development. Two of those affect hair cells. One of these mutations (named C27) shows a normal pattern of neuromast, but it has a strong lacking of hair cells in them. This is because hair cells degenerate. We are currently mapping all these mutations and we have identified at least 3 of these genes responsible for the phenotypes found in the screening. C27 has been mapped to the linkage group 25 in an interval containing 25 genes where no one has been previously related to hair cell development. These findings indicate that C27 represents a new degenerative mutation.

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Program/Abstract # 300
prdm1a regulates Rohon-Beard neuron and neural crest cell fate at the neural plate border
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Neural crest (NC) cells and Rohon-Beard (RB) sensory neurons are induced at the neural plate border (NPB) in zebrafish, but the molecular mechanisms that are required for their induction remain unclear. Analysis of the *prdm1a* mutation has demonstrated that RB neurons are completely absent and neural crest cells and their derivatives are reduced. Since *prdm1a* does not affect cell proliferation or cell death at the neural plate border, we have completed a series of double fluorescent in situ hybridization experiments to determine which genes *prdm1a* may regulate. These data along with other genetic studies provide evidence supporting the hypothesis that RB and NCC specification are linked. It is unknown whether precursor cells exist that give rise to both cell types, or if a separate pool of precursor cells reside in the same developmental domain. Here we use fate mapping and live imaging techniques to investigate the lineage relationships between neural crest cells and Rohon-Beard sensory neurons. Combined with loss-of-function experiments, we hope to elucidate how *prdm1a* regulates RB and NCC specification at the neural plate border.

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Program/Abstract # 301
Zebrafish *paf1* is required for neural crest specification
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The Neural Crest (NC) is a uniquely vertebrate population of multipotent embryonic cells that gives rise to a diverse array of tissues, including cartilage and bone of the face, cells of the PNS, and endocrine cells. NC development is a highly orchestrated process that includes multiple tissue interactions, growth factor signaling pathways and transcription factors. Perturbations in NC development can severely impact human health. Many human diseases, such as congenital malformations and cancers, can be linked to impaired NC development. Therefore understanding what factors and pathways contribute to early neural crest development may provide insight into the pathogenesis of human NC diseases. We have a zebrafish NC mutant, *alyron* (*aln*), which fails to generate the full complement of premigratory NC and their derivatives. We show that NC is normally induced, but specification of NC is disrupted in *aln* mutants. *aln* encodes the *RNA Polymerase II Associated Factor 1* (*Paf1*) gene, a member of the multifactor Paf1 complex (Paf1C). Paf1C members have many roles including, histone modification, modification of transcriptional elongation, tumor suppression and control in maintaining embryonic stem cell identity. Although, *paf1* is widely expressed during embryogenesis, our rescue experiments indicate that *paf1* is required cell autonomously in the NC domain for proper NC formation. Further studies are aimed at determining how *paf1* is functioning to control NC specification and if other Paf1C members are functioning in a similar manner during NC development.

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Program/Abstract # 302
The methyltransferase NSD3 regulates neural crest cell specification and migration
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Neural crest cells are a multipotent vertebrate cell population that originates in the dorsal neural tube and migrates extensively to form multiple structures, including the peripheral nervous system, melanocytes, and parts of the craniofacial skeleton and heart. While a gene regulatory cascade that specifies neural crest cells has been described, not all cells expressing these factors eventually migrate, revealing an incomplete picture of neural crest development. Our data demonstrate an unexpected role for methylation reactions, mediated by the methyltransferase nuclear receptor-binding SET domain-containing 3 (NSD3), in neural crest cell development. NSD3 is expressed in premigratory neural crest cells in the neural folds and in migratory neural crest cells in the midbrain and in r2/r4 streams. NSD3 knock down caused a dramatic reduction in Sox10 expression in the neural folds, demonstrating that neural crest specification requires NSD3. Meanwhile, migration is drastically reduced and often absent in the few neural crest cells that form. In addition, NSD3 is required for Sox10 expression in the otic placode. As NSD3 is a lysine methyltransferase localized to both the nucleus and the cytoplasm of neural crest cells, we propose that both histone methylation and dynamic cytoplasmic protein methylation regulate neural crest cell formation and migration. We are currently assessing the importance of NSD3 subcellular localization and methyltransferase activity for neural crest formation and migration. Together, our data reveal NSD3 as a novel regulator of neural crest cell development.

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Program/Abstract # 303
Regulation of neural crest development by the putative phosphatase, paladin
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Neural crest cells arise in the dorsal neural tube and migrate into the periphery to form a variety of structures including sensory ganglia and facial bones. Efforts to characterize neural crest formation at the molecular level have focused on the transcription factors that specify neural crest cells in the ectoderm. However, neural crest gene expression does not guarantee eventual migration as a neural crest cell. We propose that differential protein activity, rather than differential gene expression, regulates neural crest cell migratory properties. One possibility is that the phosphorylation status of proteins important for neural crest migration determines the ability of neural crest cells to migrate. We have identified a