interaction between *Hoxc13* and *Soat1* may establish a paradigm for demonstrating the use of defined hair follicle abnormalities as a diagnostic tool for determining the risk of developing complex diseases affecting multiple organ systems.

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Program/Abstract # 173
Uncovering developmental gene regulatory networks in the *Drosophila* CNS midline

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Understanding how neuronal and glial diversity arises is an important goal of developmental neurobiology. The *Drosophila* CNS midline cells constitute a simple model system to uncover gene regulatory networks that regulate neurogenesis, cell fate acquisition, and neuronal function. In the *Drosophila* CNS, the LIM homeodomain transcription factor *tailup* (also known as *islet*) is required for proper axon pathfinding of a subset of motor neurons and for dopamine and serotonin synthesis. We report the role of *tailup* in the development of a single midline neuron, the H cell. The H cell is a dopamine-producing neuron that receives serotonergic, glutamatergic and peptidergic inputs. We show that *tailup* is expressed in the H cell. Consistent with previous reports, *tailup* regulates the expression of *ple*, which is a dopamine biosynthetic enzyme. We also have identified additional *tailup* target genes that are involved in dopamine synthesis and transport. Additionally, *tailup* represses *neuropeptide F receptor* expression in abdominal segments. Experiments are in progress to determine the role of *tailup* in H cell axonogenesis and how *tailup* expression is regulated. Together with the genetic analysis of *tailup* these experiments will help identify developmental gene regulatory networks that will likely be evolutionarily conserved.

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Program/Abstract # 174
Direct regulatory control of chicken Sall4 (Spalt4) in the otic placode

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The zinc finger gene *Sall4* is important in the formation of the inner cell mass, regulation of stem cell differentiation, and limb development in the mouse. In the chicken we have shown that *Sall4* is expressed in the presomitic mesoderm, pronephros, lateral plate mesoderm and later in the limbs, as well as portions of the CNS, the cranial neural crest and the otic placode. We isolated regions adjacent to the chicken *Sall4* gene that were conserved among chickens, humans, mice, and frogs. These regions were cloned into a vector in front of a minimal promoter driving GFP expression. A number of these potential cis-regulatory elements drove the expression of GFP in a tissue specific manner that recapitulated much of pattern of *Sall4* RNA expression at the stages studied, including the intermediate mesoderm and pronephros, the presomitic mesoderm and somites, and spatially and temporally restricted regions of the CNS. We also found a region that drove the expression of GFP in the otic placode and later in the otic vesicle, as well as the lateral plate mesoderm. When we looked at this element more carefully we found a number of different putative transcription binding sites, including those for *Pax2*, *Tbx5*, *TCF* and *Pea3*, that might be responsible for the enhancer activity found in this region. When the *Pax2* region was replaced by heterologous DNA, the expression in the otic placode was significantly diminished. Removing one of the *Tbx5* sites eliminated the expression in the lateral plate. This cis-regulatory region may also be responsible for *Sall4* induction by FGF signaling seen previously. This work is supported by USPHS grantDE16459.

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Program/Abstract # 175
microRNA-24a is required to repress apoptosis in the developing neural retina

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Program/Abstract # 176
The role of PAX6 in the development of the retinal pigment epithelium

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Genetic tests indicate that the paired/homeodomain protein PAX6 promotes the development of the highly proliferating retina while the bHLH-Zip protein MITF promotes the development of the less proliferating retinal pigment epithelium (RPE). In mice, however, PAX6 is also expressed in the future RPE, prompting us to explore its role genetically. While mild *Mitf* mutations show increases in PAX6 and its pro-retinogenic targets in the RPE, there are little if any changes in cell proliferation. In combination with heterozygosity for *Pax6*, however, cell proliferation is increased even though some retinal genes are decreased. Conversely, an increase in *Pax6* gene dose (as provided by a YAC transgene) corrects cell proliferation in the RPE, but, surprisingly, reduces retinal gene expression to wild type levels. We explain these observations in the following way: The increase in cell proliferation following decrease of *Pax6* is associated with a decreased expression of alpha B crystallin which is known to regulate Cyclin D1 protein levels through the regulation of the SCF^{FBX4} ubiquitin ligase complex. The decrease in retinal gene expression following an increase in *Pax6* correlates with an increase in the expression of an *Mitf* relative, *Tfec*. Hence, in mild Mitf mutations, despite increased retinal gene expression, the RPE does not develop as a retina because *Pax6* inhibits cell proliferation, and in *Pax6* over-expressors, the RPE does not develop as a retina because *Tfec* is induced. We conclude that *Pax6* has dose-dependent roles in RPE development that involve complex transcriptional and cell biological feed-back mechanisms.

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Program/Abstract # 177
Prep1 regulates lens induction via direct regulation of the Pax6 ectodermal enhancer

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