including cells of the peripheral nervous system, endocrine cells, melanocytes, and craniofacial bone and cartilage. Our current understanding of neural crest formation, specifically of cranial crest, is very limited despite their relevance in frequent birth defects (cleft pallet). Neural crest cells are thought to originate at the border between neural and non-neural ectoderm. We have initiated studies aimed at understanding the early induction of neural crest cells, including the cranial crest. To this end, we performed an in vitro specification assay under non-inducing conditions in early chick embryos. We have found that neural crest induction is underway during gastrula stages 3 to 4 in a restricted medial epiblast region, which is competent to generate neural crest cells in the absence of additional tissues or signals. This medial epiblast will soon express pax7 (stage 4+ to 5), and the expression of Pax7 will label the neural folds and migrating neural crest cells later on. By preventing Pax7 translation with morpholinos, we unveil its early requirement for neural crest formation in vivo. These data suggest that neural crest specification initiates much earlier than previously assumed and that Pax7 plays a critical role in the early steps towards crest.

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175

An NF-κB, Slug and Wnt network in *Xenopus*

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During neural crest formation and cancer cell metastasis, cells undergo a dramatic epithelial to mesenchymal transition (EMT) combined with the suppression of anoikis, a form of apoptosis that occurs when epithelial cells lose contact with their substrate. The related zinc finger transcription factors Slug and Snail have been implicated in the regulation of both processes. In the course of studies to separate these two functions during neural crest formation in *Xenopus*, we found that RelA, which encodes an NF-κB subunit protein, is positively regulated by Slug. RelA directly up-regulates expression of Slug, Snail, the neural crest marker Sox9, as well as the anti-apoptotic BclxL gene and inhibits accumulation of RNA encoding the pro-apoptotic protein p53. Both RelA and BclxL rescue the effects of blocking Slug expression on neural crest formation, and a dominant-negative form of RelA disrupts neural crest formation. Based on the use of dominant negative forms of IκB and acetyl-11-keto-β-boswellic acid, an inhibitor of IκB kinase activity, it appears that BclxL acts through an NF-κB/IκB-dependent process to regulate both RelA and Slug expression. To complete the regulatory circuit, we find that *Drosophila* WntD and *Xenopus* Wnt8 inhibit RelA RNA accumulation. Taken together, these observations indicate that a circuit analogous to the *Drosophila* Dorsal (NF-κB)/Snail (Slug) circuit is active in vertebrates and that NF-κB activity is required for neural crest formation in *Xenopus*. Work supported by NIH grant GM54001 and the American Heart Association.

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176

C. elegans Sp1 factor LEX-4 functions with the Wnt pathway

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The Sp1 transcription factors play an important role in the development of several organisms. There is evidence that the Sp1 members function in regulating Wnt pathway in both invertebrates and vertebrates. This study reports the cloning of the *C. elegans* Sp1 gene, *lex-4*, and elucidates its potential role in modulating Wnt regulated processes.

We isolated *lex-4(gu85)* in a genetic screen performed to identify partners that function with EGL-38, a Pax factor, in regulating the expression of a target gene *lin-48*. SNP mapping placed the *lex-4(gu85)* mutation on LGI, and sequencing of the genomic DNA revealed that it is a mutant allele of the Sp1 homolog Y40B1A.4. We find that *gu85* is a missense mutation that affects the DNA binding domain and abolishes the DNA binding ability of the protein. In addition, *lex-4(gu85)* mutant animals show a wide range of developmental defects that are similar to these seen in Wnt pathway mutants. Specifically, mutant hermaphrodites exhibit the Biv phenotype similar to *lin-17(frizzled)* and *lin-18(RTK)* mutants. We have used vulval cell markers that suggest that the Biv phenotype results from reversal of the P7.p cell lineage characteristic of the Wnt pathway mutants. We are currently working on dissecting the mechanism of action of the *lex-4* gene in the development of several organs that are responsive to the Wnt pathway and are attempting to determine how this gene functions with the Wnt pathway to influence development.

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177

Activation of *Goosecoid* transcription by Siamois and Twin

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In early *Xenopus laevis* development, the Spemann organizer regulates the patterning of the mesoderm at the marginal zone and is essential in the establishment of a complex and organized body plan. The transcription factors *siamois* (Sia) and *twin* (Twn) are expressed in the dorsal vegetal blastomeres in response to stabilized β-catenin and are thought to be key regulators of organizer gene expression. Sia and Twn are among the earliest organizer genes expressed following