

4 Canalization & Evolvability

4.1 Concepts

Heritability The ratio of the additive genetic variance to the total phenotypic variance.

Evolvability The genetic potential to generate adaptive heritable variation, i.e. the ability of a population to respond to selection¹. Note that evolvability is not the same as variability (see 3.1), because not all variation is adaptive. The instantaneous evolvability of a population is measured by the heritability. The evolvability can be affected by the mutation rate, recombination rate and the developmental system.

Capacitor hypothesis Successive increases and decreases in canalization can promote evolvability.

Epistasis The influence of an allele at one locus on the effects of alleles at other loci. In the absence of epistasis, alleles at different loci act independently on the phenotype. Canalization implies epistasis.

Pleiotropy The effect of an allele at one locus on more than one phenotypic trait. Pleiotropy can affect the canalization and evolvability of a trait.

4.2 How Does Canalization Evolve?

- Canalization could be adaptive, i.e. the insensitivity to perturbations might increase fitness.
- Selection on a trait can take many different forms. We can distinguish three simple types of selection on a quantitative trait:

Directional Selection for one extreme of a trait; after selection, the trait distribution shifts toward this extreme (change in mean).

Stabilizing Selection against both extremes of a trait; after selection, the trait distribution becomes narrower around the center (lower variance).

Disruptive Selection for both extremes of a trait; after selection, the trait distribution becomes wider (higher variance).

Stabilizing selection is generally expected to lead to the evolution of higher canalization; directional and directional selection are expected to lead to lower canalization.

- Certain instances of canalization may not be adaptive. For example, canalization could arise as a nonadaptive correlated byproduct of complex gene and trait networks.

4.3 Hsp90: A Canalization Gene

For the rest of the lecture we shall consider a model for canalization²:

- In most eukaryotes studied, the heat-shock protein Hsp90 is essential, abundant at normal temperatures and induced by stress. Hsp90 also interacts dynamically with a diverse set of unstable proteins, including signal transducers.
- In *D. melanogaster*, loss of Hsp90 function by mutation, pharmacological inhibition or environmental stress can produce a variety of morphological changes.
- These variants were not directly caused by Hsp90, but were based on previously silent genetic variation. It was possible to select on this polygenic variation so that the traits were expressed even after Hsp90 function was restored (genetic assimilation).
- Hsp90 might allow the storage and release of genetic variation in *Drosophila*. However, it is unclear whether Hsp90 has evolved to act as a “capacitor” of phenotypic variation, or if this effect is a by-product of its normal function.

4.4 Next Lecture

In the next lecture we will finish our exploration of the topic of canalization, by considering the robustness of genetic networks. Required reading is the paper by von Dassow *et al.* (2000)³.

4.5 Literature Cited

1. Wagner, G. P. & Altenberg, L. Complex adaptations and the evolution of evolvability. *Evolution* **50**, 967–976 (1996).
2. Rutherford, S. L. & Lindquist, S. Hsp90 as a capacitor for morphological evolution. *Nature* **396**, 336–342 (1998).
3. von Dassow, G., Meir, E., Munro, E. M. & Odell, G. M. The segment polarity network is a robust developmental module. *Nature* **406**, 188–192 (2000).