

## **Neurofeedback-induced modification of the neural network representing tinnitus distress**

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Increasing evidence confirms the theory that tinnitus sensations are linked to plasticity in central nervous system structures. On a cortical level this has been shown for the auditory cortex. In earlier work, we have discussed distortions of the tonotopic map a candidate mechanism. But more recent work from our laboratory suggests that this link is not direct and indicates that abnormal changes in ongoing spontaneous activity patterns could be causally related to tinnitus perception and its associated distress. Using MEG and magnetic source imaging, we uncovered relationships between tinnitus and enhanced slow wave and concomitant reduced tau wave activity over temporal and frontal areas. Modification of this pattern via EEG-neurofeedback significantly reduced the matched tinnitus intensity. Participants who consistently altered their oscillatory patterns benefited from the treatment even to the extent that the tinnitus was completely abolished. Tinnitus distress in these patients was of mild to moderate intensity. Extending research to patients with the full range of distress, we observed a tight relationship between the subjective report and the coupling of distinct brain regions (via phase coherence of single-trial steady state responses). In particular, severe distress was perfectly correlated ( $r=.97$ ) with network activity involving the right parietal and the anterior cingulate cortex. Based on this breakthrough in the neuroscience of tinnitus, we now propose to implement a treatment protocol that is apt to allay tinnitus distress by direct intervention into this newly uncovered tinnitus distress network. We will train a total of three groups (15 patients each) to investigate the effectiveness of this therapeutic measure and compare it to the previous treatment approaches using neurofeedback with tinnitus patients. One group will be given a training to reduce phase synchrony between brain regions representing the tinnitus distress network while they listen to an amplitude-modulated version (eliciting a steady-state response) of their tinnitus sound. A second group will get an optimised treatment of neurofeedback to enhance posterior tau. As comparison to those, we will have a control group to train phase synchrony between brain regions that are unlikely to be involved in the tinnitus distress network.

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