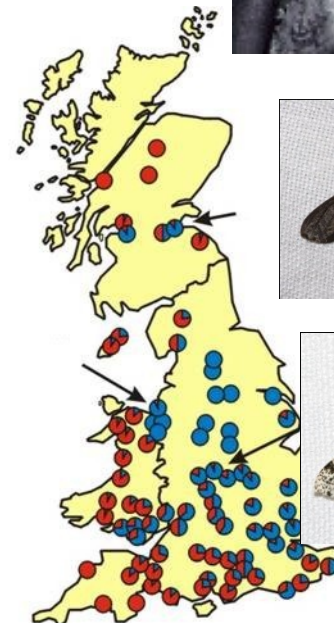
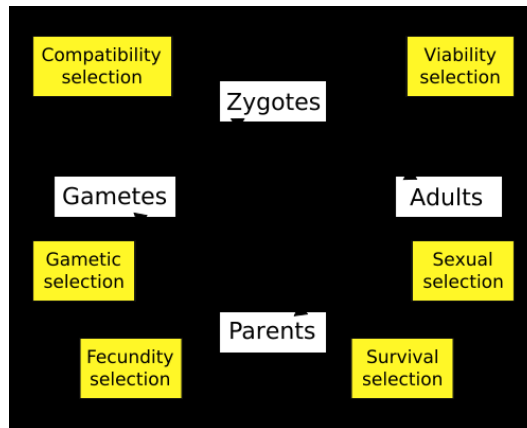
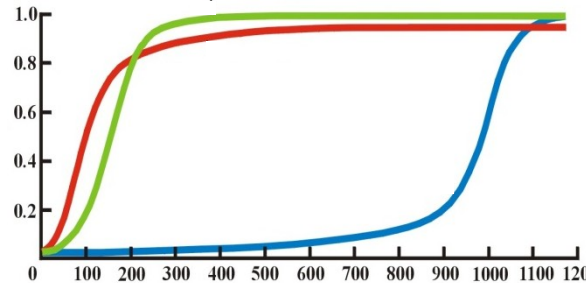
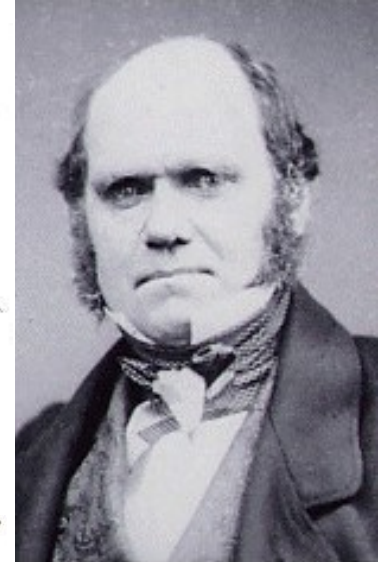
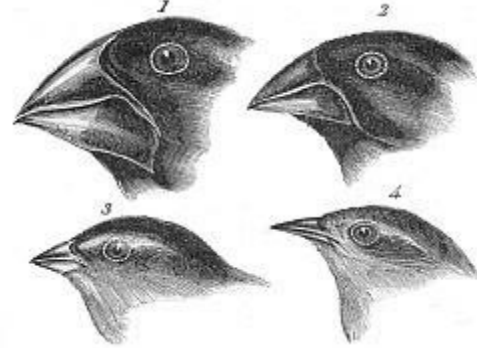


NATURAL SELECTION

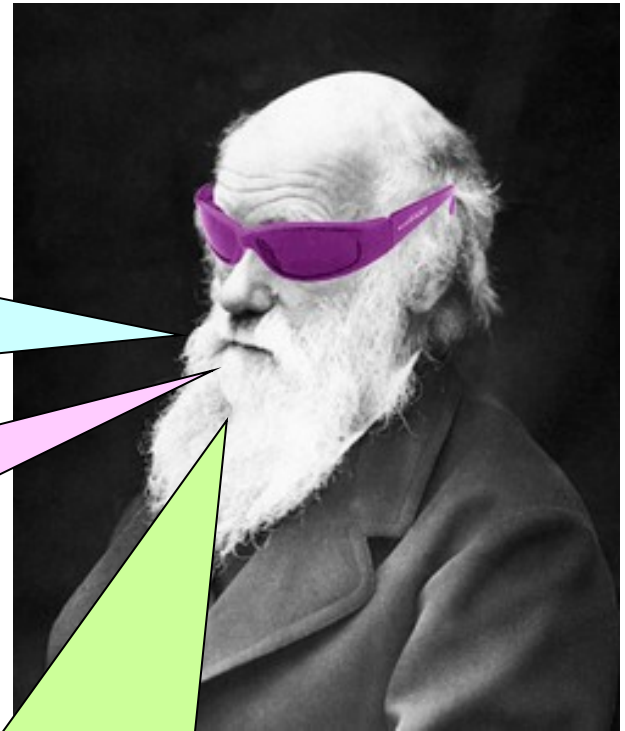


Evolution by natural selection:

All organisms produce more offspring than can survive and reproduce.

Individuals (genotypes) differ in heritable traits related to survival and reproduction.

The genotypes differ in their contribution to the next generation, ie. the most fit genotypes contribute more than the less fit ones.

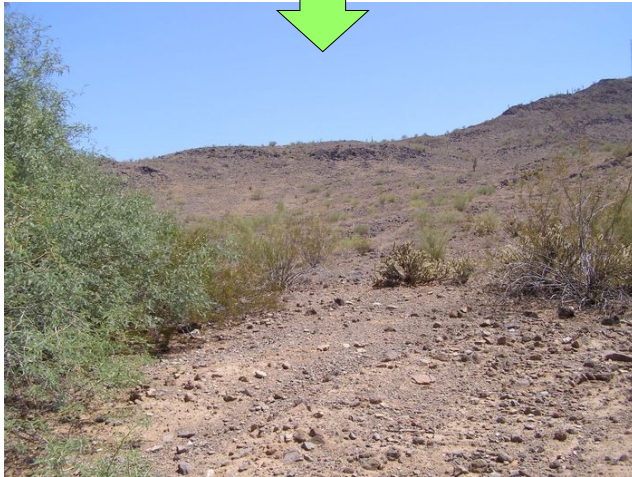


Rock pocket mouse (*Chaetodipus intermedius*): Sonoran and Chihuahuan Des.

dd



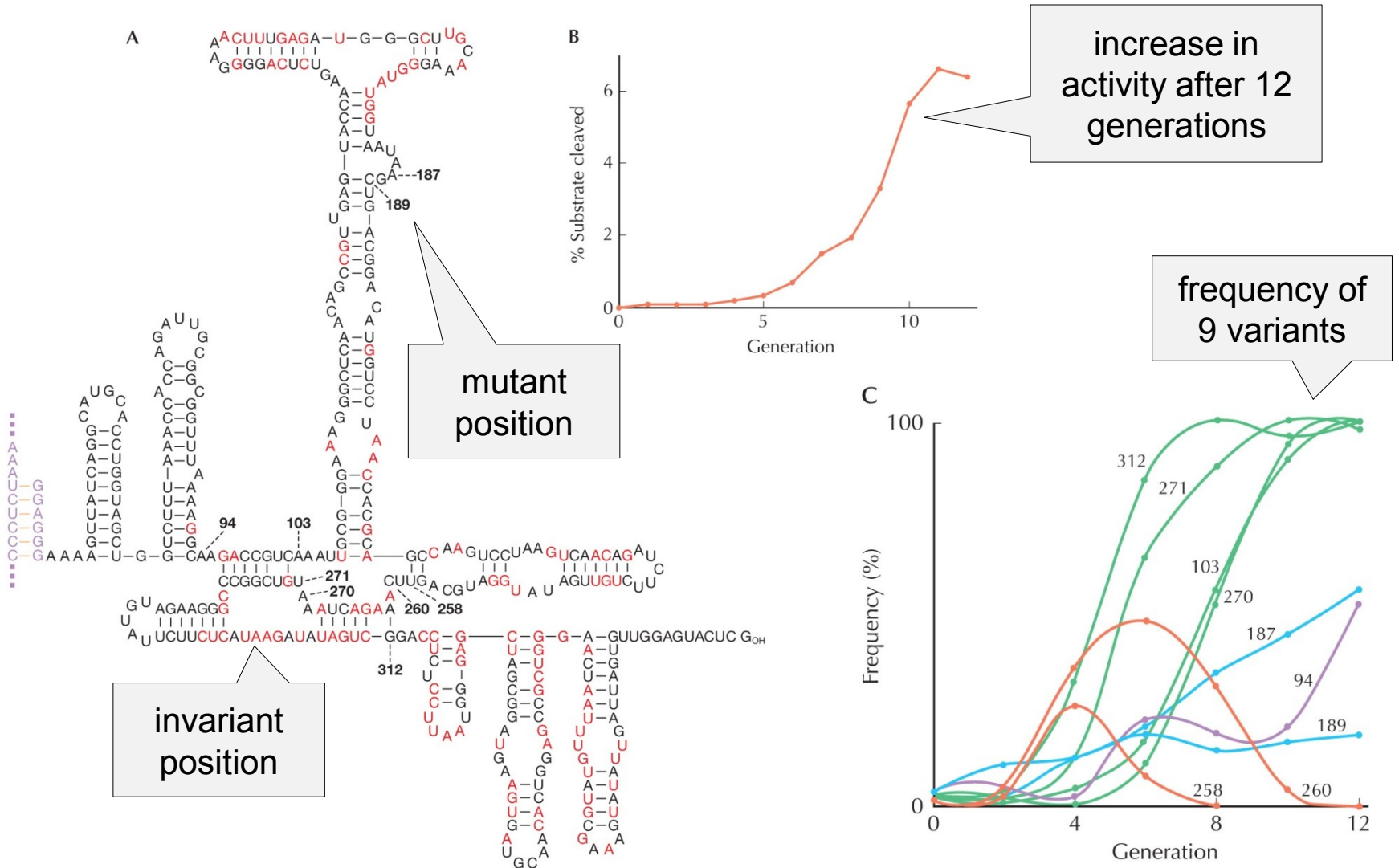
DD, Dd



60-98% survival
relative to dark
individuals

Selection on the RNA level:

intron *Tetrahymena*: Ca⁺ instead of Mg⁺ (normal state)



REPRODUCTIVE FITNESS, w

= average per capita lifetime contribution of individuals of a given genotype to the population after one or more generation

absolute number of the offspring = **absolute fitness**

discrete generations, stable population \rightarrow fitness ≈ 1 in asexual organisms, ≈ 2 in sexual organisms; even with a slight deviation the population goes either to extinction or to overpopulation

continuous time scale \rightarrow growth rate ≈ 0

in evolution relationships between genotypes in a population more important \rightarrow **relative fitness**

discrete time \rightarrow = ratio of absolute fitness; continuous time \rightarrow = difference between growth rates

usually relative fitness of the most fit genotype = 1

alternatively we may relate to the mean population fitness

Components of fitness:



Total number
of offspring

Quality of the weaned
offspring

Viability
Reproductive success
Litter size
Litter frequency
Number of litters

Resistance to pathogens
Escape from predators
Frequency of ovulations
Survival of embryos

Maternal behaviour
Milk production
Size of mammary
glands

zygotic selection:

viability

reproductive success

fertility/fecundity



gametic selection:

gamete viability

fertilisation success

segregation distortion



Change of allele frequencies and selection coefficient, s

Fitness in the pocket mouse:

AA	1
Aa	1
aa	$1 - s$

mean $s(aa) \approx 0,20$



$w = 0,60 - 0,98$
 $\Rightarrow s = 0,02 - 0,40$

biggest change when $p=q=0,5$

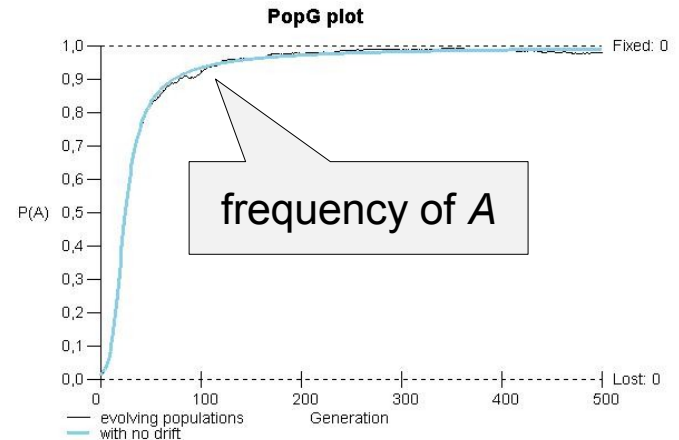
Increase of beneficial dominant allele A:

negatively proportional to the mean fitness of the population \Rightarrow with increasing frequency of the beneficial allele the evolution is slowing down; if q or $p = 0$ $\Delta p = 0$

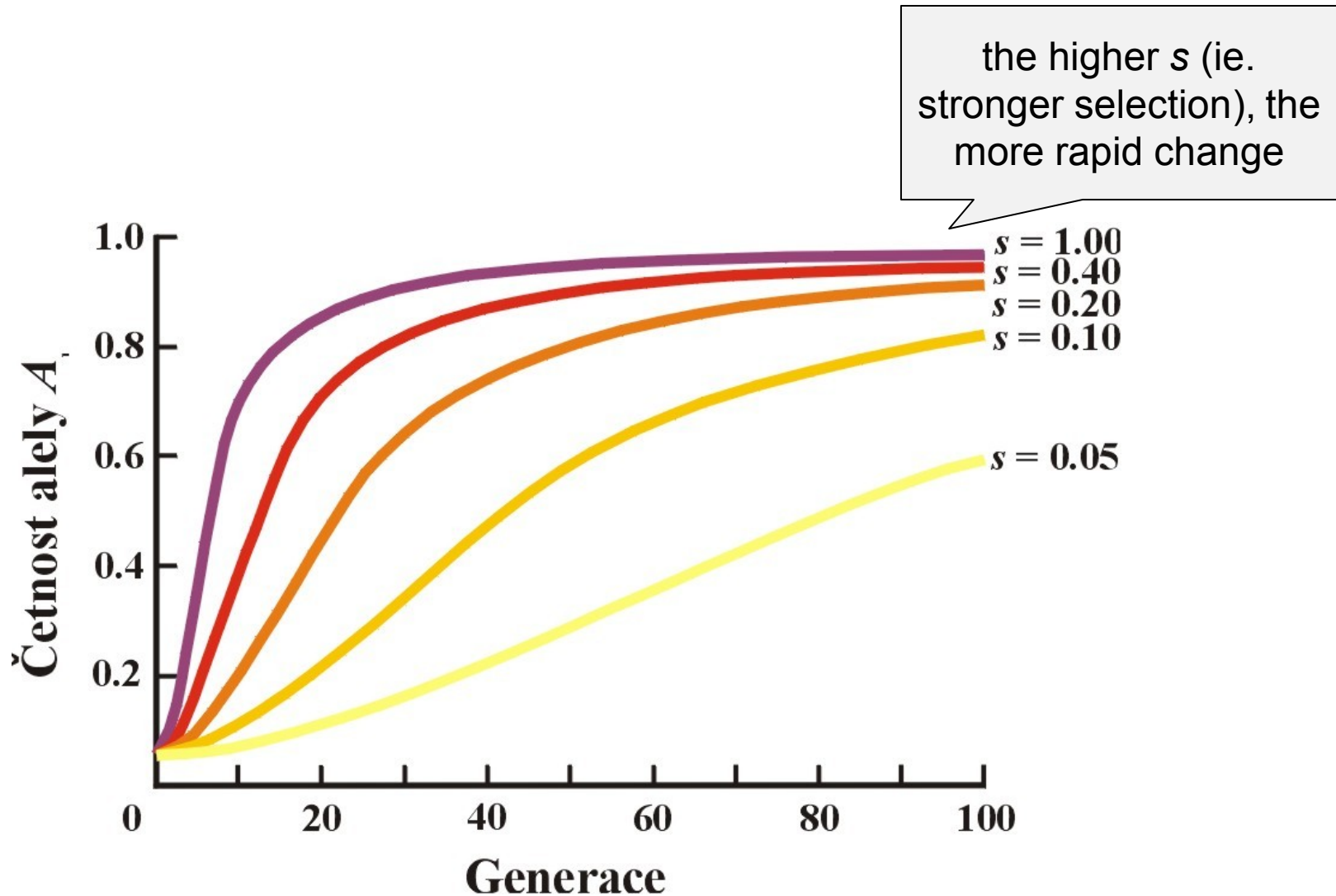
$$\Delta p =$$

$$-\frac{pqS}{\bar{w}}$$

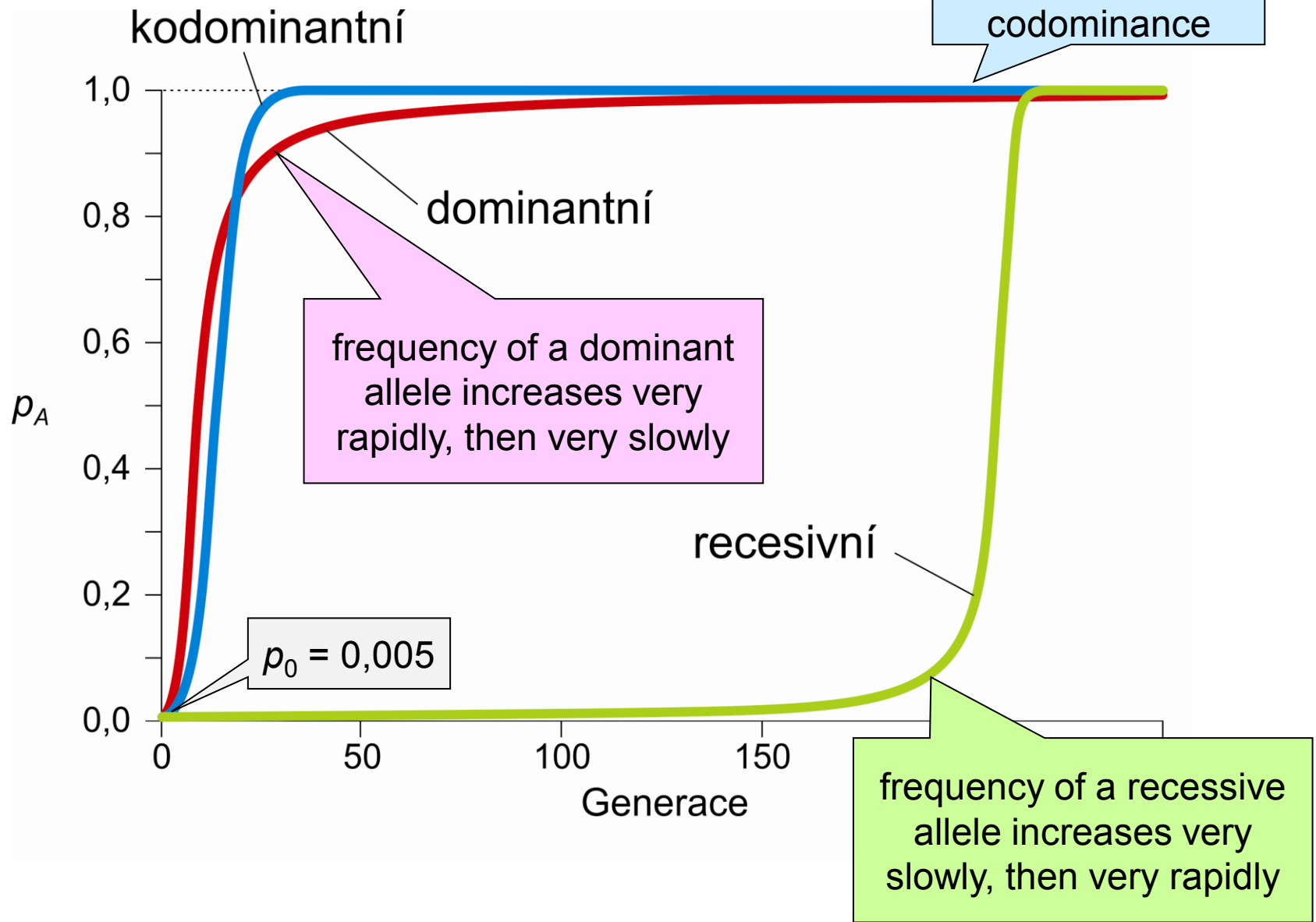
When $s = 0,20$ and initial frequency $A = 0,01$ the freq. increases to 0,95 in ca. 120 generations



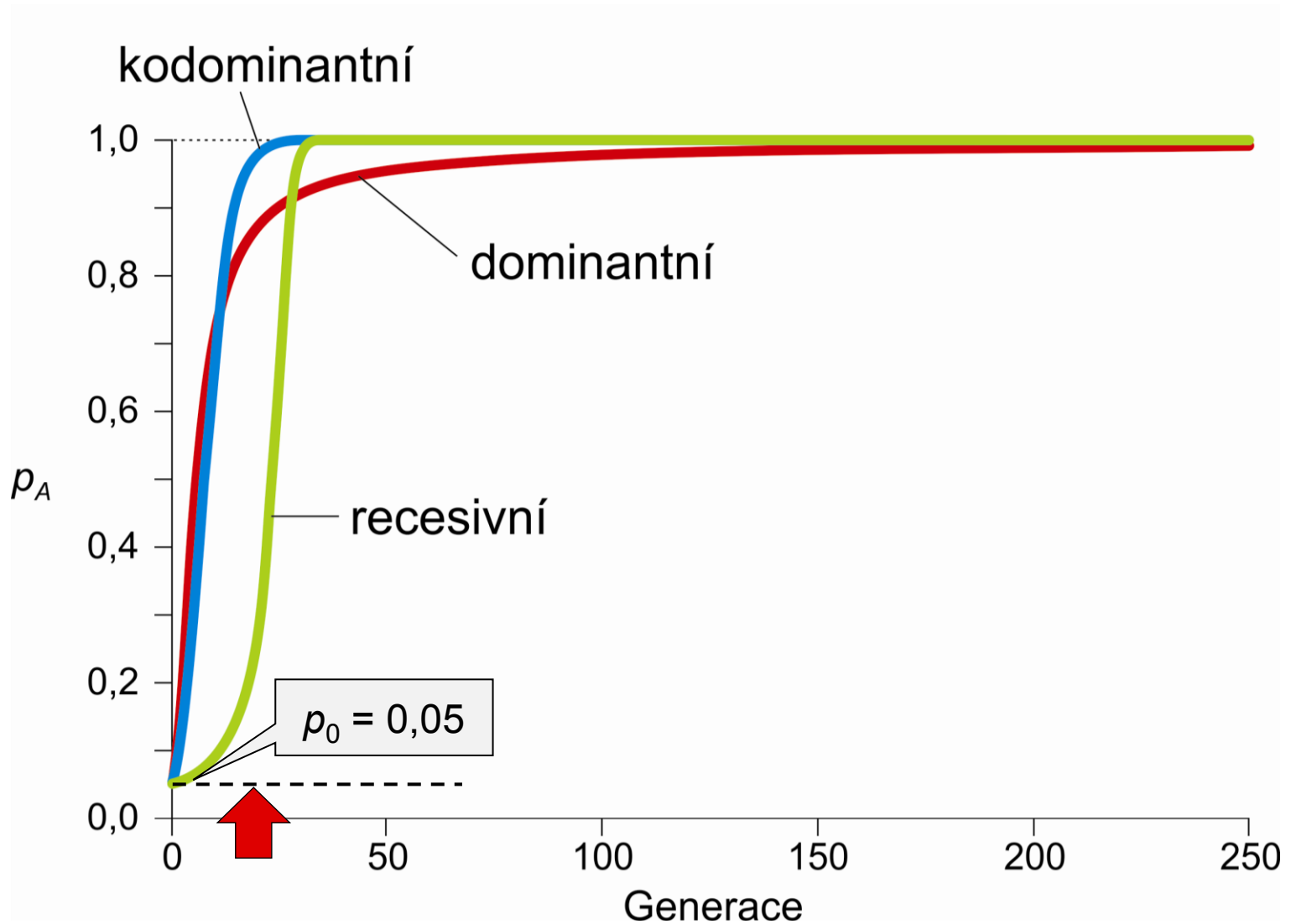
Increase of a advantageous dominant allele A :



Selection and dominance



Effect of the initial allele frequency:



STUDY OF NATURAL SELECTION:

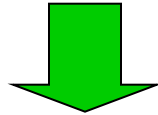
1. Correlation of allele frequencies across populations

Adh^F in *D. melanogaster*



2. Deviations from expected genotype frequencies (HW)

3. Temporal changes of a trait:

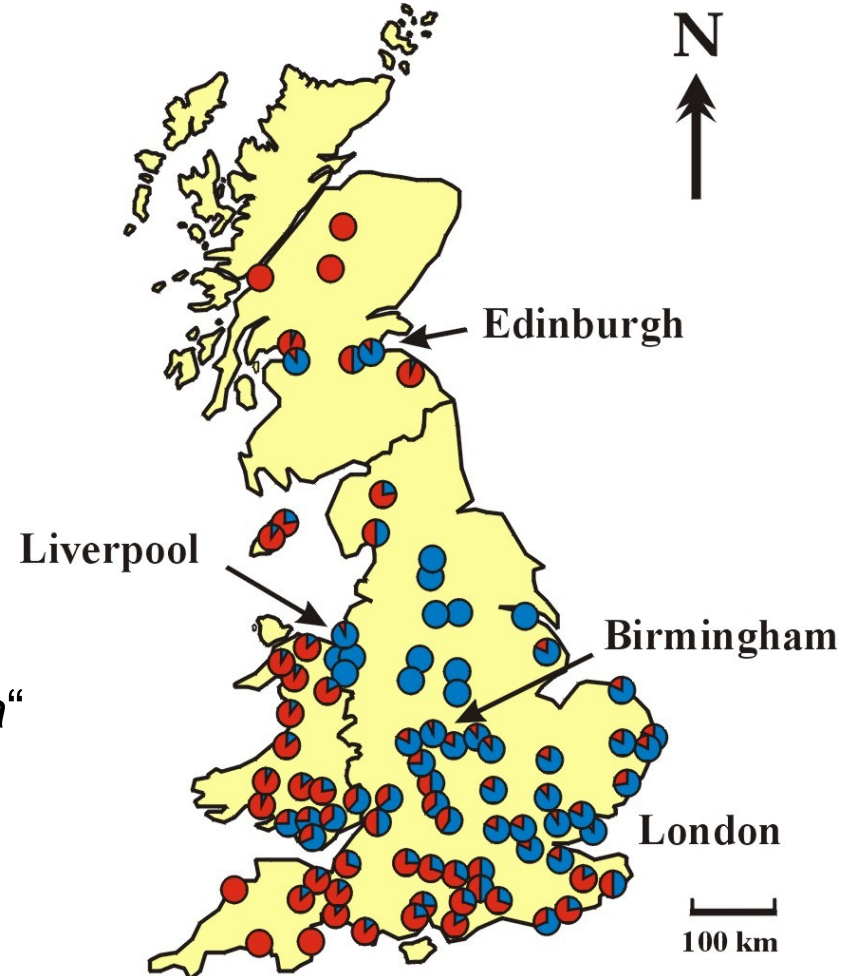


industrial melanism in the peppered moth (*Biston betularia*) in Britain



● „typica“

● „carbonaria“



4. Experimental evidence:

H.B.D. Kettlewell



Bernard Kettlewell



Light and dark Peppered moths

polluted area

light

Birmingham (znečištěná oblast)

Počet zpětně odchycených:

pozorovaný 18

očekávaný 36

Relativní míra přežívání 0,5

Relativní fitness $0,5/1,15 = 0,43$

Deanend Wood (neznečištěná oblast)

Počet zpětně odchycených:

pozorovaný 67

očekávaný 53

Relativní míra přežívání 1,26

Relativní fitness $1,26/1,26 = 1$

non-polluted area

Tmavá forma (*carbonaria*)

dark

140

122

1,15

$1,15/1,15 = 1$

Tmavá forma (*carbonaria*)

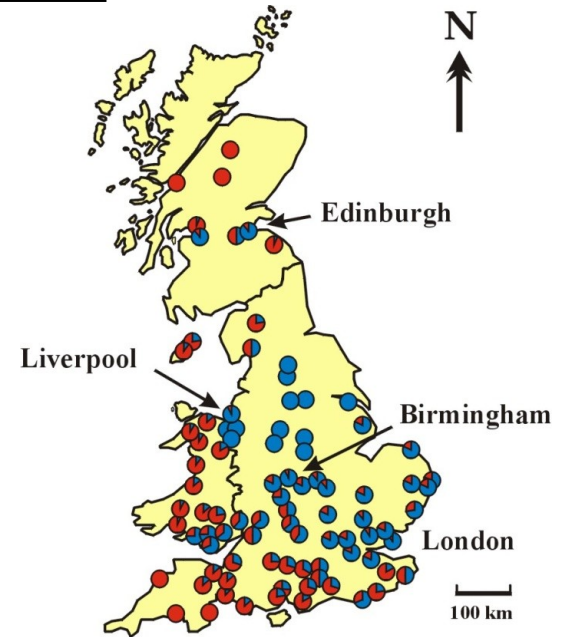
32

46

0,69

$0,69/1,26 = 0,55$

relative fitness



Problems:

industrial melanism of
B. betularia in Britain

3 alleles, not 1, affect the colouration

increase of melanism also in species not endangered by predation by insectivorous birds (pigeons, cats, some beetles)

in some areas correlation between melanism and pollution weak

errors in the experiment:

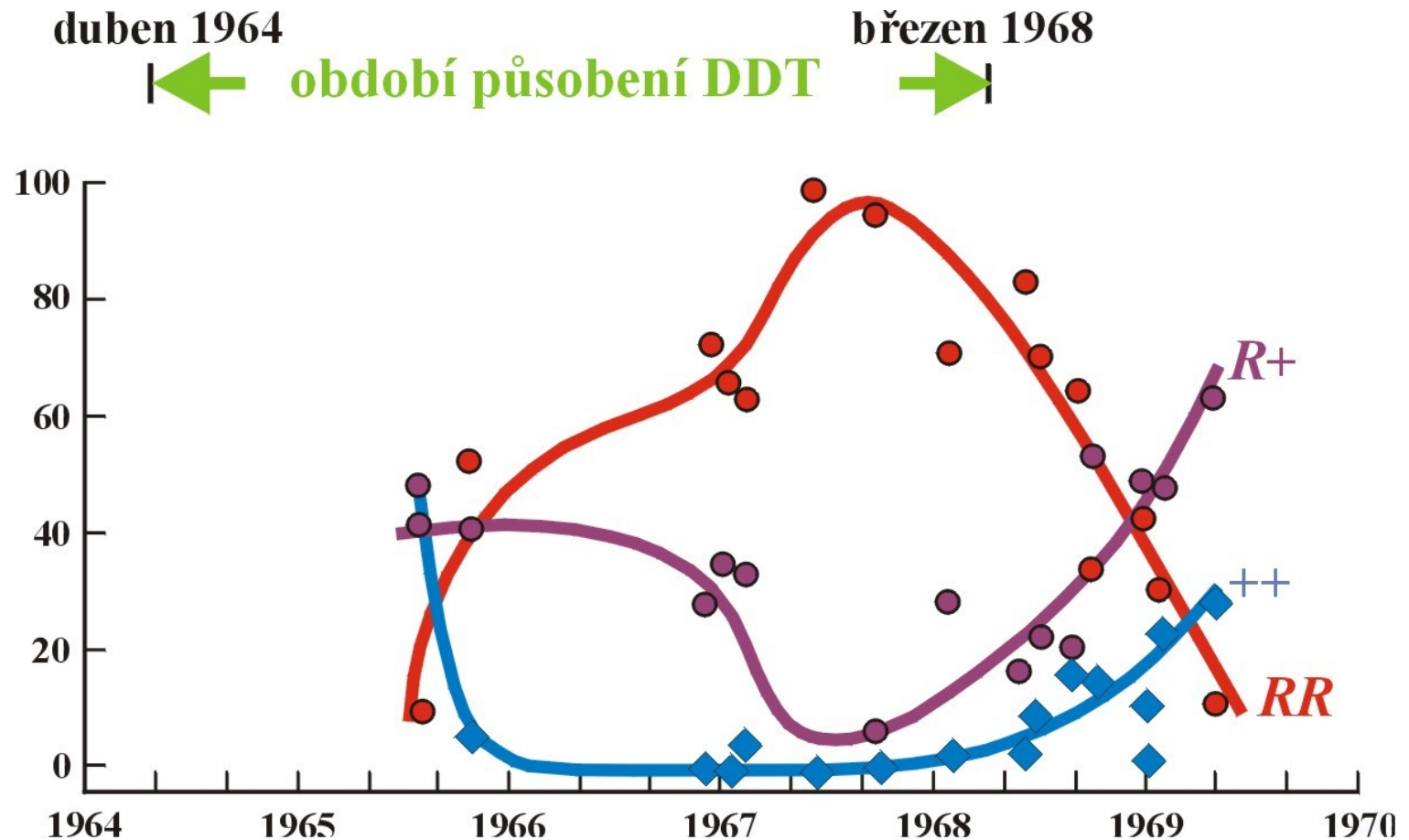
during the day, peppered moths stay on horizontal branches, not on trunks (different lichen species); in butterflies and birds different perception of UV

under laboratory conditions the *typica* viability by 30% lower than that of *carbonaria*

better absorption of solar radiation in melanic forms?
(eg. two-spot ladybird)

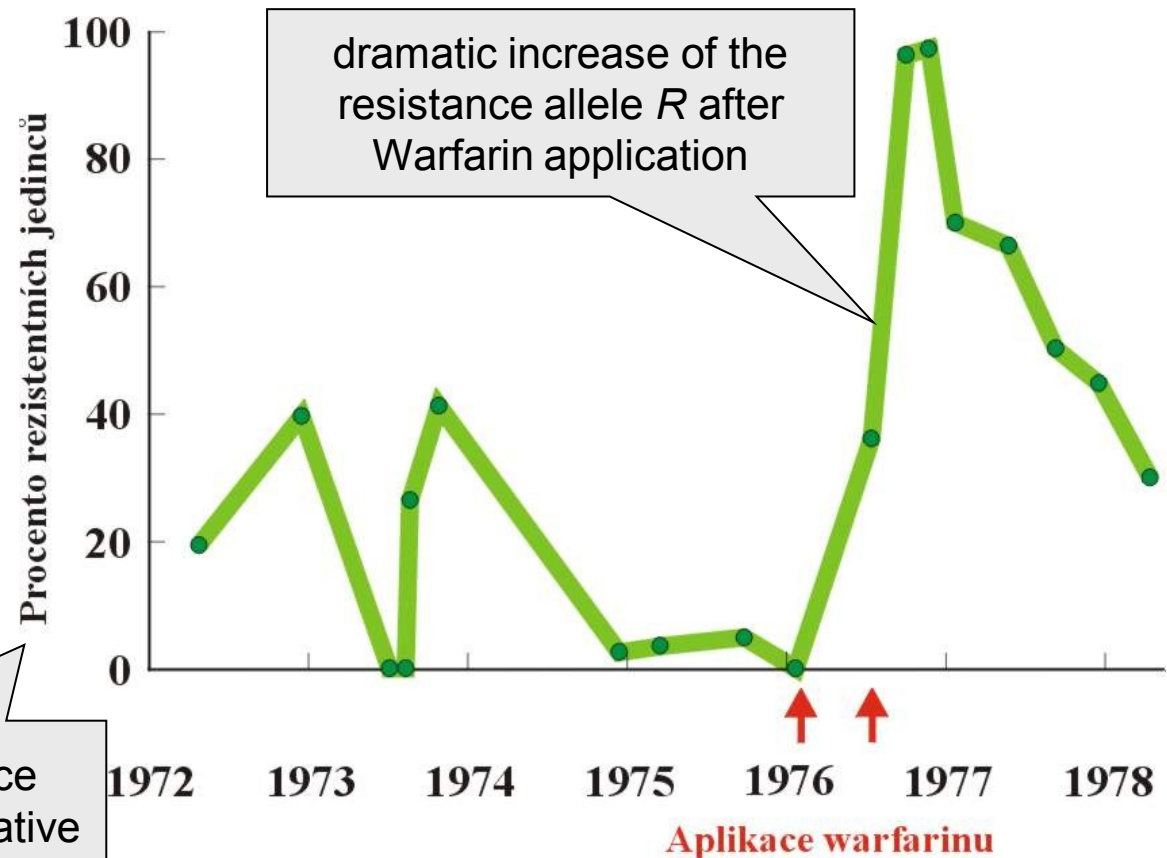
5. Resistance

eg.: DDT resistance in mosquitos (*Aedes*, *Anopheles*):



eg.: Warfarin resistance in rats:

Warfarin = blood anticoagulant, inhibiting the enzyme responsible for the recovery of vitamin K (coagulation cofactor)

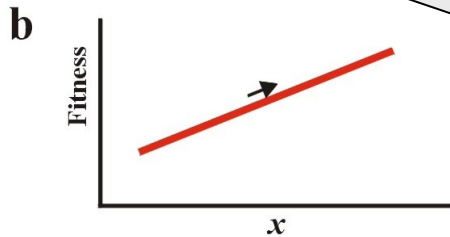
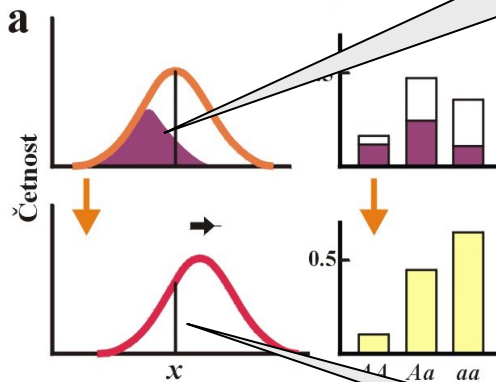


relative to the resistance *R* allele *dominant* but relative to the increased demand of vitamin K *recessive*

Relationship between phenotype and fitness: basic selection regimes

directional

these phenotypes are disadvantageous

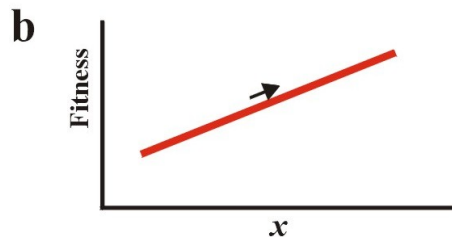
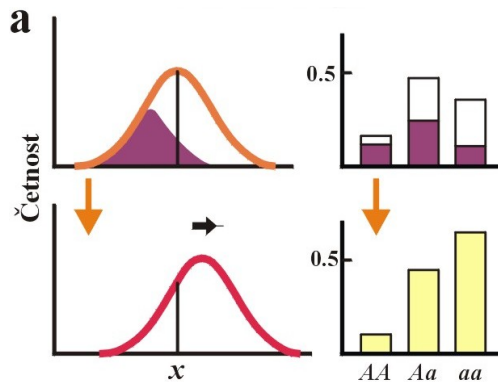


original mean

consistent change of environment
shift of the mean
variance unchanged

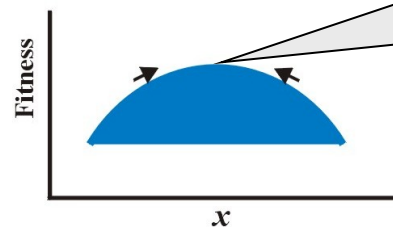
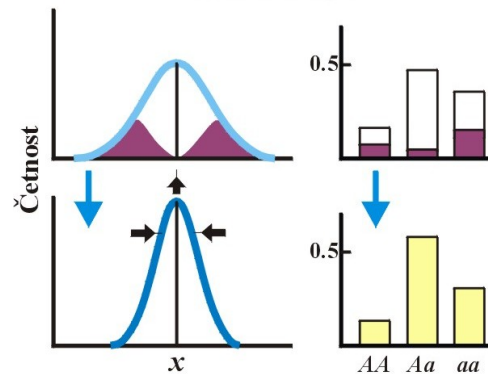
Relationship between phenotype and fitness: basic selection regimes

directional



consistent change of environment
shift of the mean
variance unchanged

stabilizing



stable environment
mean unchanged
lower variance

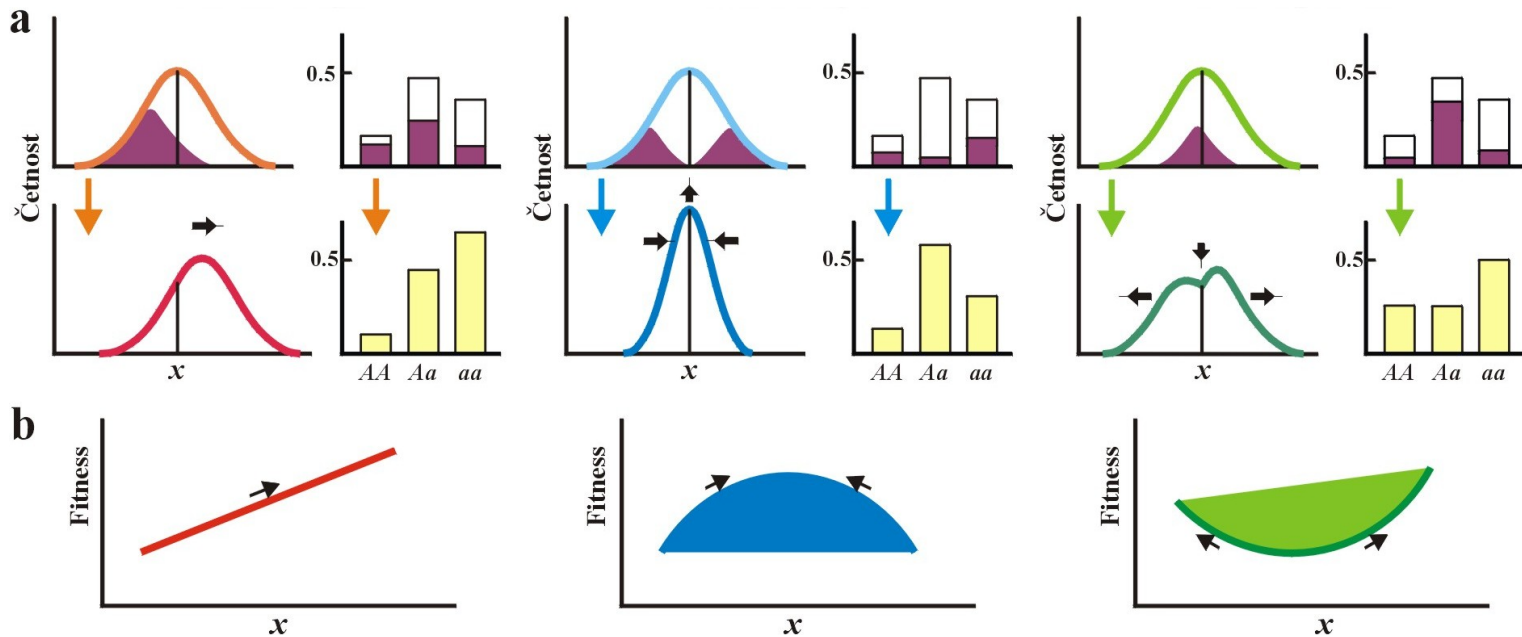
highest fitness in individuals with intermediate (mean) phenotypes

Relationship between phenotype and fitness: basic selection regimes

directional

stabilizing

disruptive

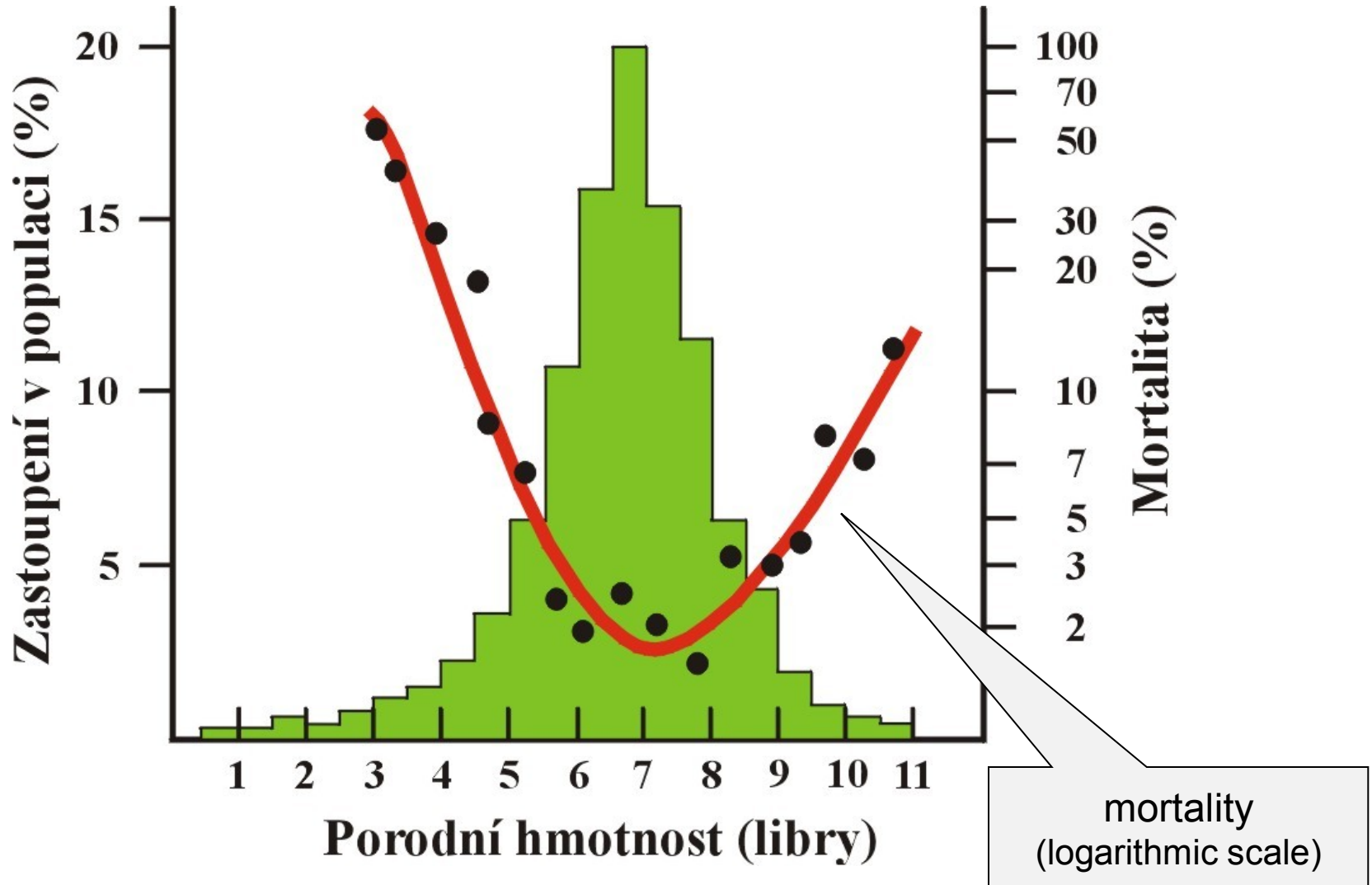


consistent change of environment
shift of the mean
variance unchanged

stable environment
mean unchanged
lower variance

heterogenous environment
intermediate phenotypes
disadvantageous
higher variance

stabilizing selection – birth weight in humans



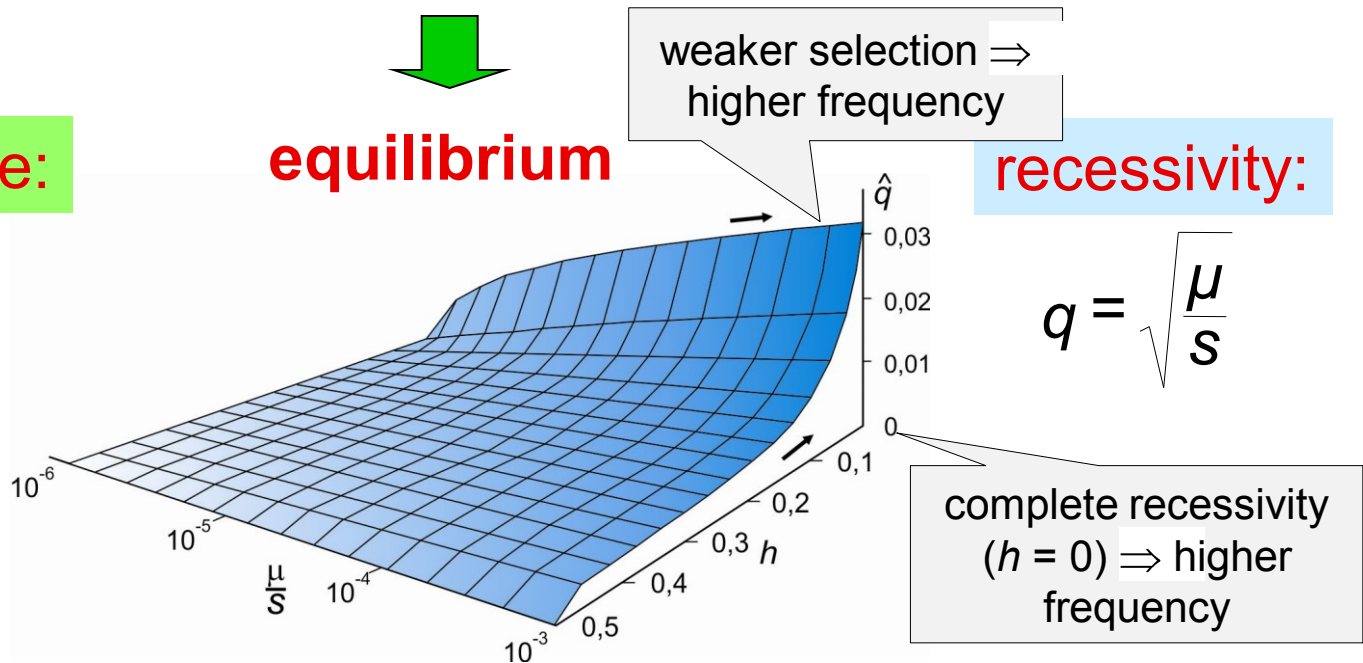
Equilibrium between selection and mutation

recurrent emergence of a deleterious mutation × elimination by selection

dominance:

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

equilibrium



Muller-Haldane principle:

Regardless of dominance/recessivity of a deleterious mutation, its impact on decreasing fitness is independent of the level of its harmfulness.

Equilibrium between selection and gene flow

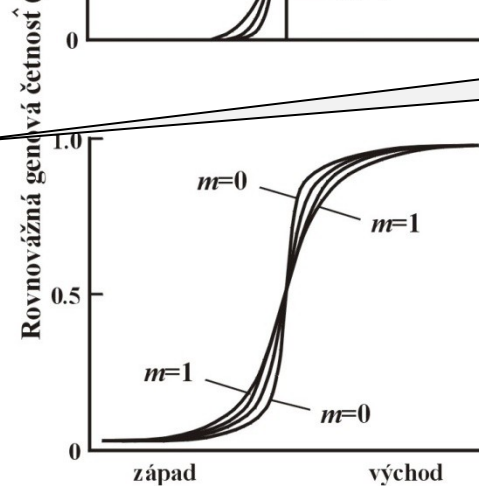
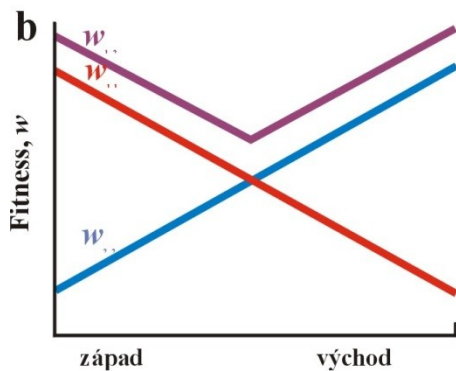
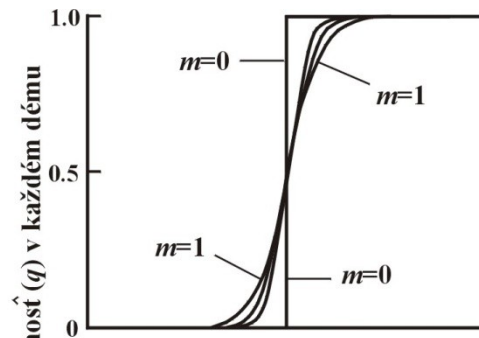
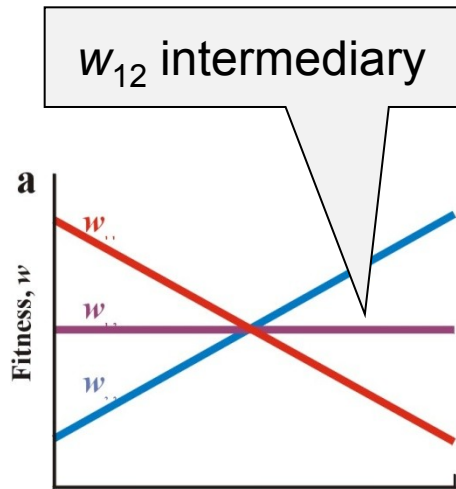
repeated „influx“ of a deleterious allele \times elimination by selection



equilibrium

- 1. $m > s \Rightarrow$ allele fixation
- 2. $m < s \Rightarrow$ allele elimination
- 3. $m = s \Rightarrow$ polymorphism

divergence among demes

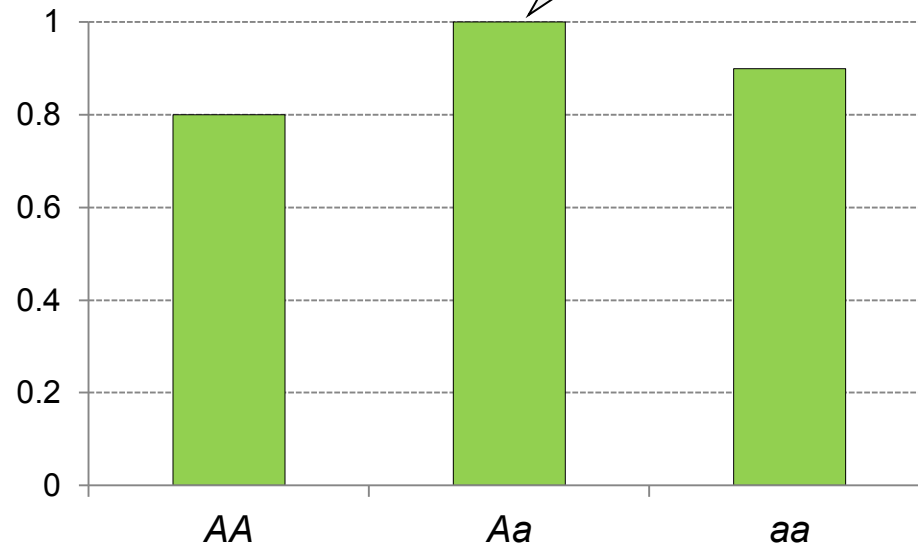


Balancing selection

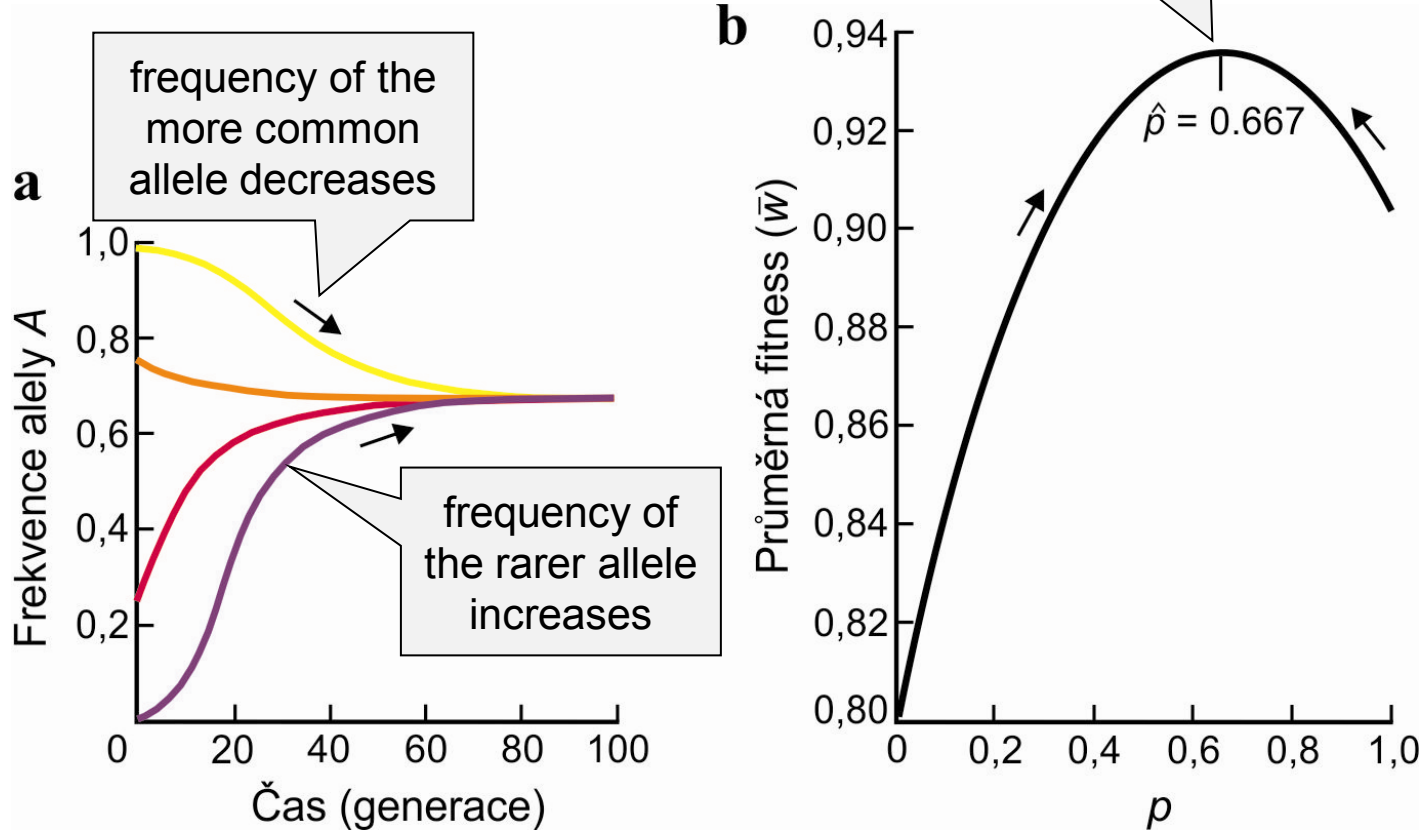
1. Selective advantage of heterozygotes = overdominance

$$W_{AA} < W_{Aa} > W_{aa}$$

heterozygote fitness
is higher than w of
homozygotes

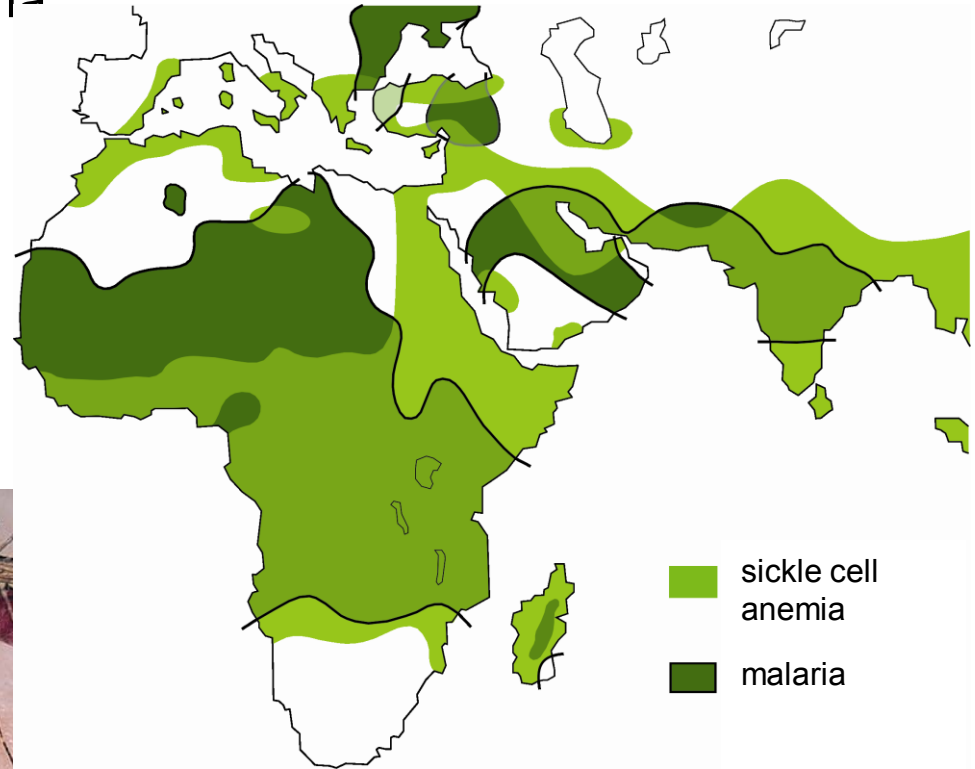
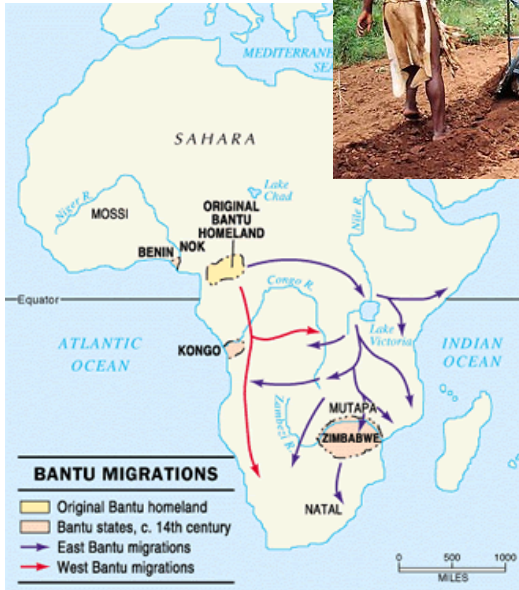


$$W_{AA} < W_{Aa} > W_{aa}$$



Selection maintains **balanced polymorphism**

Eg.: sickle cell anemia and malaria



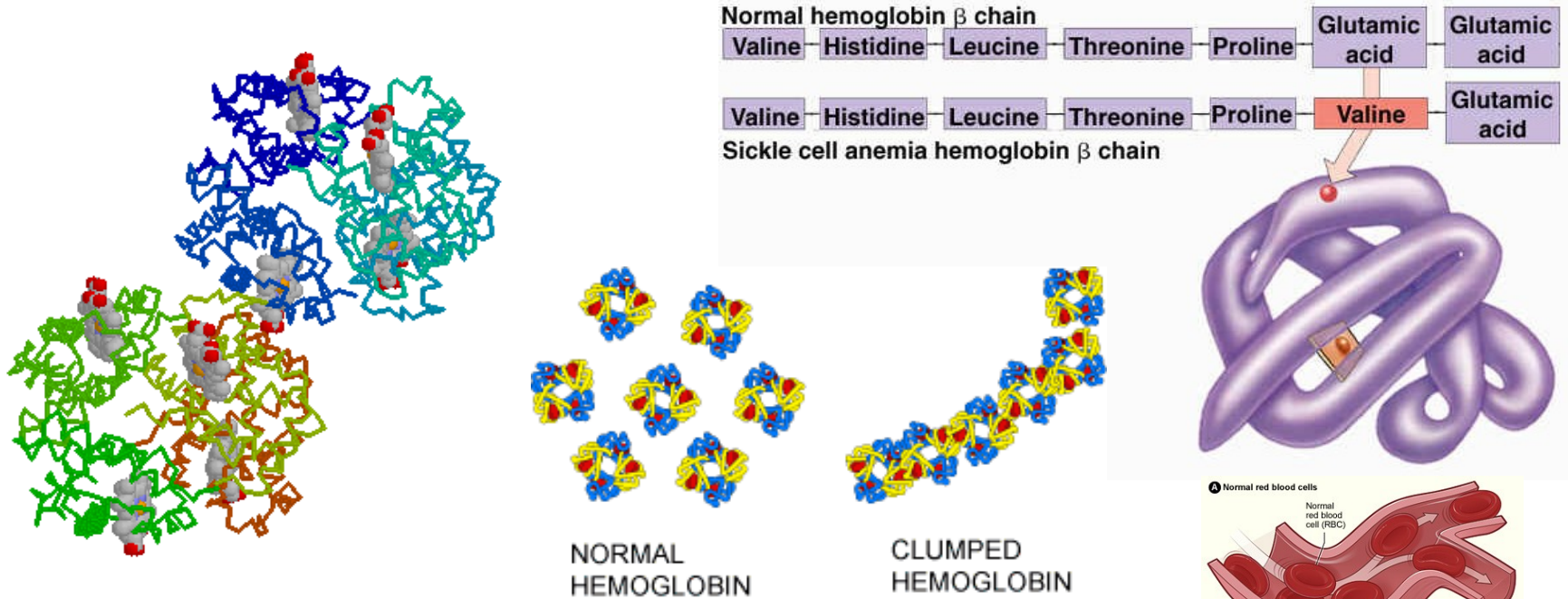
~ 2000 years ago expansion of Bantu peoples

burning off savannas and forests, increase of population density → suitable environment for *Anopheles* mosquitos (*A. gambiae*), the host of *Plasmodium falciparum*

⇒ **malaria**

Sickle cell anemia and malaria:

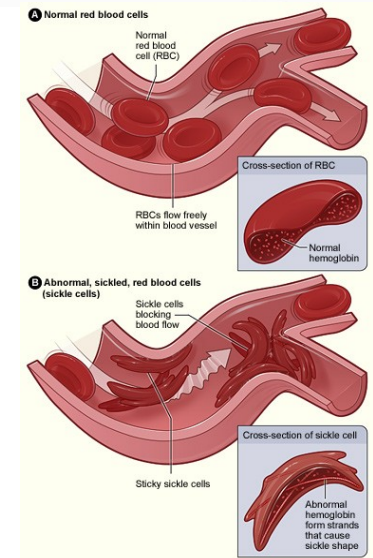
sickle cell anemia: S allele: substitution of 1 AA at 6th position in 6th codon of the β -Hb gene:



at low O_2 concentrations \rightarrow production of elongated crystals

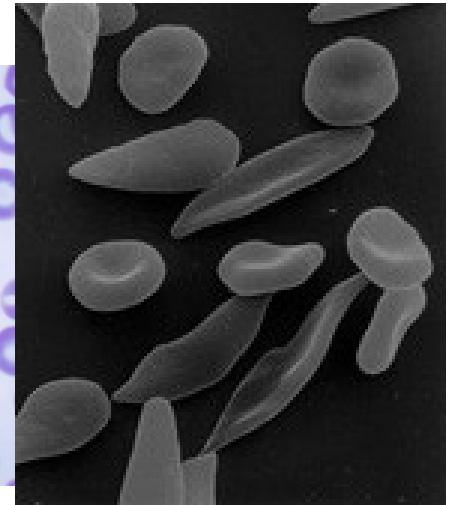
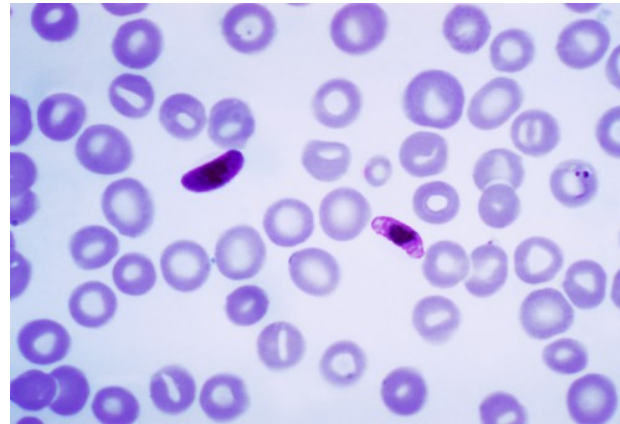
\Rightarrow **anemia**

AS – only transmission of anemia, SS – strong anemia



sickle-cell red blood cell invaded by *Plasmodium* is rapidly breaking \Rightarrow
 the parasite cannot reproduce and multiply \Rightarrow **resistance**

\rightarrow **heterozygote advantage**



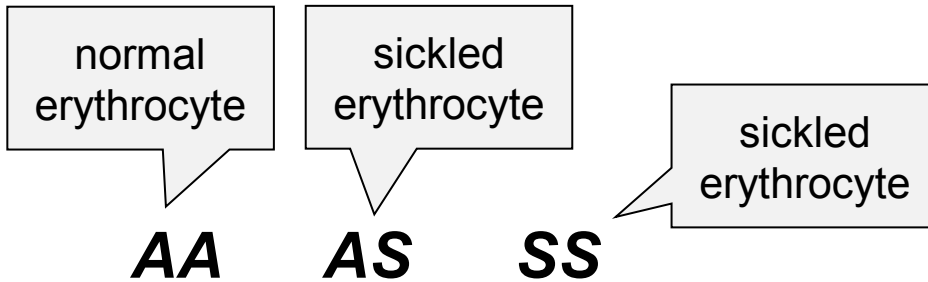
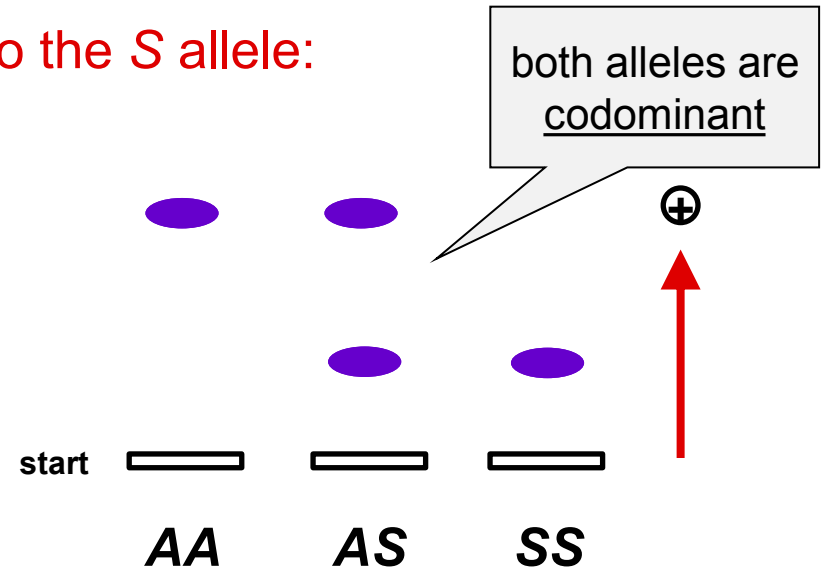
Relative fitness of genotypes related to sickle cell anemia:

Table 11.1. Phenotypic Attributes and Relative Fitnesses (Viabilities) of Six Genotypes Formed by *A*, *S*, and *C* Alleles at β -*Hb* Locus in Humans in Wet, Tropical Africa

Genotype	Phenotypic Attributes	Fitness in Nonmalarial Environment	Fitness in Malarial Environment
AA	Malarial susceptibility	1.00	0.89
SS	Hemolytic anemia	0.20	0.20

Phenotypes related to the S allele:

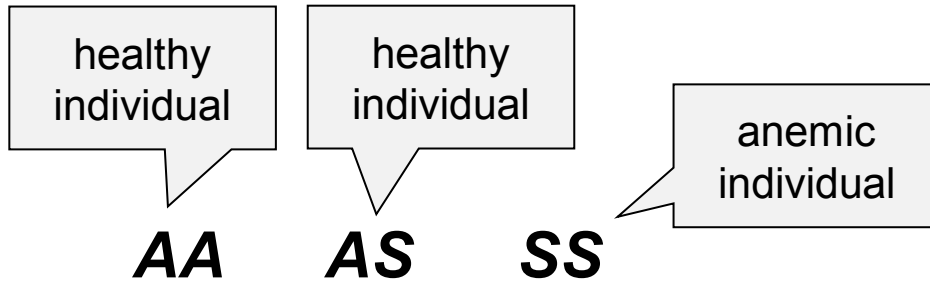
1. Electrophoretic mobility



2. Sickling

sickling in SS and AS individuals \Rightarrow with respect to deformation S dominant

Phenotypes related to the S allele:



3. Anemia

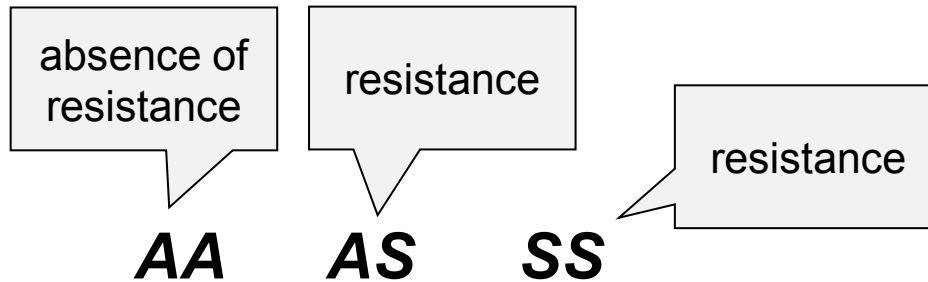
in **SS** individuals longer chains \Rightarrow stronger deformation of red blood cells
 \Rightarrow more fatal impacts on the organism: erythrocyte rupture (anemia),
clogging of capillaries etc.

clinical syndromes only in **SS** \Rightarrow S allele recessive

Phenotypes related to the S allele:

4. Resistance to malaria

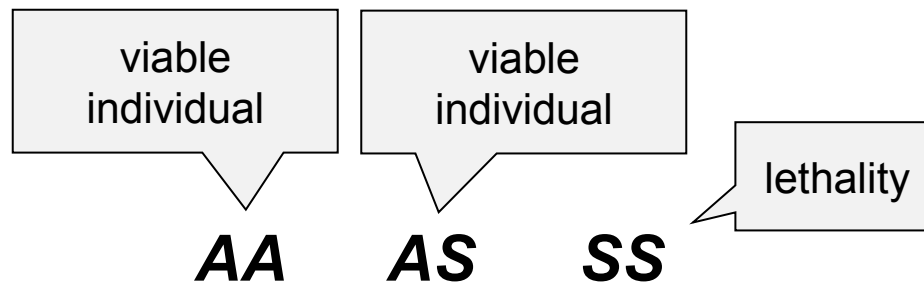
with respect to resistance the S allele dominant



Phenotypes related to the S allele:

5. Phenotype of health (viability)

nonmalarial environment: S recessive



malarial environment: SS – strong anemia; AA – malaria; AS – no anemia, weak malaria \Rightarrow S is overdominant

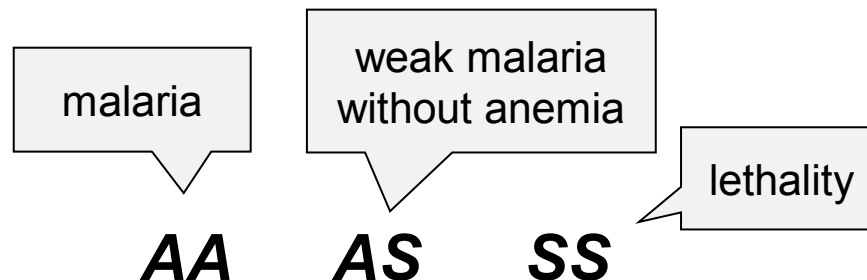


Table 11.1. Phenotypic Attributes and Relative Fitnesses (Viabilities) of Six Genotypes Formed by A, S, and C Alleles at β -Hb Locus in Humans in Wet, Tropical Africa

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AA	Malarial susceptibility	1.00	0.89
AS	Malarial resistance	1.00	1.00
SS	Hemolytic anemia	0.20	0.20

Note: The fitness of the AS heterozygote is set to 1. The malarial fitnesses are estimated from data given in Cavalli-Sforza and Bodmer (1971).

Emergence of C allele in the AS polymorphism region:

possible genotypes: $w_{AC} = 0,89$; $w_{SC} = 0,70$

$w_{AS} = 1,00 \Rightarrow$ selection acts against beneficial allele!

Although C highly beneficial, selection will decrease its frequency until it is completely removed!!

Resistance against malaria can be mediated through other mechanisms:

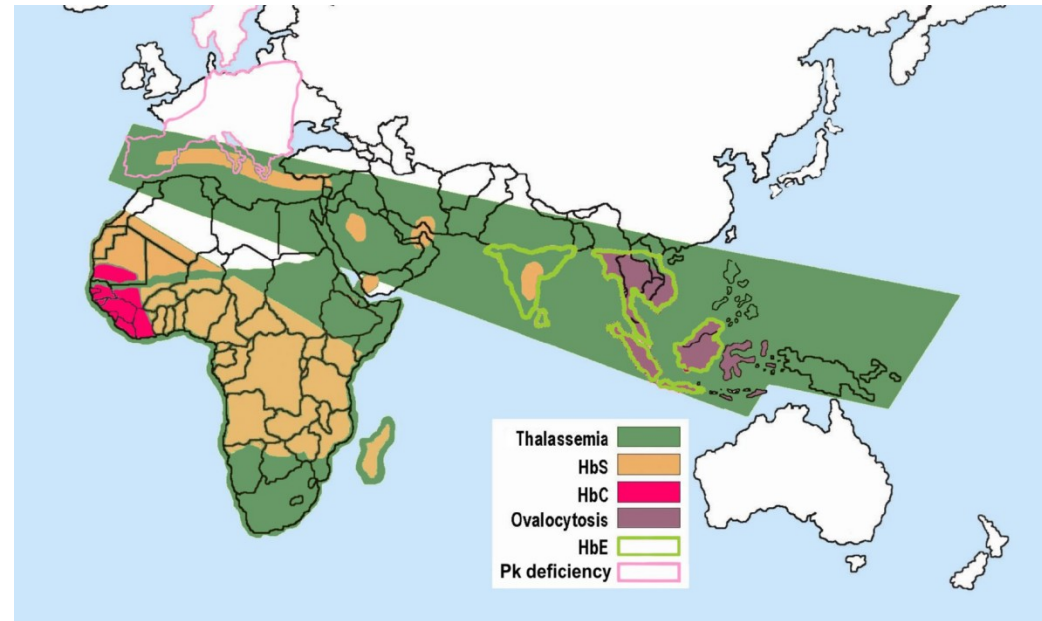
hemoglobin E (JV Asie)

α - a β -thalassemia

G6PD^{*)} deficiency

Pk^{**)} deficiency

etc. etc.



^{*)} glucose-6-phosphate dehydrogenase

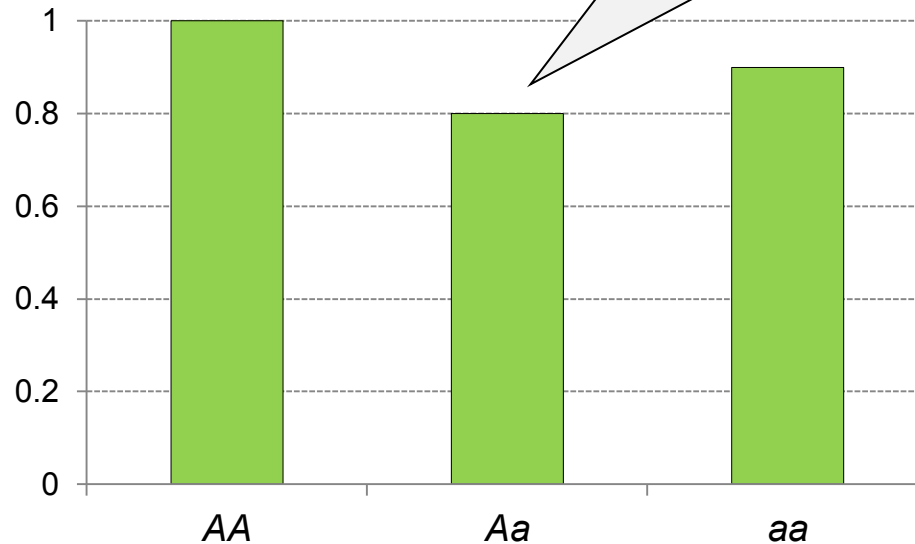
^{**)} pyruvate kinase

However, selection against heterozygotes
not widespread in nature

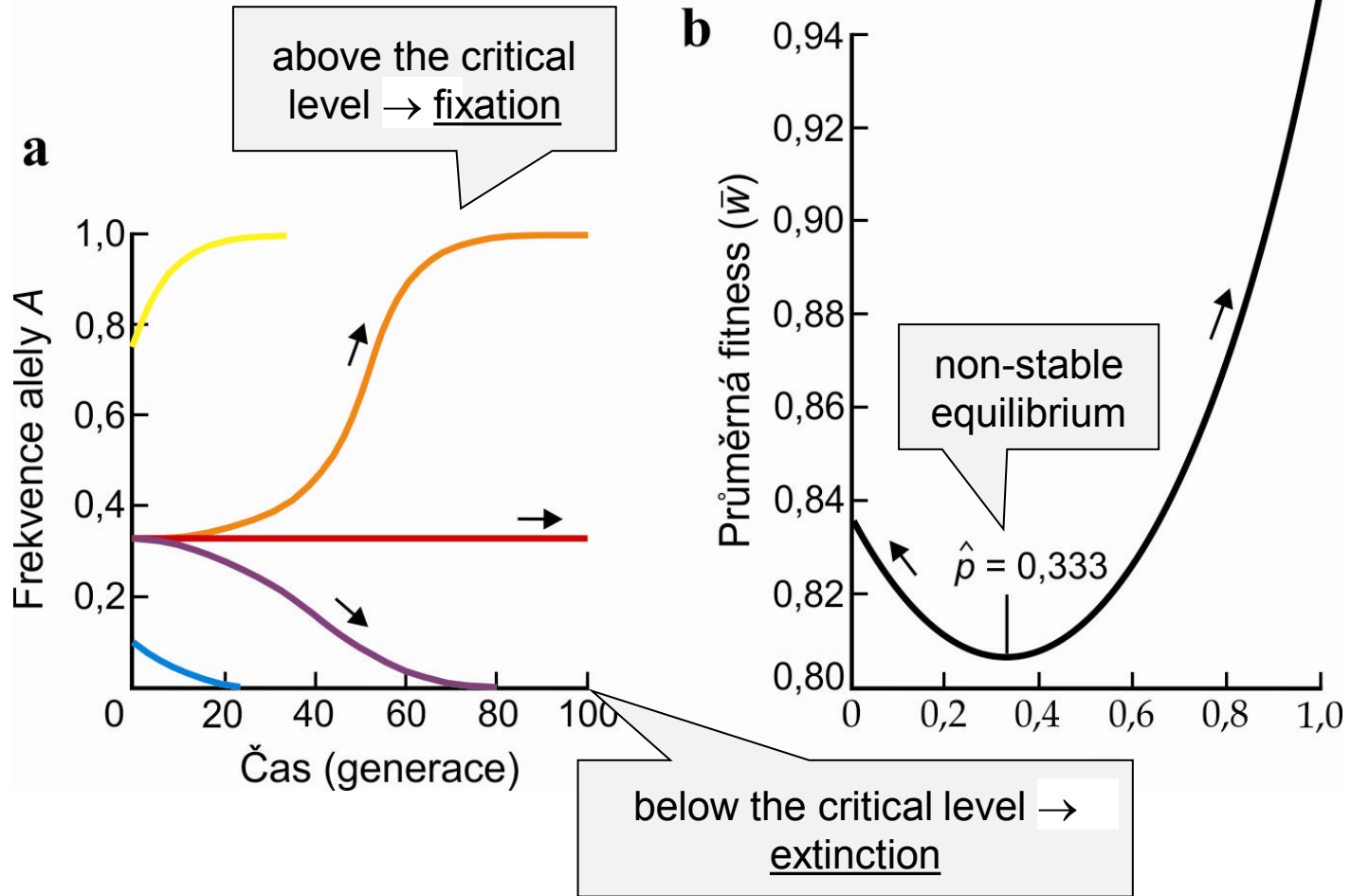
Alternative equilibrium: selection against heterozygotes (underdominance)

$$W_{AA} > W_{Aa} < W_{aa}$$

heterozygote fitness
is lower than w of
homozygotes



$$W_{AA} > W_{Aa} < W_{aa}$$



Selection results in fixation of one of the alleles
(and extinction of the other)

2. Selection in heterogeneous environment

environmental variation:

spatial

temporal

coarse-grained: single environment throughout lifetime

fine-grained: environmental heterogeneity throughout
lifetime

selection: soft

hard

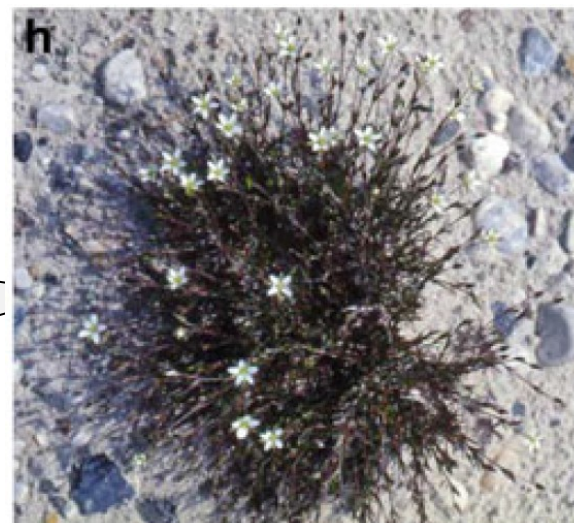
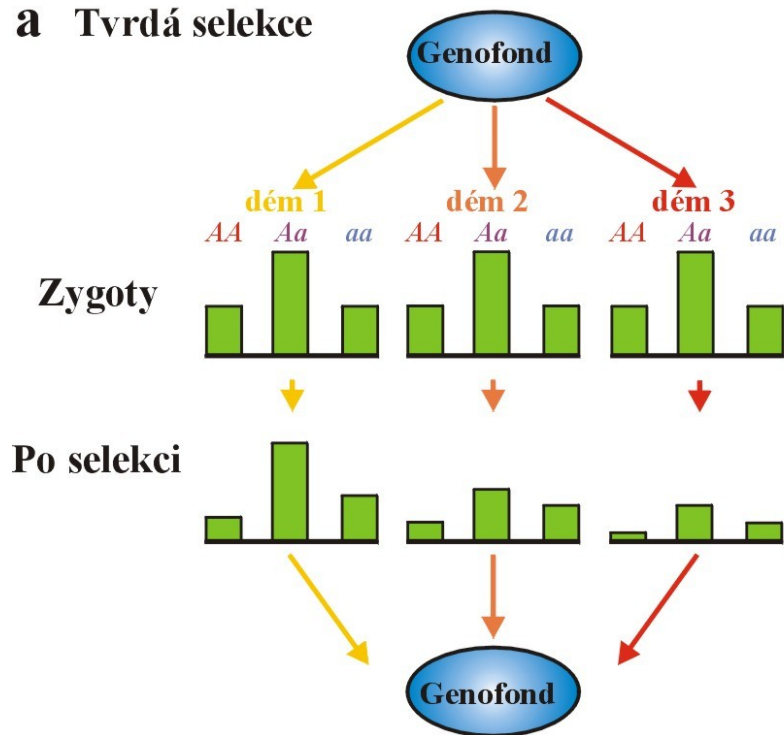
selection

hard

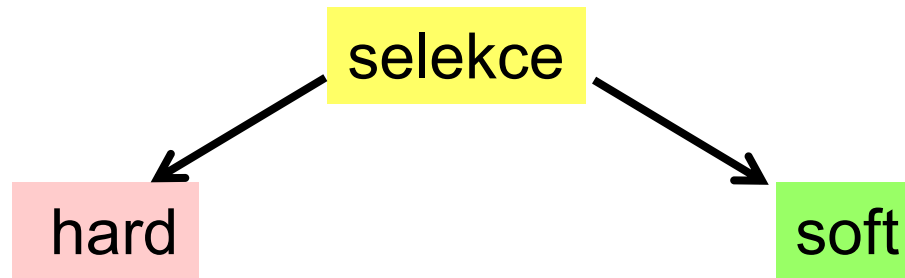
Silene vulgaris
ssp. *humilis*



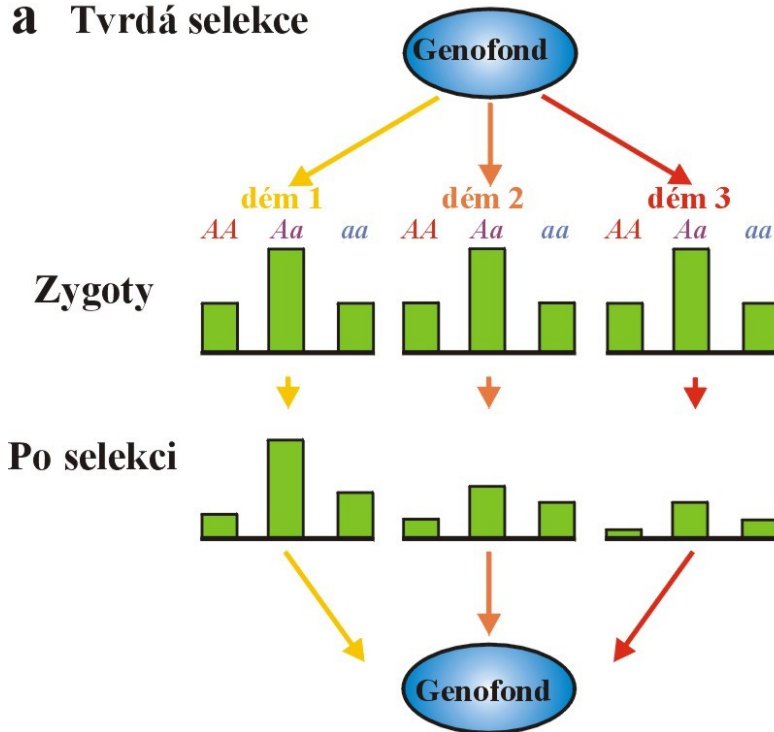
a Tvrdá selekce



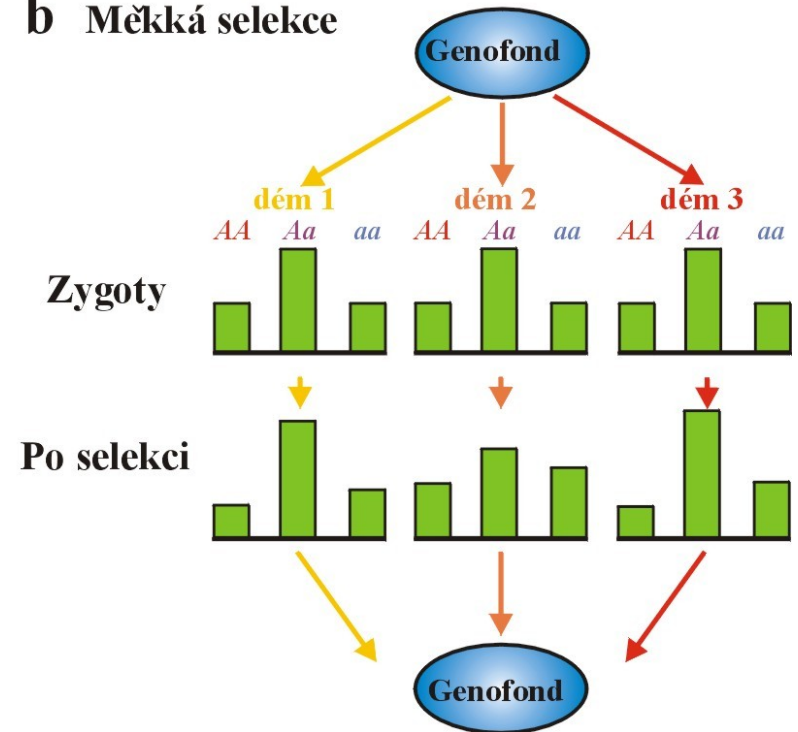
Minuartia
verna



a Tvrdá selekce



b Měkká selekce



Coarse-grained environment and **soft selection** will maintain polymorphism in the population **with higher probability** than fine-grained environment and hard selection.

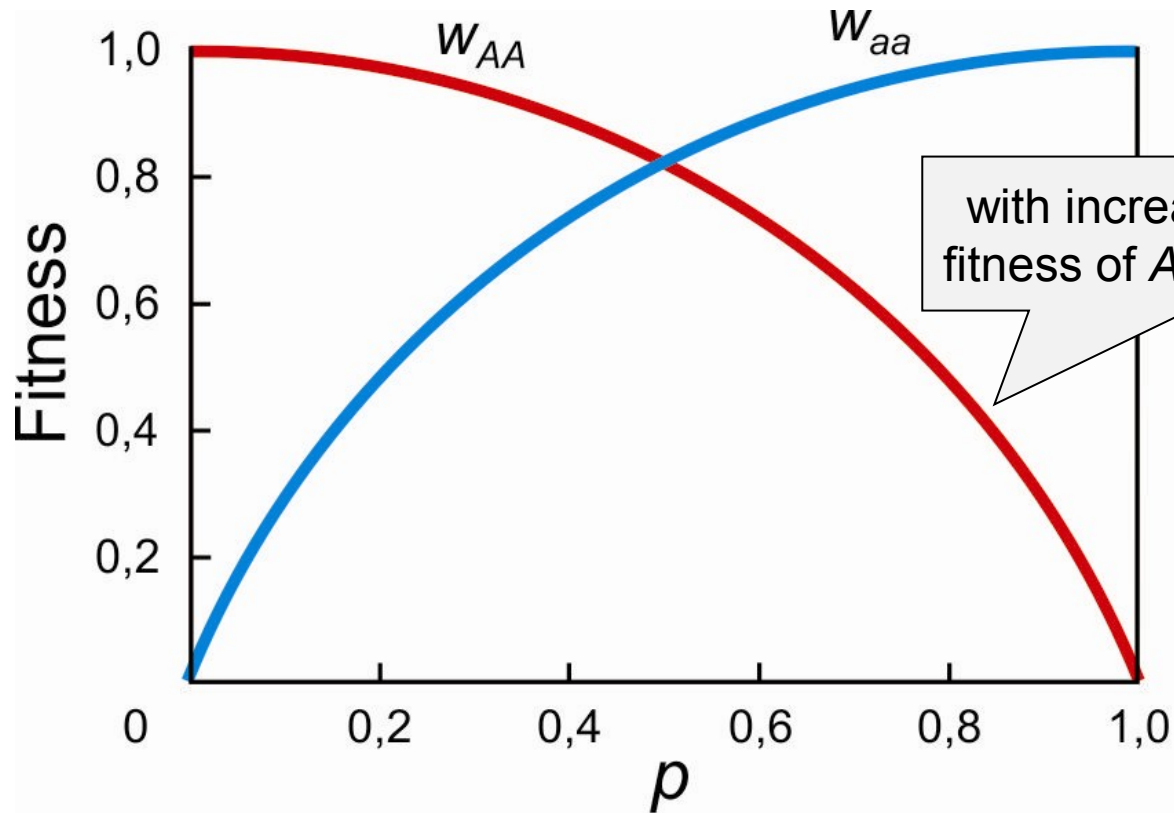
3. Antagonistic selection

different sexes

different ontogenetic stages

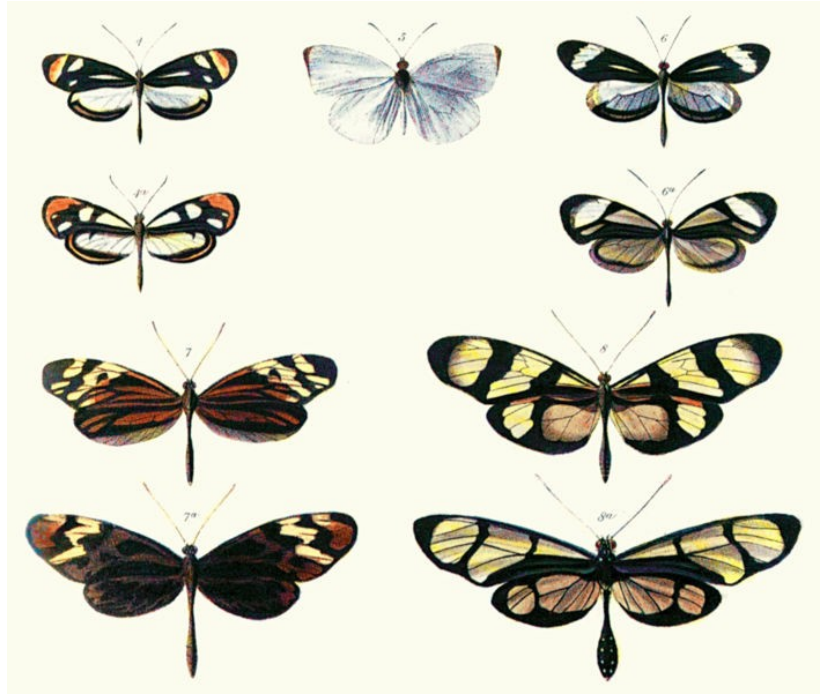
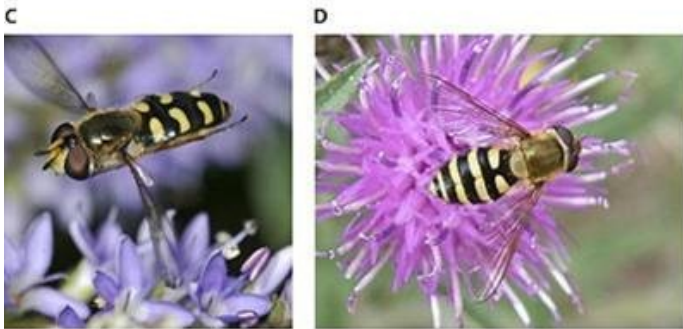
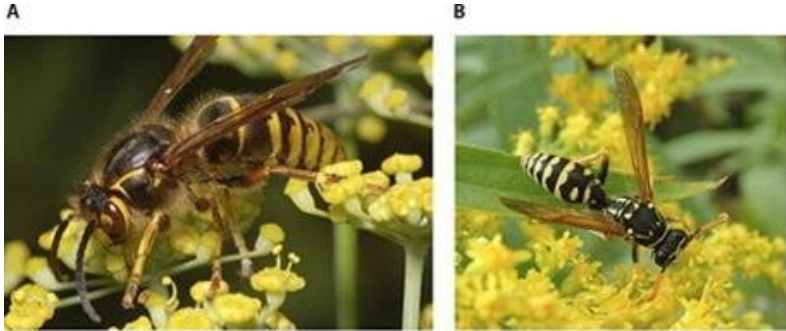
gametic × zygotic phase

4. Frequency-dependent selection I. Negative

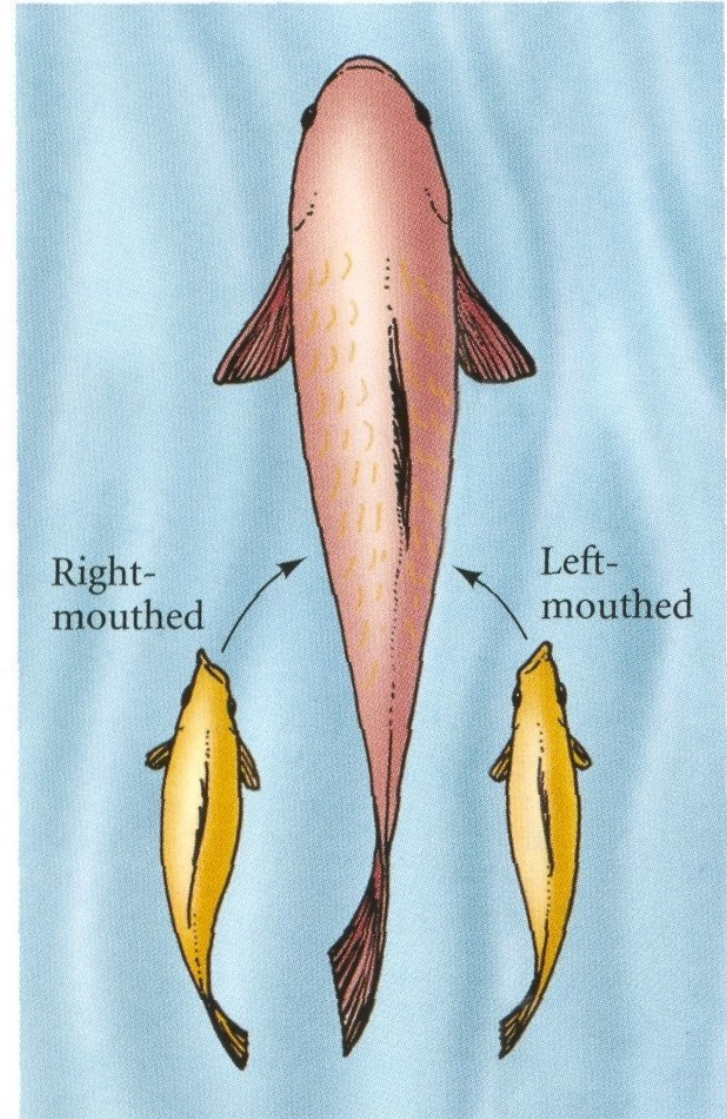
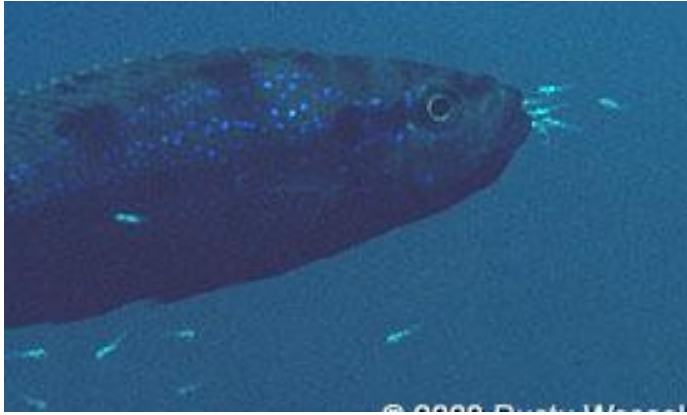


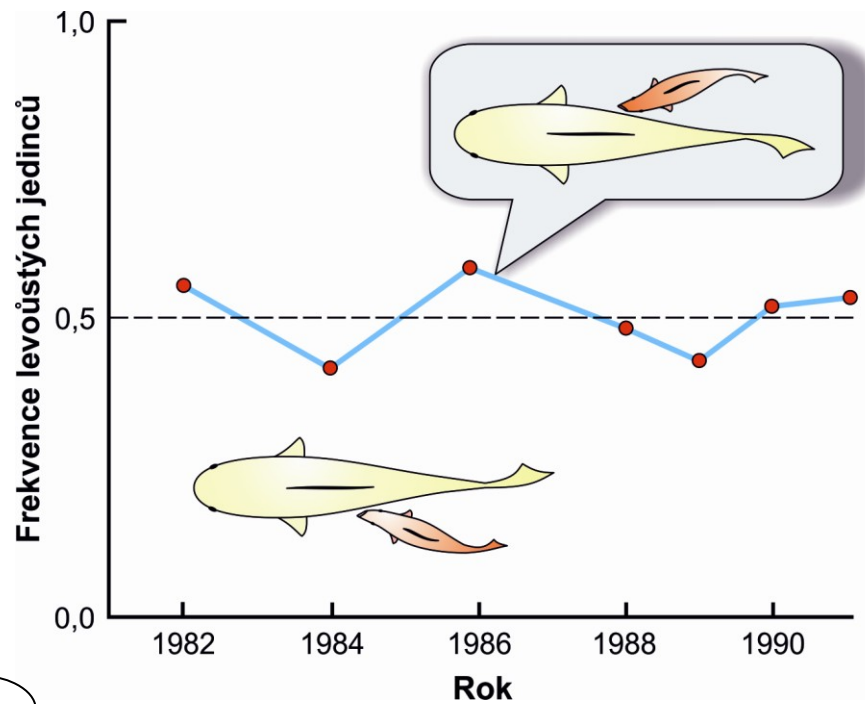
Eg.: Batesian mimicry

[in this case it is rather density-dependent selection]



Eg.: cichlid *Perissodus microlepis* (Tanganyika)



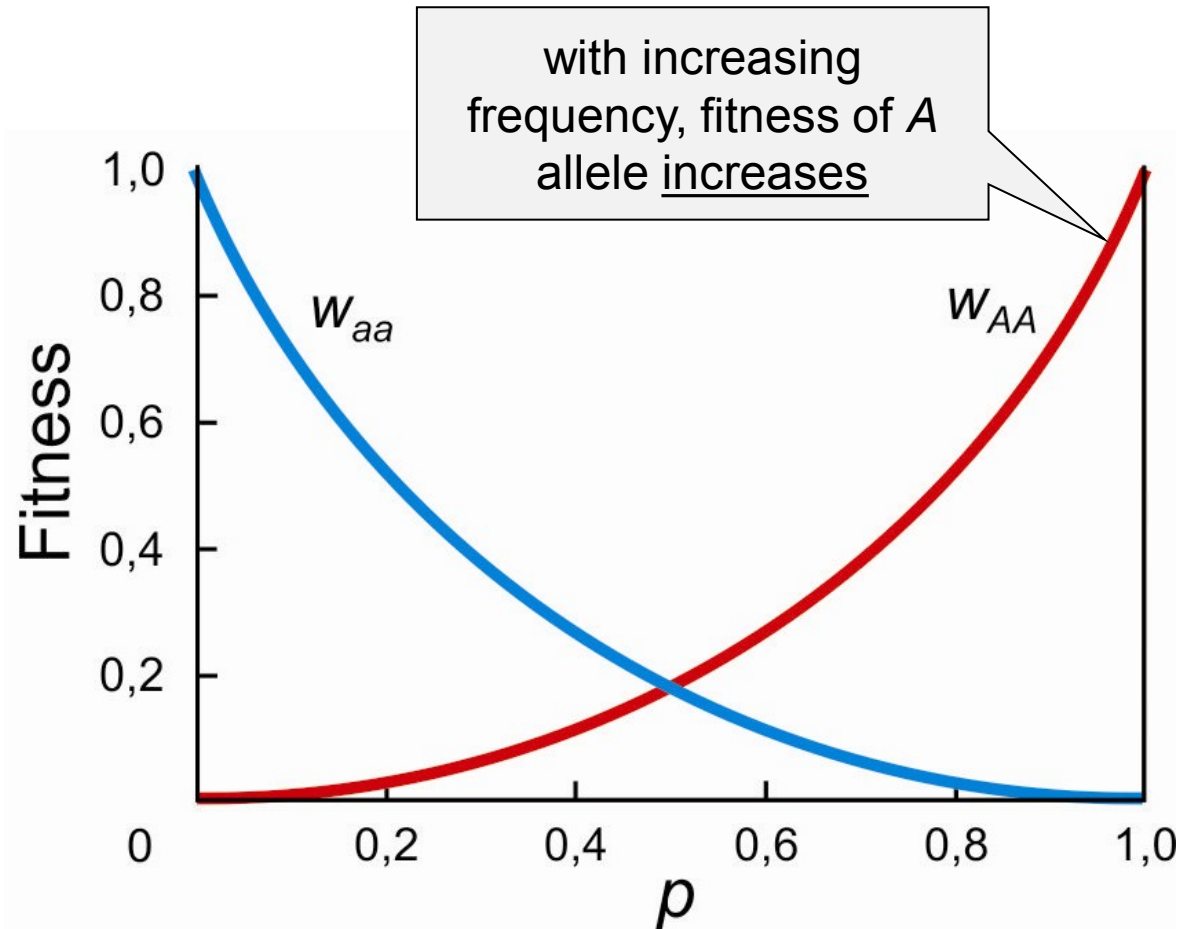


„right-mouthed“



„left-mouthed“

4. Frequency-dependent selection II. Positive



Müllerian mimicry:

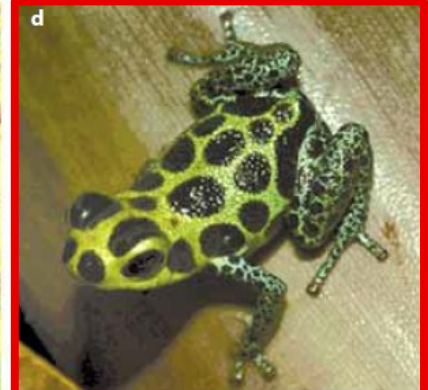
Amereega hahneli
(poisonous)



Lithodytes lineatus
(harmless; Batesian mimicry)



R. variabilis
(poisonous)

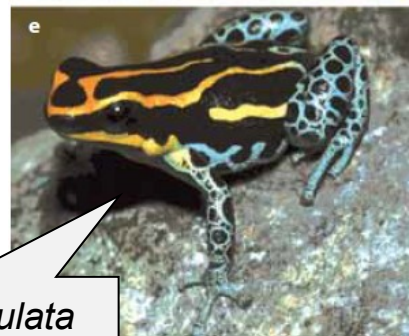


Heliconius melpomene



H. erato

R. ventrimaculata
(poisonous)

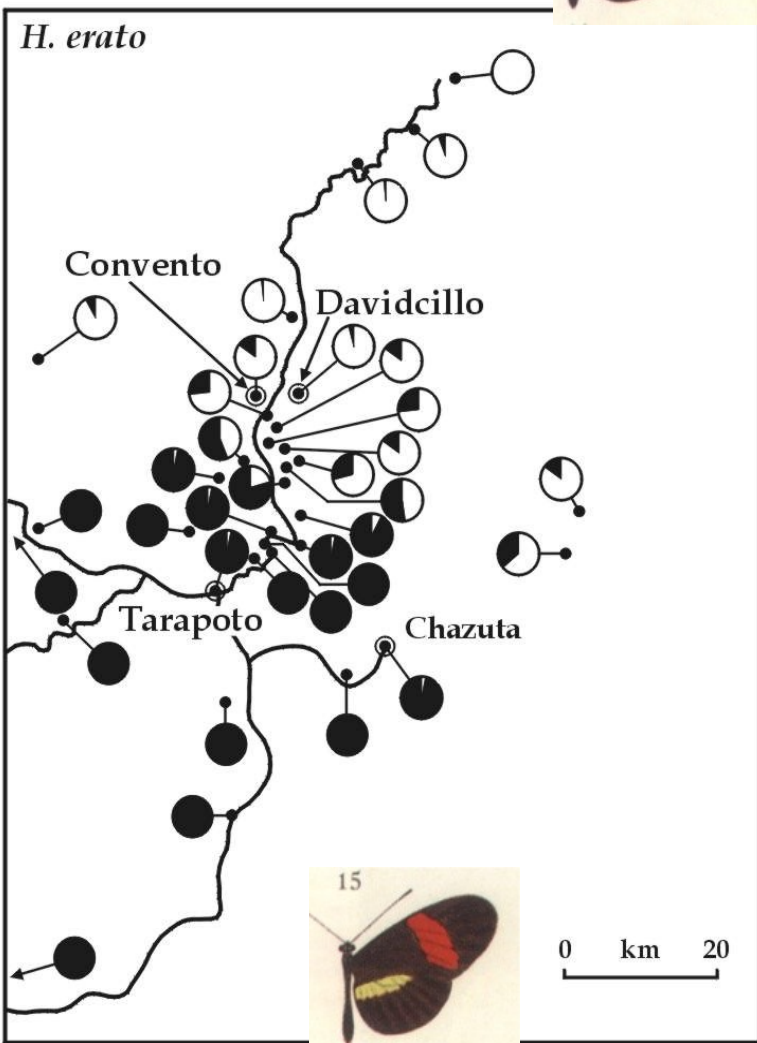


Ranitomeya imitator
(d,f; poisonous)

Heliconius erato



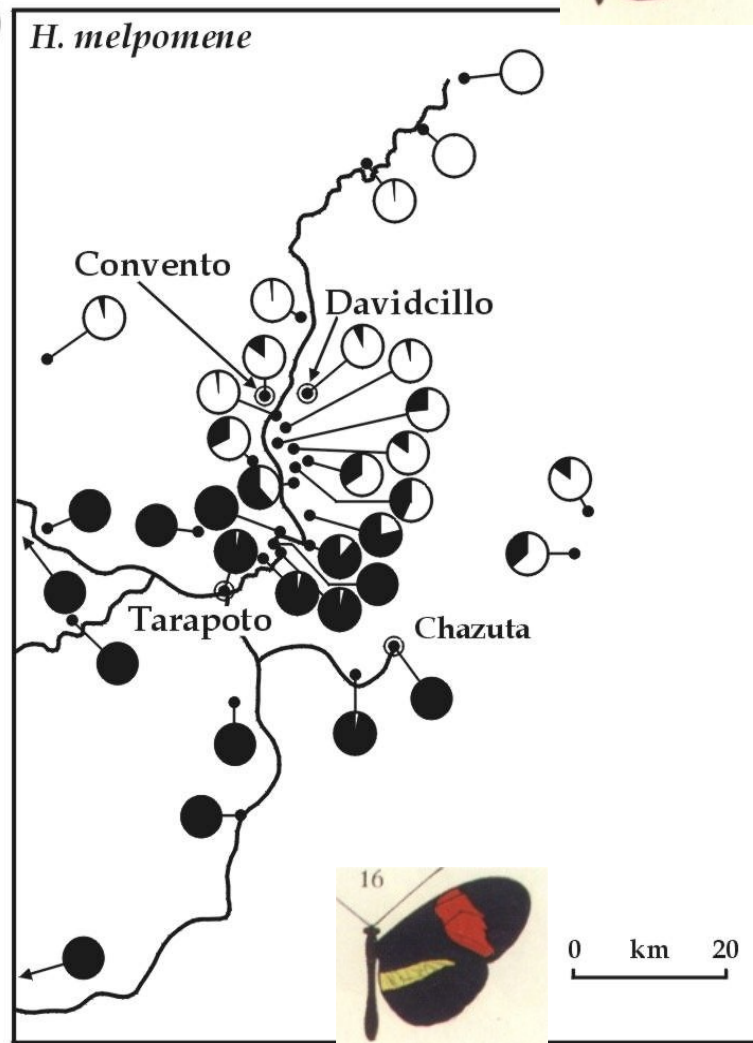
(a)



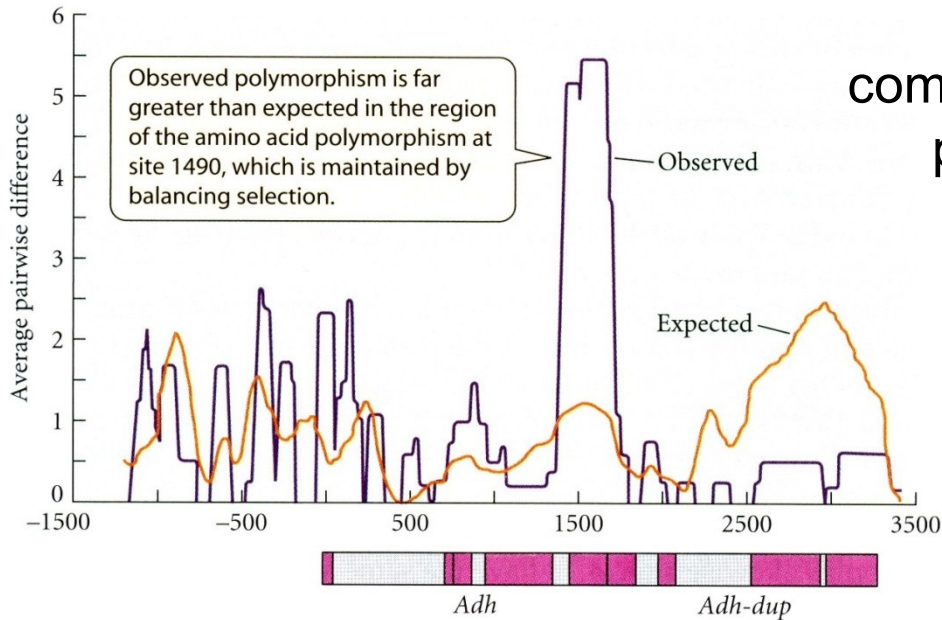
H. melpomene



(b)



Balancing selection at the molecular level:



comparison of observed and expected polymorphism in the ADH gene

chimpanzee alleles (C) more similar to human alleles (H) than other C alleles

MHC genes

