XFEB, A Direct Target of ZIC1, is Involved in Neural Crest Development

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The peripheral nervous system, melanocytes and craniofacial cartilage and bone arise from neural crest cells that develop during early embryonic neural development. Transcription and signaling factors form a network to regulate this development. For example, it has been shown that Zic1 and Pax3 in conjunction are able to induce full neural crest cell development (Monsoro-Burq et al., 2005). Xfeb and Gbx2 also play roles during neural crest cell development as they are present in the same regions and developmental stages as the neural crest (Plouhinec et al., 2014; Li et al., 2009). A microarray identified Xfeb as a direct, downstream target of Zic1 (Li et al., 2006). An additional lab also identified Xfeb as a neural crest gene induced by Zic1 (Plouhinec et al., 2014). We hypothesize that Pax3,

Xfeb, Gbx2 and Zic1 are all part of the same gene regulatory network controlling neural crest development. To investigate the relationship between the Xfeb, Pax3, Gbx2, and Zic1 genes, we first upregulated Xfeb gene expression with sense RNA and down regulated Xfeb gene expression with morpholino oligonucleotides (MO). We used in situ hybridization to visualize neural crest induction by staining for Slug RNA expression, a known neural crest marker. Our results showed that embryos injected with Xfeb sense RNA expanded Slug expression while those injected with Xfeb MO diminished Slug expression. Given other labs' results suggesting that Zic1 plus Pax3 or Zic1 plus Gbx2 induced ectopic Slug expression, we will determine whether Xfeb plus Pax3 or Xfeb plus Gbx2 genes can induce ectopic Slug expression. These experiments will allow us to determine whether Xfeb acts in neural crest induction and will allow us to place Xfeb into the gene regulatory network that drives neural crest development.