

was to test the hypothesis that a temporal shift in *cyp26b1* expression

the hope that the

findings could be applied to disease in humans because of their phylogenetic proximity to humans. An example in other vertebrates indicating retinoic acid (RA) as an essential inducer of

tooth development. In published work, manipulations in RA exposure influenced how teeth developed in rats and mice. Additionally, the Jackman lab observed that Zebrafish exposed to exogenous RA grew teeth that were longer and narrower compared to wild-type fish.

Currently, the Jackman lab is focused on learning more about *cyp26b1*, the only RA-degrading enzyme expressed in zebrafish during tooth development. In an experiment where *cyp26b1* levels had been reduced, the Jackman lab observed zebrafish with higher levels of RA and teeth that were narrower and longer than those of their wild-type siblings. The teeth of the mutant zebrafish had a remarkably

difference in tooth morphology between zebrafish and mountain minnows. The Jackman lab began investigating this theory by breeding transgenic zebrafish. This process involved transferring the mountain minnow *cyp26b1* enhancer, fused with green fluorescent protein (GFP), into zebrafish. As the transgenic fish develop, green fluorescence was observed. We observed that GFP expression was the same between the

mountain minnow and zebrafish *cyp26b1* enhancers. This result indicates that in future studies, the enhancers will need to be studied at an earlier stage of development to determine if the onset of expression differs.

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