module	Indicator/Path	b	SE	β	95% CI (b)	*p*
Model fit	χ^2 (df)	3.24 (3)				0.356
	CFI	0.998				
	TLI	0.995				
	RMSEA	0.024			0.000–0.148	0.499
	SRMR	0.023				
Measurement model	D → Barthel_rev (reference, fixed)	1.000	0.000	0.604	1.000–1.000	
	D → mRS	0.054	0.013	0.526	0.023–0.083	<0.001
Structural paths	D → PSQI	0.214	0.085	0.407	0.081–0.434	0.012
	D → FAS	0.325	0.125	0.692	0.200–0.646	0.010
	D→PHQ-9 (direct)	0.196	0.965	0.348	0.010–1.081	0.839
	PSQI → PHQ-9	0.317	0.105	0.296	0.084–0.503	0.003
	FAS → PHQ-9	0.324	1.572	0.270	−0.963–0.621	0.837
Mediation effects	Indirect: D→PSQI→PHQ-9	0.068	0.029	0.121	0.018–0.131	0.020
	Indirect: D→FAS→PHQ-9	0.105	0.942	0.187	−0.516–0.236	0.911
	Total indirect	0.173	0.943	0.308	−0.451–0.332	0.855
	Total effect	0.368	0.131	0.656	0.207–0.710	0.005
Explained variance	R^2 (PSQI)	0.166				
	R^2 (FAS)	0.479				
	R^2 (PHQ-9)	0.541				

Notes: SEM-1 was specified as the primary model with a latent functional impairment factor. The symbol "→" indicates the direction of a specified path; in indirect effect rows, expressions such as "D → PSQI → PHQ-9" indicate the hypothesized indirect pathway from the predictor to the outcome through the mediator.

Abbreviations: SEM, Structural Equation Modeling; PHQ-9, Patient Health Questionnaire-9; PSQI, Pittsburgh Sleep Quality Index; FAS, Fatigue Assessment Scale; mRS, Modified Rankin Scale; Barthel_rev, reverse-scored Barthel Index (BI); SRMR, Standardized Root Mean Square Residual; RMSEA, Root Mean Square Error of Approximation; CI, confidence intervals; D, latent functional impairment factor; b, unstandardized path coefficient; SE, standard error; β, standardized path coefficient; p, p-value; χ^2, chi-square; df, degrees of freedom; CFI, Comparative Fit Index; TLI, Tucker-Lewis Index; R^2, coefficient of determination.

FAS (mRS → PSQI: $\beta = 0.228$, $p = 0.010$; mRS → FAS: $\beta = 0.220$, $p = 0.005$). In contrast, Barthel_rev was significantly associated with FAS ($\beta = 0.345$, $p < 0.001$) but not with PSQI ($\beta = 0.090$, $p = 0.515$). After adjustment for PSQI and FAS, the direct effects of Barthel_rev and mRS on PHQ-9 were not statistically significant (Barthel_rev → PHQ-9: $\beta = 0.142$, $p = 0.256$; mRS → PHQ-9: $\beta = 0.049$, $p = 0.462$).

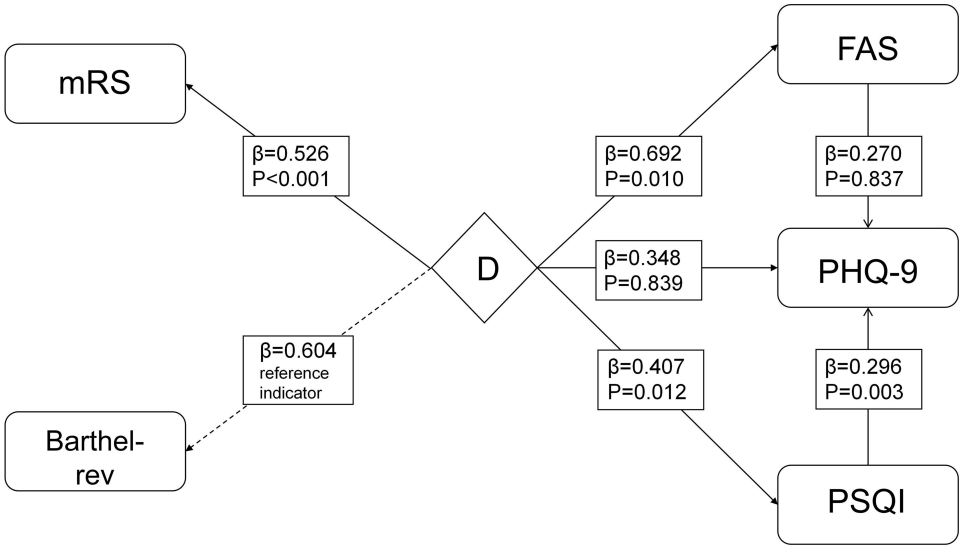

Figure 2 SEM-1: Latent functional impairment with parallel mediation by PSQI and FAS. The latent variable D (Dysfunction) consists of mRS and Barthel_rev. Solid lines denote free parameters, and the dashed line denotes the fixed reference indicator (Barthel_rev).

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Bootstrap-based mediation analysis indicated that the indirect effect of Barthel_rev on PHQ-9 through PSQI was not statistically significant (b = 0.011, β = 0.033, 95% CI −0.008–0.052, p = 0.470). In contrast, the indirect effect of Barthel_rev mediated by FAS was statistically significant (b = 0.049, β = 0.148, 95% CI 0.027–0.087, p = 0.001). Accordingly, the total indirect effect of Barthel_rev on PHQ-9 was significant (b = 0.060, β = 0.181, 95% CI 0.025–0.122, p = 0.017). The total effect of Barthel_rev on PHQ-9 demonstrated a marginal level of statistical significance (b = 0.106, β = 0.323, 95% CI 0.034–0.263, p = 0.077), indicating a modest positive association. In contrast, both specific indirect effects of mRS were statistically significant, including the pathway mediated by PSQI (b = 0.442, β = 0.083, 95% CI 0.058–0.886, p = 0.038) and the pathway mediated by FAS (b = 0.502, β = 0.095, 95% CI 0.123–0.943, p = 0.020). The total indirect effect of mRS on PHQ-9 was also significant (b = 0.944, β = 0.178, 95% CI 0.362–1.542, p = 0.002), as was the total effect (b = 1.206, β = 0.227, 95% CI 0.214–2.017, p = 0.008). Overall, in this supplementary mutually adjusted model, mRS exhibited a stronger independent and overall association with depressive symptoms, as measured by PHQ-9, than Barthel_rev. Both functional measures were associated with depressive symptoms through the fatigue pathway (FAS), whereas mRS additionally demonstrated a significant association through the sleep quality pathway (PSQI).These findings were interpreted as an exploratory comparison of the two functional scales rather than as the primary basis for inference.

The model accounted for 7.4% of the variance in PSQI, 22.0% of the variance in FAS, and 47.5% of the variance in PHQ-9. Overall model fit was suboptimal ($\chi^2(1)$ = 7.59, p = 0.006; SRMR = 0.052; RMSEA = 0.219, 95% CI 0.095–0.376) and Model 2 was therefore interpreted as a supplementary mutually adjusted model rather than as a primary model. Detailed results are presented in Table 3, and the corresponding path diagram is presented in Figure 3.

Table 3 Path Coefficients and Effect Estimates for the Supplementary Mutually Adjusted Model 2 (Bootstrap 95% CI)

Module	Indicator/Path	b	SE	β	95% CI (b)	*p*
Model fit	χ^2 (df)	7.59 (1)				0.006
	SRMR	0.052				
	RMSEA	0.219			0.095–0.376	0.015
Structural paths	Barthel_rev →PSQI	0.029	0.044	0.090	−0.021–0.151	0.515
	mRS (Rankin)→PSQI	1.162	0.450	0.228	0.227–1.958	0.010
	Barthel_rev→FAS	0.098	0.024	0.345	0.064–0.161	<0.001
	mRS (Rankin)→FAS	1.001	0.360	0.220	0.293–1.703	0.005
	Barthel_rev (direct effect)→PHQ-9	0.047	0.041	0.142	−0.003–0.158	0.256
	mRS (direct effect)→PHQ-9	0.262	0.356	0.049	−0.498–0.930	0.462
	PSQI→PHQ-9	0.381	0.089	0.366	0.195–0.545	<0.001
	FAS→PHQ-9	0.501	0.091	0.430	0.319–0.687	<0.001
Parallel mediation (Barthel_rev)	Barthel_rev → PSQI → PHQ-9	0.011	0.015	0.033	−0.008–0.052	0.470
	Barthel_rev → FAS → PHQ-9	0.049	0.015	0.148	0.027–0.087	0.001
	Total indirect (Barthel_rev)	0.060	0.025	0.181	0.025–0.122	0.017
	Total effect (Barthel_rev)	0.106	0.060	0.323	0.034–0.263	0.077
Parallel mediation (mRS)	mRS → PSQI → PHQ-9	0.442	0.213	0.083	0.058–0.886	0.038
	mRS → FAS → PHQ-9	0.502	0.215	0.095	0.123–0.943	0.020
	Total indirect (mRS)	0.944	0.304	0.178	0.362–1.542	0.002
	Total effect (mRS)	1.206	0.452	0.227	0.214–2.017	0.008
Explained variance	R^2 (PSQI)	0.074				
	R^2 (FAS)	0.220				
	R^2 (PHQ-9)	0.475				

Notes: SEM-2 was specified as a supplementary mutually adjusted model. The symbol "→" indicates the direction of a specified path; in indirect effect rows, expressions such as "Barthel_rev → PSQI → PHQ-9" and "mRS → FAS → PHQ-9" indicate the hypothesized indirect pathway from the predictor to the outcome through the mediator.

Abbreviations: SEM, Structural Equation Modeling; PHQ-9, Patient Health Questionnaire-9; PSQI, Pittsburgh Sleep Quality Index; FAS, Fatigue Assessment Scale; mRS, Modified Rankin Scale; Barthel_rev, reverse-scored Barthel Index (BI); SRMR, Standardized Root Mean Square Residual; RMSEA, Root Mean Square Error of Approximation; CI, confidence intervals.

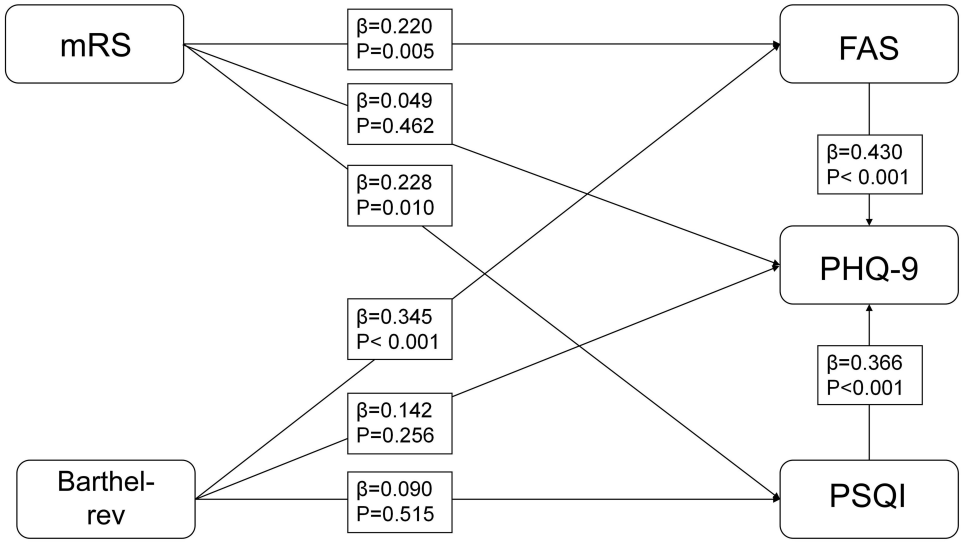

Figure 3 Supplementary SEM-2: mutually adjusted model with Barthel_rev and mRS as independent predictors and PSQI/FAS as intermediary variables. Because overall model fit was suboptimal, this figure should be interpreted as a supplementary result. Solid lines denote free parameters.

Discussion

The present study extends existing research on PSD by examining whether sleep quality and fatigue were statistically associated with the relationship between functional impairment and depressive symptoms within a cross-sectional SEM framework. The contributions of this study can be summarized in three key aspects. First, by integrating information from Barthel_rev and mRS into a latent construct of functional impairment and examining indirect association patterns involving PSQI and FAS within a unified SEM framework, the findings move beyond a simple descriptive comparison between functional impairment and depressive symptoms.This approach provides theory-informed evidence for clinically relevant association patterns linking functional impairment, sleep quality, fatigue, and depressive symptoms, rather than confirming a mechanistic pathway. Second, the study elucidates the differentiated roles of commonly used functional measures across these mediating pathways. The distinct patterns observed for mRS and Barthel_rev in relation to PSQI, FAS, and PHQ-9 highlight that these instruments capture different dimensions of post-stroke functional status. These differences have important methodological implications, indicating that the choice of functional assessment tool may influence the identification of relevant mediators and underlying mechanisms in PSD research.

Third, from a clinical perspective, the findings indicate that sleep quality may represent a relatively stable and proximal pathway linking functional impairment to depressive symptoms, whereas the contribution of fatigue appears to be more sensitive to the specific functional dimension assessed. When functional impairment is examined using separate indicators, fatigue emerges as a prominent mediator, particularly in relation to ADL limitations. These observations underscore the potential value of targeting sleep disturbances and fatigue as complementary but distinct intervention targets in patients with PSD.

Relatively More Consistent Sleep-Related Association Pattern

In Model 1, the statistically significant indirect association involving PSQI, together with the non-significant FAS-related pathway, suggested that sleep quality may represent a more consistent association pattern than fatigue within the primary model. Functional limitations following stroke are frequently accompanied by difficulties with turning or repositioning, frequent nighttime urination and caregiving needs, and reduced daytime activity levels, all of which may disrupt circadian rhythms and impair sleep regulation. In addition, common post-stroke nocturnal symptoms, including pain, numbness, muscle spasms, and positional discomfort, further compromise sleep continuity and quality.

Evidence from meta-analyses indicates that the prevalence and severity of post-stroke sleep disturbances vary dynamically over time, with individuals experiencing comorbid depression or anxiety demonstrating more pronounced

insomnia symptoms.[11,12] Notably, several risk factors for sleep disturbance substantially overlap with indicators of functional impairment, such as hemiplegic shoulder pain, spasticity, limited mobility, and reduced social participation.[13] These factors have also been shown to adversely affect rehabilitation outcomes, underscoring the interconnected nature of physical function, sleep, and recovery.[14]

In the general population, a wide range of sleep disturbances, including prolonged wakefulness, shortened sleep duration, and frequent nocturnal awakenings—have been consistently associated with impaired emotional functioning.[15] Similarly, PSD has been shown to be positively associated with post-stroke sleep disturbances.[16,17] At the same time, because both PSQI and PHQ-9 are self-report measures and may capture overlapping aspects of sleep-related burden and emotional distress, the relatively stronger PSQI-related association observed in the present study may partly reflect shared method variance rather than a uniquely dominant mechanism. Certain sleep disorders, such as obstructive sleep apnea (OSA), may further contribute to depressive symptoms post-stroke; studies conducted in hospitalized populations with ischemic stroke have reported significant associations between OSA severity and the risk of PSD.[18]

Collectively, these findings are consistent with a close and potentially bidirectional relationship between sleep disturbance and depressive symptoms in the context of post-stroke functional impairment. Sleep disturbance may arise as a consequence of functional limitations while simultaneously contributing to the development or exacerbation of depressive symptoms. Such interdependence may partly explain why the PSQI-related indirect association appeared more prominent in the present statistical model. Among individuals with comparable levels of functional impairment, poorer sleep quality is generally associated with greater depressive symptom severity. Accordingly, systematic assessment and targeted management of sleep disturbances may remain clinically relevant for individuals with functional limitations after stroke, although the present study does not establish temporal directionality. This interpretation is consistent with the high burden of post-stroke sleep disturbances and aligns with existing evidence emphasizing the central role of sleep in emotional regulation and depression risk.[15,16]

Differences in Scale Dimensions and Model-Dependent Fatigue Associations

PSF is among the most prevalent and clinically consequential subjective symptoms following stroke and has been consistently characterized by high prevalence, persistence, and adverse effects on rehabilitation outcomes and quality of life.[16,19,20] In the present analyses, distinct patterns emerged regarding the role of fatigue across different modeling frameworks. In SEM-1, although fatigue severity was significantly predicted by the latent functional impairment variable (D), the path from FAS to PHQ-9 was not statistically significant when modeled in parallel with sleep quality. In contrast, the pathway from D to PHQ-9 mediated by PSQI remained robust, indicating that within a composite functional impairment framework, sleep quality may serve as a more proximal mediator of depressive symptoms. In SEM-2, when Barthel_rev and mRS were entered simultaneously as parallel and mutually adjusted predictors, the association between FAS and PHQ-9 became statistically significant. Accordingly, the fatigue-related findings should be interpreted as model-dependent and hypothesis-generating rather than as evidence of a stable intermediary role across model specifications.

In this model, Barthel_rev primarily predicted FAS, whereas mRS demonstrated significant associations with both PSQI and FAS, resulting in a more stable overall indirect effect through dual mediating pathways. These findings indicate that the relationship between fatigue and depressive symptoms depends on both the dimensional structure of functional impairment and the measurement instrument applied. When functional impairment is further disaggregated, the contribution of fatigue becomes more evident, particularly among individuals with limitations in ADL performance.[21–23] This pattern is consistent with the conceptual distinctions between the two functional scales.

The mRS primarily captures global disability, encompassing overall dependency and limitations in social participation, whereas the BI focuses more specifically on performance in basic ADL. Although the two measures are highly correlated, they are not interchangeable and differ in scoring structure, cut-off values, and sensitivity to functional outcomes.[24–27] When included concurrently and mutually adjusted, the model distinguishes global disability from ADL-specific limitations. Global disability reflects dependency, restricted social functioning, and overall disease burden, whereas ADL-specific limitations more directly reflect physical capacity and daily activity demands.

ADL limitations are more likely to manifest as fatigue through mechanisms such as activity compensation, reduced physical reserve, and increased energy expenditure, thereby strengthening the Barthel_rev → FAS → PHQ-9 pathway. In contrast, global disability appears to contribute concurrently to both sleep disturbance and fatigue, supporting the dual pathways observed for mRS (mRS → PSQI/FAS → PHQ-9).[23–26,28] This distinction helps explain why the indirect and total effects of mRS were more stable across pathways, whereas the effects of Barthel_rev were concentrated primarily through fatigue.

Taken together, these findings suggest that the role of fatigue in relation to depressive symptoms was model-dependent and varied according to the functional construct being emphasized. Fatigue does not represent a uniform mediator across all dimensions of functional impairment; rather, its influence depends on the specific functional components assessed. ADL-related limitations primarily affect depressive symptoms through fatigue, whereas global disability contributes through both sleep and fatigue pathways. Clinically, these results underscore the importance of assessing both overall disability and ADL-specific limitations, as each may indicate distinct and potentially modifiable targets, such as sleep disturbance or fatigue, for more precise stratification and management of PSD risk and symptom burden.

Clinical Implications

Building on the preceding findings, this study provides cross-sectional SEM-based evidence that the observed data were consistent with a "function–sleep/fatigue–depressive symptoms" association pattern. The results suggest that sleep quality and fatigue may represent clinically relevant intermediate correlates of the association between functional impairment and depressive symptoms, rather than establishing a fixed temporal sequence. These intermediate factors represent clinically actionable targets for early identification and intervention.

Based on these findings, a structured screening framework may be proposed: (1) stratified functional assessment using BI and mRS; (2) prioritized screening for sleep disturbance and fatigue using PSQI and FAS; and (3) evaluation of depressive symptom severity using PHQ-9 to inform subsequent clinical management. PHQ-9 has demonstrated good screening performance for PSD in previous studies and may be effectively applied for rapid risk stratification and longitudinal monitoring in rehabilitation settings.[29] With respect to intervention prioritization, sleep quality appears to represent a relatively stable transmission link between functional impairment and depressive symptoms. Accordingly, early identification and management of sleep disturbances should be emphasized in clinical practice. Feasible sleep-focused interventions may include sleep hygiene education, management of pain, spasticity, and nocturnal discomfort, optimization of environmental and behavioral routines, and evaluation for comorbid sleep-disordered breathing. In addition, cognitive behavioral therapy for insomnia, including digital delivery formats, has demonstrated promise in stroke populations for improving both sleep and mood-related outcomes.[30]

Furthermore, findings from SEM-2 indicate that the contribution of fatigue to depressive symptoms is dependent on the functional dimension assessed. Among individuals with marked ADL limitations, fatigue management emerges as a critical intervention target. Comprehensive approaches may include energy conservation strategies, task segmentation, individualized adjustment of rehabilitation intensity and rest periods, and systematic evaluation of nutritional status and comorbid conditions, such as anemia, infection, or medication-related adverse effects, that may exacerbate fatigue. When indicated, structured psychosocial interventions should be integrated. Cognitive Behavioral Therapy (CBT) has been shown in experimental settings to improve PSF and sleep-related symptoms.[31] Given that PSF is widely recognized as a prevalent and clinically significant post-stroke symptom, systematic assessment and multidisciplinary management are warranted.[32]

In summary, the findings support a refined stratified management approach. Patients with higher mRS scores, reflecting greater global disability and dependency, may benefit from dual-targeted interventions addressing both sleep disturbance and fatigue, along with regular monitoring of PHQ-9 scores. In contrast, patients with more pronounced ADL limitations based on BI scores may require prioritization of fatigue management through individualized rehabilitation planning, while concurrently addressing sleep quality. These recommendations represent model-informed strategies for risk-chain management. They should be integrated with clinical judgment, taking into account stroke stage, severity, pharmacological treatment, and psychosocial context.

Strengths, Limitations, and Future Directions

This study has several notable strengths. First, constructing the latent variable D to represent overall functional impairment integrates information from multiple functional scales. This approach reduces measurement error associated with individual instruments and enhances the stability and interpretability of the proposed "function–sleep/fatigue–depressive symptoms" association framework. Second, mediation effects were evaluated using nonparametric bootstrap procedures with 2,000 resamples to estimate confidence intervals. This approach provides more robust statistical inference than conventional normal-theory approaches and improving the reliability of indirect effect estimation. Third, SEM-2 was implemented as a sensitivity analysis by simultaneously including and mutually adjusting Barthel_rev and mRS. This approach clarified the relative contributions of different functional measures within the PSD pathway. As a result, it enhanced the interpretability, reproducibility, and translational relevance of the findings.

Several limitations should also be acknowledged. First, the single-center, retrospective, cross-sectional design involved concurrent assessment of all variables. Although SEM supports internal consistency of hypothesized pathways, it cannot establish causal or temporal relationships. These require confirmation through longitudinal designs. This caution is also consistent with recent longitudinal symptom-network research in new-onset stroke patients, in which cross-lagged panel network analysis showed that depressive symptoms may interact dynamically over time rather than follow a simple unidirectional sequence.[33] Second, key variables (PHQ-9, PSQI, and FAS) were assessed using self-report instruments. These may be subject to common-method bias and symptom overlap, potentially attenuating the specificity of estimated path coefficients. Third, several potentially important confounders such as pain severity, medication use, OSA, anxiety symptoms, and stroke lesion characteristics were not fully controlled, which may have influenced the estimated indirect associations and attenuated the strength of the conclusions. Fourth, the relatively modest sample size limited statistical power for more complex stratified analyses, and findings derived from an inpatient rehabilitation population should be generalized cautiously to outpatient or community-based settings.

Future research may build on these findings in several directions. First, prospective longitudinal studies with repeated assessments of functional status, sleep quality, fatigue, and depressive symptoms at multiple time points are needed. Approaches such as cross-lagged panel network analysis may be particularly helpful for capturing temporal symptom interactions that cannot be addressed in a single-time-point SEM framework.[33] Application of cross-lagged panel models or longitudinal SEM would allow verification of temporal sequencing within the proposed "sleep/fatigue → depression" pathways. Second, incorporation of objective and more granular indicators such as wearable device–derived sleep parameters, characterization of OSA subtypes, and biological markers related to fatigue may reduce common-method bias and enhance mechanistic interpretation. Third, interventional studies evaluating integrated sleep- and fatigue-focused management strategies, including insomnia-focused CBT and individualized optimization of rehabilitation prescriptions, are warranted to determine their effects on PHQ-9 scores and functional recovery, thereby translating pathway-based evidence into clinical benefit.

Conclusion

Overall, this study provides theory-informed cross-sectional evidence that functional impairment, sleep quality, fatigue, and depressive symptoms are interrelated in a real-world inpatient rehabilitation population. The findings suggest that sleep quality may represent a more consistent statistical pathway linking functional impairment and depressive symptoms, whereas the role of fatigue appears to depend on the functional measure and model specification. By highlighting sleep quality and fatigue as clinically relevant and potentially modifiable correlates within this association pattern, the study may help inform comprehensive post-stroke rehabilitation and psychological management. These findings should be interpreted as hypothesis-generating and supportive of targeted screening considerations, rather than as evidence for a confirmed intervention pathway.

Abbreviations

PHQ-9, Patient Health Questionnaire-9; PSQI, Pittsburgh Sleep Quality Index; mRS, Modified Rankin Scale; PSD, Post-stroke depression; ADL, Activities of Daily Living; PSF, Post-stroke fatigue; BI, Barthel Index; FAS, Fatigue Assessment Scale; SEM, Structural Equation Modeling; BMI, Body Mass Index; NIHSS, National Institutes of Health Stroke Scale; TIA, Transient Ischemic Attack; ML, Maximum likelihood; SRMR, Standardized Root Mean Square Residual; CI, confidence intervals; RMSEA, Root Mean Square Error of Approximation; OSA, Obstructive Sleep Apnea; CBT, Cognitive Behavioral Therapy.

Data Sharing Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki (as was revised in 2013). The study was approved by Ethics Committee of the Affiliated Hospital of Hebei University (Approval Number: HDFYLL-KY-2023-138). Written informed consent was obtained from all participants.

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Disclosure

The authors declare that they have no competing interests.

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