Brain and Neural Mechanisms 2025

Tinnitus as a disorder of altered neural synchrony, impaired inhibition and large scale network reorganisation

A total of 46 publications were categorised under brain and neural mechanisms. These studies used neuroimaging, electrophysiology, neurotransmission analysis, network modelling and biochemical approaches to investigate the central origins of tinnitus. Despite methodological diversity, their conclusions consistently depict tinnitus as a disorder of aberrant central processing rather than a passive consequence of cochlear injury.

Large scale network connectivity and reorganisation

Many studies examined functional connectivity between large scale brain networks. Resting state functional MRI analyses concluded that tinnitus is associated with abnormal coupling between the default mode network, salience network, limbic network and auditory network. Graph theoretical studies reported increased clustering and reduced network efficiency, findings that suggest reorganisation toward less flexible neural information flow.

Comparisons between recent onset and chronic tinnitus indicated that network redistribution becomes more widespread as tinnitus persists. Increased connectivity between the precuneus, inferior temporal gyrus and auditory cortex was repeatedly linked with higher tinnitus distress.

Oscillatory dynamics and spectral signatures

Studies of neural oscillations concluded that tinnitus is characterised by elevated gamma activity and reduced alpha and beta synchrony. This pattern was interpreted as evidence of high frequency cortical hyperactivity combined with reduced inhibitory control. EEG microstate analyses showed temporal instability in microstate dynamics, which could classify tinnitus participants from controls.

Other work demonstrated abnormal temporal correlations of alpha oscillations over multiple time scales. Authors concluded that these patterns reflect disrupted inhibitory regulation. Sleep related studies further showed that oscillatory abnormalities persist across sleep stages, indicating that tinnitus related dysregulation extends beyond conscious perception.

Thalamocortical dysrhythmia

A substantial group of studies investigated thalamocortical dysrhythmia. These papers concluded that disrupted gating between thalamus and auditory cortex contributes to phantom auditory perception. Magnetoencephalography and combined imaging studies showed reduced sleep spindle synchronisation, interpreted as impaired thalamic inhibition. Deep learning EEG classification models using these features reported that thalamocortical dysrhythmia signatures can differentiate tinnitus severity levels.

Limbic and affective circuits

Another important subgroup focused on limbic involvement. Volumetric and functional MRI studies reported that tinnitus distress correlates with alterations in the anterior cingulate cortex, insula, hippocampus and amygdala. Magnetic resonance spectroscopy demonstrated shifts in anterior cingulate glutamate to glutamine ratios. Multiple multimodal studies concluded that tinnitus distress is better predicted by limbic activity than by primary auditory cortex function. Additional work showed that gamma and alpha coupling in frontal regions reflects increased attention shifting demands, linking emotional processing with perceptual persistence.

Neurotransmission and central inhibition

Several studies addressed inhibitory signalling. Auditory gating work showed reduced P50 suppression in tinnitus, indicating impaired sensory inhibition. Cortical auditory evoked potential studies reported reduced inhibitory control compared with controls. Basic science research demonstrated reduced GABA(A) receptor cell counts in the dorsal cochlear nucleus of tinnitus models. Studies of BK channels and ryanodine receptors found that deficits in these inhibitory pathways increase susceptibility to tinnitus.

Neural prediction of treatment response

A growing number of publications evaluated whether neural markers can forecast treatment outcomes. Brain connectivity studies concluded that reduced coupling between auditory and limbic networks predicts improvement following sound therapy. EEG microstate metrics and graph theoretical signatures were reported to predict responsiveness to cognitive behavioural therapy and music based interventions. Studies of repetitive transcranial magnetic stimulation found that pre treatment volumetric and connectivity profiles correlated with treatment magnitude.

Developmental, plasticity based and longitudinal mechanisms

A smaller group of studies focused on developmental and plasticity driven pathways. Combined structural and functional imaging demonstrated that auditory cortex and brainstem plasticity occurs in patients who develop tinnitus after surgical unilateral deafness, while those with equivalent deafness but no tinnitus did not show these changes. Longitudinal imaging analyses concluded that structural abnormalities in auditory and non auditory regions evolve gradually from early onset to chronic tinnitus. Functional near infrared spectroscopy showed altered auditory related haemodynamic responses in chronic tinnitus patients compared with controls.

Overall interpretation

Across all 46 studies, tinnitus emerges as a disorder of persistent alterations in neural synchrony, connectivity and inhibition within distributed cortical and subcortical networks. Rather than a single focal lesion, tinnitus reflects aberrant sensory prediction, pervasive network reorganisation and impaired top down control. These mechanisms stabilise the phantom auditory percept over time.

Key Mechanistic Brain Insights in Tinnitus 2025

TINNITUS IS A DISTRIBUTED NETWORK DISORDER

Salience, default mode, limbic and attention networks are consistently involved.

"New insight: tinnitus is a multi-network systems disorder."

THALAMOCORTICAL DYSRHYTHMIA NOW HAS STRONG SUPPORT

Altered gating, sleep spindle disruption and characteristic frequency coupling recur across studies.

"New insight: thalamocortical dysrhythmia is a reproducible neurophysiological signature."

TINNITUS ABNORMALITIES PERSIST DURING SLEEP

Oscillatory disturbances remain active across sleep stages rather than fading with awareness.

"New insight: tinnitus represents a sustained change in brain function."

TINNITUS DISTRESS ARISES AT THE LIMBIC–AUDITORY INTERFACE

Distress maps to ACC, hippocampus, amygdala and insula more than auditory cortex.

"New insight: emotional circuits explain the suffering, auditory circuits explain the sound."

REDUCED CENTRAL INHIBITION IS A SHARED MECHANISM

P50 deficits, cortical inhibition loss, reduced GABA signalling and ion channel changes converge.

"New insight: tinnitus reflects impaired inhibitory control rather than simple over-excitation."

NEURAL MARKERS CAN FORECAST TREATMENT RESPONSE

Connectivity patterns, EEG microstates and graph indices predict improvement with sound therapy or neuromodulation.

"New insight: neural biomarkers may enable precision treatment selection."