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Objective Neurophysiological Stratification in ADHD: Integrating Quantitative EEG and ERP Biomarkers

Krista Casazza PhD[1], Slav Danev[2] and Jonathan RT Lakey PhD, MSM[1]*

[1]*Department of Surgery and Biomedical Engineering, University of California Irvine, California, USA.*

[2]*Medeia Inc, Santa Barbara, California, USA.*

***Correspondence:**
Jonathan RT Lakey, PhD, MSM., Professor Emeritus, Department of Surgery and Biomedical Engineering, 333 City Blvd West, Suite 1600, Orange, California, USA, Phone: 1-949-824-8022/ 714 851 8856.

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ABSTRACT

Attention-Deficit/Hyperactivity Disorder (ADHD) is a heterogeneous neurodevelopmental condition with substantial interindividual variability in symptom expression, cognitive control, and neurophysiological signatures. Despite decades of evidence implicating EEG-derived markers—such as altered theta, alpha, and beta power and event-related potentials (ERPs) including N2 and P3 components—routine clinical translation has remained limited. Variability in methods, populations, and data interpretation has hindered standardization and reproducibility. Converging meta-analytic findings, however, indicate that EEG-based neurofeedback offers additive short-term benefits when combined with medication, particularly for parent-rated global and inattention symptoms. Yet these effects tend to attenuate at six months, highlighting the need for objective, standardized monitoring frameworks. The BrainView platform represents a pragmatic, FDA-cleared ecosystem for embedding quantitative EEG (qEEG) and ERP measures into ADHD assessment, treatment monitoring, and research. Here, we outline how BrainView enables neurophysiology-informed phenotyping, individualized treatment optimization, and objective tracking of neural change. By integrating standardized spectral and ERP metrics with clinical outcomes, BrainView bridges the translational gap between laboratory EEG biomarkers and actionable clinical decision-making, supporting reproducibility, harmonization, and meta-science in ADHD neurophysiology.

Keywords

Attention-Deficit/Hyperactivity Disorder (ADHD), Neurodevelopmental disorder, Cognitive control, Symptom variability, Clinical translation, Neurophysiological assessment

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a common, heterogeneous neurodevelopmental disorder characterized by pervasive difficulties in attention regulation, inhibitory control, and hyperactivity–impulsivity that emerge in childhood and often persist into adulthood. ADHD affects approximately 5–7 % of children and 2–5 % of adults worldwide, though prevalence estimates vary according to methodology and diagnostic criteria [1]. Recent epidemiologic data suggest prevalence of diagnosed ADHD among U.S. youth increased steadily between 2017 and 2022, underscoring growing recognition and service demand. Despite its high prevalence and functional impact, ADHD diagnosis remains largely behavioral, grounded in structured clinical interviews, DSM-5 criteria, and parent- or teacher-rated symptom scales [1-4]. These tools provide essential descriptive anchors but are subjective, prone to rater bias, and insensitive to the underlying neurobiological heterogeneity that differentiates clinical subtypes and comorbid profiles [1,5]. Moreover, diagnostic assessments are typically static, (i.e., performed at a single time point), offering limited capacity to monitor neural change or treatment response objectively [6,7]. In adults, underdiagnosis and phenotypic overlap with affective and cognitive disorders further complicate diagnostic accuracy [1].

Consequently, there is a pressing need for objective, scalable

biomarkers capable of indexing core neurocognitive processes (e.g., attention, inhibition, arousal) and supporting dimensional, neurobiologically informed stratification [8,9]. Among candidate tools, electroencephalography (EEG) has emerged as one of the most promising modalities for bridging research and clinical practice. EEG provides a non-invasive, temporally precise measure of brain activity that can capture rapid neural dynamics underlying attentional allocation, inhibitory control, and cortical arousal regulation. Resting-state quantitative EEG (qEEG) and task-evoked event-related potentials (ERPs) are particularly well suited to ADHD research, given their sensitivity to executive-attention circuits and catecholaminergic modulation [10].

Despite robust literature, EEG biomarkers have not yet achieved routine clinical implementation. Across decades of studies, characteristic EEG abnormalities have been consistently observed in ADHD. The principal barriers include methodological heterogeneity (e.g., differences in recording hardware, reference montages, preprocessing algorithms, and frequency band definitions), small and demographically narrow samples, and inconsistent statistical normalization [11,12]. Moreover, the absence of FDA-cleared normative databases and standardized analytic pipelines has limited the reproducibility and interpretability of EEG findings across laboratories and clinical sites [13]. Resting-state analyses reveal elevated theta power, reduced beta power, and altered theta/beta ratios, patterns interpreted as markers of cortical hypoarousal and dysregulated top-down control [14,15]. Event-related potential studies, employing cognitive paradigms such as Go/No-Go and oddball tasks, demonstrate reduced P3 amplitudes (reflecting impaired attentional resource allocation) and delayed N2 latencies (reflecting deficient conflict monitoring and inhibitory control) [16,17]. These electrophysiological signatures align with the neurocognitive models of ADHD implicating frontostriatal, cingulo-opercular, and parietal control networks [18]. A recent meta-review from the European ADHD Guidelines Group emphasized that although EEG-based neurofeedback can provide additive short-term benefits when combined with pharmacotherapy its effects tend to be attenuated at six-month follow-up, highlighting the need for ongoing objective monitoring frameworks [15,19]. These findings reinforce the importance of standardized, longitudinal EEG platforms capable of capturing dynamic neural changes associated with treatment response, adherence, and relapse.

The objective of this review is to advance ADHD care by identifying a pathway for standardized qEEG and ERP biomarkers, into clinical and research workflows. Specifically, we seek to (1) establish a pragmatic neurophysiology-informed phenotyping framework within outpatient ADHD clinics; (2) demonstrate the potential impact of incorporating qEEG/ERP endpoints into prospective adjunctive-therapy protocols; and (3) highlight how the development of shareable, FDA-anchored analytic pipelines and reporting templates may be leveraged to enhance reproducibility and cross-site comparability and improve outcome measurement. This translational effort aims to bridge the gap between neurophysiological insight and individualized clinical care, accelerating the integration of EEG biomarkers into evidence-based ADHD management.

EEG Biomarkers in ADHD: Evidence and Limitations

Resting-state quantitative EEG (qEEG) provides a window into large-scale neural oscillatory dynamics that index cortical arousal, excitation–inhibition balance, and network coordination. Across multiple meta-analyses, ADHD has been consistently associated with elevated absolute and relative theta power (4–7 Hz) and reduced beta power (13–30 Hz), particularly over frontocentral regions [15,17,20]. The resulting increase in the theta/beta ratio (TBR) was historically viewed as a potential biomarker of cortical hypoarousal, reflecting delayed cortical maturation or underactivation of executive control networks [21]. However, more recent high-resolution EEG and source-space analyses have nuanced this interpretation. Studies using current-source density mapping and individualized frequency bands demonstrate that spectral alterations are not globally uniform but instead localize to specific functional networks (i.e., frontoparietal, dorsal attention, default mode networks), implicating dysregulated oscillatory coupling between task-positive and task-negative systems [22,23]. Connectivity-based qEEG studies show that children and adults with ADHD exhibit reduced beta and alpha coherence between frontoparietal nodes and abnormal theta-phase synchrony, correlating with attentional variability and executive dysfunction [24,25]. Importantly, the magnitude and directionality of spectral deviations vary across ADHD presentations. Individuals with the predominantly inattentive subtype often display greater theta excess and alpha slowing, whereas hyperactive–impulsive presentations exhibit elevated beta activity linked to heightened motor excitability [18,26]. Developmental stage also modulates EEG signatures, TBR abnormalities tend to normalize with age, suggesting state dependence rather than a fixed trait marker [27,28]. These findings underscore that EEG spectral features likely capture subtype- and symptom-specific neurophysiological states rather than a universal biomarker of ADHD [29,30].

Methodological variability further complicates interpretation. Differences in reference montages, sampling rates, artifact rejection, and spectral parameterization contribute to cross-study heterogeneity [18]. Moreover, many early TBR studies lacked age-matched controls or failed to account for confounding factors such as drowsiness, medication status, or comorbid anxiety, all of which substantially influence resting EEG power [31]. Consequently, although TBR remains a sensitive indicator of altered arousal regulation, its specificity and clinical utility as a diagnostic biomarker are limited under current evidence standards [32].

ERPs provide temporally precise markers of cognitive operations underlying attention allocation, error monitoring, and inhibitory control, domains central to ADHD pathophysiology. Across Go/No-Go, stop-signal, and oddball paradigms, attenuated P3 amplitudes and delayed N2 latencies are among the most robust findings in both pediatric and adult ADHD [33,34]. The N2 component (200–350 ms) reflects conflict monitoring and inhibitory control, primarily generated in the anterior cingulate and right inferior

frontal cortices, while the P3 component (300–500 ms) indexes attentional resource allocation and context updating within parietal–frontal networks [35]. Meta-analyses confirm medium-to-large effect sizes for reduced P3 amplitude in ADHD, correlating with inattention severity and omission error rates [36,37]. Recent high-density ERP studies have expanded this framework to include earlier components (e.g., diminished N1 and P2 amplitudes linked to sensory gating) and later components such as ERN/Pe reflecting performance monitoring deficits [38]. Moreover, ERP heterogeneity tracks ADHD subdimensions: inattentive symptoms are associated with blunted parietal P3, whereas hyperactive–impulsive traits show pronounced N2 deficits and altered frontal inhibition signatures [39]. Beyond amplitude and latency, single-trial variability in ERP waveforms has emerged as a sensitive index of attentional instability, offering a dynamic measure of neural noise [40]. Computational EEG approaches combining ERPs with time–frequency decomposition and machine learning classifiers have achieved moderate accuracy in distinguishing ADHD from controls and predicting treatment response, though generalizability remains limited without harmonized pipelines [41,42].

Despite substantial progress, EEG biomarkers remain under-translated into routine ADHD diagnosis or treatment stratification. The major barriers include: (1) a lack of standardized acquisition and preprocessing protocols, leading to inconsistent findings across laboratories; (2) an absence of large, demographically representative normative databases with rigorous age and sex correction; (3) insufficient integration into clinical decision-support frameworks linking neurophysiological markers to actionable outcomes; and (4) the limited availability of longitudinal validation, as most studies are cross-sectional [18,43].

To move from biomarker discovery to utility, the field requires standardized qEEG/ERP pipelines embedded in clinical and research infrastructures. This is precisely the gap addressed by the BrainView platform. BrainView's FDA-cleared analytics, normative z-score mapping, and automated ERP extraction offer a reproducible framework for longitudinal phenotyping and treatment monitoring [44]. By capturing both resting-state and task-evoked dynamics, BrainView enables characterization of state-dependent neurophysiological change across medication and neurofeedback interventions, potentially transforming EEG from an exploratory signal into a clinically actionable biomarker system [45].

The BrainView Platform: Standardization, Integration, and Clinical Application

The BrainView platform is an FDA-cleared qEEG and ERP system developed to overcome major methodological and translational barriers in EEG biomarker implementation. Designed as a portable, integrated hardware–software solution, BrainView combines high-fidelity EEG acquisition, automated signal processing, and normative database comparisons to generate objective neurophysiological profiles for both clinical and investigational use. Its architecture integrates validated algorithms for artifact reduction and spectral decomposition with FDA-anchored statistical mapping, enabling reproducible quantification of brain activity in real-world settings. These design principles (i.e., standardization, automation, and normative benchmarking) align with emerging best practices for reproducible biomarker translation in psychiatry and neurology.

BrainView supports both resting-state qEEG and task-based ERP analyses within a single, clinic-feasible session (~20 minutes). The qEEG module quantifies canonical spectral parameters (i.e., theta, alpha, and beta power and their ratios) normalized against age- and sex-corrected normative distributions to produce z-scores that characterize cortical arousal, excitation–inhibition balance, and attentional control. Complementary source-space modeling using low-resolution electromagnetic tomography (LORETA) infers regional activation within frontostriatal and parietal networks, core systems implicated in ADHD pathophysiology. The ERP module employs standardized auditory and visual oddball paradigms to elicit the N2 and P3 components, reflecting conflict monitoring and attentional allocation, respectively. Automated pipelines extract latency and amplitude metrics for these components, providing interpretable indices of inhibitory control and cognitive engagement relative to normative expectations. Together, these measures enable a multidimensional assessment of neural function across both resting and task-evoked domains, bringing laboratory-grade electrophysiology into outpatient clinical practice.

A defining feature of BrainView's translational capacity is its large-scale normative and discriminant database. As detailed in [44], the system incorporates over 28,000 EEG recordings, including nearly 8,000 healthy controls, to construct multivariate models generating discriminant z-scores and diagnostic likelihoods. Supplementary manufacturer documentation cites more than 200,000 EEG reports contributing to its machine-learning architecture, an unprecedented scale in commercial clinical EEG systems. This normative infrastructure allows probabilistic characterization of individual EEG profiles relative to population distributions, addressing a central limitation in biomarker reproducibility: the historical absence of harmonized, age-corrected reference frameworks.

BrainView's analytic suite enhances reliability through automated artifact rejection, spectral and ERP statistical mapping, and 2D/3D visualization of deviations from normative baselines. Standardized "brain maps" integrate neurophysiological and behavioral metrics, facilitating interpretation and reducing inter-operator variability. These features mirror design principles from scalable EEG applications in other clinical domains. For example, algorithmically guided EEG frameworks for status epilepticus and post-COVID-19 neurocognitive syndromes [46,47], which emphasize automation, quality control, and integration into care workflows. BrainView extends this scalable architecture to psychiatry and neurodevelopmental disorders, creating a foundation for reproducible, cross-site electrophysiological data capture.

Clinical Implementation in ADHD

BrainView enables objective, mechanism-informed phenotyping

at clinical intake. Deviations in theta/beta ratios or attenuated N2/P3 components identify specific deficits in arousal regulation or inhibitory control, domains critical to ADHD heterogeneity. When combined with behavioral assessments, these neural profiles refine subtype classification and guide personalized treatment strategies. For example, individuals exhibiting cortical hypoarousal (excess theta, low P3 amplitude) may benefit from stimulant therapy or arousal-targeted neurofeedback, whereas hyperactive beta profiles may respond better to cognitive-behavioral interventions.

Longitudinally, BrainView supports objective monitoring of treatment response across pharmacologic and behavioral interventions. Serial qEEG/ERP assessments quantify neural changes that mirror symptom improvement or reveal early drift. Meta-analyses indicate that EEG-based neurofeedback combined with medication produces short-term additive benefits for global and inattentive symptoms, yet effects often attenuate within six months. BrainView's z-score outputs allow detection of early neural regression (e.g., theta/beta ratios, P3 amplitude) informing booster neurofeedback sessions or medication recalibration. This establishes EEG as a dynamic treatment biomarker, enabling feedback-informed clinical management rather than static diagnosis.

The platform's compatibility with digital behavioral and physiological monitoring systems situates it within broader precision psychiatry ecosystems. Integration with ecological momentary assessment, actigraphy, cognitive testing, and wearable physiological sensors enables concurrent tracking of subjective and objective markers of attention, arousal, and executive function. This multidimensional dataset supports predictive modeling of treatment trajectories, relapse risk, and comorbidity emergence. Moreover, BrainView's intuitive visualization reports enhance patient engagement, allowing clinicians to communicate neurophysiological progress directly to patients and families, reinforcing adherence and motivation.

Research and Data Harmonization

In research contexts, BrainView provides a reproducible infrastructure for multi-site data harmonization and mechanistic inquiry. Its standardized acquisition and FDA-cleared analytic pipelines ensure consistent preprocessing, spectral quantification, and normative scaling across laboratories, enabling pooling of large datasets. This supports computational modeling of ADHD neurophysiology, cross-modal integration with fMRI or MEG, and cross-diagnostic comparisons aligned with Research Domain Criteria (RDoC) frameworks. In adjunctive therapy trials, qEEG/ERP metrics serve as objective secondary endpoints, clarifying neural mechanisms underlying interventions such as neurofeedback, mindfulness, or sleep optimization. The ability to link changes in P3 amplitude or theta–beta power to clinical outcomes enhances biological validity and accelerates mechanism-based therapy development.

Furthermore, BrainView's standardized data outputs facilitate federated learning and meta-analytic aggregation, refining predictive models for subtype classification, treatment response, and longitudinal neural trajectories. The platform thus functions not only as a clinical diagnostic and monitoring tool but also as a meta-scientific engine advancing reproducibility, transparency, and open data science in electrophysiology.

Future Directions and Translational Outlook

To realize the full potential of EEG biomarkers in ADHD, several developments remain essential. Expansion of BrainView's normative datasets across age, sex, ethnicity, and comorbidity profiles will improve external validity and reduce bias. Multi-center calibration studies should establish reproducibility benchmarks for spectral and ERP measures, ensuring cross-site consistency. Integration of EEG biomarkers with genetic, behavioral, and imaging data will support multimodal models of ADHD heterogeneity. Finally, real-time feedback systems capable of streaming BrainView outputs to clinical dashboards could enable adaptive treatment guidance and neurophysiologically informed decision support. Addressing these priorities will require coordinated collaboration among clinicians, data scientists, and regulatory bodies.

EEG biomarkers offer a transformative pathway toward objective, mechanistically grounded ADHD assessment and treatment monitoring. The BrainView platform operationalizes this vision by embedding standardized qEEG and ERP acquisition, normative benchmarking, and automated analytics within a scalable clinical workflow. Through reproducibility, automation, and rigorous statistical mapping, BrainView bridges the gap between experimental neurophysiology and practical psychiatry, advancing the field toward precision, data-driven, and individualized ADHD care.

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