Genetics Day Poster Submission

Presenter: Emiliano Martí

Category: Graduate Student

Authors: EMILIANO MARTÍ, Christina A. Muirhead, Beatriz Navarro Dominguez, Benjamin E. Goulet, Lori Wright, Daven C. Presgraves

Title: DEREPRESSION OF R1 AND R2 NON-LTR RETROTRANSPOSONS CAUSES HYBRID INCOMPATIBILITY IN DROSOPHILA

Abstract: As noted in Darwin's Origin of Species, the evolution of hybrid incompatibility- the intrinsic sterility or inviability of species hybrids- poses a paradox: how could such maladaptive phenotypes evolve by natural selection? The Bateson-Dobzhansky-Muller (BDM) model shows that hybrid fitness problems can result from genetic incompatibilities, as substitutions that accumulate in one species may not be compatible with alleles present only in the other. Recent work has implicated genetic conflicts mediated by pathogens and selfish genetic elements (SGEs) as the evolutionary force driving divergence between closely related species. The study of hybrid incompatibilities between young species can therefore provide insights into the fastest-evolving sequences in the genome and the factors responsible for their evolution. The three species of the Drosophila simulans clade- D. simulans, D. mauritiana and D. sechellia- diverged ~240kya and are important models for speciation research. All pairwise crosses between species produce fertile hybrid females and sterile hybrid males, consistent with Haldane's rule. Hybrid male sterility factors accumulate faster than other kinds of hybrid incompatibilities; however, even between these young species, a small number of strong hybrid lethality factors has evolved. One of these, hybrid lethal on the X (hlx), maps to the pericentric heterochromatin of the X chromosome of D. mauritiana and causes male lethality in the genetic background of its sibling species. By combining genetic and genomic approaches, we started to dissect the basis of this hybrid incompatibility. We have discovered that hlx-mediated hybrid lethality is caused by the derepression of two non-LTR retrotransposons, R1 and R2, that insert site-specifically into the 28S subunits of rRNA genes, rendering them non-functional. The mass derepression of R1 and R2 in hlx hybrid males results in new transposon insertions, incidental deletions of rRNA genes, and therefore a large decrease in the number of functional rDNA subunits. Our results suggest a direct role of retrotransposons as a proximal cause of hybrid incompatibilities.