

Neuroimaging Biomarkers in Sleep Disruptions: Current Evidence from a Radiology Perspective

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Received: 2026-08-30.

Accepted: 2026-09-23.



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J Clin Med Kaz 2026; 23(5):103-109

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ABSTRACT

Sleep disruption has emerged as an important contributor to cognitive, neurovascular, and metabolic dysfunction, yet its acute cerebral correlates remain incompletely characterized. This review synthesizes current evidence on structural, diffusion, functional, metabolic, and optical neuroimaging biomarkers associated with acute sleep deprivation from a radiology perspective. Structural magnetic resonance imaging studies show rapid but often reversible changes in gray matter morphology and perivascular spaces, whereas white matter signal abnormalities predominantly reflect underlying chronic vascular vulnerability. Diffusion-based approaches, including diffusion tensor and diffusion kurtosis imaging, and diffusion tensor image analysis along the perivascular space, reveal alterations in white matter microstructure and glymphatic transport that may reflect impaired waste clearance and increased vulnerability to sleep debt. Functional magnetic resonance imaging demonstrates disruptions in the default mode, thalamic, and frontoparietal networks, alongside impaired task-related activation and reduced attentional performance due to acute sleep deprivation. Positron emission tomography and magnetic resonance spectroscopy provide complementary metabolic and neurochemical evidence of reduced glucose utilization, incomplete recovery after deprivation, and sleep-related shifts in cerebral metabolites. Functional near-infrared spectroscopy further highlights deficits in prefrontal neurovascular coupling during acute sleep deprivation. Taken together, these modalities indicate that even short-term sleep disruption produces measurable and potentially clinically relevant changes in the brain. Determining whether these transient neuroimaging shifts predict long-term neurological consequences requires prospective, longitudinal studies with standardized acquisition and analysis pipelines to accelerate the clinical translation of quantitative sleep debt biomarkers.

Keywords: sleep deprivation, magnetic resonance imaging, biomarkers, prefrontal cortex, diffusion tensor imaging, glymphatic system, diagnostic imaging.

Introduction

Sleep is fundamental for maintaining neurophysiological homeostasis and supporting multi-systemic physiological functions [1]. Sleep disturbances are prevalent and can present as either quantitative or qualitative deficits. Conditions that compromise sleep continuity are collectively classified as sleep disruptions, which encompass acute insomnia, transient sleep deprivation (SD), sleep fragmentation, circadian disruptions such as jet lag, and stress-induced

parasomnias (which secondarily disrupt sleep architecture through nocturnal awakenings) [2]. The etiology of these disruptions is multifactorial, involving lifestyle factors, environmental stressors, primary sleep disorders, and underlying medical comorbidities [3].

Chronic sleep disruption and poor sleep quality are recognized risk factors for cardiovascular, metabolic, and cognitive morbidity and are directly associated with reduced life expectancy and diminished quality of life [4]. Even short-term or acute SD leads to cognitive decline,

cellular stress, impaired immunity, and emotional dysregulation [3,5,6].

Polysomnography (PSG) is the gold standard for objective sleep staging, measurement of sleep duration, fragmentation analysis, and diagnosis of obstructive sleep apnea. This technique simultaneously records multiple physiological parameters, including neurological activity (electroencephalography (EEG)), cardiac rhythms (electrocardiography), muscle tone (electromyography), ocular movements (electrooculography), oxygenation, respiratory effort, airflow, and motor activity. Monitoring these diverse signals is essential, as sleep quality directly impacts systemic health and long-term clinical outcomes [7]. Despite its status as the reference standard for sleep neurophysiology, PSG is limited by high operational costs, substantial equipment and personnel requirements, and restricted accessibility, which constrain its use for longitudinal or periodic monitoring [8].

While PSG and wearable devices give metrics of sleep quality, to estimate the effects of acute SD and sleep debt on the brain, neuroimaging is a valuable approach for assessing the structural and functional correlates of sleep insufficiency, as well as for mapping cerebral responses to sleep disruptions and therapeutic interventions [9,10]. Modalities such as structural, diffusion, and functional magnetic resonance imaging (MRI), positron emission tomography (PET), and functional near-infrared spectroscopy (fNIRS) (Table 1) provide direct insight into the underlying pathophysiological mechanisms of sleep disorders [11].

This review evaluates how advanced neuroimaging modalities detect and quantify the transient, short-term neurological shifts caused by acute SD and accumulated sleep debt. While our primary focus centers on acute experimental sleep loss protocols in healthy adults, established chronic sleep pathologies (such as chronic sleep fragmentation, obstructive sleep apnea, and insomnia) are deliberately synthesized as comparative baselines to highlight how acute sleep debt interacts with underlying structural and metabolic brain vulnerability. Using a modality-driven perspective, we assess how structural and diffusion MRI, functional MRI (fMRI), metabolic [PET/Magnetic resonance spectroscopy (MRS)], and optical (fNIRS) imaging modalities track acute

alterations in gray matter morphometry, white matter tracts, network connectivity, and biochemical balance. Finally, we highlight technical acquisition challenges, including scanner noise, fragmentation, and multi-site protocol harmonization, and outline how deep learning and radiomics can transition radiological reporting toward objective, quantitative sleep-debt profiling.

Methods

This narrative review synthesizes current evidence on neuroimaging biomarkers of acute SD. A comprehensive literature search was conducted across PubMed/MEDLINE, Scopus, and Web of Science databases for articles published between January 2010 and March 2026, with landmark historical papers included where relevant. The search string utilized combinations of Medical Subject Headings (MeSH) and free-text terms, including: ("sleep deprivation" OR "acute sleep loss" OR "sleep debt") AND ("neuroimaging" OR "magnetic resonance imaging" OR "positron emission tomography" OR "fNIRS" OR "diffusion tensor imaging" OR "radiomics").

Inclusion criteria focused on human neuroimaging studies evaluating healthy adult cohorts subjected to acute total or partial SD protocols. Studies examining chronic neurodegenerative disorders, movement disorders, or obstructive sleep apnea were excluded unless included as explicit baseline comparative controls.

Modalities and neuroimaging biomarkers of acute sleep debt

Structural neuroimaging biomarkers

A T1-weighted MRI is a foundational imaging sequence that provides high-resolution anatomical and structural details of the human brain, allowing quantification of transient cortical thinning, gray matter volume shifts, and structural alterations induced by acute SD, especially in the prefrontal cortex (PFC) (Figure 1), hippocampus, and thalamus [12,13].

In terms of specific brain changes, one night of SD induces rapid but reversible alterations in perivascular space volume

Table 1 Current neuroimaging modalities for evaluating acute sleep deprivation and sleep debt

Modality	Primary metric	Key advantages	Challenges in sleep context
Structural MRI	Gray matter volume, cortical thickness	High spatial resolution; excellent for quantifying long-term atrophy/ neurodegeneration.	Provides only a "snapshot" of structure; cannot capture dynamic sleep-state changes.
Diffusion MRI	White matter integrity, indices related to perivascular fluid transport	Maps structural connectivity and provides a diffusivity-based proxy for perivascular fluid transport and glymphatic clearance (via DTI-ALPS).	Technically demanding; sensitive to micro-movements during long scans.
Functional MRI	Neural activity (hemodynamic)	Superior spatial resolution; maps network connectivity (default mode network, executive control network) across sleep states.	Sensitive to motion; loud scanner noise can fragment sleep; requires a constrained environment.
PET	Glucose metabolism, proteinopathy (Tau/Amyloid)	Direct metabolic and molecular insight (clearance rates, protein accumulation).	Radiation exposure; requires injection of tracers; poor temporal resolution.
MRS	Neurometabolite levels (GABA+, glutamate+glutamine, N-acetylaspartate)	Direct, non-invasive quantification of cerebral neurochemistry and energy balance.	Low spatial resolution; spectral overlap; sensitive to motion artifacts during post-deprivation drowsiness.
fNIRS	Cortical hemodynamics	Portable and quiet; allows more "naturalistic" sleep recording than MRI.	Limited penetration depth (surface cortex only); lower spatial resolution.
EEG-functional MRI	Combined electrical & hemodynamic	Unmatched multimodal precision; synchronizes macro-sleep architecture with network dynamics.	Extremely complex setup; EEG cap interference with MRI; high participant discomfort.

DTI-ALPS – Diffusion tensor image analysis along the perivascular space; EEG – electroencephalography; fNIRS – functional near-infrared spectroscopy; GABA+ – Gamma-aminobutyric acid+; MRI – magnetic resonance imaging; MRS – magnetic resonance spectroscopy; PET – positron emission tomography.

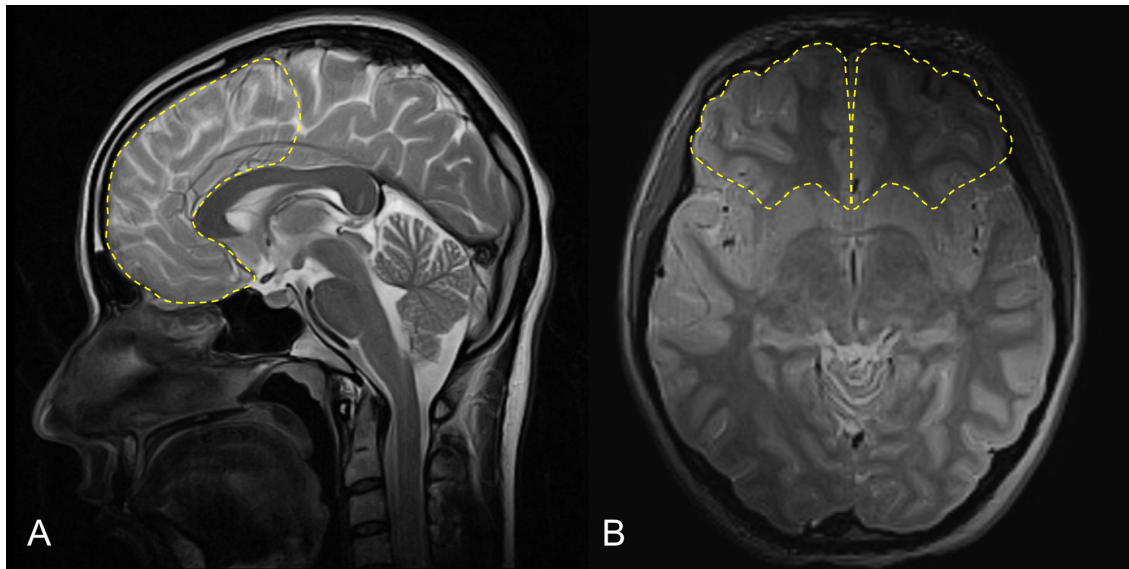


Figure 1 – Didactic anatomical baseline of the prefrontal cortex (PFC) on T2-weighted brain MRI: A – Sagittal scan delineating the boundaries of the PFC, serving as a structural reference for regions exhibiting acute resting-state functional (ALFF) drops and metabolic hypometabolism following sleep loss; B – Axial scan highlighting the bilateral PFC. This cortex regulates attention, working memory, and executive function, all of which decline with sleep debt.

ALFF – Amplitude of low-frequency fluctuations; MRI – magnetic resonance imaging; PFC – prefrontal cortex.

that correspond to microstructural tissue changes detectable as shifts in quantitative T1 and quantitative susceptibility maps [12]. This rapid structural plasticity reflects diurnal morning-to-evening thickness increases in the right prefrontal cortex during standard wakefulness, whereas subsequent overnight SD (23 hours) drives significant cortical thinning across bilateral medial parietal regions, specifically the precuneus and posterior cingulate cortex, which correlates directly with post-deprivation sleepiness [13]. T1-weighted MRI also showed a significant correlation between SD and age in the right anterior insula, while the main effect of disruptions was in the left postcentral gyrus, precuneus, and right thalamus [14]. These structural changes reflect known functional network disruptions and suggest how brain morphology responds to acute SD, depending on age and time of day.

T1-weighted MRI also allows the assessment of the impact of SD on human cerebral molecular clearance using intrathecal contrast-enhanced MRI [15]. Brain tracer enrichment has been tracked up to 48 hours following intrathecal administration of gadobutrol (0.5 mL, 1.0 mmol/mL) by quantifying percentage changes in normalized T1 signal intensity across 85 anatomical brain regions [16].

In contrast to the rapid, reversible structural changes observed following acute SD, T2/FLAIR white matter hyperintensities serve as markers of chronic ischemia and small vessel disease driven by chronic sleep fragmentation or hypoxemia [17]. This pathology is caused by chronic tissue hypoperfusion and blood-brain barrier breakdown [18], representing a baseline structural factor that increases brain vulnerability to acute sleep debt rather than an acute consequence of single-night sleep loss.

Microstructural diffusion imaging biomarkers

Conventional diffusion tensor imaging (DTI) models water mobility using a symmetric second-order 3D tensor, assuming unhindered, Gaussian water displacement. Consequently, this classical model assumes a monoexponential decay of the diffusion-weighted signal relative to the applied b-value [19]. To contextualize acute microstructural fluid shifts against chronic

baseline pathology, DTI metrics demonstrate that reduced brain network transmission efficiency in chronic sleep disruption associated with small vessel disease is linked to microstructural damage in frontal, thalamic, cingulum, and cuneal white matter tracts. DTI parameters and network topology features thus hold significant promise as non-invasive neuroimaging biomarkers in this population [20].

Diffusion kurtosis imaging (DKI) extends the classical DTI framework by incorporating the dimensionless metric kurtosis to quantify the degree of non-Gaussian water diffusion within a voxel [21]. DKI provides enhanced sensitivity over DTI for mapping sleep-related brain changes, revealing more extensive signal alterations on statistical maps in healthy adults during induced sleep (post-sleep deprivation and zolpidem 10 mg). High-order diffusion analysis confirmed that sleep-induced expansions of the extracellular space lead to measurable drops in diffusion kurtosis, predominantly in slow-wave generating regions [22].

Applying advanced diffusion frameworks, such as DTI and DKI, enables characterization of the microstructural substrates driving observed white matter shifts. While time-of-day effects were evident within both intra- and extra-axonal compartments, the extra-axonal microenvironment demonstrated heightened vulnerability to acute SD [23].

The glymphatic system drives waste clearance in the brain by moving cerebrospinal fluid along perivascular pathways. This process removes soluble proteins, such as amyloid-beta, and metabolic waste while distributing glucose, lipids, amino acids, and neuromodulators [24]. Disruptions in glymphatic transport driven by acute sleep debt may impair parenchymal solute clearance, potentially predisposing the central nervous system to microstructural vulnerability and cognitive decline [16,25]. Using diffusion MRI, the non-invasive diffusion tensor image analysis along the perivascular space (DTI-ALPS) technique proposed by Taoka et al. [26] provides an indirect, diffusivity-based proxy for perivascular fluid transport potentially related to glymphatic function via the analysis along the perivascular space (ALPS) index, bypassing the need for intrathecal contrast agents. Recent literature reveals that the

ALPS index is significantly lower in individuals experiencing chronic sleep disruption or acute SD than in healthy controls. Within sleep-disrupted groups, this imaging biomarker shows a strong negative correlation with objective and subjective metrics, including total Pittsburgh Sleep Quality Index scores, sleep latency, and sleep medication use [27].

Functional network biomarkers

Resting-state fMRI (rs-fMRI) captures intrinsic, spontaneous blood-oxygen-level-dependent (BOLD) signal fluctuations in the brain without task intervention and reveals that acute SD alters intrinsic baseline activity within the thalamus, default mode network [28], and frontal-parietal attention network [29]. The amplitude of low-frequency fluctuations (ALFF) drops in frontoparietal and default-mode hubs but rises in the thalamus, visual, and motor cortices, suggesting a compensatory response to combat sleepiness. Essentially, baseline fluctuations in the dorsolateral PFC and thalamus predict vulnerability to attention deficits. In contrast, weakened connectivity between the frontal cortex and primary sensory inputs (the thalamus and visual cortex) directly tracks with declines in psychomotor vigilance task performance [30].

Comparing 24-hour SD to rested wakefulness in 22 female subjects, rs-fMRI revealed regional changes in spontaneous BOLD signal oscillations. Specifically, the acute SD state was associated with lower ALFF values in the right anterior cerebellum, paired with elevated ALFF signals across the bilateral inferior occipital gyrus, left thalamus, left insula, and bilateral postcentral gyrus during attentional testing [31]. Moreover, following 36 hours of total SD, rs-fMRI demonstrated that modafinil and caffeine administration modulated time-domain correlations between specific thalamic subregions and the frontal and temporal lobes compared to placebo [32]. These findings highlight the importance of rs-fMRI as a valuable tool for mapping activity in brain regions linked to SD and their responses to interventions.

Working memory is a foundational cognitive process mediated by a distributed network including the parietal lobe and cingulate cortex. Acute SD markedly reduces working memory accuracy, increases omission rates in task-based fMRI [33], and reveals convergent functional hypoactivation predominantly localized to the right intraparietal sulcus and superior parietal lobule [34]. An emotional task-based fMRI study demonstrated age-specific reorganizations of local network efficiency following SD. Younger adults showed pronounced increases in local efficiency within prefrontal nodes of the emotion regulation network, whereas older adults displayed preferential increases in lower-order cortical and subcortical regions [35].

Molecular, metabolic, & physiological modalities

PET combined with either computed tomography or MRI provides direct in vivo quantification of cerebral metabolic and molecular processes, making it highly suitable for capturing the neurochemical sequelae of sleep disruption [36]. Early work by Wu et al. using 18F-fluorodeoxyglucose demonstrated that approximately 32 hours of total SD resulted in significant reductions in absolute glucose metabolism across the frontal and temporal lobes, thalamus, basal ganglia, and cerebellum. Subsequent analysis revealed that recovery sleep resulted in only partial metabolic restoration: frontal lobe hypometabolism persisted after a full night of recovery, while subcortical deficits showed minimal reversal [37].

As a comparative counterpart to acute 24–36-hour SD protocols, a recent study using 8-cyclopentyl-3-(3-18F-fluoropropyl)-1-propylxanthine PET-MRI data indicates that chronic sleep restriction (5 days) induces reversible structural

plasticity in frontal, temporo-occipital, and thalamic gray matter. However, this plasticity response was mitigated or reversed by concurrent caffeine intake. Furthermore, individual baseline subcortical availability of A1 adenosine receptors may modulate this caffeine-associated grey matter impact [38], confirming that the neurobiological effects of sleep debt are highly sensitive to confounding pharmacological factors.

MRS quantifies cellular metabolites to assess tissue metabolic status and monitor neurochemical shifts across disease states. In sleep science, it provides essential diagnostic utility by mapping cerebral metabolic changes that reflect insults to brain integrity, specifically capturing markers of axonal viability, energy metabolism, and cell membrane stability [39]. Serving as a clinical baseline comparison, MRS findings in patients with chronic insomnia do not demonstrate altered gamma-aminobutyric acid+ (GABA+) levels as a core pathological feature. However, exploratory analyses indicate that GABA+ concentrations correlate positively with habitual sleep duration, while glutamate/glutamine levels may track rising diurnal arousal in this population [40].

In recent years, fNIRS has emerged as a promising tool for sleep research [41] due to its significant utility for probing neural correlates of operational performance during state-dependent challenges such as fatigue and sleep debt [42]. Acute 24-hour SD significantly degrades sustained attention and reaction speed while sparing cognitive performance in working memory, visual memory, and short-term visual matching. Neuroimaging, including transcranial Doppler and fNIRS, revealed altered cerebral blood flow and attenuated neurovascular coupling responses within the PFC, highlighting a primary hemodynamic mechanism underlying these selective cognitive impairments [43].

Multimodal synthesis & acquisition challenges

Simultaneous EEG-fMRI bridges electrophysiological and hemodynamic measures of neural activity. This hybrid setup overcomes the low temporal sampling of fMRI (seconds) and the limited spatial localization of EEG (milliseconds), leveraging the complementary strengths of both modalities to enable high-precision multimodal brain mapping [44]. Moreover, combining EEG and oculomotor features within an attention-optimized convolutional neural network accurately classifies distinct forms of mental fatigue. Key findings delineate task-induced cognitive exhaustion from sleep loss and reveal strong neurophysiological and subjective convergence between 24-hour and 36-hour SD states [45].

Cross-study comparisons and replication in neuroimaging are currently hindered by the absence of standardized preprocessing and analytical pipelines [46]. Discrepancies in hardware, acquisition settings, software platforms, and spatial approaches (whole-brain vs. region-of-interest) introduce significant analytical noise. As a result, applying different processing pipelines to identical MRI datasets can yield conflicting results that stem from analytical strategy rather than underlying neurophysiology [47]. For example, ultra-high-field 7T MRI promises greater sensitivity in detecting subtle structural, perivascular, and network alterations caused by a single night of sleep restriction in both healthy young adults [12] and adolescents [48].

Beyond hardware and analytical software discrepancies, demographic heterogeneity serves as a significant, underreported source of cross-study variability in sleep neuroimaging [46]. Age markedly modulates structural and functional vulnerability

to sleep loss: younger adults demonstrate localized prefrontal network reorganization to preserve executive performance, whereas older adults exhibit preferential efficiency shifts across lower-order subcortical and cortical hubs [14,35]. Furthermore, sex-specific differences in baseline resting-state BOLD oscillations and stress responsiveness, highlighted by studies in female-only cohorts [31], underscore that demographic variance can obscure microstructural and network-level signals if not systematically controlled or disaggregated in sample designs.

Future horizons: AI, radiomics, and clinical translation

Radiomics converts standard medical images into high-dimensional quantitative data by extracting and modeling textural and spatial features, thereby connecting radiological phenotyping directly to clinical and biological targets [49]. In particular, it can utilize structural and functional feature extraction to build predictive models for objective sleep debt or early cognitive decline [50,51]. In exploratory neuroimaging workflows, radiomics derived from modern deep learning-based segmentation methods have shown promising discriminatory performance in distinguishing sleep-deprived subjects from non-deprived controls, though Poirot et al. [52] demonstrated that subtle segmentation variations significantly affect feature stability and classification performance (77-84%), highlighting the need for rigorous standardization.

In routine clinical practice, neuroimaging evaluations of individuals presenting with sleep disruptions are frequently qualitative, often noting non-specific white matter hyperintensities, markers strongly linked to chronic unhealthy sleep behaviors and microstructural injury in large cohort studies [53], rather than extracting dynamic functional or glymphatic biomarkers. Transitioning toward quantitative sleep debt profiling requires integrating automated deep-learning segmentation into standardized radiological reporting [54]. By incorporating standardized quantitative metrics for perivascular space volume fraction, glymphatic ALPS index, and frontoparietal functional network efficiency, benchmarked against population-specific normative databases to eliminate demographic and morphological bias, modern neuroimaging suites may support the future development of candidate quantitative imaging biomarkers to evaluate sleep-related brain health.

Conclusion

Evidence from structural, diffusion, functional, metabolic, and optical neuroimaging demonstrates that even acute SD produces measurable changes in the brain, particularly in the prefrontal cortex, thalamus, white matter, and glymphatic systems. Although these alterations are often subtle and dynamic, they consistently correlate with impaired attention, executive function, and neurovascular regulation. Methodological limitations of this narrative review include the non-systematic

literature selection, absence of formal risk-of-bias/quality appraisal, and restriction to English-language publications, which may introduce selection bias. Nevertheless, continued progress in the field will require standardized acquisition and analysis pipelines, while radiomics and deep learning present promising approaches for the future quantitative assessment of sleep debt, pending rigorous validation prior to routine clinical implementation.

Abbreviations

SD – Sleep deprivation
 PSG – Polysomnography
 EEG – Electroencephalography
 MRI – Magnetic resonance imaging
 PET – Positron emission tomography
 fNIRS – Functional near-infrared spectroscopy
 fMRI – Functional magnetic resonance imaging
 MRS – Magnetic resonance spectroscopy
 PFC – Prefrontal cortex
 DTI – Diffusion tensor imaging
 DKI – Diffusion kurtosis imaging
 DTI-ALPS – Diffusion tensor image analysis along the perivascular space
 ALPS – Analysis along the perivascular space
 rs-fMRI – Resting-state functional magnetic resonance imaging
 BOLD – Blood-oxygen-level-dependent
 ALFF – Amplitude of low-frequency fluctuations
 GABA+ – Gamma-aminobutyric acid+

Author Contributions: Conceptualization, N. Z. and S. B.; methodology, N. Z. and A. B.; validation, J. A. and S. B.; formal analysis – not applicable; investigation – not applicable; resources, S. B.; data curation – not applicable; writing – original draft preparation, N. Z., E. A. and A. B.; writing – review and editing, N. Z. and A. B.; visualization, N. Z.; supervision, S. B.; project administration, A. B.; funding acquisition, S. B. All authors have read and agreed to the published version of the manuscript.

Disclosures: The authors have no conflicts of interest.

Acknowledgments: None.

Funding: This study was funded by a research grant of the Ministry of Science and Higher Education of the Republic of Kazakhstan (No. AP26199361), “Investigating the Relationship Between Sleep Quality and Brain Imaging Biomarkers”.

Data availability statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

Artificial Intelligence (AI) Disclosure Statement: The authors declare no AI Tools used for preparation of this work.

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