BMC Neuroscience

Poster presentation

Open Access

Tinnitus-related hyperactivity through homeostatic plasticity in the auditory pathway

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from Sixteenth Annual Computational Neuroscience Meeting: CNS*2007
Toronto, Canada. 7–12 July 2007

Published: 6 July 2007

BMC Neuroscience 2007, **8**(Suppl 2):P89 doi:10.1186/1471-2202-8-S2-P89

This abstract is available from: http://www.biomedcentral.com/1471-2202/8/S2/89

Hearing loss through acoustic trauma or administration of ototoxic drugs leads to the development of increased spontaneous firing rates (hyperactivity) in neurons of the auditory pathway. Hyperactivity in the first processing stage, the dorsal cochlear nucleus (DCN), is correlated to behavioral signs of tinnitus, and the distribution of hyperactivity along the tonotopic axis of the DCN corresponds to the patterns of cochlear damage. Recently, we have proposed that the development of hyperactivity after hearing loss is a consequence of activity stabilization through homeostatic plasticity [1].

We now include inhibitory interneurons in our model to reproduce the basic neuronal circuit of the DCN where projection neurons (PNs) are inhibited by type-II and wide-band inhibitory units. By altering the strengths of the inhibitory connections, we can tune the PN responses to resemble the response characteristics of DCN principal cells like type-III and type-IV responses. We then analyze how the activity of the model neurons is changed by hearing loss through different kinds of cochlear damage. After hearing loss, the mean activity of the model neurons depends on the severity of cochlear damage and the strengths of excitation and inhibition.

In our model, homeostatic plasticity stabilizes the mean firing rate of the PNs by scaling the strengths of excitatory and inhibitory synapses, which also influences the spontaneous firing rate. After hearing loss and homeostasis, the spontaneous firing rate of PNs depends on the type and severity of cochlear damage and on the ratio of the mean to the spontaneous firing rate before hearing loss. Only those PN types where excitation dominates over inhibition become hyperactive. We observe hyperactivity in type-III and type-IV-T PNs, but not in type-IV PNs whose mean rate is close to the spontaneous rate.

Finally, we apply our model to data from tinnitus patients [2] and predict changes in spontaneous firing rates of auditory neurons from the patients' audiograms. Estimates of tinnitus pitch based on the hyperactivity patterns in the model DCN are consistent with observed tinnitus pitch. We conclude that hyperactivity through the action of homeostatic plasticity after hearing loss may be the basis for a tinnitus sensation.

References

1. Schaette R, Kempter R: **Development of tinnitus-related neuronal hyperactivity through homeostatic plasticity after hearing loss: a computational model.** *Eur J Neurosci* 2006, **23:**3124-3138.
2. König O, Schaette R, Kempter R, Gross M: **Course of hearing loss and occurrence of tinnitus.** *Hear Res* 2006, **221:**59-64.