Modulation of cochlear amplifier performance by contralateral suppression

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Sound delivered to the opposite ear is known to activate medial olivocochlear (MOC) efferent neurons in the auditory system that causes suppression of outer hair cell (OHC) electromotiilty (reverse transduction) and micromechanics in the ipsilateral ear which underlies the 'cochlear amplifier'. This is referred to as 'contralateral suppression' (CS). This modulation of OHC function can be measured as a decrease in the amplitude of the Distortion Product Otoacoustic Emissions (DPOAE). The CS adaptation is an extremely rapid response (ms to seconds). CS contributes to protection against noise-induced hearing loss (Rajan, 2000) as well as for antimasking in noisy environments for attentive hearing (Kawase, Delgutte & Liberman, 1993). Here we describe a new model for evaluating the regulation of the 'cochlear amplifier' by CS which matches the fast adaptation profile of CS to the dynamics of transient noise loading on the cochlea.

Quadratic (f1-f2) DPOAEs were measured in 129/Bl6/J mice prior to, and immediately following exposure to 20 seconds of ipsilateral sound (band limited) with, or without, contralateral stimulation. These experiments were conducted on mice under ketamine / xylazine / acepromazine anaesthesia as previously described (Cederholm *et al.*, 2012) and in accordance with University of New South Wales Animal Care and Ethics Committee approval.

Our results to date (n = 9) show that contralateral acoustic stimulation given along with the ipsilateral sound mitigates the noise-induced suppression of the cochlear amplifier, promoting a more rapid recovery of sensitivity in the immediate post-noise period. The initial test with ipsilateral noise caused a reduction in the quadratic DPOAE of 9.7 ± 1.59dB from an average of 104.2 ± 1.00dB. When CS was added to the ipsilateral noise, the DPOAE recorded at cessation of the noise was reduced by 6.6 ± 1.26dB. Thus CS produced on average 3.1 ± 0.80dB less noise-induced reduction in 'cochlear amplifier' adaptation (P = 0.005 paired t-test). This is consistent with the proposed role of CS in enabling rapid modulation of sound transduction in the face of a dynamic acoustic environment. This model could prove extremely useful when studying fast cochlea dynamics and MOC activity.

Rajan, R. (2000) Centrifugal pathways protect hearing sensitivity at the cochlea in noisy environments that exacerbate the damage induced by loud sound. *Journal of Neuroscience* **20:** 6684-93.

Kawase T, Delgutte B, Liberman MC. (1993) Antimasking effects of the olivocochlear reflex. II. Enhancement of auditory-nerve response to masked tones. Journal of Neurophysiology **70:** 2533-49.

Cederholm JM, Froud KE, Wong AC, Ko M, Ryan AF, Housley GD. (2012) Differential actions of isoflurane and ketamine-based anaesthetics on cochlear function in the mouse. *Hearing Research* doi: 10.1016/j.heares.2012.08.010.

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