Studying Tooth Development in Vertebrates
Tommy Reynolds, Class of 2026

This past summer, I worked in the Jackman Lab investigating vertebrate tooth morphogenesis in collaboration with the Gilbert lab at UMMC. The research for this project was conducted primarily on zebrafish (*Danio rerio*) and white cloud mountain minnows (*Tanichthys albonubes*) with the hope that the findings could be applied to diseases in humans because of their phylogenetic proximity to humans. An example of one such disease is Cleidocranial Dysplasia (CCD), a condition in humans leading to the underdevelopment of teeth because of genetic mutations that are poorly understood currently. However, there has been promising research in 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 similar shape to mountain minnow teeth which are exposed to cyp26b1 at a later stage in development. The comparison across species led to the hypothesis that regulation in the timing of cyp26b1 expression may have evolved differently between zebrafish and mountain minnows and explains why their teeth are different lengths.

My role this past summer was to test the hypothesis that a temporal shift in cyp26b1 expression is responsible for the differences 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 fluorescent light emits from the GFP protein when the attached cyp26b1 enhancer activates. Our original hypothesis was that GFP expression would be turned on by the zebrafish enhancer before the mountain minnow enhancer during tooth development. Studying the fish at 72 and 77 hours post fertilization, 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.

Faculty Mentor: Professor William Jackman
Funded by the Kibbee Science Fellowship