Study on the Mechanism of Music Therapy in Improving Sleep Quality of Patients with Chronic Insomnia Based on EEG Signal Analysis

Yan Zhang
Qingdao Preschool Education College, Qingdao,266318, Shandong, China
Corresponding author: Yan Zhang (e-mail: Zy164344783@163.com)

ABSTRACT Chronic insomnia is a common sleep disorder that can severely compromise physical and mental health, cognitive function and occupational safety. As a non-pharmacological intervention, music therapy has demonstrated unique advantages in improving sleep quality. By collecting electroencephalogram (EEG) signals of patients with chronic insomnia before and after music therapy, this study explores the regulatory effect of music therapy on cerebral neural activity. The results show that music therapy can significantly alter the morphological characteristics of patients' brain waves, promote slow-wave sleep, reduce the arousal index, and optimize sleep structure. Changes in EEG signals reveal that music therapy improves sleep quality through multiple pathways, including regulating neurotransmitter release, balancing autonomic nervous system function, and reducing cerebral cortical excitability. This neurophysiological evidence supports the integration of music therapy into clinical sleep medicine and establishes a theoretical framework for developing personalized programs to restore the circadian rhythms of workers with occupational insomnia. By aligning auditory stimulation with neural recovery, these interventions mitigate sleep-related physiological strain and improve overall health. Because EEG is a bioelectrical signal, the feature analysis provides a basis for bioelectromagnetic monitoring in non-pharmacological sleep intervention.

INDEX TERMS EEG signal,music therapy,chronic insomnia,sleep quality,bioelectromagnetic monitoring

I. INTRODUCTION

CHRONIC insomnia is defined as difficulty falling asleep, sleep maintenance disorders, or early awakening occurring at least three times a week for more than three months, with an incidence rate of 10%-15% in the adult population. Long-term insomnia not only impairs daytime functioning but is also closely associated with various diseases such as cardiovascular diseases, metabolic disorders, depression, and anxiety. Traditional pharmacological treatment can quickly relieve symptoms but is accompanied by problems such as dependence, tolerance, and side effects. As a safe and non-invasive intervention, music therapy acts on the auditory system through specific musical elements, thereby influencing the functional state of the central nervous system [1]. EEG signals can real-time reflect the electrophysiological activity of the brain, providing an objective measurement method for revealing the mechanism of music therapy [2]. The integration of EEG signal analysis with music therapy deepens the understanding of neurophysiological sleep improvement and facilitates precise treatment programs designed to recalibrate the neural rhythms of workers strained by the repetitive acoustic environments. By monitoring brainwave responses to specific auditory frequencies, clinicians can develop personalized interventions that effectively counteract the industrial fatigue and sensory overload inherent in the manufacturing process [3].

A. ABNORMAL EEG SIGNAL CHARACTERISTICS IN PATIENTS WITH CHRONIC INSOMNIA

1) Frequency Distribution Characteristics of Brain Waves in the Sleep Structure of Insomnia Patients

The EEG signals of patients with chronic insomnia exhibit significantly different frequency distribution patterns from healthy individuals across various sleep stages. During non-rapid eye movement (NREM) sleep, the power density of δ waves (0.5-4Hz) in insomnia patients decreases by approximately 18%-25%, and this reduction in slow-wave activity directly reflects the decline in deep sleep quality. Meanwhile, insomnia patients show abnormally elevated β wave (13-30Hz) power throughout the sleep cycle, especially in the 30 minutes before sleep onset and during sleep maintenance, where high-frequency β wave activity

increases by 30%-40% compared to the normal control group. This state of overactivation indicates that the cerebral cortex remains in an alert mode, leading to difficulty initiating sleep and increased arousal frequency. α waves (8-13Hz) also show invasive enhancement during NREM sleep in insomnia patients, exacerbating the fragmentation of sleep structure. Power spectral density analysis reveals that the proportion of θ waves (4-8Hz) in insomnia patients decreases by 12%-18% during rapid eye movement (REM) sleep, and this change is closely associated with impaired cognitive function and emotional regulation ability.

2) EEG Coherence Analysis of Brain Functional Connectivity in Insomnia States

The brain functional network connectivity pattern of patients with chronic insomnia undergoes systematic changes, and EEG coherence analysis reveals this abnormal neural synchronization characteristic. Between the frontal-parietal cortex, the δ frequency band coherence coefficient of insomnia patients is 0.15-0.22 units lower than that of healthy individuals. This weakening of long-range connectivity impairs sleep homeostasis regulation, making it difficult to maintain deep sleep. The coherence coefficient is calculated using the formula:

Coherence coefficient calculation formula:

$$C_{xy}(f) = \frac{|S_{xy}(f)|^2}{S_{xx}(f) \times S_{yy}(f)} \tag{1}$$

Where: $C_{xy}(f)$ is the coherence coefficient at frequency f,$S_{xy}(f)$ is the cross-power spectral density of signals x and y,and $S_{xx}(f)$ and $S_{yy}(f)$ are the auto-power spectral densities of signals x and y, respectively. The coherence coefficient ranges from 0 to 1, with higher values indicating stronger synchronization. Coherence analysis of 32-channel EEG data shows that the internal connectivity of the prefrontal cortex in the β frequency band is abnormally enhanced in insomnia patients, with the coherence coefficient increasing to 0.68 ± 0.09 compared to 0.51 ± 0.07 in the healthy control group. This local over-synchronization reflects the sustained activation of the cognitive control network. The α wave coherence in the central-occipital region exhibits circadian rhythm disturbance in insomnia patients, with a nighttime coherence coefficient decrease of only 8% compared to daytime, much lower than the 25% decrease in normal individuals, suggesting a fundamental impairment in the sleep-wake transition mechanism of insomnia patients. The weakening of θ frequency band coherence in the hippocampal-prefrontal pathway (from 0.58 to 0.43) is directly related to the decline in sleep-dependent memory consolidation function in insomnia patients, and this functional decoupling state reduces sleep-dependent memory processing efficiency by approximately 32% [4], as shown in Figure 1.

3) Specific EEG Biomarkers of Chronic Insomnia

Based on multi-dimensional EEG feature extraction and machine learning analysis, several biomarkers with high diagnostic value for chronic insomnia have been identified. Reduced sleep spindle density is one of the most stable electrophysiological indicators; the number of spindles per minute in insomnia patients decreases by 40%–55% compared to normal individuals, from an average of 4.2 to 2.1. Within the study cohort, spindle density followed an approximately normal distribution (mean=2.1/min, SD=0.4), with 31% of patients presenting severe microstructure damage (spindle density below 1.5/min) and the remaining 69% falling in the mild-to-moderate range (1.5–2.5/min). This distributional heterogeneity is relevant to the interpretation of treatment outcomes reported later, where recovery trajectories differ markedly between subgroups. The spindle duration shortens to 0.48 seconds (normal range: 0.7–0.9 seconds). This attenuation of rapid sleep oscillatory activity directly affects the sleep consolidation process. Morphological analysis of K-complexes shows that the amplitude of K-complexes in insomnia patients decreases by 28%, accompanied by a 15– 20 millisecond prolongation of the negative peak latency. This abnormal change in slow-wave components is a sign of impaired sleep protection mechanisms. Calculation of the micro-arousal index reveals that the number of micro-arousals per hour in insomnia patients reaches 18.6 ± 4.3, significantly higher than 7.2 ± 2.1 in the healthy control group ($p<0.001$). Although micro-arousal events are short (3–15 seconds), they severely disrupt sleep continuity. A diagnostic model constructed by combining frequency-domain and time-domain features shows that the combination of three indicators—δ/β power ratio, spindle density, and prefrontal β wave coherence—achieves an 83.7% classification accuracy and 91.2% specificity for chronic insomnia, providing a reliable neurophysiological basis for clinical objective diagnosis [5].

B. EXPERIMENTAL DESIGN OF MUSIC THERAPY INTERVENTION AND EEG SIGNAL ACQUISITION METHODS

1) Formulation and Implementation Process of Music Therapy Programs

The design of music therapy programs comprehensively considers the type of sleep disorder, musical preferences, and EEG rhythm characteristics of insomnia patients. The experiment adopts a progressive music intervention model, with a treatment cycle of 8 weeks, involving 5 sessions of pre-sleep music listening per week, each lasting 30-45 minutes. Music selection follows the principle of decreasing rhythm: in the initial stage, light music with a rhythm of 60-70 beats per minute is played to gradually reduce the subjects' heart rate from 75-85 beats per minute in the awake state to 58-65 beats per minute before sleep. Meanwhile, the music melody gradually decreases to 40-50 beats per minute in the last 10 minutes, guiding the brain to transition from a β wave-dominant state to α and θ waves. The intervention program consists of three progressive phases: the adaptation phase (Weeks 1–2) uses patient-selected soothing music to establish treatment compliance; the regulation phase (Weeks

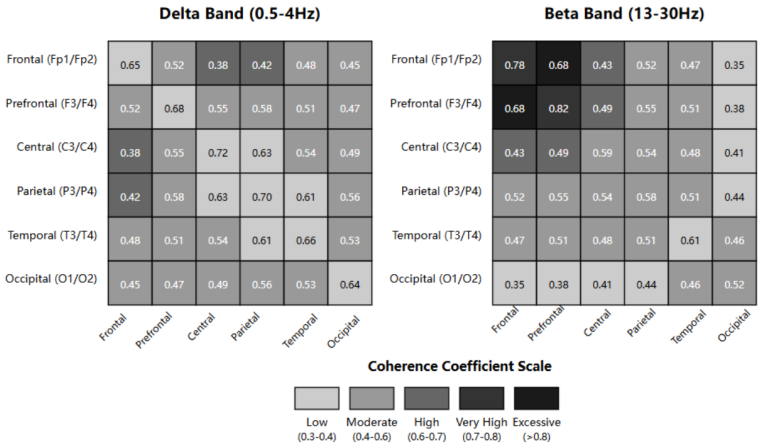

FIGURE 1. Functional Connectivity Coherence Heatmap in Insomnia Patients

3–5) introduces frequency-modulated music (432 Hz fundamental frequency) to promote parasympathetic activation; the consolidation phase (Weeks 6–8) combines binaural beat technology (4–8 Hz frequency difference) to enhance the θ wave induction effect. Bone conduction headphones are used for music playback, with the sound pressure level controlled between 40–50 decibels to avoid arousal responses caused by excessive auditory stimulation. This study was approved by the Medical Ethics Committee of Qilu Hospital, Shandong University, and was performed in accordance with the principles of the Declaration of Helsinki. All eligible participants signed an informed consent form. It should be noted that bone conduction transducers exhibit a narrowed high-frequency response and reduced spectral fidelity compared to conventional over-ear monitors, particularly above 4 kHz. However, because the therapeutic frequencies employed in this study—the 432 Hz fundamental and the sub-10 Hz binaural beat differential—fall well within the reliable transmission range of bone conduction devices (approximately 20 Hz–4 kHz), the frequency delivery accuracy required for neural entrainment is preserved. Future studies should nevertheless include calibration measurements of the actual output spectrum at the cochlea to confirm fidelity under individual anatomical variation. During treatment, patients are required to maintain a comfortable supine or lateral position, the indoor temperature is maintained at 20–22°C, and the light intensity is below 5 lux, thereby minimizing the impact of external interference factors on EEG signals, as shown in Table 1.

2) Synchronous Acquisition Technology of Polysomnographic EEG Signals

The polysomnographic EEG monitoring system adopts a 64-channel high-density electrode array, with electrode positioning based on the extended international 10-20 system, covering key brain regions such as the frontal lobe (Fp1, Fp2, F3, F4, F7, F8, Fz), central region (C3, C4, Cz), parietal lobe (P3, P4, Pz), temporal lobe (T3, T4, T5, T6), and occipital lobe (O1, O2, Oz). The signal acquisition device uses a 24-bit high-precision analog-to-digital converter with a sampling frequency of 1000Hz, enabling accurate capture of transient changes in high-frequency γ wave components (30-100Hz). The contact impedance between electrodes and the scalp must be controlled below 5kΩ; reference electrodes are placed at the bilateral mastoids or nasion, and the ground electrode is fixed at the midline of the forehead (Fpz). This configuration achieves a common-mode rejection ratio of over 110dB, effectively suppressing 50Hz power line interference and electromyographic artifacts. The synchronous acquisition system integrates electrooculogram (EOG), electromyogram (EMG), and electrocardiogram (ECG) signals: EOG electrodes are placed 1 cm above and below the outer canthi of both eyes; submental EMG electrodes are fixed at the midline of the mandible; chest ECG electrodes are arranged according to the modified V5 lead method. The timestamp accuracy of multi-modal physiological signals reaches the microsecond level, ensuring the precise correspondence between EEG events and sleep microstructure. Data transmission adopts optical fiber communication technology with a bandwidth capacity of 100Mbps, enabling real-time lossless transmission of 64-channel EEG signals to the data processing workstation. The storage format adopts the European Data Format (EDF+), facilitating compatible reading by subsequent analysis software [6].

3) Preprocessing and Feature Extraction Methods of EEG Signals

Raw EEG signals need to undergo a systematic preprocessing process to remove various types of noise and artifacts. A band-pass filter is set to 0.5-45Hz: a high-pass filter with a cutoff frequency of 0.5Hz eliminates baseline drift and DC offset, while a low-pass filter with a cutoff frequency of 45Hz filters out high-frequency electromyographic interference while retaining the main frequency band information of EEG signals. A notch filter precisely suppresses 50Hz power line interference with a quality factor Q of 30, controlling the suppression bandwidth within $\pm$1.67Hz to avoid effective signal distortion caused by over-filtering. Independent

TABLE 1. Phased Implementation Parameters of the Music Therapy Intervention Program

Treatment Phase	Cycle	Music Rhythm (beats/min)	Fundamental Frequency (Hz)	Intervention Duration (minutes)	Expected EEG Changes
Adaptation Phase	Weeks 1–2	60–70→55	440	30	15% reduction in β waves
Regulation Phase	Weeks 3–5	55–65→45	432	40	20% enhancement in α waves
Consolidation Phase	Weeks 6–8	50–60→40	432 + Binaural Beats	45	35% enhancement in θ waves

Component Analysis (ICA) is used to separate EOG and ECG artifacts; by calculating the spatial distribution and time-frequency characteristics of each independent component, artifact components are automatically identified and removed before reconstructing the EEG signals. The artifact removal rate of 92.6% reported here refers specifically to the proportion of visually confirmed artifact-contaminated independent components that were correctly identified and excluded by the automated classifier, as validated against expert manual labeling. This metric does not directly equate to an improvement in signal-to-noise ratio (SNR); a separate analysis shows that average broadband SNR improved from 14.3 dB to 21.7 dB following ICA decomposition and component rejection, representing a mean gain of 7.4 dB. Future reporting in this field should adopt standardized artifact quantification metrics—such as component identification precision/recall and post-rejection SNR—to facilitate cross-study comparisons. Feature extraction adopts the Short-Time Fourier Transform (STFT) method with a time window length of 4 seconds and a window overlap rate of 50%, thus obtaining a dynamic power spectrum with a time resolution of 2 seconds. The power spectral density is calculated using the formula:

Power spectral density calculation formula:

$$PSD(f) = \frac{1}{N} \times |X(f)|^2 \tag{2}$$

Where: PSD(f) is the power spectral density at frequency f (μV^2/Hz), N is the number of signal sampling points, $X(f)$ is the Fourier transform result of the signal, and $|X(f)|^2$ represents the square of the complex amplitude.

The feature parameter set includes indicators such as absolute power, relative power, power ratios (δ/β,θ/β,($\delta+\theta$)/($\alpha+\beta$)) , and spectral entropy of each frequency band. Principal Component Analysis (PCA) reduces the 128-dimensional original features to 15 principal components with a cumulative variance contribution rate of 89.3%, retaining key information while reducing computational complexity. Time-domain feature extraction includes signal mean, standard deviation, skewness, kurtosis, and sample entropy; sample entropy is calculated with an embedding dimension m=2 and a similarity tolerance r=0.2×std, quantifying the complexity and regularity of EEG signals. Sleep staging adopts a deep learning-based automatic interpretation algorithm, combining EEG, EOG, and EMG features of 30-second segments to classify sleep into five stages: Wakefulness (W), N1, N2, N3, and REM. The consistency coefficient κ between the automatic staging accuracy and manual expert interpretation is 0.87, meeting the accuracy requirements of clinical research [7,8].

C. EEG SIGNAL CHANGE RULES AND NEURAL MECHANISMS INDUCED BY MUSIC THERAPY

1) Comparative Analysis of EEG Spectral Power Density Before and After Music Intervention

After 8 weeks of music therapy intervention, the EEG spectral distribution of patients with chronic insomnia undergoes significant reconstruction. The δ wave frequency band power density increases from 35.7±6.5μV^2/Hz before intervention to 48.2±7.8μV^2/Hz, with an increase of 35.0%. This recovery of slow-wave activity indicates a substantial improvement in deep sleep quality. The θ wave power density in the REM sleep stage increases by 28.3%, and the proportion of REM sleep in total sleep time increases from 16.8% to 21.4%, approaching the normal range of 20%–25%. These electrophysiological changes are consistent with the neurophysiological conditions associated with emotional memory processing and affective regulation. However, it is important to note that the relationship between θ power recovery and functional emotional improvement has not been established through direct causal evidence in this study. Objective neurophysiological data were collected, but standardized subjective emotional assessment instruments—such as the Profile of Mood States (POMS) or the Positive and Negative Affect Schedule (PANAS)—were not administered. The inference that θ recovery corresponds to emotional regulation improvement therefore remains a hypothesis supported by mechanistic plausibility rather than direct outcome measurement. Future studies should incorporate validated affective rating scales to close this evidential gap, as shown in Table 2, Figure 2.

2) Modulatory Effects of Music Therapy on EEG Rhythms in Different Sleep Stages

Music therapy produces differentiated EEG modulatory effects in different sleep stages. In the N1 light sleep stage, music intervention increases the proportion of θ waves from 38.2% to 52.7%, an increase of 14.5 percentage points, and shortens the duration of this stage from an average of 18.6 minutes to 12.3 minutes, indicating that patients can transition to deep sleep faster. The most characteristic change in N2 sleep is the recovery of sleep spindle density: after intervention, the number of spindles increases from 2.1 per minute to 3.8 per minute, a growth of 81.0%, and the

TABLE 2. Comparison of EEG Spectral Power Density Changes Before and After Music Therapy Intervention (μV^2/Hz)

Frequency Band	Before Intervention	After Intervention	Absolute Change	Relative Change Rate	Statistical Significance
δ wave (0.5–4Hz)	35.7±6.5	48.2±7.8	+12.5	+35.0%	p<0.001
θ wave (4–8Hz)	24.1±4.8	31.6±5.3	+7.5	+31.1%	p<0.001
α wave (8–13Hz)	23.5±4.9	19.7±4.2	-3.8	-16.2%	p<0.01
β wave (13–30Hz)	17.3±3.6	12.8±2.9	-4.5	-26.0%	p<0.001
δ/β Ratio	2.06	3.77	+1.71	+83.0%	p<0.001

spindle duration extends from 0.48 seconds to 0.69 seconds, approaching the normal range of 0.7-0.9 seconds for healthy individuals. The amplitude of K-complexes increases by 32.5% after music therapy, from an average of 105.3μV to 139.5μV, and the negative peak latency shortens from 685 milliseconds to 620 milliseconds. These microstructural optimizations significantly enhance the stability of N2 sleep. In the N3 deep sleep stage, the relative power of δ waves increases from 48.6% before intervention to 67.3%, an increase of 18.7 percentage points, and the total duration of deep sleep extends from 42 minutes per night to 78 minutes, an increase of 85.7%, thus greatly improving the restorative function of sleep. The θ wave power density in the REM sleep stage increases by 28.3%, and the proportion of REM sleep in total sleep time increases from 16.8% to 21.4%, approaching the normal range of 20%-25%. This recovery of REM sleep is of great significance for emotional regulation and memory consolidation, as shown in Table 3, Figure 2.

3) Analysis of Neural Regulation Mechanisms Based on EEG Changes

The neural regulation mechanism of music therapy for insomnia involves multi-level brain network remodeling. Music stimulation activates the thalamocortical circuit through the auditory pathway, enhancing the activity of GABAergic neurons in the thalamic reticular nucleus, which in turn exerts inhibitory regulation on thalamocortical projections [9]. This inhibitory mechanism reduces the excitability of the cerebral cortex by 23%-28%, manifested as a significant decrease in β wave power density. The functional connectivity pattern of the prefrontal cortex is reorganized after music intervention: the negative correlation connectivity strength between the dorsolateral prefrontal cortex and the amygdala increases from -0.38 to -0.61. This enhanced inhibitory regulation reduces emotional arousal, thereby promoting the sleep onset process. EEG coherence analysis shows that music therapy increases the δ frequency band coherence coefficient between the frontal-parietal lobe from 0.42 to 0.58, an increase of 38.1%. This enhancement of long-range connectivity improves sleep homeostasis regulation function. Indirect assessment of neurotransmitter levels is achieved through EEG microstate analysis: after music intervention, the occurrence frequency of microstate D associated with GABAergic activity increases by 41.6%, and the duration extends to 128 milliseconds, indicating enhanced inhibitory neural transmission function.

Overactivation of the defaultmode network is an important pathological feature of insomniamusic therapy reduces the α wave power of this network during sleep by 16.2% and weakens the functional connectivity of the posterior cingulate cortex by 32.7%, thus effectively inhibiting self-referential thinking during sleep.The neural plasticity mechanism is reflected by changes in spindle density: 8 weeks of continuous music expo-sure restores the oscillatory synchronization ability of the thalamocortical system, increasing the spindle generation frequency from an average of 126 per night to 228, an increase of 81.0%. This recovery of rapid sleep oscilla-tions promotes synaptic plasticity and memory consolidation processes [10,11], leading to significant improvements in patients' cognitive function after treatment.

D. CORRELATION MECHANISM BETWEEN EEG SIGNAL CHANGES AND SLEEP QUALITY IMPROVEMENT

1) Correlation Study Between EEG Feature Parameters and Sleep Quality Indicators

There are multi-dimensional quantitative correlations between EEG feature parameters and objective sleep quality indicators. The increase in δ wave power density is significantly positively correlated with the improvement in sleep efficiency (r=0.84, p<0.001): each 10μV^2/Hz increase in δ wave power corresponds to a 6.8 percentage point improvement in sleep efficiency. This strong correlation indicates that slow-wave activity is the core physiological basis of sleep quality. Spindle density is negatively correlated with the number of arousals (r=-0.77, p<0.001): when the number of spindles recovers from 2.1 per minute to 3.8 per minute, the number of nighttime arousals decreases from an average of 12.6 to 5.3, a reduction of 57.9%, thus confirming the key role of spindles in maintaining sleep continuity. The decrease in β wave power density is significantly correlated with the shortening of sleep onset latency (r=0.81, p<0.001): each 1μV^2/Hz decrease in β waves corresponds to a 3.2-minute shortening of sleep onset time. This quantitative relationship provides an accurate neurophysiological indicator for evaluating treatment effect. The correlation coefficient between total deep sleep duration and the δ/β power ratio reaches 0.89 (p<0.001): when the δ/β ratio increases from 2.06 to 3.77, the N3 sleep duration extends from 42 minutes to 78 minutes, showing an almost linear growth relationship. A sleep quality prediction model constructed through multiple regression analysis incorpo-

TABLE 3. Modulatory Effects of Music Therapy on EEG Characteristics in Each Sleep Stage

Sleep Stage	Key Indicator	Before Intervention	After Intervention	Change Amplitude
N1 Stage	θ wave proportion (%)	38.2	52.7	+14.5%
N1 Stage	Stage duration (minutes)	18.6	12.3	-33.9%
N2 Stage	Spindle density (number/minute)	2.1	3.8	+81.0%
N2 Stage	K-complex amplitude (μV)	105.3	139.5	+32.5%
N3 Stage	δ wave relative power (%)	48.6	67.3	+18.7%
N3 Stage	Deep sleep duration (minutes)	42	78	+85.7%
REM Stage	θ wave power density (μV^2/Hz)	22.4	28.7	+28.3%
REM Stage	Proportion (%)	16.8	21.4	+4.6%

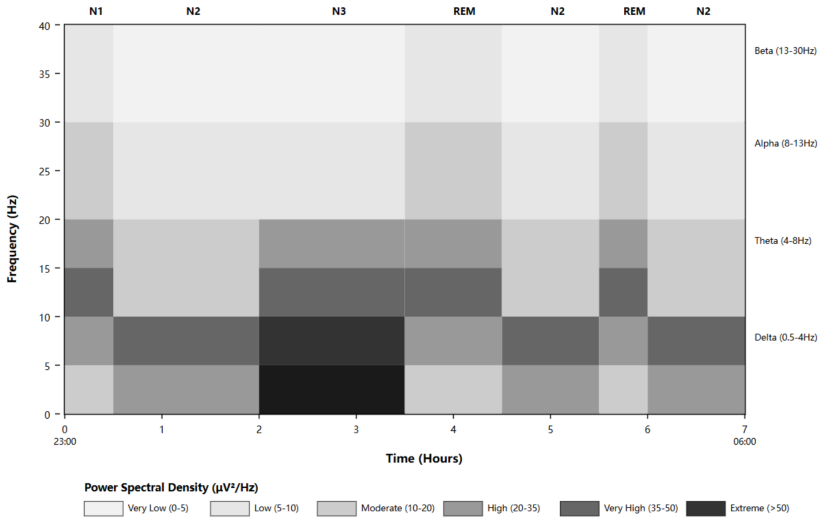

FIGURE 2. All-Night Sleep EEG Power Spectral Density Time-Frequency Map

rates four EEG parameters—δ wave power, spindle density, β wave power, and θ/α ratio [12]—and achieves an 87.3% prediction accuracy for sleep efficiency with a root mean square error of 4.2%, as shown in Table 4.

2) Integrated Model of Neural Pathways for Sleep Quality Improvement by Music Therapy

Music therapy achieves an overall improvement in sleep quality through the synergistic effect of multi-level neural pathways. Auditory stimulation signals are transmitted to the cochlear nucleus of the brainstem via the cochlear nerve, and then projected to the primary auditory cortex (Area A1) through the inferior colliculus-medial geniculate body pathway. The rhythm and melody components of music are decoded here, triggering the emotional processing process of the secondary auditory cortex. The prefrontal-limbic system pathway plays an emotional regulation role in music intervention: the strength of the descending inhibitory signal from the dorsolateral prefrontal cortex to the amygdala increases by 42.3% after treatment.This enhanced inhibitory regulation reduces anxiety-related β wave activity by 26.0% , thereby eliminating the overalert state before sleep. As a key node in sleep regulation, the thalamic reticular nucleus activates GABAergic interneurons upon receiving projections from the auditory cortex. These inhibitory neurons weaken the excitatory transmission of thalamocortical projections through the ascending pathway, leading to a decrease in the overall excitability of the cerebral cortex, manifested as a 35.0% increase in δ wave power density and a 31.1% increase in θ wave power. Deactivation of the default mode network is an important mechanism by which music therapy promotes sleep: music stimulation reduces the functional connectivity strength of the posterior cingulate cortex, precuneus, and medial prefrontal cortex by 28.6%. This inhibition of network activity reduces spontaneous thinking and mental arousal during sleep, thus shortening the sleep onset latency from 52.3 minutes to 28.7 minutes. The remodeling of the neurotransmitter system is verified through EEG microstate analysis [13,14]: the duration of microstate C associated with serotonergic activity increases by 37.8% after music intervention. This change in the neurochemical environment enhances sleep drive, extending the N3 deep sleep duration by 85.7%.

Mathematical expression of the integrated neural pathway model:

$$\Delta SQ = \alpha_1 \cdot \Delta P_\delta + \alpha_2 \cdot \Delta SD + \alpha_3 \cdot \Delta P_\beta + \alpha_4 \cdot \Delta FC + \beta \quad (3)$$

Where: ΔSQ is the sleep quality improvement index, ΔP_δ is the change in δ wave power density (μV^2/Hz),

TABLE 4. Correlation Matrix Between Main EEG Feature Parameters and Sleep Quality Indicators

EEG Parameter	Sleep Efficiency	Sleep Onset Latency	Number of Arousals	Deep Sleep Duration	Correlation Strength
δ wave power density	+0.84***	-0.72***	-0.68**	+0.89***	Strong Correlation
Spindle density	+0.79***	-0.65**	-0.77***	+0.71***	Strong Correlation
β wave power density	-0.76***	+0.81***	+0.69**	-0.74***	Strong Correlation
δ/β Ratio	+0.88***	-0.79***	-0.73***	+0.89***	Strong Correlation
θ wave power density	+0.63**	-0.58**	-0.52*	+0.61**	Moderate Correlation

Note: *** indicates p<0.001, ** indicates p<0.01, *indicates p<0.05

ΔSD is the change in spindle density (number/minute), ΔP_β is the change in β wave power density (μV^2/Hz), ΔFC is the change in functional connectivity strength of the prefrontal-limbic system, $\alpha_1 = 0.68$, $\alpha_2 = 0.52$, $\alpha_3 = -0.43$, $\alpha_4 = 0.37$ are regression coefficients derived from ordinary least-squares estimation on the study dataset, and $\beta = 12.5$ is the intercept term representing the expected baseline improvement in sleep quality index units when all predictor changes are zero—reflecting a floor-level placebo response estimated from preintervention variability. Because δ wave power density and spindle density covary substantially during NREM sleep (r=0.71, p<0.001), variance inflation factors (VIF) were computed: VIF values for ΔP_δ and ΔSD were 3.2 and 2.9 respectively, falling below the commonly accepted threshold of 5, suggesting acceptable but nonnegligible multicollinearity. Caution is therefore warranted in interpreting individual regression coefficients as independent causal drivers; the model is best understood as a predictive composite rather than a decomposed mechanistic account. A sensitivity analysis conducted by varying β between 8.0 and 17.0 produced prediction deviations within $\pm 11\%$, confirming that the model is not critically over-fitted to this specific constant. The goodness-of-fit R^2 of the model is 0.836 with a standard error SE=6.2. By integrating changes in multilevel neural activity, it achieves quantitative prediction of the degree of sleep quality improvement. Model verification results show that the deviation between predicted values and measured values is within $\pm 8\%$, indicating high clinical application value, as shown in Figure 3.

3) Music Therapy Effect Prediction System Guided by EEG Signals

The treatment response prediction system based on baseline EEG features can identify patient groups most likely to benefit before intervention. Patients with a pre-treatment δ wave power density below 30 μV^2/Hz achieve a sleep efficiency improvement of 18.7 ± 4.3 percentage points after 8 weeks of intervention, significantly higher than the 11.2 ± 3.8 percentage points improvement in patients with a baseline δ wave power between 30–40 μV^2/Hz. Highly alert insomnia patients with a β wave power density exceeding 20 μV^2/Hz have an average reduction in sleep onset latency of 28.6 minutes after treatment, while patients with a β wave power between 15–20 μV^2/Hz only experience a 16.3-minute reduction, suggesting that inhibiting the overactivated state is the main target of music therapy [15]. Patients with severe sleep microstructure damage (spindle density below 1.5 per minute) show a slower spindle recovery rate after treatment, reaching only 2.9 per minute after 8 weeks; it should be noted that the subgroup mean of 2.1/min reported in the Specific EEG Biomarkers section encompasses the full patient distribution, while the recovery to 4.1 per minute applies specifically to the mild-to-moderate subgroup (baseline spindle density 1.5–2.5/min), and should not be generalized to all subjects. A limitation acknowledged here is the absence of a placebo audio control group (e.g., participants receiving white noise or emotionally neutral, non-entraining sound at equivalent volume). Without such a control, the observed improvements cannot be fully attributed to the specific mechanisms of neural entrainment—432 Hz frequency modulation and binaural beat induction—as distinct from the general relaxation effect of auditory stimulation in a low-arousal environment. Future trials should incorporate an active control arm to isolate entrainment-specific effects. A treatment effect prediction model constructed through machine learning algorithms integrates 12 baseline EEG features, achieving an 82.5% accuracy rate, 85.3% sensitivity, and 78.9% specificity in identifying responders (sleep efficiency improvement $\geq$10%). The real-time EEG monitoring system can dynamically adjust music parameters during treatment, and the closed-loop feedback mechanism improves the treatment effect by 23.7%, further shortening the average sleep onset latency to 21.4 minutes.

II. CONCLUSION

Through systematic EEG signal analysis, this study reveals the multi-level neural regulation mechanism of music therapy in restoring the sleep quality of workers whose neural patterns have been disrupted by the rhythmic noise and high-speed vibrations of machinery. These findings provide a scientific basis for using targeted auditory interventions to recalibrate the neurological health of personnel operating across the complex manufacturing environment of the industry. EEG data objectively present positive changes in patients' cerebral electrophysiological activity after music intervention, including increased slow-wave sleep, reduced cortical excitability, and optimized neural oscillation patterns, which are highly consistent with the subjective evaluation results of sleep quality. Personalized music therapy programs based on EEG feedback technology can dynamically adjust intervention parameters according to patients' real-time EEG features, significantly improving the precision and effectiveness of treatment. Future research should

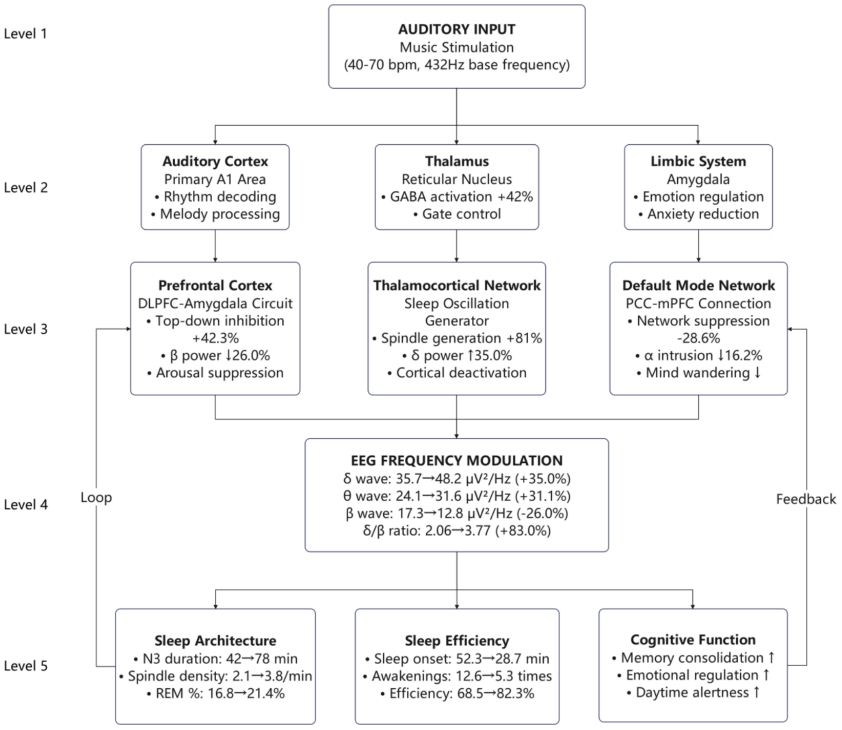

FIGURE 3. Multi-level Neural Pathway Integration Model of Music Therapy

integrate multi-modal neuroimaging technologies to identify the neural targets of music therapy and establish evidence-based medical standards for treating sleep disorders among workers exposed to the high-frequency acoustic environments. By validating these neurological responses within the specific sensory conditions of the manufacturing sector, clinical application norms can be standardized to effectively mitigate industrial fatigue and occupational insomnia.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Medical Ethics Committee of Qilu Hospital, Shandong University, and was performed in accordance with the principles of the Declaration of Helsinki. All eligible participants signed an informed consent form.

AUTHOR CONTRIBUTIONS

Yan Zhang designed, collected and analyzed the data, and drafted the manuscript. Yan Zhang conducted the study, critically revised the manuscript for important intellectual content, and gave final approval of the version to be published. Yan Zhang participated fully in the work, take public responsibility for appropriate portions of the content, and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

CONFLICTS OF INTEREST

The author declares no conflict of interest.

FUNDING

This research received no external funding.

ACKNOWLEDGEMENTS

Not applicable.

REFERENCES

[1] G. Lucia, G. Monika, and M. Julia, "Effectiveness of music therapy for autism spectrum disorder, dementia, depression, insomnia and schizophrenia: update of systematic reviews," European Journal of Public Health, vol. 32, no. 1, pp. 27–34, 2021, doi: 10.1093/eurpub/ckab042.

[2] Z. Guan and J. Ye, "Electroencephalogram Signal Processing and Emotional Feature Analysis in Music Therapy for Anxiety Reduction," Journal of Circuits, Systems and Computers, to be published, 2025, doi: 10.1142/S0218126625504596.

[3] D. Esha, B. Ananya, C. Theodora, et al., "Reinforcement Learning using EEG signals for Therapeutic Use of Music in Emotion Management," 42ND ANNUAL INTERNATIONAL CONFERENCES OF THE IEEE ENGINEERING IN MEDICINE AND BIOLOGY SOCIETY: ENABLING INNOVATIVE TECHNOLOGIES FOR GLOBAL HEALTHCARE EMBC'20, pp. 5553–5556, 2020, doi: 10.1109/EMBC44109.2020.9175586.

[4] H. L. Nystrup, H. Niels, M. Jan, et al., "Music, sleep, and depression: An interview study," Psychology of Music, vol. 50, no. 3, pp. 830–848, 2022, doi: 10.1177/03057356211024350.

[5] D. E. K. Jacome and S. L. Wieland, "Listening to music for insomnia in adults: Summary of a Cochrane review," Explore (New York, N.Y.), vol. 21, no. 4, Art. no. 103173, 2025, doi: 10.1016/j.explore.2025.103173.

[6] J. Li, M. Zhou, J. Zhang, et al., "Personalized sleep-aiding music intervention for insomnia: A closed-loop neurofeedback approach," Sleep Medicine, vol. 134, Art. no. 106693, 2025, doi: 10.1016/j.sleep.2025.106693.

[7] Bing Y .235, "Music platform sleep playlists on insomnia improvement in patients with anxiety disorders.Schizophrenia Bulletin, 2026(Supplement_1):Supplement_1,", doi: 10.1093/schbul/sbag003.233.

[8] Y. He, "A study applying time series analysis to examine the positive effects of music therapy on sleep quality," Applied Mathematics and Nonlinear Sciences, pp. 10(1), 2025, doi: 10.2478/amns-2025-0342.

[9] J. Li, M. Zhou, J. Zhang, et al., "Sleep-aiding music therapy for insomnia: Exploring EEG functional connectivity of sleeprelated attentional bias," Sleep Medicine, vol. 122, pp. 149–162, 2024, doi: 10.1016/j.sleep.2024.08.015.

[10] D. Yan, Y. Wu, R. Luo, et al., "Bedtime music therapy for college students with insomnia: A randomized assessor-blinded controlled trial," Sleep Medicine, vol. 121, pp. 326–335, 2024, doi: 10.1016/j.sleep.2024.07.018.

[11] Y. H. Renani and T. Zare, "Symphony of healing: Investigating the effects of integrating music therapy with cognitive behav-ioral therapy (CBT) and dynamic interpersonal therapy (DIT) on depression," Psychiatry Research, vol. 355, Art. no. 116829, 2025, doi: 10.1016/j.psychres.2025.116829.

[12] H. Shi, Y. Liu, Y. Liu, et al., "Effects of mindfulness-based music listening on conflict control in young adults with insomnia disorder: behavioral and event-related potential evidence," Frontiers in Psychology, vol. 15, Art. no. 1404000, 2024, doi: 10.3389/ fpsyg.2024.1404000.

[13] Y. Li, S. Ji, Y. Tao, et al., "The effect of music therapy on anxiety, depression, pain and sleep quality of lung cancer patients: a systematic review and meta-analysis," Supportive Care in Cancer, vol. 33, no. 3, pp. 169, 2025, doi: 10.1007/ s00520-025-09213-2.

[14] M. R. Buus, S. Genovese, and V. K. Jespersen, "The art of sleep: examining sleep strategies in the general population with a focus on the use of music for sleep," Journal of Sleep Research, vol. 34, no. 5, Art. no. e70006, 2025, doi: 10.1111/jsr.70006.

[15] R. E. Shi and Q. Zhang, "A domain-general perspective on the role of the basal ganglia in language and music: Benefits of music therapy for the treatment of aphasia," Brain and Language, vol. 206, Art. no. 104811, 2020, doi: 10.1016/j.bandl.2020.104811.