ABSTRACTS OF THE THIRTY-SECOND ANNUAL
MIDWINTER RESEARCH MEETING

ASSOCIATION FOR RESEARCH IN OTOLARYNGOLOGY

February 14-19, 2009

Baltimore Marriott Waterfront

Baltimore, Maryland, USA

ABSTRACTS OF THE THIRTY-SECOND ANNUAL MIDWINTER RESEARCH MEETING OF THE

Association for Research in Otolaryngology

February 14 – 19, 2009
Baltimore, Maryland, USA

Peter A. Santi, PhD
Editor

Association for Research in Otolaryngology
19 Mantua Road, Mt. Royal, NJ 08061 USA

134 Decomposition of Mechanical Stresses in Acoustic Trauma

Qunfeng Cai[1], Bo Hua Hu[1]
[1]*State University of NY at Buffalo*

Acoustic overstimulation generates multiple forms of mechanical stresses, including stretching, shearing, bending, compression/decompression. Interaction of these mechanical stresses can activate acute hair cell (HC) apoptosis. However, it remains unclear how each individual stress induces acute apoptosis. Here, we reported a cochlear model of compression/decompression injury induced by exposure to intense noise. The model was generated by surgical occlusion of the round window of the cochlea of the Sprague Dawley rat. Blocking the round window of the cochlea eliminated the induction of the pressure difference between the scala typmpani and the scala vestibuli, the driving force for basilar membrane vibration during acoustic stimulation. Under this condition, noise stimulation will generate only the pressure fluctuation without inducing the basilar membrane motion, the source of stretching and shearing stresses. Consequently, only the compression/decompression stress was induced. After the surgery, we found 10 to 15 dB threshold shifts across the frequency range between 5 and 40 kHz, which were probably due to the reduction of basilar membrane motion. The animals with the round window closure were exposed to a broadband noise at 120 dB SPL for one hour. After the noise exposure, the cochleae were examined for assessment of HC membrane permeability and apoptotic activity. As compared with the cochleae without the round window closure, the cochleae having the round window closure exhibited a marked reduction in the membrane permeability. The number of apoptotic cells was also reduced. The reduction was more evident in the second cochlear turn, the initial site of HC pathogenesis. The results suggest that removing stretching and shearing stresses reduces acute membrane damage, which in turn prevents the cells from entering the apoptotic pathway.(Supported by NOHR funds and New Faculty Startup funds from University at Buffalo)

135 Changes in E-Cadherin in the Cochlea After Traumatic Noise Exposure

Chiemi Tanaka[1], Guang-Di Chen[1], Bo Hua Hu[1], Richard Salvi[1], Donald Henderson[1]
[1]*Center for Hearing and Deafness, SUNY at Buffalo*

Leonova and Raphael (1997) reported that the adhesion molecule, E-cadherin, alters distribution patterns resulting from scar tissue formation in the cochlea after administration of kanamycin. They speculated that E-cadherin may play an important role in the transmission of sound and the process of the scar tissue formation in the cochlea after ototoxic drug administration. We investigated changes in E-cadherin in the organ of Corti in rats immediately after exposure to a traumatic noise (10-20 kHz broad band noise at 110 dB SPL for 4 hours) using a confocal microscope. Similar to the previous study (Leonova and Raphael, 1997), E-cadherin was found in the reticular lamina. However, intense E-cadherin staining that outlined the apical part of the outer hair cells (OHCs) was observed in the reticular lamina in the noise-induced damage lesion (basal turn) immediately after the noise exposure. This phenomenon was not observed in the unexposed control animals. The OHCs that showed localization of E-cadherin, were found to be apoptotic or have missing nuclei. This E-cadherin localization may be related to the mechanical stress caused by the intense noise since this phenomenon was not previously observed in the kanamycin-treated cochlea.
This study was supported by NIOSH grant 1R01OH008113-01A1.

136 Noise-Induced Focal Lesions in the Organ of Corti: Distribution and Cell Death Pathways

Barbara A. Bohne[1], Gary W. Harding[1]
[1]*Washington University School of Medicine*

Studies have been conducted to identify what death pathways OHCs follow after moderate-severe noise exposures. Identified pathways include oncosis/necrosis, apoptosis & a non-apoptotic, non-oncotic pathway. IHCs, pillar & Deiters cells are also destroyed by noise. In order to develop treatment strategies that will minimize noise-induced hearing loss, it is important to identify death pathways in all cell types in the organ of Corti (OC). Eighteen chinchillas were exposed for 1 h to a 4-kHz OBN at 108 dB SPL. Recovery times were < 1 d to 30 d post-exposure. At termination, animals were anesthetized; their cochleae surgically exposed & fixed in-vivo with 1% buffered OsO_4. The intact cochleae were dehydrated, embedded in plastic & dissected into flat preparations. For each ear, OC length was measured & losses of cells were quantified throughout the OC. Focal hair-cell lesions were identified [≥ 50% loss of OHCs, IHCs or both (i.e., combined) over at least 0.03 mm] and death pathways in dying cells determined. All but one cochlea had one or more focal hair-cell lesions. In different cochleae, some lesions covered a narrow portion of the OC & were close to the 4-kHz OBN location, while some lesions were spread over ~ 50% of the OC. In cochleae with two or more lesions, variable-length areas of reduced damage separated the lesions. Twice as many OHC lesions as combined & IHC lesions were identified. OHC & combined lesions were greater in length than IHC lesions & usually included missing pillars & Deiters cells. IHC lesions rarely involved other cell types. This suggests that IHC lesions are generated by a different mechanism than OHC & combined lesions. Within the OHC & combined focal lesions, dying hair cells were identified that were following the oncotic, apoptotic & non-apoptotic, non-oncotic death pathways. The identification of death pathways followed by IHCs in IHC focal lesions & by supporting cells in OHC & combined focal lesions is in progress.

137 The Effect of Acoustic Trauma on Cochlear Pericytes

Xiaorui Shi[1]
[1]*Oregon Health & Science University*

Cochlear blood flow is markedly affected by acoustic trauma, but the concomitant changes in cochlear pericytes are unknown. In this study, we investigated the effect of