These results suggest that the ahl mutation in the Cdh23 gene affects the hair cells in the cochlea, but not the vestibular system. Supported in part by NIH grant R01 DC005827-05 to KJ.

580 Effect of Chronic Salicylate Treatment on Age-Related Cochlear Degeneration

Guang-Di Chen[1], Manna Li[1], Chiemi Tanaka[1], Eric Bielefeld[1], Mohammad Habiby Kermany[1], Richard Salvi[1], Donald Henderson[1]
[1]*SUNY at Buffalo*

Salicylate (aspirin) is a widely used drug in clinics. Acute application of salicylate may cause reversible hearing loss, reduction of distortion product otoacoustic emission (DPOAE), and loss of outer hair cell (OHC) electromotility. A long-term application may cause tinnitus. Interestingly, it has been reported that a long-term salicylate application up-regulated expression of prestin, the OHC motor protein, consequently leading to an increase of OHC electromotility and DPOAE. Salicylate has also been shown to have protective effect on ototoxicity induced by noise, cisplatin and gentamicin. In the current study, aging Fischer 344 rats (18 months old) were treated with sodium salicylate at a dose of 100 mg/kg for 2 times per day for 5 days per week for 3 weeks. DPOAE and auditory brainstem response (ABR) were recorded and compared before and after the treatment. The OHC-related cochlear functions including cochlear microphonics (CM) and cochear amplification were also determined. Finally, prestin levels in OHCs were examined immunohistochemically. It appeared that the treatment delayed some aging processes.
This study was supported by NIOSH grant 1R01OH008113-01A1

581 Glycine Receptor Subunit Changes in DCN of Rats with Behavioral Evidence of Presbycusis

Hongning Wang[1], Lynne Ling[1], Jeremy G. Turner[1,2], Jennifer L. Parrish[1,3], Larry F. Hughes[1], Donald M. Caspary[1]
[1]*SIU school of medicine,* [2]*Illinois College, Jacksonville,* [3]*Illinois State University*

Presbycusis, age-related hearing loss, can be considered a consequence of progressive peripheral auditory deafferentation. Previous studies suggest that temporal processing deficits observed in presbycusis may partially result from functional loss of the inhibitory glycinergic neurotransmission in dorsal cochlear nucleus (DCN). The present study assessed age-related behavioral gap detection and neurochemistry changes of postsynaptic glycine receptor (GlyR) subunits and their anchoring protein gephyrin over fusiform cells of young (7-8 month) and aged (28-29 month) Fischer Brown Norway (FBN) rats. Aged rats showed significantly (20-30dB) higher ABR thresholds across all tested frequencies and longer gap detection thresholds compared to young FBN rats. *In situ* hybridization and quantitative immunocytochemistry were used to measure GlyR subunit message and protein levels. There was a significant age-related increase in $GlyR\alpha_1$ subunit message but significant decreases in protein levels. $GlyR\alpha_2$ showed significant age-related decreases in both message and protein levels. Gephyrin message and protein were significantly increased in aged DCN fusiform cells. The pharmacologic consequences of these age-related subunit changes were assessed using [^{3}H] strychnine binding. In support of the age-related decrease of α_1 subunit protein, there was a significant age-related decrease in the number of GlyR binding sites with no significant change in affinity.
Previous studies suggest that gephyrin may act as a retrograde GlyR intracellular transporter contributing to an age-related decrease in $GlyR\alpha_1$ protein and binding in DCN. These changes may reflect an effort to re-establish a homeostatic balance between excitation and inhibition impacting on fusiform cells in aged animals by down-regulating glycinergic inhibition. This age-related down-regulation comes at the cost of accurate temporal acuity observed at the single cell and behavioral level.
Supported by NIH DC00151 and ATA.

582 Behavioral Effects of Unilateral Noise Exposure on Young and Aged Mice

Richard Meyerholz[1], Deb Larsen[1], Jennifer L. Parrish[1], Larry F. Hughes[1], Jeremy G. Turner[1,2]
[1]*SIU School of Medicine,* [2]*Illinois College*

Young (n=12) and aged (n=13) mice were anesthetized with isoflurane and given unilateral noise exposure for one hour using the same acoustic parameters used in previous studies with rats (116 dB SPL, 16 kHz octave band signal). Age-matched control mice were sham exposed for one hour (n=5 young, n=7 aged). ABR thresholds were collected immediately before and after noise exposure for each ear. Mice were behaviorally tested before noise exposure and at 1, 3, 7 and 14 days after noise exposure. Behavioral measures included prepulse inhibition and gap-induced inhibition of the startle reflex. Previous studies have used such tests to assess tinnitus and hyperacusis-like behaviors in rats. Frequency bands tested included 1 kHz bandpass signals centered at 4, 8, 10, 12, 16, 20, 24, and 32 kHz, as well as broadband noise, each presented at 60 dB SPL. Preliminary analyses suggest young and aged mice responded differently to the noise exposure, but that in both cases the results were consistent with noise inducing a hyperactive auditory system. At two weeks post noise exposure, aged noise-exposed mice exhibited significant gap detection deficits at 8 kHz (as well as a trend at 10 kHz). However, young mice exhibited significant gap detection improvements at 20 kHz (as well as trends for better responses throughout the entire 12-24 kHz range). These age-related responses, and their temporal development following noise exposure, are discussed in the context of the possible relationship between tinnitus and hyperacusis.

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
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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.
2. Understand current controversies in research methods and findings that bear on this understanding.
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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