migration using the Matlab software. One major challenge to quantifying this data is that CNC explants inherently possess polarity, making it difficult to decipher between the polarity of the explant vs. cells moving toward a target. Our goal is to resolve cells that are moving with directionality from those that migrate randomly so that we can determine if CNC cells are moving toward a potential chemoattractant.

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Program/Abstract # 287
Regulation of rear retraction and nucleokinesis during interneuron migration

Jacqueline C. Simonet, Jeffrey A. Golden
Department of Cell and Dev. Biol., Univ. of Pennsylvania, Philadelphia, PA, USA

The highly organized structure of the human brain is achieved through precisely directed migration of neurons during development. Failed neuron migration is believed to underlie many human conditions including some forms of mental retardation, developmental disability, and epilepsy. In contrast to the amoeboid-like migration of other cells such as fibroblasts, migrating interneurons exhibit a complex migratory behavior with discontinuous movements. First, the leading process extends, then the nucleus moves forward, and finally, the trailing process retracts. The mechanisms of nuclear movement (nucleokinesis) and trailing process retraction (rear retraction) and how that process coordinates with nucleokinesis is poorly understood. It has previously been shown that non-muscle myosin II motor protein is localized to the rear of migrating interneurons and inhibition of myosin II halts nucleokinesis. RhoA, a Rho GTPase, is necessary for rear retraction in migrating monocytes, lymphocytes, and endothelial cells. In monocytes and lymphocytes, RhoA regulates detachment of adhesion at the rear of the cell and in endothelial cells RhoA regulates myosin II contractility. Here we show that RhoA and its specific kinase, ROCK, are involved in regulation of rear retraction and nucleokinesis in migrating interneurons. RhoA through ROCK activates myosin contraction at the rear of these interneurons. This contraction causes retraction of the trailing process and pushes the nucleus forward. These data indicate that interneurons, although having distinct migration morphologies from other migratory cell types, are regulated by similar molecular pathways.

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Program/Abstract # 288
Analysis of the role of Prickle1b during facial branchiomotor neuron migration

Oni Mapp[a], Anna Griffith[b], Monica Rohrschneider[a], Victoria Prince[a,b]
[a]*Committee on Dev. Bio., Univ. of Chicago, Chicago, IL, USA*
[b]*Department of Organismal Bio. and Anat.; Univ. of Chicago, Chicago, IL, USA*

The facial neurons are a subset of branchiomotor neurons that undergo a characteristic tangential migration through the hindbrain during development. The goal of this research is to investigate several aspects of this migration in zebrafish. Recently, our lab has identified a cell-autonomous role for the gene *prickle1b* (*pk1b*) during facial neuron migration. Pk1 orthologues have well-demonstrated roles in establishing planar cell polarity (PCP), however previous work has demonstrated that several other PCP genes are required primarily in the surrounding neural tissue in order to mediate facial neuron migration. Subcellular localization of Pk1 is important for its various functions; while localization at the cell membrane appears to be required for PCP processes, Pk1 also functions in the nucleus. We find that deletion of the C-terminal prenylation domain of Pk1b eliminates its nuclear localization, but does not affect Vangl2-mediated localization at the cell membrane. Correspondingly, analysis of embryos deficient in isoprenoid synthesis demonstrates a role for prenylation during facial neuron migration. Using mutagenesis and transgenic techniques, we have designed a rescue experiment to test the functional requirements for Pk1b domains during facial neuron migration, and our preliminary data suggests that the prenylation domain is required in this context. This suggests that Pk1b may function in a partially PCP-independent fashion, and our ongoing experiments will test this hypothesis.

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Program/Abstract # 289
Impaired neural crest migration contributes to midgestation lethality of beta-actin knock-out mouse

Davina Tondeleir[a,b], Matthias Müller[c], Veronique Jonckheere[a,b], Davy Waterschoot[a,b], Karima Bakkali[a,b], Joël Vandekerckhove[a,b], Christophe Ampe[a,b], Anja Lambrechts[a,b]
[a]*Department of Medical Protein Research, Flanders Institute for Biotechnology (VIB), Ghent, Belgium*
[b]*Department of Biochemistry, Faculty of Medicine and Health Sciences, Ghent Univ, Ghent, Belgium*
[c]*Novartis-Phama, Basel, Switzerland*

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Program/Abstract # 290
Regulation of neural crest migration by the putative phosphatase, paladin

Julaine Roffers-Agarwal, Karla Hutt, Laura S. Gammill
Department of Genetics, Cell Biology and Development, University of Minnesota, Minneapolis, MN 55455, USA

Neural crest cells arise in the dorsal neural tube and migrate into the periphery to form a variety of structures including sensory ganglia, bones of the face and heart septa. Efforts to characterize neural crest formation at the molecular level have focused on the transcription factors that specify neural crest cells in the ectoderm. However, neural crest gene expression does not guarantee eventual migration as a neural crest cell. We propose that differential protein activity, rather than differential gene expression, regulates neural crest cell migratory properties. One possibility is that the phosphorylation status of proteins important for neural crest migration determines the ability of neural crest cells to migrate. We have identified a putative phosphatase, paladin, that is expressed by premigratory neural crest cells and upregulated at the initiation of neural crest migration in both chick and mouse embryos. Paladin knockdown in chick embryos inhibits neural crest migration and delays expression of the neural crest transcription factor snail-2, but does not affect the expression of other markers of neural crest specification. Additionally, we have begun to characterize neural crest migration in a mouse knockout and to analyze the phosphatase activity of paladin. Together, these data indicate that paladin is an important regulator of neural crest migration and support the notion that phosphorylation plays an important role in this process.

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