type and Lfng-null cells, the pattern of cyclic genes was more severely affected compared with the DI1 chimera. In addition, I found that Lfng might work in non-cell autonomous manner, since Notch activity was found to be positive or negative in Lfng KO cells in the chimera embryo, although all cells show Notch activity in a simple Lfng KO embryo. These results suggest that Lfng is not only a key regulator of the clock but also plays an important role in the coupling mechanism.

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Program/Abstract # 102
Roles of Tbx2b during asymmetric brain development
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The molecular processes involved in establishing left-right (L-R) asymmetry of the vertebrate nervous system are not well understood. The zebrafish epithalamus is comprised of the bilateral habenular nuclei, left-sided parapineal, and medially located pineal. The parapineal is apparent 28-31 hours post fertilization (hpf) as a group of cells migrating leftward from the anterior medial region of the pineal anlage, to lie adjacent to the left habenula. The parapineal is required for many asymmetries in anatomy and gene expression between the habenular nuclei. We have shown that T-box containing transcription factor 2b (Tbx2b) is required for parapineal formation. In tbx2b KO mutants, parapineal cells are not correctly specified and fail to migrate. To investigate the roles of Tbx2b in parapineal migration, we are conducting high-resolution lineage labeling, and have found a left-of-midline bias for their origin. We are currently examining the fate of parapineal precursor cells in tbx2b KO mutants. We are also testing candidate regulators and targets of Tbx2b in the retina, the transcription factor Nr2e3 is an antagonist of Tbx2b during photoreceptor specification. Our results suggest a similar antagonism between Nr2e3 and Tbx2b in parapineal development. Total cadherin activity is reported to be downregulated in Tbx2b morphants; our data suggests that N-cadherin is specifically required for parapineal cohesion. Future studies will involve tissue-specific tbx2b over-expression studies as well as high throughput transcriptome analysis to identify tbx2b transcriptional targets.

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Program/Abstract # 103
Cell shortening, basal constriction and epithelial relaxation, in the developing vertebrate brain, are regulated by non-muscle myosins
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type and Lfng-null cells, the pattern of cyclic genes was more severely affected compared with the DI1 chimera. In addition, I found that Lfng might work in non-cell autonomous manner, since Notch activity was found to be positive or negative in Lfng KO cells in the chimera embryo, although all cells show Notch activity in a simple Lfng KO embryo. These results suggest that Lfng is not only a key regulator of the clock but also plays an important role in the coupling mechanism.

Vertebrate brain morphogenesis includes a complex set of processes that result in correct folding of the neuroepithelium, so that it packs correctly into the skull, and correctly shapes the brain ventricles, a system of connected cavities containing cerebrospinal fluid. Multiple cell shape changes underlie these processes, including cell shortening and basal constriction, a previously undescribed morphogenetic mechanism. Myosins and their regulatory proteins comprise a huge and diverse gene family, which control these shape changes. We are analyzing two regions of the developing zebrafish brain in which we have shown that myosin regulation is pivotal. The first region is the midbrain-hindbrain boundary constriction (MHBC), which forms through cell shortening and basal constriction. At the MHBC both non-muscle myosin IIA and IIB have necessary and highly synergistic function. MHBC cells with decreased myosin II function neither shorten nor basally constrict. This is the first demonstration that myosin II function is required for cell shortening during brain development. The second region is the hindbrain, where, in contrast to the MHBC, inhibition of myosin II function is required for normal morphogenesis. In particular, inhibition of myosin activity by myosin phosphatase is required for normal cell shape and relaxation of the hindbrain epithelium to allow for ventricle expansion. These data indicate that regulation of myosin II, by creating a balance between myosin activation and inactivation, is critical for multiple processes during brain morphogenesis.

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Program/Abstract # 104
Vgl-2a is Required for Neural Crest Cell Survival During Zebrafish Craniofacial Development
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The development of the vertebrate cranial skeleton results from the specification, growth, patterning, and morphogenesis of tissues derived from all three germ layers in response to a complex network of reciprocal signaling. While many genes involved in these processes have been identified, others remain uncharacterized. We have identified a gene, vgl-2a, which is expressed in the pharyngeal endoderm and ectoderm surrounding the neural crest derived mesenchyme of the pharyngeal arches in zebrafish. We have found that reducing expression of vgl-2a in zebrafish embryos using Morpholino antisense oligonucleotides results in increased neural crest cells death, a defect in endodermal pouch morphogenesis, and subsequent reduction of cranial cartilages. We have also demonstrated that expression of vgl-2a within the arches is regulated by FGFs and retinoic acid, suggesting that vgl-2a may represent an intersection of these signaling pathways in pharyngeal arch development.

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Program/Abstract # 105
Requirements for fat4 and atr2a in shaping the zebrafish craniofacial skeleton
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Little is known about the mechanisms of cell-cell communication necessary to assemble skeletal elements of appropriate size and shape. Skeletal progenitors may behave as coherent units by communicating via the planar cell polarity (PCP) pathway. In Drosophila, two sets of factors control PCP independently: the Fat and the Stan systems. While a requirement for components of the Stan system was recently demonstrated in regulating the oriented divisions of chondrocytes and cellular intercalation in long bones, a role for the Fat system in skeletal development has not been reported. We find that mutants in two zebrafish orthologs, fat4 and atr2a, have defects in the neural crest-derived craniofacial skeleton: mutant skeletal elements have irregular edges and chondrocytes fail to flatten or stack normally. Co-expression of fat4 and atr2a in the pharyngeal