changes at E10.5. From these results, we hypothesize that the migration of the CNCC is arrested prematurely because they fail to activate specific genes necessary for their migration and to distinguish themselves from the CNCC in the pharyngeal arch arteries. In the future, we will identify genes differentially expressed in the two subpopulations of CNCC regulated by Ednra signaling, which could explain CNCC behavior and the Ednra mouse phenotype.

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Program/Abstract # 315
Calcium transients in trunk neural crest reveal the dynamics of cell migration and aggregation during peripheral nervous system development
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Examining calcium events during neural crest (NC) migration has the potential to shed light on cell communication during migration and patterning events. However, typical analysis of calcium transients during embryogenesis has been limited to cultured cells using fluorescent indicators added to the culture media which make the cells easy to visualize but not necessarily representative of their in vivo behavior. Here we studied the spontaneous calcium transients during NC migration in vivo using a genetically encoded calcium sensor (GECI), GCaMP3 which is not toxic to the embryo like synthetic indicators but is still easily imaged. The GCaMP3 vector was electroporated into pre-migratory NC cells and calcium transients were visualized in vivo in whole chick embryos and in sagittal slice trunk explants using confocal time-lapse imaging. First we conclude trunk NC cells displayed significantly more calcium transients than cranial NC cells, especially once trunk NC cells reached the dorsal aorta and during aggregation into sympathetic ganglia (SG). Second, blocking of N-cadherin activity in trunk NC cells near the presumptive SG led to a dramatic decrease in the frequency of calcium transients. Finally, we found that calcium transients were predictive of trunk NC cells aggregating into a cluster to form an SG anlagen. Our data suggest a model in which calcium transients are correlated with trunk NC cell migration and aggregation of cells into discrete sympathetic ganglia and highlight the power of using GECIs during an embryonic event.

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Program/Abstract # 316
Migration and transcriptional profiling of sacral neural crest derivatives in the lower urinary tract
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The neural crest (NC) gives rise to the peripheral nervous system including the innervation of the lower urogenital tract (LUT) which is essential for proper function of the bladder and genitalia. However, remarkably little is known about the molecular events that control the migration of sacral NC-derived cells that populate the LUT to produce pelvic neural elements. We utilized transgenic mice that express a Histone2BVenus (H2BVenus) reporter from Sox10 regulatory regions to visualize migration of sacral NC and capture these progenitors for analysis of gene expression in the developing bladder and pelvic ganglia. We imaged Sox10-H2BVenus embryos from E12-16 to determine migration routes and timing of NC cell entry into the LUT. H2BVenus+ progenitors form the anlagen of the pelvic ganglia by E12, enter the bladder by E13 and reach the bladder dome by E15. Using robust flow sorting methods, we isolated H2BVenus+ progenitors from LUT regions of transgenic embryos to obtain stage and sub-domain specific transcriptional profiles by microarray hybridization. To identify pathways that control development of LUT innervation, we compared the gene expression profiles of NC-derived progenitors against total embryo RNA. Subsequent comparisons of the up- and down-regulated genes were made between each stage and subdomain isolated. These simple two by two comparisons allowed identification of genes that are differentially expressed between NC progenitor populations. Understanding the factors that guide migration and differentiation of NC-derived progenitors during innervation of pelvic organs will help provide insight into increasingly prevalent LUT malformations and conditions, such as neurogenic bladder.

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