John Bills and Nicholas Norton
Presentation Overview

December 2nd, 2012
Biology 519

Does robustness promote evolvability?

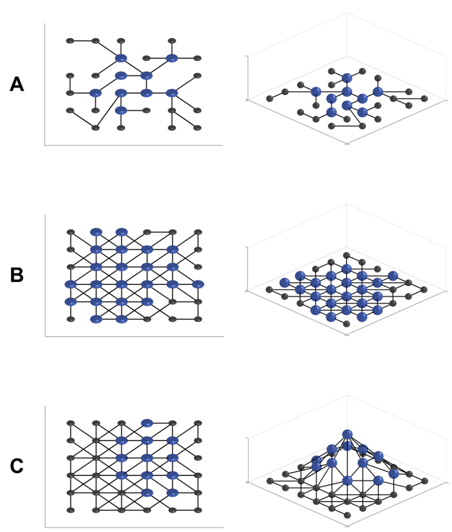
Introduction

What do the terms ‘evolvability’ & ‘robustness’ mean?

- **Evolvability** is the capacity of a population to produce heritable phenotypic variation of a kind that is not unconditionally deleterious.
- **Robustness** is the average effect of a specific perturbation on a specific phenotype relative to some control.
    - **Genotype robustness** appears to have an antagonistic relationship with evolvability
    - **Phenotype robustness** does appear to promote evolvability

High phenotypic robustness allows for **cryptic genetic variation** (standing genetic variation that does not ordinarily contribute to the phenotype) to accrue within populations.

Neutral networks (Fig 1) depict how low robustness **(A)** and high robustness **(B)** affect genotypes in a population. Blue circles represent genotypes within a population & black circles represent potential genotypes that are not present within a population. All of these genotypes confer the same phenotype. **(C)** represents the effects of perturbations on genotypes and phenotypes. Circles that are higher indicate a higher fitness.



Capacitor: biological switch that hides and reveals heritable phenotypic variation

- Capacitors form the link between robustness, cryptic genetic variation, and evolvability
- Stress, gene knockout mutations, and hybridizations are all examples of factors that may act as switches that change neutral alleles into adaptive alleles

Literature

Masel, Joanna, and Meredith V. Trotter. "Robustness and Evolvability." *Trends in Genetics* 26.9 (2010): 406-14. Print.

Wagner, Andreas. "Robustness and Evolvability: A Paradox Resolved." *Proceedings of the Royal Society B: Biological Sciences* 275.1630 (2008): 91-100. Print.

An Empirical Example of Cryptic Genetic Variation:

Suzuki, Y, and HF Nijhout. "Evolution of a Polyphenism by Genetic Accommodation." Science (new York, N.y.). 311.5761 (2006): 650-2. Print.

Introduction: The authors wanted to test the hypothesis that a polyphenism could evolve through genetic stabilization of a stress induced phenotype. Furthermore, they reasoned that this could be accomplished by releasing genetic variation in a monophonic line that has an ancestral polyphenic character for a particular trait. The tobacco hornworm, *Manduca sexta*, is exclusively green during its fifth instar larval stage (i.e.monophenic), even in response to heat shock, and has relatives polyphenic for this particular trait. A *M. sexta black* mutant shows reduced juvenile hormone (JH) secretions and is almost entirely black but exhibits a range of color phenotypes when heat-shocked.

Experiments/Results:

The authors created different *black* mutant lines, one with individuals showing increased color response to heat shock (polyphenic line), another with individuals showing reduced color response to heat shock (monophonic line), and an unselected control line. After 13 generations, the different lines showed a heritable difference in color response to heat shock as well as a different sensitivity to high temperatures.

To look at the physiological basis of these phenotypic differences, the authors looked at the color response of the two lines in response to increasing levels of a JH analog. Additionally, they looked at the role of JH concentration in creating color change in heat-shocked lines. The polyphonic line showed a higher sensitivity to JH and higher levels of JH in response to heat shock.

Conclusions:

1) Selection for or against polyphenism in heat shock produced lines with **a heritable difference in hormonal regulation**.

2) Genes that maintain expression of a developmental hormone above threshold activity can mask mutations in genes that respond to hormone!

3) Mutations in hormone control mechanisms can shift phenotypic thresholds and reveal this cryptic genetic variation!