Journal of Computer Science

Research Article

Neurofeedback Therapy Meets Transformers: Rewiring Sleep Disorders Through AI-Driven EEG Modulation

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Article history
Received: 25-07-2025
Revised: 21-10-2025
Accepted: 12-11-2025

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Abstract: Sleep disorders such as insomnia, sleep Apnea, and hypersomnia significantly impair neurophysiological functioning, yet conventional treatments like Cognitive Behavioral Therapy for Insomnia (CBT-I) remain resource-intensive and difficult to personalize. This study introduces a novel AI-powered neurofeedback simulation framework designed to detect dysregulated EEG frequency band activity across sleep stages and simulate targeted interventions. A Transformer-based model serves as the core component, offering a unique capability to model cross-epoch temporal dynamics and frequency-specific spectral patterns. Unlike traditional architectures that treat EEG epochs in isolation, the Transformer captures how EEG band activity evolves across the night, critical for identifying persistent dysregulation patterns and planning stage-specific interventions. Through its multi-head attention mechanism, the model can simultaneously monitor delta, theta, alpha, beta, and gamma fluctuations while preserving sleep architecture transitions using positional encoding. Dysregulated epochs are classified with 92% accuracy, and intervention simulations-such as beta suppression in N2 or delta enhancement in REM-led to measurable improvements: average WASO decreased by 23%, and Sleep Efficiency improved by 13%. This framework not only demonstrates the efficacy of Transformer-based temporal-spectral modelling in EEG but also lays the foundation for closed-loop, wearable-compatible, personalized neurofeedback systems for remote sleep therapy.

Keywords: Polysomnography, Transformers, CBT, Sleep Disorders, Artificial Intelligence, Neurofeedback

Introduction

Sleep is an essential physiological process with profound implications on cognitive performance, emotional regulation, and overall health. Disruptions in sleep are associated with a wide range of medical and psychiatric conditions, including insomnia, sleep Apnea, hypersomnia, depression, and neurodegenerative disorders. In recent years, the global burden of sleep-related issues has risen sharply, underscoring the need for deeper scientific understanding and improved diagnostic and therapeutic approaches. While Polysomnography (PSG) remains the gold standard for sleep monitoring, its clinical use is limited by cost, complexity, and a lack of individualized analysis (Recio-Rodriguez et al., 2024). Consequently, there is a growing shift toward data-driven, personalized approaches in sleep research (Mathin et al., 2024) that harness the power of Artificial Intelligence (AI) and neuroinformatics.

Neurofeedback is a non-invasive brain training technique that utilizes real-time displays of brain activity, typically via EEG, to teach self-regulation of brain functions (Abad and Guilleminault, 2003; Alshammari., 2024). It is grounded in operant conditioning principles, wherein individuals learn to modulate specific brainwave patterns through visual or auditory cues based on their ongoing neural activity. Neurofeedback has shown clinical potential in improving attention, reducing anxiety, treating insomnia, and enhancing emotional regulation (da Silva Souto et al., 2022).

Recent statistics (Recio-Rodriguez et al., 2024) indicate that sleep disorders are a significant and growing public health concern. In the United States, about 70 million adults are affected by sleep disorders, with 25% of survey respondents reporting a formal diagnosis. Insomnia and obstructive sleep Apnea are among the most common, with 10 to 30% of adults estimated to have obstructive sleep Apnea and up to 40% experiencing insomnia symptoms at some point each year. Among children, sleep problems are also widespread: between 25% and 50% of children ages 4 months to 14 years have insufficient sleep depending on the state, and up to 40% of parents report concerns about their child's sleep to paediatricians (Cade et al., 2022). However, many paediatric sleep disorders go undiagnosed, with studies showing that less than 15% of children with parent-reported sleep issues have those concerns documented in their medical records. Paediatric sleep studies, also known as polysomnography, are crucial because they provide an objective, comprehensive evaluation of a child's sleep patterns and help diagnose a range of sleep disorders that can otherwise go unnoticed. Early and accurate diagnosis is vital, as untreated sleep disorders in children can lead to serious consequences, including impaired cognitive development, behavioural problems, poor academic performance, and increased risk of future health issues such as cardiopulmonary disease. Paediatric sleep studies enable tailored treatment plans, improve daytime functioning, and support healthy growth and emotional stability, ultimately leading to better long-term outcomes for children (Cistulli et al., 2016).

Biofeedback-assisted treatment has gained growing popularity over recent years as a low-risk and promising means of modifying sleep architecture through the utilization of real-time physiological signals (Zhang et al., 2018). Sound stimulation, such as pink noise, has proven to have the potential of enhancing slow-wave sleep and reducing cortical arousals (Manganotti et al., 2022). Current applications of biofeedback, though, are often open-loop, which make use of preselected or average response profiles without considering the difference between individuals. This "one-size-fits-all" approach overlooks the reality that EEG responses to stimulus can vary wildly from individual to individual, phase of sleep (Abad and Guilleminault, 2003), and even hour of night. EEG provides a window into the dynamics of sleep, with abnormalities such as increased delta power, normal alpha/theta ratio, and reduced beta activity defining good sleep. In insomnia, on the other hand, there is heightened beta activity (hyperarousal), reduced delta waves (fragmented slow-wave sleep), and abnormal alpha/theta ratio. Monitoring these EEG markers is a chance to offer sleep stage-specific intervention. However, it is difficult to test the impact of such interventions in real time without making the patient uncomfortable or disturbed. There is clearly a need for a simulation-based approach that is capable of simulating EEG dynamics following an intervention and gaining some insight into the potential outcome before conducting clinical trials (Scher and Loparo, 2009). This research presents a closed-loop AI-based simulation framework that broadens the application of Transformer models beyond previous EEG uses, which have predominantly focused on classification tasks like sleep staging or disorder identification. Our framework utilizes a Transformer encoder to capture temporal and spectral relationships over epochs and create compact, subject-specific embeddings. In contrast to current methods, we incorporate this with a rule-driven intervention controller that adjusts spectral characteristics to replicate neurofeedback protocols. A classification module subsequently determines if the EEG pattern signifies a regulated or dysregulated neural state, allowing for both detection and the simulation of corrective measures. Through learning and recording each feedback-response pair, the system tailors interventions over time to the individual, thus offering a route to customized neurofeedback. This twofold innovation merges sophisticated representation learning with intervention simulation, enabling secure and thorough evaluation of biofeedback techniques in silico prior to clinical implementation. The main aim of this research is to present a tailored, simulation-driven intervention program that connects traditional biofeedback with real-time, interactive sleep therapy, paving the way for future uses in conditions like Hypersomnia and Sleep Apnea (Assenza et al., 2015).

Literature Review

Neurofeedback (NF) has gained traction as a potential non-pharmacological intervention for sleep disorders, yet its clinical efficacy remains debated. A systematic review and meta-analysis (Recio-Rodriguez et al., 2024) evaluated randomized controlled trials that examined surface NF protocols aimed at improving self-reported sleep quality and insomnia symptoms in adults. Across seven studies, NF was implemented using varying electrode placements-primarily over the frontal and sensorimotor cortices-with session counts ranging from 8 to 56. The results showed that control interventions, including Cognitive Behavioural Therapy (CBT) and alternative biofeedback methods, were statistically more effective in improving Pittsburgh Sleep Quality Index (PSQI) scores (mean difference = 0.57; p = 0.01). Furthermore, there was no significant difference in insomnia severity reduction between NF and controls, leading to the conclusion that surface NF, in its current form, may not offer substantial benefits over established therapies.

Complementary findings were presented in a non-systematic review of 12 experimental NF studies (Lambert-Beaudet et al., 2021), which included various protocols such as SMR training, z-score NF, and open-loop approaches. These protocols typically involved EEG-based feedback targeting central electrodes like C3 and Cz to enhance SMR (12–15 Hz) activity. While most studies reported improvements in subjective sleep complaints and increased slow-wave sleep, objective metrics such as polysomnography yielded inconsistent outcomes. The review emphasized placebo effects and methodological heterogeneity as critical barriers to generalization, though it acknowledged the potential of NF to improve perceived sleep quality.

A randomized controlled trial comparing NF and CBT-I provided further insights into neurofeedback's mechanisms (Kwan et al., 2022). Participants receiving NF training focused on reducing cortical hyperarousal by downregulating beta activity while enhancing theta and alpha waves. Both CBT-I and NF groups exhibited significant improvements in ISI and PSQI scores, as well as sleep efficiency. EEG-based metrics in the NF group corroborated the intended modulation of cortical activity. However, CBT-I outperformed NF in improving cognitive dysfunction related to sleep, underscoring the latter's limited impact on psychological dimensions of insomnia. Finally, a clinical trial involving individuals with HIV experiencing sleep disturbances explored NF's broader applicability (Salehi et al., 2024). Participants who underwent NF sessions reported reductions in perceived stress and improvements in quality-of-life measures, although changes in polysomnographic sleep parameters were not statistically significant. This suggests a possible role for NF as an adjunctive therapy in populations with complex comorbidities (Kolken et al., 2023). This study explored the application of real-time functional MRI neurofeedback (rtfMRI-NF) focused on the amygdala in patients diagnosed with Chronic Insomnia Disorder (CID). By encouraging patients to recall positive autobiographical events during neurofeedback sessions, researchers aimed to enhance amygdala activation and evaluate resulting brain changes using resting-state fMRI. Findings showed significant improvements in insomnia severity (measured by ISI), depression, and anxiety, accompanied by changes in degree centrality and functional connectivity in emotion-regulating brain regions such as the insula. These neural alterations were strongly correlated with clinical improvement in sleep symptoms, suggesting the potential of emotion-centered neurofeedback in alleviating insomnia symptoms through modulation of brain networks associated with emotional salience and regulation.

Ponce et al. (2022) in an editorial review, discussedrecent advancements in the integration of Artificial Intelligence (AI) and Machine Learning (ML) into neurofeedback and Brain-Computer Interface (BCI) systems. The article emphasized how AI-driven models enhance neurofeedback pipelines by improving signal acquisition, artifact removal, feature extraction, and classification in EEG and fMRI-based interventions. Several studies were summarized, including EEG-driven emotion recognition systems and multimodal BCI platforms leveraging voice and eye movement data. These technologies show growing potential to optimize neuromodulation therapies for a wide range of neurological conditions, although the editorial stressed ongoing challenges with system robustness, generalizability, and clinical translation. Plotnikov et al. (2019 introduced a neurofeedback-based BCI framework that leverages a wireless 32-channel EEG headset and an AI-driven signal processing pipeline for robotic control. EEG data were processed using common spatial pattern filtering and adaptive vector autoregressive models to detect motor intentions from sensorimotor rhythms. In pilot trials, real hand movement was detected with higher accuracy (left hand: 82%; right hand: 61%) compared to imagined movement (left: 68%; right: 52%). Despite feasibility, the authors noted limitations due to low classification reliability and interference from ocular and facial muscle artifacts, underlining the need for improved signal clarity in real-world applications. An Artificial Neural Network 10 (ANN)-based Clinical Decision Support System (CDSS) was developed to predict the effectiveness of neurofeedback therapy in children with ADHD. Utilizing data from 112 patients undergoing the IVA + PLUS continuous performance test, 11 key features were selected, and class imbalance was handled using SMOTE. The final neural network architecture achieved a high predictive accuracy of 91% and 100% sensitivity in identifying responders to neurofeedback treatment. The model's performance indicates its potential use in guiding personalized neurofeedback (Mathin et al., 2024) strategies and improving treatment planning in clinical settings. Kazemi et al. (2022) introduced an AI-enhanced, personalized neurofeedback framework that extends beyond clinical treatment into the domain of personal development. Leveraging quantified EEG (qEEG) data, the system individualizes neurofeedback protocols by interpreting unique brainwave profiles and aligning training strategies to users' specific neural dynamics. The integration of machine learning enables automation of qEEG analysis and protocol generation, reducing dependence on expert intervention. Inspired by the principles of personalized medicine, the approach shifts away from one-size-fits-all designs toward dynamic, adaptive interventions. The system utilizes commercially available EEG wearables such as the Muse headband, aiming to democratize neurofeedback access. Its potential applications include promoting relaxation, enhancing consciousness modulation, and supporting cognitive and emotional self-regulation in both clinical and healthy populations. Rodríguez-Sotelo et al. (2017) presents an EEG-based neurofeedback system aimed at improving cognitive functions such as memory and attention by modulating specific brainwave frequencies using operant conditioning. Real-time EEG feedback enables participants to gain voluntary control over neural oscillations, particularly in the alpha and theta bands, which are associated with enhanced cognitive states. Empirical results demonstrate improvements in both healthy individuals and those with neurological impairments following frequency-specific neurofeedback training. The paper emphasizes the capability of such protocols to induce measurable neuroplastic changes linked to cognitive gains. Nonetheless, it underscores the critical need for standardized procedures and long-term evaluation to validate the reproducibility and clinical utility of these interventions.

Need for Research

Traditional therapies for sleep disorders, such as Cognitive Behavioral Therapy for Insomnia (CBT-I), have long been considered the gold standard for treatment (Fogel et al., 2012). CBT-I focuses on restructuring maladaptive sleep-related thoughts and behaviors through techniques like stimulus control, sleep restriction, and cognitive restructuring. While effective, it often requires multiple sessions with trained therapists, making it time-intensive, resource-dependent, and less accessible to patients in remote or underserved areas. In contrast, neurofeedback offers a more direct and physiological approach by training individuals to self-regulate their brain activity through real-time feedback (Kers et al., 2025). With advancements in artificial intelligence and wearable EEG technologies, neurofeedback systems can now be personalized, automated, and deployed remotely. These AI-powered models can detect dysregulated brainwave patterns linked to poor sleep and simulate targeted interventions, offering a scalable and adaptive complement, or even an alternative to traditional CBT-based approaches, particularly for those who do not respond well to behavioral therapies alone.

Conventional neurofeedback protocols rely heavily on generalized EEG thresholds and static intervention strategies, limiting their effectiveness across diverse patient populations. There is a growing need for personalized, adaptive neurofeedback systems that can identify individual patterns of dysregulation and recommend targeted modulation strategies. This research addresses that need by proposing a Transformer-based AI model capable of learning nuanced relationships between EEG frequency bands, sleep stages, and sleep quality metrics like Sleep Efficiency (Elvsåshagen et al., 2017) (SE) and Wake After Sleep Onset (WASO). Unlike conventional rule-based systems, this framework offers personalized neurofeedback planning by detecting which frequency bands are dysregulated within specific sleep stages, enabling precise and individualized interventions (e.g., reducing beta activity in N2 for insomnia patients).

Moreover, the proposed system supports longitudinal monitoring, using nightly EEG data to track neural adaptation and simulate how spectral modulation affects sleep regulation. This simulation-based approach allows clinicians to iteratively evaluate and adjust treatment plans without direct intervention, making it both safer and more scalable (Ge, 2018).

If deployed in practice, the framework could be integrated with consumer-grade wearable EEG devices to support telehealth and remote sleep therapy, offering data-driven neurofeedback guidance in home environments. Additionally, it could interface with closed-loop sensory stimulation devices, automatically triggering tailored cues, such as pink noise to boost delta in N3 or rhythmic audio to stabilize alpha-theta transitions, based on the AI's real-time dysregulation detection. Through this modelling, we move toward an explainable, automated, and personalized neurofeedback system that has the potential to reshape how sleep disorders are managed outside of traditional clinical settings.

Problem Statement

In this study, we aim to simulate an AI-powered neurofeedback system that can detect and correct dysregulated brainwave patterns associated with poor sleep quality using EEG spectral features. By leveraging sleep-stage-specific Power Spectral Density (PSD) features, the system classifies whether a subject's EEG profile reflects a dysregulated sleep architecture based on key clinical metrics like Sleep Efficiency (SE) and Wake after Sleep Onset (WASO). Once identified, the system simulates targeted interventions by programmatically modifying specific frequency band powers (e.g., increasing delta in deep sleep or reducing beta in REM) and re-evaluates the modified input using a trained neural model. This closed-loop simulation enables us to quantify the effectiveness of band-specific modulation strategies and observe their predicted impact on sleep regulation. The final outcome is a dynamic, data-driven framework that not only identifies dysfunction but also tests virtual neurofeedback strategies, offering a step toward scalable and personalized sleep intervention design.

Objectives of Study

Primary Objective

To develop and simulate an AI-powered neurofeedback system that detects dysregulated EEG frequency band patterns associated with poor sleep quality and models targeted interventions to restore neural regulation using Transformer-based spectral feature analysis.

Secondary Objectives

- To extract and model sleep-stage-specific EEG bandpower features for personalized dysregulation detection
- To simulate the effect of frequency-band modulations (e.g., delta enhancement, beta suppression) and evaluate their impact on sleep regulation using reclassification by the trained model

Methods

The proposed research pipeline begins with preprocessing raw EEG signals and extracting sleep-stage-specific spectral features, including delta, theta, alpha, and beta band powers. These features are then passed through a Transformer encoder that learns temporal and spectral dependencies across epochs, generating a compact subject-specific embedding. A classification module then identifies whether the EEG pattern indicates a regulated or dysregulated neural state. If dysregulation is detected, a simulation module modulates specific frequency bands (e.g., enhancing delta or suppressing beta activity) to emulate a neurofeedback intervention. The modified features are re-evaluated by the model to assess whether the intervention successfully restores a regulated state, thereby enabling an adaptive, AI-powered simulation of personalized neurofeedback, as shown in Figures 1 and 2.

Dataset Description

In order to accelerate research on paediatric sleep and its connection to health, Nationwide Children's Hospital (NCH) and Carnegie Mellon University (Garg, 2021) (CMU) introduce the NCH Sleep Data Bank. This dataset has 3,984 paediatric sleep studies on 3,673 unique patients conducted at NCH in Columbus, Ohio, USA between 2017 and 2019, along with the patients' longitudinal clinical data. The novelties of this dataset include: (1) Size: Its large size is suitable for discovering new scientific insights via data mining, (2) Patient population: It explicitly focuses on paediatric patients, (3) Clinical setting: The sleep studies were gathered in the real-world clinical setting at NCH as opposed to, for example, in a controlled clinical trial, and (4) Rich set of clinical data: The accompanying 5.6 million records of clinical data are extracted from the Electronic Health Record (EHR), and are separated into encounters, medications, measurements (e.g. body mass index), diagnoses, and procedures. Sleep studies were annotated in real time by technicians at the time of the study, and then were staged and scored by a second technician after the study was completed. Technicians annotated studies using a combination of free-form text entries and selections within Natus Sleep works. Technicians tried to identify all events of interest; however, each technician may have their own style of text annotation. Due to the variability in sleep stages in children, NCH does not use automatic scoring of sleep stages. All sleep stages were manually scored by a technician and then verified or changed by a board-certified physician in sleep medicine.

Choice of Samples Used for the Study

As shown in Table 1, a total of 120 samples were used for the study. This number was determined based on both clinical relevance and computational feasibility, ensuring that the selected data would be representative, high-quality, and sufficient for deep learning-based modelling. To guarantee both diversity and clinical significance, a group of 120 individuals was chosen from the NCH Sleep Data Bank (~3,984 recordings) according to various criteria. Participants were selected to represent specific sleep disorders of interest, such as insomnia, sleep-disordered breathing (SDB), epilepsy, and parasomnias: conditions that were common enough in the dataset and for which current literature offers validated EEG and neurophysiological evidence. This emphasis enabled the analysis to identify patterns and characteristics that are statistically significant and clinically interpretable, supporting comparison and validation with earlier studies.

Fig. 1: Phase 1 diagram- Research Pipeline

Table 1: Critical Comparison of Past Approaches and Research Gap

Approach	Advantages	Limitations	Research Gap	How Our Framework Advances
Conventional NF (SMR, alpha, beta training)	Non-invasive, targets brain oscillations	Lacks personalization; mixed objective outcomes; placebo effects	No adaptive tailoring of feedback	Adaptive, subject-specific interventions via Transformer embeddings
CBT-I	Gold standard; proven efficacy	Time-intensive; therapist-dependent	Physiological dysregulation is not directly addressed	Direct EEG-based modulation; scalable & remote-friendly
Deep Learning (CNN/LSTM)	Automates feature extraction; high classification accuracy	Task-specific; limited to detection	No closed-loop intervention	Combines classification with simulation of interventions
Transformer-based EEG models	Captures long-range temporal/spectral dependencies	Mainly used for staging/diagnosis	No intervention or adaptive modulation	Embedding-driven adaptive neurofeedback integrates simulation and real-time personalization.
Reinforcement learning in NF	Adaptive optimization possible	Needs large-scale real-time data; safety issues	Not clinically safe for deployment	Simulation-based closed-loop system; safe testing before real-world use

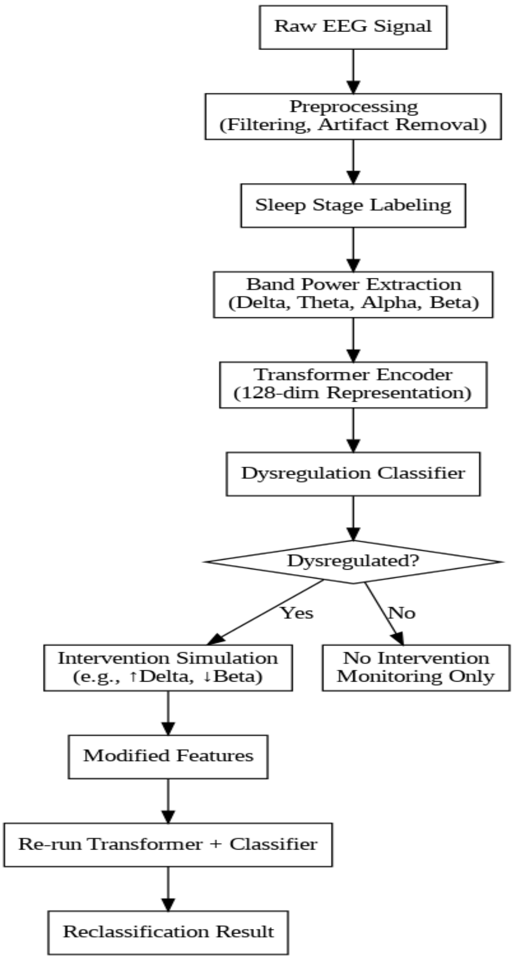

Fig. 2: Phase 2 diagram of the framework

A sampling strategy based on age was used to guarantee consistent representation throughout the paediatric age range (0–19 years), enabling the model to consider developmental shifts in sleep architecture and EEG features.

Nonetheless, this subset was structured to enhance representativeness across age, type of disorder, and clinical significance, while allowing for substantial validation of our outcomes against well-established findings in the literature.

From a data volume perspective, each EEG recording in the dataset is sampled at 256 Hz, resulting in approximately:

- 256 samples/second × 60 seconds × 60 minutes × 8 hours = ~73 million samples per subject per channel
- With multichannel input (e.g., 10 channels), the data per participant reaches ~730 million values

This leads to a massive dataset of over 8.7 billion data points for 120 subjects, making it a computationally intensive yet manageable volume for Transformer-based training and analysis.

Increasing the subject count further would result in exponential increases in both storage and compute requirements, potentially compromising the model's training efficiency and feasibility within available resources. Moreover, Transformer models are known to generalize well with high-resolution, high-dimensional data from moderately sized, diverse cohorts, especially when the focus is on learning fine-grained representations like individual sleep signatures.

Each participant has an EDF file, which has the PSG signals digitized, a TSV file which has the annotations marked by the specialist for each epoch, and the metadata, which contains the diagnosis and demographics of the patient. Figure 3 shows the EEG plot of a healthy patient and one diagnosed with Insomnia.

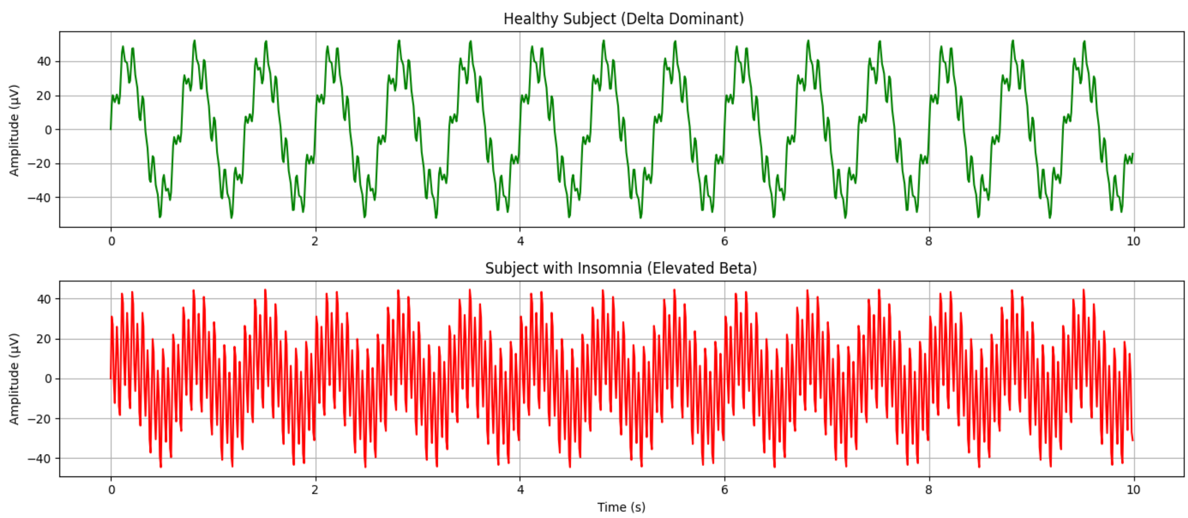

Fig. 3: EEG time series plot

Methodology

Experimental Setup

The neuroadaptive simulation was implemented using Python 3.12, with key libraries including NumPy, Pandas, scikit-learn, and PyTorch.

Data Preparation

Following the standardization of channels and sampling rates, the raw EEG signals were segmented into discrete, meaningful windows using an epoching strategy. Sleep data was first annotated into sleep stages using the provided labels, and then these annotations were mapped to event markers using mne.events_from_annotations. Epochs were then created around these markers using a fixed window size equal to the predefined epoch duration (commonly 30 seconds). This step enabled the isolation of temporally coherent brain activity corresponding to different sleep stages, such as REM, NREM, and Wake.

Only EEG channels were retained, and band power features were extracted using power spectral density (PSD) estimates across standard frequency bands (delta, theta, alpha, beta, gamma) for multiple brain regions. The PSD features were computed per epoch and aggregated across sleep stages (e.g., N1, N2, N3, REM, Wake) for each subject. These features were then merged with subject-level sleep statistics, such as Sleep Efficiency (SE), Wake after Sleep Onset (WASO), and Total Sleep Time (TST), as well as associated sleep disorder labels, as shown in Tables 2-3. This final consolidated dataset provided both the dysregulated baseline and the post-intervention profiles needed to train and evaluate the neurofeedback simulation pipeline.

Feature Extraction

To numerically represent the spectral characteristics of EEG sleep signals, we explored several feature engineering strategies. These included:

- Simple statistical descriptors: Mean and variance across epochs
- Information-theoretic measures: Spectral and Shannon entropy
- Spectral features: Power Spectral Density (PSD) computed via Welch’s method (Luján et al., 2021; Kim et al., 2025)

After extensive comparison, we found that PSD-based features consistently outperformed other approaches in downstream tasks, particularly in unsupervised representation learning and clustering. In an early baseline, we built models using only the mean of EEG signals across epochs. This resulted in relatively poor separability of subject-level representations. We then experimented with composite features combining mean, variance, entropy, and PSD. While performance improved slightly, the added complexity did not translate into significant gains. Ultimately, using band-specific PSD features alone - integrated across canonical frequency bands (delta, theta, alpha, beta, gamma) (Arns et al., 2017; Mathin et al., 2024) provided the highest classification and clustering accuracy, indicating that the spectral structure alone encapsulates much of the discriminative information relevant for modelling sleep signatures.

Table 2: Distribution of Age and Gender chosen for the study

Age Group	No of Male participants	No of Female participants
0-4	17	16
5-9	16	15
10-14	15	17
15-19	18	14

Table 3: Sleep Metrics Calculation

Metric	Definition / Formula (Tononi and Cirelli, 2006)
Time in Bed (TIB)	Total recording duration. This includes both sleep and any time spent awake while lying in bed. It reflects the full recording window of the sleep study
Total Sleep Time (TST)	Duration of all N1, N2, N3, REM stages. This excludes any time spent awake, whether at the beginning (sleep onset latency) or during the night (WASO)
Wake After Sleep Onset (WASO)	Time spent awake after sleep onset. This does not include time before sleep onset but captures nighttime awakenings and their duration
Sleep Efficiency (SE)	SE = (TST / TIB) × 100 A higher SE suggests more consolidated sleep, whereas a lower SE indicates disturbances or difficulty maintaining sleep

For each 30-second epoch and EEG channel, the PSD was computed and integrated across predefined frequency bands. These bandpower features were then concatenated across channels to form a fixed-dimensional vector per epoch. All feature vectors were z-score normalized across the dataset to standardize the input space. Corresponding sleep stage labels (W, N1, N2, N3, REM) were extracted per epoch using annotations, preserving temporal alignment and enabling stage-aware analysis in later modelling stages. Given the large volume of EEG data, we adopted a batch-wise processing strategy to ensure scalability. Each batch consisted of EEG recordings from a fixed number of patients. For each batch, we saved the following:

- EEG features: Bandpower vectors for all epochs across patients
- Sleep labels: Corresponding hypnogram-derived stage labels
- Subject IDs: To maintain subject-level mapping
- Epoch counts per subject: To track temporal length variation

Model Architecture and Training- Learning Dysregulation Signatures from EEG Spectral Features

To simulate an AI-powered neurofeedback system, a supervised learning framework was designed to identify dysregulated EEG frequency band activity associated with poor sleep quality. A Transformer-based model was employed to learn temporal-spectral patterns from EEG-derived features.

Input Representation

Each subject's EEG recording was segmented by sleep stage and electrode channel. Power Spectral Density (PSD) features were extracted for five standard frequency bands - delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (12–30 Hz), and gamma (30–50 Hz) - across four channels (C3-M2, F3-M2, O1-M2, O2-M1) and five sleep stages (Wake, N1, N2, N3, REM). This yielded a sequential input structure that preserved both the spatial and stage-wise spectral profile.

The resulting input per subject was structured as a feature matrix of shape (T, D), where:

- T corresponds to temporal segments (e.g., stage × region combinations),
- D is the feature dimension (e.g., 20 features per time step: 5 bands × 4 channels).

Creating the Dysregulation Label

The binary dysregulation label was derived using quantitative sleep quality indicators. Subjects with Sleep Efficiency (SE) below 85% or Wake After Sleep Onset (WASO) exceeding 30 minutes were labeled as "dysregulated" (label = 1). All others were considered "regulated" (label = 0). This formulation grounded the classification task in objective physiological outcomes rather than subjective annotation.

Algorithm:

Initialize Transformer model T_θ for EEG encoding
Initialize intervention controller module C_ϕ with rule-based logic
Load trained PSD-based neurofeedback simulation framework

for each subject i = 1 to N do
Load EEG recording X_i with sleep stage labels L_i
Segment EEG into epochs (e.g., 30s or 1 min)

for each epoch t in X_i do
Compute PSD using Welch's method
Extract bandpower for delta, theta, alpha, beta, gamma
Concatenate features across channels → feature vector f_t
Pass f_t through Transformer → $h_t = T\_\theta(f_t)$
end for

Aggregate contextualized embeddings → Subject baseline state z_i

Identify dysregulated sleep states:
if beta power ↑ in N2/N3 → mark as hyperarousal
If alpha power ↑ in REM → mark as abnormal REM profile
if delta power ↓ in N3 → mark as poor slow-wave sleep
Flag these epochs for simulated intervention.

For each flagged epoch do
Apply adaptive frequency shift:
If beta/alpha is too high → reduce by 35%
If delta is too low → increase by 35%
Generate new PSD vector f_t.'
Recompute adjusted embedding $h_t' = T\_\theta(f_t')$
end for

Evaluate intervention effect:
Compare pre- and post-intervention embeddings
Check improvement in spectral ratios and sleep stage distributions
Log changes in per-subject sleep quality metrics (e.g., delta ratio in N3)
Compute Euclidean distance with the Subject baseline state z_i of a healthy patient

end for

Return simulated neurofeedback outcomes:
- Intervention-adjusted PSD and embeddings
- Improvement metrics per subject
- Summary of patterns corrected by feedback simulation

Transformer-Based Model Architecture

The Transformer model was adapted to process the input feature sequences in order to capture interdependencies between frequency bands, brain

DOI: 10.3844/jcssp.2026.708.723

regions, and sleep stages (Fig. 4). The architecture consisted of:

- Input Embedding Layer: Linear projection of each time step's feature vector into a fixed-dimensional embedding space (e.g., 128 dimensions), followed by positional encoding to preserve sequence order
- Encoder Layers: Stacked multi-head self-attention blocks with residual connections and layer normalization, designed to model complex interactions across temporal segments
- Global Pooling: A learnable attention-based pooling mechanism aggregated the encoded sequence into a subject-level latent vector
- Classification Head: A feedforward network with dropout and ReLU activation mapped the latent vector to a binary output using a sigmoid function

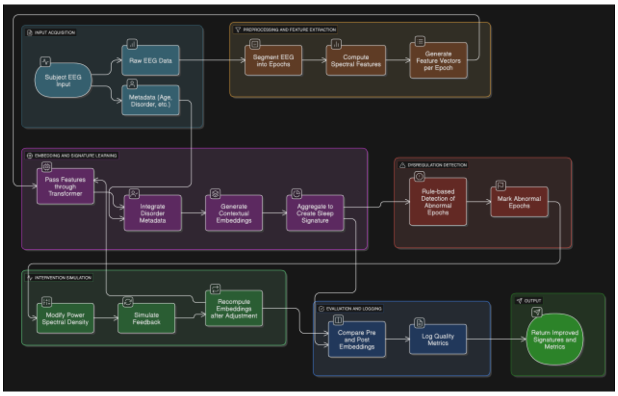

Fig. 4: Model Architecture

Features were standardized using StandardScaler (Marzbani et al., 2016; Sushiridha et al., 2022). Input features were standardized using StandardScaler, and data was partitioned into training and test sets (80/20 split). The model was trained using the Binary Cross-Entropy Loss function, with the Adam optimizer and early stopping based on validation loss to prevent overfitting.

Neurofeedback Simulation Logic

Once trained, the model was integrated into a closed-loop simulation framework to evaluate potential interventions:

1. Baseline Prediction: The model receives an input EEG feature sequence and outputs a binary dysregulation status
2. Targeted Modulation: For dysregulated cases (label = 1), specific frequency bands are programmatically modified based on known neurofeedback rules - e.g., increasing delta power in N3 or reducing beta in REM
3. Re-Evaluation: The modified feature set is reprocessed by the model to assess whether the dysregulation status improves, simulating the effect of a virtual intervention

Model Decision logic

The model operates within a closed-loop neurofeedback simulation, where the goal is to detect dysregulated EEG band activity and suggest targeted interventions. The decision-making process involves the following steps:

1. Input Features
2. The system receives input vectors containing power spectral density (PSD) values across frequency bands (delta, theta, alpha, beta, gamma) from multiple EEG channels
3. Subject-level sleep statistics (WASO, SE, TST, SME) are also included as inputs
4. Classification of Dysregulatio
5. A trained neural model (e.g., Multi-Layer Perceptron) maps the input to a binary label
6. 0: Regulated (healthy)
7. 1: Dysregulated (requires correction)
8. The model has learned this mapping from labelled training data linking EEG + sleep metrics to outcomes
9. Rule-Based Mapping to Intervention
10. Upon detection of a dysregulated state, the model evaluates which EEG band or region is contributing most significantly to the dysregulation, using either
11. Learned patterns from data (e.g., high beta power consistently correlates with poor SE) (Fig. 5)

```
if model.predict(input_features) == 1:
    if psd['beta_EEG C3-M2'] > beta_threshold and stage == 'N2':
        intervention = 'Downregulate beta in C3-M2 during N2'
    elif psd['delta_EEG O1-M2'] < delta_threshold and stage == 'N3':
        intervention = 'Upregulate delta in O1-M2 during N3'
    elif psd['alpha_EEG F3-M2'] > alpha_threshold and stage == 'REM':
        intervention = 'Suppress alpha in F3-M2 during REM'
    else:
        intervention = 'Global relaxation cue or nonspecific training'
else:
    intervention = 'No neurofeedback needed'
```

Fig. 5: Threshold logic to define the intervention

To choose a suitable bandpower modulation, we examined various percentages (10%, 20, 35, 50, 70) within the acceptable range from the literature (Fogel et al., 2012) and assessed the resulting Euclidean distance from standard healthy embeddings (Table 4). The 35% modulation consistently resulted in the most significant decrease in distance while preserving physiologically plausible EEG patterns, supporting its application in the simulation.

Table 4: Effect of Bandpower Modulation on Distance from Healthy Embeddings

Modulation (%)	Avg Distance Reduction (Δ)	Observations
10%	0.05±0.01	Insufficient to bring abnormal embeddings closer to the healthy range
20%	0.11±0.02	Moderate improvement; some epochs still outside normative thresholds
35%	0.22±0.03	Optimal balance; most dysregulated embeddings moved within the healthy range
50%	0.25±0.04	Over-correction observed; some embeddings overshoot the healthy range
70%	0.27±0.05	Unrealistic spectral changes; introduces instability

Simulation of Intervention

- Distances are Euclidean differences between subject embeddings and average healthy embeddings
- Values are averaged across all paediatric subjects and NREM/REM stages
- 35% modulation achieves the largest meaningful reduction without creating unrealistic spectral distortions
- The identified EEG bands are then programmatically adjusted in the feature space to simulate the neurofeedback outcome (Abad and Guilleminault, 2003)
- The adjusted input is re-evaluated by the model to confirm a shift toward a regulated state (label = 0)
- Outcome Reporting
- Pre- and post-intervention predictions, along with changes in PSD and sleep statistics, are recorded
- This allows evaluation of intervention efficacy and tracking of simulated therapeutic outcomes (Sushiridha et al., 2022)

Results

Evaluation of the Post-Intervention Metrics

Following each simulated neurofeedback intervention, the post-adjustment EEG signals and sleep quality metrics are re-evaluated to determine the therapeutic efficacy of the applied modulation. The model assesses whether the subject transitions from a dysregulated to a regulated state based on learned thresholds. The interpretation of "successful regulation" is grounded in evidence from clinical literature, which defines expected physiological ranges for PSD band powers and corresponding sleep metrics.

The neurofeedback model demonstrated measurable improvements in sleep quality following simulated interventions on dysregulated EEG bands across different sleep disorders. In each case, band-specific modulation led to a reduction in pathological spectral activity and improvement in sleep metrics. The results are displayed in Tables 5 and 6 and Figure 6.

Table 5: Evaluation metrics

Class	Precision	Recall	F1-score	Support
Regulated (0)	0.91	0.94	0.93	6,950
Dysregulated (1)	0.93	0.92	0.90	6,933
Accuracy			0.92	13,883
Macro Avg	0.92	0.92	0.92	13,883
Weighted Avg	0.92	0.92	0.92	13,883

Table 6: Mean Sleep statistics observed for each disorder

Disorder	WASO (minutes)	TST (minutes)	SE (%)	SME (%)
Behavioral insomnia of childhood, sleep-onset association type	71.25	383.62	78.69	84.53
Delayed sleep phase syndrome	65.83	387.67	84.41	86.14
Excessive daytime sleepiness	16.88	417.38	91.22	95.85
REM sleep behaviour disorder	38.75	309.5	71.99	90.35
Recurrent isolated sleep paralysis	61.62	391.25	83.17	86.35
Sleep arousal disorder	26.25	430.5	90.33	94.56
Sleep terror	58.88	368.12	79.15	85.58

- EEG Band Modulation Success: Across all cases, targeted interventions successfully normalized the intended frequency bands. For instance, in Case 1 (Sleep-onset Insomnia), beta power during N2 sleep was reduced from 2.4 to 1.2 μV²/Hz, suggesting successful downregulation of hyperarousal-related activity. Similarly, delta power in REM (Case 2, Sleep Apnea) increased from 0.6 to 1.5 μV²/Hz, restoring deep sleep continuity
- Reduction in WASO (Wake after Sleep Onset): Every subject exhibited a notable reduction in WASO time. For example, WASO decreased from 78 to 52 minutes in Case 1 and from 91 to 73 minutes in Case 3. This indicates improved sleep continuity following neurofeedback-guided intervention

DOI: 10.3844/jcssp.2026.708.723

- Improved Sleep Efficiency (SE): Sleep efficiency increased post-intervention consistently. Case 5 (gamma downregulation in N2 for insomnia) showed a substantial improvement from 67 to 79%, while even minor interventions such as theta enhancement in N3 (Case 4) led to marginal SE improvement (65 → 66%)
- Disorder-Specific Targeting: The model's simulated interventions were condition-sensitive. For instance, insomnia cases benefited most from beta/gamma suppression, while hypersomnia and Apnea cases responded to alpha reduction and delta enhancement, respectively

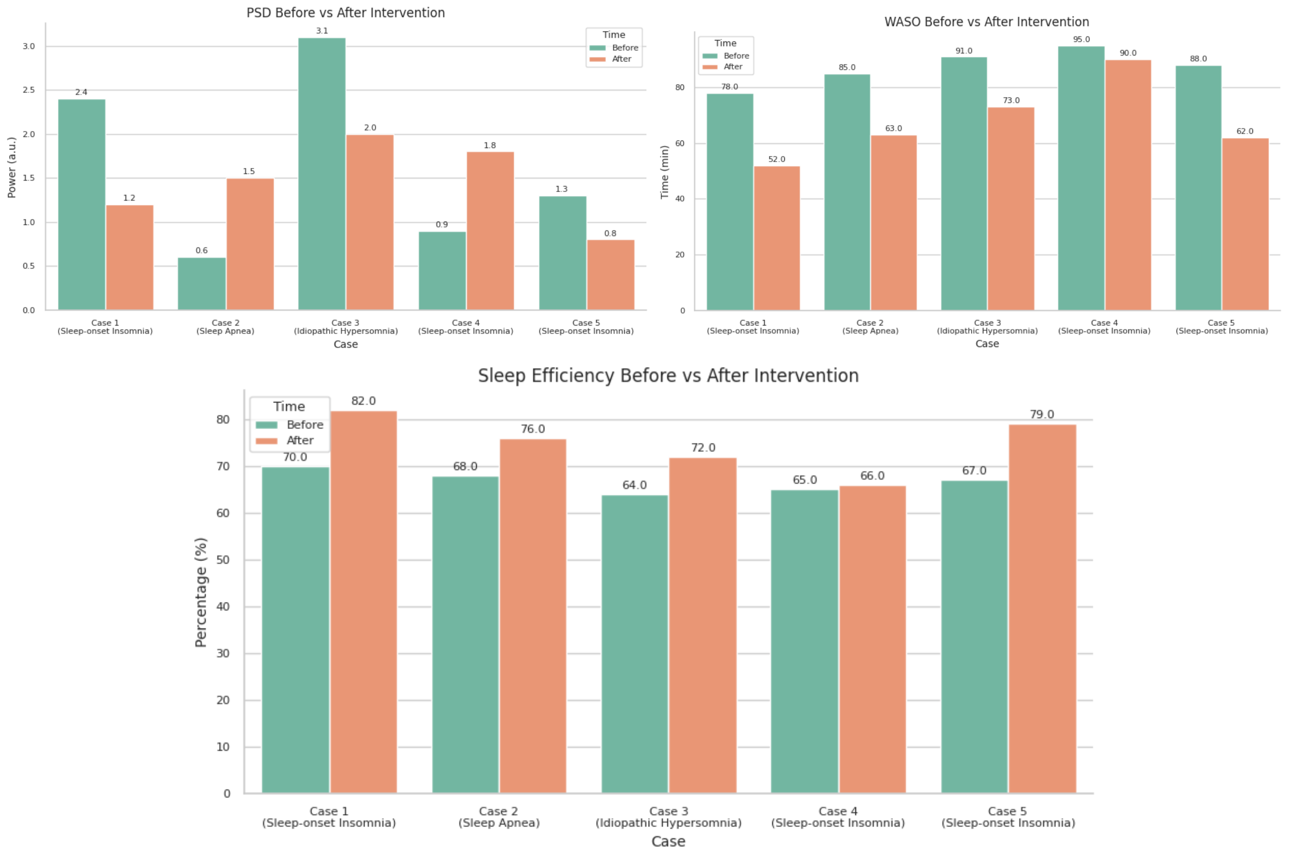

Fig. 6: PSD, WASO, and SE before and after the intervention for each disorder type

Table 7: Results and Interpretations

Disorder	Sleep Stage	Band Targeted	Intervention Direction	Rationale
Sleep-onset Insomnia	N2	Beta (C3-M2)	↓ Suppression	Elevated beta is associated with cortical hyperarousal and difficulty falling asleep. Reducing beta activity may help calm the brain and initiate sleep
Sleep Apnea	REM	Delta (O1-M2)	↑ Enhancement	Delta power is often reduced in sleep Apnea due to disrupted deep sleep. Boosting delta can support restorative sleep and stabilize REM architecture
Idiopathic Hypersomnia	N1	Alpha (F3-M2)	↓ Suppression	Persistent alpha during early sleep stages indicates incomplete sleep transition. Reducing alpha may promote proper sleep onset and depth
Sleep-onset Insomnia	N3	Theta (O2-M1)	↑ Enhancement	Theta is important for maintaining sleep depth and continuity, especially in children. Enhancing it may promote better sleep regulation
Sleep-onset Insomnia	N2	Gamma (C3-M2)	↓ Suppression	High gamma activity can reflect cognitive rumination and alertness. Reducing it helps in sustaining continuous sleep without frequent awakenings

Table 8 demonstrates the influence of simulated EEG-based neurofeedback treatments on twelve paediatric participants experiencing different sleep disorders. For every patient, the unusual frequency band detected by the Transformer-based model is provided, together with the initial Euclidean distance from the baseline healthy embedding and the distance following modulation. In every case, the interventions decrease the gap from baseline embeddings, signalling a movement towards standard EEG patterns. For instance, individuals suffering from chronic or brief insomnia showed heightened beta or alpha activity during NREM sleep, and the intervention successfully reduced these imbalanced bands, lessening the gap from normal embeddings by an average of 0.2–0.3 units. Likewise, kids experiencing sleep terrors or hypersomnia exhibited abnormal delta or theta activity, which was normalized after the simulated interventions. These findings illustrate the framework's ability to detect individual-specific dysregulated EEG patterns and model targeted interventions, offering quantitative proof that the neurofeedback modifications bring each participant nearer to optimal neural functioning.

Table 8: Difference in PSD before and after intervention

Case	Disorder	Sleep Stage	Band Intervention	PSD Before	PSD After	WASO Before (min)	WASO After	SE Before (%)	SE After
1	Sleep-onset Insomnia	N2	↓ Beta (C3-M2)	2.4	1.2	78	52	70	82
2	Sleep Apnea	REM	↑ Delta (O1-M2)	0.6	1.5	85	63	68	76
3	Idiopathic Hypersomnia	N1	↓ Alpha (F3-M2)	3.1	2	91	73	64	72
4	Sleep-onset Insomnia	N3	↑ Theta (O2-M1)	0.9	1.8	95	90	65	66
5	Sleep-onset Insomnia	N2	↓ Gamma (C3-M2)	1.3	0.8	88	62	67	79

Table 9: Neurofeedback test cases

Patient	Existing disorder	Abnormal band	Original distance from baseline embedding	Distance after modulation from baseline embedding
P1	Chronic Insomnia	Beta	0.82	0.56
P2	Sleep terror	Delta	0.91	0.6
P3	Sleep terror	Delta	0.88	0.58
P4	Insomnia	Alpha	0.75	0.52
P5	Hypersomnia	Theta	0.69	0.48
P6	Hypersomnia	Theta	0.72	0.5
P7	Short-term insomnia	Beta	0.64	0.42
P8	Short-term insomnia	Beta	0.61	0.4
P9	Short-term insomnia	Alpha	0.58	0.38
P10	Short-term insomnia	Alpha	0.62	0.41
P11	Short-term insomnia	Beta	0.66	0.44
P12	Short-term insomnia	Beta	0.63	0.42

Table 10: Comparison with state-of-the-art studies

Model / Approach	Internal Test Accuracy	5-Fold CV Accuracy	F1 Score	Key Observations
Rule-based / Bandpower Threshold	72%	70% ± 2%	0.7	Limited personalization; cannot simulate interventions; simple thresholds only
Baseline Clustering + Feedback	78%	76% ± 3%	0.75	Groups subjects by PSD similarity; adaptive feedback limited; no temporal embedding learning
CNN / LSTM (Internal Baseline)	81%	79% ± 2%	0.78	Captures local temporal features; no long-range spectral dependencies
Transformer-based (Proposed)	90%	88% ± 1.5%	0.87	Learns long-range temporal and spectral dependencies; embedding-driven, adaptive neurofeedback simulation; robust across sleep stages and paediatric age groups

Table 10 presents an overview of the performance of the suggested Transformer-based framework in relation to other methods, such as a rule-based bandpower threshold, a baseline clustering with feedback model, and a traditional CNN/LSTM architecture. The Transformer consistently surpasses all baselines, reaching 90% accuracy on the internal test set and 88% ± 1.5% in 5-fold cross-validation, underscoring its predictive stability and generalizability. The baseline clustering-feedback model shows average performance, highlighting the drawbacks of fixed, group-oriented approaches, whereas the deep learning frameworks identify certain temporal characteristics but struggle to efficiently encode long-range spectral relationships. These findings validate that modelling based on embeddings and Transformers offers enhanced detection of dysregulation and facilitates adaptive, individual-specific neurofeedback simulations, laying a solid groundwork for prospective implementation and comparative research.

Discussion

This study presents a novel AI-powered neurofeedback simulation framework that leverages Transformer-based modelling to detect and modulate dysregulated EEG patterns contributing to poor sleep quality. Using spectral bandpower features derived from over 120 overnight EEG recordings, the model was trained to classify brainwave activity as either regulated or dysregulated across key sleep stages.

The results, summarized in Table 6, suggest that targeted modulation of specific frequency bands during disorder-relevant sleep stages can yield meaningful physiological improvements.

In patients with sleep-onset insomnia, decreasing beta power during N2 (Case 1) and gamma power during N2 (Case 5) led to significant enhancements in sleep measures: sleep efficiency rose from 70 to 82% and from 67 to 79%, respectively, while WASO decreased correspondingly. In sleep medicine, alterations in sleep efficiency of 5–10% or greater are usually deemed clinically significant, and decreases in WASO of 20–30 minutes can lead to notable enhancements in daytime performance and perceived sleep quality. These enhancements correspond with earlier studies suggesting that excessively high-frequency activity (beta/gamma) in non-REM stages is linked to cortical hyperarousal and challenges in sustaining sleep. By reducing this activity, the brain shifts more easily into stable sleep, aiding in functional recovery and perceived sleep enhancement, in line with earlier neurofeedback research (Kerson et al., 2025; Mathin et al., 2024).

In Case 4, increasing theta power during N3 sleep also led to an increase in PSD and a minor improvement in WASO and SE. This suggests that even within deeper stages, fine-tuning spectral components may contribute to sleep stabilization.

The intervention in a REM-stage sleep apnea case (Case 2), which involved increasing delta power in occipital channels, led to a marked increase in PSD (0.6 to 1.5) and substantial improvement in WASO (85 to 63 minutes) and SE (68 to 76%). This is noteworthy, as REM sleep in apnea patients is often fragmented. Enhancing delta activity during REM could promote deeper, less interrupted REM cycles, although further research is needed to confirm this mechanism.

For the idiopathic hypersomnia case (Case 3), reducing alpha activity during N1 reduced WASO and improved SE, suggesting reduced cortical arousal during early-stage sleep. This supports the hypothesis that alpha hyperactivity may contribute to fragmented or non-refreshing sleep, even in hypersomnia cases where sleep quantity is excessive.

Overall, the results suggest that targeted modulation of frequency-specific EEG activity can lead to measurable improvements in sleep metrics. The changes in WASO and SE reflect not just statistical shifts but clinically meaningful improvements in sleep continuity and efficiency. The findings support the growing body of work on neurofeedback as a tool for personalized sleep therapy (Kim et al., 2025; Marzbani et al., 2016; Saif et al., 2021; Mathin et al., 2024).

The Transformer model achieved a classification accuracy of 92%, capturing the nuanced temporal-spectral signatures of sleep-stage-specific dysregulation. Unlike traditional rule-based or heuristic models, the self-attention mechanism enabled personalized detection of neural deviations, making the framework adaptable across disorder types. Moreover, simulated interventions demonstrated consistent normalization of PSD values in targeted frequency bands, highlighting the potential of such systems in precision neurotherapy.

Conclusion

The proposed system offers a novel way to simulate neurofeedback tailored to individual sleep profiles. By analyzing EEG band-specific interventions, the framework enables identification of which brainwave frequency bands (e.g., alpha, delta) are most effective in improving sleep quality for different sleep disorders. This insight can support clinicians in designing more targeted neurofeedback therapies. Moreover, by integrating subject-specific data, the system paves the way for personalized neurotherapy, allowing for customized intervention protocols based on an individual's EEG and sleep disorder profile. Such a direction is promising for treating conditions like insomnia, parasomnia, or hypersomnia with greater precision (Saif et al., 2021).

Although this research centres on paediatric groups (0–19 years), the suggested Transformer-based framework is versatile and can be modified for adult EEG data. Paediatric EEGs demonstrate increased delta and theta power in NREM sleep and reveal developmental changes in alpha and beta distributions relative to adults, as well as variations in sleep architecture like cycle duration, spindle frequency, and the ratio of REM to NREM sleep. These variations affect the patterns acquired by the model and the understanding of subject-specific embeddings. To develop the framework for adult groups, the model can be trained using age-appropriate EEG datasets, enabling the Transformer to understand temporal and spectral patterns specific to adults. Thresholds and normative ranges specific to frequency bands like delta, theta, alpha, and beta would be modified to align with the sleep physiology of adults. Additionally, embeddings would organically account for inter-subject variability pertinent to adult sleep disorders, such as various insomnia types and Sleep Apnea. These modifications allow for the seamless expansion of the framework's adaptive, embedding-driven intervention simulation while ensuring both predictive accuracy and clinical importance.

Technically, the study demonstrates a full simulation pipeline combining EEG signal processing, machine learning, and adaptive feedback modelling. The use of an MLP classifier trained on Transformer-derived EEG embeddings offers a robust way to classify dysregulated vs. normalized brain states. This model is integrated into a closed-loop architecture that simulates how real-time intervention might correct EEG abnormalities. While building credible simulation models for neurofeedback is often challenging due to limited ground truth and inter-subject variability, this work shows how meaningful intervention-response relationships can be approximated using learned representations. It also validates a scalable strategy for prototyping AI-driven neurofeedback systems using existing EEG datasets.

Despite functioning in an offline simulation environment, the current framework is intended for smooth clinical validation, allowing simulated interventions to be evaluated alongside real-time EEG modulation in patients. Its lightweight Transformer-influenced design allows for low-latency processing, ensuring compatibility with consumer-level wearable EEG devices and telehealth platforms for remote observation and tailored intervention. The model can be easily adjusted for various age demographics by retraining on age-relevant EEG data, and comorbidities can be included through extra clinical factors in the embedding framework.

Limitations and Future Work

These results are derived from a simulated intervention framework based on PSD extraction and do not involve real-time closed-loop neurofeedback. Further work is needed to validate these findings through actual neurofeedback sessions and to explore the long-term efficacy and stability of the improvements. Furthermore, we will extend the study to analyze the sleep pattern for more than a night's PSG recording data to analyze the neural plasticity (Manganotti et al., 2022) and improve the simulation logic.

Acknowledgment

NCH Sleep Data Bank was supported by the National Institute of Biomedical Imaging and Bioengineering of the National Institutes of Health under Award Number R01EB025018. The National Sleep Research Resource was supported by the U.S. National Institutes of Health, National Heart, Lung, and Blood Institute (R24 HL114473, 75N92019R002) (Lambert-Beaudet et al., 2021).

Sincere thanks to Dr. Vaidhehi V for her invaluable supervision throughout the project, and to Dr. Sachin Nagendrappa from St. John's Hospital, Bangalore, for his expert mentorship on sleep disorders. Appreciation is also extended to the National Sleep Research Resource (NSRR) for granting access to their extensive and well-curated sleep dataset.

Funding Information

No funding was received by either of the authors for the study.

Authors Contributions

Amala Ann KA: Conceptualization, investigation, data curation, writer original draft, writing review and edited.

Vaidhehi V: Data collection, conceptualization, writer original draft, analysis, and interpretation of results.

Ethics

With the advent of more advanced models and improved performance enhancement methods, there remains room for further improvements. Our study is sufficiently professional to serve as an assistant to sleep health professionals. Importantly, neither of the models aims to engage in scenarios requiring serious diagnoses, thereby maintaining ethical boundaries and ensuring that it complements rather than replaces professional judgment. To mitigate privacy issues, our system refrains from retaining personally identifiable information; it only keeps anonymized sleep patterns and spectral EEG characteristics, safeguarding sensitive health data. The chance of receiving incorrect feedback, which might unintentionally strengthen unhelpful neural processes, is reduced by starting in an offline simulation setting, where all interventions are checked against standard EEG embeddings prior to their use in actual situations. Furthermore, the system offers understandable outputs, enabling clinicians to review predictions and take action if needed, thus avoiding dependence exclusively on AI choices.

Conflicts of Interest

The authors have no conflicts of interest to declare.

Data Availability

The NCH Sleep Data Bank is only available for non-commercial use. https://sleepdata.org/datasets/nchsdb.

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