arches with two other key-components of the PCP pathway – fj1 and das1 – suggests that morphogenesis of the craniofacial skeleton is controlled through a conserved PCP mechanism. Time-lapse analysis of cranial NC cell behavior in transgenic embryos to reveal division orientation and polarity during cartilage condensation, as well as cell transplantation to determine cell autonomous and non-autonomous requirements for fat4 and atr2a, will be presented.

doi:10.1016/j.ydbio.2010.05.144

Program/Abstract # 106
Morphogenesis of post-embryonic neural crest-derived pigment cell precursors revealed by time lapse imaging during zebrafish adult pigment pattern formation
Erine H. Budi, Larissa B. Patterson, David M. Parichy
\textsuperscript{a}Department of Molecular and Cellular Biology
\textsuperscript{b}Department of Biology, University of Washington, WA, USA

The neural crest gives rise to numerous cell types and organ system in the vertebrate embryo and adult, including bone and cartilage, peripheral neurons and glia, and pigment cells. The early morphogenesis of neural crest-derived cells in the embryo has been well documented. Yet, little is known about the morphogenesis of these cells during post-embryonic development, or precisely how they contribute to the development of adult form. To address these outstanding questions, we are studying the pigment pattern of adult zebrafish and how post-embryonic pigment cell precursors are established, maintained, and recruited to particular fates and locations. We previously showed that distinct populations of pigment cells generate the embryonic/early larval pigment pattern and the adult pigment pattern. Here, we use fate mapping and time lapse imaging in wild type and mutant backgrounds to identify the locations and migratory pathways of latent precursors to adult pigment cells, and we test the molecular and proliferative phenotypes of these cells. Our analyses reveal novel post-embryonic pools as well as migratory and proliferative behaviors during adult pigment pattern formation. These studies are a first step towards a fuller understanding of roles of neural crest-derived latent precursor populations in the development and evolution of adult form.

doi:10.1016/j.ydbio.2010.05.145

Program/Abstract # 107
\textit{zic1} and \textit{zic4} expression in the somite regulates dorsalization of the fish trunk structures
Toru Kawanishi, Yuuta Moriyama, Ryohei Nakamura, Atsuko Shimada, Hiroyuki Takeda
Dept. of Biol. Sci., Grad. Sch. of Sci., Univ. of Tokyo, Japan

The dorsoventral pattern of early animal embryos is determined by the gradient of BMP activity, and then interpreted by late developing organs such as the neural tube and somite. However, it remains unknown how the dorsal/ventral-specific characters such as fins and pigmentation emerge in late embryogenesis. \textit{Double anal fin} (Da) is a spontaneous medaka mutant which exhibits a unique ventralized phenotype in the dorsal trunk-tail region, with ventralized shape of the dorsal fin and ventral-type pigmentation on the dorsal side. The previous study genetically demonstrated that candidate genes of the mutant are \textit{zic1} and \textit{zic4}, but no mutation was found in the coding regions (Ohtsuka et al., 2004). Here we found a transposon-like DNA fragment inserted 9 kb downstream of \textit{zic4}, suggesting that \textit{Da} is an enhancer mutant of \textit{zic1}/\textit{zic4}. In \textit{Da} mutants, the expression of \textit{zic1} and \textit{zic4} is specifically reduced in the dorsal part of the somite. Somite transplantation experiments showed that when transplanted into \textit{Da} larva, wild-type somite locally rescued the \textit{Da} phenotype such as pigmentation pattern and fin morphology, demonstrating that affected somites are indeed responsible for the phenotype. Furthermore, the transplanted somites, randomly oriented upon transplantation, later re-acquired the dorsal expression of \textit{zic1}/\textit{zic4}, indicating that the expression of \textit{zic1}/\textit{zic4} is regulated by the surrounding tissue(s). Taken together, we conclude that the dorsoventral information is inherited in the somite which then regulates dorsalization of the fish trunk including fin shape and pigmentation.

doi:10.1016/j.ydbio.2010.05.146

Program/Abstract # 108
Myotome patterning and growth in zebrafish - The role of t\textit{bx}24
Stefanie E. Windner, Stephen H. Devoto
\textsuperscript{a}Dept. of Biol., Wesleyan Univ., Middletown, CT, USA
\textsuperscript{b}Dept. of Organismic Biol., Univ. of Salzburg, Austria

Vertebrate skeletal musculature is organized as a series of segmentally repeated compartments called myotomes. In most teleosts each myotome is subdivided into a medial fast fiber domain, the major proportion of the muscle mass, and a smaller slow fiber domain superficial to the fast fibers. The transcription factor Tbx24/fused somites (fss) is required for segmentation of the zebrafish musculature into myotomes. Tbx24 is also required for proper formation of the dermomyotome, which is the source of precursor cells for embryonic and larval muscle growth. We investigated the formation of slow and fast fiber domains and muscle growth in \textit{tbx24} mutant zebrafish by examining the distribution and proportion of both fiber types in \textit{fss} embryos and larvae and comparing their growth rates to heterozygous siblings. We find that Tbx24 is required for the establishment of a dorsoventral slow fiber domain superficial to the fast fibers. While wildtype embryos have slow fibers exclusively lateral to the fast fibers, slow fibers are found media to fast fibers along the entire body axis in \textit{tbx24} mutant embryos. The defect is most prominent in the central region of the trunk, which is devoid of superficial slow fibers. This defect spatially coincides with a lack of DM cells observed in \textit{fss} mutants and is retained into larval life. In addition, \textit{fss} larvae frequently show a deficit in muscle mass and are transiently smaller than their heterozygous siblings. We speculate that this results from the significantly reduced number of DM cells in \textit{fss} mutants.

doi:10.1016/j.ydbio.2010.05.147

Program/Abstract # 109
Identifying mechanisms which control asymmetric development of the zebrafish heart
Jessica R. Rowland, Kari Baker, Rebecca D. Burdine
Department of Molecular Biology, Princeton University, Princeton, NJ, USA

During zebrafish development, asymmetries in the heart are established through two distinct morphological events. The first event, cardiac jogging, consists of the conversion of the cardiac cone into the linear heart tube and is directly determined by the expression the nodal gene southpaw (\textit{spaw}) in the lateral plate mesoderm. Recent work from our lab has shown that the laterality of cardiac jogging is determined by a directional migration of the left atrial that this process is affected in mutants with absent or altered Nodal signaling. The second event, cardiac looping, establishes the relative positions of cardiac chambers and their
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.

doi:10.1016/j.ydbio.2010.05.148

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 effectors. 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 NCH, AHA Founders Affiliate Undergraduate Research Fellowship 09-15 to ON, NIH MARC U-Star Program).

doi:10.1016/j.ydbio.2010.05.149

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.

doi:10.1016/j.ydbio.2010.05.150

Program/Abstract # 112
Studying the potential dual role of adhesion G protein-coupled receptors in early zebrafish embryogenesis
Xin Li, Heidi Hamm, Liliana Solnica-Krezel
1Neuroscience Graduate Program, Vanderbilt University, Nashville, TN, USA
2Department of Pharmacology, Vanderbilt University, Nashville, TN, USA
3Department 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.

doi:10.1016/j.ydbio.2010.05.151

Program/Abstract # 113
Proper initiation of zebrafish epiboly requires the T-box transcription factor Eomesoderm 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 Eomesoderm A (MZeomesa). We find that epiboly initiation is delayed in MZeomesa 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,