Three alternative transcripts were identified, at least one of which appears to be due to the use of an alternative brain-specific promoter and first exon. Localization with an anti-peptide antibody indicated membrane association of TMIE that was ectopically expressed in cultured fibroblasts, consistent with the predicted transmembrane domain in the protein. TMIE in the fibroblasts was concentrated in filopodia-like projections of the cortical actin cytoskeleton, suggesting the protein may have some role in anchoring cytoskeletal elements to the membrane.

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515 Processing and Secretion of Normal and Mutated Cochlin, the Affected Protein in the Sensorineural Deafness and Vestibular Disorder, DFNA9

*Nahid Ganjei Robertson[1], Sara A. Hamaker[1], Vytas Patriub[2], Jon C. Aster[3], Cynthia C. Morton[4] [1]Ob/Gyn, Brigham & Women's Hosp., Boston, MA, [2]Pathology, Brigham & Women's Hospital, Boston, MA, [3]Pathology, Brigham & Women's Hosp., Harvard Medical School, Boston, MA, [4]Pathology & Ob/Gyn, Brigham & Women's Hosp., Harvard Medical School, Boston, MA

Missense mutations in the FCH/LCCL domain of cochlin result in the autosomal dominant hearing loss and vestibular dysfunction disorder, DFNA9. Recombinant FCH/LCCL has a novel fold disrupted by 3 of 4 previously analyzed DFNA9 mutations. Characteristic eosinophilic deposits in DFNA9-affected inner ear structures could be the result of aberrant folding, secretion, or solubility of mutated cochlins, as misfolded proteins accumulate and aggregate, causing toxicity in certain other pathologic states. To study the biological consequences of cochlin misfolding, we expressed normal and mutated cochlins in cultured mammalian cells. Three missense mutations associated with DFNA9 were introduced separately into full-sized cochlin cDNAs, which were transfected into 293T-HEK, COS-7, and 3T3 fibroblast cell lines. Immunocytochemistry revealed localization of normal and mutated cochlins in perinuclear structures consistent with the Golgi apparatus/endoplasmic reticulum.

Western blot analysis of lysates prepared from transfected cells and conditioned media showed equal amounts of normal and mutated cochlins. Normal and mutated cochlins were proteolytically processed and glycosylated equivalently, and non-reducing SDS-PAGE failed to detect any evidence of abnormal cross-linking of the mutated polypeptides. These findings suggest that the pathology associated with cochlin mutations is unlikely to result from abnormalities in secretion, processing, or cross-linking. Alternatively, DFNA9 mutations may disrupt protein-protein interactions involving the FCH/LCCL domain and other components of the extracellular matrix of the inner ear.

516 Calcium-Activated Potassium Channel Genes in *Xenopus laevis* and *Xenopus tropicalis*

*D R Sultemeier, H M Khawaja, W M Bullock, C Trujillo-Provencio, E E Serrano Biology, New Mexico State University, Las Cruces, NM

The calcium-activated potassium channel (*slowpoke*, KCNMA1, KCa1.1) plays a key role in establishing the acoustic frequency selectivity of auditory hair cells (Fettiplace and Fuchs, 1999). We are interested in the regulation of *slo* expression during development of the *Xenopus* inner ear and present here preliminary data comparing *xslo* genes between two *Xenopus* species, *X. laevis* and *X. tropicalis.* The diploid species, *X. tropicalis*, has a shorter generation time than the tetraploid *X. laevis*, and has been selected by the DOE for shotgun sequencing of the genome. We identified expressed *xslo* inner ear sequence by synthesizing and probing a *Xenopus* inner ear SMART (Clontech) cDNA library (Serrano *et al.*, 2001) with a *xslo* cds partial clone. Isolated clones shared 98% nucleotide identity with a full length *xslo* coding sequence (AF274053). We have begun to use PCR-based techniques (Genomewalker, Clontech) to clone the *X. laevis* and *X. tropicalis slo* promoters. Recent studies in *Drosophila* demonstrate that the *slo* transcriptional control region is large and complex (Bohm *et al.*, 2000). To date, we have cloned over 1 kb of *xslo* genomic sequence upstream of the translational start site, and this region shares 96% nucleotide identity between the two species. We determined *xslo* gene copy number using *Xenopus* muscle to prepare genomic DNA for Southern blots. The genomic DNA was restricted with *Bam*HI, *Eco*RI, *Hin*dIII, and *Pst*I, and probed with a *xslo* cds partial clone. In both species, genomic Southern analysis provided strong support for the presence of a single copy of the gene. Taken together, the data provide insight regarding the conservation of *xslo* genes between divergent *Xenopus* species.

Supported by NIDCD R29 DC03292 to EES.

517 Expression of a Dominant-Negative Connexin26 in Mice Causes Disorganization of Organ of Corti and Non-syndromic Deafness

Takayuki Kudo[1], *Katsuhisa Ikeda[2], Yukio Katori[3], Toshimitsu Kobayashi[4], Shigeo Kure[5], Yoichi Matsubara[6] [1]Department of Otorhinolaryngology - Head & Neck Surgery, Sendai, Japan, [2]Department Otorhinolaryngology - Head & Neck Surgery, Tohoku University Graduate School of Medicine, Sendai, Japan, [3]Department of Otorhinolaryngology - Head and Neck Surgery, Tohoku University Graduate School of Medicine, Sendai, Japan, [4]Department of Otorhinolaryngology, Tohoku University School of Medicine, Sendai, Japan, [5]Department of Medical Genetics, Tohoku University Graduate School of Medicine, Sendai, Japan, [6]Department Medicasl genetics, Tohoku University Graduate School of Medcine, Sendai, Japan

Hereditary deafness affects about 1 in 2,000 children and mutations in the GJB2 gene, which encodes gap junction protein connexin26, are the major cause in various ethnic groups. However, the pathogenesis of deafness due to GJB2 mutations remains obscure. Mice with targeted disruption of the gene were embryonic lethal in a previous study. To elucidate the pathological role of connexin26 in the inner ear, we produced transgenic mice carrying a R75W mutation in the GJB2 gene, which was identified in a hereditary deafness pedigree and showed a deleterious dominant-negative effect.

The R75W+mice showed severe hearing loss from an early stage of development. Histological analysis of the mutants revealed hyperplasia of supporting cells, failure in the formation of the tunnel of Corti, and degeneration of sensory hair cells. Despite robust expression of the transgene, no obvious structural change was observed in the stria vascularis and spiral ligament that are rich in connexin26 and generate the endolymph. The high resting potential in cochlear endolymph essential for hair cell excitation was normally sustained.

These results indicate that the GJB2 mutation associated with sensorineural deafness affects the differentiation of supporting cells resulting in disorganization of the organ of Corti, rather than affecting endolymph homeostasis, in mice and probably in human.

518 A Proposed Mechanism of the Gene *Ahl* for Increased Susceptibility to Noise-Induced Hearing Loss.

*Rickie R. Davis[1], Peter Kozel[2], Lawrence C. Erway[3] [1]Hearing Loss Prevention Section, NIOSH, Cincinnati, OH, [2]NIDCD, NIH, Rockville, MD, [3]Biological Sciences, University of Cincinnati, Cincinnati, OH

Animals and humans show differing susceptibility to noise damage even under very carefully controlled exposure conditions. This difference in susceptibility may be related to an uncontrolled genetic component. Common experimental animals (rats, guinea pigs, chinchillas, cats) are outbred?their genomes contain an admixture of many genes.

About 10 years ago Erway et al (1993) demonstrated a recessive gene associated with early presbycusis in inbred mice: *Ahl*. A series of studies have shown that mice homozygous for *Ahl* are more sensitive to the damaging effects of noise.

Recent work has shown that mice homozygous for *Ahl* are not only more sensitive to noise, but also are probably damaged in a different manner by noise than mice containing the wild-type gene.

Recent work in Noben-Trauth?s lab (Di Palma et al., 2001) has shown that the wild-type *Ahl* gene codes for an outer-hair cell specific cadherin. Cadherins are calcium dependent proteins which hold cells together at adherins junctions to form tissues and organs. The cadherin of interest is localized to outer hair and has been termed otocadherin or cdh-23. Reduction in, or missing otocadherin may allow stereocilia to be more easily physically damaged by loud sounds and by aging.

519 Towards Functional Analysis Of Protocadherin 15, The Gene Associated With Ames Waltzer (av) Mouse Mutation And Usher Syndrome 1F

*Kumar N. Alagramam[1], Nithin Adappa[2], Karen S Pawlowski[3], Charles G. Wright[4] [1]Otolaryngology-HNS, The Research Institute, University Hospitals of Cleveland, Case Western Reserve University, Cleveland, OH, [2]Otolaryngology-HNS, Case Western Reserve University, Cleveland, OH, [3]Human Development and Communication Sciences, University of Texas at Dallas, TX, OH, [4]Otolaryngology - Head & Neck Surgery, UT Southwestern Medical Center, Dallas, TX

The av mutation causes deafness and circling behavior in mice. The gene that harbors the av mutation codes for a protocadherin, Pcdh15. Mutation in the human homologue of the mouse Pcdh15 causes Usher syndrome type 1F, establishing the av mouse as a model for deafness in USH1F. Analysis of the predicted amino acid sequence of Pcdh15 shows 11 cadherin repeats, a transmembrane domain and a unique intracellular domain with 2 proline-rich regions, which could serve as binding sites for domains such as SH3 (Src Homology 3). SH3 domains regulate protein localization and often participate in the assembly of multi-component signaling complexes. Using the yeast 2-hybrid system interacting proteins are being screened and candidates genes, such as myosin VIIa, are being tested for interaction in this system. To investigate gene regulation, we analyzed the 'TATA-less' promoter region of Pcdh15 for critical regulatory elements and characterized alternatively spliced products derived from Pcdh15. Results from these experiments will be presented. In the cochlea, electron microscopy of hair cells from the null allele av-3J show severe disorganization of stereocilia bundles as early as E17.5. Hair cell stereocilia from alleles carrying less deleterious mutations (ex. av-J& av-2J) appear fairly normal at P0. However, some cuticular plates of hair cells from av-J and av-2J alleles appear rotated on the apical cell surface by P2, compared to age matched controls. By P5, this rotation is more conspicuous. Although abnormal function of the vestibular system is apparent electrophysiologically (see abstract by Jones SM et al) and behaviourally (waltzing), stereocilia of all vestibular receptors in all alleles studied appear normal into adulthood. Based on current observations it appears that protocadherin 15 is required for hair cell development and function and that Pcdh15 may mediate its function through interacting proteins.

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520 Identification Of The Rodent USH2A And USH3 Genes And The Cellular Source Of The USH2A And USH3 Transcripts

*Janos Sumegi[1], Dali Huang[2], Catherine B. Talmadge[3], Randall R Fields[4], Eva Uzvolgyi[5], William J. Kimberling[6] [1]Hematology/Oncology, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio, [2]Pathology and Microbiology, University of Nebraska Medical Center, Omaha, Nebraska, [3]Pathology and Microbiology, University of Nebraska Medical Center, Omaha, NE, [4]Center for the Study and Treatment of Usher Syndrome, Boys Town National Research Hospital, Omaha, NE, [5]Biochemistry and Molecular Biology, University of Nebraska Medical Center, Omaha, NE, [6]Otolaryngology and Genetics, Boys Town National Research Hospital, Omaha, Nebraska

The autosomal recessive disorder, Usher syndrome is defined by the association of sensorineural deafness and visual impairment due to retinitis pigmentosa (RP). Variation between families corresponds to three clinical types distinguishable by difference in the severity of hearing loss and vestibular dysfunction; all three forms have RP. Each subtype is heterogeneous and twelve genetic loci have been identified. Five genes have been already cloned and characterized: USH1B, USH1C, USH1D, USH2A and USH3. We have recently identified and characterized the human USH2A and USH3 genes. Murine and rat orthologues of these genes were also identified. To identify the cellular origin of the USH2A and USH3 transcripts we visualized the distribution of the transcripts by in situ hybridization. The transcripts for USH2A are found in the rod and the cone cells of the outer nuclear layer. The USH3 transcripts were detected in the outer and inner nuclear layers. Laser capture microdissection (LCM) coupled with reverse transcription-polymerase chain reaction (RT-PCR) data confirmed the in situ results. Both USH2A and USH3 proteins are highly conserved in human, mouse and rat. Immunohistochemistry suggests that the USH2A protein is excreted into the interphotoreceptor cell matrix (IPM) of the retina. Antibodies against the USH2A protein specifically bound only to the apical surface of the retinal pigment epithelial cells. The USH3 protein was immunodetected in the cells of the outer nuclear layer of the retina.

521 Overexpression of Fibroblast Growth Factor (FGF2) in the Adult Mouse Protects Against Synaptic Degeneration in the Cochlear Nucleus Following Acoustic Overstimulation.

*Chrystal Stanislaus D'sa, Waheeda A. Hossain, Julie S Gross, D. Kent Morest Neuroscience, University of Connecticut Health Center, Farmington, CT

FGF2 has been implicated in the early development of the cochlear nucleus in the fetal mouse, but there is relatively little of it expressed there postnatally. Previous experiments in the adult (C57/BxCBA/J) mouse cochlear nucleus demonstrated a loss of synaptophysin-stained synaptic endings within 7-14 days after noise damage to the cochlea, while FGF2 expression increased in astrocytes. On this basis we hypothesize that FGF2 upregulation represents a compensatory reaction on the part of cochlear nucleus cells to the degenerative changes. If so, one might prevent synaptic degeneration by providing FGF2 to the cochlear nucleus challenged by overstimulation. To test this hypothesis, we noise-exposed transgenic mice, in which the full-length cDNA for FGF2 is overexpressed and higher levels of the FGF2 protein might protect synaptic endings against damage. The wild type from the same strain and the FGF2 overexpressor heterozygous mice, aged 3-4 months, were exposed to 115 dB SPL for 6 hours. After 7 and 14 days' survival, the brains were fixed and the cochlear nucleus sections were immunostained for SV2 (a synaptic vesicle protein, used as a measure of the number of synaptic endings). The cochleograms of both the wild type and FGF2 overexpressor showed complete loss of outer hair cells and loss of inner hair cells restricted to the region of noise exposure. In the wild type mouse there was a loss of synaptic endings in the parts of

ABSTRACTS OF THE TWENTY-SIXTH ANNUAL MIDWINTER RESEARCH MEETING OF THE

Association for Research in Otolaryngology

February 22-27, 2003

Daytona Beach, Florida, USA

Peter A. Santi, Ph.D.
Editor

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

CONFERENCE OBJECTIVES

After attending the Scientific Meeting participants should be better able to:

1. To understand current concepts of function of normal and diseased ears and other head and neck structures.
2. To understand current controversies in research methods and findings that bear on this understanding.
3. To understand what are considered to be the key research questions and promising areas of research in otolaryngology.

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Christove, Gomez and Anderson, Genotype-phenotype Correlations for the Horizontal VOR in Spinocerebellar Ataxia 1-8, Assoc. Res. Otolaryngol., Abs.: 1077.

President's Message

Welcome to the 26th Midwinter meeting of the Association for Research in Otolaryngology! For the first time the meeting is held in Daytona Beach. It is a testament to the success of the organization and its MidWinter Meeting that a move had to be made from small quarters on the west coast of Florida to larger quarters on the east coast. Even with more abstracts than ever before, the poster sessions will now all be warm and dry, independent of the weather.

A perusal of the abstracts shows that the meeting promises to be diverse and interesting. Before the meeting officially begins, there will be a short course entitled "Vestibular System 101: Introduction to Vestibular System Structure and Function for Non-Experts." The meeting will begin with the Presidential Symposium on the "Calyceal Synapses of the Brain Stem." This symposium will highlight synapses whose specializations are not only remarkable for the role they play in hearing but that have also made possible biophysical studies of neurotransmission in the mammalian central nervous system. Speakers include E. Neher, Nobel Laureate, from Germany, J.G.G. Borst from the Netherlands, P. X. Joris from Belgium as well as L.G. Wu, E. von Gersdorff, L. O. Trussell, and R. Shannon from the United States. Other symposia occur over the following days. On Monday these include "Development of the Inner Ear" organized by D. Wu, "Tinnitus: Mechanisms, Models and Therapy" organized by M. C. Liberman, and "Afferent Synaptic Transmission in the Cochlea" organized by T. Parsons. On Tuesday there will be two symposia, "Functional Organization of the Auditory Cortex in Humans and Primates" organized by D. Hall and A. Palmer, and "Auditory Learning: Principles, Applications and Mechanisms" organized by D. Moore and B. Wright. On Wednesday a symposium entitled "Stem Cells and Progenitors: Identification, Isolation and Use" will be presented under the guidance of N. Segil and A. Groves.

The winner of the Award of Merit is David Kemp, whose work on otoacoustic emissions has not only contributed to an understanding of the cochlea but has also led to the development of sensitive, objective hearing tests. On Sunday afternoon he will present the Presidential Lecture, "Otoacoustic Emissions – A 25 year Overview." His contributions will be celebrated on Tuesday evening at the Award of Merit Ceremony.

The Program Book provides details about other highlights. These include workshops and special events.

The ARO owes its vitality to the people it attracts and to the support it receives. It is fortunate to be served by a group of exceptionally talented, generous and committed people. Much of the work of organizing this meeting and running the ARO is done by members of the Council and by those who serve on committees. The MidWinter Meeting of the ARO is supported in part by a grant from the National Institute for Deafness and Other Communication Disorders. Support for students and residents comes from the National Institute for Deafness and Other Communication Disorders as well as from the American Academy for Otolaryngology-Head and Neck Surgery Foundation and from the Deafness Research Foundation. The ARO also gratefully acknowledges the contributions of Springer Verlag, the publisher of the Journal of the Association for Research in Otolaryngology (JARO) and host of a reception on Sunday evening. The ARO is ably managed by the Talley Management Group, Inc..

Donata Oertel, PhD

David T. Kemp, Ph.D.

2003 Award of Merit Recipient

David T. Kemp Ph.D.
2003 Award of Merit Recipient

David T. Kemp was trained initially in physics and then in geophysics at Kings College, University of London. His graduate studies on naturally occurring low frequency electromagnetic radiation led to a number of novel observations. These included developing a method for global thunderstorm mapping and for localizing individual lightning bolt strikes, along with the characterization of rare, massive electrical discharges. These latter resonant events have since been identified as being giant electrical discharges from clouds that extend up to the ionosphere. Soon after completing his doctoral studies, to follow up on his interests in audio-frequency signals, Dr Kemp joined the research group at London's Royal National TNE Hospital that was attempting to quantify auditory perception in children. He soon extended his research to include the study of low-level hearing in adults, and it was his observations of the fine 'microstructure' of auditory sensitivity that led him to postulate sound 'reflection' inside the cochlea. Dr Kemp further hypothesized the reality of a biologically 'active' signal-processing mechanism, and predicted the consequential existence of evoked otoacoustic emissions (OAEs) from the ear. As the result of a series of experiments conducted in 1977, he demonstrated that the human ear really did generate sound as a part of the normal hearing process. After describing the existence of stimulus-frequency OAEs that mimicked the psychoacoustical microstructure and supported his internal-reflection hypothesis, he went on to observe both spontaneous and distortion product OAEs. To illustrate this new class of physiological response to the hearing field, Dr Kemp devised the transient evoked OAE method and described it, in 1978, in the initial peer-reviewed report on emitted responses. This original publication led to a series of grant awards from the United Kingdom's Medical Research Council, which permitted him to establish the auditory biophysics laboratory at the University of London that focused on further exploration of the biophysics of cochlear function using OAEs. There is no question that Dr Kemp's discovery of OAEs led to new avenues of research into the fundamental mechanisms of hearing including the subsequent finding by others of the outer hair cell's electromotility. In addition, the discovery of OAEs gave new life to the concept of the 'cochlear amplifier' and stimulated further research on the biomechanical nonlinearities of the cochlea. Moreover, the breakthrough finding of OAEs also led to the development by Dr Kemp of a new objective hearing test based on the detection of OAEs. By introducing the first commercial OAE instrument, he set the 'gold standard' for developing such devices for clinical testing. Another major contribution that Dr Kemp has made to clinical service has been his commitment to universal newborn hearing screening. There is no doubt that his interest in, and support of, this important endeavor have contributed to its visibility around the world. Most recently, Dr Kemp along with his auditory research colleagues at University College London (UCL), has spearheaded the development of a new facility called the UCL Centre for Auditory Research. This center, due to open in early 2004, will be a major national and European focus for integrated cross-disciplinary hearing research aimed at understanding the causes of deafness. In all, Dr Kemp has received a number of awards and prizes for his considerable scientific work on OAEs from professional societies, learned institutions, charitable foundations, and industry. It is fitting that the Association for Research in Otolaryngology honor David T. Kemp as the recipient of the 2003 Award of Merit for his significant contributions to both the basic and clinical hearing sciences.

Brenda Lonsbury-Martin
Susan Norton

Association for Research in Otolaryngology

Executive Offices
19 Mantua Road, Mt. Royal, NJ 08061 USA
Phone: (856) 423-0041 Fax: (856) 423-3420 E-Mail: headquarters@aro.org
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2000	David M. Green, PhD
2001	William S. Rhode, PhD
2002	A. James Hudspeth, MD, PhD
2003	David T. Kemp, PhD

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*** Indicates presenting author**