Abstracts

**Cell fate specification**

**Program/Abstract # 210**

Linking cell polarity to competence during heart specification

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To ensure proper development, coarse growth factor signals must be refined through a precise cellular response. Here we explore how asymmetric division generates differential competence within daughter cells, reinforcing their distinct identities. In embryos of the simple chordate, *Ciona intestinalis*, FGF/MAP kinase signaling causes four founder cells to undergo an asymmetric division, generating four small heart precursor cells and four large tail muscle precursors. Continued FGF/MAPK signaling in the smaller daughter cells leads to heart specification. Studies of dissociated cells indicate that when founder cells are uniformly exposed to FGF, heart specification occurs in both daughters. However, once the founder cells are polarized through their initial contact with endogenous FGF, subsequent exposure to uniform FGF has no effect on differential cell fate. This leads to a model in which FGF/MAPK generated cell polarity affects how daughter cells respond to further FGF signaling. We are currently studying the mechanisms that generate founder cell polarity and whether this polarity determines the distribution of FGF signaling components.

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**Program/Abstract # 211**

Vitamin overdose: Vitamin B3 processing by PNC-1 regulates *C. elegans* organ development

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Cells must integrate a variety of signals to develop functional organ complexes. To understand how molecular disturbances manifest physiological and anatomical defects, we use *C. elegans* as a model system. *C. elegans* provide a well-studied, tractable system for investigation of organogenesis with cellular resolution. Here I describe a novel role of the gene *pnc-1* in organogenesis. *PNC-1* is a nicotinamidase that converts the amide form of vitamin B3, nicotinamide (NAM), to nicotinic acid (NA) in the NAD⁺ salvage pathway. In *pnc-1* hermaphrodites gonadogenesis is temporally delayed and the four uv1 cells, which are an integral part of the uterine-vulva connection, die by necrosis (Huang and Hanna-Rose, 2006). Male *pnc-1* mutants are copulation defective, which may be due in part to the crumpled morphology of the copulatory spicules. We hypothesize that loss of PNC-1 activity should result in increased NAM and decreased NAD⁺. NAM is a potent inhibitor of Sirtuin deacetylases and PARPs, and thus may play an NAD⁺ independent role in development (Ruf et al., 1998; Avalos et al., 2005). I have found that supplementing NAM to worms can recapitulate the uv1 defect in wildtype worms, whereas feeding NA to *pnc-1* mutants can rescue the gonad delay. These results suggest that both NAM and NAD⁺ levels are relevant for *pnc-1*-mediated organ development. Also the mammalian functional ortholog of *pnc-1*, NAMPT, can partially rescue the developmental defects of *pnc-1* mutants. Therefore our studies of vitamin B3 processing will provide insight into its role regulating development.

References


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**Program/Abstract # 212**

An extrinsic cue regulates neuronal temporal identity in the *Drosophila* mushroom body

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The mushroom body is an insect brain structure formed by four neuroblasts, each of which divides to generate three major neuronal subtypes sequentially during development. We have used dietary and genetic experiments that uncouple mushroom body neuroblast divisions from organismal growth to show that the switch between neuronal subtypes is regulated by an extrinsic cue. Expression of a dominant negative version of one ecdysone receptor isoform disrupts the fate of later born neuronal subtypes. Furthermore, loss and gain of function experiments demonstrate that the ecdysone-responsive transcription factor *broad* influences the numbers and subtypes of neurons produced. Taken together, these results suggest that the steroid hormone ecdysone regulates the switch between mushroom body subtype development.

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