lack trunk somites, and *spt*/*ntl* double mutants lack all trunk and tail mesoderm, including tissues that do form in either single mutant. Thus *ntl* and *spt* are required for and work together to regulate the formation of all posterior mesoderm. To identify T-box downstream targets, we performed and published microarray analyses to generate an extensive list of putative target genes. Characterization of target gene promoters showed that several drive mesodermal expression largely controlled by Ntl- and Spt-responsive elements. This published work increased the number of known direct targets, and we are now further examining the role of these target genes as mediators of Ntl and Spt function. Through functional analysis of a selected set of genes, we hope to determine their place and importance in the hierarchy of mesoderm development.

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Program/Abstract # 135
p53 involvement in early cranial neural crest development in the chick
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Program/Abstract # 136
Regulation of chick neural crest cell migration by the Snail2 target alpha-N-catenin
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The neural crest, a population of migratory cells derived from the future central nervous system in the developing vertebrate embryo, gives rise to a diverse range of cell types, including most of the peripheral nervous system, melanocytes, and the craniofacial skeleton. Initially existing as adherent epithelial cells in the embryonic dorsal neural tube (the premigratory neural crest), these cells undergo an epithelial-to-mesenchymal transition (EMT), characterized by the loss of intercellular contacts and the generation of motile, mesenchymal neural crest cells. The Snail family of transcriptional repressors is known to play key roles during the EMTs underlying both normal embryonic development and disease. We have identified neural alpha-catenin (alpha-N-catenin), a component of the adherens junction, as a Snail2 target gene whose repression is critical for chick neural crest cell EMT and migration. Knock-down and overexpression of alpha-N-catenin enhances and inhibits neural crest cell migration, respectively, both in vivo and in vitro. Furthermore, our overexpression results identify actively migrating neural crest cells in the lumen of the neural tube, suggesting a role for alpha-N-catenin in the appropriate detachment of neural crest cells and movement away from the neural tube. Taken together, our data point to a novel function of an adherens junction protein in modulating neural crest cell EMT/emigration and proper migration of neural crest cells during chick embryogenesis.

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Program/Abstract # 137
PI15 modulates patterning of avian face
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Our lab has shown that applying Noggin and Retinoic acid to the early avian embryo causes a homeotic transformation in the face where the side of the beak is replaced by duplicated set of midline. *PI15* (Peptidase Inhibitor 15/Sugarcrisp) was one of the most highly induced genes that came out when a microarray experiment was performed on the facial mesenchyme after RA/Noggin treatment. In this study we investigated the hypothesis that PI15 patterns the face by modifying the activity of other proteins or morphogens. We found that *PI15* is expressed in all the regions of face but at older stages it is restricted only to the frontonasal mass. We first validated the microarray results by assaying the effects of RA and Noggin beads on *PI15* expression in embryos. We found that RA beads induced *PI15* to a much greater extent than Noggin suggesting that PI15 mediates the effects of RA. We also found that BMP4 and SHH induce their expression whereas FGF8 inhibits *PI15*. Over expression of PI15 in the face caused inhibition of formation of maxillary bones which is half of the transformation process. The other half is the gain of midline skeletal elements. To see whether *PI15* can replace RA and synergize with Noggin to induce transformation, we overexpressed *PI15* with virus-infected cells and simultaneously implant a bead soaked in a 10-fold lower concentration of Noggin (unable to induce any pattern changes). As hypothesized the combination of PI15 and low concentrations of Noggin induced a full set of duplicated elements. The focus of this study identified a new gene that mediates the effect of RA signalling pathway in controlling the jaw identity in the face.

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