Evolutionary Genetics

LV 25600-01 | Lecture with exercises | 4KP

Natural Selection

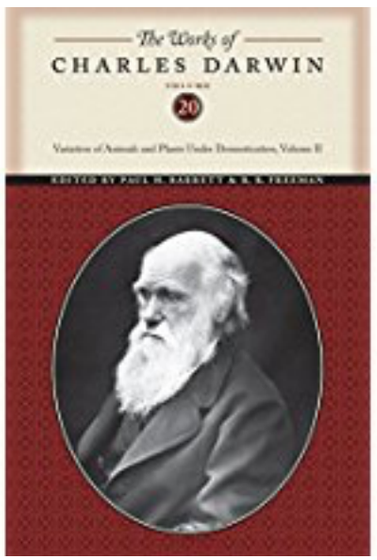
Darwin Awards

The Petri Dish: Darwin Awards

The **Darwin Awards** are a tongue-in-cheek tribute to individuals who have contributed to human evolution by removing themselves from the gene pool through an act of incredible stupidity, often resulting in their own death. The awards are named after Charles Darwin, the father of the theory of evolution, and humorously highlight instances where individuals remove themselves from reproduction through exceptionally foolish actions.

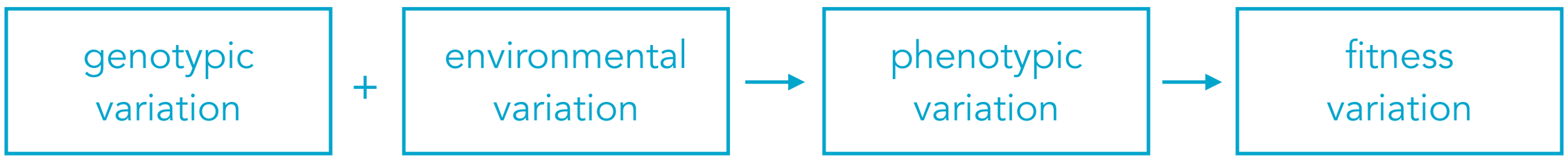
These "awards" are not official awards, but rather a concept used in a humorous way to highlight instances where people engage in highly risky or absurd behaviour that leads to their demise. The stories often involve acts of astonishing recklessness, bizarre accidents or extreme lack of common sense.

While the Darwin Awards aren't an actual ceremony with prizes, they have become part of popular culture, shared through books, websites and social media to entertain and sometimes warn against dangerous or ill-advised actions. The intention isn't to celebrate tragedy, but to highlight the importance of making safe and sensible choices in everyday life.

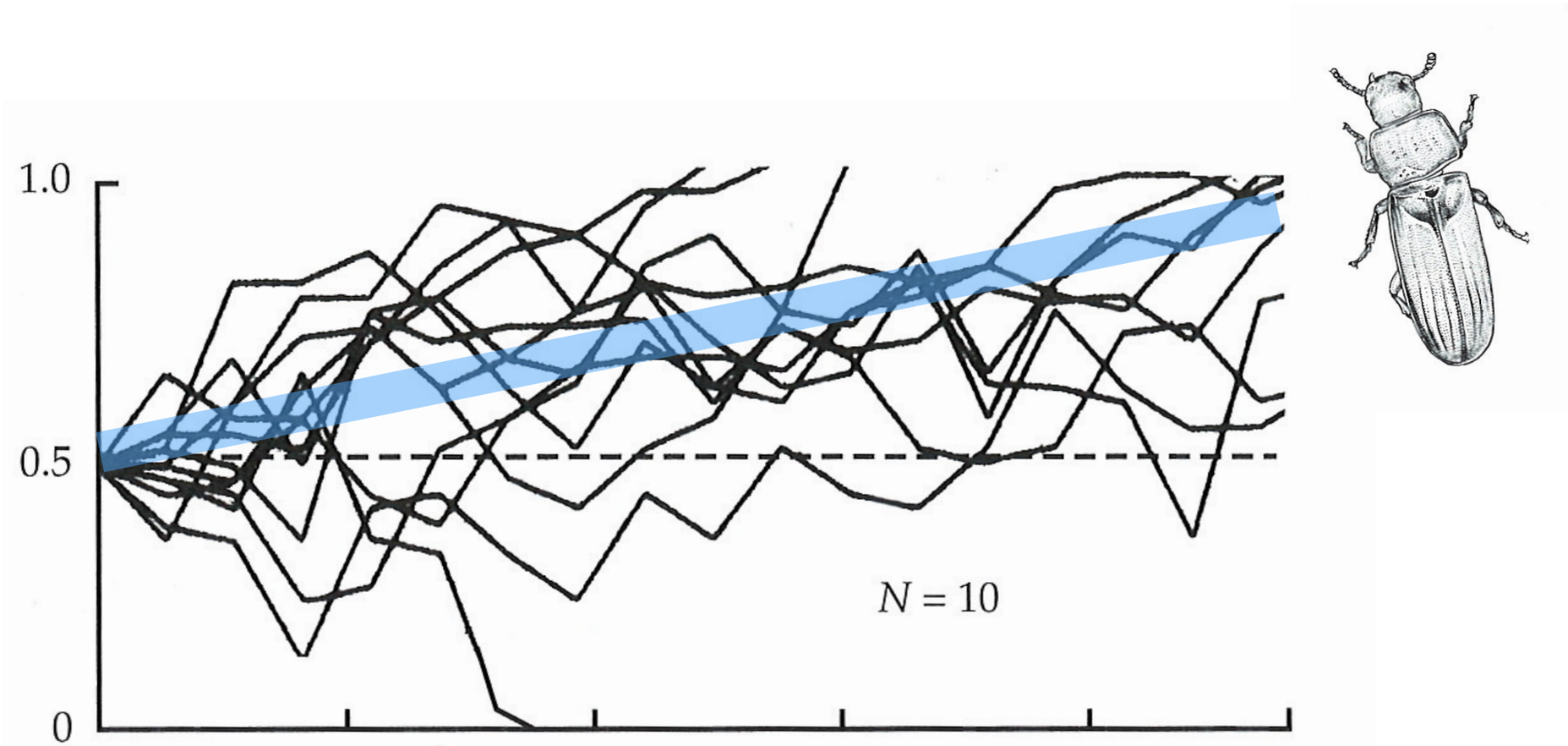


"The importance of the great principle of selection mainly lies in the power of selecting scarcely appreciable differences, which nevertheless are found to be transmissible, and which can be accumulated until the result is made manifest to the eyes of every beholder."

Charles Darwin (1859)



So far we have assumed that different genotypes have an equal chance of surviving and passing on their alleles to future generations. In other words, we have assumed that **natural selection** does not operate.



Conner and Hartl (2004) page 54

Biston betularia betularia morpha typica
(the white-bodied peppered moth)

Biston betularia betularia morpha carbonaria
(the black-bodied peppered moth)

The **peppered moth** (*Biston betularia*) is a famous example of natural selection and adaptation in response to environmental change during the Industrial Revolution in Britain.

The difference in colour between the white and black forms of the peppered moth is mainly due to genetic variation. **Both forms existed before the Industrial Revolution**, but **the light-coloured form (typica) was more common and better camouflaged against the lichen-covered tree bark that was prevalent in its natural habitat.**

However, as industrialisation progressed, soot and pollutants from factories darkened the tree trunks, causing the lichen to die off. In this changed environment, the darker variant of the peppered moth (carbonaria), which had always existed but was less common in the population, gained a survival advantage. **The darker colouration provided better camouflage against the darkened tree trunks, making the black form less visible to predators.**

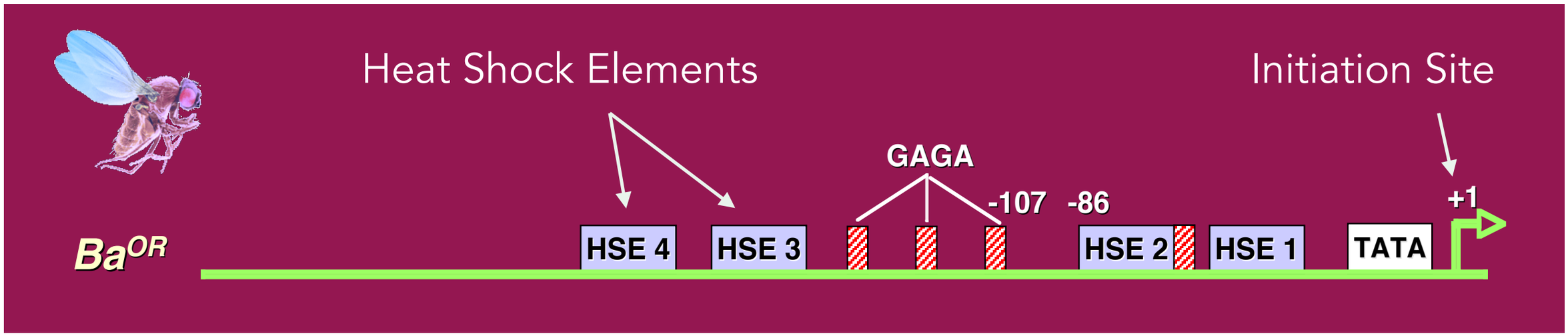
This shift in the moth population from predominantly light to predominantly dark individuals in a relatively short period of time is an example of natural selection driven by environmental change. **Predation pressure acted as a selective force**, favouring the survival and reproduction of the better camouflaged dark moths in the polluted environment.

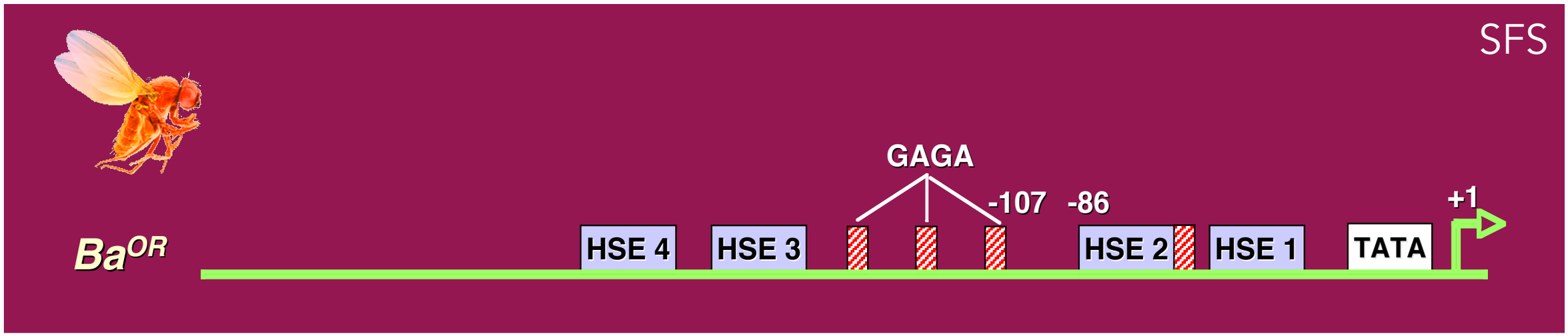
The **peppered moth became a textbook example of evolution by natural selection**, demonstrating how populations can adapt to changing environments through the prevalence of certain genetic traits that provide a survival advantage.

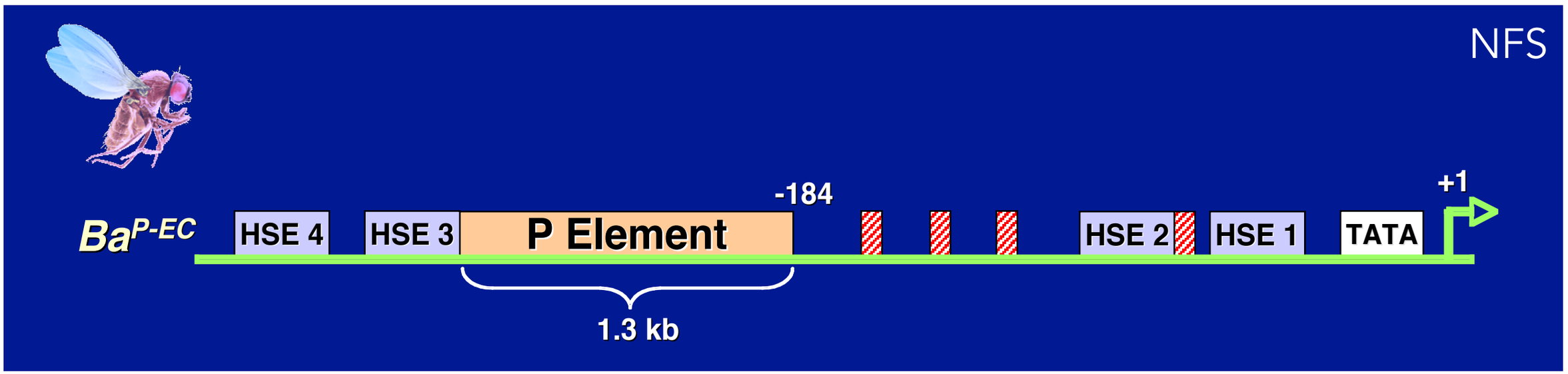
Hsp70 expression

Source: Michalak et al. (2001) Proc Natl Acad Sci USA 98(23): 13195-13200.

Hsp70 expression regulation

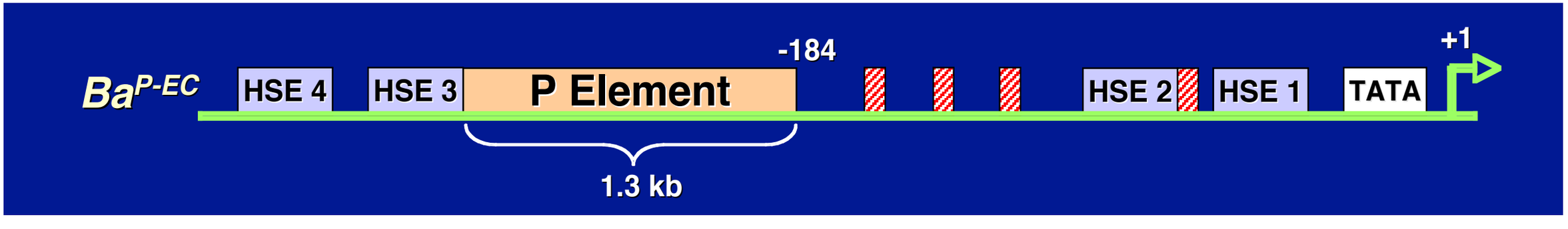



SFS
GAGA
-107
-86
+1
Ba^OR
HSE 4
HSE 3
HSE 2
HSE 1
TATA


NFS
-184
+1
Ba^P-EC
HSE 4
HSE 3
P Element
HSE 2
HSE 1
TATA
1.3 kb

freq(*Hsp70Ba*^{P})=**33.6%**

freq(*Hsp70Ba*^{P})=**1.2%**



OPEN ACCESS Freely available online

PLoS GENETICS

Heat-Shock Promoters: Targets for Evolution by *P* Transposable Elements in *Drosophila*

Jean-Claude Walser[1], Bing Chen[1], Martin E. Feder[1,2*]

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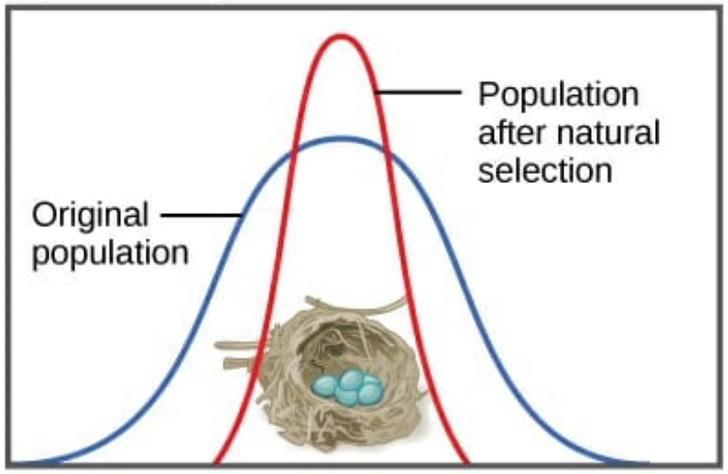
Transposable elements are potent agents of genomic change during evolution, but require access to chromatin for insertion—and not all genes provide equivalent access. To test whether the regulatory features of heat-shock genes render their proximal promoters especially susceptible to the insertion of transposable elements in nature, we conducted an unbiased screen of the proximal promoters of 18 heat-shock genes in 48 natural populations of *Drosophila*. More than 200 distinctive transposable elements had inserted into these promoters; greater than 96% are *P* elements. By contrast, few or no *P* element insertions segregate in natural populations in a "negative control" set of proximal promoters lacking the distinctive regulatory features of heat-shock genes. *P* element transpositions into these same genes during laboratory mutagenesis recapitulate these findings. The natural *P* element insertions cluster in specific sites in the promoters, with up to eight populations exhibiting *P* element insertions at the same position; laboratory insertions are into similar sites. By contrast, a "positive control" set of promoters resembling heat-shock promoters in regulatory features harbors few *P* element insertions in nature, but many insertions after experimental transposition in the laboratory. We conclude that the distinctive regulatory features that typify heat-shock genes (in *Drosophila*) are especially prone to mutagenesis via *P* elements in nature. Thus in nature, *P* elements create significant and distinctive variation in heat-shock genes, upon which evolutionary processes may act.

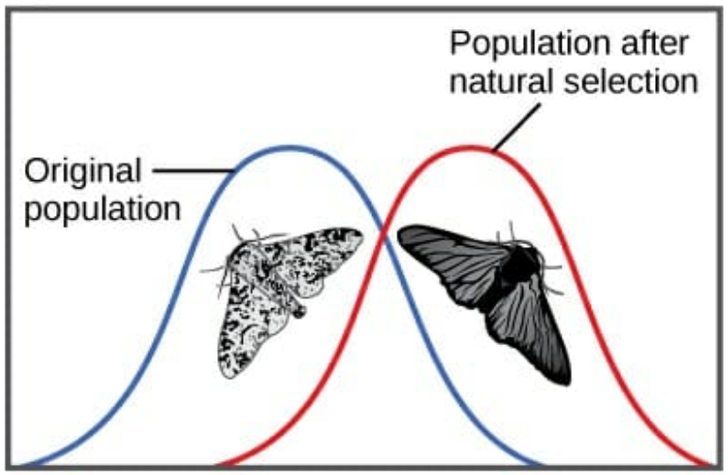
Citation: Walser JC, Chen B, Feder ME (2006) Heat-shock promoters: Targets for evolution by *P* transposable elements in *Drosophila*. PLoS Genet 2(10): e165. DOI: 10.1371/journal.pgen.0020165

(a) **Stabilizing selection**



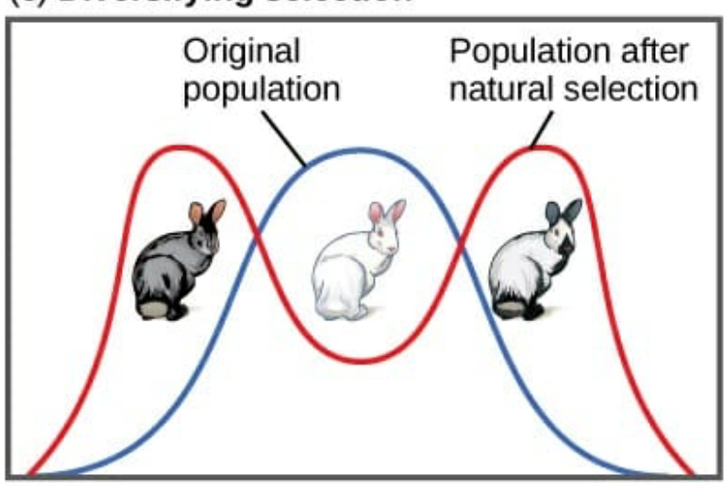
Robins typically lay four eggs, an example of stabilizing selection. Larger clutches may result in malnourished chicks, while smaller clutches may result in no viable offspring.

(b) **Directional selection**



Light-colored peppered moths are better camouflaged against a pristine environment; likewise, dark-colored peppered moths are better camouflaged against a sooty environment. Thus, as the Industrial Revolution progressed in nineteenth-century England, the color of the moth population shifted from light to dark, an example of directional selection.

(c) **Diversifying selection**

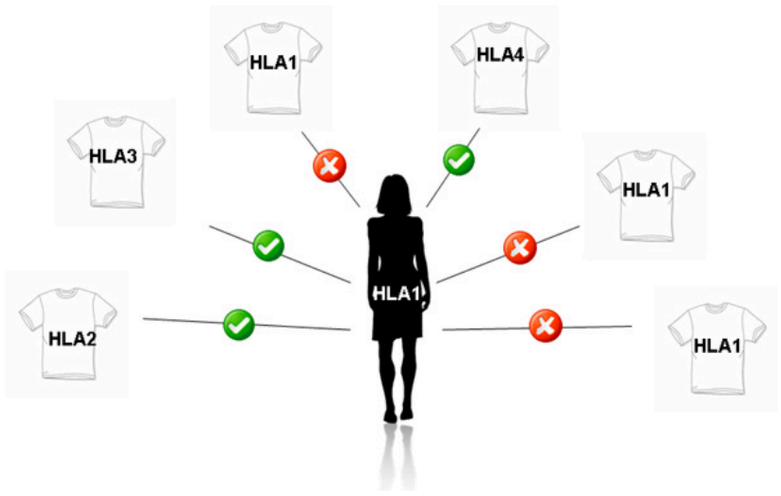


In a hyphothetical population, gray and Himalayan (gray and white) rabbits are better able to blend with a rocky environment than white rabbits, resulting in diversifying selection.

biology dictionary

Balancing selection is a type of natural selection that maintains genetic diversity in a population by favouring the persistence of multiple alleles (different versions of a gene) rather than promoting the fixation of a single allele. This can happen when heterozygotes have a fitness advantage over homozygotes, or when different alleles are favoured in different environments or under different conditions.

Balancing selection is essential to maintain genetic diversity and adaptability within populations. It prevents the fixation of a single allele, which can be beneficial in changing or heterogeneous environments. This diversity can provide a reservoir of genetic variation for future adaptation and evolution.



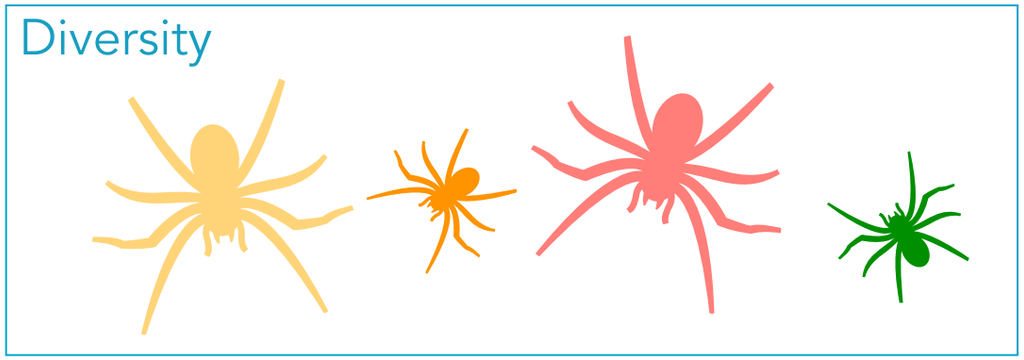
The MHC (Major Histocompatibility Complex) system is a prime example of how balancing selection can maintain genetic diversity within a population by favouring the presence of multiple alleles. This diversity is crucial for an effective and adaptable immune response to the ever-changing landscape of pathogens.

What is selection?

What does selection need?

What does selection do?

Can we avoid selection?



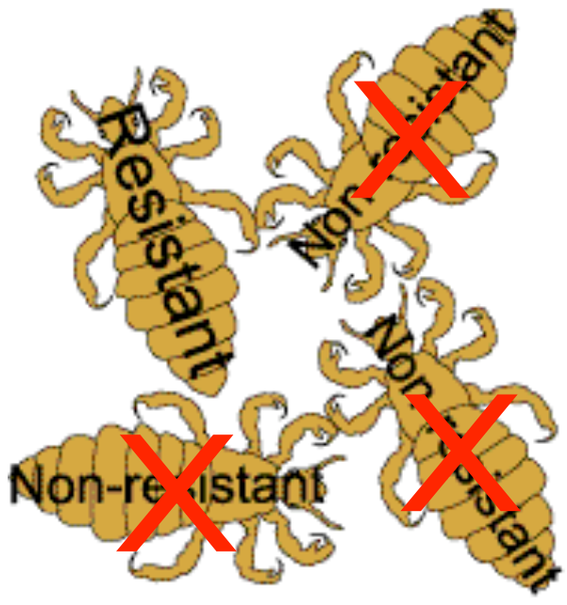
Variation - Within a population, there is genetic diversity, resulting in variations in traits among individuals.

Environmental Pressures - The environment poses challenges and pressures on the population, such as competition for resources or predation.

Differential Survival and Reproduction - Individuals with traits that are better adapted to their environment have a greater chance of surviving and reproducing, passing on their advantageous traits to the next generation.

Accumulation of Adaptations - Over time, natural selection can lead to the accumulation of beneficial traits, resulting in the adaptation of a population to its specific environment.

Hypothesis A

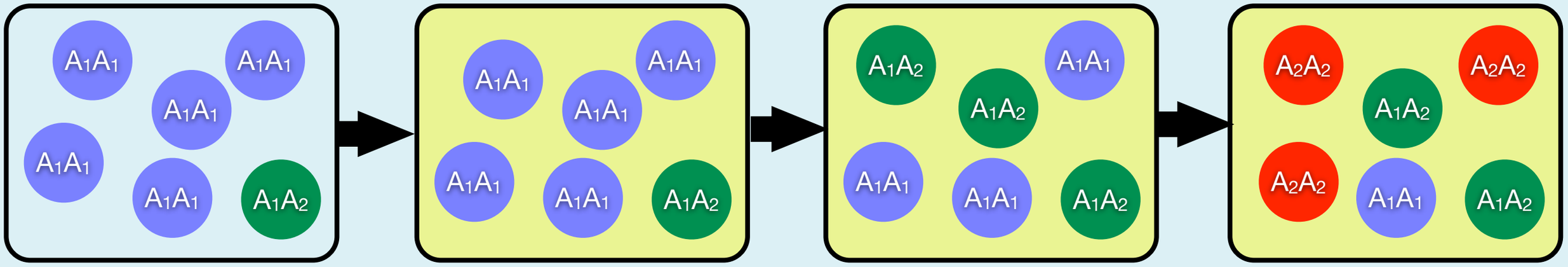


Resistant strains of lice have always been there, they are just more common now because all the non-resistant lice have died a sudsy death.

Hypothesis B

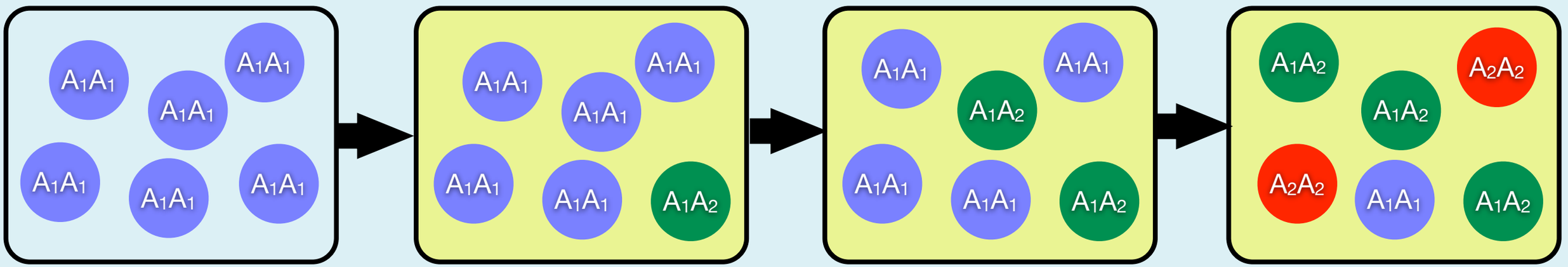
Exposure to lice shampoo actually caused mutations for resistance to the shampoo.

Standing Genetic Variation



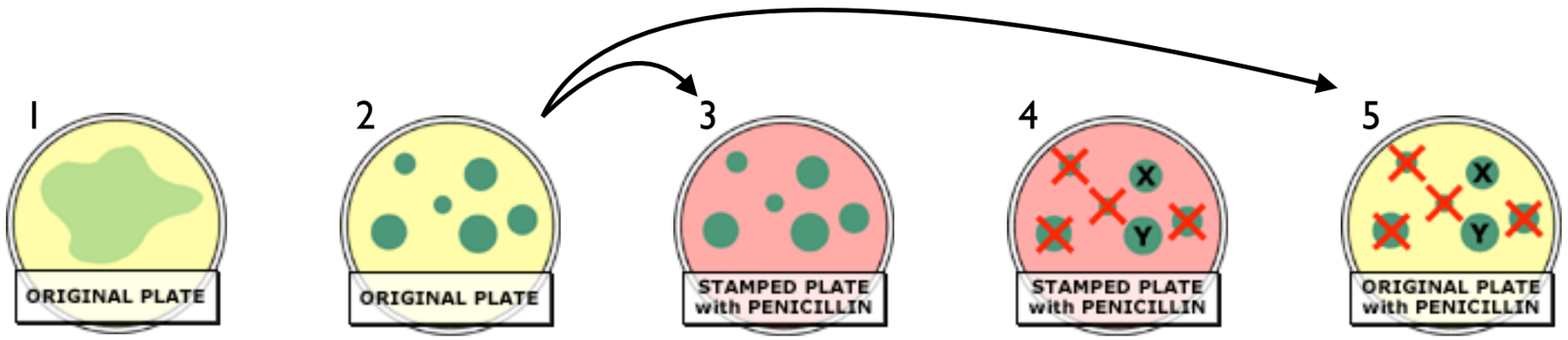
e.g. pesticide resistance in Norway rat, human blood types

New Adaptive Mutants



e.g. resistance to insecticides among mosquitos, human lactase persistence

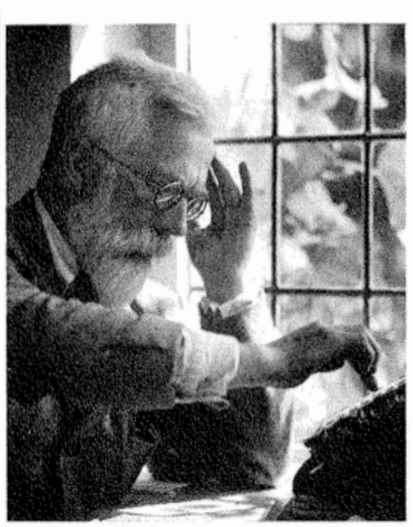
In 1952, Esther and Joshua Lederberg performed an experiment that helped show that many mutations are random, not directed. The hypothesis for the experiment is that antibiotic-resistant strains of bacteria that survive a course of antibiotics had the resistance before they were exposed to the antibiotics, not as a result of the exposure.



1. Bacteria are spread out on a plate, called the "original plate."
2. They are allowed to grow into several different colonies.
3. This layout of colonies is stamped from the original plate onto a new plate that contains the antibiotic penicillin.
4. Colonies X and Y on the stamped plate survive. They must carry a mutation for penicillin resistance.
5. The original plate is washed with penicillin, the same colonies (those in position X and Y) live—even though these colonies on the original plate have never encountered penicillin before.

Conclusion: The penicillin-resistant bacteria were present in the population before they were exposed to penicillin. They did not develop resistance in response to exposure to the antibiotic.

Natural Selection - The **differential survival and reproduction** of classes of organisms that differ from one another in one or more usually **heritable** characteristics. Through this process, the forms of organisms in a population that are **best adapted to their local environment** increase in frequency relative to less well-adapted forms over a number of generations. This difference in survival and reproduction is **not due to chance**.



©Photograph by Antony Barrington Brown. Reproduced with permission of the Fisher Memorial Trust.

R. A. FISHER (1890–1962)

Sir Ronald Fisher is perhaps most widely known for his contributions to statistics (shown here at his desk calculator). Throughout his lifetime, one of his major interests was evolutionary genetics, and his early book *The Genetical Theory of Natural Selection* (Fisher, 1930) was a landmark of synthesis of Darwinian selection and genetics. His contributions to genetics were theoretical (such as the development of concepts of adaptive selection and inbreeding) or statistical (such as the estimation of allele frequencies, selection intensity, or inbreeding coefficients). He also developed the "fundamental theorem of natural selection," part of an effort by him and others to provide a unifying conceptualization for evolution. Fisher was born and lived nearly all of his life in England, where he worked at University College London and then Cambridge University (after retirement, he moved to Australia). He and Sewall Wright had a substantial disagreement, starting in the 1930s, on the factors important in evolution. It is therefore ironic that the general model used in contemporary population genetics is usually termed the Wright–Fisher model (see p. 198). Fisher, for most of his life, was a strong advocate of eugenics. His research papers have been published as a collection (Bennett, 1971–1974), and a biography by his daughter details much of his life and contributions to genetics (Box, 1978; see also Crow, 1990; Edwards, 2003; Ford, 2005.)

Although the name of Charles Darwin is synonymous with natural selection, it was **R. A. Fisher** who did much to demonstrate the power of this process at the genetic level.

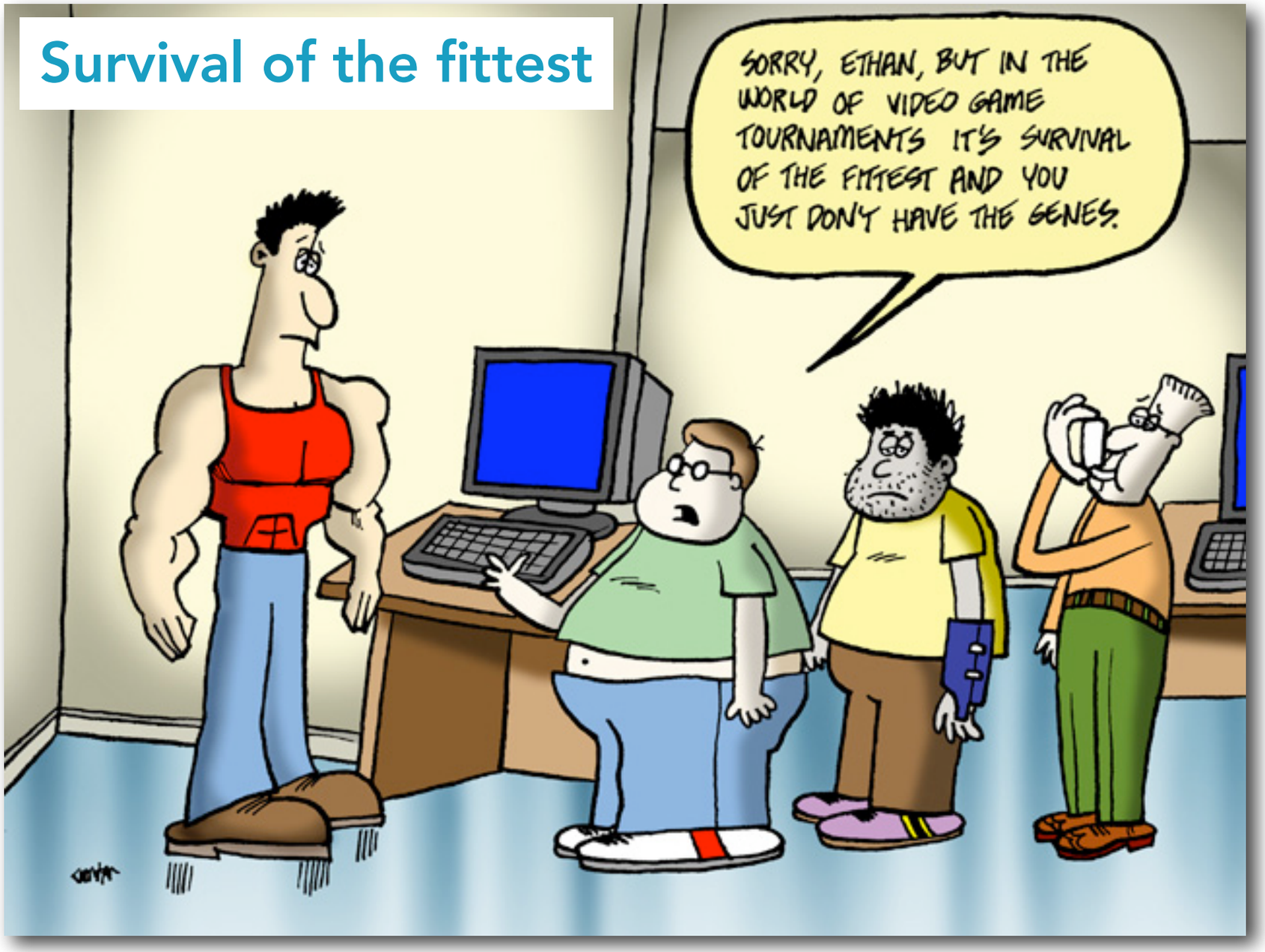
Three conditions must be met for selection to occur in a population:

1. **Variation**: Individuals in the population must differ with respect to the trait in question. Without this variation, all individuals will have the same trait value and cannot be distinguished with respect to that trait.

2. **Heritability**: The variation found in the population must be (at least partially) heritable, i.e. transmitted from parent to offspring. For example, if the variation in the trait were entirely due to the environment, changes in the parent population would not affect the characteristics of the offspring population.

3. **Differential Mortality**: Finally, individuals must have a probability of survival that is a function of the value of the trait in question. If all individuals, regardless of their trait value, had the same probability of survival and fecundity, there would be no predictable change in the population mean.

Heritable variation with fitness consequences.

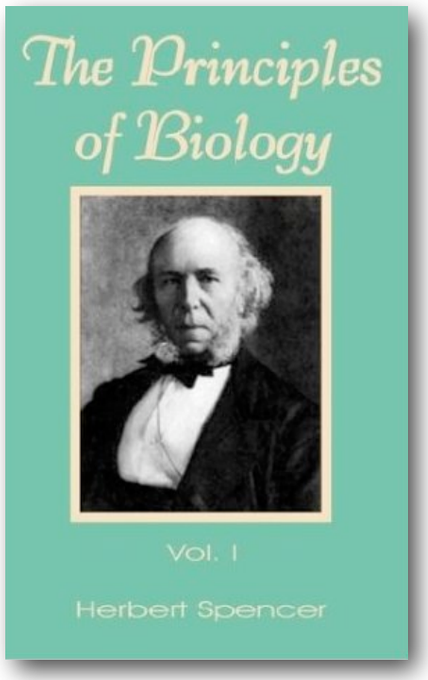

Survival of the fittest
SORRY, ETHAN, BUT IN THE WORLD OF VIDEO GAME TOURNAMENTS IT'S SURVIVAL OF THE FITTEST AND YOU JUST DON'T HAVE THE GENES.

The idea that species adapt and change by natural selection with the best suited mutations becoming dominant - often called “survival of the fittest” is often attributed to Charles Darwin and, although it appears in the fifth edition of his *Origin of Species*, 1869, it is there attributed to **Herbert Spencer**:

> *“The expression often used by Mr. Herbert Spencer of the survival of the fittest is more accurate ... ”*

Spencer had published *The principles of biology* in 1864. In that he referred to “survival of the fittest” twice:

> *“This survival of the fittest, implies multiplication of the fittest.”*
>
> *“This survival of the fittest... is that which Mr. Darwin has called 'natural selection', or the preservation of favored races in the struggle for life.”*



Natural selection is the differential success of genotypes in contributing to the next generation. In the simplest conceptual model, there are two major life history components that bring about selective differences between genotypes: **viability** and **fertility**.

The effect of natural selection on genotypes is measured by fitness. Fitness is the **average number of offspring produced by individuals of a particular genotype**. Fitness can be calculated as the product of viability and fertility, as defined above, and we can define fitness for a di-allelic locus as:

Genotype	Viability	Fertility	Fitness
A_1A_1	V_{11}	f_{11}	$(v_{11})(f_{11})=w_{11}$
A_1A_2	V_{12}	f_{12}	$(v_{12})(f_{12})=w_{12}$
A_2A_2	V_{22}	f_{22}	$(v_{22})(f_{22})=w_{22}$

Genotype	Viability	Fertility	Fitness
A_1A_1	V_{11}	f_{11}	$(v_{11})(f_{11})=w_{11}$
A_1A_2	V_{12}	f_{12}	$(v_{12})(f_{12})=w_{12}$
A_2A_2	V_{22}	f_{22}	$(v_{22})(f_{22})=w_{22}$

Genotype	Viability	Fertility	Absolute Fitness	**Relative Fitness (*w*)**	**Selection (*s*)**
A_1A_1	0.9	3	2.7	**1**	**0**
A_1A_2	0.9	2	1.8	**0.67**	**0.23**
A_2A_2	0.45	2	0.9	**0.33**	**0.67**

The relative contribution of the three genotypes to the next generation is determined by the product of the relative fitness and the frequency before selection of that genotype.

A_1A_1	A_1A_2	A_2A_2
frequency after selection:		
$p^2 \frac{\omega_{11}}{\bar{\omega}}$	$2pq \frac{\omega_{12}}{\bar{\omega}}$	$q^2 \frac{\omega_{22}}{\bar{\omega}}$

The **mean fitness** of the population is the sum of the relative contribution of the different genotypes:

$$\bar{\omega} = p^2\omega_{11} + 2pq\omega_{12} + q^2\omega_{22}$$

ideal population

$$\omega_{11} = \omega_{12} = \omega_{22} = 1$$

$$\Delta p = p_1 - p_0 = p_0 - p_0 = 0$$

$$f(A_1A_1) = \frac{p^2\omega_{11}}{\bar{\omega}} \qquad f(A_1A_2) = \frac{2pq\omega_{12}}{\bar{\omega}} \qquad f(A_1A_1) = \frac{q^2\omega_{22}}{\bar{\omega}}$$

$$p' = \frac{p^2\omega_{11}}{\bar{\omega}} + \frac{1}{2}\left(\frac{2pq\omega_{12}}{\bar{\omega}}\right) = \frac{p^2\omega_{11} + pq\omega_{12}}{\bar{\omega}}$$

$$q' = \frac{1}{2}\left(\frac{2pq\omega_{12}}{\bar{\omega}}\right) + \frac{p^2\omega_{22}}{\bar{\omega}} = \frac{pq\omega_{12} + q^2\omega_{22}}{\bar{\omega}}$$

$$p' = \frac{p^2\omega_{11}}{\bar{\omega}} + \frac{1}{2}\left(\frac{2pq\omega_{12}}{\bar{\omega}}\right) = \frac{p^2\omega_{11} + pq\omega_{12}}{\bar{\omega}}$$

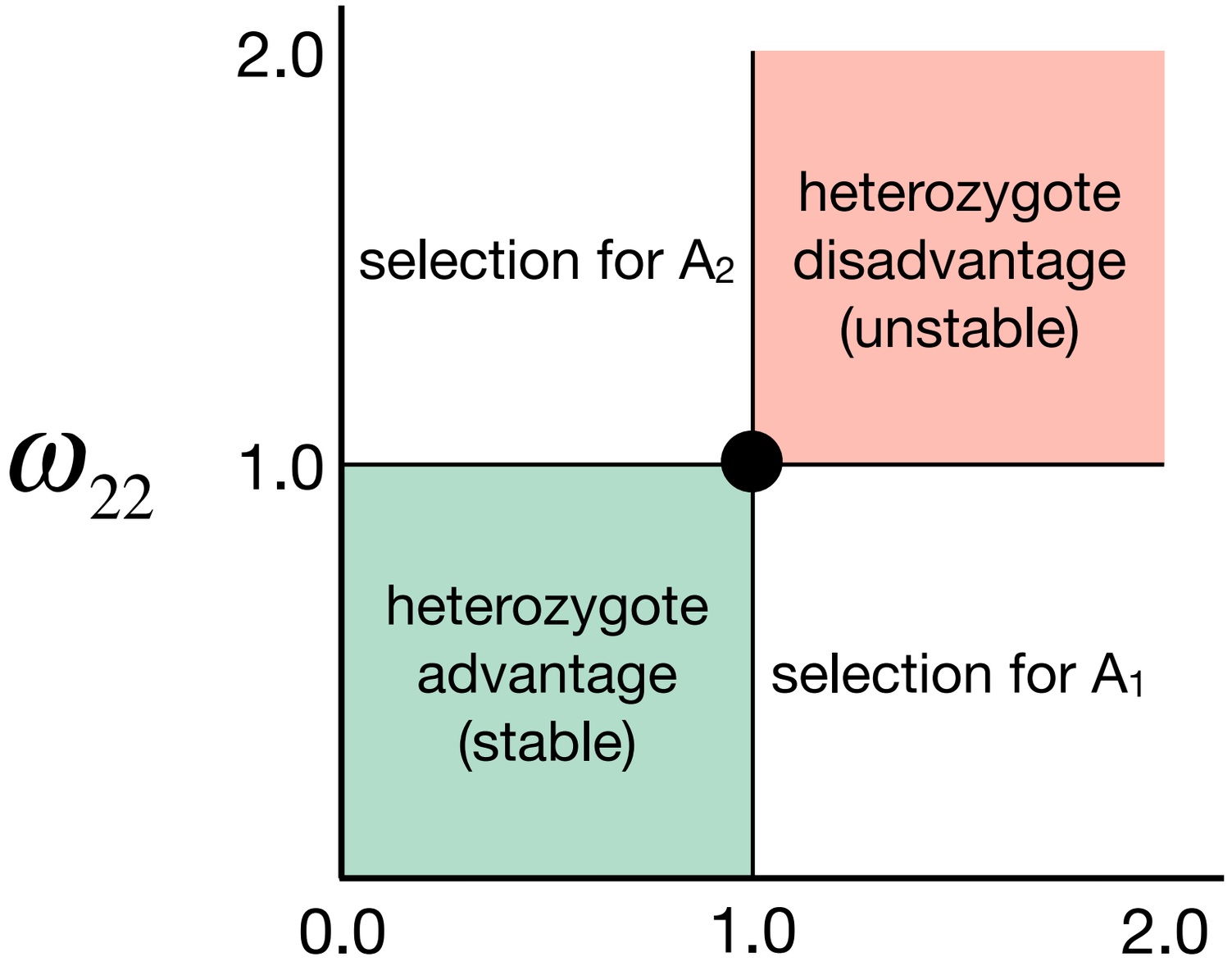
$$\Delta p = p' - p = \frac{pq\left[p(w_{11} - w_{12}) + q(w_{12} - w_{22})\right]}{\bar{\omega}}$$

$$\omega_{11} = 1;\ \omega_{12} = 1;\ \omega_{22} = 1$$

$$q_1 = \frac{p_0 q_0(1) + q_0^2(1)}{\bar{\omega}} = \frac{(1-q_0)q_0 + q_0^2}{\bar{\omega}} = \frac{q_0}{p_0^2(1) + 2\,p_0 q_0(1) + q_0^2(1)} =$$

$$\frac{q_0}{(1-q_0)^2 + 2q_0(1-q_0) + q_0^2} = \frac{q_0}{1 - 2q_0 + q_0^2 + 2q_0 - 2q_0^2 + q_0^2} = q_0$$

$$\Delta q = q_1 - q_0 = q_0 - q_0 = 0$$


2.0
1.0
0.0
1.0
2.0
ω_{22}
ω_{11}
selection for A_2
heterozygote disadvantage (unstable)
heterozygote advantage (stable)
selection for A_1

$$\omega_{12} = 1$$

Heterozygote disadvantage refers to a situation where individuals carrying two different alleles for a particular gene have a lower fitness than individuals who are homozygous for both alleles. This scenario can lead to an unstable balance in the genetic make-up of a population.

Heterozygote disadvantage would result from the expression of deleterious or less favourable traits associated with being heterozygous for that gene. In such cases, the two different alleles may interact in a way that produces an unfavourable phenotype or reduces the fitness of the individual relative to those who are homozygous for one of the alleles.

Heterozygote disadvantage is considered unstable because it can lead to shifts in allele frequencies and potentially drive evolutionary changes within a population due to the reduced fitness of individuals carrying both alleles. This instability can affect the genetic equilibrium and the overall genetic diversity within a population.

Natural selection favours genetic diversity and can lead to phenomena such as overdominance (heterozygote advantage) or balanced polymorphism, where multiple alleles are maintained at relatively stable frequencies in a population.

Heterozygous advantage (overdominance)

$$W_{11} < \mathbf{W_{12}} > W_{22}$$

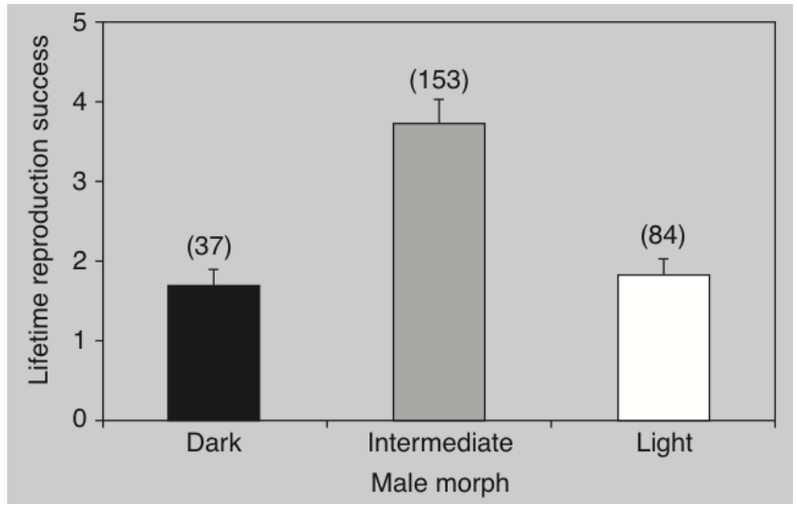
	A_1A_1	A_1A_2	A_2A_2
Fitness	1-s	1	1-s

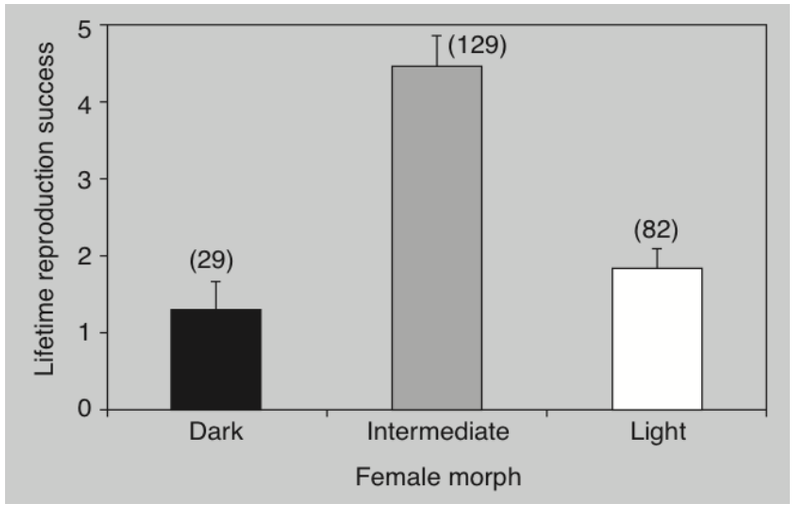
s measures the reduction in fitness compared with the best genotype in the population: for instance, an s of 0.01 means that the genotype has a 1% less chance of survival than the best genotype-it is 99% as fit.

$$s = 1 - \omega$$

ω : relative fitness
s : selection coefficient

European common buzzard (*Buteo buteo*)





Boerner and Krüger (2009)

HLA and HIV-1: Heterozygote Advantage and *B**35-Cw*04* Disadvantage

Mary Carrington,[1] George W. Nelson,[1] Maureen P. Martin,[1]
Teri Kissner,[1] David Vlahov,[2] James J. Goedert,[3]
Richard Kaslow,[4] Susan Buchbinder,[5] Keith Hoots,[6]
Stephen J. O'Brien[7*]

A selective advantage against infectious disease associated with increased heterozygosity at the human major histocompatibility complex [human leukocyte antigen (*HLA*) class I and class II] is believed to play a major role in maintaining the extraordinary allelic diversity of these genes. Maximum *HLA* heterozygosity of class I loci (*A*, *B*, and *C*) delayed acquired immunodeficiency syndrome (AIDS) onset among patients infected with human immunodeficiency virus–type 1 (HIV-1), whereas individuals who were homozygous for one or more loci progressed rapidly to AIDS and death. The *HLA* class I alleles *B*35* and *Cw*04* were consistently associated with rapid development of AIDS-defining conditions in Caucasians. The extended survival of 28 to 40 percent of HIV-1–infected Caucasian patients who avoided AIDS for ten or more years can be attributed to their being fully heterozygous at *HLA* class I loci, to their lacking the AIDS-associated alleles *B*35* and *Cw*04*, or to both.

Heterozygous disadvantage (underdominance)

$$\mathbf{w_{11}} > w_{12} < \mathbf{w_{22}}$$

A_1A_1	A_1A_2	A_2A_2
Fitness	1	1-s	1

$s = 1 - \omega$

ω : relative fitness
s : selection coefficient

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PLoS COMPUTATIONAL BIOLOGY

Stability Properties of Underdominance in Finite Subdivided Populations

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Abstract

In isolated populations underdominance leads to bistable evolutionary dynamics: below a certain mutant allele frequency the wildtype succeeds. Above this point, the potentially underdominant mutant allele fixes. In subdivided populations with gene flow there can be stable states with coexistence of wildtypes and mutants: polymorphism can be maintained because of a migration-selection equilibrium, i.e., selection against rare recent immigrant alleles that tend to be heterozygous. We focus on the stochastic evolutionary dynamics of systems where demographic fluctuations in the coupled populations are the main source of internal noise. We discuss the influence of fitness, migration rate, and the relative sizes of two interacting populations on the mean extinction times of a group of potentially underdominant mutant alleles. We classify realistic initial conditions according to their impact on the stochastic extinction process. Even in small populations, where demographic fluctuations are large, stability properties predicted from deterministic dynamics show remarkable robustness. Fixation of the mutant allele becomes unlikely but the time to its extinction can be long.

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Biased sex-ratio and sex-biased heterozygote disadvantage affect the maintenance of a genetic polymorphism and the properties of hybrid zones

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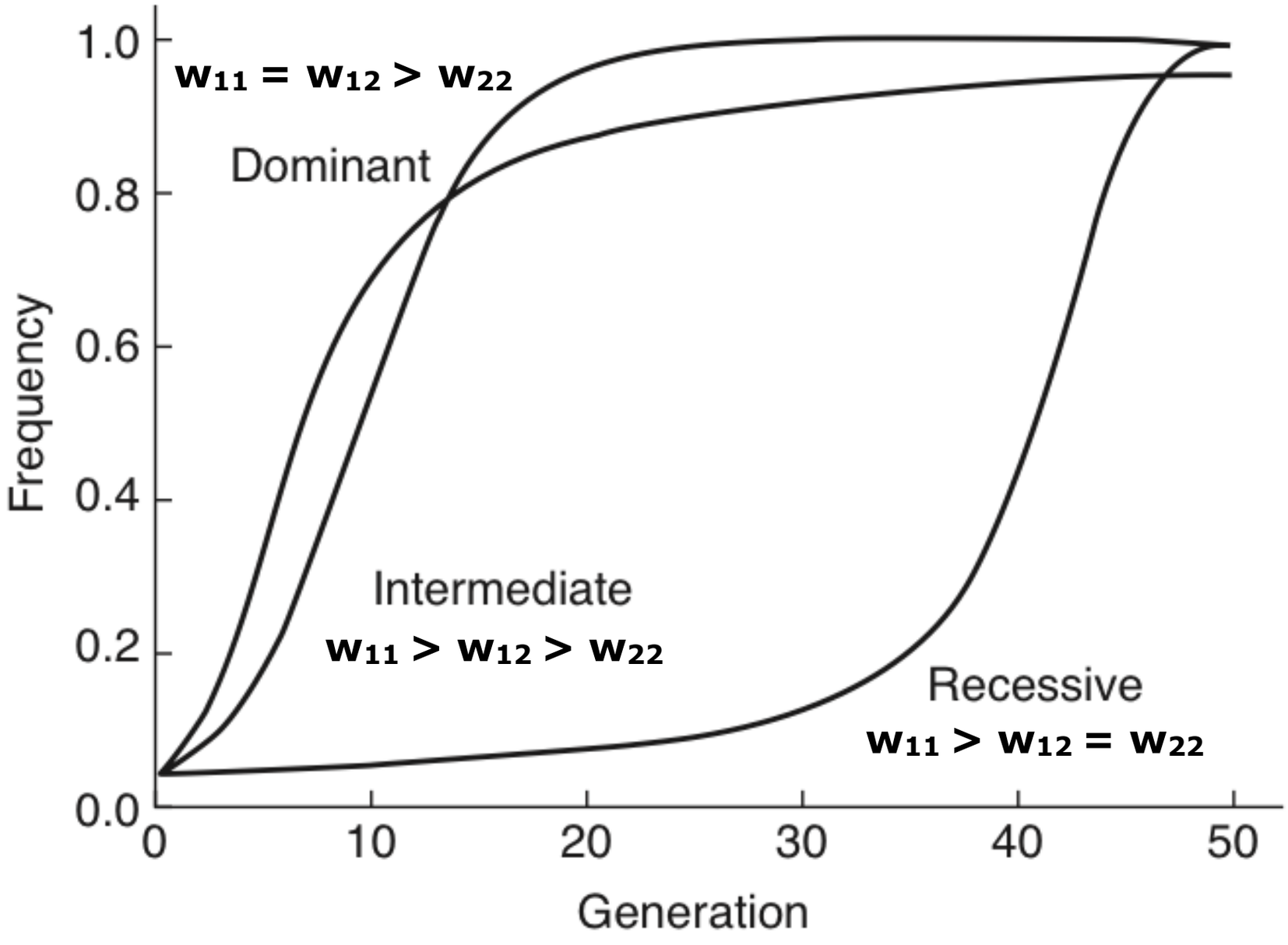
Abstract

The evolution of biodiversity is a major issue of modern biology, and it is becoming increasingly topical as the ongoing erosion of diversity puts serious threats on human well-being. An elementary mechanism that allows maintaining diversity is the interplay between dispersal and heterozygote selective disadvantage, which can lead to self-sustainable spatial genetic structures and is central to the stability of hybrid zones. Theoretical studies supporting the importance of this mechanism assume a balanced sex-ratio and a heterozygote disadvantage equally affecting both sexes, despite the multiplicity of empirical evidence that (i) adult sex-ratio is usually biased towards either male or female and that (ii) heterozygote disadvantage often affects a single sex. We expanded the existing theory by weighting the strength of selection against heterozygote according to the biased in sex-ratio and in heterozygote disadvantage. The range of conditions allowing for the maintenance of polymorphism can then either double or vanish. We discuss the implications of such finding for birds, mammals and insects diversity. Finally, we provide simple analytical predictions about the effect of those biased on the width and speed of hybrid zones and on the time for the spread of beneficial mutations through such zones.

Dominant:	$\mathbf{w_{11} = w_{12}} > w_{22}$
Intermediate:	$\mathbf{w_{11}} > w_{12} > w_{22}$
Recessive:	$\mathbf{w_{11}} > w_{12} = w_{22}$
Overdominance:	$w_{11} < \mathbf{w_{12}} > w_{22}$
Underdominance:	$\mathbf{w_{11}} > w_{12} < \mathbf{w_{22}}$

MORE
THINGS
CONSIDERED



Change in allele frequency under directional selection when the homozygote for the favored allele has twice the fitness of the homozygote for the unfavored allele (1.00 vs. 0.50). The heterozygote can have the same fitness as the favored allele (1.00, dominant), the same fitness as the unfavored allele (0.50, recessive), or has intermediate fitness (0.75). The initial frequency of the favored allele is 0.03.

A_1A_1	A_1A_2	A_2A_2	Fitness
1	1	1	neutral
1	1	0	recessive lethal (complete dominance)
1	1	1-*s*	recessive (lethal-neutral)
1	1-*s*/2	1-*s*	additive
1	1-*hs*	1-*s*	purifying selection
1+*s*	1+*hs*	1	positive selection
1-s_1	1	1-s_2	heterozygot advantage (overdominance)
1+s_1	1	1+s_2	heterozygot disadvantage (underdominance)

h: level of dominance, hs:amount of selection against the heterozygote

$$\omega_{11} = 1;\ \omega_{12} = 1;\ \omega_{22} = 0$$

$$q_1 = \frac{q_0}{1+q_0} \Rightarrow q_2 = \frac{q_1}{1+q_1} = \frac{\frac{q_0}{1+q_0}}{1+\frac{q_0}{1+q_0}} = \frac{q_0\left(\frac{1}{1+q_0}\right)}{1+2q_0\left(\frac{1}{1+q_0}\right)} = \frac{q_0}{1+2q_0}$$

$$q_t = \frac{q_0}{1+tq_0}$$

the mean fitness of a population:

$$\bar{\omega} = p^2\omega_{11} + 2pq\omega_{12} + q^2\omega_{22}$$

genotype frequency after selection:

$$f(A_1A_1) = \frac{p^2\omega_{11}}{\bar{\omega}} \qquad f(A_1A_2) = \frac{2pq\omega_{12}}{\bar{\omega}} \qquad f(A_2A_2) = \frac{q^2\omega_{22}}{\bar{\omega}}$$

allele frequency after selection:

$$q' = \frac{1}{2}\left(\frac{2pq\omega_{12}}{\bar{\omega}}\right) + \frac{q^2\omega_{22}}{\bar{\omega}} = \frac{pq\omega_{12} + q^2\omega_{22}}{\bar{\omega}}$$

allele frequency change:

$$\Delta q = q' - q = \frac{pq\left[q(\omega_{22} - \omega_{12}) - p(\omega_{11} - \omega_{12})\right]}{\bar{\omega}}$$

Balancing selection refers to a number of selective processes by which multiple alleles are actively maintained in the gene pool of a population at frequencies above that of gene mutation. There are several mechanisms by which balancing selection works to maintain polymorphism. The two major and most studied are **heterozygote advantage** and **frequency-dependent selection**.

Heterozygote advantage - A situation in which a single disadvantageous allele is not selected out of a population, because, when a individual is heterozygous for that allele, it gains some sort of local advantage by having the disadvantageous allele. For example, the allele for **sickle-cell anemia** offers "resistance" to malaria. If a person in an area high in malaria is heterozygous for sickle-cell anemia, the "resistance" they gain to malaria outweighs the disadvantage of having heterozygous sickle-cell anemia. A person homozygote for the mutant allele will not have a greater advantage, even if they are completely resistant to malaria. What's the use of being resistant to malaria if you're blood can't carry oxygen?

Frequency-dependent selection occurs when the fitness of a trait depends on its frequency in a population. It is possible for the fitness of a genotype to increase (**positively frequency-dependent**) or decrease (**negatively frequency-dependent**) as the genotype frequency in the population increases. Positive frequency dependent selection occurs when a trait has higher fitness when it is common than when it is rare. Negative frequency dependent selection occurs when a trait has higher fitness when it is rare than when it is common. Negative frequency dependent selection will turn out to have some important implications for evolution of other traits.

• Natural selection may favor non-poisonous butterflies that have the same color pattern as poisonous butterflies. This system is called Batesian mimicry. When they are rare, birds will tend to avoid the mimics, because they will have already have encountered a poisonous butterfly of the same appearance. But when the non-poisonous type is common, the previous encounters of birds with butterflies of their appearance are more likely to have been rewarding; the birds will not avoid eating them, and their fitness will be lower. The fitness of the mimics is **negatively frequency-dependent**.

• In other butterflies, such as in central and south American Heliconius, there are several morphs within a species, each morph having a different color pattern. All the morphs are poisonous. When a morph is common, it will be more likely that birds will have already learned to avoid them, whereas birds will not yet have learned to avoid a rare morph. An individual of a rare morph is therefore more likely to be the unlucky prey that educates the bird, and gets killed in the process. The fitness of each morph is **positively frequency-dependent**.

But with negatively frequency-dependent fitnesses (as in Batesian mimicry), it is possible for natural selection to maintain a polymorphism. When a genotype is rare, it is relatively favored by selection and it will increase in frequency; as it becomes more common, its fitness decreases and there may come a point at which it is no longer favored. At that point, the fitnesses of the different genotypes are equal and natural selection will not alter their frequencies: they are at **equilibrium**.

A different example of negative frequency dependent selection occurs in fruit flies, the flies that have been commonly studied by geneticists, and is called the "**rare male advantage**". Female fruit flies in a population prefer to mate with a male with an unusual phenotype. Suppose for example that most individuals in the population have red eyes, but a few have white eyes. White eyed males will attract more females. This is not just because females like white eyes. If most males in the population have white eyes but a few have red eyes, females will mate preferentially with the red-eyed males.

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The Mating Advantage of Rare Males in *Drosophila*

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Communicated by Theodosius Dobzhansky, November 20, 1969

Abstract. The mating advantage of rare *Drosophila* males is tested using two eye color mutants. In one experiment, the flies remained for three hours in observation chambers containing 25 pairs; in another experiment they stayed for 24 hours in mass cultures of 200 individuals. The outcome of this latter experiment was followed for ten generations, with all competition other than that for mates eliminated. For initial frequencies of 80 per cent for the common and 20 per cent for the rare type, the frequencies converged to approximate equality because the rare males were favored as mates. When the formerly rare type increases in frequency, it loses its mating advantage, and a balanced equilibrium is eventually attained.

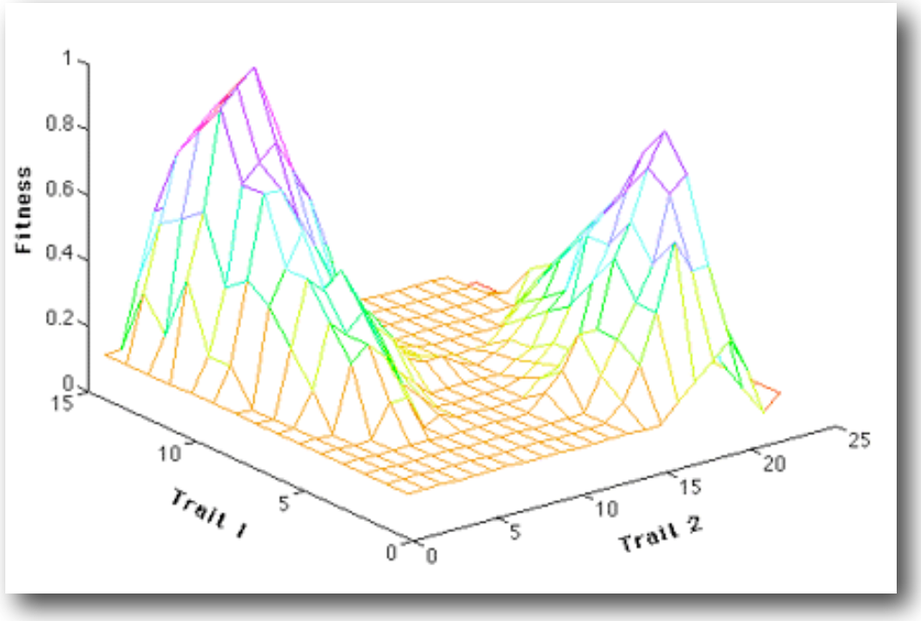
Genotype	A_1A_1	A_1A_2	A_2A_2	A_1A_3	A_2A_3	A_3A_3
Fitness	w_{11}	w_{12}	w_{22}	w_{13}	w_{23}	w_{33}
Frequency	p^2	$2pq$	q^2	$2pr$	$2qr$	r^2

$$p' = \frac{(p^2\omega_{11} + pq\omega_{12} + pr\omega_{13})}{\bar{\omega}}$$

$$q' = \frac{(q^2\omega_{22} + pq\omega_{12} + qr\omega_{23})}{\bar{\omega}}$$

$$r' = \frac{(r^2\omega_{33} + pr\omega_{13} + qr\omega_{23})}{\bar{\omega}}$$

In a population, there may be just one coadapted gene complex, or there might be several different combinations of traits, each of which could have high fitness. This latter possibility gives rise to another concept: that of an adaptive landscape. An adaptive landscape is the description of the fitnesses of all possible combinations of different traits in a population. Adaptive landscapes are frequently represented graphically; fitness is plotted on a vertical axis and trait values for different genes are plotted on other axes. Combinations of traits that have high fitness thus appears as peaks, and combinations that have low fitness appear as valleys. Here is an example of an adaptive landscape:



This example shows two traits, and a situation in which there are two combinations of traits, the peaks in the graph, shown in purple, that have high fitness, while other combinations of the traits have low fitness. These peaks in the adaptive landscape can be called adaptive peaks; note that they are also combinations of different genetic traits that, together, have high fitness, so they are coadapted gene complexes. An adaptive peak and a coadaptive gene complex are thus basically the same thing.

genotype	A_1A_1	A_1A_2	A_2A_2	$\overline{\omega}$	q_1	Δq
recessive lethal	1	1	0	1	$\frac{q_0}{1+q_0}$	$-\frac{q_0^2}{1+q_0}$
complete dominance	1	1	1-*s*	$1-sq_0^2$	$\frac{q_0(1-sq_0)}{1-sq_0^2}$	$\frac{sq_0^2(1-q_0)}{1-sq_0^2}$
additive effect	1	1-*s*/2	1-*s*	$1-sq_0$	$\frac{q_0[2-s(1+q_0)]}{2(1-sq_0)}$	$\frac{sq_0(q_0-1)}{2(1-sq_0)}$
purifying selection	1	1-*hs*	1-*s*	$1-sq_0^2-2q_0hs(1-q_0)$	$\frac{q_0(1-hs+q_0hs-sq_0)}{1-sq_0^2-2q_0hs(1-q_0)}$	$\frac{q_0s(1-q_0)(2q_0h-q_0-h)}{1-sq_0^2-2q_0hs(1-q_0)}$
positive selection	1+*s*	1+*hs*	1	$1+s(1-q_0)(1+2hq_0)$	$\frac{q_0(1+hs(1-q_0))}{1+s(1-q_0)(1-q_0+2hq_0)}$	$\frac{sq_0(1-q_0)(h-1+q_0-2hq_0)}{1+s(1-q_0)(1-q_0+2hq_0)}$
heterozygote advantage	1-s_1	1	1-s_2	$1-s_1+2s_1q_0-q_0^2(s_1+s_2)$	$\frac{q_0-s_2q_0^2}{1-s_1p_0^2-s_2q_0^2}$	$\frac{p_0q_0(s_1p_0-s_2q_0)}{1-s_1p_o^2-s_2q_0^2}$
heterozygote disadvantage	1+s_1	1	1+s_2	$1+s_1-2s_1q_0+q_0^2(s_1+s_2)$	$\frac{q_0+s_2q_0^2}{1+s_1p_0^2+s_2q_0^2}$	$\frac{p_0q_0(s_2q_0-s_1p_0)}{1+s_1p_o^2+s_2q_0^2}$

A_1A_1	A_1A_2	A_2A_2
1	1	0

The number of generation (t) needed to reduce the allele frequency from an initial value of q_0 to q_t for a **recessive lethal**.

q_0	q_t	t
0.5	0.25	2
	0.1	**8**
	0.001	98
0.1	0.05	10
	0.01	90
	0.001	**990**
0.01	0.005	100
	0.001	900
	0.0001	9900

A_1A_1	A_1A_2	A_2A_2
1	1-0.5s	1-s

The number of generation (t) needed to reduce the allele frequency from an initial value of q_0 to q_t for **additivity when s=0.1**.

q_0	q_t	t
0.9	0.5	44
	0.1	89
	0.01	136
0.5	0.25	22
	0.1	**44**
	0.01	92
0.1	0.05	15
	0.01	48
	0.001	**194**

Sexual selection is a "special case" of natural selection. Sexual selection acts on an organism's ability to obtain (often by any means necessary!) or successfully copulate with a mate.

Selection makes many organisms go to extreme lengths for sex: peacocks maintain elaborate tails, elephant seals fight over territories, fruit flies perform dances, and some species deliver persuasive gifts.

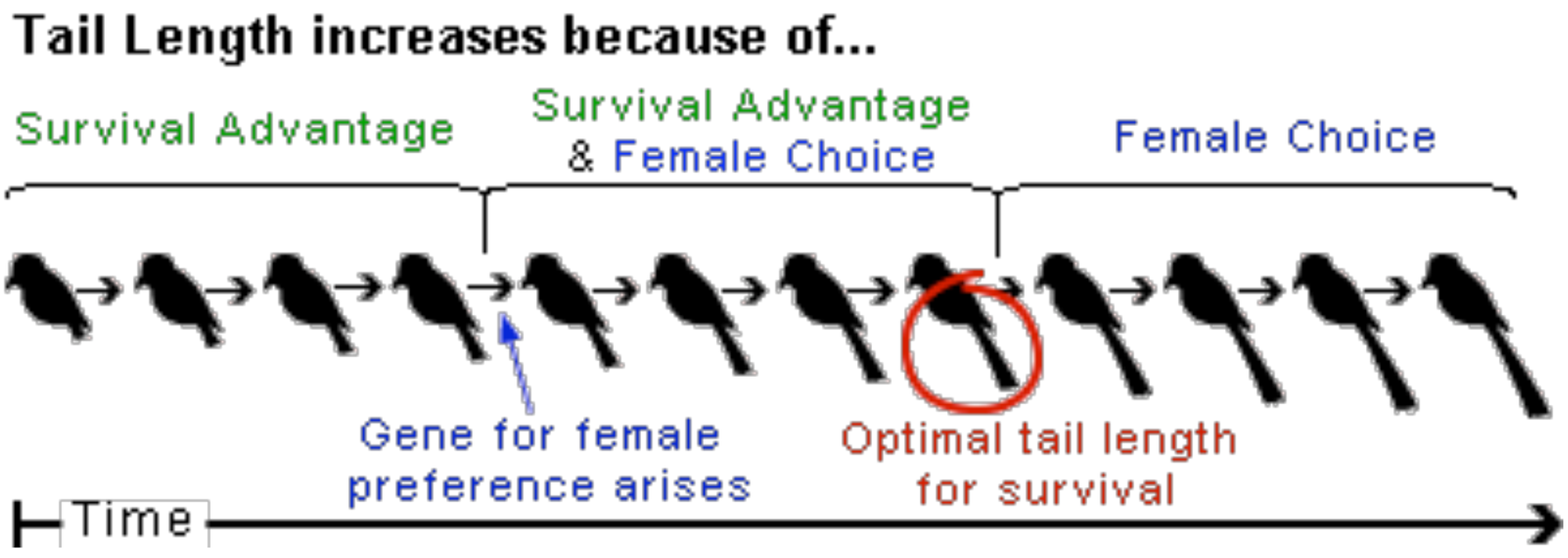
Sexual selection is often powerful enough to produce features that are harmful to the individual's survival. For example, extravagant and colorful tail feathers or fins are likely to attract predators as well as interested members of the opposite sex.

How did female choice for traits like a long, colorful tail evolve? After all, if a female chooses a male with a long, awkward tail, her sons will probably have a similar tail—and that tail might hurt their chances of survival by attracting predators. How could natural selection act to produce a preference for a disadvantageous trait?

It makes sense for a female to choose a mate based on **traits that help to survive**. For example, a female bird would do well to choose a strong-looking, disease-free mate. That male likely carries "good" genes that allow him to resist disease and get sufficient food—and he will pass those genes on to his offspring.

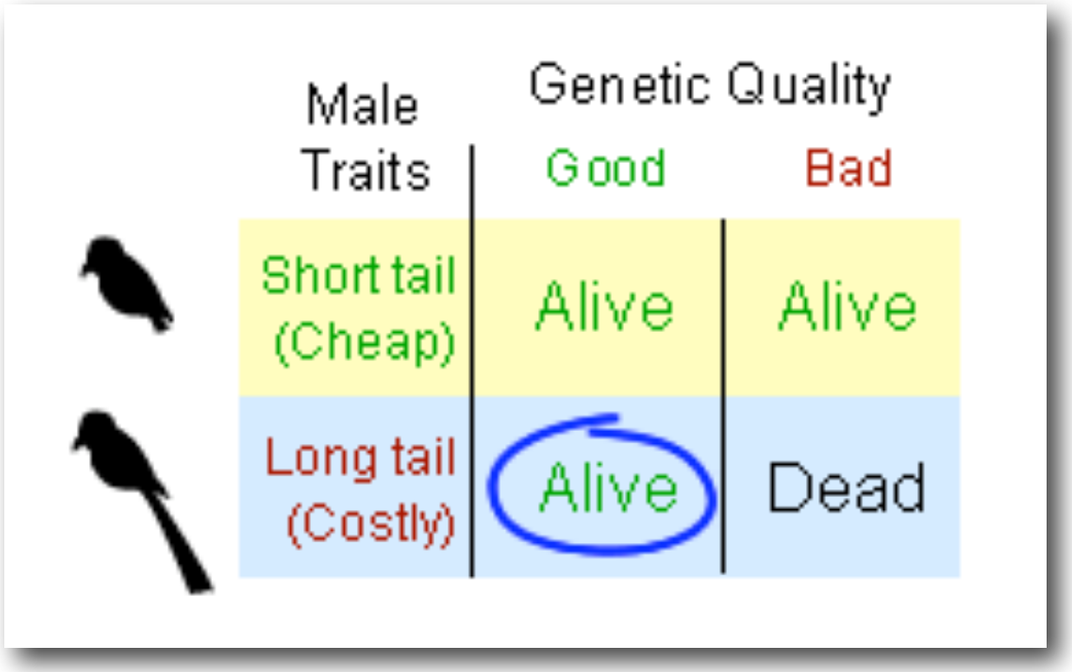
However, there are **many examples of females choosing mates based on less useful traits** (e.g. song complexity) or even traits detrimental to survival (e.g., brightly colored plumage). These cases present evolutionary biologists with a bit of a puzzle. How did these preferences arise in the first place? If a female chooses a male with bright feathers, her sons will have bright feathers, which are likely to attract predators. A gene for choosing brightly colored males would seem to be disadvantageous. How do such genes spread through a population?

Imagine a bird population in which females choose mates at random. Males with slightly longer tails fly a little more adeptly, avoid predation, and so, survive better than males with slightly shorter tails. In this situation, a gene for female choosiness (longer tail = sexier) will be favored, since—by choosing a long-tailed male—she will have sons with longer tails. This trait will spread through the population until most males have long tails and most females prefer long-tailed mates. So far so good.



However, once this has happened, the process may run out of control, until the male trait becomes so exaggerated that it is disadvantageous. In other words, female preference, instead of survival advantage, may begin to drive the evolution of ever-longer tails, until males are encumbered by showy plumage that no longer helps them avoid predation.

Imagine another bird population in which females choose mates at random. Some males in the population have better genes for survival than others, but it is difficult to tell whether a male has good genes or not. In this scenario, long tails make it more difficult to survive—they are costly to produce and maintain. Because they are so costly, only males with good genes have the extra resources to produce them. In this situation, a long tail is an indicator of good genes. A gene for female choosiness (longer tail = sexier) will be favored, since —by choosing a long-tailed/good gene male—she will have sons with good genes. This trait will spread through the population until most females choose long-tailed mates and males that are able to produce long tails are favored.



Behavior can also be shaped by natural selection. Behaviors such as birds' mating rituals (see Figure), bees' wiggle dance, and humans' capacity to learn language also have genetic components and are subject to natural selection.

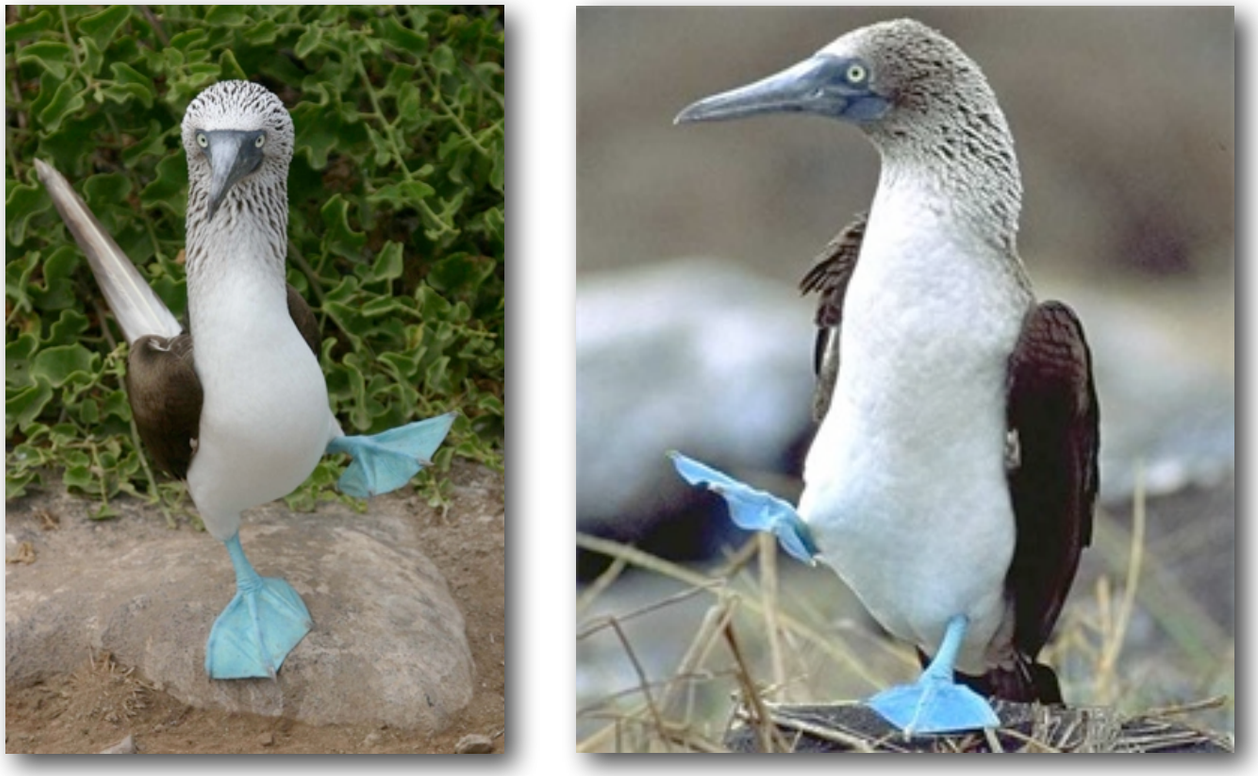
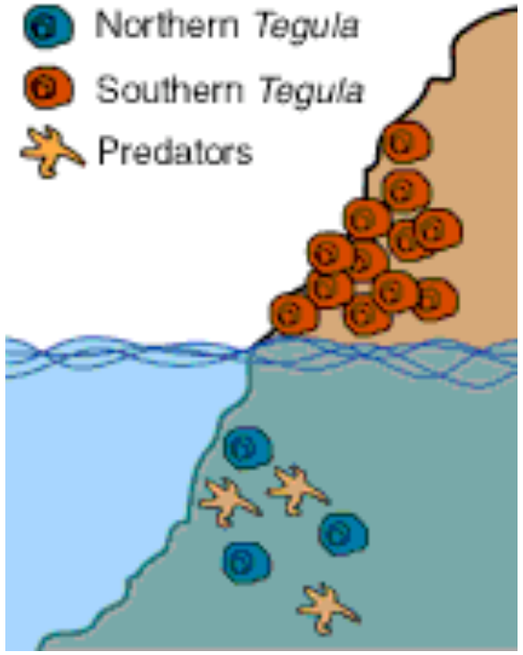


Figure: The male blue-footed booby (*Sula nebouxii*) exaggerates his foot movements to attract a mate.

In some cases, we can directly observe natural selection. Very convincing data show that the shape of finches' beaks on the Galapagos Islands has tracked weather patterns: after droughts, the finch population has deeper, stronger beaks that let them eat tougher seeds.



For example, on rocky shores, animals have ranges that form clear spatial patterns. Some species live only in deep water, and some only live much higher up the shore. A snail common on California shores (*Tegula funebralis*) can be found in both ranges. In Southern California, Tegula live high up on the shore, while in Northern California, they live in deeper water.

Hypothesis:

Fawcett et al. (1984) found that predators, such as octopi, starfish, and crabs, were more abundant in southern California than in northern California. Perhaps intense predation in the south selected for snails that lived higher up the shore, out of reach of many predators. In the north, selection might not have been as strong, so the snails were not selected to live high on the shore.

Experiment:

The hypothesis was tested by transplanting snails (reciprocal transplant experiments). Northern and southern snails were released in deep water and were watched. If predators were around, all the snails high-tailed it towards higher ground (snails can probably sense the chemicals exuded by predators). But southern snails moved further up the shore faster than northern snails. Because the northern snails were slower and didn't move high enough, they were more likely to be eaten by predators.

What did this experiment show?

1. There is an innate difference between southern and northern snails (i.e., some difference that is not merely a function of being on a southern or northern shore). This difference is probably genetic (but we would need to do more experiments to be absolutely sure).

2. This difference can lead to differential survival. If predation is intense, snails that move higher faster are more likely to survive.