Lineage analysis of Rohon–Beard sensory neurons and neural crest cells

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Neural crest cells (NCCs) and Rohon–Beard sensory neurons (RBs) both arise from the same region in developing embryos. NCCs migrate extensively and ultimately give rise to several cell types, including most of the peripheral nervous system. RBs are mechanosensory neurons whose cell bodies reside in the central nervous system of anamniote vertebrate embryos. Analysis of several zebrafish mutations suggests that these cell types may share a common progenitor, but it remains to be determined when and if this is the case. I hypothesize that a population of precursor cells exists prior to differentiation of cells at the neural plate border that is capable of giving rise to both NCCs and RB neurons, and that some of these precursors will ultimately give rise to both cell types. If the hypothesis is supported, a link will be established between cells in the central nervous system and the neural crest. The current project addresses this hypothesis using the lipophilic dye DiI to label neural plate and axial and paraxial mesoderm to determine when and if this is the case. I hypothesize that these cell types may share a common progenitor, but it remains to be determined when and if this is the case. I hypothesize that a population of precursor cells exists prior to differentiation of cells at the neural plate border that is capable of giving rise to both NCCs and RB neurons, and that some of these precursors will ultimately give rise to both cell types. If the hypothesis is supported, a link will be established between cells in the central nervous system and the neural crest. The current project addresses this hypothesis using the lipophilic dye DiI to label regions in the developing zebrafish embryo and map precursor cells for RBs and NCCs. Embryos are labeled in the stages prior to and including 90% epiboly, at which time the progenitors are thought to reside at the border of the neural plate and non-neural ectoderm, and are later examined for the presence of labeled RBs and/or NCCs. Preliminary data suggest the trunk neural crest precursors reside in the ventral posterior region of the embryo, and RB neurons potentially come from this same region.

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Epithelial–mesenchymal transition regulators Snail and Twist are required for PMC ingression in the sea urchin embryo

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Epithelial–mesenchymal transitions (EMTs) are fundamental to embryonic morphogenesis throughout the animal kingdom. At the onset of gastrulation in the sea urchin embryo, micromere-derived primary mesenchyme cells (PMCs) undergo an EMT process to ingress into the blastocele, and these cells later become the larval skeleton. Transcriptional regulators such as Snail and Twist have emerged as important molecules for controlling EMTs in many systems. Sea urchin snail and twist genes were cloned and characterized from Lytechinus variegatus. As expected, functional knockdown analyses of Snail with morpholino-substituted antisense oligonucleotides in whole embryos and chimeras demonstrated that Snail is required in micromeres for PMC ingression. We proceeded to place Snail in the sea urchin micromere–PMC gene regulatory network (GRN) as a key regulator of PMC ingression. Snail is expressed late in micromere specification downstream of Alx1 and is essential for ingestion. Phenotypes observed in Twist-deficient embryos indicate that Twist is also required for PMC ingression. Current efforts are to place Snail and Twist in the context of the PMC specification program and show these transcription factors to occupy central positions in the subnetwork that regulates EMTs in the sea urchin embryo.

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Clearing the way: The small GTPase RhoA and endomesodermal specification

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The monomeric GTP-binding protein RhoA is best known for its role in morphogenesis and cytoskeletal remodeling. Further research identifies RhoA involvement in cell polarity, cytokinesis, and dorsal closure. Studies in the sea urchin L. variegatus suggest a novel role for RhoA in endomesodermal specification. Near the top of the network specifying endomesoderm in the sea urchin is Wnt8, which positively regulates beta-catenin signaling in a community effect that drives the entire network. We have discovered that RhoA works in the Wnt8 pathway and identified a role for RhoA in endomesodermal SoxB1 clearance. For endomesodermal cells to be fully specified, SoxB1 must be progressively cleared from the nuclei of vegetal cells, where SoxB1 is thought to interfere with beta-catenin signaling. Our data reveals that when co-expressed with dominant negative Wnt8, activated RhoA is capable of rescuing SoxB1 clearance (as well as the expression of the downstream endodermal