forming primitive streak, notably FGF8. Inhibition of FGF signalling, through depletion of FGFs or inhibition of FGF8 expression results in a failure of mesoderm differentiation and streak formation, while increased FGF signalling stimulates axial mesoderm formation, but inhibits streak formation. In order to dissect the signalling pathways to mesoderm differentiation and movement we have inhibited several downstream signalling pathways. We show that FGF-mediated mesoderm induction is dependent on signalling through both the ERK/MAP kinase and the PI3 kinase pathway. Inhibition of either of these pathways results in inhibition of mesoderm formation as well as streak formation. However, overexpression of Sprouty2, a negative regulator of FGF receptor signalling, does not inhibit mesoderm formation, but does effectively inhibit the cell movements associated with streak formation. The underlying mechanisms are now under further investigation.

Reference

Chuai, M., et al., 2006. Cell movement during chick primitive streak formation. Dev. Biol. 296, 137–149.

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Program/Abstract # 424
Visualization of the epithelial-to-mesenchymal transition of individual trunk neural crest cells

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During avian neural morphogenesis at in the trunk at E2, neural crest cells undergo an epithelial-to-mesenchymal transition (EMT) in order to leave the dorsal region of the neural tube. To accomplish this EMT, neural crest cells must lose adhesion to the apical lumen of the neural tube and leave the neural tube at its basal surface. This developmental event has long fascinated biologists, yet it has not been observed directly. This leaves certain questions about the avian trunk EMT unanswered. Are neural crest cells required to lose apical attachments before they leave the neural tube? Is an asymmetric mitosis responsible for the EMT of trunk neural crest cells? We have imaged individual dorsal neural tube cells labeled with membrane-localized EGFP using time-lapse, laser-scanning confocal microscopy. No instances of asymmetric mitosis among neural tube cells have been observed. In some cases, neural crest cells leave behind small pieces of themselves as their trailing process breaks away from the apical surface. Some neural crest cells exit the neural tube at the position of their basal process. However, other neural crest cells form a new process and leave the neural tube at an entirely different location. While we verify that neural crest cells do eventually lose contact with the apical lumen of the neural tube, our observations suggest that the activation of cell motility may be more important in the EMT of trunk neural crest cells than the loss of cell–cell adhesion.

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Program/Abstract # 425
Molecular and cellular mechanisms of cranial skeleton development

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While the mechanisms of skeletal development in the trunk of an embryo are well understood, the molecular program that directs analogous process in the head remains largely uncharacterized. In the axial skeleton of the trunk, cartilaginous elements originate from the sclerotome. The situation in the head is more complex as the cranial skeleton receives contributions from cephalic paraxial mesoderm, neural crest and occipital somites. The aim of this investigation is to elucidate the molecular and cellular interactions that result in the induction of the cranial base using the developing chick embryo as an experimental system. Our results show that, like craniofacial myogenesis, development of the cranial base is delayed relative to the trunk. Yet, whilst Shh, the principal inducer of cartilage differentiation in the trunk, is expressed along the entire rostro-caudal axis in the notochord and in the neural tube, analysis of the expression of the Shh transcriptional targets, Ptc1 and -2, indicates that Shh signalling is not active in the early cranial mesenchyme. This suggests that Shh activity is antagonised by, as yet, unidentified signals. In the chick embryo, the chondrocranium starts to differentiate at stage 25. Recent data in zebrafish has indicated a role for Shh in the development of the chondrocranium. This suggests that although Shh signalling is initially blocked it may be later required for development of the cranial base. Analysis of Ptc1 and -2 expression indicates that this is very likely. Functional studies are in progress to analyse the role of Shh and to identify the tissue interactions required for chondrogenic differentiation.

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Program/Abstract # 426
The roles of tenascin-W in osteogenesis

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Tenascins are a family of large glycoproteins found in the extracellular matrix (ECM). We are characterizing the final