Xfeb, a Direct Target of Zic1, is Involved in Neural Crest Development

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During early embryonic development, neural crest cells give rise to the peripheral nervous system, melanocytes, bone and craniofacial cartilage. A network of signaling and transcription factors regulate early neural crest development, including Zic1, Pax3, Gbx2, and Xfeb. Combinations of Zic1 plus Pax3 and Gbx2 plus Pax3 are able to induce ectopic neural crest development. We hypothesized that Xfeb also contributes to neural crest development, as it is present in the same region at the correct time. Besides being a direct downstream target of the transcription factor Zic1, Xfeb was also identified as a potential neural crest gene induced by Zic1in genomic screens. We hypothesize that pax3, Xfeb, gbx2 and zic1 are all part of a gene regulatory network controlling neural crest development. To investigate these relationships, we overexpressed the Xfeb gene using Xfeb sense RNA and inhibited Xfeb 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 show that embryos injected with Xfeb sense RNA expanded slug expression while those injected with Xfeb MO diminished slug expression. In further experiments, we injected embryos with pax3 sense RNA without and with Xfeb MO. Injection with pax3 sense RNA alone expanded slug expression, while embryos injected with pax3 sense RNA plus Xfeb MO showed a decrease in slug expression. This suggests that Xfeb acts downstream of Pax3 in the neural crest gene regulation network. Our next step will be to determine if upregulation of gbx2 or zic1 will rescue neural crest development in the absence of Xfeb. This research will contribute to our understanding of gene regulatory networks, and how these contribute to early neural crest development.