injected after the onset of neural development, to assess the role Sip1 during cranial neural crest development. With this approach we can determine whether Sip1 functions similarly in cancer cells and neural crest EMT and if it has any additional functions during embryonic development. Our results show that loss of Sip1 has no effect on cranial crest specification, but it prevents or delays migration of the crest out of the dorsal neural tube. Future studies will identify the mechanism by which Sip1 regulates the onset of migration in these cells.

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Program/Abstract #455
Interaction between Cdx transcription factors and the Retinoic Acid pathway in patterning the posterior neural plate
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In vertebrates, the central nervous system (CNS) originates from a uniform neural tube that becomes patterned along the anterior–posterior axis to give rise to the distinct structures of the forebrain, midbrain, hindbrain and spinal cord. While the anterior portion of the neural tissue gives rise to the forebrain and midbrain, the posterior neural plate is subdivided to form both a segmented hindbrain and an unsegmented spinal cord. We have previously shown that Cdx transcription factors are required for the patterning of the posterior neural plate, where in the absence of Cdx, the spinal cord is replaced by a mirror image duplicated posterior hindbrain. Extensive data have indicated that Cdx transcription factors play a conserved role in regulating posterior hox gene expression in the CNS. However, overexpression of posterior hox genes in Cdx-deficient embryos failed to rescue spinal cord identity, suggesting that other genes required for spinal cord specification may be under the control of Cdx transcription factors. To identify these downstream targets we performed a microarray experiment by comparing the gene expression profile of wild-type and Cdx deficient embryos. Based on the microarray results, we are currently investigating the role that Cdx transcription factors play in modulating the Retinoic Acid (RA) signaling pathway and the requirement of RA signaling in spinal cord specification.

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Program/Abstract #456
Merging anterior–posterior and dorsal–ventral markers to trace neuronal lineages in the mouse brainstem
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In the developing neural tube early anterior–posterior (A–P) and dorsal–ventral (D–V) patterning is thought to specify unique neural progenitor identities. In this study we trace the fate of neurons that develop at two distinct A–P locations but within the same D–V neural progenitor domain. We use a D–V cell fate marker to express the expression of cyan fluorescent protein upon CRE mediated recombination. The D–V marker we selected is a homeobox domain factor called Chx10 which is expressed in V2a neurons of the ventral spinal cord and hindbrain. The CRE is driven by the endogenous En1 promoter to target neurons in rhombomere 1 (r1) or by a minimal rhombomere 2 (r2) specific transgenic promoter. Locations of V2a neurons that originate from r1 and r2 were mapped by analyzing serial sections through the hindbrain. These analyses clearly demonstrate that V2a neurons which originate from r1 constitute three separate nuclei in the pontine tegmentum and one in the ventral midbrain. In the pontine tegmentum r1-derived V2a neurons are found in the pedunculopontine tegmental nucleus, lateral dorsal tegmental nucleus and subpeduncular tegmental nucleus. In the midbrain r1-derived neurons are restricted to the interpeduncular nucleus. In contrast all r2-derived neurons migrate into the pontine reticular formation. Despite localization to distinct nuclei, most V2a neurons whether derived from r1 or r2 are glutamatergic. However, their axonal projections are distinct and appear to be determined based on their final location within the pontine tegmentum or the midbrain. Together these mapping studies demonstrate that even within a narrowly defined domain these progenitors can generate functionally distinct populations of neurons.

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Program/Abstract #457
GDF11 regulates temporal progression of neurogenesis but not anterior–posterior patterning in the zebrafish spinal cord
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Generation of distinct types of neurons from progenitor cells during the development of the nervous system follows a precise spatial and temporal order. The spatial and temporal identity of spinal neurons along the anterior–posterior (A–P) axis of the embryo is influenced by intrinsic properties of the progenitors and by extra- cellular cues. Currently, little is known about the interactions between intrinsic and extrinsic factors in the control of spinal A–P identity. Here we investigate the function of the secreted molecule Growth Differentiation Factor (GDF) 11 in the A–P patterning of the zebrafish spinal cord. While GDF11 is essential for both temporal progression of neurogenesis and proper A–P patterning of the mouse spinal cord, we find that zebrafish GDF11 only regulates the temporal progression of neuronal differentiation and not spinal cord A–P patterning. Currently we are investigating possible redundancy of GDF11 with the close family members GDF8/Myostatin-A and -B in the A–P patterning of the zebrafish spinal cord.

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Program/Abstract #458
Regulating the function of Twist, an essential factor in neural crest development and tumor progression
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Neural crest cells are multipotent, proliferative stem cells that undergo an epithelial–mesenchymal transition (EMT) and migrate to distant regions of the embryo where they give rise to a large and diverse set of derivatives essential to the vertebrate body plan. Interestingly, a number of transcription factors that control EMTs during neural crest development, including Twist, Snail, Slug, and Sip1, also regulate tumor cell metastasis. Twist is a bHLH (basic helix-loop-helix) protein expressed during neural crest formation, migration, and fate diversification. Depletion of Twist in Xenopus leads to defects in cranial neural crest formation and migration, demonstrating its necessity for the normal development of these cells. The mechanisms via which Twist regulates cell fate decisions, as well as EMTs in both embryonic development and cancer metastasis, remain poorly understood. Here we show that an E3 ubiquitin ligase, Ppa, that had previously been shown to regulate Slug/Snail protein levels,