right, and dorsal–ventral) that function to provide polarity cues for cell fate specification and migrations. The *C. elegans* body wall muscle cells are a great system in which to study spatial polarity cues because this tissue extends the length of the animal in four quadrants, occupying left and right positions on both the dorsal and ventral sides of the animal. One cue known to originate from body wall muscle is TGF-beta/UNC-129 that is produced at higher levels in dorsal versus ventral muscle. This pattern of *unc-129* expression is, in turn, generated by the activity of the transcriptional repressor UNC-130 that is preferentially produced in ventral bodywall muscle. To understand how these dorsal–ventral differences in expression are established, we determined the temporal pattern of factors required to establish ventral polarity by using a combination of reporter genes, imaging in live embryos, and antibody staining of fixed embryos. We can also define the cis-acting elements regulating *unc-130*, by identifying an evolutionary conserved sequence that is also sufficient to impose ventral polarity on a promoter element that normally is expressed uniformly in all body wall muscle. In order to find the trans-acting factor, we are performing a RNAi screen of candidate transcription factors in *C. elegans*. By identifying cell autonomous and non-autonomous factors regulating *unc-130* expression we hope to understand the logic behind the establishment and maintenance of spatial and temporal polarity cues important for animal development.

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Program/Abstract # 317

Signaling through BMP receptors promotes respiratory identity in the foregut through repression of SOX2

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The mammalian foregut gives rise to the dorsally-located esophagus and stomach, and the ventrally-located trachea and lung. Proper patterning and morphogenesis of the common foregut tube and its derived organs is essential for viability of the organism at birth. Previous studies suggest that bone morphogenetic protein (BMP) signaling is important for early stages of respiratory development. However, its precise role in this process remains unclear. Here we show that conditional inactivation of BMP Type I receptor genes *Bmpr1a* and *Bmpr1b* (*Bmpr1a;b*) in the ventral endoderm led to tracheal agenesis and ectopic primary bronchi. Molecular analyses show that these mutants exhibit a reduction of ventral endodermal marker NKX2.1 and a complementary expansion of dorsal markers SOX2 and p63 in the prospective trachea and primary bronchi. Furthermore, we found that inactivation of Sox2 rescued tracheal formation, but did not suppress ectopic lung budding in *Bmpr1a;b* mutant mice. Together, our data suggest that signaling through BMPRA1A:B performs at least two roles in early respiratory development: first, it promotes tracheal formation through repression of Sox2, and second, it restricts the site of lung bud initiation.

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Program/Abstract # 318

Ngn3 as a dosage sensitive driver for beta cell differentiation during embryogenesis and regeneration

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Ngn3 drives endocrine differentiation from endodermal progenitors during embryogenesis. We study how a specific portion of pancreatic progenitors is selected to activate Ngn3 expression, how Ngn3 level with each progenitor cells is controlled and how Ngn3 regulates islet cell differentiation. We show that during embryogenesis, Ngn3 must reach a high level to push pancreatic progenitor cells to endocrine islet cell fates. Reducing Ngn3 dosage pushes Ngn3+ cells to pancreatic duct and acinar cell fates, demonstrating that pancreatic progenitors maintain their multipotency before Ngn3 expression reaches a high level. Furthermore, we show that an absence of Ngn3 proteins reduces the transcription of Ngn3 in individual cells while increasing the number of progenitors that activate Ngn3 transcription, suggesting that Ngn3 utilizes both feed-forward and feed-back mechanisms to regulate its expression. In the adult stages, regenerative conditions, including partial pancreatectomy and partial duct ligation, could activate a low level of Ngn3 expression in both pancreatic duct and acinar cells. However, cell lineage tracing showed that most of these Ngn3+ cells do not differentiate into endocrine islet cells, as in embryonic pancreas. These data suggest that although the adult pancreatic cells maintain a degree of plasticity, a simple injury is not sufficient to induce these cells to become endocrine islet cells. These findings also suggest that microenvironments (factors) in the embryonic pancreas can cell autonomously reprogram the adult pancreatic cells.

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