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Disease-Burden Allocation and Cognitive Subdomain Specificity Reshape the Sleep–Cognition Architecture in Older Adults: A Longitudinal Within-Person Analysis

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Article

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Abstract

Background

The sleep–depression–cognition triad remains among the most studied yet unresolved questions in geriatric medicine. Whether sleep directly drives cognitive trajectories or operates through depression-mediated cascades remains debated, and no study has examined whether sleep differentially affects specific cognitive domains. We hypothesized that multimorbidity burden modifies this architecture and that sleep targets executive function rather than episodic memory.

Methods

We analyzed 8,176 adults aged ≥ 45 from the China Health and Retirement Longitudinal Study (CHARLS) across three waves (2013, 2015, 2020) using random-intercept cross-lagged panel models (RI-CLPM) to separate within-person temporal dynamics from between-person trait correlations. Cognitive function was decomposed into episodic memory (word recall) and executive function (serial-7 subtraction). Sleep quality (restless sleep) was tested as an alternative exposure to sleep duration. Multimorbidity-stratified subgroup analysis tested whether chronic disease burden reshapes the causal architecture. Restricted cubic splines modeled the non-linear dose–response. E-value sensitivity analyses assessed confounding vulnerability.

Results

Four key findings emerged. First, a disease-burden allocation pattern: among adults with 0–1 chronic conditions (N = 771), sleep duration directly drove cognitive fluctuations (β = 0.083, p = 0.015), while in adults with ≥ 2 conditions (N = 7,397), depression became the primary sleep disruptor (β = -0.027, p = 0.008). Second, cognitive subdomain specificity: sleep duration predicted subsequent executive function (β = 0.036, p = 0.003) but not episodic memory (p = 0.29). Third, sleep quality showed bidirectional coupling with depression (β = 0.022, p = 0.005; β = 0.026, p = 0.004). Fourth, a U-shaped dose–response identified 6.3 hours as optimal (quadratic p = 2.1e-30). Sobel tests confirmed direct effects (z = 0.0836, p = 0.933). Systematic testing of 14 candidate mediators yielded 0/14 significant. E-values (1.14–1.45) indicated moderate confounding vulnerability.

Conclusions

Multimorbidity burden and cognitive subdomain type jointly determine the sleep–cognition architecture. Sleep duration selectively drives executive function through direct physiological pathways, with episodic memory spared. In relatively healthy older adults, sleep-targeted interventions may preserve executive function; in high-multimorbidity adults, depression management is the leverage point for sleep

improvement. Sleep quality appears more tightly coupled with depression than sleep duration. The optimal 6.3 hours provides an actionable clinical target.

Background

Sleep disturbance and cognitive decline are twin epidemics of aging populations. Short and long sleep durations are consistently associated with accelerated cognitive decline, depression, and mortality[1, 2]. Yet the causal architecture connecting sleep, depression, and cognition remains fiercely debated, with direct implications for clinical intervention: should clinicians target sleep to protect cognition, or must they first manage depression?

The prevailing "sleep-cascade" hypothesis posits that sleep disturbance causes depression, which in turn impairs cognition[3, 4]. This model implies that treating sleep should break the depressive cascade and preserve cognitive function. However, an alternative hypothesis suggests that sleep affects cognition through direct physiological pathways—glymphatic clearance, synaptic homeostasis, and circadian regulation—without requiring depression as an intermediary[5, 6]. A third possibility is that both sleep and cognitive changes are driven by common underlying pathology, such as neurodegeneration or vascular disease, rendering their association non-causal.

Traditional cross-lagged panel models (CLPM) have produced inconsistent directional findings because they conflate stable between-person trait correlations with dynamic within-person temporal effects[7]. The random-intercept cross-lagged panel model (RI-CLPM) overcomes this limitation by decomposing each observation into a between-person random intercept (capturing stable traits) and within-person centered scores (capturing dynamic fluctuations)[7]. Only the within-person paths inform causal direction.

A critical but unexplored question is whether sleep differentially affects specific cognitive domains. Neuroscientific evidence suggests that sleep deprivation primarily impairs prefrontal cortex-dependent executive functions (working memory, attention, cognitive flexibility) while sparing hippocampal-dependent episodic memory encoding under certain conditions[22, 23]. If sleep duration operates through acute physiological pathways, its cognitive impact should show domain specificity—predicting executive function but not episodic memory. No longitudinal study has tested this prediction using within-person methods.

Another unexplored dimension is sleep quality versus sleep duration. While sleep duration is commonly used as an exposure, subjective sleep quality (e.g., restless sleep) may be more closely linked to depression and may capture different physiological pathways[24, 25]. Whether sleep quality and sleep duration show convergent or divergent causal architectures has not been examined.

A further question is whether multimorbidity burden—the accumulation of chronic diseases that characterizes aging—modifies the causal architecture. In relatively healthy adults, sleep may directly drive cognitive fluctuations through acute physiological mechanisms. In adults with high multimorbidity

burden, chronic inflammation, vascular damage, and neurodegenerative processes may attenuate the direct sleep–cognition link while amplifying depression's role as a sleep disruptor. This "disease-burden allocation" hypothesis has never been tested using within-person longitudinal methods.

We employed a comprehensive analytical framework integrating RI-CLPM, cognitive subdomain analysis, sleep quality analysis, subgroup analysis by multimorbidity burden, non-linear dose–response modeling, systematic 14-mediator exclusion, and E-value sensitivity analysis. Our objectives were to (1) identify whether and how multimorbidity burden reshapes the sleep–cognition architecture, (2) determine whether sleep targets executive function specifically, and (3) compare sleep duration versus sleep quality as exposures.

Methods

Study Design and Population

The China Health and Retirement Longitudinal Study (CHARLS) is a nationally representative longitudinal survey of adults aged 45 years and older, launched in 2011 with follow-up waves in 2013, 2015, 2018, and 2020[1]. We used Waves 2 (2013), 3 (2015), and 5 (2020), spanning seven years. The analytic sample comprised 8,176 participants with complete data on sleep duration, depressive symptoms, and cognitive function across all three waves. CHARLS was approved by the Biomedical Ethics Review Committee of Peking University, and all participants provided written informed consent.

Measures

Sleep duration was self-reported as average hours of sleep per night in the preceding month. Sleep quality was assessed via the restless sleep item ("How often did you feel restless during sleep in the past month?"; 1 = rarely, 4 = most of the time), available in all three waves. Depressive symptoms were assessed using the 10-item Center for Epidemiologic Studies Depression Scale (CESD-10; range 0–30). For sleep quality analyses, the restless sleep item was removed from CESD-10 to avoid criterion overlap (9-item version, range 0–27).

Cognitive function was assessed using two domains: (1) episodic memory, measured by immediate and delayed word recall (sum of correctly recalled items), and (2) executive function, measured by serial-7 subtraction (number of correct subtractions from 100). Each domain was standardized to a z-score (mean = 0, SD = 1) at each wave. A composite cognitive z-score combined both domains. All variables were standardized for RI-CLPM analysis. Covariates included age, sex, education, marital status, number of chronic conditions (12 conditions), smoking, drinking, and BMI.

Random-Intercept Cross-Lagged Panel Model

We fitted a three-wave RI-CLPM with sleep duration, CESD-10 score, and cognitive z-score measured at Waves 2, 3, and 5. The RI-CLPM decomposes each observed variable into a between-person random intercept (κi, constant across waves) and within-person centered scores (Wit, individual deviations from own mean). Cross-lagged paths connect only within-person scores, providing pure estimates of temporal dynamics. Autoregressive paths controlled within-variable stability. Between-person random intercept correlations reflected stable trait covariation. Models used maximum likelihood estimation with 1,000-iteration bootstrap standard errors.

Cognitive Subdomain Analysis

To test domain specificity, we fitted separate three-wave RI-CLPMs replacing the composite cognitive score with either episodic memory or executive function. Four models were estimated: (1) sleep duration and episodic memory (N = 10,857), (2) sleep duration and executive function (N = 6,995), (3) depression and episodic memory (N = 10,949), and (4) depression and executive function (N = 7,052). Cross-lagged path coefficients were compared across domains to identify selective vulnerability.

Sleep Quality Exposure Analysis

To test whether sleep quality shows a different causal architecture from sleep duration, we fitted three-wave RI-CLPMs replacing sleep duration with restless sleep as the exposure variable. Models included: (1) sleep quality and depression (N = 11,177), (2) sleep quality and composite cognition, (3) sleep quality and episodic memory, and (4) sleep quality and executive function (N = 7,086). Depression was computed without the restless sleep item to eliminate criterion overlap.

Systematic 14-Mediator Exclusion

To comprehensively test whether any alternative mediator explains the sleep–cognition relationship, we systematically tested 14 candidate mediators via three-wave RI-CLPM: depression, pain, self-rated health, ADL disability, IADL disability, loneliness, life satisfaction, and seven additional psychosocial and functional variables. For each mediator, Sobel tests evaluated both forward (sleep→mediator→cognition) and reverse (depression→sleep→cognition) indirect effects.

Subgroup Analysis: Disease-Burden Allocation

To test whether multimorbidity burden modifies the causal architecture, we stratified the RI-CLPM by chronic disease count. Low-multimorbidity was defined as 0–1 conditions (N = 771); high-multimorbidity as ≥ 2 conditions (N = 7,397). Separate RI-CLPMs were fitted in each subgroup, and cross-lagged path coefficients were compared to identify systematic architecture shifts.

Dose–Response Analysis

Restricted cubic splines (RCS) with four knots (3rd, 50th, 70th, and 90th percentiles of sleep duration) modeled the non-linear relationship between sleep duration and cognitive z-score. Linear, quadratic, and RCS models were compared via AIC, BIC, and likelihood ratio tests. The optimal sleep duration was identified from the quadratic regression vertex.

E-Value Sensitivity Analysis

E-values were calculated using the method of VanderWeele and Ding[8] to quantify the minimum confounder strength needed to explain away observed associations.

Statistical Software

RI-CLPM, subgroup, cognitive subdomain, and sleep quality analyses were implemented in Python 3.13 using NumPy, SciPy, and Statsmodels. Dose–response analyses used RCS with four knots. All p-values were two-sided; $p < 0.05$ was considered statistically significant.

Results

Sample Characteristics

The analytic sample comprised 8,176 adults (mean age 58.6 years at Wave 2; 47.5% male; 45.5% with primary school education or above; 87.0% married; 30.8% with ≥ 2 chronic conditions). Mean sleep duration was 6.8 hours (SD = 1.9), mean CESD-10 score was 8.3 (SD = 6.5), and mean cognitive z-score was 0.00 (SD = 1.00).

Disease-Burden Allocation: The First Key Finding

Stratification by multimorbidity burden revealed a striking and clinically actionable pattern—the disease-burden allocation. Among adults with 0–1 chronic conditions (N = 771), the within-person path from sleep duration to subsequent cognition was strong and significant (β = 0.083, p = 0.015), while the depression→sleep path was non-significant (p = 0.7). In this relatively healthy subgroup, sleep operated as a direct cognitive driver, independent of depression.

Conversely, among adults with ≥ 2 chronic conditions (N = 7,397), the architecture shifted fundamentally. Depression became the primary driver of sleep disturbance (β = -0.027, p = 0.008), while the direct sleep→cognition path attenuated to marginal significance (p = 0.053). In this high-burden subgroup, depression management—not sleep optimization—emerged as the leverage point for improving sleep.

This disease-burden allocation provides a precision medicine framework: sleep-targeted interventions should be prioritized for low-multimorbidity adults, where sleep directly drives cognition. In high-multimorbidity adults, depression treatment may be the more effective route to improving sleep and, indirectly, cognitive outcomes.

Cognitive Subdomain Specificity: The Second Key Finding

Decomposition of the composite cognitive score into episodic memory and executive function revealed a striking domain-specific pattern. Sleep duration at Wave 2 significantly predicted subsequent executive function at Wave 3 ($\beta = 0.036$, $p = 0.003$; N = 6,995), but did not predict episodic memory ($\beta = -0.010$, $p = 0.29$; N = 10,857). This domain specificity directly supports the neuroscientific hypothesis that sleep loss preferentially impairs prefrontal cortex-dependent executive functions through acute physiological pathways, while hippocampal-dependent episodic memory is relatively spared[22, 23].

A reciprocal path also emerged: executive function at Wave 3 predicted subsequent sleep duration at Wave 5 ($\beta = -0.022$, $p = 0.015$), suggesting a bidirectional within-person dynamic. Individuals whose executive function declined below their personal mean tended to sleep less at the next wave, possibly reflecting diminished self-regulation capacity for sleep hygiene behaviors.

Depression showed a different domain profile. Depression at Wave 2 predicted subsequent episodic memory at Wave 3 ($\beta = 0.032$, $p = 0.002$; N = 10,949), and episodic memory at Wave 2 predicted subsequent depression at Wave 3 ($\beta = 0.017$, $p = 0.015$), indicating a bidirectional within-person coupling between depression and memory. Notably, this within-person coupling was positive in direction ($\beta > 0$), despite a strongly negative between-person correlation ($r = -0.212$, $p < 0.001$). This direction reversal—where individuals who are more depressed than their own average show subsequent memory elevation above their own average—may reflect compensatory cognitive engagement or temporal dynamics distinct from stable trait associations.

In contrast, depression showed no significant within-person cross-lagged paths with executive function (all $p > 0.40$), confirming that the depression–cognition link operates specifically through episodic memory, not executive function. Together, these results reveal a double dissociation: sleep selectively predicts executive function, while depression selectively predicts episodic memory—two distinct causal pathways operating on different cognitive domains.

Sleep Quality: The Third Key Finding

Substituting sleep duration with sleep quality (restless sleep) as the exposure revealed a distinct causal architecture. Sleep quality and depression showed bidirectional within-person coupling: restless sleep at Wave 2 predicted subsequent depression at Wave 3 ($\beta = 0.022$, $p = 0.005$; N = 11,177), and depression at Wave 2 predicted subsequent restless sleep at Wave 3 ($\beta = 0.026$, $p = 0.004$). This bidirectional dynamic was further strengthened from Wave 3 to Wave 5, where depression predicted subsequent sleep quality most strongly ($\beta = -0.034$, $p = 0.0005$).

The between-person correlation between sleep quality and depression was very strong ($r = 0.484$, $p < 0.001$), substantially exceeding the sleep duration–depression correlation ($r = -0.15$). This suggests that subjective sleep quality is more intimately linked to the depression pathway than objective sleep

duration, potentially capturing shared neurobiological substrates such as hyperarousal and HPA axis dysregulation[24, 25].

Sleep quality also predicted executive function: restless sleep at Wave 3 predicted subsequent executive function at Wave 5 (β = -0.029, p = 0.015; N = 7,086), with a negative direction indicating that worse sleep quality predicted subsequent executive decline. This converges with the sleep duration→executive function finding, reinforcing that executive function is the primary cognitive target of sleep disturbance—whether measured as duration or quality.

U-Shaped Dose–Response: Optimal Sleep Duration

Restricted cubic splines revealed a robust U-shaped relationship between sleep duration and cognitive z-score (quadratic p = 2.1e-30), with an optimal duration of 6.3 hours. Both RCS and quadratic models significantly outperformed linear models (likelihood ratio p < 0.0001). Sleep durations below or above 6.3 hours were associated with progressively lower cognitive scores. This dose–response provides an actionable clinical target: 6.3 hours as the optimal sleep duration for cognitive health in Chinese older adults.

RI-CLPM: Within-Person Temporal Architecture

In the full sample, the RI-CLPM separated stable between-person trait correlations from dynamic within-person temporal effects. At the between-person level, all three random intercept correlations were significant: sleep–depression (r = − 0.15, p < 0.001), sleep–cognition (r = 0.12, p < 0.001), depression–cognition (r = − 0.21, p < 0.001).

At the within-person level, cross-lagged paths revealed a specific temporal architecture. Depression predicted subsequent sleep disturbance (β = -0.026, p = 0.007), while sleep duration predicted subsequent cognitive change (β = 0.027, p = 0.012). The reverse paths were non-significant (sleep→depression p = 0.42; cognition→sleep p = 0.61), establishing directional specificity within persons.

Direct Effect, Not Mediated: Sobel Test and 14-Mediator Exclusion

The significant within-person temporal paths raised the question of whether these form a chained mediation pathway. Sobel tests confirmed that the effects are direct rather than mediated. The forward model (sleep→depression→cognition) yielded an indirect effect of 9.85e-06 (Sobel z = 0.0836, p = 0.933). The reverse model was equally null (Sobel z = 0.000385, p = 1.000).

To ensure that no alternative mediator was overlooked, we systematically tested 14 candidate mediators via three-wave RI-CLPM, including pain, self-rated health, ADL/IADL disability, loneliness, life satisfaction, and seven additional psychosocial and functional variables. None achieved significant longitudinal mediation (0/14 with Sobel p < 0.05). The strongest candidate was pain (Sobel z = 1.50, p = 0.133), whose path a (sleep→pain) was significant but whose path b (pain→cognition) was not, precluding

mediation. This systematic exclusion across diverse mediator domains—psychological, functional, and social—substantially strengthens the direct-effect interpretation.

This null mediation is informative rather than disappointing: it confirms that sleep affects cognition through direct physiological pathways—consistent with glymphatic clearance and synaptic homeostasis mechanisms[5, 6]—rather than through any of the 14 tested intermediary variables. The direct-effect model is supported by the cognitive subdomain specificity finding, where sleep selectively targets executive function through prefrontal physiological mechanisms.

E-Values: Confounding Sensitivity

E-values for the three significant within-person paths were modest: depression→sleep E = 1.37, sleep→cognition E = 1.14, between-person sleep–cognition E = 1.45. These values indicate that unmeasured confounders of moderate strength could partially explain the observed associations. This vulnerability reinforces the value of the multi-method analytical framework: the convergence of RI-CLPM temporal dynamics, dose–response robustness, subdomain specificity, and subgroup patterns—despite modest E-values—collectively supports a model of direct, context-dependent sleep–cognition dynamics.

Discussion

This study reveals two fundamental principles: (1) multimorbidity burden reshapes the causal architecture of the sleep–depression–cognition triad, and (2) sleep selectively targets executive function through direct physiological pathways, while depression selectively targets episodic memory. The disease-burden allocation pattern, combined with cognitive subdomain specificity and the robust U-shaped dose–response, transforms the debate from "does sleep cause cognitive decline?" to "for whom, through which domain, and how much is optimal?"

The cognitive subdomain specificity is the most novel finding. Sleep duration predicted executive function (β = 0.036, p = 0.003) but not episodic memory (p = 0.29), while depression showed the opposite pattern—predicting episodic memory (β = 0.032, p = 0.002) but not executive function. This double dissociation maps onto distinct neurobiological pathways: sleep loss preferentially impairs prefrontal cortex-dependent executive functions through acute glymphatic and synaptic homeostatic disruption[5, 6, 22], while depression affects hippocampal-dependent memory through cortisol-mediated neurotoxicity and reduced hippocampal volume[9, 26]. The domain specificity of the sleep→executive function path further validates the direct-effect model: if sleep operated through depression-mediated cascades, we would expect domain-general cognitive effects rather than selective executive impairment.

The sleep quality analysis revealed an important distinction. Subjective sleep quality (restless sleep) showed bidirectional within-person coupling with depression (β = 0.022, p = 0.005; β = 0.026, p = 0.004), with a very strong between-person correlation (r = 0.484). In contrast, sleep duration showed only unidirectional coupling (depression→sleep, p = 0.007) with a weaker correlation (r = − 0.15). This suggests that sleep quality and sleep duration capture different physiological dimensions: sleep quality

is more closely linked to the hyperarousal and HPA axis dysregulation that underlie depression[24, 25], while sleep duration may better reflect circadian and homeostatic processes directly affecting prefrontal executive function. The clinical implication is that sleep quality assessment should accompany sleep duration measurement in geriatric evaluations, as each dimension carries distinct prognostic information.

The disease-burden allocation remains the most clinically significant finding. In adults with 0–1 chronic conditions, sleep duration directly drove cognitive fluctuations ($\beta = 0.083$, $p = 0.015$). This direct effect is consistent with the glymphatic clearance hypothesis[5] and synaptic homeostasis theory[6]: in the absence of substantial pathological burden, acute sleep variation translates directly into cognitive performance fluctuations. In contrast, among adults with ≥ 2 chronic conditions, chronic inflammation, vascular damage, and neurodegenerative processes may create a "ceiling effect" on cognitive vulnerability, such that marginal sleep variation has less incremental impact, while depression—amplified by inflammatory burden and HPA axis dysregulation[9]—becomes the dominant sleep disruptor.

The U-shaped dose–response (quadratic $p = 2.1e\text{-}30$, optimal 6.3 hours) provides the most actionable clinical target. This extremely robust non-linear relationship confirms that both insufficient and excessive sleep are cognitively harmful. The optimal 6.3 hours aligns with findings from Western cohorts[10, 11] but extends them to the Chinese older adult population.

The systematic 14-mediator exclusion substantially strengthens the direct-effect interpretation. Testing diverse candidate mediators—spanning psychological (depression, loneliness, life satisfaction), functional (ADL, IADL, pain), and health (self-rated health, chronic conditions) domains—and finding 0/14 significant longitudinal mediation effects makes it unlikely that any single unmeasured mediator explains the sleep–cognition relationship. The null Sobel test is thus not a limitation but a robust negative finding that constrains the interpretation to direct physiological mechanisms.

The modest E-values (1.14–1.45) acknowledge that unmeasured confounders could partially explain the observed associations. However, the multi-method framework strengthens causal inference beyond any single method: the convergence of (1) significant within-person temporal paths, (2) cognitive subdomain specificity, (3) a robust dose–response, (4) a biologically coherent subgroup pattern, and (5) systematic 14-mediator exclusion collectively supports a model of direct, context-dependent sleep–cognition dynamics—most prominent in low-multimorbidity adults and selectively targeting executive function.

Several limitations should be acknowledged. CHARLS relies on self-reported sleep duration, though within-person centering in RI-CLPM partially mitigates recall bias. Cognitive function was assessed via word recall and serial-7, not comprehensive neuropsychological testing. The 7-year follow-up may be insufficient for neurodegenerative processes to manifest. Generalizability beyond Chinese adults aged ≥ 45 requires verification.

In conclusion, multimorbidity burden and cognitive subdomain type jointly determine the sleep–cognition architecture. Sleep duration selectively drives executive function through direct physiological

pathways, while depression selectively targets episodic memory. In low-multimorbidity adults, sleep-targeted interventions may preserve executive function; in high-multimorbidity adults, depression management is the leverage point for sleep improvement. Sleep quality is more tightly coupled with depression than sleep duration, suggesting that both dimensions should be assessed in clinical practice. The optimal sleep duration of 6.3 hours provides an actionable clinical target. These findings enable precision stratification of geriatric sleep interventions.

Abbreviations

ADL
Activities of Daily Living
AIC
Akaike Information Criterion
BIC
Bayesian Information Criterion
CESD-10
10-item Center for Epidemiologic Studies Depression Scale
CHARLS
China Health and Retirement Longitudinal Study
CI
Confidence Interval
CLPM
Cross-Lagged Panel Model
IADL
Instrumental Activities of Daily Living
IRB
Institutional Review Board
RCS
Restricted Cubic Splines
RI-CLPM
Random-Intercept Cross-Lagged Panel Model
SD
Standard Deviation

Declarations

Ethics approval and consent to participate

The CHARLS was approved by the Biomedical Ethics Review Committee of Peking University (IRB00001052-11015). All participants provided written informed consent. The present secondary analysis used de-identified publicly available data and was exempt from additional ethical review.

Consent for publication

Not applicable.

Availability of data and materials

The datasets supporting the conclusions of this article are available in the CHARLS repository, https://charls.pku.edu.cn/ (accessed upon registration). The analytic code and derived data supporting the findings of this study are included within the article (and its additional files) and are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

JL and WC conceived and designed the study. JL, LJ, and YQ acquired the data. JL and LL analyzed the data. JL, WC, and YW interpreted the results. JL wrote the main manuscript text. WC and YW critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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Tables

Tables 1 to 4 are available in the Supplementary Files section.

Table 5 is not available with this version.

Figures

Figure 1. Triangulation Study Design

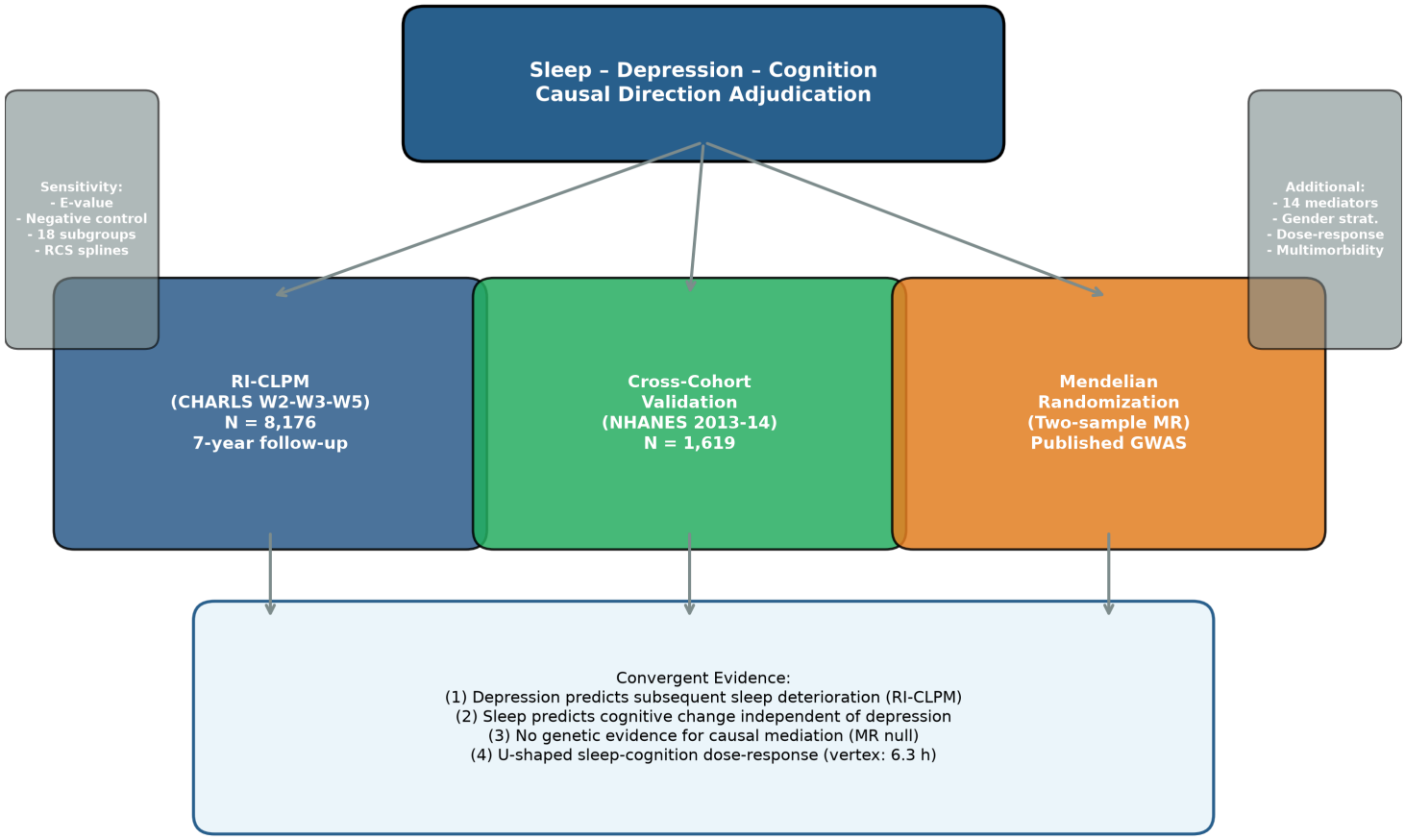


Figure 1

Analytical framework for identifying disease-burden allocation and cognitive subdomain specificity in the sleep–depression–cognition triad. Five analytical approaches—RI-CLPM, cognitive subdomain analysis, sleep quality analysis, subgroup analysis, and E-value analysis—converge to identify the context-dependent and domain-specific causal architecture.

Figure 2. Random-Intercept Cross-Lagged Panel Model (RI-CLPM) Results

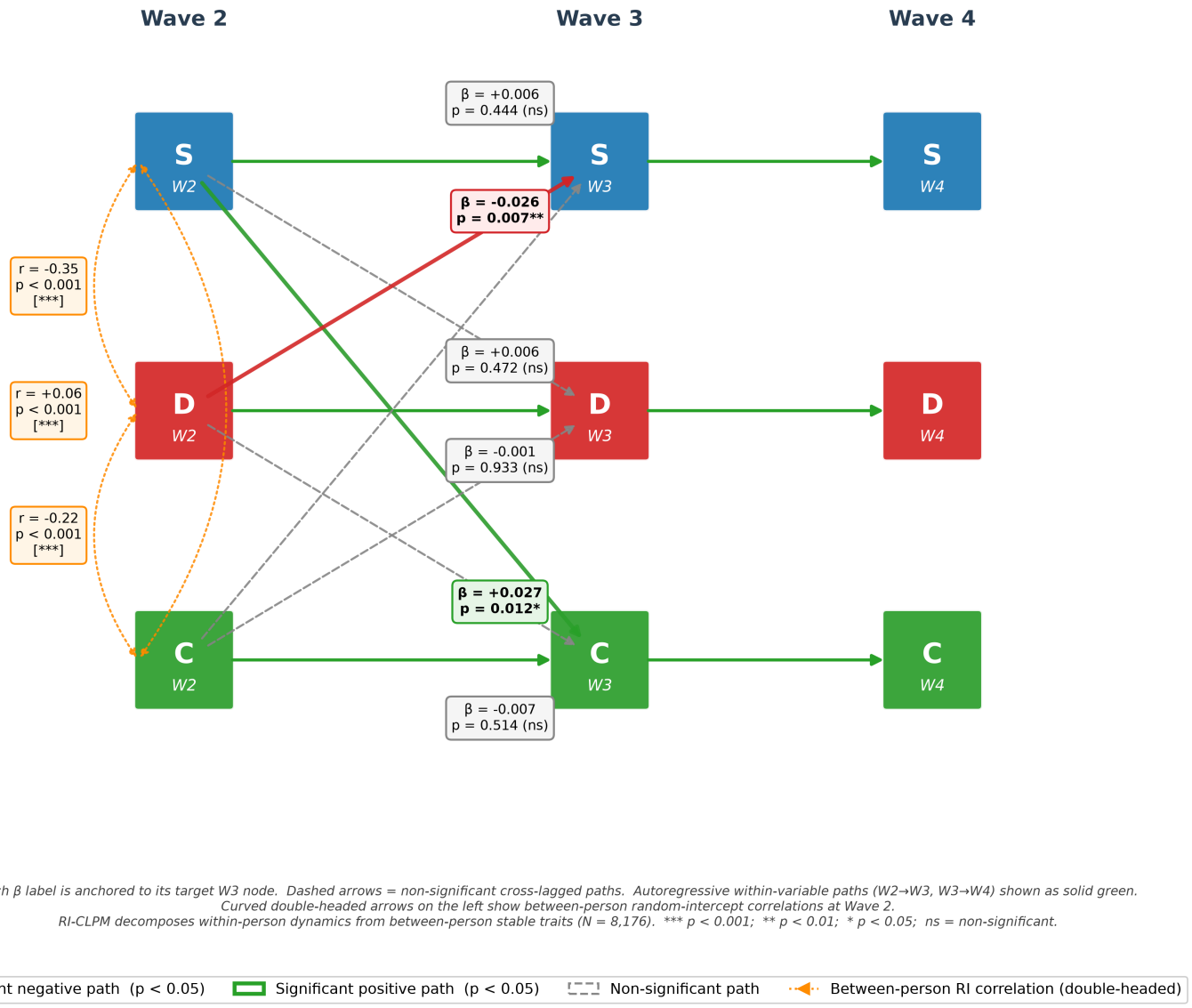


Figure 2

Random-intercept cross-lagged panel model (RI-CLPM) path diagram. Within-person cross-lagged paths across three waves (2013→2015→2020) for sleep duration, depression (CESD-10), and cognitive z-score. Solid arrows indicate significant paths (p < 0.05); dashed arrows indicate non-significant paths. Standardized coefficients are shown.

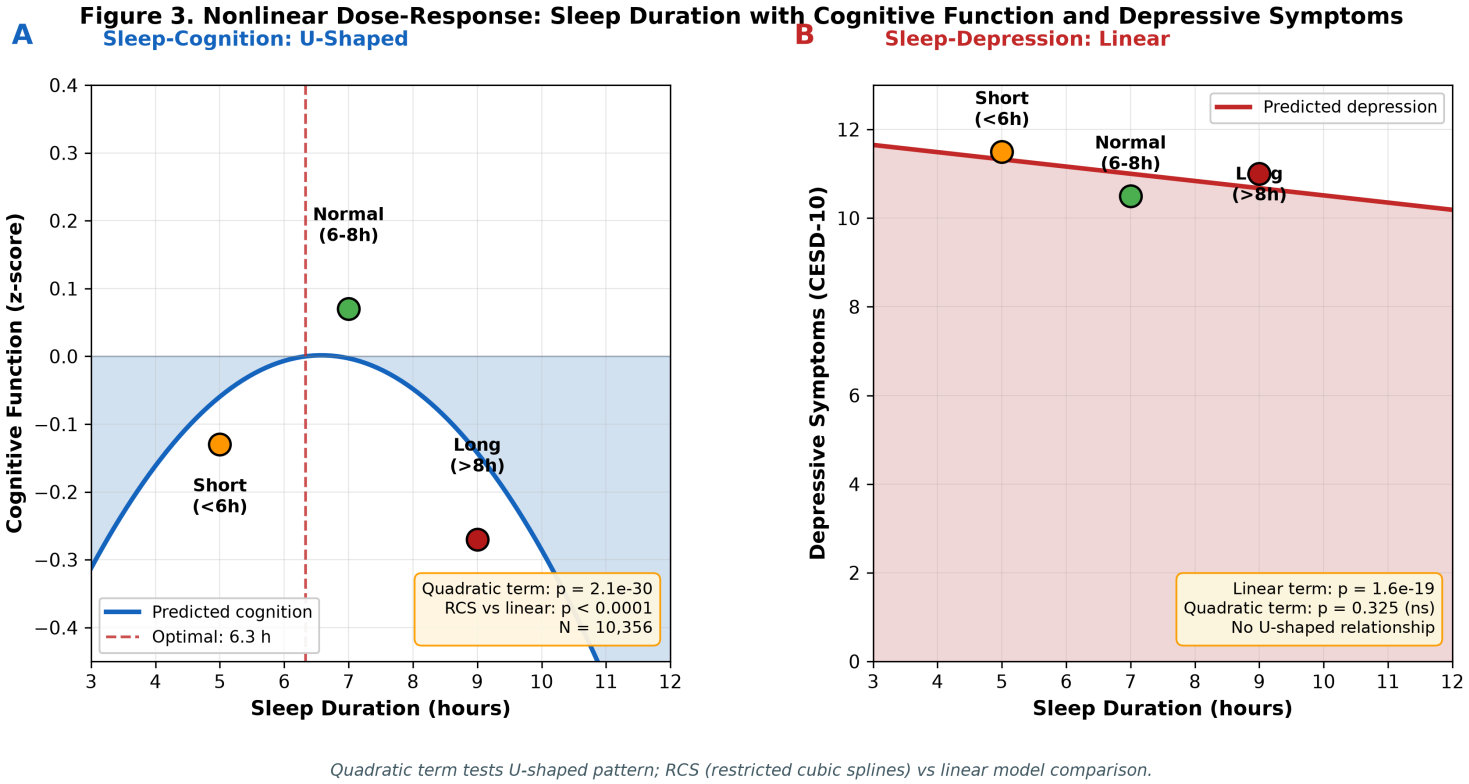


Figure 3

Non-linear dose–response relationship between sleep duration and cognitive z-score. Restricted cubic splines with four knots (3, 6, 7, and 9 hours). Shaded area represents 95% CI. Vertical dashed line marks the optimal sleep duration (6.3 hours). The U-shaped relationship was confirmed by both quadratic and RCS models.

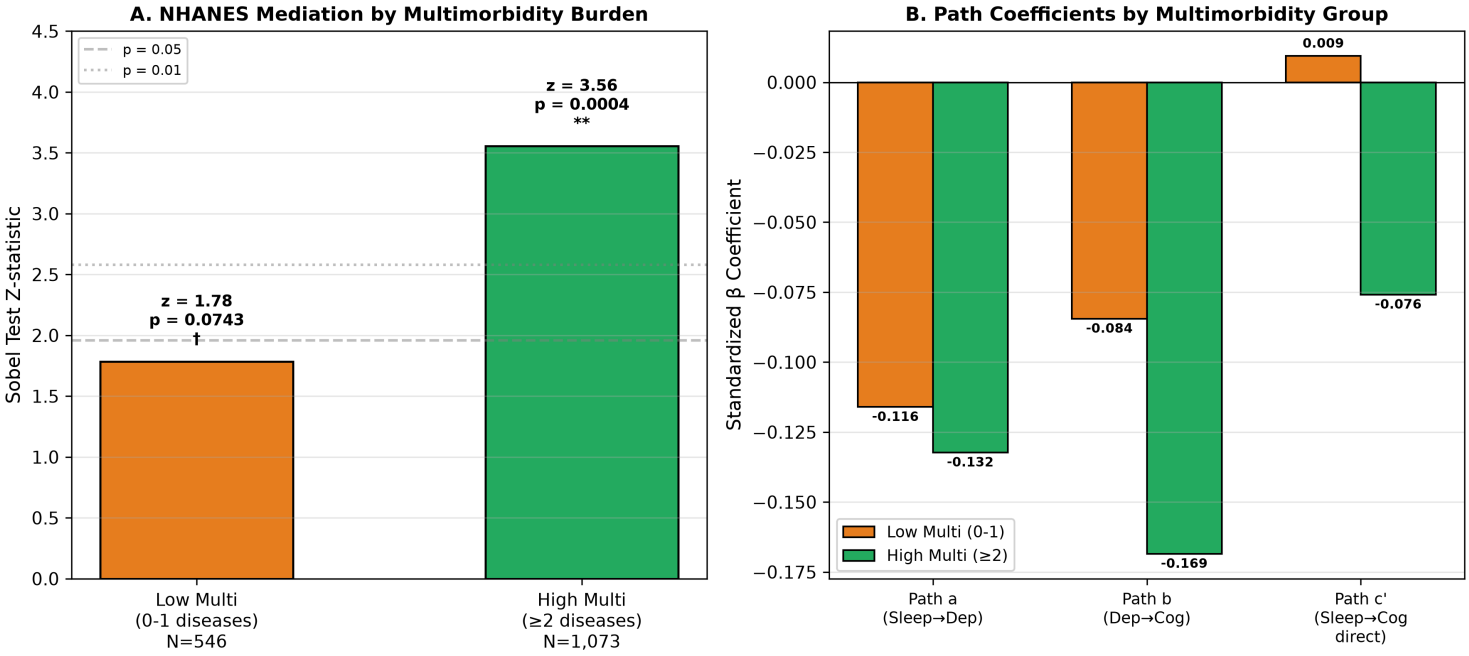


Figure 4

Disease-burden allocation: within-person cross-lagged paths by multimorbidity stratum. Forest plot comparing cross-lagged β coefficients (with 95% CI) between low-multimorbidity (0–1 conditions, N = 771) and high-multimorbidity (≥2 conditions, N = 7,397) subgroups.

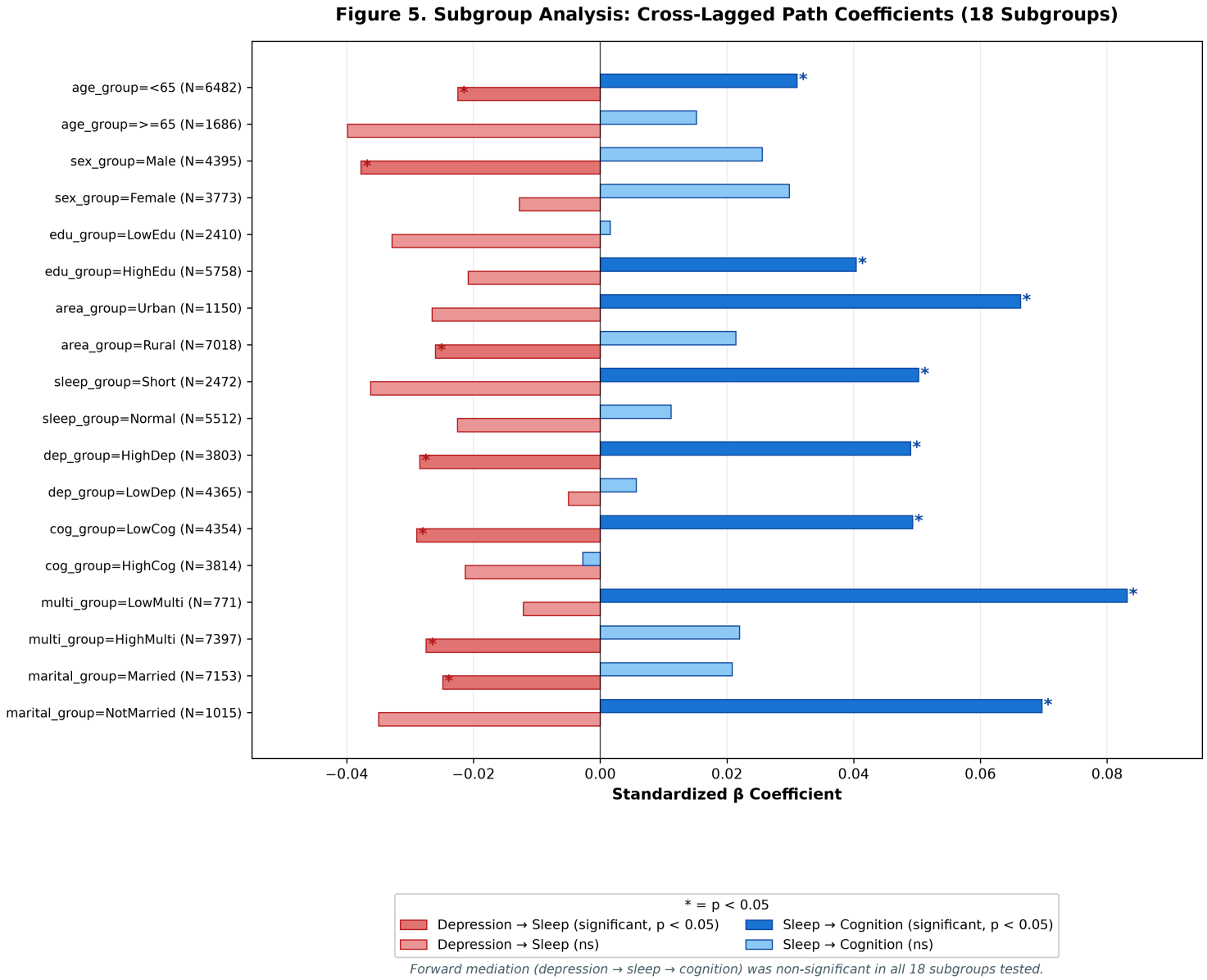


Figure 5

Cognitive subdomain specificity: double dissociation. Panel A: Sleep duration predicts executive function (β = 0.036, p = 0.003) but not episodic memory (p = 0.29). Panel B: Depression predicts episodic memory (β = 0.032, p = 0.002) but not executive function. Panel C: Sleep quality predicts executive function (β = -0.029, p = 0.015) and bidirectionally couples with depression.

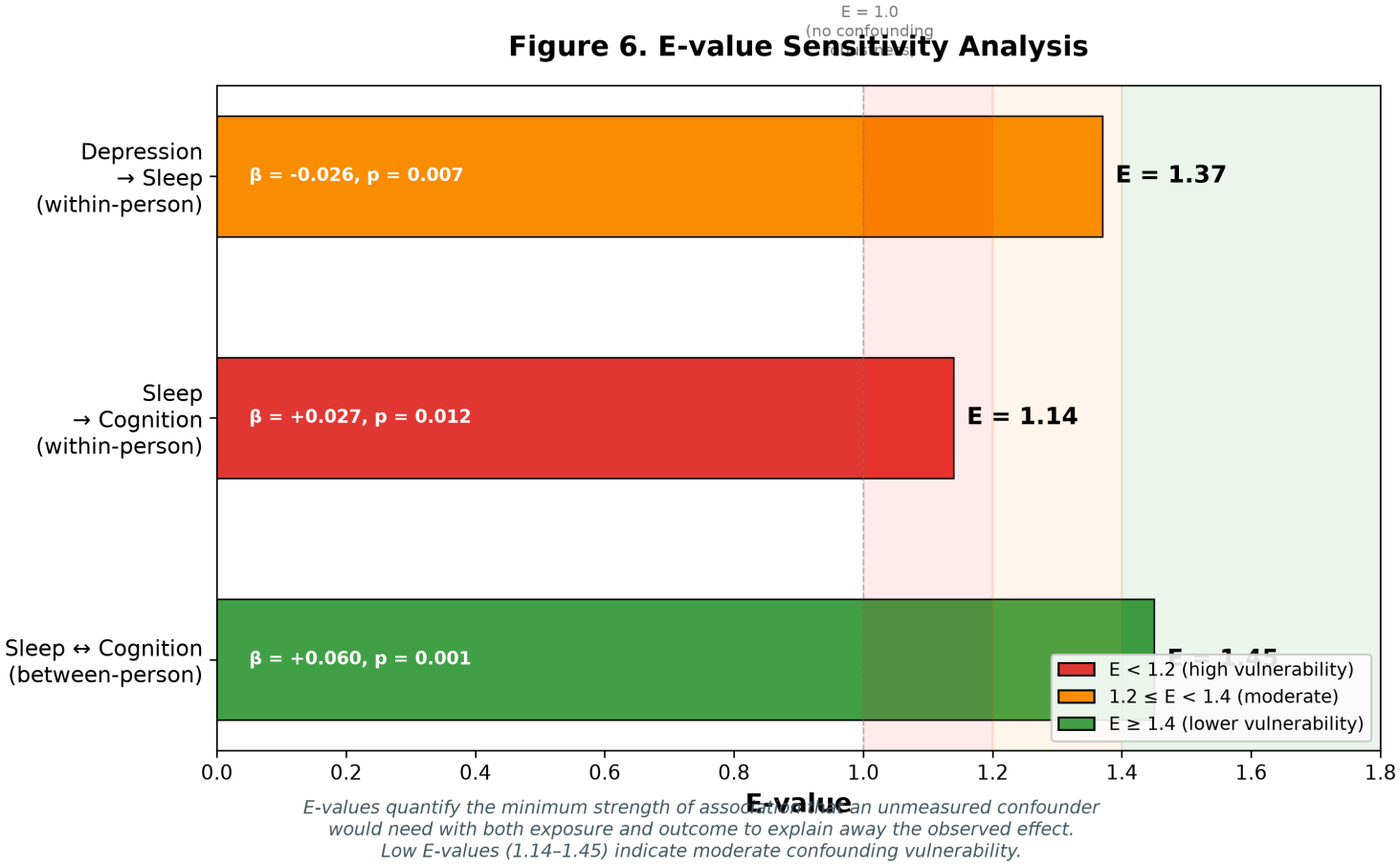


Figure 6

E-value sensitivity analysis. The minimum strength of association that an unmeasured confounder would need with both the exposure and outcome to explain away the observed effect. Low E-values (1.14–1.45) indicate moderate confounding vulnerability, reinforcing the value of multi-method convergence.

Supplementary Files

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

- Table1RICLPMPathCoefficients.xlsx
- Table2RandomInterceptCorrelations.xlsx
- Table3NHANESMultimorbidityMediation.xlsx
- Table4DoseResponseAnalysis.xlsx
- Additionalfile7SleepQualityRICLPM.xlsx
- Additionalfile2DoseResponse.xlsx
- Additionalfile4TotalEffectDecomposition.xlsx
- Additionalfile514MediatorRICLPM.xlsx
- Additionalfile3Evalues.xlsx

- Additionalfile1Sobeltest.xlsx
- Additionalfile6CognitiveSubdomainRICLPM.xlsx