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GENETIC LABELING OF STRESS-REGULATING CELLS IN THE ZEBRAFISH HYPOTHALAMUS

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When cortisol levels increase in response to stress, neurons in the hypothalamus release corticotropin-releasing hormone binding protein (*crhbp*) to the pituitary gland. The c*rhbp* inhibits cortisol release, decreasing stress response. Zebrafish lef1 mutants that exhibit anxiety-like behaviors have been shown to be missing crhbp expression in the posterior hypothalamus. We hypothesize that loss of *crhbp+* neurons in these mutants leads to increased stress response. To test this, we will see whether *crhbp+* neurons in the hypothalamus project to the pituitary gland and determine whether they are required for stress regulation. The *crhbp* upstream regulatory region was cloned by PCR, and Multisite Gateway Technology was used to create a construct driving enhanced green fluorescent protein (EGFP) expression. Zebrafish embryos were injected with the construct and observed under a fluorescence microscope to confirm EGFP expression. These founders were raised to adulthood, and EGFP expression was observed in the brains of their progeny at 3 days post-fertilization. Data gathered thus far shows that the *crhbp* construct is capable of driving expression in neurons and axonal projections in the posterior hypothalamus. In situ hybridization results will be analyzed in order to confirm that cells expressing EGFP are indeed *crhbp+*. Transcription factor binding sites in the *crhbp* regulatory region will be identified, to learn more about the regulation of this gene. Understanding how *crhbp+* neurons are generated during development will provide insight into developing more targeted treatments for anxiety and other stress disorders that affect the general population.