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Genetic changes cause morphological divergence between species, however, little is known about the genes involved. During my internship I investigated the genetic basis of sex and species-specific wing size morphology in closely related species of *Nasonia*.

In nature, *Nasonia* are reproductively isolated due to a bacterial endosymbiant, *Wolbachia.* After curing of the bacteria, the four closely related species of *Nasonia* can be successfully crossed. Inter-species crosses between *N. vitripennis* and *N. giraulti* enable insertion of *giraulti* alleles into an otherwise *vitripennis* background. These hybrid strains are called SILs, segmental introgression lines. During my research, I used SILs that contain regions of inserted *giraulti* DNA known to contribute to wing size.

My first aim was to identify gene(s) involved in changes to growth in the wing. Two major quantitative trait loci (QTL) found to affect wing size in male *Nasonia* have been previously identified, *ws1* and *wdw*. A third gene, found to influence sex and species-specific wing size, wing-size 2 (*ws2*) has been crudely mapped. By phenotype-based mapping of the *ws2* region using a visible marker I have fine-scale mapped *ws2* and identified several candidates. During my work at the Werren Lab, I have reduced the region known to contain *ws2* from 3.4 Mb to 250 kb in size. Although the exact identity of *ws2* remains unknown, we now have a more precise idea of its location.

My second objective was to quantify changes to wing growth caused by *ws2*. Overall area of the wing increases by 29%. Partial width measurements along the A-P axis reveal major growth is localized within the anterior-central region of the wing, a 158% increase. This finding supports the claim that changes to male wing size by *ws2* is driven by localized changes to size and shape. Changes to wing cell size and number were calculated by measuring thin hair-like structures (setae) on the wing. These results confirm more numerous cells, but not larger cells, is responsible for increased growth at the anterior-central region of the wing. Future studies related to expression patterns of candidate genes will provide further evidence for the mechanisms involving *ws2* and their relation to cell cycle progression.

My last aim was to do *in situ* hybridization to determine whether *giraulti* and *vitripennis* alleles of candidate genes are differential expressed in developing male wings. With most of my focus geared towards fine-scale mapping *ws2* and quantifying changes to growth, I made less progress with *in situ* hybridizations. I was able to familiarize myself with the protocol and began to prepare all the necessary material for performing the experiment. During my upcoming semester at the Werren lab, I plan to begin executing this step of my research.

During my time as an intern I was able to broaden my understanding of developmental biology both academically and practically. I examined current literature and mastered various laboratory techniques. I gained skills in referencing genomic databases and other bioinformatics applications. I am thankful for the great opportunity my EDEN internship has provided me.