was initiated in the diphyodont pig, the dental lamina between the oral epithelium and the primary tooth became disconnected and fragmented into several pieces. The degradation of the pig lamina occurred during mid-gestation and injection of DiI into the dental lamina revealed high migratory potential of the lamina cells towards the mesenchyme. The dental lamina of all examined reptiles was not interrupted and kept a compact morphology, connecting the individual tooth generations, including fang teeth, during prehatching and posthatching stages. From such observations it is possible that the degradation of the dental lamina plays a role in restricting the number of tooth generations. MB and OZ are supported by GAASCR (grant KJB601110910) and IRP IPAG No. AVOZ 5045015, AST is supported by an International Joint grant from the Royal Society (JP080875).

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Program/Abstract #514 Filling in the gaps: First look at neural crest migration in a non-Avian reptile

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The morphological architecture of all species results from a genomic-environmental interaction, from which the best fit is selected for a particular environmental niche. In vertebrates, the skull is the most architecturally diverse and embryologically complex anatomical feature of an organism and can tell us much about its life history. Neural Crest Cells (NCCs), a population of cells arising from the dorsal neural tube, migrate and provide the progenitor cells for most of the craniofacial tissues of vertebrates. Thus, playing a key role in cranial development, their highly conserved migratory patterns are of importance for comparative and medical study. Herein, we provide the first analyses of the patterns of neural crest cell migration and development in a squamate (i.e., phylogenetic group comprising lizards and snakes), the Veiled Chameleon (Reptilia: Squamata: Chamaeleonidae). Due to the phylogenetic placement of Reptilia as the sister group to Mammalia, using a squamate as an outgroup to study craniofacial development and evolution of NCCs provides the advantage of maintaining a more 'typical' diapsid cranial architecture (though still missing the lower temporal bar present in Sphenodontia) and thus lacking the extremely derived avian cranial architecture. Developmentally, this species provides a more suitable system for studying early embryonic development within squamata and also maintains a unique array of cranial, trunk and appendicular skeletal morphologies for studying functional and ecological adaptations.

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Program/Abstract #516 Uncovering the ancestral role of FGF signaling in neural development Doreen D. Cunningham, Elena S. Casey

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FGF signaling plays prominent roles in early neural development including in A-P patterning and neural induction. The role of FGF signaling during deuterostome neural induction, however, remains controversial and varies in the organism studied. In *Ciona* and chick, FGF is a key instructive signal for neural induction, whereas in Xenopus, it facilitates induction by inhibition of BMP signaling (neural default) and is necessary to maintain neural progenitor proliferation. It has proven difficult to tease out a role for FGF in neural induction that is independent of its role to reinforce the default pathway by either inhibiting BMP signaling or inducing mesodermal tissue. To help clarify these complexities, we are studying the role of FGF in the neural development of Saccoglossus kowalevskii, an organism in which neural induction is not dependent on BMP/Chordin antagonism. Furthermore, Saccoglossus is ideally situated at the base of the deuterostome phyla to elucidate the ancestral role of FGF signaling in neural development. While the long term goal of this work is to determine how the specification of a diffusely organized neuronal cell population is achieved independent of the conserved BMP/Chordin antagonism, here, we analyze the role of the FGF pathway in neural development.

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Program/Abstract #517 Vertebrate kidney innovation by ponzr1 Victoria M. Bedell^a, Anthony Person^b, Jon Larson^c, Anna McLoon^d, Darius Balciunas^e, Karl Clark^f, Katie Nelson^f, Brent Bill^g, Lisa Schimmenti^c, Soraya Beiraghi^c, Stephen Ekker^f ^aMayo Clinic Biochemistry and Molecular Biology, Rochester, MN, USA ^bMadison, WI, USA ^cUniversity of Minnesota, Minneapolis, MN, USA ^dCambridge, MA, USA ^eTemple University, Philadelphia, PA, USA ^fMayo Clinic, Rochester, MN, USA ^gUniversity of Minnesota, Los Angeles, CA, USA

The homeobox (hox) and paired box (pax) master regulatory gene families provide a foundation for animal body plan and organ structure. Master regulatory genes establish core pathways that control organogenesis. Despite these conserved genes, organs are expanded and modified in response to evolutionary pressures. How organ variation is encoded in the genome remains an open question. We molecularly and functionally characterized one member of an evolutionarily dynamic gene family, plac8 onzin related protein 1 (ponzr1), in the zebrafish. Genetically, ponzr1 functions downstream of pax2a and forms a feedback loop that modifies pax2a expression. Morpholino knockdown reveals ectopic midline expression of pax2a and wt1a at 24 hour post-fertilization (hpf). At 72hpf, ponzr1 knockdown results in a modified kidney with loss of the glomerulus and disrupted podocytes. Despite glomerular loss, the resulting kidney is a functioning structure reminiscent of the kidney found in aglomerular fish. Therefore, we propose a new model of kidney development wherein pax2a signals for kidney differentiation in the pronephric ducts and tubules, while ponzr1 serves as a switch to signal for a more complex kidney that filters with an integrated glomerulus. Examining a second organ system involved in osmotic homeostasis, we find that the wt1b -expressing pharyngeal arches, which will develop into the gills, do not form in ponzr1 morphants. We functionally demonstrate that ponzr1 can act as a transcription factor or cofactor. Together this work provides experimental evidence of an additional mechanism that incorporates evolutionarily dynamic, lineage-specific gene families into master regulatory gene networks to create functional organ diversity.

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