Effects of Embryological Exposure to Cortisol on Gene Expression during Tailfin Regeneration in Adult Zebrafish

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Cortisol is a hormone released in response to acute stress that regulates blood pressure, controls the circadian rhythm, increases blood sugar, and controls many other processes at basal levels. However, prenatal stress in mothers can cause sustained elevated cortisol levels during pregnancy, leading to the early development of chronic diseases in the offspring. Additionally, while it is known that elevated cortisol levels impair wound healing in adult humans, we do not know how prenatal cortisol exposure can affect the wound healing process in the offspring. We investigated the hypothesis that early life cortisol exposure affects later life gene expression during regeneration in a zebrafish model. Specifically, zebrafish embryos were treated with cortisol via DMSO (vehicle) or DMSO only. Zebrafish tailfins were amputated and tissue samples were collected by our lab at multiple time points for RNA-sequencing. Principal component analysis (PCA) of the RNA-seq data revealed a strong effect of cortisol at 2 days post-amputation (dpa) of the tailfin. This corresponds to the blastema stage of regeneration, known to be critical for successful regeneration. Gene ontology analyses revealed that genes downregulated at 2 dpa were involved in processes including viral response, mitosis, and chromosomal organization. Notably, the suppression of mitotic activity resulted in incomplete tailfin regeneration overall. These results suggest that early life cortisol exposure affects regenerative capacity during adulthood, particularly through effects on gene regulation during the critical blastema stage of regeneration.

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