Auditory and Vestibular Research

From Decibels to Phons: A Comprehensive Study on Tinnitus Loudness and Brain Activity

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Highlights

Resting-state EEG oscillations correlate with tinnitus loudness.

Phon-based tinnitus loudness inversely relates to delta power and theta/alpha ratio.

Phon might more accurately reflect perceived loudness than dB SL.

Abstract

Background and Aim: Given that the loudness obtained through the conventional methods cannot accurately represent the loudness of tinnitus due to the phenomenon of recruitment, and that the self-reported loudness is more reflective of tinnitus-related distress, and considering that previous studies have not demonstrated a significant correlation between tinnitus loudness and specific brain regions, the present study may provide a more appropriate method for evaluating tinnitus loudness and reveal stronger associations between loudness and brain areas. In this study, we addressed the relationship between tinnitus loudness estimates and resting-state electroencephalography (EEG) activity in tinnitus-associated brain networks.

Methods: In this cross-sectional correlational study, twenty-one subjects with normal to moderate high-frequency hearing loss and tinnitus underwent psychoacoustic assessments including the Visual Analog Scales

for Loudness (VAS-L), pitch matching, and loudness matching in both dB SL and phon units. Subsequently, EEG was recorded from all participants. The collected data were analyzed using statistical methods to examine the correlation between study variables and brain networks.

Results: Loudness in the phon was inversely related to delta band power and theta/alpha ratio in the frontal and central regions, but loudness in dB SL was positively correlated with delta band power in the right frontal region. Furthermore, self-reported loudness on the VAS was negatively correlated with theta-band power in the temporal and central regions.

Conclusion: These results emphasize the multifactorial nature of tinnitus perception and suggest that phon may be a more sensitive measure of tinnitus loudness.

Keywords: Tinnitus, electroencephalography, tinnitus loudness, visual analog scales, tinnitus distress

Introduction

Subjective tinnitus occurs in the ear or brain without any external acoustic source, affecting approximately 14% of the population affected, with about 2% experiencing more severe symptoms [1, 2]. As many patients complain about the loudness or severity of their tinnitus, measuring tinnitus loudness is an important step to take prior to completing any intervention. The loudness of tinnitus can be determined using various methods. One such approach is the Visual Analog Scale-Loudness (VAS-L), a patient-reported measure of perceived loudness [3]. This subjective assessment represents more of the impact of tinnitus on patient's life than its actual loudness [3]. A more widely used method is to define tinnitus loudness in decibel sensation level (dB SL), which is the level above the hearing threshold. However, it has been shown that tinnitus loudness assessed in dB SL usually does not reflect the actual loudness perceived by patients [4]. This discrepancy raises the question of why a mismatch exists. There are several reasons for this discrepancy. First, loudness measurement may be invalid. Second, loudness is not necessarily related to distress expressed by the patient. Third, the loudness of tinnitus may be high, but it is underestimated because of the recruitment in the matching frequency [4]. These factors contribute to the challenges of accurately measuring tinnitus loudness and provoke a search for alternative measurement methods.

Tinnitus is a heterogeneous phenomenon, with multiple proposed neural mechanisms underlying its generation and perception. Based on Thalamo-Cortical Dysrhythmia (TCD) theory, tinnitus is a cause of either impaired central noise cancellation or an afferent disorder. This disruption abnormally alters brain wave activity [5]. Additionally, the Global Workspace Hyperactivity, Tinnitus Network, and chaos theory posits that tinnitus is a result of activity in multiple or whole brain regions, including auditory and important non-auditory structures, such as auditory perception, salience, and stress networks, which contribute to its perception and various consequences, thereby linking neural oscillatory dynamics directly to the subjective experience of tinnitus loudness [6]. Electroencephalography (EEG) has therefore become a key method for investigating the neural correlates of tinnitus loudness. As tinnitus most likely results from aberrant activity in networks distributed over the brain and manifests in the spontaneous activation patterns of the cortex, resting-state EEG has become an invaluable means of investigating tinnitus [7].

While several studies have been conducted regarding the correlation between tinnitus loudness and brain wave activity, the specific relationship between perceived tinnitus loudness and neural oscillations within brain regions remains poorly understood, and the results remain inconsistent. For example, Ridder et al. reported that the brain creates a network representing perceived subjective loudness, as determined by a rating scale. However, no significant correlations were found between tinnitus loudness measured in dB SL and activity in EEG bands [8]. On the other hand, Balkenhol et al. found that higher tinnitus loudness in dB SL was associated with higher gamma and delta band activities contralateral to unilateral tinnitus, though no significant correlation appeared between tinnitus loudness, as measured at 1 kHz, and activity within any of the band activities [2]. Similarly, Van der Loo et al. suggested that gamma band activity in the contralateral auditory cortex may be involved in the neural representation of tinnitus loudness reported subjectively, although they suggested that this activity might reflect intensity coding rather than conscious tinnitus perception [8]. The inconsistent findings regarding the relationship between tinnitus loudness and activity in the tinnitus network may be due to limitations in the current

loudness measurement scales, such as the dB SL. This inconsistency emphasizes the need for alternative methods to more accurately measure tinnitus loudness.

One such method is the use of Equal Loudness Levels proposed by Fletcher and Munson in 1933 [9]. The loudness level of a sound is defined as the level in dB SPL of a 1000 Hz tone that is perceived to be equal to that of sound [10]. The loudness unit, called the phon, provides a psychoacoustic measure of loudness. Any sound that is as loud as a 1 kHz tone at 40 dB SPL has a loudness of 40 phon [11]. Given that tinnitus is a psychoacoustic phenomenon, assessing its loudness using a phon unit could be a more accurate method. Previous studies on the correlation between tinnitus loudness and EEG activity have primarily relied on the VAS-L and dB SL unit to quantify tinnitus loudness [12]. As previously mentioned, this unit may not accurately reflect the actual tinnitus loudness for various reasons. Considering tinnitus as a psychoacoustic phenomenon, employing a loudness scale, such as phon, may reveal a stronger correlation between neural activity within the tinnitus network and perceived tinnitus loudness.

Therefore, this study aimed to analyze the correlation between tinnitus loudness measured in dB SL at tinnitus pitch and tinnitus loudness measured in phon, and subsequently investigate their respective correlations with neural activity in various regions of the tinnitus network. By addressing these aspects, this study sought to provide a more comprehensive understanding of the neural and psychoacoustic correlates of tinnitus loudness. These findings could have significant implications for the development of more effective measurement techniques and personalized treatment strategies for individuals with tinnitus.

Methods

Participants

Twenty-one patients (17 male, 4 female) with tinnitus participated in this study. Informed consent to participate in this study was obtained from all subjects. Of these participants, eight presented with bilateral asymmetrical tinnitus with the same tinnitus pitch in both ears. The remaining patients had unilateral tinnitus. Patients were selected from individuals who were willing to participate in the study with normal hearing thresholds (auditory pure tone thresholds less than 25 dB HL across the frequency range of 0.250–8 kHz) or at most moderate high-frequency hearing loss (evaluated using GSI 61 Clinical audiometer manufactured by Interacoustics (Denmark)). The effects of hearing loss were reduced as much as possible by excluding patients with significant hearing loss; however, high-frequency hearing loss could not be completely eliminated because participants with tinnitus naturally have at least some degrees of high-frequency hearing loss. Also, the hearing loss effects could not be fully controlled due to the nature of the study. As previously noted, one factor contributing to discrepancies between loudness measurement methods is the recruitment phenomenon caused by hearing impairment. Therefore, it was both logical and necessary to include participants with some degree of high-frequency hearing loss, as there have been some studies, that did not control for the effects of hearing loss [7]. A comprehensive medical history was obtained for each patient to control for otological, neurological, and psychological disorders. All patients completed the Persian version of the Tinnitus Handicap Inventory (THI) Questionnaire [13]. Participants with no history of medical disorders and HADS scores < 11 were included in the study. They were also asked to rate the loudness of their tinnitus on a scale of 0 to 10 (VAS-L). Subsequently, all participants underwent psychoacoustic tinnitus measurements. This study was approved by the Research Ethics Committee of IRAN University of Medical Sciences (Ethics code: IR.IUMS.REC.1402.314).

Psychoacoustic Assessment of Tinnitus

After selecting the eligible patients, psychoacoustic evaluations of tinnitus were conducted. These evaluations included the following:

Pitch Matching: Pitch matching was conducted using the two-frequency comparison method. In the subjects with unilateral tinnitus, matching was performed in the contralateral ear. For those with bilateral tinnitus, these procedures were performed in the ear with lower tinnitus perception. Patients whose tinnitus matched to the narrow-band noise (noise-like tinnitus) were excluded. The others were asked to match their tinnitus pitch to a 1000 Hz pure tone presented at 15 dB above the hearing threshold and indicate whether their tinnitus frequency was higher or lower than the presented stimulus frequency. If tinnitus was higher, the frequency comparison

proceeded to higher octave frequencies and vice versa. Frequency comparison was continued until tinnitus pitch was confirmed.

Loudness Matching: Loudness matching was performed in two ways: 1) In the conventional loudness matching method in dB SL, a pure tone at the tinnitus pitch was presented to the patient. Tone intensity increased in 1 dB steps until it was perceived by the patients as loudness as their tinnitus. The difference between this level and the absolute hearing threshold level at tinnitus pitch represents tinnitus loudness in dB SL. 2) The loudness matching method in phon was conducted in a similar manner, except that the loudness adjustment was determined at 1 kHz instead of tinnitus pitch. Because the equal-loudness contours were measured at the sound pressure level (SPL), the loudness measured in dB SL was converted to dB SPL. The SPL of the reference tone at 1000 Hz represents tinnitus loudness in phon. The tinnitus characteristics of patients are shown in Table 1.

Electroencephalography Data Collecting and Processing

After collecting the primary information of the patients, the brain waves of the patients with tinnitus were collected. The steps for data acquisition and processing were as follows: 1) Methodology employed for EEG data acquisition. 2) Protocols for data preparation and pre-processing. 3) Data processing involving segmenting brain regions to enable region-specific analysis and description of the signal power computation technique. 4) Presenting the statistical analysis framework applied in this study. A workflow summarizing this sequence of analytical stages is shown in Figure 1.

Electroencephalography Recording: EEG data were recorded in an acoustically, electrically, and magnetically shielded room using the Liv Intelligent Technology EEG device. Based on the collected medical history, no EEG was recorded on patients with a history of epilepsy, sleep disorders, or other metabolic and neurological conditions (such as Alzheimer's disease and multiple sclerosis). Participants sat comfortably with their eyes closed and were instructed to stay relaxed, reduce neck tension and eye blinks, and limit jaw movements. All EEG recordings were performed in the morning, from which approximately two minutes of artifact-free resting-state data were selected for analysis. Participants were advised to wash their hair prior to their visits and to abstain from caffeine intake. A total of 30 electrodes were placed at locations according to the international 10-20 system: FP1, FP2, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T7, C3, Cz, C4, T8, TP7, CP3, CPz, CP4, TP8, P7, P3, Pz, P4, P8, O1, O2, Oz, and Fpz as ground electrode with reference electrodes on the mastoids [14]. Data were sampled at 512 Hz and bandpass filtered between 0.4 and 200 Hz. Electrode impedance was maintained below 10 kΩ.

Data Pre-Processing: The EEG data preprocessing pipeline was performed in MATLAB R2020a (Version 9.8, The MathWorks Inc., Natick, Massachusetts, USA) using EEGLAB and comprised five steps to optimize signal integrity. First, the raw recordings were visually reviewed to identify major artifacts (e.g., disruptions in the electrodes) and malfunctioning channels. Although the Fp2 electrode showed operational status during acquisition, its signal could not be recovered and was subsequently excluded, leaving 29 active channels for analysis. The subsequent step was the application of a zero-phase FIR (Finite Impulse Response) notch filter (50 Hz) to reduce the electrical interference, followed by a broad-spectrum FIR bandpass filter (0.5-45 Hz) targeting both low-frequency oscillations and high-frequency noise. The data underwent source separation via Independent Component Analysis (ICA; runica algorithm), enabling manual identification and removal of physiologically irrelevant components (e.g., eye movements and muscle activity). A final quality check verified the successful artifact elimination and restored defective channels through spherical spline interpolation. This methodology emphasizes maintaining signal timing fidelity through phase-conscious filtering and comprehensive artifact mitigation to yield reliable neural signals for subsequent investigations.

Data Processing: Pre-processed EEG signals were divided into five distinct frequency bands: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (12–30 Hz), and gamma (30–45 Hz). Spectral analysis was performed using Welch's method to compute absolute and relative power (the absolute power of a specific band relative to the total electrical activity of the brain) values for each sub-band, as well as gamma/alpha and theta/alpha power ratios across all channels. This approach utilized a Hamming window (15-second duration, 50% overlap) and FFT parameter matching the sampling frequency (512 Hz) to optimize the spectral resolution and minimize noise. Five clusters were identified to align with established tinnitus-related neural networks (Figure 2): (1) left frontal (Fp1, F7, F3, Fz), (2) right frontal (Fz, F4, F8), (3) left temporal (FT7, T7, TP7), (4) right temporal (FT8, T8,

TP8), and (5) central (FC3, FCz, FC4, C3, Cz, C4, CP3, CPz, CP4). These clusters were chosen to focus on the brain regions previously implicated in tinnitus pathophysiology [15].

Statistical Analysis: The derived EEG metrics, including power values, frequency band ratios, and demographic data, were integrated for statistical analysis. The analysis was based on the Spearman's correlation coefficient (ρ), and the significance level α was set at 0.05. The analysis evaluated two related questions: 1) how is the relationship between phon and other tinnitus measurements, and 2) how do these measurements relate to activity across the tinnitus network and specific clusters of brain sections. Both analyses yielded detailed findings regarding the associations between neurophysiological features and tinnitus variables, aiming to identify potential biomarkers or patterns linked to tinnitus.

Results

This study examined the associations between tinnitus loudness (measured in decibel sensation level and phon units) and neural activity in brain regions involved in auditory processing and non-auditory processes, respectively, in the concurrent computation of tinnitus loudness

A moderate negative correlation was found between delta band absolute power and tinnitus loudness in phon ($\rho = -0.441$, p-value = 0.044). No significant correlations were observed between the average absolute power of the frequency bands and other tinnitus loudness measurements (dB SL and VAS-L). The results suggest that delta absolute power may specifically relate to psychoacoustic loudness perception.

The analysis of relative frequency band power revealed that EEG bands did not show significant correlations with dB SL and VAS-L. However, tinnitus loudness in phon was significantly and negatively correlated with the relative theta/alpha ratio ($\rho = -0.473$, p-value = 0.030), indicating that a higher proportion of theta power relative to alpha power is associated with a reduced perception of tinnitus loudness. While the relationship between phon and alpha may appear positive, phon is, in fact, inversely related to both waves. however, due to the stronger correlation with theta, its impact on the phon unit is more pronounced (p-value and ρ of theta = 0.07, -0.39 respectively and p-value and ρ of alpha= 0.08, -0.38 respectively).

The analysis of the absolute power of clusters and tinnitus variables revealed a localized relationship between brain oscillatory activity and tinnitus loudness measured in dB SL. In particular, only the delta band power in the right frontal region showed a statistically significant positive correlation with tinnitus loudness ($\rho = 0.461$, p-value = 0.035), indicating that higher slow-wave activity in this area was associated with greater perceived loudness. In contrast, no other frequency band or brain region was significantly associated with tinnitus loudness in dB SL. Furthermore, when tinnitus loudness was quantified in phon as well as for perceptual ratings (VAS for loudness), none of the correlations achieved significance (all p-values > 0.05). Although a trend toward negative correlations was observed in the left temporal region for tinnitus loudness in phon (e.g., delta: $\rho = -0.383$, p-value = 0.086), this did not meet the significance threshold.

For relative power, no significant correlations were observed for tinnitus loudness measured in dB SL across any brain region or frequency band. However, tinnitus loudness measured in phon showed significant negative correlations with the theta/alpha ratio in the right frontal ($\rho = -0.461$, p-value = 0.035) and center clusters ($\rho = -0.486$, p-value = 0.025). Additionally, VAS-L was significantly negatively correlated with theta power in the right temporal ($\rho = -0.499$, p-value = 0.021), left temporal ($\rho = -0.531$, p-value = 0.013), and center clusters ($\rho = -0.438$, p-value = 0.046). These findings suggest that in terms of relative power, specific alterations in the theta band and its ratio with alpha in certain regions may be linked to perceptual measures of tinnitus loudness.

Complementing the detailed statistical results, Figures 3 and 4 synthesize our findings using two complementary panels. Figure 3 panel (a) features a topographical brain map, with color gradients representing the strength of correlations between tinnitus loudness (measured in phon) and the theta/alpha power ratio, and panel (b) presents the corresponding scatterplots. Figure 4 panel (a) shows the subjective tinnitus loudness (assessed via VAS-L) and theta-band relative spectral power, and panel (b) presents the corresponding scatterplots. In each figure, scatter plots are overlaid with regression lines and 95% confidence intervals, depicting the linear relationships between these paired variables. Together, the spatial patterns in panel (a) and quantitative trends in panel (b) offer a dual perspective on the significant neurophysiological associations identified in our study, bridging localized neural activity with perceptual and subjective features of tinnitus loudness.

Initial analyses revealed no significant correlation between tinnitus loudness in phon and other tinnitus loudness measurements.

Discussion

Given that present study is a preliminary investigation, our primary objective was to explore initial hypotheses rather than to establish definitive relationships. As an exploratory research effort, the main focus was on assessing the possible relationship between tinnitus loudness, as measured in both phon and decibel sensation level, and EEG activity across different brain regions. One of the key findings of this study was the negative correlation observed between tinnitus loudness in phon and the mean absolute power of delta band activity, as well as the mean theta-to-alpha ratio in the right frontal and central regions. Conversely, a significant positive correlation was identified between tinnitus loudness measured in dB SL and absolute power of the delta rhythm in the right frontal region. Additionally, the study found a significant relationship between VAS-L score and the relative power of the theta band in the right and left temporal lobes as well as in the central region.

As mentioned, the current investigation revealed negative correlations between tinnitus loudness in phon and EEG band powers. One study has investigated the correlation between tinnitus loudness in phon and brain activity [2]. Balkenhol et al. investigated the relationship between tinnitus loudness, assessed through various methods, including matching tinnitus loudness with a 1 kHz pure tone, and brain frequency band power across different brain regions. Controlling for age, anxiety, and hearing threshold, the study found no significant correlation between tinnitus loudness and activity within various frequency bands [2]. Conversely, the current findings indicate that with increasing loudness in phon, the power of delta activity and the ratio of theta to alpha band powers decrease. Phon demonstrates an inverse relationship with both theta and alpha, as mentioned earlier. Low-frequency theta band power (4–8 Hz) is dominant in states of drowsiness and meditation, and the frontal region is typically dominant in various cognitive processes, such as mental effort. The alpha rhythm, defined by oscillatory activity within the 8–13 Hz frequency band, is indicative of a state of relaxed wakefulness and serves as a neural correlate of cognitive inactivity. Likewise, delta band power with a very low frequency (0.5–4 Hz), which is characteristic of deep sleep and neurological pathologies, is recognized for its primary role in inhibitory neural mechanisms [16]. Considering the known roles of delta, theta and alpha band powers, the observed decrease in these bands in line with increased loudness in individuals with tinnitus might suggest a possible reduction in inhibitory activities in the auditory neural system, leading to an increase in neural excitability and tinnitus. However, drawing a definitive conclusions from these findings is challenging; because no significant correlation was observed between phon and the absolute power of theta and alpha band activities. Consequently, the relationship appears to be mediated by the relative balance between these frequencies (the theta-to-alpha ratio) rather than their isolated absolute powers, complicating the interpretation of specific neural mechanisms involved.

On the other hand, analyses demonstrated that increased perceived tinnitus loudness measured in dB SL was positively correlated with increased delta wave activity within the right frontal region. This finding might be explained within the framework of the TCD model. Given that dB SL measurements are typically measured at tinnitus frequency, which often coincides with regions of hearing loss in patients with tinnitus, the observed increase in delta activity could possibly represent a compensatory response to deafferentation, consistent with the TCD model. According to this theory, reduced sensory input, such as that resulting from hearing loss, can induce alterations in neural oscillatory activity, resulting in reduced alpha rhythm activity and increased theta and delta band powers [5]. Indeed, delta band power is known to be modulated by sensory deafferentation, exhibiting an increase in response to diminished sensory input, such as hearing loss [17]. Therefore, it may be assumed that the observed increase in delta band activity with increasing loudness in dB SL at tinnitus pitch reflects the underlying hearing loss at that specific frequency. Although efforts were made to mitigate the influence of hearing loss in this study, the effects of deafferentation cannot be entirely eliminated in auditory evaluations. However, Ridder et al. reported no significant correlation between tinnitus loudness (dB SL) and brain activity across delta, theta, alpha, beta, and gamma frequency bands [7]. Other neuroimaging studies that utilized EEG and functional magnetic resonance imaging (fMRI) have implicated a distributed network of brain regions involved in tinnitus loudness perception, including the primary and secondary auditory cortices, hippocampus, and cingulate cortex [5]. Although the temporal lobe includes areas related to auditory processing (primary and secondary auditory cortices) [18] and areas related to memory/emotion regulation (hippocampus and amygdala) [19], the spatial

resolution of this study precludes precise localization of the observed delta band power correlation with tinnitus loudness to any single subregion within the temporal lobe.

A further significant finding of the present study was the negative correlation between the VAS-L score (tinnitus loudness as determined by the individual) and the relative power of the theta band in the right/left temporal lobes and central region. In this regard, the literature presents inconsistent results. For instance, Van der Loo et al. reported a correlation between subjective tinnitus loudness (VAS-L) and gamma band activity in the contralateral auditory cortex. However, they further proposed that gamma band activity within this region primarily encodes tinnitus loudness rather than the perceptual experience itself [8]. Conversely, in a study conducted by Ridder et al., it was found that VAS-L is determined by the functional connection of the theta band between the left primary auditory cortex and the parahippocampus [7]. Subsequent work by Ridder and Vanneste identified correlations between subjective loudness and beta and gamma activity within the auditory cortex [5]. Despite these discrepancies, a general consensus exists regarding the significant role of the temporal lobe in the subjective perception of tinnitus loudness, as assessed by VAS-L, a finding that aligns with that of the current study. Therefore, it is imperative to acknowledge the perceptual dimensions of tinnitus. VAS-L relies on individual self-assessment and is thus susceptible to the influence of affective and emotional factors [20]. The brain regions examined in this study encompass extensive neural networks. For example, the temporal lobe includes areas associated with auditory processing, memory, and emotional regulation, while the central areas of the brain contribute to the processing of affective states [21]. Although theta band power can be increased according to the TCD theory in response to increasing hearing loss, the observed negative correlation between VAS-L and theta band power may possibly reflect the dominant influence of subjective loudness perception and its associated emotional impact. Consequently, it is plausible that with increasing VAS-L scores, theta band power decreases within the right and left temporal and central regions because this band power is susceptible to modulation not only by auditory deafferentation but also by emotional and cognitive processing [22].

As mentioned, the results revealed no statistically significant correlation between tinnitus loudness measured in phon and loudness measured in dB SL at tinnitus frequency. This dissociation suggests that, while both methods can be employed to assess tinnitus loudness, they likely address distinct aspects of this phenomenon. The results are interpretable in light of the distinct nature of intensity and loudness. As Stanley and Gelfand explained in their book, sound intensity and loudness are distinct concepts. Intensity is a physical stimulus parameter and loudness is the associated perceptual experience [11]. Furthermore, Fletcher and Munson highlighted that sounds of equal intensity can exhibit varying loudness (in phon) at different frequencies [23]. Consequently, a direct linear relationship between these two measures is not anticipated. As mentioned previously, the observed discrepancy may be attributable to the influence of recruitment. Meikle et al. highlighted the limitations of conventional tinnitus loudness matching (dB SL) when significant hearing loss was present at tinnitus frequency. Their findings demonstrated substantially elevated matching values by measuring loudness in the frequency region of normal hearing, which is more consistent with patient reports of high tinnitus loudness [24]. This suggests that the presence of hearing loss at tinnitus pitch can increase the likelihood of recruitment, potentially leading to an underestimation of tinnitus loudness, a phenomenon documented in the studies [25]. Given that the hearing threshold of 1 kHz is less susceptible to recruitment at normal hearing thresholds (as was the case for all participants in the present study), loudness measurements at this frequency are expected to differ from those obtained at other frequencies, particularly tinnitus frequency. Therefore, loudness measurements based on dB SL may not accurately reflect the true tinnitus loudness.

Another finding of the current study was the lack of any relationship between tinnitus loudness based on phon and VAS-L scores. The present findings align with existing literature. Hall et al. concluded that loudness rating (a self-report measure) and loudness matching measured at 1 kHz represent distinct constructs, with loudness matching assessing perceptual tinnitus characteristics and loudness ratings reflecting its subjective impact [26]. Similarly, Rabau et al. suggested that subjective tinnitus loudness reflects not only loudness itself, but also the impact of tinnitus on daily life [27]. Consequently, while loudness (based on phon) may be related to perception, subjective tinnitus loudness (VAS-L) may be modulated by additional factors, such as emotional state. Asutay and Västfjäll demonstrated that negative emotions can influence sound perception and increase individuals' sensitivity to loudness [28]. Therefore, the absence of a significant correlation between loudness measured in phon and VAS-L scores likely arises from the different nature of these measures. Phon is a physical measure of

loudness, whereas VAS-L is a psychometric scale designed to assess subjective loudness perception, which can be influenced by a multitude of factors.

Conclusion

Unlike earlier studies on tinnitus, where dB SL was measured, this study measured another loudness measurement (in phon). This study imply that each measurement may provide an index of specific perceptual mechanisms and biased by emotional/cognitive factors. Therefore, the results highlight the need for deeper consideration and further research into the accurate assessment of tinnitus loudness and expand upon these preliminary results. Despite the controversial findings, this study was certainely limited: the study's sample size limits the generalizability of the results, and EEG's outstanding temporal resolution (time-locked) cannot provide high spatial mapping in return. Larger participant cohorts and multimodal imaging approaches, such as fMRI-EEG fusion, are recommended to better understand tinnitus-associated phenomena. Lastly, the existing models of tinnitus that developed based on values of dB SL can also be evaluated using the phon unit that encompasses frequency-weighting designs.

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Tables

Table 1. The tinnitus characteristics of patients

	min	max	Ave	SD
Age (year)	22	64	42.8	13.8
Tinnitus Duration (month)	2	156	42	37.3
Tinnitus Pitch (Hz)	750	8,000	5,000	3.4
Tinnitus loudness (dB SL)	2	17	8.4	3.3
Tinnitus loudness (phon)	6	46	24.5	9.7
VAS-L	1	9	4.4	1.9
THI	6	86	28.1	21.3

min; minimum, max; maximum, Ave; average, SD; standard deviation

Figures

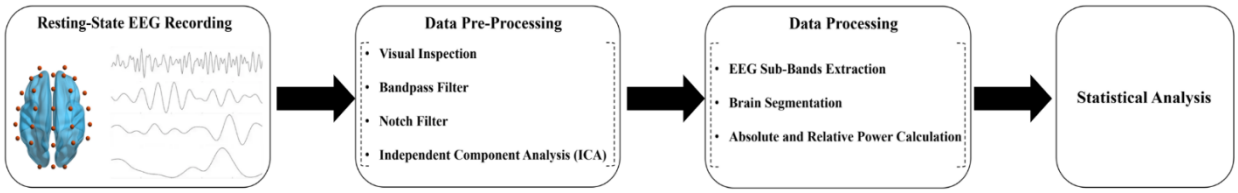

Figure 1. The paradigm's workflow architecture.

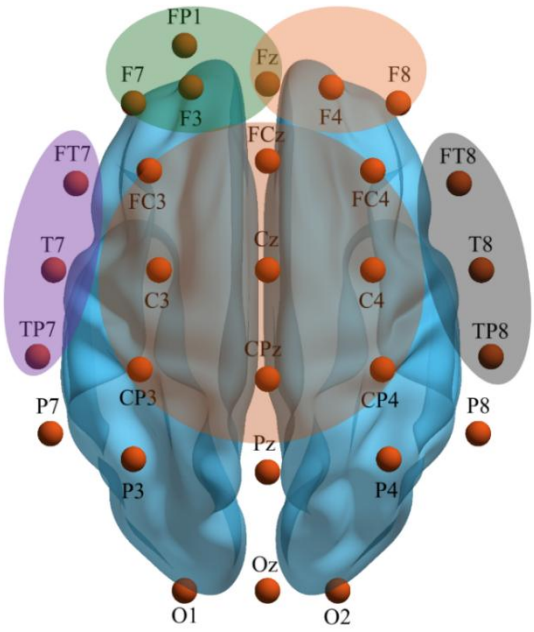

Figure 2. Scalp topography of electrode clusters (colored zones) analyzed for spectral power metrics. The cluster definitions integrate prior evidence of tinnitus-associated functional alterations in the frontal, temporal, and central cortices.

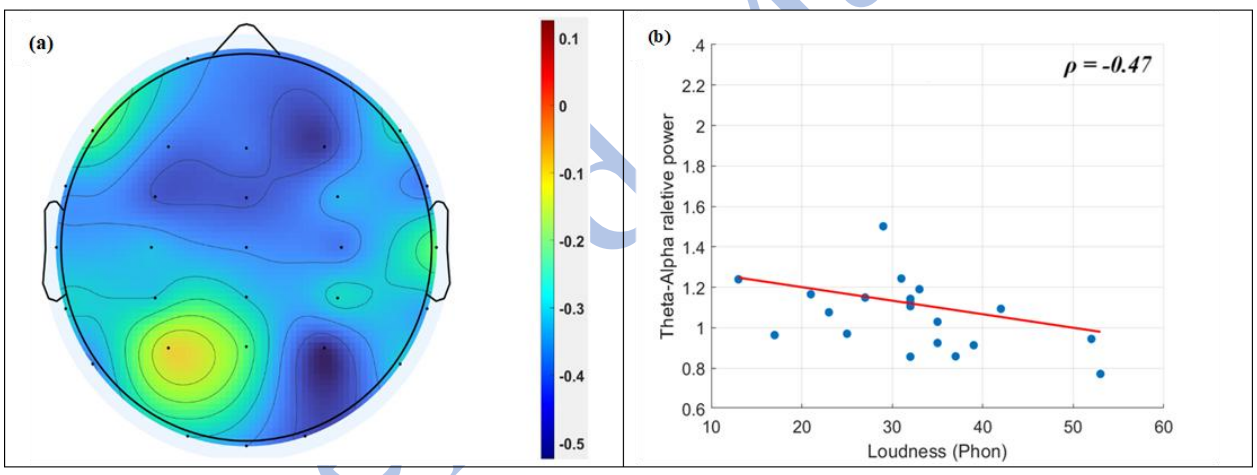

Figure 3. Panel (a) features a topographical brain map, with color gradients representing the strength of the correlations between tinnitus loudness (measured in phon) and the theta/alpha power ratio, and panel (b) presents the corresponding scatterplots.

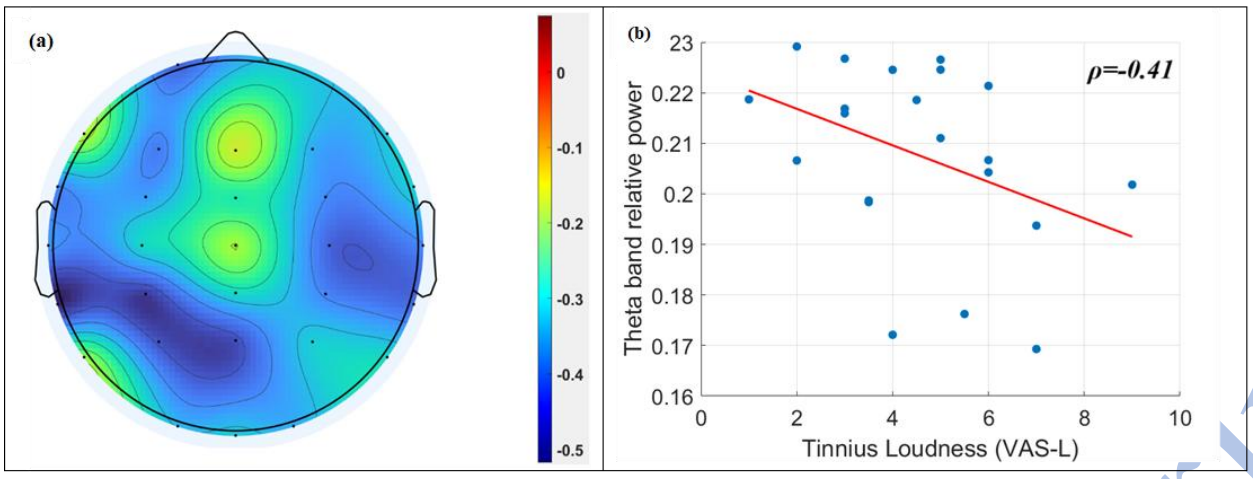

Figure 4. Panel (a) represents subjective tinnitus loudness (assessed via VAS-L) and the theta-band relative spectral power, and panel (b) presents the corresponding scatterplots.