using this system to study the process. We are analyzing the zebrafish mutant, *schnitter*, that fails to initiate ventricle formation. Interestingly, these mutants also show defects in the dorsal diencephalon. Specifically, the pineal organ is disrupted and preliminary evidence suggests that the habenular nuclei do not form. Mutants also disrupt left–right patterning of the diencephalon. The pineal organ and the habenular nuclei normally display characteristic asymmetries. These asymmetries are preceded by left-sided gene expression of Nodal signaling pathway members in the developing diencephalon. We assayed Nodal gene expression in schnitter mutants and observed abnormal bilateral expression. We are cloning the gene disrupted in *schnitter* mutants and further characterizing their mutant phenotype. Together, these data will elucidate our understanding of the connections among ventricle formation, left–right asymmetry and early brain patterning.

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Program/Abstract # 383
Repression of genes by Snail2 during avian neural crest emigration

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Neural crest cells are a transient population of migratory cells that arise during neurulation at the border of the neural and non-neural ectoderm in the developing vertebrate embryo. Initially existing as adherent, epithelial cells in the dorsal neural tube (premigratory neural crest), these cells undergo an epithelial-to-mesenchymal transition (EMT), generating motile neural crest cells that subsequently differentiate into various structures, including the craniofacial skeleton and peripheral nervous system. The Snail family of transcriptional repressors is known to play key roles during the EMTs underlying both normal embryonic development and disease. In the chick, Snail2 functions to repress gene expression in premigratory neural crest cells to facilitate EMT and neural crest cell emigration. We have previously shown that the cell adhesion molecule *cadherin6B* is a direct target of Snail2 repression during neural crest EMT. Using a combination of bioinformatics as well as biochemical and embryological assays, we aim to identify Snail2 target genes during avian neural crest cell emigration whose repression is critical for proper regulation of neural crest EMT, and avian embryonic development as a whole. Future experiments will include the functional characterization of these genes during neural crest EMT, with the goal of understanding the molecular mechanism by which Snail2-mediated repression of these genes fosters neural crest cell emigration in the avian embryo.

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Program/Abstract # 384
Early neurotransmitter phenotype specification in *Xenopus laevis*

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The means by which a neuron progressively acquires and maintains a neurotransmitter phenotype, particularly the earliest stages of this process, is not well understood. There is evidence supporting the presence of a "default" phenotype or phenotypes that are subsequently restricted; alternatively, neurons may gradually acquire a specific neurotransmitter fate through a series of cell–cell interactions. In order to test the validity of these different models, we conducted primary cell culture and tissue culture experiments using early *X. laevis* embryos. *X. laevis* animal caps and whole embryos that have been dissociated and reaggregated both display the neural marker neural β-tubulin, but only the whole embryo reaggregates express GABAergic (*xGAT1*) and glutamatergic (*xVGluT1*) markers. Dissociated animal cap primary cell cultures express the early neural markers *Sox2* and *Sox3* but not neural β-tubulin, *xGAT1*, or *xVGluT1*. Treatment of animal cap cultures with FGF-8b results in expression of neural β-tubulin and GABAergic and glutamatergic markers, indicating these phenotypes are induced and neither is a default. Dissociated presumptive neural plate cultures are also being examined using *in situ* hybridization. Early neural plate cultures express several neurotransmitter markers, including *xVGluT1*, *xGAT1*, *xVIAAT*, and *xGlyT2*. These cells also coexpress markers for different neurotransmitter types, including *xGAT1* with *xGlyT2* and *xVGluT1*, and *xVGluT1* with *xGlyT2* and *xVIAAT*, suggesting that the identities of these cells may not be fully determined and lending support to aspects of several of the models.

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Program/Abstract # 385
Primary heart forming region of early avian embryo revealed by real-time positional fate map

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In HH 5–6 avian embryos, the traditional primary heart forming regions (HFRs) derived from several positional-fate-mapping studies do not agree with the expression pattern of putative cardiogenic molecular markers Bmp2 and Nkx2.5. To address the discrepancy, we followed heart progenitor cells in avian embryos between HH stage 3+, when they first ingress through the primitive streak, and HH stage 10, when the heart takes a tubular shape. Instead of two separate HFRs at HH stages 5–6, our results suggest a continuous HFR, which overlaps with the Nkx2.5 and Bmp2 expression pattern to a great degree with some discrepancies. In the mean time, our data revealed how the heart is assembled in real time.

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Program/Abstract # 386
A unifying concept of heart tube formation for avians and mammals

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In human development, it is postulated based on histological sections, that the cardiogenic mesoderm rotates 180° with the pericardial cavity. This is also thought to be the case in mouse development where gene expression data suggests that the progenitors of the right ventricle and outflow tract invert their position with respect to the progenitors of the atria and left ventricle. However, the