**Direct mapping of a cortical tinnitus network**

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**Introduction**

Tinnitus occurs when damage to the peripheral auditory system leads to spontaneous brain activity that is interpreted as sound. Many types of brain activity show abnormalities in association with tinnitus, but it is not clear which of these relate to the phantom sound itself, as opposed to predisposing factors or secondary consequences. Direct demonstration of the core tinnitus correlates requires high-precision recordings of neural activity combined with a behavioral paradigm in which the perception of tinnitus is manipulated and accurately reported upon by the subject. This has thus far not been possible in animal or human research. Here we present extensive intracranial recordings from an awake, behaving tinnitus patient during short-term modifications in perceived tinnitus loudness, permitting a robust characterization of the core tinnitus brain network.

**Methods**

A 50-year-old male patient with moderate-to-severe bilateral hearing loss (Fig.1) and longstanding bilateral tinnitus underwent electrocorticographic epilepsy monitoring. He had tinnitus unrelated to his hearing loss (Fig.1) and longstanding bilateral tinnitus underwent diagnostic electrocorticography. The subject provided repeatedly transiently suppressed using residual inhibition (RI) to a stimulus in the left hemisphere. Electrodes in left primary auditory cortex and grid electrodes over large parts of left auditory cortex (164 total intracranial electrodes). To investigate dynamic tinnitus correlates, the subject’s tinnitus was repeatedly transiently suppressed using residual inhibition (RI) to a stimulus in the left hemisphere. Electrodes in left primary auditory cortex and grid electrodes over large parts of left auditory cortex (164 total intracranial electrodes).

**Conclusions**

In a tinnitus patient with typical symptomatology, we provide the first clear demonstration of a distributed cortical ‘tinnitus system’, which incorporates a large proportion of the cerebral cortex, and all of the major oscillatory frequency bands. Tinnitus modulations were associated with complex restructurings of widespread within- and between-region neural activity. The anterior and cortical areas found to be involved in tinnitus change are in general agreement with recent proposals of a tinnitus network of brain regions.

**References**


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**1. Tinnitus characteristics and experimental paradigm**

**2. Power-masking power changes**

**3. Power changes with tinnitus suppression across days**

**4. Power changes with tinnitus suppression**

**5. Delta phase locking value changes with tinnitus suppression**

**6. Local cross-frequency coupling changes with tinnitus suppression**

**7. Summary of findings and proposed tinnitus networks**

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Oscillatory power changes correlating with tinnitus suppression are shown for two experimental sessions and for combined results. Three data matrices are displayed, corresponding to (1) days 1 and 2 of the experiment, and the combined results thresholded at a $p < 0.05$ corrected. Rows in the matrices represent individual electrodes (numbers correspond to those in Figure 4) and columns individual frequency bands (PF). Color values denote the correlation coefficients (Pearson's r) between partial tinnitus suppression and power in any specific electrode-frequency combination. Color bars indicate power decreases, with tinnitus suppression, and bar colors indicate power increases. In the thresholded plot, gray bars indicate the absence of significant power change. Significant changes in magnitude are shown as the size of the circle and changes in angle as rotation of the arrow. Red is baseline and black is during tinnitus suppression.

The number of each plot indicates the electrode number at which coupling is illustrated, and the Greek letters show which frequency bands the coupling being illustrated is between. HG = Heschl’s gyrus, TP = temporal pole, IPC = inferior parietal cortex, STG = superior temporal gyrus, PHC = parahippocampal cortex.