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Abstracts

Symposium 7: Organ systems in vertebrate development

Program/Abstract # 52
The role of GPI-anchored proteins in chondrogenesis and cell polarity

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Proteins that are localized to the cell surface via glycosylphosphatidylinositol (GPI) linkages have been proposed to regulate cell signaling and cell adhesion events involved in tissue patterning. Here we demonstrate that during skeletal development GPI anchored proteins play limited, focal roles in chondrogenesis. Conditional deletion of Piga, an essential enzyme in the GPI biosynthetic pathway, in the lateral plate mesoderm results in limbs that display chondrodysplasia. Analysis of mutant and mosaic Piga cartilage revealed two independent cell autonomous defects. First, loss of Piga function interferes with signal reception by cells and results in delayed chondrocyte maturation. Second, the proliferative chondrocytes, while present, fail to flatten and arrange into columns of clones as in wildtype tissue. We present evidence that the abnormal organization of mutant proliferative chondrocytes results from errors in cell intercalation following cell division. Consistent with the known relationship between cell polarity and cell intercalation, we additionally show that GPI anchored proteins regulate cell polarity in other tissues. Collectively, our data suggest that the distinct morphological features of the proliferative chondrocytes result from regulated cell polarity that is controlled independent of chondrocyte maturation.

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Program/Abstract # 53
Organ size control in mice

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Vertebrate organs reproducibly achieve a size that is proportionate to the entire body, but it is unclear how a target size is so precisely and reproducibly achieved. One possibility is that tissues have growth sensors that measure size and respond to deviations. To determine how accurately growth is measured during development, we developed techniques to perturb the number of progenitor cells in the primordia of the mouse pancreas and liver. Surprisingly, the two tissues differed in their ability to compensate for an early loss of progenitor cells (the liver did, the pancreas did not). These results suggest that embryonic tissues differ in their ability to sense size and make corrections; the presence or absence of such a sensor might underlie growth regulation during adult tissue regeneration.

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Program/Abstract # 54
Hoxb5b acts downstream of retinoic acid signaling in the forelimb field to restrict heart field potential in zebrafish

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Proper determination of organ size is critical for organ function. In the vertebrate embryo, retinoic acid (RA) signaling is required for the proper formation of many organs. We have recently found that RA signaling plays an important role in restricting heart size in zebrafish. However, the mechanism through which RA acts to limit cardiac cell number is not understood. Here, we demonstrate that RA signaling acts within the forelimb (fin) field to indirectly limit the size of the heart field. When RA signaling is reduced, fin progenitors are lost and the numbers of atrial and ventricular cardiac progenitors are increased, implying a link between the fin and heart lineages. Despite this inverse correlation, we find that RA signaling functions differently in fin and heart: RA signaling is required cell-autonomously to promote fin formation and non-autonomously to restrict heart formation. These results suggest that the fates of fin and cardiac progenitors are not directly linked and that the role of RA signaling in restricting cardiac cell number is indirect. Consistent with this hypothesis, we show that RA signaling positively regulates *hoxb5b* expression in the fin field and that *hoxb5b* is required to non-autonomously restrict atrial cell number. Therefore, our results indicate a novel mechanism by which communication between organ fields limits organ size and provide a new perspective on the possible causes of congenital syndromes affecting both the heart and the forelimb.

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Program/Abstract # 55
Finding closure: Visualizing the cell behaviors and uncovering the genetics of neural tube closure

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