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Original Research Article

Graph-based learning for sleep microarchitecture: a hybrid graph autoencoder and graph attention network approach

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ABSTRACT

Background: Sleep plays a vital role in cognitive function, memory consolidation, and overall neurological health. Analysis of sleep microarchitecture including features such as sleep spindles, K-complexes, slow waves, and EEG bandpower components provides critical insights into sleep disorders and genetic diseases. However, the complex interactions between sleep architecture and underlying genetic abnormalities remain underexplored. This study aims to investigate these interactions by leveraging advanced graph-based deep learning methods to uncover hidden relationships within EEG signals.
Methods: We developed a graph autoencoder (GAE) combined with a Graph attention network (GAT) to analyze polysomnography (PSG) data from the National Children's Hospital (NCH) dataset. EEG epochs were modelled as graph nodes, while edges were constructed based on bandpower similarity between epochs, enabling dynamic representation of sleep activity. The GAE learned latent embeddings that capture subtle patterns in sleep microarchitecture, and the GAT applied attention mechanisms to classify and interpret relationships between EEG events, sleep disorders, and genetic abnormalities. Three core analyses were conducted: (1) identifying differences in sleep microarchitecture across sleep disorders, (2) detecting EEG event changes associated with genetic disorders, and (3) exploring shared patterns linking sleep and genetic abnormalities.
Results: The model achieved classification accuracies of 92.4%, 91.2%, and 88.6% across the three tasks, respectively. The approach successfully identified distinct EEG event patterns in subjects with co-occurring sleep disorders.
Conclusions: This work presents a scalable, automated, and interpretable framework for analyzing the interplay between sleep microarchitecture, sleep disorders, and genetic disorders.

Keywords: Polysomnography, Paediatrics, Graph attention networks, Genetic disorders, Sleep microarchitecture

INTRODUCTION

Sleep plays a vital role in cognitive processes such as memory consolidation, emotion regulation, and overall brain activity. New research indicates that an estimated 70 million American adults are experiencing either one or another sleep disorder, and most teenagers are getting less than the requisite amount of sleep. Across the globe, over one-third of adults describe themselves as suffering from insomnia, two-thirds indicate disturbed sleep on a nightly basis, and 80% say that they want to sleep better.

Sleep is a very intricate physiological condition with distinctive stages, i.e., rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. Slow-wave sleep (SWS), or deep sleep, occurring during NREM Stage 3, is particularly important for brain repair and synaptic plasticity. Slow waves, i.e., high-amplitude, low-frequency oscillations of the EEG signal, are an elementary component of sleep microarchitecture and are inextricably interwoven with cognitive repair and neurological health. Electroencephalography (EEG) is one of the foremost research tools of sleep. The EEG signals

can be separated into bands of frequencies: delta (0.5-4 Hz), theta (4-8 Hz), alpha (8-13 Hz), beta (13-30 Hz), and gamma (>30 Hz).[1]

Sleep microarchitecture refers to the microscopic characteristics of brain activity during sleep, as they appear on electroencephalography (EEG) records, beyond the traditional sleep categorization into distinct stages. Sleep microarchitecture is directly associated with EEG frequency bands. Different EEG bands prevail in different stages of sleep and contribute to the more specific information regarding brain activity that defines microarchitectural features.

In this paper, we propose a dynamic graph-based model that views EEG epochs as nodes, with edges drawn based on the similarity between bandpower profiles. The model draws on a GAE to map data to its latent representations that preserve the inherent structure of data. The classification is improved through the GAT, which incorporates the most crucial EEG features with a focus on the microarchitectural alterations across groups. We will aim at three major goals: (1) Determination of differences in sleep microarchitecture among sleep disorders, (2) Examination of changes in EEG events among patients with uncommon genetic disorders, and (3) Identification of common patterns that connect particular abnormalities of sleep with genetic conditions.

Research gap identified

Despite extensive research on sleep disorders, several critical gaps remain unaddressed. One notable limitation is the lack of studies focusing on paediatric sleep disorders, as most research primarily targets middle-aged and older populations. Understanding sleep disturbances in children is crucial for early intervention and developmental outcomes. Another new direction yet to be thoroughly addressed is applying GNN to the field of sleep science. GNNs have the potential to identify complex patterns among sleep epochs, sleep disorders, and comorbid conditions and offer a new insight into the identification of patterns in high-dimensional PSG data.

Need for research

What makes this a persistent research gap?

Genome-wide association studies (GWAS), which scan markers across the entire genome of large populations to detect gene variations linked to diseases, have been highly effective in identifying around 14 susceptibility loci for sleep disorders such as narcolepsy and restless legs syndrome.[6] These findings have enhanced the understanding of the genetic etiology of sleep-related disorders and their possible associations with other illnesses, enabling the development of more targeted strategies for diagnosis, treatment, and prevention. Sleep disorders are complex traits influenced by multiple genes and environmental factors, unlike simple Mendelian disorders caused by single gene mutations. GWAS have uncovered numerous genetic variants associated with insomnia often overlapping with psychiatric disorders: variants linked to restless legs syndrome clarifying its pathophysiology, and immune-related genes implicated in narcolepsy. In addition, genes influencing sleep duration and chronotype have been identified, offering deeper insights into the genetic mechanisms underlying sleep regulation.

Problem statement

To develop a hybrid GAE-GAT model, which learns dynamically graph structures from sleep microarchitecture and predicts associations between sleep disorders and genetic disorders.

To create a single pipeline that identifies major EEG microarchitecture events, predicts latent relations with GAE, and conducts node classification with GAT for the study of the effect of genetic mutations on sleep disorders.

METHODS

Research design

The overall research pipeline is given in Figure 1.

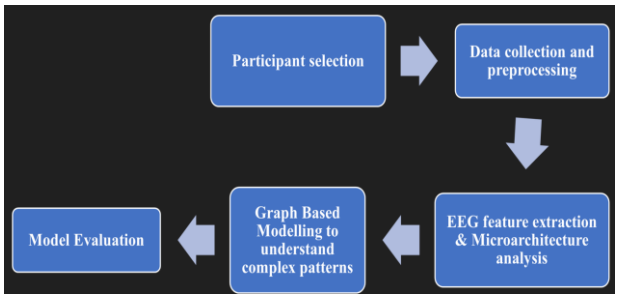

Figure 1: Research design.

Data

A total of 44 participants were selected based on stratified random sampling from six neuro-disorders. Since the availability of the participants was because of the prevalence of the gene-related disorders was low, every disorder was sampled randomly for five subjects and an additional 14 healthy controls for the sake of balance in the NCH dataset.[7,8]

Choice of selection of the EEG channels

According to the 10-20 electrode placement system of AASM, Electrodes are named based on the area of the brain they record from, letters denoting various areas.[9,10] Each area has its own importance, represented in Figure 2.

Since this research is concerned with genetic disorders that impact cognitive processes, decision-making, sensory

integration, and executive functions, EEG C3-M2 and EEG C4-M1 were excluded from analysis based on their correspondence to the Centro-Parietal (CP) area and its role in these functions.[11]

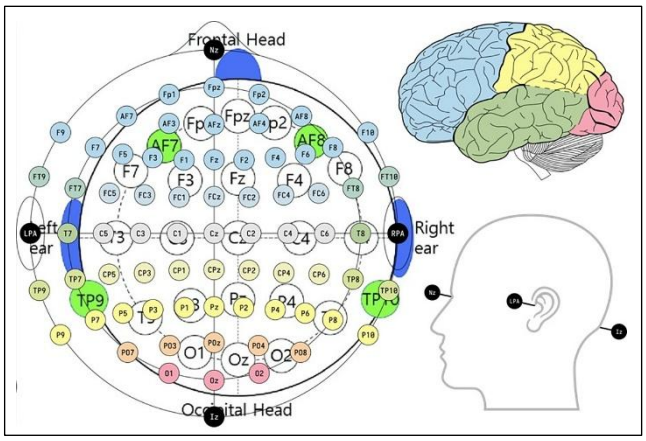

Figure 2: Strategic placement of sensors.[12]

Experimental setup

The experiment was executed in Python 3.12, and dependencies were installed for the model accordingly. Training was done with two RTX 2080-Ti GPUs. In the training procedure, the RAdam optimizer was utilized with a learning rate of 0.001 for 100 epochs. The batch size for both training stages was fixed at 128, and the model dimension (d model) was also fixed at 128. The random seed was set to 42 for the entire training process for both the training processes to achieve reproducibility.

Data preprocessing

Because this research involves EEG bandpower, only EEG channels out of the polysomnographic (PSG) signal data were utilized for preprocessing. To remove noise and keep important frequency components, a bandpass filter 0.3-45 Hz was used in the EEG signals. This ensured that the data obtained stayed within the standard EEG frequency range for use in sleep studies. For every subject, pre-processed EEG signals were segmented into 30-second epochs according to the sampling rate of the dataset. The typical recording rate of NCH data is 256 Hz, and thus all signals were resampled to ensure compatibility across subjects. To calculate the average bandpower for the delta band, an estimate of the power spectral density (PSD) had to be made. This was done using Welch's periodogram, which was accomplished by averaging the consecutive, small overlapping windows of the signal's Fourier transforms.[12] Sample PSD of EEG F3-M2 is given in Figure 3.

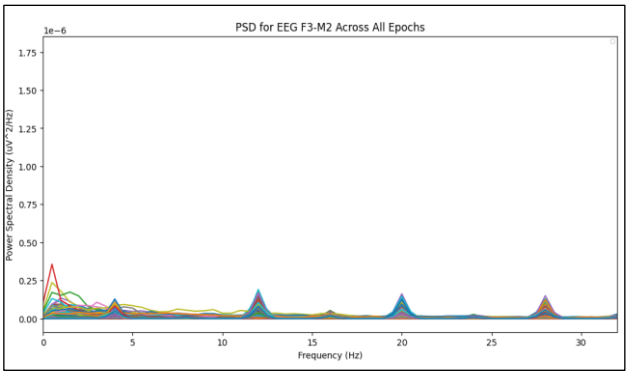

Figure 3: Power spectral density across EEG.

RESULTS

A GAE + GAT model was developed to explore relationships between EEG bandpower, sleep microarchitecture abnormalities, sleep disorders, and rare genetic disorders. By integrating event-level EEG with participant-level data, the model uncovered patterns overlooked by traditional methods. Distinct EEG signatures were identified across sleep disorders, while genotype-specific alterations such as decreased slow-wave activity in Joubert syndrome and spindle density changes in Neurofibromatosis-Noonan syndrome highlighted genetic influences on sleep. Common EEG abnormalities across conditions suggested shared pathways between genetic mutations and sleep disruptions.

The freqs vector contains the x-axis (frequency bins) and the psd vector contains the y-axis (power spectral density)

After the PSD is established, the bandpower can be calculated by integrating the power values within the frequency range of the given band. After the bandpower is established, they are scaled using the

StandardScaler of sklearn. First five rows of the bandpower for the EEG channels are given in Table 1.

Table 1: Sample EEG bandpower data of first participant.

Chan	Delta	Theta	Alpha	Sigma	Beta	Gamma
EEG LOC-M2	0.840	0.119	0.016	0.008	0.012	0.005
EEG F3-M2	0.875	0.097	0.016	0.005	0.005	0.002
EEG F4-M1	0.877	0.097	0.015	0.005	0.005	0.002
EEG C3-M2	0.875	0.104	0.011	0.003	0.005	0.001
EEG C4-M1	0.873	0.107	0.010	0.004	0.005	0.001
EEG O1-M2	0.795	0.156	0.027	0.010	0.010	0.003

Model architecture

Overview of the proposed model

Rather than manually setting the graph structure from pre-specified similarity measures (e.g., cosine similarity), the model uses GAE to predict graph edges dynamically, the GAT subsequently analyzes the created graph to label subjects according to their EEG bandpower patterns. The pipeline includes two main parts: GAT for edge prediction and GAT for classification through attention-based message passing. The schematic design flow is given in Figure 4.

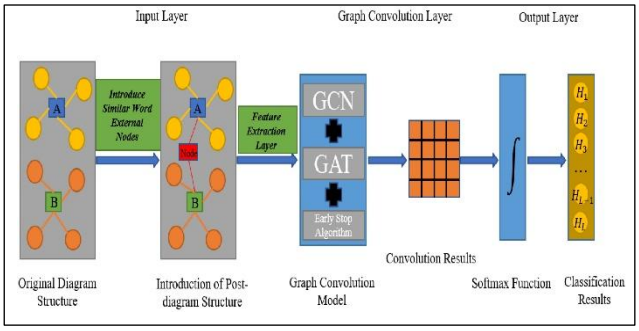

Figure 4: GAE-GAT model architecture.

Training

For the examination of sleep microarchitecture, we identified three important events from EEG bandpower per epoch:

Sleep spindles: Detected employing sigma band (11–16 Hz) with amplitudes higher than a pre-set threshold (75th percentile) and lasting 0.5 to 2 seconds. This is calculated by calculating the moving root mean square (RMS) of the signal over a window size of 0.3 seconds. Spindles are labelled where RMS is higher than the 75th percentile for more than 0.5 to 2 seconds (duration threshold).[13]

Identify spindles with RMS above the 75th percentile for a minimum of 0.5 to 2 seconds (threshold duration).

K-Complexes: Identified in the delta band (<1 Hz) as a large negative peak followed by a positive deflection. This is done using a low-pass filter with cut-off:1 Hz.

Slow waves: Drawn from the delta band (0.5–4 Hz) where thresholds higher than the 80th percentile were designated as significant. The bandpass filter (0.5–4 Hz) is used to pick out the slow-wave components. The peaks lower than the 80th percentile in terms of amplitude are detected as Slow waves.

Training parameters

Training undergoes in three major aspects:

Analysis of sleep microarchitecture variations in sleep disorders.

Analysis of sleep microarchitecture changes in uncommon genetic disorders.

Identifying shared patterns connecting sleep and genetic disorders.

The following are the input features for each participant:

EEG bandpower: Delta, Theta, Alpha, Sigma, Beta, Gamma (over epochs).

Detected sleep events: Density and amplitude of sleep spindles, K-complexes, and slow waves.

Clinical diagnosis: Diagnosed sleep and related genetic condition.

The database consists of EEG bandpower scores for five sleep stages (Wake, N1, N2, N3, REM) and has six genetic conditions. The classification results for the selected 44 participants are given in Tables 2, 3, 4.

Goal 1: Examination of sleep microarchitecture variation between sleep disorders

Each participant is represented as a node with EEG bandpower and sleep event features, while edges are based on feature similarity using cosine distance. The GAE learns latent embeddings by reconstructing graph edges with Binary Cross-Entropy loss and L2 regularization. These embeddings are then used by a GAT to classify sleep disorders using Cross-Entropy loss for multi-class prediction.

Table 2: Goal 1 evaluation metrics.

Sleep disorder	Precision	Recall	F1-Score
Obstructive sleep apnea	0.910	0.880	0.895
Insomnia	0.930	0.923	0.926
Restless leg syndrome	0.930	0.920	0.925
Narcolepsy	0.870	0.850	0.860
Sleep paralysis	0.920	0.890	0.905
Sleep disordered breathing	0.890	0.900	0.895

Average accuracy = 90.1%

Goal 2: Analyzing alterations in sleep microarchitecture in rare genetic disorders

Each participant is modelled as a node with EEG features and detected events, while edges are defined by the clinical co-occurrence of sleep and genetic disorders. The GAE learns embeddings by reconstructing the disorder co-occurrence graph. These embeddings are then used by a

GAT to classify participants based on their genetic disorder.

Table 3: Goal 2 evaluation metrics.

Genetic disorder	Precision	Recall	F1-Score
Dandy-walker syndrome	0.930	0.920	0.925
Di George syndrome	0.880	0.880	0.880
Joubert syndrome (tmem67 mutation)	0.940	0.920	0.930
Intractable Lennox-Gastaut syndrome with status epilepticus	0.890	0.920	0.905
Neurofibromatosis-Noonan syndrome	0.920	0.890	0.905
Chromosomal deletion syndrome	0.910	0.930	0.920
Healthy controls	0.910	0.910	0.934

Average accuracy =91.4%

Goal 3: Discovering shared patterns connecting sleep with genetic disorders

Participants are represented as nodes using EEG and event-based features, with dynamic edges formed based on similarities in EEG patterns and shared sleep or genetic disorders. The GAE learns hidden embeddings to predict new relationships between sleep and genetic disorders, while the GAT uses attention-based message passing to identify participants with similar phenotypic characteristics.

Table 4: Goal 3 evaluation metrics.

Sleep Disorder	Associated genetic disorder	Confidence score
Obstructive sleep apnea	Dandy-Walker syndrome	0.92
Sleep disordered breathing	Di George syndrome	0.87
Narcolepsy	Joubert syndrome (TMEM67 Mutation)	0.89

The overall average Precision, recall and F1 score are given in Table 5.

Table 5: Overall performance.

Metric	Goal 1	Goal 2	Goal 3
Accuracy	90.1%	86.90%	91.4%
Precision	0.908	0.918	0.91
Recall	0.894	0.910	0.89
ROC-AUC (Link Pred.)	-	-	0.92

Link prediction accuracy between the pair of nodes in the graph is computed as: the dot product of. the two embedding vectors. This function is applied to the pair of node embeddings.[13] A higher dot product indicates a higher probability of a link. Figure 5 shows how the similar embeddings are grouped together.

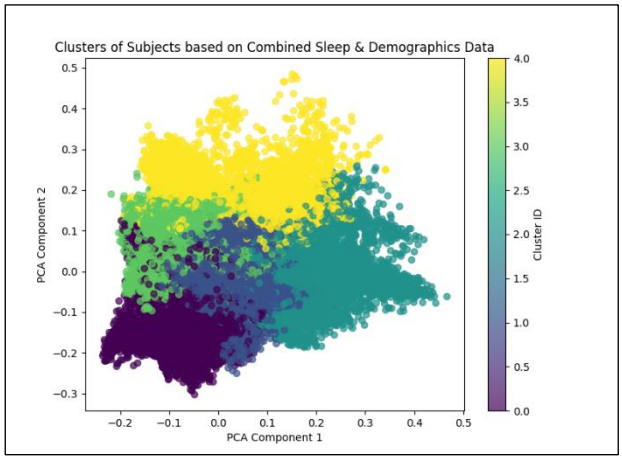

Figure 5: 3D visualization of the node embeddings.

Comparison with other solutions

To begin with, it is particularly commendable that to the best of our knowledge, no work has fully explored how genetic diseases affect sleep microarchitecture as evidenced in the sleep microarchitecture. The available work mainly addresses isolating sleep disorders or genetic syndromes separately without exploring their relationship through neurophysiological signals. Our research fills this gap by studying the common patterns among EEG event abnormalities, sleep disorders, and orphan genetic diseases. It has been previously investigated using EEG biomarkers for sleep disorder diagnosis and graph models for sleep stage estimation, but not in terms of how genetic effects interfere with sleep physiology.[14,15]

DISCUSSION

Literature for this research aggregates studies between 2019 and 2024 into three dimensions of relevance to the research. First, research on the prediction of sleep disorders is discussed, highlighting the use of machine learning and deep learning models for early diagnosis and risk estimation. Second, sleep microarchitecture studies are reviewed, focusing on features such as sleep spindles, K-complexes, and slow waves, their neurophysiological roles, and associations with sleep disorders. Lastly, studies applying GNN in biomedicine are overviewed, with particular emphasis on modelling complex relationships in PSG data and their strengths in disorder classification and individualized sleep analysis.

Related work on sleep disorder classification

Wara et al in their research identifies 183 articles, whose topic of discussion is sleeping disorder classification

through AI. Key findings are the use of brain waves, where convolutional neural networks were the predominant model utilized, with a performance metric as high as 83.75% accuracy.[16] Deep learning techniques such as long short-term memory (LSTM) and temporal convolutional networks (TCN) have demonstrated the ability to learn temporal relationships in sleep data, improving disorder prediction accuracy while making models more explainable through methods such as SHAP for counterfactual explanations, which are essential for clinical decision-making.[17,18] Apart from deep learning techniques, feature selection techniques such as the dipper throated optimization algorithm have been applied to maximize classification performance for the prediction of sleep disorders. Feature set optimization of sleep health and lifestyle data resulted in a minimum average error rate of 0.719 with this approach, while logistic regression yielded 95% accuracy, emphasizing measures of personalized treatment and early detection.[19]

Related work on sleep microarchitecture

A study on chronic insomnia disorder (CID) participants and comorbid major depressive disorder (MDD) highlighted strong associations between cognitive functioning and polysomnography (PSG) scores. Specifically, declarative memory correlated positively with total sleep time (TST), while visuospatial memory negatively correlated with rapid eye movement (REM) sleep latency. Of note, the degree of depression and insomnia was unrelated to cognitive impairment, and changes in sleep architecture may be more relevant to cognitive dysfunction than the severity of these illnesses.5 A survey research of 496 men with MS has found that 90% of the subjects had low sleep quality, and there was a high positive correlation between sleep facilitative behaviours and sleep quality. Regression analysis also indicated that sleep facilitative behaviours and age were predictors that were significant, accounting for 15.2% of the variance in sleep quality scores. However, the male orientation of the study limits the generalizability of the findings to women and individuals with other types of MS.[20]

Related work on association of genetic disorders and sleep

Sleep disorders such as REM sleep behaviour disorder (RBD), insomnia, excessive daytime sleepiness (EDS), and periodic leg movement in sleep (PLMS) have been positively linked with neurodegenerative diseases, with the microarchitecture of sleep being disturbed in affected individuals. For instance, SCA3 participants exhibit decreased sleep efficiency, elevated arousal index, and fragmented sleep compared to normal controls, reflecting a role of important neurodegeneration in sleep disturbances.[21] Epigenetic modifications could also be implicated in the concomitance among sleep, depression, and brain plasticity. The findings corroborate the role of sleep in synaptic plasticity and cognitive processes, particularly during neurodevelopment. In spite of the restriction caused by a small sample, the study provides initial evidence of a relationship between sleep disorders and epigenetic dysregulation of psychiatric and neurodevelopmental disorders.[22]

Limitations and future scope of the study

One of the main limitations of this research is the omission of other biomarkers (e.g., respiratory patterns, heart rate variability) that might yield more accurate associations between sleep abnormalities and genetic disorders. The inclusion of these other biomarkers might enhance the model's capacity to explain the intricate interplay between sleep and genetic factors. Future work will involve extending this method by combining a biofeedback therapy model with GNN and EEG signals.

CONCLUSION

This framework enhances the understanding of how genetic mutations affect sleep architecture, improving diagnostic accuracy and offering novel biomarkers for early detection. The findings support precision sleep medicine by linking genetic markers to specific sleep disturbances, paving the way for personalized interventions and better health outcomes in children with neurodevelopmental and neurodegenerative disorders.

Impact of this research

Early diagnosis and intervention

If genetic disorders interfere with normal sleep patterns and result in sleep disorders, early detection via EEG bandpower classification can provide early diagnosis in children, allowing for timely interventions and better long-term health outcomes.

Precision sleep medicine

By associating genetic markers with particular sleep microarchitecture changes, this work can propel precision medicine, enabling genotype-directed treatment strategies that treat the particular sleep issues of rare genetic disorder participants.

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