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Correlation between chronic tinnitus severity, magnetic resonance imaging findings, and anxiety/depression scores

Chidananda Mishra[1], Bapini Patra[2], Fakir Mohan Mohanta[3], Sayambika Mishra[4]

[1]Department of Radiodiagnosis, Government Medical College and Hospital, Sundargarh, Odisha, India, [2]Department of Psychiatry, Government Medical College and Hospital, Sundargarh, Odisha, India, [3]Department of ENT, Government Medical College and Hospital, Sundargarh, Odisha, India, [4]Department of ENT, Institute of Medical Sciences and Sum Hospital, Bhubaneswar, Odisha, India

Address for correspondence: Dr. Fakir Mohan Mohanta, Assistant Professor, Department of ENT, Goverment Medical College And Hospital, Sundargarh, Odisha, India. E-mail: fakirsaachin@gmail.com

Abstract

Background: Chronic tinnitus, also known as the experience of sound in the absence of an external source, is experienced by around 10–15% of the population and can cause a substantial impact on quality of life. Neuroimaging studies suggest that tinnitus is the result of maladaptive neuroplastic changes within the congruent auditory and limbic systems. Both emotional and cognitive distress associated with tinnitus seems to manifest as symptoms of anxiety and depression, which seems to engender overlapping or common neural circuitry associated with the perceptions of tinnitus and affective processing.

Objective: The objective of the study is to examine the relationship between chronic tinnitus severity, magnetic resonance imaging (MRI)-identified brain structural and functional changes, and anxiety and depression scores.

Materials and Methods: In this prospective, observational study, we enrolled 80 patients with chronic subjective tinnitus of at least 6-month duration. The tinnitus severity was assessed using the tinnitus handicap inventory (THI). All patients underwent standardized audiologic protocols and high-solubility 3-Tesla MRI protocols including T1-weighted volumetric and resting-state functional sequences. Cortical thickness, white matter integrity, and abnormal activation were reviewed among MRI parameters in auditory and non-auditory locations. The Beck Anxiety Inventory (BAI) and Beck Depression Inventory-II (BDI-II) gauged occupied psychological distress. Correlations of THI were evaluated with MRI changes and psychological scores using paired *t*-test and multivariable regression models.

Results: A strong positive correlation was found between THI scores and BAI scores ($r = 0.68$, $P < 0.001$) and BDI-II scores ($r = 0.63$, $P < 0.001$), indicating that the severity of tinnitus increased anxiety and depression. MRI findings indicated reduced gray matter volume in the left auditory cortex, increased functional connectivity between the auditory cortex and amygdala, and abnormalities in microstructural and volumetric properties of the corpus callosum and hippocampus. The patients with high severity tinnitus (THI >56) were the most affected both structurally and functionally. Regression analysis indicated that amygdalar hyperconnectivity was the strongest predictor of distress ($\beta = 0.44$, $P < 0.01$).

Conclusion: Individual severity of chronic tinnitus is related to MRI-detected neural changes and increased anxiety and depressive symptoms. These findings point to the interconnected neurobiological substrates underpinning tinnitus perception and the associated/related clinically significant dysregulation of affect and the need for treatment to reflect this both neurobiological and psychological.

Keywords: Anxiety, Auditory cortex, Beck anxiety inventory, Beck depression inventory, Depression, Functional connectivity, Magnetic resonance imaging, Neuroplasticity, Tinnitus, Tinnitus handicap inventory

Introduction

Chronic tinnitus is the ongoing awareness of sound with no external source and is generally associated with sleep disturbance, trouble concentrating, and decreased quality of life. Current models do not merely consider it as an otologic symptom, but rather as the byproduct of aberrant central auditory processing molded by neuroplasticity.[1] Peripheral deafferentation (from, e.g., noise exposure, presbycusis, or ototoxicity) can provoke gain adjustments and spontaneous hyperactivity throughout the auditory pathway, beginning at the dorsal cochlear nucleus and continuing to the inferior colliculus and the auditory cortex. Ultimately, these alterations may become stable and provide an ongoing perception of tinnitus, without their initiating peripheral insult becoming progressively smaller.[2] A major tenet of all current pathophysiological frameworks is the identical interface of auditory and non-auditory networks. Salience and attention networks, e.g., anterior insula and dorsal anterior cingulate, can facilitate exposure of tinnitus-related signals while the limbic network, e.g., amygdala, hippocampus, and ventromedial prefrontal cortex, can moderate discomfort and the memory encoding of the percept of tinnitus.[3] This "auditory-limbic coupling" offers an explanation why different individuals with the same audiogram and sustained acoustic trauma have different amounts of annoyance and handicap from the tinnitus and corresponds with what clinicians and researchers are able to observe, which is that cognitive and emotional factors (e.g., catastrophizing, attention variability, and expectancy) shape symptom severity and subsequent coping strategies.[4]

Magnetic resonance imaging (MRI) provides non-invasive access to these mechanisms. Structural MRI (sMRI) has been reported on differences in gray matter volume and cortical thickness in auditory and limbic regions, and diffusion-weighted imaging (DWI) is used to assess the white-matter tracts including the corpus callosum and cingulum.[5] Resting-state functional MRI (rs-fMRI) captures intrinsic functional connectivity within and between auditory, salience, default mode, and limbic networks. Although task-based fMRI is used less often in tinnitus due to the confounding impacts of the stimulus, task-based fMRI is more suitable for engaging processes such as attention and the processing of emotional response to symptom salience. Taken together, these methodologies allow for multi-level characterization of neuroplasticity changes that may parallel clinical severity.[6]

Tinnitus severity is typically assessed in the clinical setting using standardized measures completed by the patient. One such measure, the tinnitus handicap inventory (THI), is a valid and commonly used assessment of functional, emotional, and catastrophic aspects. Since distress is a key component of the experience of impairment related to tinnitus, anxiety and depression can be assessed, as well.[7] Instruments for anxiety and depression, such as the Beck Anxiety Inventory (BAI) and Beck Depression Inventory (BDI-II), have been established in medical patient populations and enable comparisons with existing broader psychopathology evidence. Notably, not all tinnitus patients will meet criteria for a psychiatric disorder, but elevated symptom counts can lead to an attribution of attention, increased perception of loudness or intrusiveness, and changes in treatment response.[8]

Across studies, several converging observations have emerged. First, the severity of tinnitus consistently appears to relate more strongly to measures of distress (e.g., anxiety or depression) than to audiometric thresholds alone. This suggests considerable influence from affective and cognitive systems on symptom burden. Second, while MRI studies have found changes in auditory cortex morphology, limbic volumes, and large-scale network connectivity, findings have not been consistent across samples and methods.[9] Third, heterogeneity in study design remains a significant limitation: Cohorts differ in the chronicity and causation of tinnitus, in the strength and preprocessing of MRI, and psychometric tools are not always harmonized. Each of these factors makes direct comparison difficult, with heterogeneity in the association with reported loci of structural or functional change also likely playing a role.[10]

There may also be important translational implications. If MRI evidence for higher amygdalar connectivity in the auditory cortex, or impaired integrative regulatory integrity in the prefrontal cortex, tracks severity of tinnitus and anxiety/depression scores, they may represent candidate biomarkers to better stratify patients, to optimize multimodal therapy (e.g., sound therapy plus cognitive-behavioral approaches), and to gauge outcomes. On the other hand, demonstrating weak or absent associations in rigorously psychiatrically phenotyped cohorts may advance mechanistic storytelling and highlight the clinical need to refocus on behavioral and audiologic contributors.[11]

Despite the increasing body of work, there are still relatively few integrated studies that have concurrently quantified tinnitus burden, detailed MRI indices, and standardized anxiety/depression scales within the same cohort. Many reports focus on one or two of these areas, draw upon small sample sizes, or do not employ multivariable modelling techniques to separate common variance across neuroimaging, symptom, and mood indices. In addition, the majority of studies are not explicitly structured to establish clinically meaningful cut points (e.g., THI severity bands) when examining the relationships between imaging features and symptoms and even less have examined whether particular MRI features predict distress independent of hearing loss and demographic factors.

Thus, we are motivated to systematically investigate the relationships between chronic tinnitus severity (by way of the THI), MRI-derived structural and functional brain measures, and standardized anxiety/depression scores (BAI/BDI-II) using a prospectively characterized cohort and whether features from neuroimaging variables explain variance in mood distress beyond prescribed audiometric and demographic covariates.

Aim and Objectives

Aim

To determine the relationship between chronic tinnitus severity, MRI-detected neural alterations, and psychological distress measured by standardized anxiety and depression scales.

Objectives

1. To quantify tinnitus severity using the THI in patients with chronic tinnitus of more than 6-month duration
2. To evaluate MRI-based structural changes in auditory and non-auditory brain regions, including cortical thickness, gray matter volume, and white-matter tract integrity
3. To assess functional connectivity abnormalities between the auditory cortex and limbic regions using rs-fMRI
4. To assess levels of anxiety and depression with the BAI and the BDI-II
5. To investigate relationships between tinnitus severity, MRI findings, and psychological variables
6. To investigate MRI biomarkers which uniquely predicted emotional distress in individuals with severe tinnitus adjusted for demographic and audiometric covariates.

Materials and Methods

Study design and setting

A prospective observational study took place at the Department of Otorhinolaryngology alongside the Departments of Radiodiagnosis and Psychiatry of a tertiary care academic hospital from January

2022 to December 2023. Approval for ethics was obtained from the Institutional Review Board, and before enrolment, informed consent was obtained from each participant.

Study population

Eighty adults (between 20 and 60 years of age) diagnosed with chronic subjective tinnitus for over 6 months were recruited to participate in this study. Recruitment occurred at outpatient otorhinolaryngology clinics and participants were screened based on inclusion and exclusion criteria.

Inclusion criteria

1. Male or female adult aged between 18 and 65 years
2. Unilateral or bilateral tinnitus duration of at least 6 months
3. Pure tone averages 30 dB or greater of either normal or mild sensorineural hearing loss
4. Ability to undergo MRI scanning and complete psychometric questionnaires.

Exclusion criteria

1. A history of neurological conditions (epilepsy, multiple sclerosis, head injury, etc.)
2. Presence of psychiatric conditions other than anxiety or depression
3. Contraindications to MRI (metal implants, claustrophobia, pacemakers, etc.)
4. Current otologic infections, Meniere's disease, or vestibular schwannoma
5. Ototoxic medication in the previous 6 months.

Evaluation of tinnitus severity

The severity of tinnitus was evaluated through the THI, a validated self-report questionnaire of 25 items which is intended for clinical use, and was classified as follows:

- Slight (0–16)
- Mild (18–36)
- Moderate (38–56)
- Severe (58–76)
- Catastrophic (78–100).

Every participant completed the THI under circumstances that were quiet and monitored to increase reliability and the likelihood that subjects would not be distracted by outside noise or interruptions. Psychological assessment: The subjects participated in a structured assessment using two standard measures:

- BAI: a 21-item scale used to determine the severity of anxiety symptoms
- BDI-II: a 21-item self-report tool to assess depressive symptoms.

Each tool is independently assessed based on standardized cut-off points (minimal, mild, moderate, and significant). Assessments were conducted blind, by a qualified psychiatrist of the MRI and audiological assessments.

Audiological assessment

Pure tone audiometry (PTA) with speech audiometry and tympanometry were used to establish each participant's baseline hearing status. For this determination, the average pure tone threshold across 0.5, 1, 2, and 4 kHz was calculated for each ear to confirm that tinnitus was not present with greater than mild hearing loss in relation to PTA results.

MRI protocol

All MRI scans were conducted using a 3-Tesla MRI scanner with a 32-channel head coil. These scans consisted of the following protocol:

1. High-resolution 3D T1-weighted structural imaging (MPRAGE sequence) for measuring cortical thickness and gray matter
2. DWI: to measure white-matter integrity using fractional anisotropy (FA) and mean diffusivity
3. rs-fMRI: 6-min scan where participants rested with their eyes closed to measure intrinsic functional connectivity between auditory and limbic regions.

Images were preprocessed through SPM12 and CONN toolbox pipelines. The preprocessing of images involved standard spatial normalization to the MNI template, motion repair, and smooth (6 mm FWHM Gaussian kernel).

Regions of interest (ROI) analysis

ROIs analyzed included the bilateral primary auditory cortex (Heschl's gyrus), superior temporal gyrus, amygdala, hippocampus, anterior cingulate cortex, and prefrontal cortex. Cortical thickness and gray matter volumes were extracted using the FreeSurfer software program and connectivity strength between auditory and limbic nodes was from rs-fMRI seed-based correlation maps. Statistical analysis: The data analysis was performed using the Statistical Package for the Social Sciences version 26.0. Descriptive statistics were reported as mean ± standard deviation (SD). The following tests were conducted:

- Pearson's correlation coefficient (r): To assess relationships between THI, BAI, BDI-II, and MRI parameters
- Independent *t*-test/analysis of variance: To compare the MRI measurements across the tinnitus severity groups
- Multiple linear regression: To identify independent predictors of anxiety and depression with controls for age, gender, and hearing threshold.

Statistical significance was determined at $P < 0.05$.

Ethical considerations

All participants signed an informed consent form, confidentiality was maintained throughout the study, and ethical approval was obtained from the appropriate IEC. The study adhered to principles of the Declaration of Helsinki (Revised 2013).

Results

A total of 80 patients with chronic tinnitus (48 males and 32 females) were included in the present study, with an age range of 20–60 years (mean age ± SD = 41.2 ± 10.3 years). Duration of tinnitus in the patients ranged from 6 months to 11 years (median = 3.4 years). The overall mean score on the THI was 49.7 ± 17.6, indicating a moderate level of handicap. Below, we present the description of the clinical characteristics, audiological data, MRI results, and psychological scores in 12 tables in a systematic way.

Table 1 shows that males constituted a slightly higher proportion of the study cohort. The age distribution was relatively uniform across both genders, minimizing age-related bias.

Table 2 demonstrates that most cases were chronic continuous types, with the majority experiencing symptoms for 1–5 years.

Table 3 categorizes tinnitus severity, showing that moderate and severe grades together comprised nearly two-thirds of the study population.

Table 1: Demographic profile of study participants

Parameter	Male (*n*=48)	Female (*n*=32)	Total (*n*=80)
Mean age (years)	41.7±9.8	40.4±10.9	41.2±10.3
Age range (years)	21–60	23–59	20–60
Right-sided tinnitus *n* (%)	22 (45.8)	14 (43.8)	36 (45.0)
Left-sided tinnitus *n* (%)	18 (37.5)	12 (37.5)	30 (37.5)
Bilateral tinnitus *n* (%)	8 (16.7)	6 (18.7)	14 (17.5)

Table 2: Distribution according to duration and type of tinnitus

Duration of tinnitus	*n* (%)	Type (Continuous/intermittent)	*n* (%)
6 months – 1 year	11 (13.8)	Continuous	63 (78.8)
1–5 years	46 (57.5)	Intermittent	17 (21.2)
>5 years	23 (28.7)	–	–

Table 3: Distribution of THI severity scores

THI category	Score range	*n* (%)
Slight	0–16	7 (8.7)
Mild	18–36	14 (17.5)
Moderate	38–56	28 (35.0)
Severe	58–76	23 (28.8)
Catastrophic	78–100	8 (10.0)

THI: Tinnitus handicap inventory

Table 4 reveals that higher tinnitus severity was marginally associated with elevated pure-tone thresholds, though still within the mild sensorineural loss range.

Table 5 indicates a progressive rise in mean anxiety scores with increasing tinnitus handicap.

Table 6 demonstrates a similar pattern to anxiety, with depressive symptom scores increasing significantly alongside tinnitus severity.

Table 4: Mean audiological thresholds by tinnitus severity

THI category	Mean PTA (dB HL)±SD	Range
Slight	17.4±3.1	10–22
Mild	19.1±4.6	12–28
Moderate	21.8±4.2	13–30
Severe	23.6±5.3	14–32
Catastrophic	25.1±6.4	16–34

THI: Tinnitus handicap inventory, PTA: Pure Tone Audiometry, SD: Standard deviation

Table 5: BAI scores across THI severity categories

THI Category	Mean BAI score±SD	Anxiety severity level
Slight	7.8±2.4	Minimal
Mild	10.2±3.8	Mild
Moderate	17.5±5.6	Moderate
Severe	21.9±6.7	Moderate-Severe
Catastrophic	27.6±7.3	Severe

THI: Tinnitus handicap inventory, SD: Standard deviation, BAI: Beck Anxiety Inventory

Table 6: BDI-II scores across THI severity categories

THI category	Mean BDI-II score±SD	Depression severity level
Slight	8.6±2.1	Minimal
Mild	11.3±4.0	Mild
Moderate	18.9±6.2	Moderate
Severe	23.7±7.5	Moderate-severe
Catastrophic	30.5±8.1	Severe

THI: Tinnitus handicap inventory, SD: Standard deviation, BDI-II: Beck depression inventory-II

Table 7 shows a significant reduction in cortical thickness within the left Heschl's gyrus and anterior cingulate cortex among patients with severe tinnitus.

Table 8 identifies decreased FA values in the corpus callosum and cingulum bundles, implying microstructural disruption in severe tinnitus.

Table 9 depicts hyperconnectivity between the auditory cortex and amygdala/hippocampus, particularly pronounced in severe tinnitus groups.

Table 10 confirms strong positive correlations between tinnitus handicap and both anxiety and depression indices.

Table 11 illustrates significant inverse associations between THI and cortical thickness/FA values,

Table 7: Structural MRI findings – cortical thickness in auditory and limbic regions

Region	Mean thickness (mm)±SD	Control reference (mm)	*P*-value
Left Heschl's Gyrus	2.31±0.18	2.48±0.15	<0.01
Right Heschl's Gyrus	2.33±0.16	2.41±0.17	0.09
Anterior Cingulate Cortex	2.27±0.20	2.44±0.19	<0.01
Amygdala Volume (cm^3)	1.23±0.14	1.36±0.13	<0.05

MRI: Magnetic resonance imaging, SD: Standard deviation

Table 8: Diffusion MRI findings: White matter integrity measures

White-matter region	Mean FA±SD	Control reference	*P*-value
Corpus callosum	0.451±0.034	0.487±0.030	<0.01
Cingulum bundle	0.432±0.029	0.465±0.028	<0.05
Inferior longitudinal fasciculus	0.459±0.031	0.472±0.027	0.12

MRI: Magnetic resonance imaging, SD: Standard deviation, FA: Fractional anisotropy

Table 9: Resting-state functional MRI connectivity between key regions

Connectivity pair	Mean z-score±SD	Correlation direction	*P*-value
Auditory Cortex↔Amygdala	0.42±0.11	Positive	<0.01
Auditory Cortex↔Hippocampus	0.38±0.12	Positive	<0.05
Auditory Cortex↔Prefrontal Cortex	–0.19±0.09	Negative	<0.05

MRI: Magnetic resonance imaging, SD: Standard deviation

Table 10: Correlation between THI and psychological scores

Parameter pair	Pearson's r	*P*-value	Strength of association
THI↔BAI	0.68	<0.001	Strong
THI↔BDI-II	0.63	<0.001	Strong
BAI↔BDI-II	0.74	<0.001	Very Strong

THI: Tinnitus handicap inventory, BDI-II: Beck depression inventory-II, BAI: Beck anxiety inventory

Table 11: Correlation between THI scores and MRI parameters

MRI parameter	Correlation with THI (r)	*P*-value	Interpretation
Left Heschl's Gyrus thickness	–0.49	<0.01	Moderate Negative
Corpus Callosum FA	–0.45	<0.05	Moderate Negative
Amygdala connectivity	+0.56	<0.001	Strong Positive

THI: Tinnitus handicap inventory, MRI: Magnetic resonance imaging, FA: Fractional anisotropy

alongside positive correlations with amygdalar connectivity.

Table 12 identifies amygdalar hyperconnectivity as an independent predictor of both anxiety and depression after adjusting for age, sex, and hearing level.

The research exhibited significant interrelations among tinnitus severity, changes in neuroimaging, and levels of psychological distress. Table 1 shows that there was comparability of demographic data by age and gender, meaning participants were equally represented. Table 2 indicates that most participants had had tinnitus for 1–5 years, meaning their tinnitus was primarily of the continuous variety. Table 3 shows that almost all patients rated moderately to severely on the handicap scale, directly associated with the clinical burden from chronic tinnitus. Table 4 suggests that even though hearing thresholds, while mildly behaving in relation to tinnitus severity, noted that the age-related relationship between tinnitus-related distress extended beyond peripheral hearing loss. Tables 5 and 6 show that as THI scores increased, anxiety and depression scores similarly and precisely rose in the same proportion, indicating the emotional component of the burden from chronic tinnitus. Tables 7 and 8 show neuroimaging findings indicative of cortical thinning and white matter vulnerability in auditory and limbic circuits, whereas Table 9 correlates aberrant auditory-limbic hyperconnectivity with severity and residual disorder from tinnitus. Table 10 objectively indicates strong correlations between tinnitus-related handicap and emotional measures, while Table 11 links structural and functional MRI analysis with tinnitus severity. Table 12 denotes amygdalar hyperconnectivity and anterior cingulate cortical thickness as independent neurobiological predictors of anxiety and depression, after controlling for the identified confounders.

Overall, the combined findings support the idea that the severity of tinnitus distress is closely linked to both structural and functional brain changes and to psychological comorbidity, pointing to the need for multimodal assessment and treatment.

Discussion

The present investigation shows that chronic tinnitus is a complex and multi-dimensional phenomenon, consisting of inter-related auditory,

Table 12: Multiple linear regression predicting anxiety and depression scores

Predictor variable	Standardized β	*t*-value	*P*-value	Outcome predicted
Amygdala connectivity	0.44	3.21	0.002	Anxiety (BAI)
Anterior cingulate thickness	–0.31	–2.54	0.013	Depression (BDI-II)
PTA (dB HL)	0.12	1.04	0.29	Non-Significant
Age (years)	0.09	0.81	0.42	Non-Significant

BDI-II: Beck depression inventory-II, BAI: Beck anxiety inventory, BDI-II: Beck depression inventory-II, PTA: Pure tone audiometry

limbic, and cognitive-affective components. The co-variation of tinnitus handicap with co-morbid state and trait anxiety and depression implies that such distress is much more than an epiphenomenon, but rather, a fundamentally defining feature of the clinical tinnitus phenotype. The mean level of sensorineural hearing loss in participants was at the level of mild; however, there was considerable variability in symptom burden across participants, even at "mild" levels of hearing loss, suggesting that central mechanisms account for much of the variability in day-to-day tinnitus impact.[12]

sMRI demonstrated cortical thinning within regions relevant to auditory perception and affective regulation to auditory perception and regulation, including Heschl's gyrus and anterior cingulate cortex. Both of these findings are consistent with maladaptive neuroplasticity: Phantom auditory input can drive reorganization in the sensory cortices, and reduced anterior cingulate integrity can result in reduced top-down control over salience and emotional appraisal. Diffusion metrics demonstrated compromised white-matter coherence in interhemispheric and limbic tracts, consistent with the idea that network integration may be impaired. These microstructural changes may provide a plausible substrate for tinnitus remaining "on" even when peripheral triggers are diminished or modest.[13,14]

Resting-state functional analyses demonstrated increased connectivity between the auditory cortex and limbic nodes, such as the amygdala and hippocampus, which highlight the relationship between these nodes and the sensation of tinnitus. Increased coupling with the amygdala supports a framework in which neutral sensory experience is given affective meaning and is subsequently stored in memory, in a manner that can become difficult to ignore. Increased connectivity with the hippocampus may enhance context-dependent recall of the tinnitus percept, promoting an increase in recurrence and chronification. Conversely, relatively decreased connectivity with the prefrontal control networks points to an imbalance between bottom-up salience assignment and top-down regulation.[15]

Psychometric data were closely aligned with these imaging findings. Anxiety scores increased with tinnitus severity, which is consistent with hyperarousal and vigilant observation of internal body sensations. Depression scores increased as well, which can be explained by reduced motivational drive and decreased cognitive flexibility that accompany reduced prefrontal cingulate efficiency.[16] Multivariable modeling identified amygdalar hyperconnectivity as an independent predictor of anxiety and cingulate thinning as an independent predictor of depression, after accounting for age, sex, and hearing threshold. It indicates that there may be partially dissociable pathways. In particular, subcortical limbic hyperreactivity is related to anxious arousal, and reduced prefrontal-cingulate integrity corresponds to depressive features.[17]

From a clinical perspective, the findings indicate that this necessitates an exhaustive evaluation, inclusive of audiometry, some form of validated tinnitus handicap measures, and brief anxiety/depression screening. The data support a stepped-care model that treats both perception and distress. Sound-based therapies and counseling can address habituation and reduce salience; cognitive behavioral strategies can recast catastrophic interpretations and lower attentional capture; and mindfulness styles may lower autonomic arousal and interrupt perseverative monitoring.[8] For people with notable limbic hyperconnectivity or cingulate thinning consider adjunct neuromodulatory methods that could influence cortical excitability and fronto-limbic dynamics within evidence-based protocols.[18]

From a translational perspective, the alignment of structural and functional abnormalities with symptom measures indicates candidate imaging markers for phenotyping and outcome assessment. Cortical thickness estimates in auditory and cingulate areas, FA at callosal and cingulum paths, and seed-based connectivity at auditory cortex and amygdala/hippocampus provide pragmatic candidates. For cross-study comparability, standardized acquisition and preprocessing pipelines, use of predefined severity strata (e.g., THI bands), and the inclusion of hearing thresholds as covariates are required.[19]

The key limitations cautioned interpretation. The cross-sectional methodology does not permit inference of causation regarding the timing of neural changes in relationship to symptoms: Severe symptoms may correlate with altered neural activity but it is not possible to say that the changed neural activity predated the severity of symptoms or occurred later (as was initially postulated in the introduction). The lack of a group of healthy controls to compare against with respect to MRI measures of brain activity limits conclusions about absolute deviations from normative values; as any references for controls came from other studies, we cannot reasonably conclude that the differences viewed in the input and output regions are significant, given potential variability in scanner and pipeline processing. The sample size was appropriate for a correlation and regression analysis and a larger and more etiologically homogenous sample would have benefited precision in analyses and allowed for sub-group analyses (i.e., acoustic trauma vs. idiopathic tinnitus; unilateral vs. bilateral). Finally, psychological measures took symptoms of anxiety and depression into account but did not equate to diagnoses as previously discussed; further studies that utilize structured interviews would better assure clinical rigor of characterization.

Overall, the present findings support a network approach that shows chronic tinnitus severity is a result of converging processes of reorganization in sensory cortex, limbic hyperactivation, and decreased prefrontal/cingulate modulation as revealed by the demonstrated relationship to anxiety and depression scores. These notable findings would support an integrated care approach, and justify future studies with follow-up to see if providing targeted interventions will alter the specific imaging findings and decrease distress level or severity.

Conclusion

Chronic tinnitus is an affective sensory condition with multiple neurobiological and psychopathological contributing factors. The evidence provided clearly indicates that tinnitus severity is based on much more than auditory thresholds alone. The neurobiological and psychopathological contributors are as important or more important for severity. The neuroimaging demonstrated distinct structural and functional changes of the auditory cortex, limbic system, and prefrontal-cingulate circuitry, indicating maladaptive neuroplastic changes supporting the phantom sound and concomitant distress. The strong positive correlation between tinnitus handicap scores and measures of anxiety and depression underscores emotional burden as a core component of the disorder, rather than a simple secondary reaction. The hyperconnectivity of the amygdala and thinning of the anterior cingulate cortex display a neural imbalance for emotional over-activation and diminished cognitive control. These objective markers indicate that tinnitus should be given consideration in a broader neuropsychiatric context and not as an isolated otologic condition. Clinically, the findings support utilizing an integrative treatment approach, which incorporates sound therapy, psychotherapeutic counseling, and neuromodulatory techniques to address both perceptual and affective areas. By identifying differences in reactivity or other identifiable neuroimaging phenotypes, clinicians can enhance their potential to achieve personalized treatment selection and improved outcomes. Ultimately, future longitudinal/intervention studies are required to determine whether alterations to these neural circuits can meaningfully reduce the perception of tinnitus and the associated emotional suffering. In summary, the findings lend support to managing chronic tinnitus with a comprehensive brain-based, patient-centered, integrative approach to address both neural dysfunctions and psychological distress.

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