D–Pax2, the *Drosophila* homolog of the vertebrate Pax2 gene, is expressed in a specific set of sensory system cell types during their specification and differentiation. These cells include the shaft and sheath cells of the external sensory organs, the cone and primary pigment cells of the eye, and the sheath cells of the auditory organ. As a transcriptional activator, D–Pax2 has been shown to guide the proper differentiation of at least some of these cell types; however, little is known about the gene targets that D–Pax2 regulates. Using an in silico approach, we have identified several putative targets for D–Pax2 activity. In situ hybridizations have been performed using probes for some of these genes and we have been able to identify a subset of candidate targets that are expressed in the D–Pax2+ shaft cells of external sensory organs and, in some cases, other D–Pax2+ cells. In addition, this expression is absent from D–Pax2 mutants. The functions and significance of some of these target genes will be discussed.

doi:10.1016/j.ydbio.2007.03.251

**Program/Abstract # 191**

**Genetic interaction of Foxe3 and Pitx3 genes in lens mouse development**

Olga M. Medina ¹, Rina Shah ¹, Richard Berhinger ², Milan Jamrich ¹

¹ Department of Molecular Human Genetics, Baylor College of Medicine, USA
² Department of Molecular Biology, M.D. Anderson Cancer Center, Houston, TX, USA

The forkhead gene Foxe3, has been shown as a key player in the network involved in lens development. Foxe3 is expressed almost exclusively in the lens lineage. First is expressed in the presumptive lens ectoderm and later is restricted to the anterior epithelial cells where is maintained until adulthood. Mutations in Foxe3 have been identified as the cause of the spontaneous mouse mutation dysgenic lens (dyl) and a rare congenital disease, primary aphakia in humans. Foxe3-null mutant mice have been observed to have defective lens formation. Pitx3 is a paired-like class of homeobox transcription factor and deletions in the promoter of this gene cause the phenotype in the aphakia mouse mutant, which lacks of lenses and pupils. We are analyzing the molecular basis of the Pitx3 aphakia phenotype and comparing it to the Foxe3 mutants. Using the Foxe3-null mutation and the Pitx3 aphakia mice mutants, we are investigating the interaction of these two genes, using a genetic approach. We will address how these two genes cooperate during lens development or if they have a hierarchical relationship. Our study will provide a better picture of the function of each of these genes and how they interact in the gene network involved in lens development and disease.

This work is being supported by a training grant T32 EG07102 and by the NEI grant # EY012505.

doi:10.1016/j.ydbio.2007.03.252

**Program/Abstract # 192**

**Specification of zebrafish INs**

Katharine E. Lewis, M Batista, F Weierud, S Lutter

PDN, University of Cambridge, UK

Interneurons (INs) constitute most of the neurons in the vertebrate CNS and they function in almost all neural circuits and behaviors but we know a lot less about their development than we do about motoneurons (MNs). This is partly because there are more INs than MNs and whereas MN axons leave the CNS and innervate distinct muscles, INs are contained within the CNS where their axons are harder to distinguish from one another. Zebrafish embryos are a powerful system for studying IN development as compared to amniotes, they have a relatively small number of different INs, all of which can be identified by their unique morphology and we can observe these neurons in live embryos. We have characterized the expression patterns of several transcription factors that are expressed by post-mitotic cells in specific dorsal–ventral regions of zebrafish spinal cord. The expression patterns of most of these genes are conserved in amniotes, suggesting that, as for MNs, mechanisms of IN specification are conserved across vertebrates. However, *lbx* genes are expressed differently in amniote and zebrafish spinal cords. Zebrafish have 3 *lbx* genes, all of which have distinct spinal cord expression patterns; whereas amniotes have 2 *Lbx* genes and only one is expressed in spinal cord. Therefore, we are investigating how Lbx gene number and spinal cord expression has evolved in vertebrates and how spinal cord *lbx* expression is regulated. We are also analysing the specification of a class of morphologically identified INs, CiAs. We are analysing how different signalling pathways interact to determine the correct position and number of CiAs and we are determining the functions of particular transcription factors in specifying CiA functional characteristics.

doi:10.1016/j.ydbio.2007.03.253

**Program/Abstract # 193**

**Analysis of mice kreisler mutants reveals new roles of neural tube signals in the axial patterning of the otic primordium**

Citlali Vazquez-Echeverria, Cristina Pujades

BDCEXS, UPF, BCN, Spain

The kreisler gene encodes a basic domain leucine zipper transcription factor that is expressed in two segments of the developing vertebrate hindbrain: the rhombomeres 5–6. Mutant mice with loss of *kr* expression lack identifiable r5 and r6. The r4–r7 region remains unsegmented, r5 was not formed and r6 was mis-specified. Gain-of-function experiments in chick have proposed a dual function for *kr* in both segmentation and specification of A-P identity in the hindbrain. Mice homozygous for the *kr* mutation are deficient in hearing and exhibit inner ear hypoplasia. Several lines of