Title

Evolution of Hybrid Incompatibilities in Gene Regulatory Networks

Author

Alexander Y. Tulchinsky, *University of Massachusetts Amherst* Follow

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First Advisor

Adam H. Porter

Second Advisor

Jeffrey L. Blanchard

Third Advisor

Ana L. Caicedo

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Abstract

Under the Dobzhansky-Muller model, postzygotic isolation results from incompatibility between interacting genes. Evidence points to regulatory networks as a rich source of incompatibilities that impact hybrid fitness. Pleiotropy is a natural feature of regulatory networks because regulatory elements generally have multiple targets. Both pleiotropy and hybrid incompatibility arise due to genetic interactions; therefore we can expect an intimate association between them. In the following chapters, I investigate the relationship between pleiotropy and hybrid incompatibility in the context of regulatory networks.

In chapter one, I extend a general network-based study of hybrid incompatibility by incorporating a sequence-based thermodynamic model of transcriptional regulation. In the absence of pleiotropy, hybrid misregulation of a positively selected trait evolves quickly as a consequence of non-recognition or spurious binding in regulatory interactions across species boundaries. In a conserved trait, hybrid incompatibility evolves much slower as a product of compensatory drift.

In chapter two, I show that pleiotropy can promote or constrain the evolution of hybrid incompatibility in a regulatory network depending on its fitness landscape, which emerges from the thermodynamic properties of molecular binding. Pleiotropy may promote hybrid incompatibility in accordance with the "selection, pleiotropy, and compensation model" of evolution, in which compensation for the pleiotropic side-effects of adaptation accelerates incompatibility in conserved traits. Pleiotropy can limit the evolution of hybrid incompatibility by constraining change in trans-acting regulatory elements in favor of adaptation at less pleiotropic downstream cis-regulatory targets. Without change in both interactors, incompatibility does not occur under the Dobzhansky-Muller model.

In chapter three, I evaluate the hypothesis that pleiotropy facilitates the onset of hybrid incompatibility under antagonistic coevolution, an ubiquitous and persistent source of natural selection. When infectivity and resistance in a host-parasite system are determined epistatically by network interactions, reciprocal selective pressure results in a genotypic chase. This causes pleiotropic mutations to accumulate and be compensated over time, producing intrinsic hybrid incompatibility in both species independent of local adaptation. Thus, cyclical antagonistic coevolution eventually overcomes constraint on pleiotropic loci, facilitating the evolution of regulatory incompatibilities commonly observed in hybrids.

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