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Bridging the Gap between Laboratory and Real-World Sleep Assessment: The Emerging Role of EEG-Derived Biomarkers in Clinical and Translational Sleep Neuroscience

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ABSTRACT

Assessment of sleep quality in clinical practice remains constrained by tools that are either subjective or impractical for routine use. Self-report instruments, such as the Pittsburgh Sleep Quality Index (PSQI) and sleep diaries, are vulnerable to recall bias and demonstrate limited correspondence with objective neurophysiological measures. Polysomnography (PSG), while the gold standard, is costly, labor-intensive, and disruptive to natural sleep, rendering it unsuitable for longitudinal or outpatient monitoring. Recent advances in quantitative electroencephalography (qEEG), event-related potential (ERP) analysis, and source localization have created opportunities to bridge this gap. These methods enable scalable, objective evaluation of neurophysiological processes underpinning sleep architecture, cognitive function, and emotional regulation. This review synthesizes emerging evidence that EEG-derived biomarkers (e.g., spectral power ratios (delta/alpha, theta/beta), cross-frequency coupling, and ERP dynamics) can serve as sensitive indices of sleep quality and neural health. We highlight the BrainView platform as an exemplar of this translational shift. BrainView is an FDA-cleared system combining qEEG, ERP, source localization, and normative comparison to quantify brain function non-invasively and longitudinally. By integrating neurophysiological data with actigraphy, self-report, and behavioral outcomes, EEG-based platforms such as BrainView may redefine how clinicians and researchers monitor sleep, treatment response, and neurocognitive resilience.

Keywords

Sleep quality, Quantitative EEG (qEEG), Event-related potentials (ERP), Source localization, EEG biomarkers.

Sleep is a fundamental neurobiological process essential for metabolic homeostasis, cardiovascular integrity, cognitive performance, and emotional regulation. A rapidly expanding evidence base now demonstrates that inadequate or disrupted sleep contributes to metabolic dysfunction, cardiovascular disease, neuropsychiatric illness, and premature mortality.

Epidemiologic and clinical research implicate poor sleep quality in insulin resistance, dyslipidemia, elevated inflammatory markers, hypertension, and increased risk of coronary events, stroke, and mortality [1-3]. The mechanistic underpinnings of these associations span multiple biological systems, from altered autonomic tone and oxidative stress to impaired glymphatic clearance and aberrant synaptic homeostasis.

Despite this evidence, routine clinical evaluation of sleep remains largely subjective. Instruments such as the Pittsburgh Sleep Quality Index (PSQI) and the Insomnia Severity Index (ISI) are inexpensive and easy to administer but are limited by recall bias, mood-dependent distortion, and cultural variability [4]. These tools correlate only weakly to moderately with objective physiological measures (e.g., including electroencephalography (EEG), actigraphy, and polysomnography (PSG)), thereby constraining their diagnostic and prognostic value [5].

PSG remains the canonical gold standard for diagnosing

sleep disorders and characterizing sleep architecture based on EEG, electro-oculography (EOG), electromyography (EMG), respiratory, and cardiac signals. While PSG provides unparalleled neurophysiological detail, it is resource- intensive, intrusive, and poorly scalable. The requirement for a controlled laboratory environment, specialized technologists, and overnight monitoring precludes its use in longitudinal or outpatient application [6]. Compounding these limitations is the first-night effect, a robust and reproducible alteration in sleep architecture during initial laboratory recording characterized by increased sleep latency, greater wake after sleep onset, reduced REM proportion, and diminished total sleep time [7-9]. This phenomenon can substantially distort the measurement of habitual sleep quality, reducing ecological validity and limiting PSG's translational utility in both research and practice.

As a result, clinicians and investigators face a persistent diagnostic and methodological chasm: subjective assessments offer convenience but lack physiological specificity, whereas objective methods provide mechanistic insight but remain impractical for large-scale or repeated use.

Addressing this gap necessitates innovations capable of capturing neurophysiological signatures of sleep repeatedly, unobtrusively, and cost-effectively, tools that can inform both individualized patient care and mechanistic research into the neural consequences of sleep disruption.

Electroencephalography (EEG) offers a uniquely powerful means of achieving this goal. EEG provides a direct, millisecond-resolution window into cortical oscillatory dynamics, enabling quantification of the electrophysiological states that define sleep architecture and its deviations. Sleep involves a coordinated reorganization of neural oscillations across frequency bands that reflect synaptic homeostasis, thalamocortical gating, and cortical plasticity. Traditional PSG channels capture these dynamics but vastly underutilize their analytical potential, often reducing rich, multidimensional data to categorical stage scoring.

Recent advances in quantitative EEG (qEEG) have enabled extraction of metrics such as absolute and relative spectral power within delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), and beta (12–25 Hz) bands, as well as inter-band ratios (e.g., delta/alpha, theta/beta) that correspond to sleep depth, continuity, and restorative potential. Elevated theta/beta ratios are increasingly recognized as markers of cortical hyperarousal and insomnia-related instability, whereas higher delta/alpha ratios signify enhanced slow-wave recovery and synaptic renormalization [9,10]. Moreover, EEG-derived network indices, including phase–amplitude coupling, coherence, and graph-theoretic connectivity metrics, are emerging as sensitive markers of sleep-dependent neuroplasticity and cognitive performance [11,12]. These neurophysiological biomarkers provide a mechanistic substrate for understanding how behavioral and environmental factors, collectively termed sleep hygiene, modulate cortical function and long-term brain health [10,11]. However, despite their scientific maturity, quantitative EEG methods remain underutilized in routine clinical settings due to the lack of accessible, validated, and scalable platforms. The objective of this paper is to critically review the scientific rationale and translational potential of EEG-based assessment of sleep hygiene, high-resolution neurophysiological monitoring can transform both clinical sleep medicine and research on brain health.

Poor Sleep Hygiene, Brain Health, and the Case for EEG-Based Monitoring

Poor sleep hygiene (i.e., irregular sleep timing, inadequate duration, frequent nocturnal awakenings, sleep fragmentation, and suboptimal sleep environment), has increasingly been implicated not merely in symptomatic complaints (e.g. fatigue, mood disturbance), but in longitudinal neurological and neurodegenerative outcomes. Chronic exposure to maladaptive sleep behaviors appears to contribute to a cascade of deleterious processes in the brain: impaired synaptic homeostasis, diminished glymphatic clearance, microstructural white matter injury, and accelerated neurodegeneration. This growing evidence underscores the urgency for objective, neurophysiological monitoring of sleep quality beyond self-report.

In a large neuroimaging subsample of ~40,000 middle-aged participants from the UK Biobank, suboptimal sleep durations (<7 h or ≥9 h) were associated with increased white matter hyperintensity volumes and lower fractional anisotropy, markers linked to small vessel disease and microstructural brain injury [12]. The association persisted after adjusting for vascular risk factors, pointing to sleep's role in subclinical brain injury [12]. Chronic poor sleep may thus contribute to silent structural damage that precedes overt clinical phenotypes (e.g. stroke, dementia) [13]. In a recent retrospective cohort leveraging EEG-based risk prediction models, patterns of brain activity during sleep predicted future incidence of neurologic (e.g. ischemic stroke, dementia, mild cognitive impairment) and psychiatric outcomes over 10 years [14].

Participants categorized in the "poor sleep EEG–predicted risk" quartile exhibited significantly elevated hazard ratios for neurologic and psychiatric conditions compared to "average" or "good" sleepers, even in external validation cohorts [15]. This suggests that microstructural EEG features may capture latent brain vulnerability that traditional macro sleep metrics or self- reported hygiene fail to detect. Moreover, emergent reviews argue that sleep electrophysiology serves as a window into brain health, enabling structural-EEG associations relevant to disease processes [16]. For example, in Alzheimer's disease, reductions in slow oscillation amplitude, spindle density, and coherence often precede overt atrophy [17]; in Parkinson's or Lewy-body disease, early REM sleep and sleep fragmentation abnormalities reflect degeneration in brainstem circuits that later propagate to cortical networks [18]. Thus, sleep microstructure offers mechanistic insight into disease origin and trajectory, far beyond coarse sleep architecture.

Mechanisms: From Sleep Disruption to Neural Dysfunction

Several pathophysiological routes link poor sleep hygiene to deteriorating brain health. For example, synaptic homeostasis disruption has been linked to excitatory/inhibitory imbalance; inadequate slow-wave sleep impairs downscaling of synaptic weights, fostering neural network saturation, inefficiency, and excitotoxic stress [19]. Sleep is also critical for interstitial fluid flow and clearance of metabolic byproducts (e.g. amyloid-β, tau). Sleep fragmentation or suppression of slow oscillations likely impedes this clearance, increasing accumulation of neurotoxic proteins implicated in Alzheimer's and other proteinopathies [20]. Moreover, suppression of brain metabolite clearance and impaired cognition is induced, even in the absence of disease. In addition, disrupted sleep exacerbates blood–brain barrier permeability, microglial activation, endothelial dysfunction, and small-vessel ischemic injury. These processes underlie white matter lesions, microinfarcts, and subcortical injury observed in sleep-deprived populations [21]. Further, repeated wake–sleep transitions, arousal bursts, and reduced restorative slow-wave amplitude elevate mitochondrial stress and reactive oxygen species generation, compromising neuronal energetics [22].

Importantly, these processes are not uniform. Rather, the process from sleep disruption to neural dysfunction evolve gradually and may initially manifest at the level of oscillatory microstructure (e.g. diminished spindles, reduced slow oscillations, altered aperiodic EEG slope or fractal exponent) long before macro-architectural changes (e.g. reduced total sleep time or sleep efficiency) are easily measurable [23]. In psychiatric illness, sleep disturbances have shifted from being considered secondary symptoms to core mechanistic features [24]. Recent narrative reviews highlight consistently altered EEG phenotypes across mood, anxiety, psychosis, and neurodevelopmental disorders, including reduced spindle density, diminished slow-wave activity, increased high-frequency spectral tails, and changes in the aperiodic (1/f) component of the EEG [25]. These phenotypes map onto disrupted thalamocortical, frontolimbic, and default- mode circuits implicated in psychiatric pathophysiology, and may serve as biomarkers of diagnosis, prognosis, and treatment response [25]. In the neurodegenerative domain, sleep EEG microstructure changes often precede overt cortical degeneration [26]. For instance, diminished slow oscillations and spindle coupling appear in Alzheimer's disease decades before diagnosis, while early REM-sleep behavior alterations and micro-arousals may mark synucleinopathies [27]. Given this, sleep hygiene disruption that erodes microstructure could accelerate disease trajectories or unmask latent vulnerability.

The mechanistic sensitivity of sleep microstructure, subjective or actigraphy-based measures may be insufficient to capture the earliest and most clinically relevant changes. Poor hygiene (e.g. delayed sleep timing, irregularity, insufficient duration) may manifest as subtle shifts in spectral ratios, spindle-slow oscillation coupling, or transient micro-arousals long before traditional metrics degrade. Without neurophysiological readouts, clinicians and researchers may lack the granularity to detect such early deviations, stratify risk, or tailor interventions.

EEG-based monitoring, particularly longitudinal and ambulatory modalities, allows tracking of neural signatures of sleep quality across nights and weeks. For example, declining slow oscillation amplitude, increasing high-frequency (beta/gamma) spectral power, or rising aperiodic exponent may signal accumulating neural stress, even when patient-reported sleep quality remains stable. Detection of such microstructural drift could permit just-in-time behavioral corrections, neuromodulation, or pharmacologic adjustments before irreversible injury accrues. In summary, poor sleep hygiene is not merely symptomatic; it can drive substantive and cumulative neurological harm via mechanisms that first manifest at the level of EEG microstructure. Only by capturing neural dynamics of sleep can we fully evaluate, intervene upon, and mitigate the brain's hidden cost of suboptimal sleep.

EEG-Derived Sleep Biomarkers: Evidence and Applications

A growing body of research supports spectral biomarkers as quantitative correlates of sleep quality. Automated classifiers based on EEG power spectra now achieve > 90% accuracy for stage classification compared with manual PSG scoring [28,29]. Meta-analyses confirm that insomnia is characterized by elevated beta/gamma power and reduced delta activity, reflecting cortical hyperarousal [30]. Further, Sun et al. [31]. examined data from 8,673 patients at Massachusetts General Hospital using PSG and electronic health records. They found that poor sleepers, identified by EEG-based risk scores, had roughly 3.5–3.8 times higher 10-year risks of major neurological, psychiatric, cardiovascular diseases, and mortality compared to better sleepers.

Kristjánsson et al. [32,33] showed that integrating deep learning with survival models enhances the ability to identify key predictors of outcomes. Unlike traditional survival models relying on hand-crafted sleep features, this data-driven approach leverages raw polysomnographic signals, representing a spectrum from expert-defined variables to fully automated machine learning methods. Beyond staging, spectral ratios predict treatment response: reductions in theta/beta and increases in delta/alpha following behavioral sleep interventions correspond with improvements in PSQI and actigraphy metrics. ERPs provide a complementary measure of neural information processing during sleep. The auditory P3 component, reflecting attentional resource allocation, remains detectable during lighter NREM stages and correlates with daytime cognitive recovery. Post-intervention increases in P3 amplitude have been observed alongside spectral normalization, suggesting shared mechanisms of cortical restoration[32]. Together, these findings underscore that spectral and electrophysiological biomarkers not only quantify sleep quality with high precision but also offer powerful, mechanistically informed tools for predicting health outcomes, guiding treatment response, and advancing personalized sleep medicine.

Advances in machine learning have enabled derivation of EEG-based brain age indices, which estimate neural aging trajectories from overnight recordings. Deviations between EEG-based and

chronological age predict cognitive decline and mortality [34]. Night-to-night variability in these metrics provides insight into neural resilience and vulnerability, offering a potential biomarker for early neurodegeneration. Recent transformer-based models integrate EEG features with actigraphy and heart-rate variability to achieve interpretable, uncertainty-quantified predictions of sleep state [35,36]. These methods underscore a paradigm shift from categorical staging to continuous, probabilistic modeling of brain states, aligning with precision-medicine approaches.

Bridging Research and Practice: The BrainView Paradigm

The BrainView platform exemplifies the emerging class of next-generation EEG-based systems designed to bridge the gap between laboratory PSG and real-world, longitudinal assessment of sleep and brain function. The platform integrates FDA-cleared quantitative EEG (qEEG), event- ERP analytics, source localization, and a large-scale normative database comparison within a single, outpatient-ready framework. Unlike conventional PSG, with accuracy is offset by cost, logistical constraints, and limited feasibility for repeated monitoring, BrainView enables noninvasive, repeatable neurophysiological assessments in naturalistic environments, providing clinicians and researchers with scalable access to high-fidelity brain-sleep metrics. At its analytic core, BrainView implements a multimodal qEEG pipeline that decomposes electrophysiological signals into frequency-domain biomarkers (e.g., delta/alpha, theta/beta ratios) and time-domain ERP indices (e.g., P300, N100, N400 latency and amplitude). These biomarkers are established correlates of cortical arousal, attention, and neuroplasticity [37-39]. Elevated beta and gamma power, recognized hallmarks of hyperarousal, are characteristic of insomnia and anxiety-spectrum disorders [30], whereas normalization of delta–alpha balance corresponds to behavioral and cognitive improvement following sleep hygiene and cognitive-behavioral interventions [33,40]. By automatically extracting and quantifying these features, BrainView allows clinicians to objectively monitor treatment response, stratify risk, and track neurophysiological recovery in disorders where cortical excitability and disrupted sleep architecture are mechanistically implicated, including insomnia, depression, and neurodegenerative diseases.

Recent clinical applications illustrate this translational potential. In neuropsychiatric research, BrainView's ERP analytic architecture has demonstrated diagnostic discrimination in large patient cohorts. In a study of 335 participants (85 Narcolepsy, 250 controls), BrainView-derived ERPs achieved sensitivity = 0.85, specificity = 0.820, with reductions in P300 amplitude and delays in N400 and N100 components consistent with established electrophysiological biomarkers of cortical dysfunction [41-43]. Complementary findings have been reported in cognitive aging and fatigue, where BrainView's normative database and ERP suite provided quantitative indices of cognitive slowing and cortical hypoactivation in outpatient settings [41-43]. The same analytic principles are now being extended to sleep applications, where qEEG-ERP integration can detect persistent cortical hyperarousal in insomnia or evaluate sedative pharmacodynamics through real-time electrophysiological feedback.

From a systems neuroscience perspective, BrainView aligns with the broader trend toward scalable EEG infrastructures in real-world medicine. Similar architectural approaches have been implemented in timely status-epilepticus management, demonstrating that portable EEG ecosystems can translate laboratory-grade signal fidelity into emergency and ambulatory contexts [44]. Within sleep medicine, BrainView's continuous monitoring capabilities advance a precision-sleep framework that integrates neurophysiological data with subjective sleep diaries, actigraphy, and cognitive outcomes. This multimodal synthesis supports mechanistic investigation into how sleep regularity and circadian stability, now recognized as stronger predictors of cardiometabolic and neuropsychiatric outcomes than duration alone [1], shape cortical function and neuroplasticity. By enabling nightly EEG biomarker quantification in home environments, BrainView facilitates longitudinal mapping of brain-sleep interactions across health and disease trajectories.

Despite its promise, peer-reviewed validation of BrainView specifically in sleep research remains limited. To date, most available data derive from technical white papers, clinical case reports, or performance abstracts rather than large-scale empirical trials [45]. This evidence gap underscores an urgent research opportunity: rigorous, multisite validation studies comparing BrainView-derived biomarkers against PSG standards, cognitive outcomes, and clinical endpoints. Such studies will be essential to establish test–retest reliability, cross-hardware reproducibility, and predictive validity. If confirmed, BrainView and analogous platforms could transform sleep assessment from subjective self-report to continuous, objective, and neurophysiologically grounded monitoring, ushering in a new era of precision sleep medicine that integrates scalable EEG analytics into routine clinical and translational neuroscience practice.

Current Limitations and Methodological Challenges

Despite their promise, EEG-based sleep biomarkers face several critical challenges that must be addressed before widespread clinical adoption can be realized. Foremost among these is the need for standardization and reproducibility: differences in electrode montage, reference schemes, preprocessing algorithms, and artifact rejection methods continue to hinder cross-study comparability and meta-analytic synthesis. Establishing consensus pipelines and open benchmarking frameworks is essential to ensure methodological consistency and scientific rigor. Equally important is the development of large, demographically diverse normative EEG databases to enable accurate z-score normalization and contextual interpretation across age, sex, and clinical populations. Technical limitations also persist; artifact contamination from movement, muscle activity, and environmental noise remains a key obstacle to reliable home- based recordings, underscoring the need for more sophisticated, automated quality-control algorithms and adaptive filtering techniques. In the analytic domain, while deep learning and transformer-based models have dramatically improved sleep staging accuracy, their "black-box" nature poses

challenges for interpretability, clinical trust, and regulatory validation, necessitating the design of transparent, explainable frameworks that balance performance with accountability. Finally, the translational validation gap remains substantial: large, prospective, longitudinal studies are urgently needed to confirm that EEG-derived biomarkers not only correlate with PSG metrics but also predict clinically meaningful outcomes, such as treatment response, cognitive performance, and neurodegenerative risk. Addressing these barriers will be pivotal to transforming EEG biomarkers from promising research tools into reliable, regulatory-grade instruments for precision sleep medicine and neuroscience.

Toward Precision Sleep Medicine

EEG biomarkers offer the possibility of personalized sleep profiling, allowing clinicians to tailor interventions to an individual's neurophysiological phenotype. For example, elevated beta power during NREM could signal cortical hyperarousal responsive to cognitive behavioral therapy for insomnia (CBT-I), whereas reduced slow-wave power may warrant physical-activity or nutritional interventions. Integration of EEG biomarkers with digital health ecosystems (wearables, telemedicine, AI dashboards) could enable population-level screening for sleep-related neurocognitive risk [46]. Continuous EEG monitoring may detect early deviations in sleep physiology preceding the onset of depression, Alzheimer's disease, or other neuropsychiatric conditions. As EEG monitoring becomes ubiquitous, data privacy, algorithmic transparency, and interpretive accuracy must remain priorities. Regulatory frameworks must ensure that automated EEG analytics meet the standards of medical-grade evidence and clinical accountability.

Future Directions and Conclusions

Looking ahead, the field of sleep neuroscience is poised for a major transformation as quantitative EEG technologies converge with advanced analytics and scalable deployment platforms. The next frontier will require large-scale, multisite validation studies that test EEG- derived biomarkers against heterogeneous polysomnography datasets, diverse populations, and longitudinal clinical outcomes. Such efforts will establish the reliability, generalizability, and prognostic value of neurophysiological sleep metrics across age, sex, and disease states. Parallel advances in integrative analytics, linking EEG with heart-rate variability, actigraphy, and other wearable physiological sensors, will enable a multidimensional understanding of sleep as a whole-system biological process rather than an isolated neural phenomenon.

Emerging paradigms in closed-loop neuromodulation further extend this potential, using real- time EEG feedback to modulate slow-wave activity, enhance memory consolidation, and promote restorative sleep. Meanwhile, longitudinal cohort studies leveraging EEG-derived markers of cortical dynamics will illuminate how sleep physiology shapes neuroplasticity, cognitive aging, and emotional resilience across the lifespan. To sustain transparency and accelerate innovation, the field must also prioritize open normative repositories. This necessitates curated, demographically representative EEG sleep databases that allow algorithm benchmarking, reproducibility testing, and regulatory oversight.

Collectively, these directions underscore a pivotal shift from descriptive to precision sleep medicine, in which EEG biomarkers become actionable clinical tools. The integration of quantitative EEG, advanced computational modeling, and scalable technology is transforming sleep assessment from subjective symptomatology to objective neurophysiology. By enabling clinicians and researchers to monitor brain function longitudinally, detect maladaptive patterns early, and tailor interventions based on neural physiology rather than self-report, EEG-based biomarkers are redefining how sleep is measured and managed. Platforms such as BrainView exemplify this transition, bringing the rigor of laboratory polysomnography into real-world environments. As validation expands and standards mature, EEG-derived biomarkers will become foundational to both clinical care and neuroscience research, bridging the long-standing divide between how we experience sleep and how the brain performs it.

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