vascular connections and is conserved in all vertebrates. Very little is currently known about the cellular or genetic mechanisms which direct these asymmetries during cardiac morphogenesis. Molecular modifications of the extracellular matrix, cellular proliferation, cell adhesion, cell differentiation, cell migration, and cell-matrix interaction are all thought to be involved in the development of cardiac asymmetries but the relative contributions of these cellular mechanisms remain unknown. The aim of my project is to identify downstream genetic targets of Nodal signaling within the heart as well as to investigate Nodal independent factors which may contribute to the morphogenesis of the organ.

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**Program/Abstract # 110**
**Endocardial-myocardial interactions direct cardiac morphogenesis**
Olivier F. Noel, Nathalia Glickman Holtzman
*Department of Biology, Queens College; City University of New York, Flushing, NY, USA*

Cardiac morphogenesis has been shown to require significant cooperation between the endocardium and the myocardium. Previous work has shown that proper endocardial patterning is required for proper myocardium formation. We are taking advantage of small molecule drug screening in zebrafish to further explore the interactions between these two populations by identifying endothelial affecters. Because of its multiple destructive effects, we examined the consequence of FK-506 (Tacrolimus) on cardiac morphogenesis. Our data shows that FK-506 significantly disrupts angiogenesis resulting in defects in myocardial migration and consequently a misshapen heart tube forms. Specifically, endothelial cells were present in treated embryos but were reduced in number and irregularly positioned. These endothelial defects result in failure of blood circulation and severe edema. Preliminary observations indicate linear heart tube defects consistent with early loss of endocardial-myocardial interactions. This study supports the finding that a proper myocardium is dependent on a proper endocardium. We plan to examine the cellular behaviors underlying these morphogenesis defects while pursuing other drugs of interest. We have identified another candidate drug that also results in similar endocardial and myocardial defects. We hope that, taken together, these data will provide insight into the molecular mechanisms underlying myocardial morphogenesis. (Supported by NIH Grant: R15 HL096067 to NGH, AHA Founders Affiliate Undergraduate Research Fellowship 09-15 to ON, NIH MARC U-Star Program).

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**Program/Abstract # 111**
**Notch-restricted Atoh1 expression regulates morphogenesis of the posterior lateral line in zebrafish**
Miho Matsuda, Ajay Chitnis
*LMG, NICHD, NIH, Bethesda, MD, USA*

The posterior lateral line primordium (pLLp) migrates caudally depositing neuromasts to establish the posterior lateral line organ in zebrafish. A Wnt-dependent FGF signaling center at the leading end of the pLLp initiates formation of "proneuromasts" by facilitating the reorganization of cells into epithelial rosettes and by initiating *atoh1a* expression. Expression of *atoh1a* gives proneuromast cells the potential to become sensory hair cells and lateral inhibition mediated by Delta-Notch signaling restricts its expression to a central cell in maturing proneuromasts. We show that as *atoh1* expression becomes established in the central cell, it drives expression of *fgf10* and the Notch ligand, *deltaD*, while it inhibits expression of *fgfr1*. As a source of FGF10, the central cell activates the FGF pathway in neighboring cells, ensuring that they form stable epithelial rosettes. At the same time DeltaD activates Notch in neighboring cells, inhibiting *atoh1a* expression and ensuring that they are specified as supporting cells. When Notch signaling fails, unregulated *atoh1a* expression reduces FGFR1 expression, eventually resulting in attenuated FGF signaling, which prevents effective maturation of epithelial rosettes in the pLLp. In addition, as sensory hair cell precursors expand, *atoh1a* inhibits *e-cadherin* and this contributes to loss of cohesion and fragmentation of the pLLp. Together our observations reveal that restricted *atoh1a* expression is essential for effective morphogenesis of the pLLp.

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**Program/Abstract # 112**
**Studying the potential dual role of adhesion G protein-coupled receptors in early zebrafish embryogenesis**
Xin Li[a], Heidi Hamm[b], Lilianna Solnica-Krezel[c]
[a]*Neuroscience Graduate Program, Vanderbilt University, Nashville, TN, USA*
[b]*Department of Pharmacology, Vanderbilt University, Nashville, TN, USA*
[c]*Department of Biological Sciences, Vanderbilt University, Nashville, TN, USA*

Adhesion G protein-coupled receptors (adhesion GPCRs) are novel seven-transmembrane proteins with a large extracellular region containing protein modules involved in the processes of cell-cell adhesion and cell-matrix adhesion. Owing to their unique structure and involvement in several developmental diseases, adhesion-GPCRs are proposed to have vital dual roles in cellular adhesion and signalling. There are 33 adhesion GPCR genes in humans and 32 found so far in zebrafish. Even though the functions of most adhesion GPCRs during embryogenesis remain uncharacterized, literature and our preliminary data suggest that more than half of these genes are expressed during early zebrafish embryogenesis. We aim to delineate their expression patterns and to uncover their functions during early zebrafish embryogenesis. Focusing on the Group IV adhesion GPCRs, we have identified four members in the zebrafish genome. RT-PCR revealed dynamic temporal expression profiles of these four genes in the first five days of development. And they also exhibit unique spatial expression patterns in the first three days of development. Gain-of-function and loss-of-function experiments revealed morphogenetic defects, which are specific to the tissues where the particular adhesion GPCR is expressed. Based on our present data, we propose that Group IV adhesion GPCRs play important roles during early zebrafish embryogenesis. In the future, we will devote our efforts to determine whether they function by mediating cellular adhesion and/or signal transduction.

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**Program/Abstract # 113**
**Proper initiation of zebrafish epiboly requires the T-box transcription factor Eomesodermin A**
Ashley Bruce, Susan Du
*Dept. of Cell & Systems Biology, Univ. of Toronto, Canada*

Epiboly, or the thinning and spreading of a multilayered cell sheet, is the earliest morphogenetic event during zebrafish development. Its first phase involves doming of the yolk cell up into the overlying blastoderm. We previously showed that over-expression of dominant-negative *eomesodermin a* inhibits doming. Here we report our analysis of embryos lacking both maternal and zygotic Eomesodermin A (M*Zeomesa*). We find that epiboly initiation is delayed in M*Zeomesa* mutant embryos and, when doming occurs, it is uneven and irregular. We are currently investigating the mechanisms underlying these epiboly defects. The yolk cell microtubules,