Genetics Day Poster Submission

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Title: INVESTIGATING ENHANCER AND PROTEIN DIVERGENCE AT FOLLISTATIN PARALOGS UNDERLYING GENETIC ASSIMILATION OF WING PLASTICITY

Abstract: Over the past several decades, we have gained considerable insight into how developmental gene regulatory networks produce morphological traits, bridging the gap between genotypic and phenotypic diversity between species. What remains unclear is how new traits might arise from environmentally induced variation within a single species. Some have proposed that genetic assimilation, a process by which environmentally induced phenotypes become entirely genetically determined, is a common mode of morphological innovation. However, the underlying mechanisms and evolutionary constraint on genetic assimilation in nature remain virtually unknown. In the pea aphid (Acyrthosiphon pisum), evidence suggests that separate duplications of the gene follistatin (fs) are responsible for female wing polyphenism (fs-2, arose first) and its genetic assimilation in males (fs-3, arose second). My work focuses on the functional characterization of cis-regulatory and protein-coding sequence at fs paralogs, and the evolutionary forces acting on them, to explore the mechanisms of, and constraint on genetic assimilation of wing polyphenism in pea aphids. The ancestral function of Fs proteins is to inhibit the TGF-beta ligands Myoglianin and Activin-beta (Myo and Act-beta). One possibility is that pea aphid fs paralogs have evolved induce winglessness via derived repression of another TGF-beta ligand, Decapentaplegic (Dpp), which is crucial for wing formation. Another possibility is that pea aphid fs paralogs rely on ancestral repression of Act-beta and Myo to partially block metamorphosis and growth of wing tissue. To analyze the potential contribution of protein evolution to the roles of fs-2 and fs-3 in wing polyphenism and genetic assimilation, I have overexpressed pea aphid Follistatin (Fs) protein paralogs and assessed their ability to repress Myo, Act-beta, and Dpp in Drosophila. To investigate how enhancer evolution may have modified gene regulation between the two paralogs, I use an integrative approach involving machine learning and transcription factor binding site prediction. I have identified candidate enhancers at fs-2 and fs-3 which exhibit changes in line with our hypothesis, and plan to test the in vivo activity of these enhancers in pea aphids and the fruit fly Drosophila melanogaster. This work aims to provide important insights into the molecular mechanistic basis of genetic assimilation, where control of trait variation is shifted from environment ("nurture") to genotype ("nature").