## 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

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

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# Association for Research in Otolaryngology

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**February 14 – 19, 2009**
**Baltimore, Maryland, USA**

**Peter A. Santi, PhD**
*Editor*

Association for Research in Otolaryngology
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**After attending the Scientific Meeting participants should be better able to:**

1. Understand current concepts of the function of normal and diseased ears and other head and neck structures.
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3. Understand what are considered to be the key research questions and promising areas of research in otolaryngology.

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For Example:
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**President's Message 2009**

Welcome back to Charm City! We return to the Baltimore Marriott Waterfront at Inner Harbor East for the 32nd Annual MidWinter Meeting of the Association for Research in Otolaryngology. As you know, this will continue as our odd-year meeting site through 2015. So, you can begin to establish your favorite restaurants, watering holes and entertainments. But be aware that this district of Baltimore has continued its remarkable renaissance and so will look quite different even from our last visit.

This is a good thing, with even more shops and restaurants nearby, as well as a first-run cinema and Whole Foods Market (…barbecued 'soy riblets'?). It's still an easy walk to the core of the Inner Harbor, Little Italy and Fells Point, and a short cab ride to literally hundreds of restaurants throughout Baltimore, from Charles Village to Canton. The National Aquarium is close by and the Visionary Arts Museum and Science Center are around the Inner Harbor. The Walters Art gallery is downtown. A personal favorite is the extraordinary collection of Matisse paintings (part of the Cone sisters' collection, itself a unique treasure) at the Baltimore Museum of Art. This is next to the Johns Hopkins Homewood campus (about 3 miles from the hotel). And of course there are nearby music and dance clubs ranging from reggae to rock; not to mention the ARO's own 'Hair Ball' Wednesday evening in the Marriott ballroom.

The meeting this year is graced with another stellar collection of **Symposia**. These include: 1. Importance of Temporal vs Spectral Fine Structure for Pitch; 2. Mechanisms of Deafness Caused by Genetic Mutations: What Did We Learn From the Mouse Models? ; 3. Vestibular Compensation: New Clinical and Basic Science Perspectives; 4. From Psychophysics to Speech and from Physiology to Engineering: Jack Cullen's Contributions to Hearing Science; 5. New Scientific Developments in Auditory Processing Disorder; 6. Molecular Basis of Prosensory Specification in the Mammalian Cochlea; 7. Novelty Detection in the Auditory System: Correlating Animal and Human Studies. The Presidential symposium on Sunday is titled "Comparative Studies of the Ear - of (More Than) Mice and Men" and will provide views of inner ear function from moths to monkeys. The award of Merit winner, Dr. M. Charles Liberman will present his **Presidential Lecture** on Tuesday evening entitled "Connecting Hair Cells with Brain Cells: Afferent Responses and Efferent Feedback in Hearing and Deafness". Dr. David Ryugo will provide a synopsis of Charlie's career at the Awards Ceremony. Saturday's **Short Course** will cover Advanced Microscopy Techniques and **Workshops** will be presented by the NIDCD, the Patient Advocacy Committee, Media Relations Committee and the Animal Research Committee.

Remember to attend the **Business Meeting** Monday evening at 6. In addition to an update of the Association's affairs, new members of the nominating committee are chosen and other issues of concern are highlighted. Further, at this year's Business Meeting we will draw winners of the 'Exhibitor's Scavenger Hunt', prizes to include popular gizmos such as iPod, Wii-Fit, etc. So, please attend to play your part in Association business, and for the possibility of scoring cool toys.

The mid-winter meeting could not occur without the diligent and effective administration of Talley Management. Also, many members of the ARO dedicate hours of their time to program organization, symposium and workshop development, short courses and more. We are indebted to AAO-HNSF, DRF, AAAF and the Collegium Oto-Rhino Laryngologicum Amicitiae Sacrum -US Group, Inc, for their donations of travel funds for students and fellows. The collected efforts and generosity of all these deserve our recognition and thanks.

As I read through the program book I find the dilemma of choice more acute each year. There are just too many interesting titles to choose among. We continue to see remarkable growth in scientific diversity and depth. Our mid-winter meeting is a testament to the creativity and hard work that so many dedicate to understanding our related sciences. I hope you will enjoy this 32nd ARO as much as I will.

Paul A. Fuchs

# M. Charles Liberman

# 2009 Award of Merit Recipient

# M. Charles Liberman

## 2009 Recipient of the Award of Merit

The 2009 ARO Award of Merit will be given to Charlie Liberman for his many exceptional contributions to the field of auditory neuroscience. His research has spanned many aspects of hearing and deafness, including the effects of acoustic overstimulation on the inner ear, the subtypes of auditory nerve fibers and the correlation of their structure and function, and the role of the efferent innervation to the inner ear. Time and again, he has made significant and considerable advances in our knowledge. A hallmark of Charlie's work is his insightful and careful attention to detail and how these details evolve into significant and bedrock observations. Equally important is his remarkable ability to incorporate new concepts and techniques into his assault on old and new questions. He has been a leader in our field for much of his career.

Charlie comes from a family of scholars; his father, Alvin M. Liberman was Professor of Psychology at the University of Connecticut, Professor of Linguistics at Yale University, and President of Haskins Laboratories from 1975 - 1986. His mother, Isabelle Yoffe Liberman, was also a researcher at Haskins and a Professor at the University of Connecticut. His brother, Mark Liberman, is a Professor at the University of Pennsylvania in the Department of Linguistics and the Department of Computer and Information Sciences. And his sister, Sarah Ash, is an Associate Professor in the Department of Food Science at North Carolina State University.

Charlie's scientific career has taken place entirely in Boston: at Harvard and its medical school, at the Massachusetts Eye & Ear Infirmary, and within Harvard-MIT's program in Speech and Hearing Bioscience and Technology. Charlie's introduction to auditory physiology began when, as senior majoring in Biology at Harvard College, he took a readings class with Nelson Kiang at the Eaton-Peabody Laboratory of the Massachusetts Eye & Ear Infirmary. In the same lab as a graduate student, Charlie's Ph.D. work documented how acoustic overstimulation affected the inner ear and the responses of its nerve fibers (published as a supplement to Acta Otolarygologica in 1978). After narrow-band noise was used to damage hearing in a particular frequency region, he recorded responses of single auditory nerve fibers and documented their abnormal tuning curves. After characterizing the nerve's responses, he examined in detail the histopathology of the individual cochleas of each experiment. This made possible the most important aspect of these experiments: a correlation of the changes in the hair cells with the abnormal responses of the nerve. This structure/function relationship had never before been done with the precision of single-nerve fiber recordings. These studies answered questions like, "How is a mild loss of outer hair cells reflected in the tuning curve of an auditory nerve fiber?" Later studies by Charlie took this question to a finer level, examining how damage to the stereocilia on hair cells altered the responses of the nerve fibers. In addition to examining the stereocilia in the electron microscope, Charlie developed embedding and specimen-thinning techniques to enable their examination in the light microscope, a considerable technical feat. From the noise-exposure studies came the question of whether such damage could occur during a lifetime of "routine" exposure to sound. A study, now classic, used animals that had been reared in a low-noise chamber to prevent any significant exposure. Their nerve fiber responses showed exceptionally low thresholds, indicating that routine noise exposure in fact does take its toll on hearing. Charlie's investigational talents and the ability to pose such interesting research questions are his hallmarks.

Along with these studies of the damaged hearing organ, Charlie has made a host of contributions to normal anatomy and physiology of hearing. His work demonstrates the importance of the subgroups of nerve fibers as distinguished by their rates of spontaneous discharge, which correlates with other important properties such as threshold, point of contact with the inner hair cell, and central anatomy in the cochlear nucleus. Some of these studies originated in postdoctoral work with Sandy Palay at Harvard Medical School's Department of Anatomy, where serial-section electron microscopy was used to follow the peripheral terminals of auditory nerve fibers and demonstrate the types of synapses that they receive from the hair cell and from olivocochlear fibers. One of Charlie's most elegant contributions was to establish with precision the cochlear frequency mapping of auditory nerve fibers. For this, he brought the technique of single-unit labeling to the auditory system - after obtaining the nerve fiber's tuning curve and characteristic frequency, the fiber is injected with a neural tracer that could be followed to the point of contact with the inner hair cell along the cochlear spiral. Fibers of all spontaneous rates share a common "tonotopic" mapping, which is continued in the central auditory pathway as a fundamental organizing principle.

Charlie has greatly advanced our knowledge of the olivocochlear system, which sends messages from the brain out to the organ of Corti. His work shows the large differences in responses and innervation patterns for olivocochlear neurons compared to auditory nerve fibers. For example, the olivocochlear neurons are "jazzed up" by previous sound exposures. Importantly, they protect the ear from acoustic overstimulation and lessen the effects of noise masking. His current work is beginning to untangle the possible roles and actions of the lesser-known subgroup, the lateral olivocochlear neurons. In the most recent decade, Charlie has pioneered the use of genetically engineered models in the study of hearing. He and colleagues tested the mouse lacking the gene for the alpha 9 cholinergic receptor, the receptor that normally

mediates the effects of olivocochlear neurons on outer hair cells. This “knockout” mouse lacks the usual effects of olivocochlear stimulation and is thus functionally de-efferented. He and colleagues showed that outer hair cells from the Prestin “knockout” mouse lack electromotility, and that without this molecular motor there is a hearing loss of 40-60 dB. Recent tests of the alpha 9 cholinergic receptor “knockin” show that it has exceptional olivocochlear effects and has exceptional resistance to acoustic overstimulation.

These accomplishments are remarkable, and along with them Charlie’s talents are displayed in remarkable teaching and administration. He is an exceptional teacher, having sponsored numerous graduate students and fellows, and directing the graduate course on the peripheral auditory system for over 15 years. He was the president of ARO (1996-7). In 1998, after the retirement of Nelson Kiang, Charlie became the Director of the Eaton-Peabody Laboratory and recently became the first Harold Schuknecht Professor of Otology and Laryngology at Harvard Medical School. As a lab director, he creates an exceptionally conducive environment for research and as a colleague, he takes a personal interest in our grants and manuscripts. As a scientific role model, he sets the bar high in terms of scientific rigor, thoroughness, and clarity in thought and writing. He is held in universally high regard by his colleagues.

This award of merit is a richly deserved symbol of recognition for Charlie Liberman, and on behalf of the ARO, we congratulate him for it.

M. CHRISTIAN BROWN

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