in the 30-min group, outer hair cells loss in the lower half of the basal turn in 1/4 animals in the 1-h group, and outer hair cells loss of the base turn in almost all animals in the 2-h group. Micro-thin sections of RWM in the 2-h group showed the degeneration of the outer epithelium and many erythrocytes infiltration in the scala tympani adjacent to the RWM. Surface preparation of the utricular macula showed no hair cells loss in any groups. These findings suggest that the application of Burow's solution put on the RWM for 1 h or longer induces damage to the outer hair cells in the cochlea, but not the utricular hair cell, through the damaged RWM. Physicians should pay attention to this adverse effect of Burow's solution when applied into the middle ear cavity.

600 Proteomic Analysis of Mefloquine Ototoxicity

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In previous studies, we have shown that the anti-malarial drug mefloquine damages rat cochlear hair cells and auditory nerve fibers by apoptosis. Using RT-PCR apoptosis-focused gene arrays, we have reported changes in a wide range of apoptotic and anti-apoptotic signals in the basilar membrane (hair cells-supporting cells) and spiral ganglion regions of rat cochlear organotypic cultures treated with 100 µM of mefloquine for 3 h. Comparison of sensory and neuronal mRNA responses suggested both differences and tissue specific mechanisms induced during the early states of ototoxicity. In the present study, we assayed changes in this model (same dose and time point) in 200 cell signaling proteins using an antibody microarray. Proteomic analysis also revealed tissue specific regulation of gene expression. Changes in expression of signaling proteins were much less than changes in levels of mRNA indicating the possibility of important translational control of the balance between cell survival and cell death responses. Supported in part by NIH grant R01 DC06630.

601 Amelioration of Noise-Induced and Age-Related Hearing Loss in the Augmented Acoustic Environment (AAE)

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Augmented acoustic environment (AAE) is a paradigm in which animals are exposed to a non-traumatic noise and was first introduced by Turner and Willott (1998) to ameliorate progressive genetic hearing loss (HL) in mice by initiating the treatment around the time of the manifestation of HL. AAE is also known as "acoustic enrichment" which has been shown to ameliorate noise-induced hearing loss (NIHL, Norena and Eggermont, 2005; Niu et al., 2004). We have used the AAE paradigm to examine the protective effects against NIHL and age-related HL (ARHL) and the influence on outer hair cell (OHC) death.

To examine the AAE effects on NIHL and OHC death, chinchillas (n=5) were exposed to a traumatic noise (one octave-band with the center frequency at 4 kHz at 107 dB SPL) for 1 hour and then immediately exposed to a non-traumatic continuous noise (4 – 20 kHz at 80 dB SPL) for 3 days. An acoustically-deprived group (n=5) wore earplugs for 3 days starting immediately after the same noise exposure. The results showed that the AAE/acoustic enrichment group showed significantly smaller ABR threshold shifts at 4 and 8 kHz and fewer numbers of deteriorated OHC (apoptotic, necrotic, and missing OHCs) compared to the deprived animals.

In order to investigate whether the AAE/acoustic enrichment ameliorates ARHL when it was initiated after the manifestation of HL, 16-month-old Fischer 344/NHsd rats (n=5) were exposed to the non-traumatic noise (4-20 kHz and 80 dB SPL), for 12 hours/day for 3 months. Six unexposed rats were used as controls. ABR thresholds were obtained before the treatment, and re-tested at 2, 6, 9, and 13 weeks after the initiation of the treatment. The results showed that the "vector" for ARHL was essentially stopped by the introduction of AAE and by 13 weeks of the treatment, the control group had 10-20 dB larger ABR threshold shifts at 20-40 kHz compared to the AAE group. Additionally, fewer numbers of deteriorated OHCs were observed in the AAE group compared to the control group. In conclusion, our results from both studies demonstrated that non-traumatic noise exposure can prevent deterioration of OHCs caused by a traumatic noise and aging.

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602 Effect of Modulating Vitamin C Levels on Age-Related Hearing Loss

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The free radical theory of aging asserts that buildup of macromolecular damage from oxygen free radicals leads to the functional decline associated with aging in multicellular organisms. Age-related hearing loss (ARHL) has been considered to be related to excess generation of free radicals. For example, McFadden et al. (Neurobiol Aging 1999) have shown that copper/zinc superoxide dismutase deficiencies increase the vulnerability of the cochlea to damage associated with normal aging through metabolic pathways involving the superoxide radical. Vitamin C (VC) or L-ascorbate is an essential nutrient for a large number of higher primate. It is made internally by almost all organisms including mice, but not humans. It is known that VC deficiency causes scurvy in humans. The pharmacophore of VC is the ascorbate ion and in living organisms, ascorbate acts as an antioxidant since it protects the body against oxidative stress. We have established senescence marker protein 30 (SMP30)/

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