were induced by *S. macrurus* MyoD and myogenin — an inter-MRF regulatory pattern that is similar to that observed among mammalian homologs. These data, together with the confirmed expression of *S. macrurus* MRF proteins in skeletal muscle and EO in vivo, suggest both functional conservation of *S. macrurus* MRFs as transcriptional activators of muscle-specific genes and the presence of a distinct regulatory mechanism affecting MRF activity in EO.

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Program/Abstract # 240
Lineage mapping and genetic cell ablation of post-migratory cardiac neural crest cells
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The critical requirement of the cardiac neural crest (CNC) cells during cardiovascular development is well documented, as are the severe and diverse congenital cardiovascular consequences associated with their removal and/or genetic manipulation. However, the actual role of the CNC in the outflow tract (OFT) itself and what role they play during mesenchymal cushion remodeling and generation of the mature heart remain unclear. Our study of the transcriptional regulation of *Periostin* identified a Periostin 3.9 kb transcriptional regulatory module that drives in vivo restricted transient expression in subpopulation of CNC-derived cells that colonize the distal OFT mesenchymal cushions. Given there are currently no OFT endocardially-restricted promoters (or even a cushion-specific promoter), we generated 3.9 kb Periostin-Cre transgenic mice. In combination with R26R reporter and R26-diptheria toxin-A genetic ablation mice, we have begun to analyze the role of the 3.9 kb Periostin-expressing OFT subpopulation. Lineage mapping and genetic cell ablation data will be presented. Combining these results indicates that we have identified a unique and powerful molecular tool with which to begin to understand/identify some of the master regulators that control how the OFT mesenchymal cushions are remodeled to give rise to mature heart and what are the molecular differences/origins between the OFT and atrioventricular cushions.

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Program/Abstract # 241
Notch2 controlled molecular mechanisms underlying secondary heart field differentiation and proliferation
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Our previous studies have shown that Notch2 is required for the proliferation of smooth muscle cells derived from cardiac neural crest cells and for myocardial development. In order to further study the effect of Notch2 in cardiovascular tissues, we made a conditionally activated Notch2 transgene (N2-GOF) that allows the expression of the Notch2 intracellular domain (ICD) in specific cell lineages. N2-GOF mice were crossed to mice containing a Mef2c-AHF-Cre transgene, resulting in the expression of the Notch2 ICD specifically in the secondary heart field which includes endothelial and myocardial cells of outflow tract, right ventricle and ventricular septum. At E16.5 Mef2c-cre; N2-GOF hearts exhibit profoundly reduced right ventricles and ventricular septal tissue but conversely, have enlarged valves. A very large decrease in the number of Mef2c-cre; N2-GOF myocardial cells that express α-Actinin was observed, while the number of α-Smooth Muscle Actin (α-SMA) positive cells remains constant. TUNEL assays on E16.5 Mef2c-cre; N2-GOF hearts show a large amount apoptosis in the right ventricle. These results together suggest that over expression of Notch2 prevents maturation and differentiation of the cardiomyocytes and causes apoptosis. In contrast to the myocardium, valvular cells show an increase in proliferating α-SMA positive cells in Mef2c-cre; N2-GOF mice as compared to wild type. Therefore the Notch2 ICD is exerting differential effects on cell populations derived from the secondary heart field. Our data will help elucidate the molecular mechanisms involved in heart tissue development and repair.

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Program/Abstract # 242
Constitutive activation of β-catenin signaling in embryonic surface epithelium results in global acquisition of hair follicle fate
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β-Catenin signaling is required for hair follicle development, but it is unknown whether its activation is sufficient to globally program embryonic epidermis to hair follicle fate. To address this we mutated endogenous epithelial β-catenin to a dominant active form in vivo. Hair follicle placodes were expanded and induced prematurely in activated β-catenin mutant embryos, but failed to invaginate or form multilayered structures. Eventually, the entire epidermis adopted hair follicle fate, broadly expressing hair shaft keratins in place of epidermal stratification markers. Mutant embryonic skin was precociously innervated and pigmented. Transcript profiling experiments identified elevated expression of Sp5, a direct β-catenin target and transcriptional repressor. We show that Sp5 normally localizes to hair follicle placodes and can suppress epidermal differentiation gene expression. We identified the pigmentation regulators Foxn1, Adamsts20 and Kitl, and the neural guidance genes *Sema4c*, *Sema3c*, *Unc5b* and *Unc5c*, as potential mediators of the effects of β-catenin signaling on pigmentation and innervation. Our data provide evidence for a new paradigm in which, in addition to promoting hair follicle placode and hair shaft fate, β-catenin signaling actively suppresses epidermal differentiation and directs pigmentation and innervation. Controlled downregulation of β-catenin signaling is required for normal placode patterning, and adoption of the full range of follicular fates.

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Program/Abstract # 243
Distinct sequential cell behaviours direct primitive endoderm formation in the mouse blastocyst
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The extraembryonic lineages trophectoderm (TE) and primitive endoderm (PrE) are the first two lineages to differentiate from a