

Use of activity-dependent genes for verification of tinnitus-related plasticity in the auditory cortex

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Phantom Auditory perception (tinnitus) has been associated with loss of neuronal inhibition and changes in synaptic strength in the central auditory system, leading to hyperactivity, a possible reorganization of the neuronal circuitry and finally to malperception. We recently observed a correlation between tinnitus perception in an animal behavioural model and changes in the expression of activity dependent plasticity genes (APG). Subsequent to ototoxic- or noise-induced tinnitus, brain derived neurotrophic factor (BDNF) expression was changed in cochlear neurons and the activity dependent cytoskeletal protein (Arc/arg3.1) was altered in the auditory cortex. The tinnitus induced changes in APGs (in both the cochlea and the auditory cortex) were reversed by cochlear application of distinct pharmacological drugs. The current project addresses the cortical changes associated with noise-induced tinnitus and seeks to identify the cell type and frequency specificity of cortical neurons with altered APG expression. It also aims to examine the dependency of cortical APG expression on the intensity and duration of traumatic noise stimuli and the degree of tinnitus induction. The project finally intends to find pharmacological compounds that reverse tinnitus-induced changes in APGs. We hope to elucidate a molecular tool able to detect hyperactive and silent cortical regions along the cortical frequency map and thereby increase our understanding of the origin of map plasticity in the tonotopically organized cortical areas. We furthermore hope to increase knowledge of putative novel tinnitus therapies and to find new tools to optimise other therapeutic strategies.

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