

# Summary of Introduction to Evolutionary Biology

v0.9

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## Preface

This document aims to summarize the lecture Introduction to Evolutionary Biology as it was taught in the autumn semester of 2017. The focus lies on concepts and examples from the lecture are only included when deemed necessary. Unfortunately I can't guarantee that it is complete and free of errors. You can contact me under **glebert@student.ethz.ch** if you have any suggestions for improvement. The newest version of this summary can always be found here: <https://n.ethz.ch/~glebert/>

# 1 Introduction

**Definition:** Evolution means biological change over time  
**Technical basis:** Phenotypes of individuals that are encoded by heritable genotypes vary in a population and their frequencies change

## 1.1 History

**Aristotle:** Ladder of nature / perfection  
**Carl von Linné:** Systematic classification of life  
**James Hutton & Charles Lyell:** Gradual long-term processes shaped earth (Uniformitarianism)  
**Jean-Baptiste de Lamarck:** Inheritance of acquired characteristics (Lamarckian evolution)  
**Charles Darwin:** Evolution is descent with modification and results in survival of the fittest

## 1.2 Microevolution

direct observation: small time-scales → short-term changes

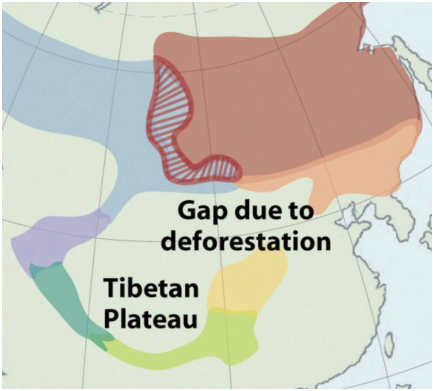
Evidence of Microevolution:

- 1) Observation from natural populations
  - Bacterial adaptation to antibiotic stress
  - Soapberry bug adaptation to fruit
- 2) Observation from living anatomy
  - Vestigial and rudimentary traits
    - Kiwi wings
    - Human coccyx (Steissbein)
    - Human arrector pili muscle (Haaraufrichter-Muskel)
    - (Appendix might be safe house for good gut bacteria)

## 1.3 Speciation

Speciation is the process that results in one species splitting into two or more. One example for this are ring species. They occur when one species spreads slowly around a geographical area to which they don't spread.

By the time they meet up again the differences between the populations are too big to interbreed. One example for this is the Siberian Greenish Warbler (the hatched area is where no interbreeding occurs with two populations present).



## 1.4 Macroevolution

indirect observation: long time-scales → long-term changes

Evidence of Macroevolution:

- 1) Successions & Extinctions
  - Law of succession: pattern of correspondence between fossil and recent forms from the same locale
  - Comparative anatomy: Georges Cuvier argued that certain species are extinct. Recent macrofauna is only a fraction of all that ever existed
- 2) Transitional forms
  - Darwinian evolution predicts intermediate forms between a species and its ancestor (e.g. Microraptor gui and Archaeopteryx between dinosaurs and modern birds)
- 3) Homologies (Owen: “the same organ in different animals under every variety of form and function”) can be found through comparative anatomy and comparative embryology. The similarity is due to inheritance from a common ancestor. They are phenotypically and genetically defined and enable the use of model organisms. Some molecular homologies are

- the universal genetic code: bases and codons
- the small-subunit (SSU) ribosomal RNA genes

# 2 Natural selection

Natural selection is the process underlying adaptive evolution

## 2.1 Darwins postulates of evolutionary change

Evolutionary change over time is a deductive implication of four postulates.

- 1) All populations contain variable individuals
- 2) Variation among individuals is, at least in part, heritable
- 3) Some individuals are more successful at surviving and reproducing than others
- 4) Survival and reproduction of individuals are not random; but individuals with the most favorable variation given the environment are those better at surviving.

These postulates can be tested in real world populations (e.g. Darwin's finches from the Galapagos islands). One has to be careful to not misinterpret biasing factors. For example heritability measures can be skewed by misidentified paternity, misidentified maternity, food quality or maternal effects such as egg quality.

## 2.2 Definition

Natural selection acts on individuals (more specifically, phenotypes), its consequences occur in populations as allele frequency changes.

2.3 Darwinian vs. Lamarckian evolution

Different processes proposed for the same pattern.

1.	No initial variation	Initial variation
2.	Individuals adapt during their lifetime	Selection acts on individuals
3.	Inheritance of aquired changes/characters (IAC)	Inheritance of surviving alleles if environment leads to adaptation
	⇒ individuals and populations evolve	⇒ populations evolve

Epigenetic modifications are attached to the genetic code and can be passed on to the offspring (up to two generations). Thus their evolutionary relevance is short-term.

2.4 Limits

- Natural selection acts on existing traits
- Natural adaptation does not lead to perfection
- Natural selection is non-random, but it is not progressive
- Natural selection is blind to the future, but tells us tales from the past

2.5 “Perfection” in nature

William Paley argued, that the vertebrate eye is too perfect and complicated to have resulted from natural processes. Thus it must be a creation of a conscious designer. By looking closely at eyes from various chordates one can see that there is a lot of variation in the complexity of the eye. Thus Paleys argument is wrong.

3 Phylogenetics

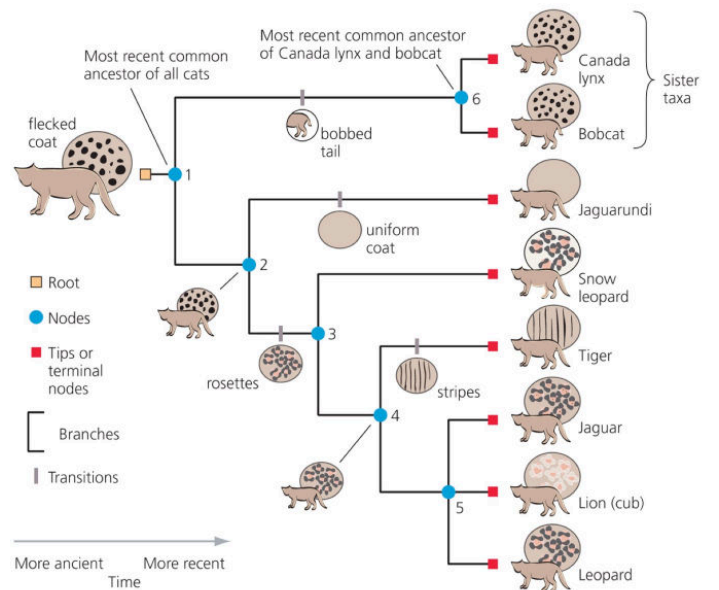
3.1 Evolutionary trees

Evolutionary trees aim to display the course of speciation over time and the relation between species. They start with one common ancestor (the root) and end with multiple species (tips or terminal nodes). Dichotomies (splits of branches into two) in between are called nodes. If phylogeny can't be resolved to dichotomies branches

split into three or more subbranches. This phenomenon is called polytomy. Branches can have transitions where a specific characteristic evolved. The first group or species to split of is called basal.

Trees come in many forms depending on the author and on the kind of data they aim to display. They can be left-right, top-down or even circular.

One should not forget that evolutionary trees are merely hypotheses.



3.2 Phylogenetic inference

- Plesiomorphy: A characteristic that is shared between a species and its ancestor
- Apomorphy: A characteristic that is different from an ancestor
- Synapomorphy: Apomorphic and shared between multiple sister taxa. Also called homology
- Autapomorphy: Apomorphic and different from sister taxa
- Monophyletic group: All descendants of one ancestor (at least two taxa). Also called clade.
- Paraphyletic group: A subset of descendants of one ancestor

Classical taxonomic groups are not necessarily monophyletic (e.g. prokaryotes, dicots and fish).

3.3 Tree reconstruction

Pitfalls of trees inferred form phenotypes alone:

- Phenotypes are influenced by genotypes and environment
- Only genotypes are heritable
- Phenotypic similarity due to convergence (analogy / homoplasy; e.g. camera eye in mollusks and teleosts)

Combination of phenotypic and genotypic data (molecular markers) leads to better results. When using molecular markers each nucleotide is seen as an independent character.

If multiple possible trees exist, one can assume that the most parsimonious (least evolutionary steps) is the correct one. If multiple equally parsimonious trees turn up one estimates uncertainty. This is done by bootstrapping which is the generation of data sets made up from the original data set. Data points can be repeated multiple times or be absent altogether. When analyzing these data sets one gets a phylogenetic tree for each. The most likely tree will be the one that comes out the most after the analysis. The certainty of a certain clade is placed at its node and given in percent. This number is the percentage of the replicates in which that particular clade appeared and is also called the bootstrap support of the clade. High bootstrap support means that the clade is a winner across our artificial data set.

Reversals or “back-mutations” can remove synapomorphies.

3.4 Answering questions with phylogeny

The following examples illustrate some of the problems that phylogeny can help to solve.

- By looking at when body lice evolved from hair

like one can estimate when humans started wearing clothes (around 107,000 years ago).

- Forensic scientists were able to conclude which patients got HIV from their dentist and which got it else where.
- In his E. coli long-term experimental evolution project (LTEE) Richard Lenski was able to show that the mutation rate in his bacteria evolved.

## 4 Science history

### 4.1 Ancient Greece

- **Hippocrates**: Importance of **good notes and categorization**
- **Socrates**: Dialectic inquiry (finding an answer by initiating a group discussion with questions)
- **Plato**: Ideal spiritual forms of real world things
- **Aristotle**: Emphasis on empirical things (**Scala Natura**)

### 4.2 Deduction and Induction

**Induction** infers generalizations base on individual instances. Reasoning in which the premises of an argument are considered to support its conclusion but do not guarantee its truth.

**Deduction** is the process of deriving consequences of what is assumed. Given the truth of the assumptions, a valid deduction guarantees the truth of the conclusion.

### 4.3 Aristotle

- pupil of Plato
- **big influence** on future course of **western science** through founding of fields of logic, biology and psychology
- generated one of the most impressive systems of thought
- heavy emphasis on rules of **deductive logic**
- lack of rigorous experimental methods
- four causes
  - 1) Material cause

- 2) Formal cause
- 3) **Efficient cause**: like modern understanding of cause-effect relation
- 4) **Final cause**: the purpose for which a thing exists of an action is done. The future end something is supposed to serve

## 5 Co-option

**Co-option** (or exaptation) is the process by which a structure or system with an original function adds or changes to a new function.

### 5.1 Myxococcus xanthus

*Myxococcus xanthus* is a social soil bacteria. It can secrete antibiotics and lytic enzymes to **externally digest other microbes**. These bacteria show social behavior in the form of **“wolf pack hunting”** and **sporulating through fruiting bodies** (only a minority of the colony becomes a spore).

Myxococcus shows two types of motility: **S-motility** drives swarming on soft agar surfaces, while **A-motility** contributes to swarming on relatively firm agar surfaces. **S-motility requires pili and fibrils**. If one knocks out pilin production the bacteria can regain the ability to swarm on firm agar. This is called **evolved cooperative motility (ECM)**. Through various test it was shown, that instead of redeveloping pili, Myxococcus **adapted A-motility to regain the ability to make fibrils**.

## 6 Sources of phenotypic variation

### 6.1 Genetic mutation

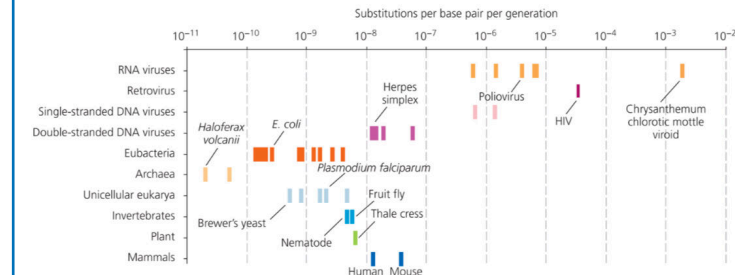
genetic differences between individuals cause phenotypic variation

#### 6.1.1 Mutation rates

Base-pair matching during synthesis leaves only **one unrepaired mismatch every 100 million times**. This results in around 38 new mutations per human gamete or 76 per

baby. Also a replacement generation of the human population would average 83 mutations per bp site.

The base substitution rate in various organisms varies by several orders of magnitude.



Mutation rates can be calculated from **Mutation Accumulation Lines**:

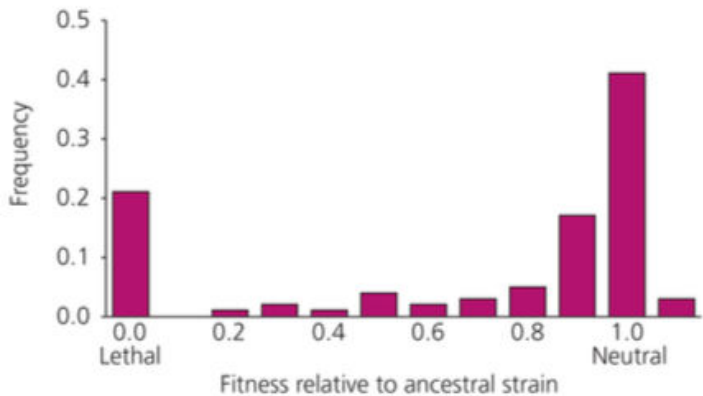
- 1) maintain 25 replicate lineages of one common ancestor
- 2) grow for 500 generations while choosing randomly which individual is transferred (no selection); in control lineages transfer many individuals per generation (selection)  $\Rightarrow$  mutations accumulate and reduce fitness
- 3) sequence one chromosome from ancestor and from individual taken from each replicate lineage
- 4) average mutation rate per bp per generation

$$= \frac{\text{total \# of mutations}}{\# \text{ of bps} * \# \text{ of generations} * \# \text{ of lineages}}$$

#### 6.1.2 Distribution of mutation fitness effects

Most mutations are **neutral or negative** in their effect on fitness. A frequency-fitness diagram might look like this:





### 6.2 Environmental variation

environmental variation in space/time cause an individual organism's phenotype to vary

### 6.3 Genotype x environment variation

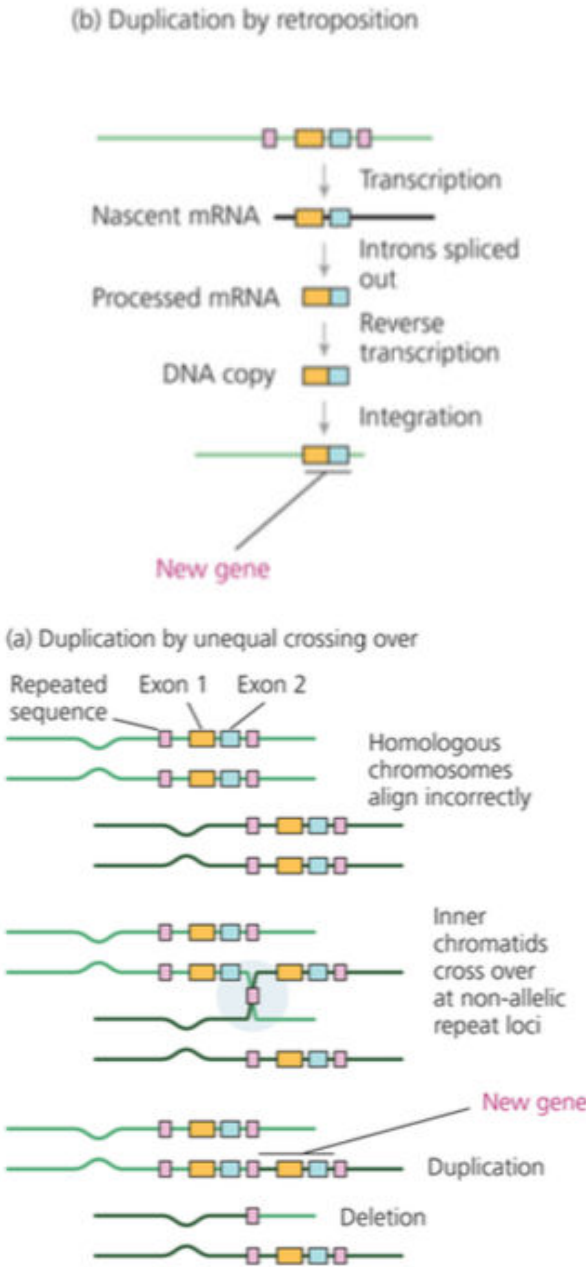
genetic differences cause distinct individuals to respond to environmental variation differently

### 6.4 vocabulary to know

complementary base pairs	neutral alleles
point mutation	gene duplication
transition	unequal crossing over
transversion	pseudogenes
replacement (non-synonymous)	paralogs, paralogous
substitutions	
silent site (synonymous)	orthologs, orthologous
substitutions loss-of-function	linkage
mutations	
indels	polymorphism
selection coefficient	frequency
polyploidy	chromosome inversion
genome duplication	

### 6.5 New genes

New gene copies can arise either by **retro-transposition**, which results in genes **without introns or regulatory regions** in eukaryotes, or by **unequal crossing over** during meiosis, which results in new copies **with introns and regulatory regions**.



By contrasting the number of duplicate genes vs. age of several species Michael Lynch arrived at an estimated duplication rate of **0.01 per gene per million years**. Considering that most organisms have a lot of genes a big amount of new genes arise every 1 million years. These new gene copies can have different fates:

- lost
- deactivated and regained as pseudogene
- diverge to serve function distinct from parent gene

### 6.6 Inversions

An **inversion** is an event when a piece of a chromosome is flipped around and refuse the other way around. They can enforce allele linkage due to lack of crossing over. Thus inversions that lock in advantageous allele combinations can be maintained by selection.

### 6.7 Whole genome duplication

**Whole genome duplications** are most commonly found in self-fertilizers. They result in a massive new input of genetic material that can evolve.

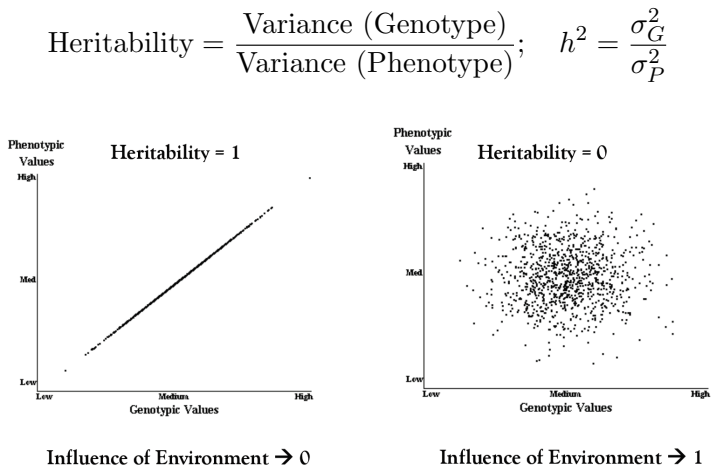
### 6.8 More impactful mutations

Name	Description	Mechanism	Significance
Point mutation	Base pair substitutions in DNA sequences	Chance errors during DNA synthesis or during repair of damaged DNA	Creates new alleles
Chromosome inversion	Flipping of a chromosome segment, so order of genes along the chromosome changes	Breaks in DNA caused by radiation or other insults	Alleles inside the inversion are likely to be transmitted together, as a unit
Gene duplication	Duplication of a short stretch of DNA, creating an extra copy of the sequence	Unequal crossing-over during meiosis or retrotransposition	Redundant new genes may acquire new functions, by mutation
Genome duplication	Addition of a complete set of chromosomes	Errors in meiosis or (in plants) mitosis	May create new species; massive gene duplication

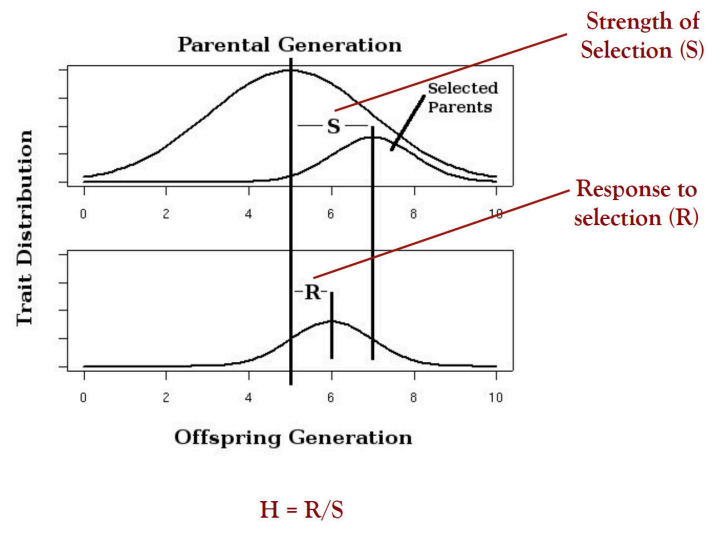
# 7 Population Genetics

Population genetics integrate Darwin's theory of evolution by natural selection and the Mendelian laws of inheritance. The field seeks to document and explain changes in allele, genotype and phenotype frequencies. It offers a quantifiable definition of evolution: It happens by allele frequency changes in the population.

## 7.1 Heritability



### 7.1.1 Selection Experiments

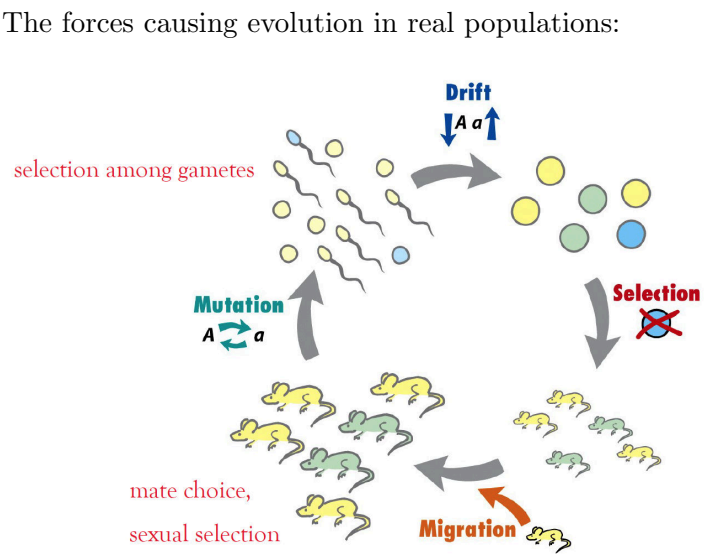


## 7.2 Calculating Genotype and Allele Frequencies

	AA	Aa	aa
$f(\text{Genotyp})$	$\frac{36}{100}$	$\frac{48}{100}$	$\frac{16}{100}$

$$f(A) = \frac{36 + 36 + 48}{200} = 0.6; \quad f(a) = \frac{48 + 16 + 16}{200} = 0.4$$
$$f(A) + f(a) = 1$$

## 7.3 Evolution of Populations



### 7.3.1 Hardy-Weinberg Population and Principle

The ideal population:

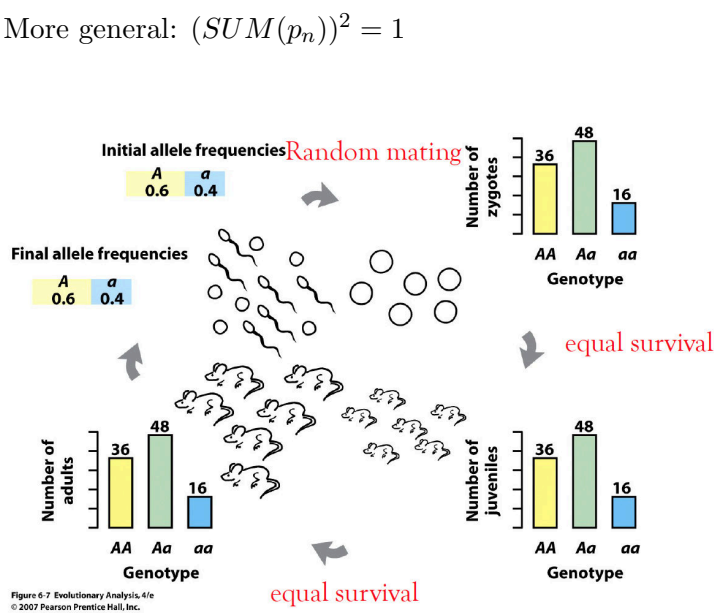
- 1) infinite population size
- 2) random mating among individuals
- 3) no evolution:
  - 1) no selection
  - 2) no mutation
  - 3) no migration
  - 4) no chance of evolution (genetic drift)

→ Does our population evolve?

If not: the population is in Hardy-Weinberg Equilibrium. (The HWP is a null model against which we test if a population is evolving or not.)

Simples case with two alleles (A and a):  $p^2 + 2pq + q^2 = 1$

Binomial equation:  $(p + q)^2 = 1$



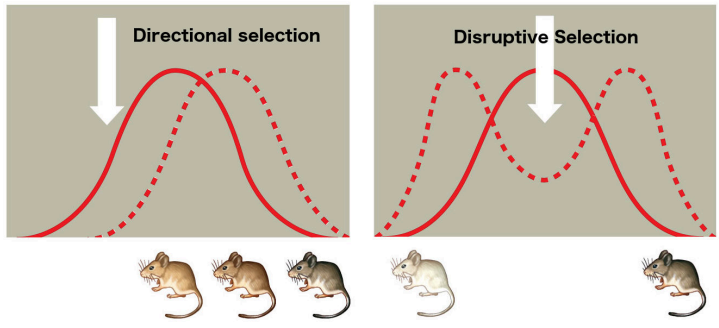
### 7.3.2 Violations of the HWE

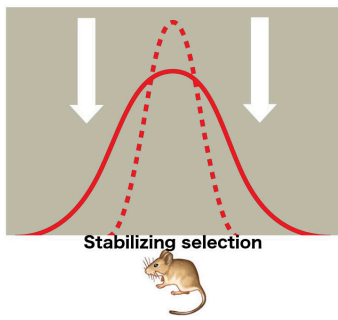
- 1) Allele frequencies change over time
- 2) Genotype frequencies do not meet expected values

### 7.3.3 Testing of the HWE

- 1) Calculate the observed allele frequencies
- 2) Calculate the expected number of each genotype under HWE
- 3) Compare expected and observed numbers
- 4) Optional but more exact: use a statistical chi-square test for significance

## 7.4 Selection





In **frequency-dependent selection**, the fitness of a phenotype depends on its frequency in the population:

- **positive FDS**: phenotype **increases** if it becomes most common in the population
- **negative FDS**: phenotype **declines** if it becomes most common in the population

#### 7.4.1 Selection coefficient $s$

The larger the **selection coefficient  $s$**  is the faster allele frequencies change. **Fitness  $w$**  (relative reproductive success) is inversely correlated to  $s$ :

$$w = 1 - s$$

#### 7.5 Calculating changes in genotype and allele frequencies due to selection

frequency of first allele:  $p = f(A) = 0.5$   
 frequency of second allele:  $q = f(a) = 0.5$   
 fitness of genotypes:  
 $w(AA) = 1.0$ ;  $w(Aa) = 0.7$ ;  $w(aa) = 0.6$

- 1) Calculate **average fitness  $w$**  for the whole population **before selection**:

$$w = p^2 * w(AA) + 2pq * w(Aa) + q^2 * w(aa)$$

- 2) Calculate **genotype frequencies after selection**:

$$f'(AA) = \frac{p^2 * w(AA)}{w}; \quad f'(Aa) = \frac{2pq * w(Aa)}{w}$$

$$f'(aa) = \frac{q^2 * w(aa)}{w}$$

- 3) Calculate **allele frequencies** among gametes **after** selection and random mating

$$f'(A) = p' = f'(AA) + 0.5 * f'(Aa)$$

$$f'(a) = q' = f'(aa) + 0.5 * f'(Aa)$$

$$\Delta p = p' - p; \quad \Delta q = q' - q$$

#### 7.6 Selection can act on different genotypes

If a flour beetle population has two alleles (+ being dominant and viable while - is recessive and lethal). The genotype fitness will play out as follows:

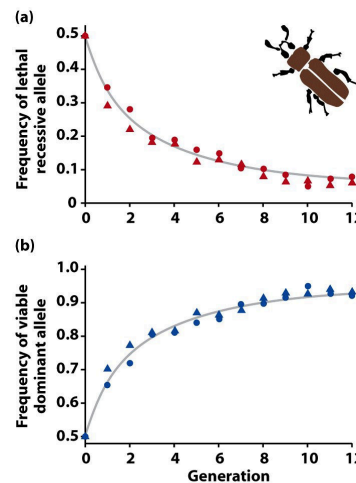
$$\begin{array}{ccc} w(+/-) & w(+/-) & w(-/-) \\ 1 & 1 & 0 \end{array}$$

One can predict how the frequencies of the alleles will change:

Generation	+/+	+/-	-/-	$p = f(+)$	$q = f(-)$	$\bar{w}_{pop}$
0	0	1000	0	0.5	0.5	
1 (HWE)	250	500	250	0.5	0.5	1
1 (Selection)	250	500	0	0.6	0.33	0.75
2 (Selection)	436	436	0	0.75	0.25	0.89

##### 7.6.1 Selection against recessive alleles

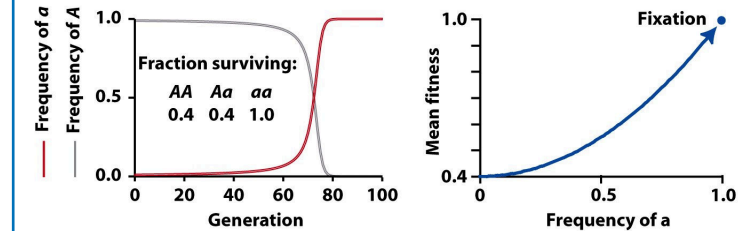
The selection against a recessive lethal allele is less and less efficient the rarer it becomes.



#### 7.6.2 Selection against dominant alleles

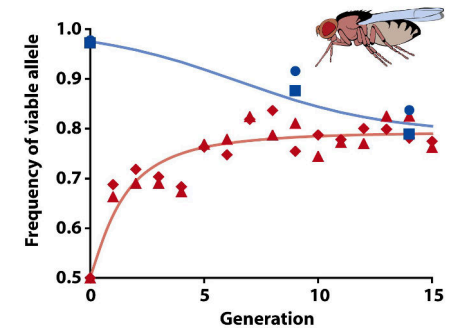
An inferior dominant allele will be eliminated from the gene pool relatively quickly.

Selection for a recessive allele and against a dominant allele ( $s = 0.6$ )

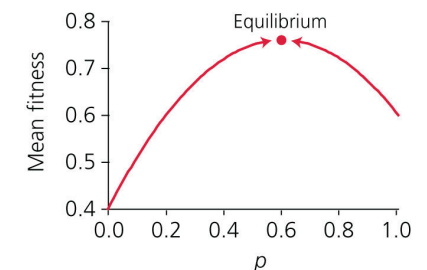


#### 7.6.3 Selection favoring heterozygotes

Heterozygote superiority (**overdominance**) leads to a stable equilibrium.



(b) Mean fitness as a function of  $p$  for overdominance



7.6.4 Selection against heterozygotes

Heterozygote inferiority (underdominance) leads to a unstable equilibrium.

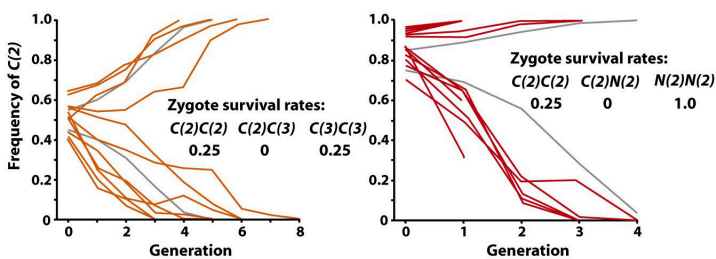
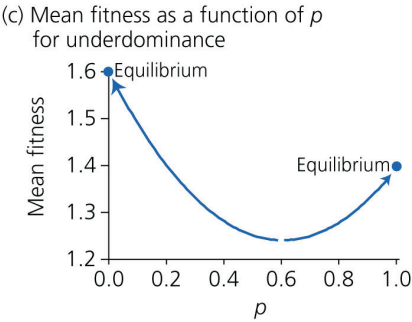
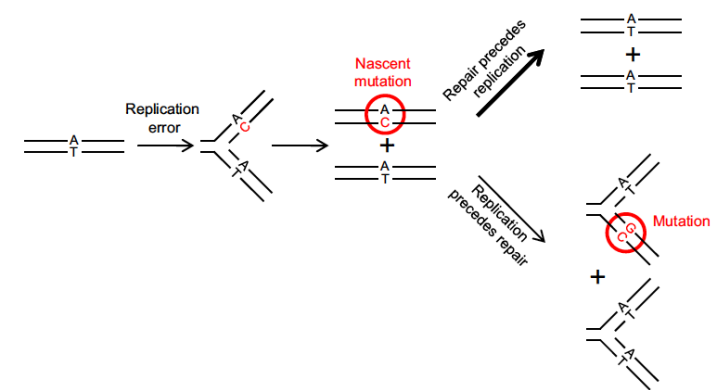


Figure 6-19f Evolutionary Analysis, 4/e © 2007 Pearson Prentice Hall, Inc.



7.7 Mutation

New mutations can arise by erroneous DNA replication

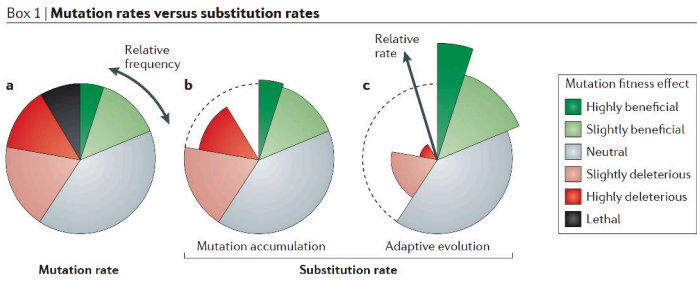


7.7.1 Mutation fitness effects

- **Positive:** rare, selected for increase

- **Neutral:** more common, fate determined by drift of hitchhiking
- **Negative:** very common, selected against, level in population determined by mutation-selection balance

After selection the frequency of different fitness levels between mutation is different from the initial mutations.



7.7.2 Induced mutagenesis

Induced mutagenesis is a stress-induced reaction to interrupted DNA replication due to UV-irradiation damage, polymerase-inhibition by antibiotics, etc. It leads to a transient increase of mutability through error-prone polymerases, increased DNA uptake / recombination or activation of “sleeping” prophages in genome

7.7.3 Mutation rate estimation

$$\mu = \frac{\text{Mutations}}{\text{Time} * \text{Genetic target}}$$

Mutation is the ultimate source of genetic variation. Over time more and more alleles are added to a gene pool. In the short run the effect may be weak, but it can slowly cause substantial change in the long-term.

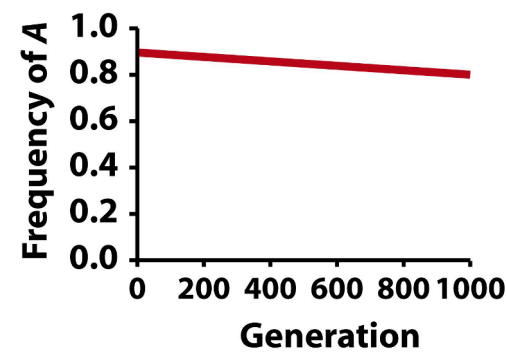


Figure 6-24 Evolutionary Analysis, 4/e © 2007 Pearson Prentice Hall, Inc.

7.7.4 Mutation-Selection Balance

After a population reaches a fitness peak a balance will be apparent between

- the rate at which new deleterious mutations occur and
- the rate at which these deleterious mutations are selected against (negative selection)

$$\hat{q} = \sqrt{\frac{\mu}{s}}$$

$\hat{q}$  = equilibrium frequency of mutant allele  
 $\mu$  = rate of mutation to mutant allele  
 $s$  = coefficient of selection against mutant allele

**Examples of autosomal recessive alleles:** SMA (spinal muscular atrophy) causes loss of muscle control through progressive atrophy (lower extremities first). Loss-of-function mutations lead to gradual death of motor neuron cells in parts of spinal chord. A high mutation rate to the loss-of-function allele leads to the maintenance of SMA at mutation-selection balance.

Cystic fibrosis can cause various complications with the most frequent cause of death being lung problems (at 80%). One in 25 people carries one copy of the recessive disease allele in CFTR. The CFTR protein is used by *Salmonella typhi* to invade gut cells which causes typhoid fever. The invasion can only happen in the wild type CFTR but not with the cystic fibrosis causing mutation. Meaning that there is a trade-off in CFTR between protection against causal agent of typhoid fever



and causing cystic fibrosis in homozygous carriers.

For dominant lethal alleles the mutation - selection balance looks as follows

$$\hat{q} = \mu$$

### 7.8 Genetic Drift

- random survival of alleles (sampling errors)
- most powerful in small populations
- strongest when natural selection is weakest
- cannot produce adaptations

Small population/sample sizes magnify the effects of change hence replication in experimental science.

#### 7.8.1 Heterozygosity is reduced over time

The probability that any given allele will drift to fixation is equal to its frequency in the population:

$$P(fix) = \frac{x}{2N}$$

where  $x$  = total number of allele copies and  $N$  = # of diploid individuals in population

#### 7.8.2 Effective population size ( $N_E$ )

Effective population sizes are especially sensitive to unequal sex ratios in populations. The higher the inequality the lower the  $N_E$  and the higher the effect of genetic drift.

$$N_E = \frac{4 * N_{males} * N_{females}}{N_{males} + N_{females}}$$

#### 7.8.3 Chance, Determinism, and Evolution

- Mutational input is largely random
- Evolution by selection is non-random
- Evolution by genetic drift is random

→ The power of drift is inversely proportional to the power of selection.

### 7.9 Migration

Migration homogenizes allele frequencies across populations if not opposed by other forces of evolution such as selection.  $F_{ST}$  values measure the degree to which separate populations are genetically distinct due to absence of gene flow.

### 7.10 Evolutionary change

The forces that cause evolutionary change:  
Forces that create variation in evolving populations:

- mutation
- recombination

Forces that determine the fate of variation:

- selection
- genetic drift
- migration
- (indirectly: non-random mating, NRM)

### 7.11 Selfing & Inbreeding

Selfing (self-fertilization) reduces heterozygote frequency. Inbreeding can depress average fitness.

### 7.12 Evolution at multiple loci: linkage disequilibrium

Extension of Hardy-Weinberg analysis to two loci through tracking of not only allele but also chromosome frequencies.

There is genetic linkage between two loci if they are on the same non-recombining stretch of a chromosome

- a in nuclear chromosomes of sexual diploids  
loci remain together after meiotic crossing
- b in non-recombining organisms / organelles  
loci are on the same single chromosome

If two loci are linked, selection on one locus can affect the evolutionary fate of the other (a hitchhiking-effect)

### 7.12.1 Linkage equilibrium

The frequency of any haplotype can be calculated by multiplying the frequencies of the constituent alleles.

$$g_{AB} = f(AB) = f(A)*f(B); \quad g_{Ab} = f(Ab) = f(A)*f(b)$$

$$g_{aB} = f(aB) = f(a) * f(B); \quad g_{ab} = f(ab) = f(a) * f(b)$$

With  $g$  being the genotype frequency, one can calculate the coefficient of linkage disequilibrium  $D$ :

$$D = g_{AB}g_{ab} - g_{Ab}g_{aB}$$

Linkage happens due to relative physical location on chromosomes, but linkage equilibrium and disequilibrium are characteristics of populations:

- In a population in linkage equilibrium the frequencies of B and b alleles are the same on A- and a-bearing chromosomes (and vice versa).
- In a population in linkage disequilibrium the frequencies of B and b alleles are different on A- vs. a-bearing chromosomes (and vice versa).

Deviations from HW expectations suggests that one of three mechanisms causing linkage disequilibrium is at work:

- selection on multi-locus genotypes
- genetic drift
- population admixture (i.e. migration)

## 8 Science History - Part 2

### 8.1 Pre-cursors of the scientific revolution

- scholarly institutions: monasteries → cathedral schools → universities
- western encyclopedic tradition
- philosophical appreciation of natural causation and regularity
- Byzantine preservation and Islamic spread of Greek literature
- 12th Century Greco-Arabic translation

- **rigorous, public scientific method:** formal integration of questions, observation, experimentation, induction and deduction
- dynamic **international university system**
- establishment of natural philosophy as core of university curriculum

## 8.2 Scientific revolution of the 16th/17th centuries

Revision or rejection of many Aristotelian ideas by:

Copernicus	Kepler	Galileo
Descartes	Francis Bacon	Newton
Vesalius		

## 8.3 Andreas Vesalius (1515 -1564)

- physician, author of *De humani corporis fabrica*
- founder of **modern human anatomy**
- attended university at Leuven and Paris
- worked at Padua, Bologna and Pisa
- thoroughly trained in Aristotelian / Galenic anatomy
- found flaws in Galen's work
- radically changed lecture format (live dissections)
- drew **detailed charts**
- **comparative anatomy:** difference between species

## 9 Why have sex?

- dilute your genes by half
- effort to find and choose a mate
- mate might have expectations
- risk of sexually transmitted diseases
- offspring might be infertile

### 9.1 The paradox of sex

**Maynard-Smith's** imaginary null population:  
sex vs. no sex does not affect

- 1) number of offspring per mother or mother's survival
- 2) survival or reproductive success of offspring

Male care of offspring can increase female fitness but in most species, males give away their genes and depart. Sex leads to recombination of genes through meiosis with crossing over and mating between non-relatives. The effect of recombination on evolutionary change is the interesting aspect of sex for population genetics.

## 9.2 Possible evolutionary reasons for sex

- **Linkage disequilibrium** happens. Sex can have no effect, and hence no benefit, in a population at linkage equilibrium. What is the process that **creates** linkage disequilibrium?
- Sex happens. Why are genes that **reduce** linkage disequilibrium favored?

Two types of models for why sex is beneficial:

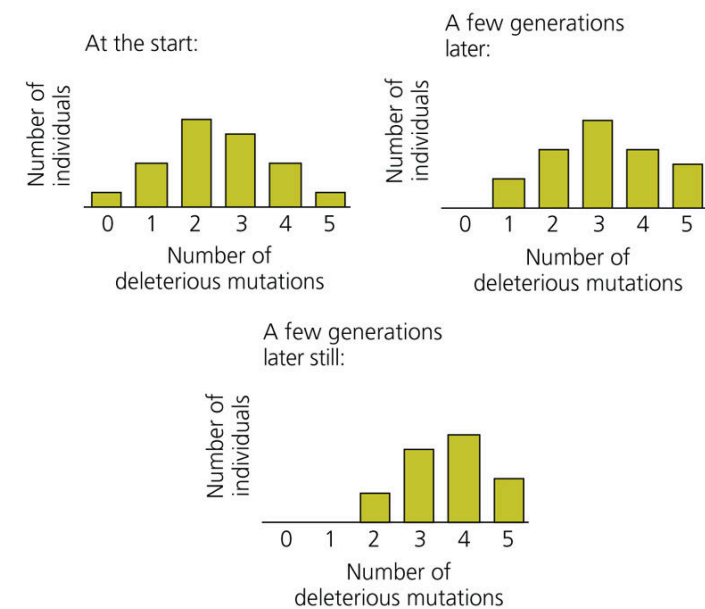
- 1) **Linkage disequilibrium by drift:** Sex allows selection to **purge unfavorable** multi-locus allele combinations that might drift to fixation in the absence of sex.
- 2) **Linkage disequilibrium selection:** Sex allows the creation of **new favorable** multi-locus allele combinations that can be promoted by selection.

Big question: **how did sex start?**

### 9.2.1 Muller's Ratchet

**Drift model** for why sex can be beneficial. Population size and mutation rate important for power of ratchet. It is strongest in small populations ( $N > \sim 1000$ ).

- **asexual populations** "ratchet" toward **accumulation** of more and more **deleterious mutations** by drift in small populations
- class of individuals with lowest number of deleterious mutations can be lost by drift
- forward mutation to deleterious alleles greater than back mutation to original state
- ratchet toward increased genetic load, decreased average fitness in population
- **sex breaks the ratchet**, as it allows resetting to zero mutation genotypes



But benefits of sex stopping Muller's ratchet are only manifested in the long term. In the short term asexual females still out-compete sexual females 2-fold in the absence of short-term benefits of sex.

### 9.2.2 Selection model

Sexual mode is more likely to rapidly create new variants that are more fit in novel environments. Sex "on the run" in an evolutionary "race" to keep adapting to changing conditions.

## 10 Adaptation

### 10.1 Identifying adaptations

How do we know that a trait is truly adaptive?

Adaptations are **traits that enhance fitness**.

Most direct: test the effect of removing a trait on reproductive fitness.

### 10.2 Adaptation caveats

Many trait variations among individuals, populations or species are likely to **not reflect adaptations** and not all

phenotypic traits are adaptations. While many adaptations are indeed remarkable from an engineering perspective, do not expect adaptations to conform to optimal engineering design by human standards.

### 10.3 Testing adaptation hypotheses

#### 10.3.1 Experimentation

Perform an experiment in which study groups differ by only one controlled variable.

Designing a **good experiment**

- 1) Ask an interesting and precise question
- 2) Generate a list of the most plausible possible answers to that question (hypotheses) in light of previous knowledge.
- 3) Design experimental treatments that will distinguish between all plausible hypotheses.
- 4) Include proper control groups, otherwise a study can be meaningless.
- 5) Subject all treatment groups to the same experiment conditions (except for the variable being tested).
- 6) Randomize to avoid bias.
- 7) Replicate sufficiently to test whether observed results are due to chance.

#### 10.3.2 Observation

Perform detailed observational studies. Find circumstances in nature that resemble an experiment.

#### 10.3.3 Comparisons

Perform comparative studies to test for correlations among variables that suggest causal relationships.

**Correlation does not imply causation**

Possible problems:

- 1) Non-independence of data points
- 2) Patterns may be shaped by phylogenetic relatedness rather than selection

If correlation holds up after adjustment for phylogenetic relationships, then correlation **may** reflect causation.

## 11 Science History - Part 3

### 11.1 William Harvey (1587-1657)

- attended university at Cambridge and Padua
- blood pumps from the heart in a closed loop (food isn't converted into it)
- bodies like machines that work by mechanisms
- encouraged finding of **mechanistic rules in biology** and using similar scientific methods to physics
- founded **modern embryology**

### 11.2 Nicholas Steno (1638-1686)

- attended Universities of Copenhagen and Florence
- showed that contracting muscles change shape, not volume
- helped founding science of **stratigraphy in geology**
- similarity of shark teeth to tooth-shaped stones
- **law of superposition**: horizontal rock layers represent a time sequence with oldest on bottom
- principles of **original horizontality** and **lateral continuity**
- fossils are **snapshots of life** at different times

### 11.3 Carolus Linnaeus (1707-1778)

- Swedish botanist, physician, zoologist
- modern scheme of nomenclature / modern taxonomy
  - **universal naming conventions**
  - hierarchical, binomial nomenclature
  - classified **humans with other primates**
- helped found modern ecology

### 11.4 Comte de Buffon (1707-1788)

- naturalist, mathematician, biologist, cosmologist
- influenced Lamarck and Darwin
- *Historie Naturelle*: goal of integrating all knowledge of nature
- theories about planet and its inhabitants (e.g. age of 70'000)
- integrated calculus into probability theory

- **life has long history**, spontaneous generation
- common ancestry of **humans and apes**
- **species change** might be possible by **various forces**
- different regions can have different biological communities (**biogeography**)

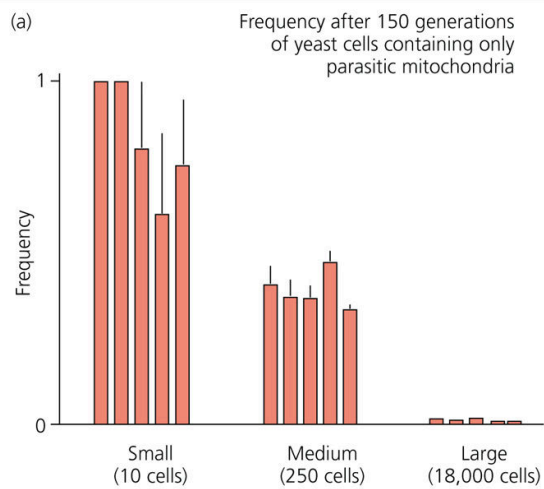
### 11.5 Erasmus Darwin (1731-1802)

- Charles Darwin's grandfather
- one of the first formal theories on evolution in *Zoonomia* (1795)
- discussed how **competition and sexual selection** could cause **changes within species**
- "integrative" approach: observations of animals, paleontology, biogeography, systematics, embryology, and comparative anatomy

## 12 Multi-level selection

Selection can happen on **multiple levels at once**. Take the following examples of yeast cells and their mitochondria: Yeast cells can harvest energy through their mitochondria by either **respiration** (more efficient) or **fermentation** (less efficient). There are **many mitochondria per yeast cell**. They **replicate independently** of the cell and can turn **parasitic** by deletion of genes necessary for respiration. Parasitic mitochondria **replicate faster**. Yeast cells with only respiring mitochondria grow faster than yeast cells with only parasitic, non-respiring mitochondria.

A selection experiment was done with three evolutionary treatments that differed in the strength of selection among yeast cells (small, medium and large effective population sizes), but did not differ in the strength of selection among mitochondria within yeast cells. All yeast cells started out with a mix of respiring and parasitic mitochondria.



## 13 Sexual selection

Selection on relative mating success that affects overall reproductive success. It is strongest when mating opportunities limit reproductive success.

### 13.1 Asymmetric Parental Investment

- sperm and ejaculate generation
- egg generation
- embryo & fetus carriage and nourishment
- care for mate providing direct offspring care
- protection of & provision for born offspring

The sex that invests more in offspring (usually females) is limited by resources and time, not potential mates, which should lead to greater choosiness about mates. The sex that invests less in offspring (usually males) is limited by number of mates, not resources and time, which should lead to greater intra-sex competition for mates.

### 13.2 Intra-sex Sexual Selection

Competition among males by

- physical combat
- sperm competition
  - sperm amount

- disposing of competitors sperm
- guard mates
- long copulation
- make mate unattractive to competitors
- infanticide
- being sneaky

### 13.3 Inter-sex Sexual Selection

Choosy females may exist because

- “good genes” hypothesis
- males bearing gifts: resource acquisition
- building on sensory wiring: evolving to exploit pre-existing bias
- reinforcement of sexually selected traits: “sexy sons” will in turn have a higher chance of mating

### 13.4 Sexual Selection on Females

Sexual selection on females can occur when having more mates rather than few increases fitness. **Polyandry** (female with multiple males) is very common in nature.

- Females can benefit from multiple matings
- Males can be choosy (e.g. body size, health)

### 13.5 Humans

- in pre-modern societies, maximum reproductive success of males is often higher than that for females
- males kill more often and kill more males than females
- depending on the culture, killers can either have more wives and offspring (amazonian Yanomamö) or aren't more reproductively successful (equatorial Waorani)
- height correlates weakly but significantly with the probability of having children among Polish men between 20 and 50 years old as well as among U.S. military officers
- British women of intermediate height were found to have more children on average than tall or short women

- during ovulation, women (on average):
  - become more attracted to men with deep voices and chiseled, symmetric faces (more extreme when the woman is in a relationship)
  - are more likely to cheat
  - become more interested in going to social gatherings
  - speak at a higher pitch
  - change walking gait
  - wear more revealing clothes
- men produce more testosterone after smelling clothes from ovulating women than from non-ovulating women