preplacodal ectoderm. Blocking expression of all three Bmp antagonists severely impairs subsequent placodal development. These and other data challenge the conventional model that preplacodal ectoderm is specified by positional readout of a static Bmp gradient. Instead, Bmp acts locally and transiently to establish a broad zone of competence, after which Bmp antagonists plus other signals promote preplacodal development.

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Program/Abstract # 446
FGF signalling is involved in the induction and morphogenesis of the inner ear

Raj K. Ladher, Sabine Freter, XaioRei Sai
Lab for Sensory Development, RIKEN CDB, 2-2-3 Minatojima-Minamimachi, Chuo-ku, Kobe, Japan

The inner ear is induced within non-neural ectoderm adjacent to the hindbrain of all vertebrates as a thickened ectodermal disk, the otic placode. Once induced, the otic placode undergoes morphogenesis to become internalised into the head of the embryo. Signals emanating from mesoderm and neural ectoderm, in particular Fgf and Wnt family members, have been shown to instruct the adjacent non-neural ectoderm to adopt an otic fate. We demonstrate that the action of FGF and wnt is sequential, and that their roles support a model of hierarchical fate decisions that restrict the developmental potential of the ectoderm going through an intermediate state before full otic commitment. We show that signalling by FGF3 and FGF19 is required to initiate a region that can be regarded as a precursor to both otic and epibranchial fates. Subsequent to the induction of the precursor domain, we identify a role for FGF signalling in directing morphogenesis of the otic placode. We find that local FGF signalling triggers a phosphorylation cascade that activates basal myosin II through the activation of phopholipase Cγ. Myosin II exhibits a non-canonical activity and results in the local depletion of actin filaments. Significantly, the resulting apical actin enrichment drives morphogenesis of the inner ear. Thus, FGF signalling coordinates both induction and morphogenesis of the inner ear.

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Program/Abstract # 447
Hindbrain rhombomere 4 induces authentic inner ear vesicles in the chick

Yingcui Li [a], S.R. Hilfer [b]
[a] *Center for Regenerative Medicine and Skeletal Development, MC3705, Department of Reconstructive Sciences, University of Connecticut Health Center, School of Dental Medicine, Farmington, CT, USA*
[b] *Department of Biology, Temple University, Philadelphia, PA, USA*

Identification of the source of signals that produce unique organ primordia from a seemingly identical ectoderm remains a major question in embryology. Existing evidence supports the conclusion that the signals initiate unique cascades of gene expression which give each organ its special character. Identification of hindbrain segments through molecular markers gave rise to the possibility to test a specific role for each of hindbrain rhombomere (R) for initiation of inner ear, the otic vesicles. The ability of R4 to induce inner ear vesicles in foreign ectoderm was tested by transplanting R4 segments into the midbrain of 10 somite chicken embryos. Evidence showed it was R4 by itself, neither adjacent R3 nor R5 nor contamination of otic ectoderm in the transplant, that initiated otic primordial from ectoderm adjacent to the transplant. Molecular identity of the ectopic vesicles confirmed them as authentic inner ear vesicles. The temporal expression pattern of these marker genes was also tested from embryos after 6 to 18 h' post micro-surgery culture, but a compressed temporal pattern was found. Possible extended expression of a putative transcriptional signal in R4 was also tested but was not detected in the transplant as early as 6 h after the operation. Overall, combining micro-surgery with current molecular tools, we have showed that R4 as the key source of signals during induction of the inner ear.

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Program/Abstract # 448
Inner ear auditory progenitors are directly dependent on Hedgehog Signaling

Alexander S. Brown, Martin Riccomagno, Douglas J. Epstein
Department of Genetics, University Pennsylvania School of Medicine, USA

In mouse embryos lacking Sonic Hedgehog (Shh), several genes in the ventral portion of the otic vesicle have reduced expression, eliminating cochlear duct outgrowth and reducing the size of the cochlear-vestibular ganglion (cvg), the sensory nerve that relays sound and positional information to the brain. In addition, dorsal otic derivatives are malformed or absent in $Shh^{-/-}$ embryos. Since inner ear patterning and morphogenesis depend on signals derived from neighboring tissues that are compromised by the loss of Shh, the extent to which Shh signaling directly acts on inner ear development is unclear. To address whether Shh acts directly on the otic epithelium we generated embryos in which Smoothened (Smo), an essential transducer of Shh signaling, was conditionally inactivated in the otic vesicle (Smo^{ecko}). While ventral otic derivatives including the cochlear duct and saccule fail to form in Smo^{ecko} embryos, vestibular structures develop properly. These findings indicate that the otic epithelium is directly dependent on Shh signaling for the formation of the cochlea and saccule, but indirectly dependent on Shh for canal and utricle development. Interestingly, the composition of neuroblasts that make up the cvg is altered in Smo^{ecko} embryos. Normally, vestibular neuron progenitors delaminate from the otic epithelium, followed by cochlear neuron progenitors. Surprisingly, in Smo^{ecko} embryos, vestibular neurons form and project to their proper targets, whereas, the cochlear ganglia is severely reduced or absent. This raises the intriguing prospect that Shh signaling is selectively required for the specification of cochlear neuroblasts.

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Program/Abstract # 449
The role of Ldb complexes in lens development

Tsadok Cohen, Heiner Westphal
LMGD, NICHD, NIH, Betheda, MD, USA

The LIM-domain binding protein 1 (Ldb1) is a multifunctional transcriptional co-factor that mediates the function of LIM-homeodomain (LIM-HD), LIM only proteins (LMO), SSDPs, OTXs and GATAs transcription factors. As such, Ldb1 is essential for embryonic development exerting roles in cell-fate determination, tissue development and cytoskeletal organization. A targeted deletion of the Ldb1 gene in the mouse results in early embryonic lethality (E9.5–10). These mutants do not form a heart and exhibit truncated anterior head structures. Although Ldb1 expression is detected at E9.5–10