was less than or equal to 10 days; 2) The exposure level was less than or equal to 108 dB SPL; & 3) Focal lesions were less than 1.5 mm in size. The data sets included a variety of exposures ranging from those that were high-level, short duration to those that were moderate-level, moderate duration. The % location of the center of each focal lesion was determined. Means, SDs & medians were calculated for lesion size for each OBN. Histograms were then constructed from the % location data using 2.0% bins & the counts were graphed relative to total number of lesions. For the 4-kHz OBN, 94% of the lesions were in the basal half of the OC & 6% were in the apical half. For the 0.5-kHz OBN, 29% of the lesions were in the apical half of the OC & 71% were in the basal half. The mean lesion size was 1.48% & 0.68% for the 4-kHz & 0.5-kHz OBN, respectively, with medians of 1.10% & 0.50%. The mean lesion size (in mm) for the 0.5-kHz OBN was less than half that for the 4-kHz OBN. For the 4-kHz OBN, a histogram of the % location of lesions showed that most occurred in the 5-7-kHz region, at & just above the upper edge of the OBN. Clusters of lesions were also found around 8 & 12 kHz. A cluster was present at & just below the lower edge of the OBN, as well as in the 1.5-kHz region. For the 0.5-kHz OBN, a histogram of the % location of lesions showed clusters at 0.25, 0.75 & 1.5 kHz in the apical half. In the basal half, the pattern was very similar (Pearson's r=0.69) to that seen with the 4-kHz OBN. The distribution of basal-turn lesions suggests that the 4-kHz & 0.5-kHz OBN are damaging that region of the cochlea in the same way.

33 Histopathological Changes in the Cochlea Following Exposure to Low-Frequency Noise

Steve Lee[1], Barbara A. Bohne[1], Gary W. Harding[1]

[1]*Washington University School of Medicine*

Thirteen chinchillas were exposed for 24 h to a 0.5-kHz OBN at 95 dB SPL. The cochleae of 8 animals were fixed at 0-d post-exposure; 5 were fixed after 1-2 wks of recovery. To keep cellular debris from washing away, all cochleae were plastic-embedded before being dissected into flat preparations. By phase-contrast microscopy, hair-cell losses were determined from apex to base. Damage consisted of scattered loss of OHCs in the apical half of the organ of Corti (OC) & small focal lesions (i.e., > 50% hair-cell loss over at least 0.03 mm) in the basal half. These specific patterns of loss suggest that noise damaged the apex & base by different mechanisms. In order to estimate the timing of cell loss, differential counts of missing & severely injured cells were performed. The presence of immature phalangeal scars & necrotic, oncotic & apoptotic hair cells indicates a recent loss while mature phalangeal scars indicate a long-standing loss. In the apical half of the OC in both groups of animals, many of the phalangeal scars replacing the missing OHCs were immature. Cellular debris was seen in the OC fluid spaces beneath these scars. By TEM, the debris was found dispersed in the Nuel spaces & consisted of vesicles of various sizes, small granules, shrunken & swollen mitochondria &, rarely, fragments of plasma membrane. TEM also revealed the presence of cellular debris in the endolymphatic space near the reticular lamina. This latter finding indicates that the barrier function of the reticular lamina broke down temporarily when the hair cells degenerated, before phalangeal scars formed. Debris in the Nuel spaces was not surrounded by plasma membrane as would be the case if the hair cells had been apoptotic before they died & then formed apoptotic bodies. In the basal half of the OC, focal lesions were found in 2 of eight 0-day & 3 of five 1-2 wk animals. In the 0-day ears, oncotic OHCs were found at the edges of the basal-turn lesions. In the 1-2 wk ears, the basal-turn lesions primarily consisted of immature phalangeal scars. The appearance of debris in the apex & base suggests that many of the OHCs were oncotic rather than apoptotic before they disappeared.

34 Intense Noise Causes Damage to the OHC Lateral Wall Leading to Hearing Loss

Guang-Di Chen[1]

[1]*SUNY at Buffalo*

The cochlear active process results in a 40-60 dB cochlear amplification. Outer hair cells (OHCs) constitute an important part of cochlear micro-mechanics and are believed to be the driving force of the cochlear active process by way of their electromotility. The OHC membrane skeleton, consisting of F-actin and spectrin, maintains the unique OHC cylindrical shape and provides stiffness to the cell. In addition to its well known basic functions, the OHC plasma membrane is a main contributor to OHC axial stiffness. Prestin, a membrane protein, has been recently recognized as the OHC motor protein. Changes in the OHC lateral wall may affect OHC electromotility and/or cochlear micromechanics and, subsequently cochlear sensitivity. In this report OHC membrane fluidity (by laser bleach approach), gene expression of beta-actin, beta-spectrin, and prestin were determined after noise exposure. The noise exposure caused a reduction of OHC membrane fluidity and a time-dependent gene expression of the proteins in the OHC lateral wall. The noise-induced changes were associated with permanent threshold shifts. The motor protein appeared to be the most sensitive to noise trauma among the three proteins. The data suggest that non-lethal injuries in the OHC lateral wall may cause loss of the OHC electromotility or the cochlear micromechanics leading to a reduction of cochlear amplification and then cochlear sensitivity.

35 Mechanisms of Oxidative Stress in the Potentiation of Noise-Induced Hearing Loss by Acrylonitrile

Benoit Pouyatos[1], Laurence Fechter[1]

[1]*Loma Linda VA Medical Center*

Acrylonitrile (ACN), one of the top 50 chemicals produced in the world, is a very powerful pro-oxidant compound whose metabolism leads to a profound glutathione (GSH) depletion and to a production of cyanide (CN) which, in turn, can inhibit superoxide dismutase (SOD). ACN, by itself, is not ototoxic, but we have shown that it can strongly promote noise-induced hearing loss (NIHL), even at noise levels that do not produce auditory impairment. The mechanism by which ACN renders the cochlea more

vulnerable to noise damage is still unknown, but we hypothesize that the decrease of GSH and/or the inhibition of SOD reduce the cochlear intrinsic anti-oxidant defenses and ultimately create oxidative stress.

In this study, we investigated in adult Long-Evans rats the effect of the alteration of CN production through the ACN metabolism on the promotion of NIHL by ACN. For this purpose, two different drugs were used: (1) Sodium thiosulfate (STS), a CN antidote commonly used in humans in case of CN poisoning and (2) 4-methylpyrazole (4MP) which blocks ACN metabolism through the oxidative pathway, preventing CN formation.

We observed that, while both STS and 4MP drastically reduced CN production by more than 80%, they did not protect against the potentiating effect of ACN. This result suggests that CN is not implicated in the potentiation of NIHL by ACN. Further studies are proposed to determine the role of GSH depletion in the increase of vulnerability to noise induced by ACN.

(supported in part by VA grant C3575R and NIOSH grant OH03481).

Key words: cochlea, noise, oxidative stress, acrylonitrile, superoxide dismutase, glutathione, cyanide.

36 Nuclear Factor-Kappa B Nuclear Translocation in the Cochlea of Mice Following Acoustic Overstimulation

Masatsugu Masuda[1], Reiko Nagashima[2], Sho Kanzaki[1], Masato Fujioka[1], Kiyokazu Ogita[2], Kaoru Ogawa[1]

[1]Keio University, [2]Setsunan University

The mechanisms of noise trauma include mechanical damage, ischemia, excitotoxic damage, metabolic exhaustion, or perturbation of cochlea fluid ion homeostasis. There is increasing evidence to suggest that expression of many molecules in the lateral wall of the cochlea plays important roles in noise-induced stress responses. Since a variety of stress responses occur, there is a possibility that simultaneous modulation of multiple mechanisms can lead to better outcomes of treatment for the hearing impairment resulting from noise trauma than modulation of a single mechanism. This may be provided by modulation of transcription factors, because one transcription factor regulates the expression of many genes in response to a single stimulus that induces tissue damage. In this study, activation of the nuclear transcription factor nuclear factor-kappa B (NF-κB) was investigated in the cochlea of mice treated with intense noise exposure. To assess effects of noise exposure on NF-κB/DNA binding activity in the cochlea, we prepared nuclear extracts from the cochlea at different time points after noise exposure and carried out electrophoretic mobility shift assay. NF-κB/DNA binding was significantly enhanced in several hours after noise exposure. Supershift analysis demonstrated that enhancement of NF-κB/DNA binding was at least in part due to nuclear translocation of p65. An immunohistochemical study also showed that nuclear translocation of p65 was observed in the lateral wall after noise exposure. These results suggest that NF-κB may be involved in expression of genes in response to acoustic overstimulation in the cochlea of mice. Some reports suggest that NF-κB acts as a protective role in the cochlea. However, some reports suggest that it acts as a cytotoxic role. Future research will be necessary to reveal whether NF-κB serves in protecting, killing, or both.

37 A BAD Link to Mitochondrial Cell Death in Noise-Induced Hearing Loss

María Angeles Vicente-Torres[1], Su-Hua Sha[1], Jochen Schacht[1]

[1]Kresge Hearing Research Institue, The University of Michigan, Ann Arbor, MI, USA

In outer hair cells (OHCs), acoustic overstimulation induces calcium overload and activation of mitochondria-mediated death pathways but it remains unknown whether these are interrelated or independent events. We have recently reported that calcineurin is activated (Minami et al., 2004) and total BAD (Bcl-2-associated death promoter) decreased (Vicente-Torres et al., 2005) in the cochlea following noise exposure. We now postulate that the calcium overload associated with acoustic overstimulation can trigger mitochondria-mediated death pathways through the dephosphorylation and, therefore activation of BAD, by the calcium-dependent phosphatase calcineurin and subsequent translocation to the mitochondria.

To explore this hypothesis, CBA/J mice were exposed to a broadband noise (2-20 kHz) causing a permanent threshold shift of about 40 dB at 12 and 20 kHz indicative of damage to the middle and upper basal turns respectively. Widespread loss of OHC was apparent in the basal turn of the cochlea. In unexposed controls, BAD immunostaining showed an essentially cytoplasmic distribution in the cells of the organ of Corti, the lateral wall and the spiral ganglion. Five hours after acoustic overstimulation, mitochondria and BAD were redistributed to the perinuclear region of OHCs in the basal and middle turns while BAD in the apical turn remained unaffected. Furthermore, upregulation of total BAD and the non-apoptotic phospho-BAD (Ser 112) were detected in the stria vascularis and the supporting cells of the organ of Corti.

Our data establish a connection between calcium and mitochondria-mediated death pathways in OHCs and also suggest a dual role for BAD. The translocation of BAD to the mitochondria in degenerating cells is indicative of the activation of the pro-apoptotic capacity of BAD while upregulation of phospho-BAD is consistent with a non-apoptotic role of BAD in these less vulnerable cells.

This work was supported by research grant DC-06457 and core center grant DC-05188 from the National Institute on Deafness and other Communication Disorders, NIH.

38 The Effect of Acoustic Trauma on VR1 Expression in Rat Spiral Ganglion and Cochlear Nucleus

Kristin Myers[1], Thomas Brozoski[1], Carol Bauer[1]

[1]SIU School of Medicine

The vanilloid receptor type 1 (VR1) is one of several members of a family of non-specific cation ion channels

ABSTRACTS OF THE TWENTY-NINTH ANNUAL
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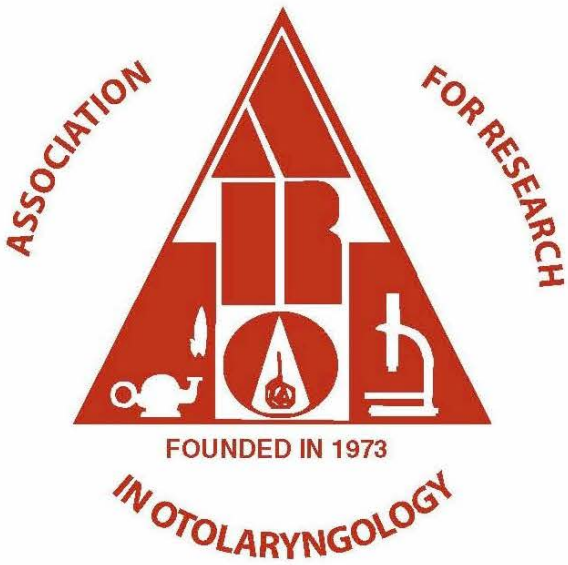

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Peter A. Santi, Ph.D.
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After attending the Scientific Meeting participants should be better able to:

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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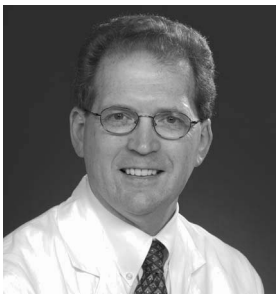

Welcome to Baltimore and to the 29th Annual MidWinter Meeting of the Association for Research in Otolaryngology. Our path to Baltimore for this year's meeting has been complicated and merits some review. In March of 2005, the ARO and the Pyramid Management Group reached an agreement that released the ARO from returning to the Adam's Mark (now Hilton) Hotel in Daytona Beach, Florida. As we have described in previous email messages to the membership, the ARO is making payments to the Pyramid Group as a part of this agreement. Our 2005 meeting at the Fairmont Hotel in New Orleans was successful, and evaluations of the hotel and the conference facilities were very positive. We had hoped to return to the Fairmont in New Orleans for the 2006 MidWinter Meeting. The tragic events surrounding hurricane Katrina forced a change in these plans. Staff members from the ARO Headquarters were in contact with representatives of the Fairmont Hotel soon after the hurricane. We wanted to return to New Orleans for the 2006 MidWinter Meeting to show our support for the people of the city who have endured so much hardship. As events unfolded in September of 2005, it became clear that it would be impossible for us to have our 2006 meeting in New Orleans, and the Fairmont Hotel released us from our contract. We are fortunate that the Waterfront Marriott in Baltimore had availability and appropriate facilities for our meeting.

The program committee under the leadership of John Middlebrooks has done excellent work in putting together an exciting scientific program. Special thanks also go to Bob Shannon who, although he officially rotated off the committee last year, provided valuable insight and advice in the organization of the program. The meeting begins with the presidential symposium on Sunday morning entitled "Vestibular Mechanisms: Achieving Balance in the Ear". Speakers in this symposium will explore aspects of vestibular function from sensory transduction in the periphery to vestibular disorders in patients. Other symposia in the meeting cover molecular biology of ear development, efferent innervation of hair cell systems, protein-protein interactions, toll-like receptors, and activity-dependent plasticity in the auditory brainstem. The workshop addresses topics in adult-onset hearing loss.

Robert Fettiplace has been selected by the Award of Merit Committee and the ARO Council to receive this year's Award of Merit. The title of Robert's Presidential Lecture is "Ion Channel Properties and the Second Filter". We will have the opportunity to celebrate Robert's accomplishments and those of other award recipients at the reception that will follow his lecture.

Eric Young has done wonderful work in leading the *Journal of the Association for Research in Otolaryngology* on a path of excellence. Eric has decided to step down as the editor of *JARO*. Under the guidance of Art Popper and Jerry Popelka, the Publications Committee conducted a thorough search for a new editor and presented recommendations to the ARO Council. The interest expressed by a number of highly qualified scientists in the editorship of *JARO* provides strong evidence of the important

place that the journal holds in our scientific community. We are pleased that Ruth Anne Eatock has accepted the position of editor. Ruth Anne and Eric are working together on the transition process.

Travel awards provide support for research trainees to attend the MidWinter Meeting. We are grateful for the generosity of the NIDCD, Deafness Research Foundation, American Academy of Otolaryngology—Head & Neck Surgery Foundation, and American Academy of Audiology/American Academy of Audiology Foundation (AAAF). The support from these organizations enables the ARO to provide travel awards and to hold the Travel Award Luncheon and Program which honors the recipients of the travel awards and their mentors.

Exhibitors at our MidWinter Meeting keep us informed about products that may benefit our research. They also provide us with information about publications and grants. In some cases they sponsor special receptions and events. Their participation helps us to keep our meeting costs down. Please show your support by visiting their exhibits.

Members of the ARO Council deserve special thanks for their role in organizing this meeting: Bob Shannon (president elect), Bill Brownell (past president), Steve Rauch (secretary treasurer), Peter Santi (editor), David Lim (historian), Ashley Wackym, Doug Cotanche, and Karen Steel. On behalf of all of us, I want to extend very special thanks to Bill Brownell who did enormously important work for the ARO during the interactions with the Pyramid Management Group.

The ARO continues to benefit from the expert organizational activities of the Talley Management Group. Darla Dobson and Lisa Astorga provide outstanding guidance and support for the ARO. We are very fortunate to have them as a part of our team. Members of the Council are working with Lisa Astorga on identification of future meeting sites. We want to keep room prices and expenses down so that as many people as possible can participate in the meeting. At the same time, we want to select cities and hotels that will be pleasant for our meetings.

These next few years are going to be especially critical for the ARO. The finances of our organization are challenged by our exodus from Daytona Beach. The support of every ARO member is vital. During these times, it is especially important for each of us to think about how much the ARO means to us. Our strength lies in the character and the commitment of our members. These attributes will enable us to meet our current challenges and move the organization forward to new levels of excellence.

Lloyd Minor

Robert Fettiplace, PhD

2006 Award of Merit Recipient