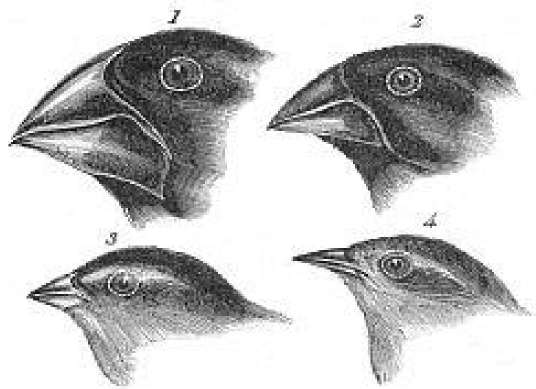
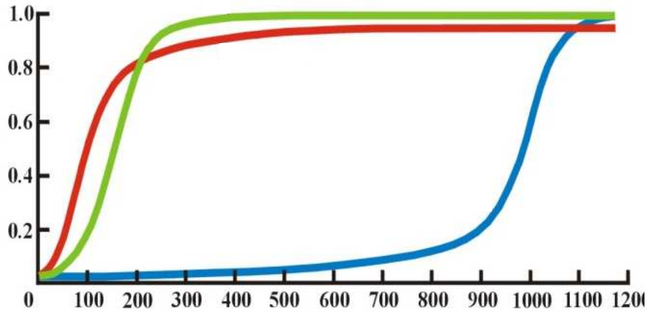
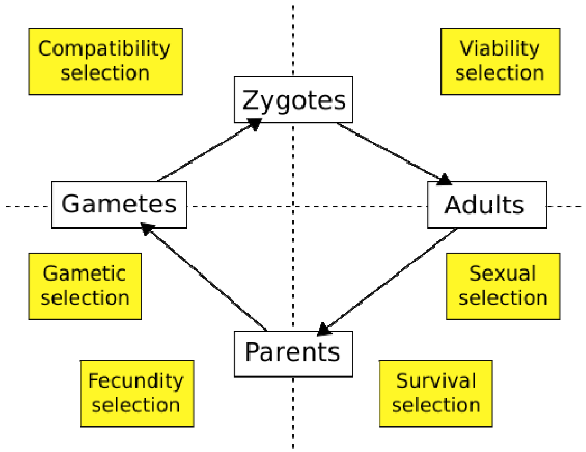
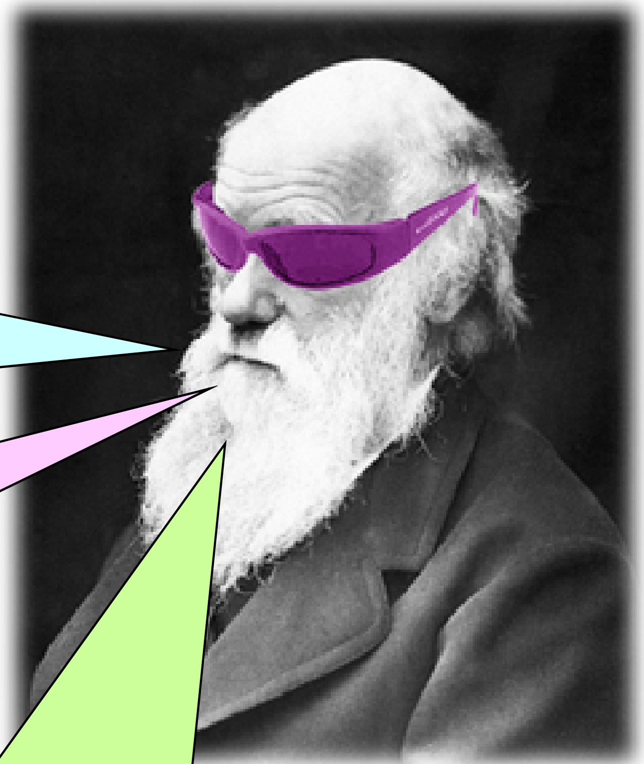
NATURAL SELECTION







Evolution by natural selection:



All organisms produce more offspring than can survive and reproduce.

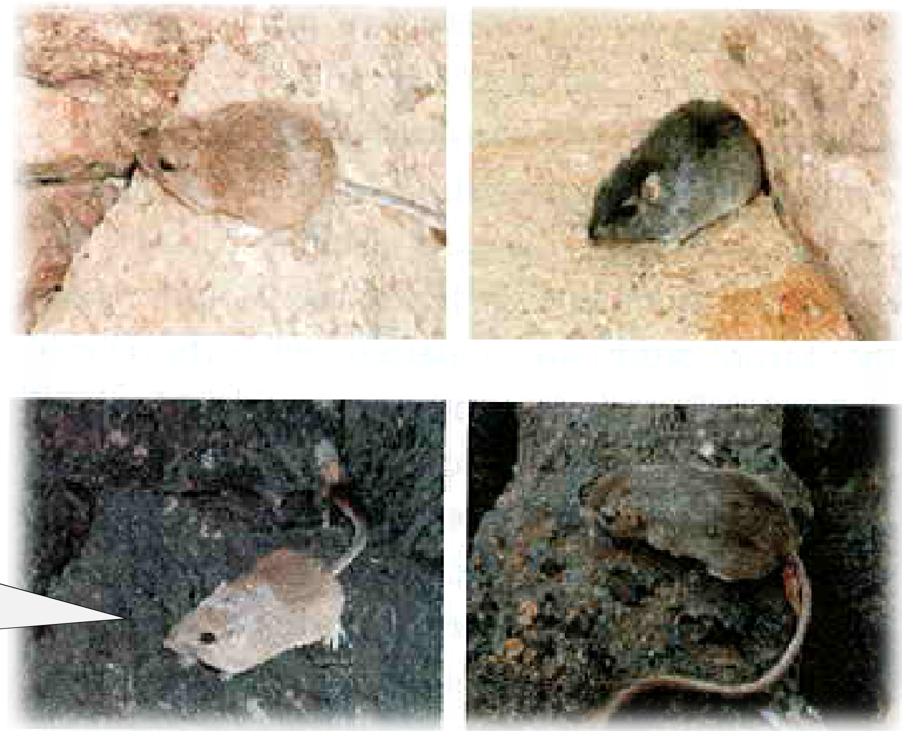
Individuals (genotypes) differ in heritable traits related to survival and reproduction.

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

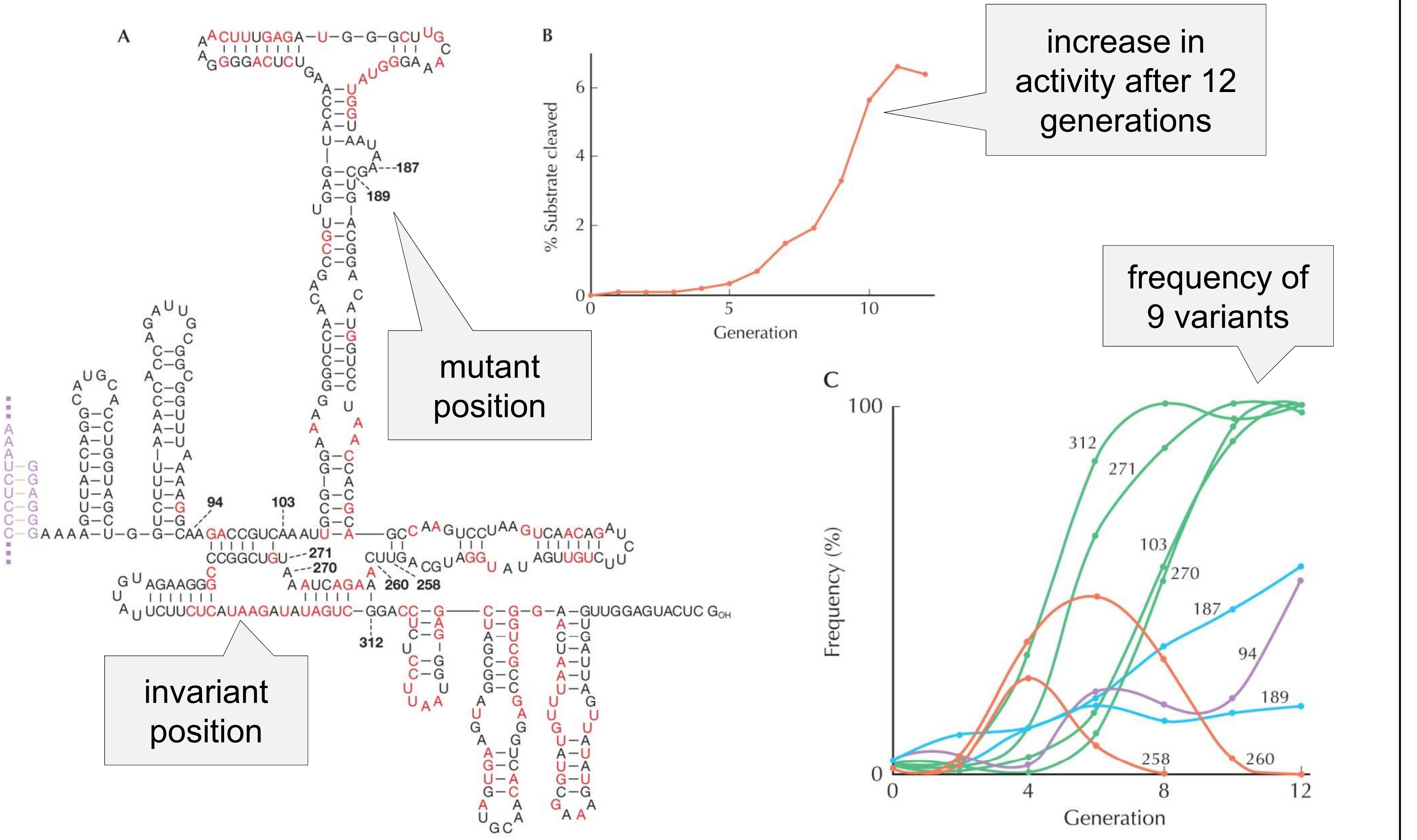
DD, *Dd*



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 → 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 → growth rate ≈ 0

in evolution relationships between genotypes in a population more important → relative fitness

discrete time → = ratio of absolute fitness; continuous time → = 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

- Viability
  - Resistance to pathogens
  - Escape from predators
- Reproductive success
  - Frequency of ovulations
  - Survival of embryos
- Litter size
- Litter frequency
- Number of litters

Quality of the weaned offspring

- 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*) ≈ 0,20

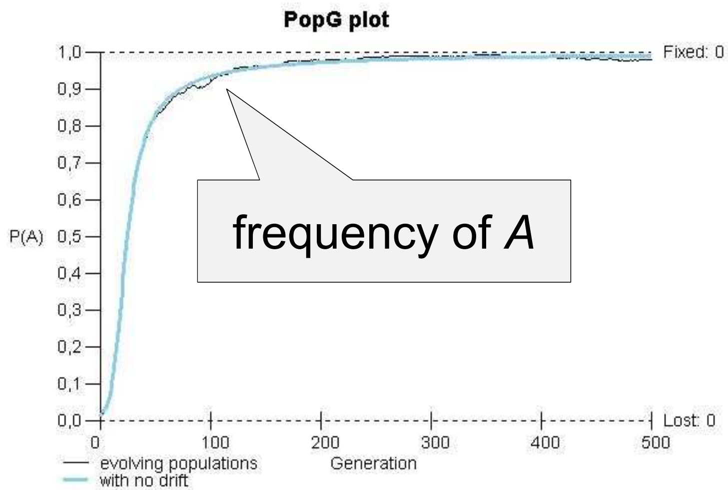
w = 0,60–0,98
⇒ *s* = 0,02–0,40

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

Increase of beneficial dominant allele *A*:

$$\Delta p = p' - p = \frac{pq^2 s}{1-q^2 s}$$

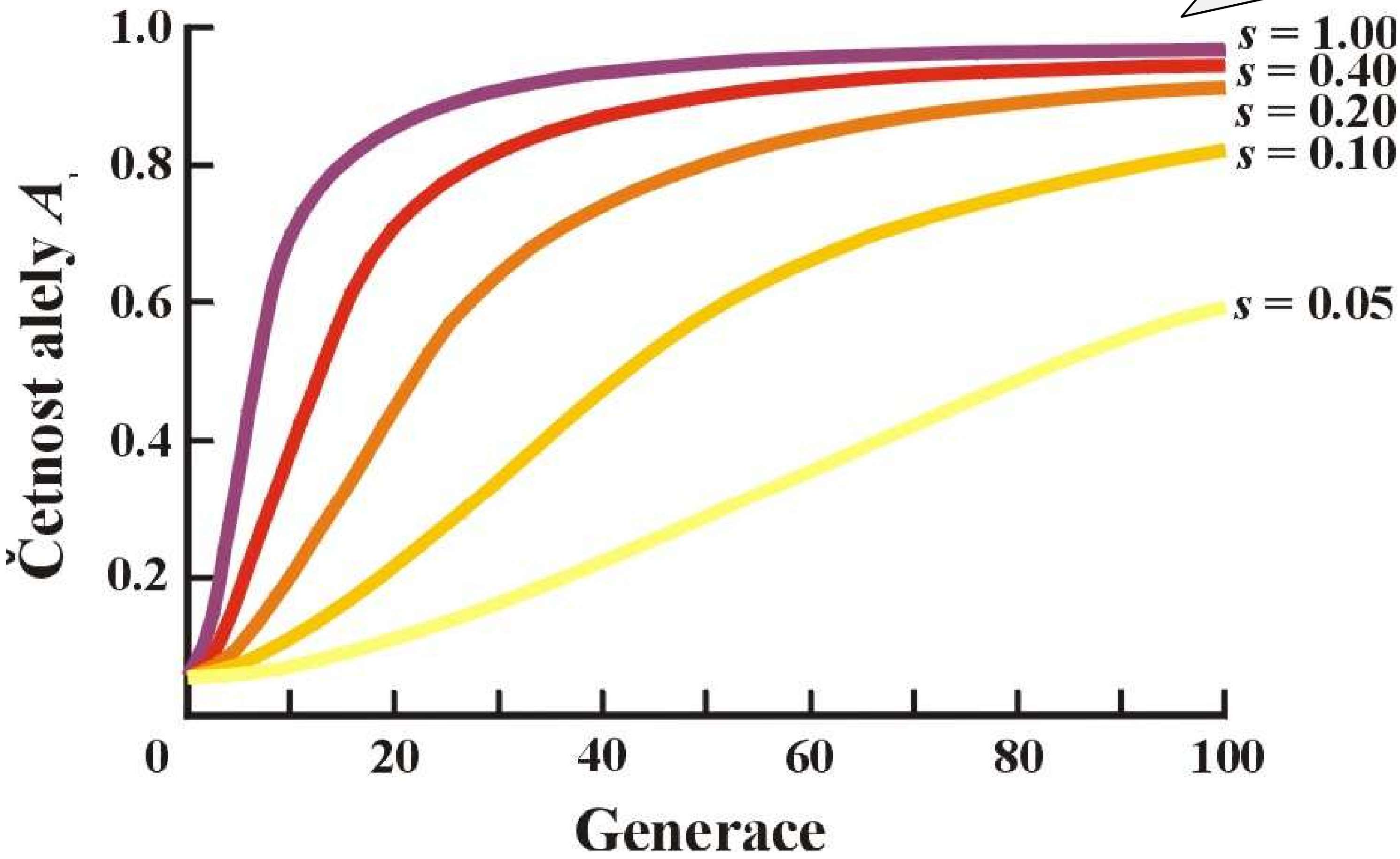
negatively proportional to the mean fitness of the population ⇒ with increasing frequency of the beneficial allele the evolution is slowing down; if *q* or *p* = 0 Δp = 0



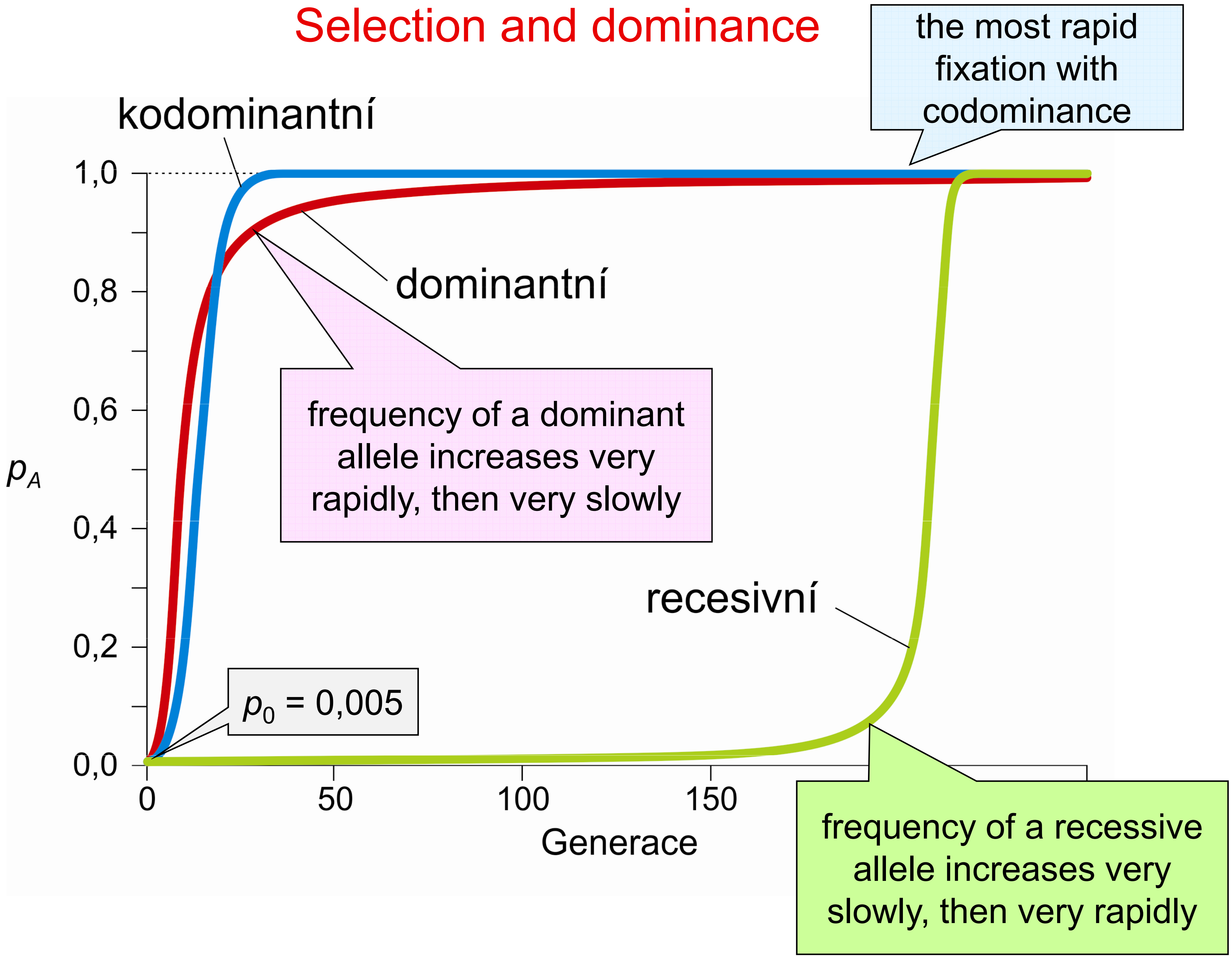
When *s* = 0,20 and initial frequency *A* = 0,01 the freq. increases to 0,95 in ca. 120 generations

Increase of a advantageous dominant allele *A*:

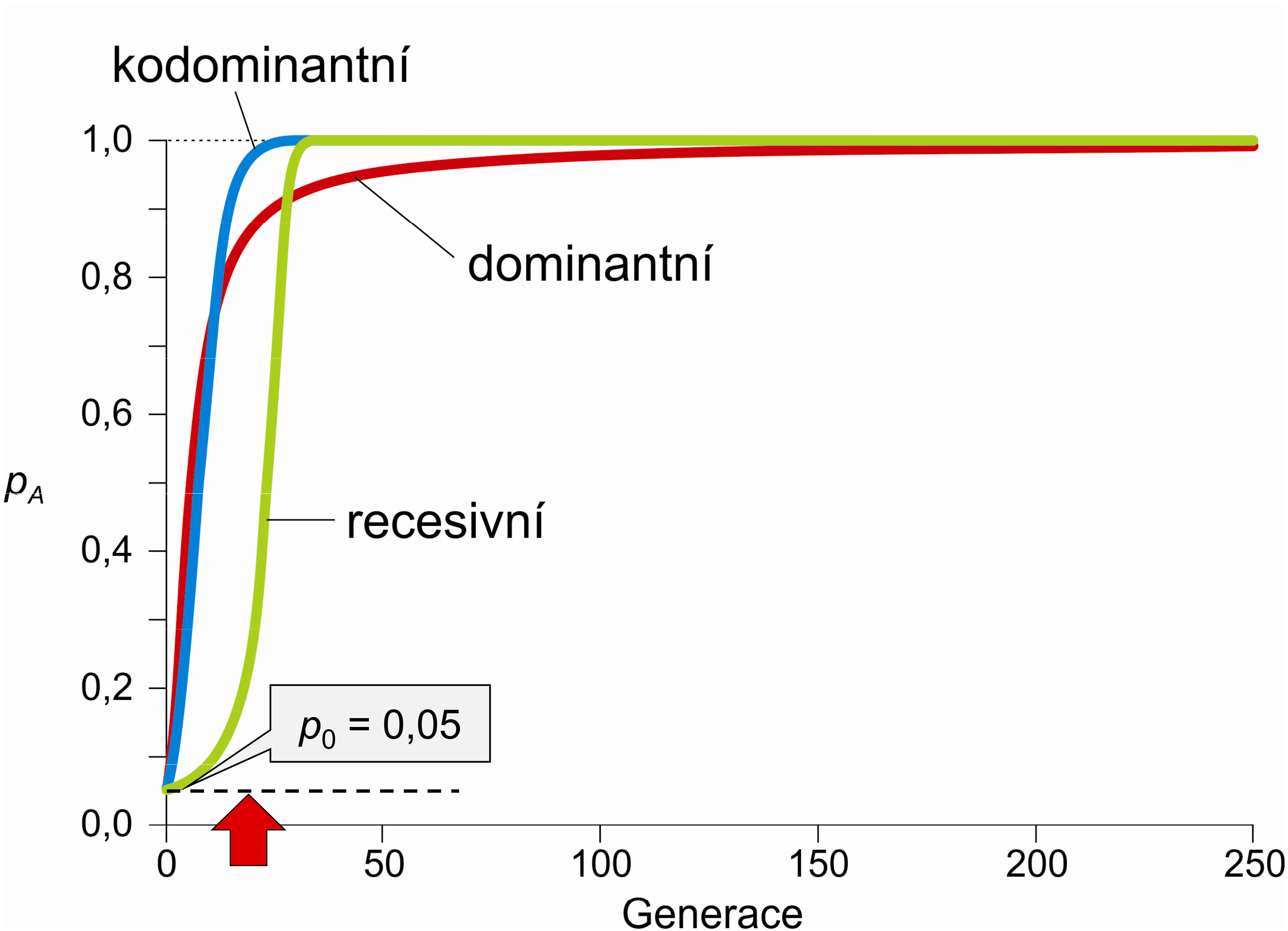
the higher *s* (ie. stronger selection), the more rapid change



Selection and dominance



Effect of the initial allele frequency:



STUDY OF NATURAL SELECTION:

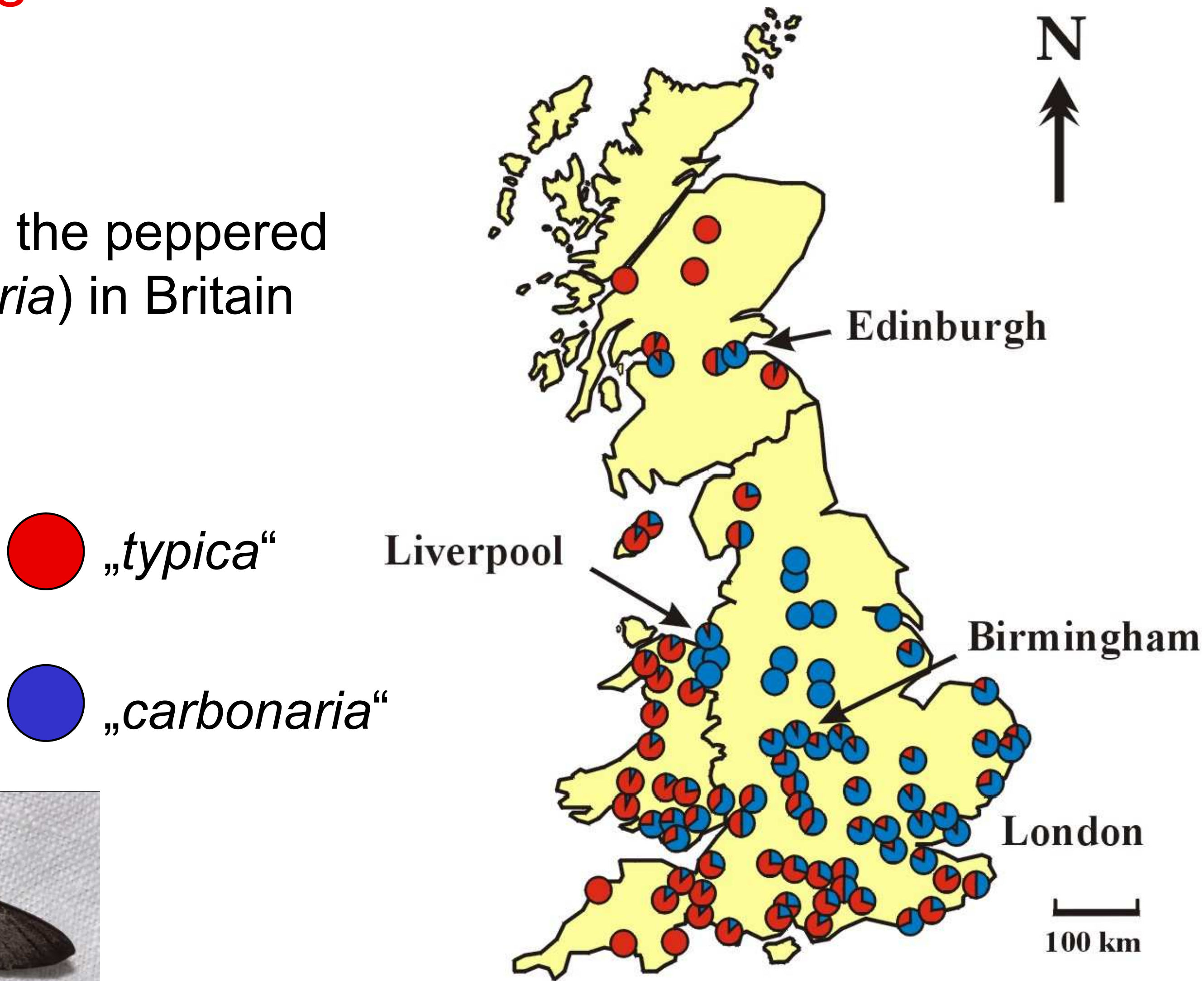
1. Correlation of allele frequencies across populations

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



4. Experimental evidence:

H.B.D. Kettlewell

Bernard Kettlewell

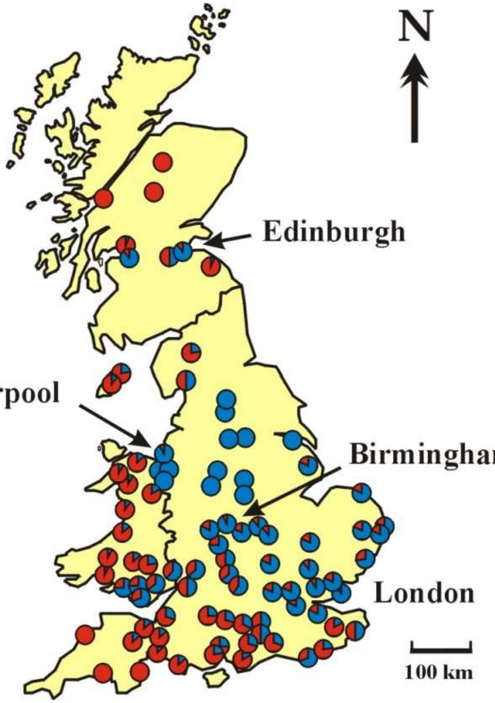
Light and dark Peppered moths

polluted area

light

dark

Birmingham (znečištěná oblast)	Světlá forma (*typica*)	Tmavá forma (*carbonaria*)
Počet zpětně odchycených:		
pozorovaný	18	140
očekávaný	36	122
Relativní míra přežívání	0,5	1,15
Relativní fitness	0,5/1,15 = 0,43	1,15/1,15 = 1
Deanend Wood (neznečištěná oblast)	**Světlá forma (*typica*)**	**Tmavá forma (*carbonaria*)**
Počet zpětně odchycených:		
pozorovaný	67	32
očekávaný	53	46
Relativní míra přežívání	1,26	0,69
Relativní fitness	1,26/1,26 = 1	0,69/1,26 = 0,55



non-polluted area

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

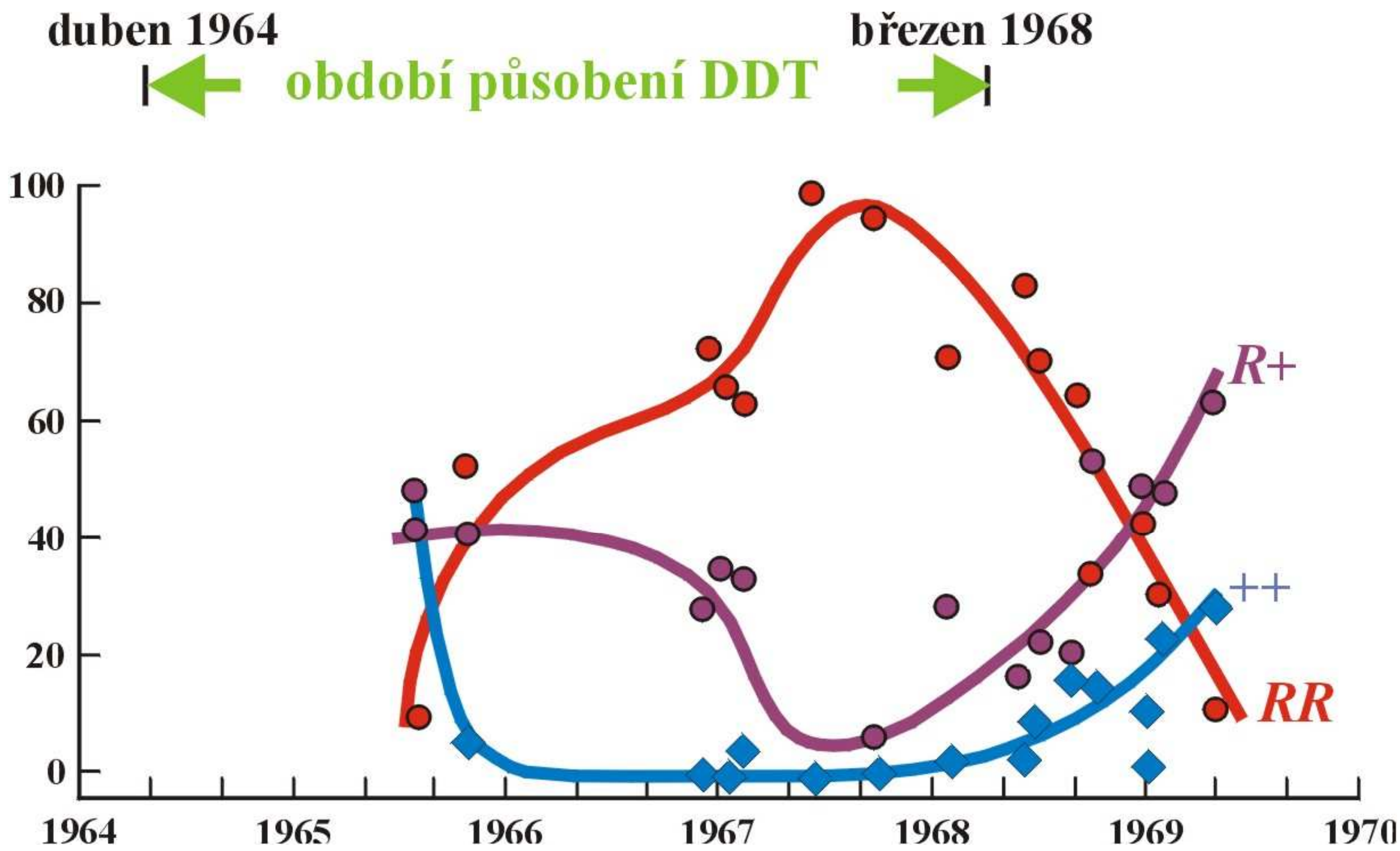
errors in the experiment:
during the day, peppered moths stay on horizontal branches, not on trunks (different lichen species); in butterflies and birds different perception of UV

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)

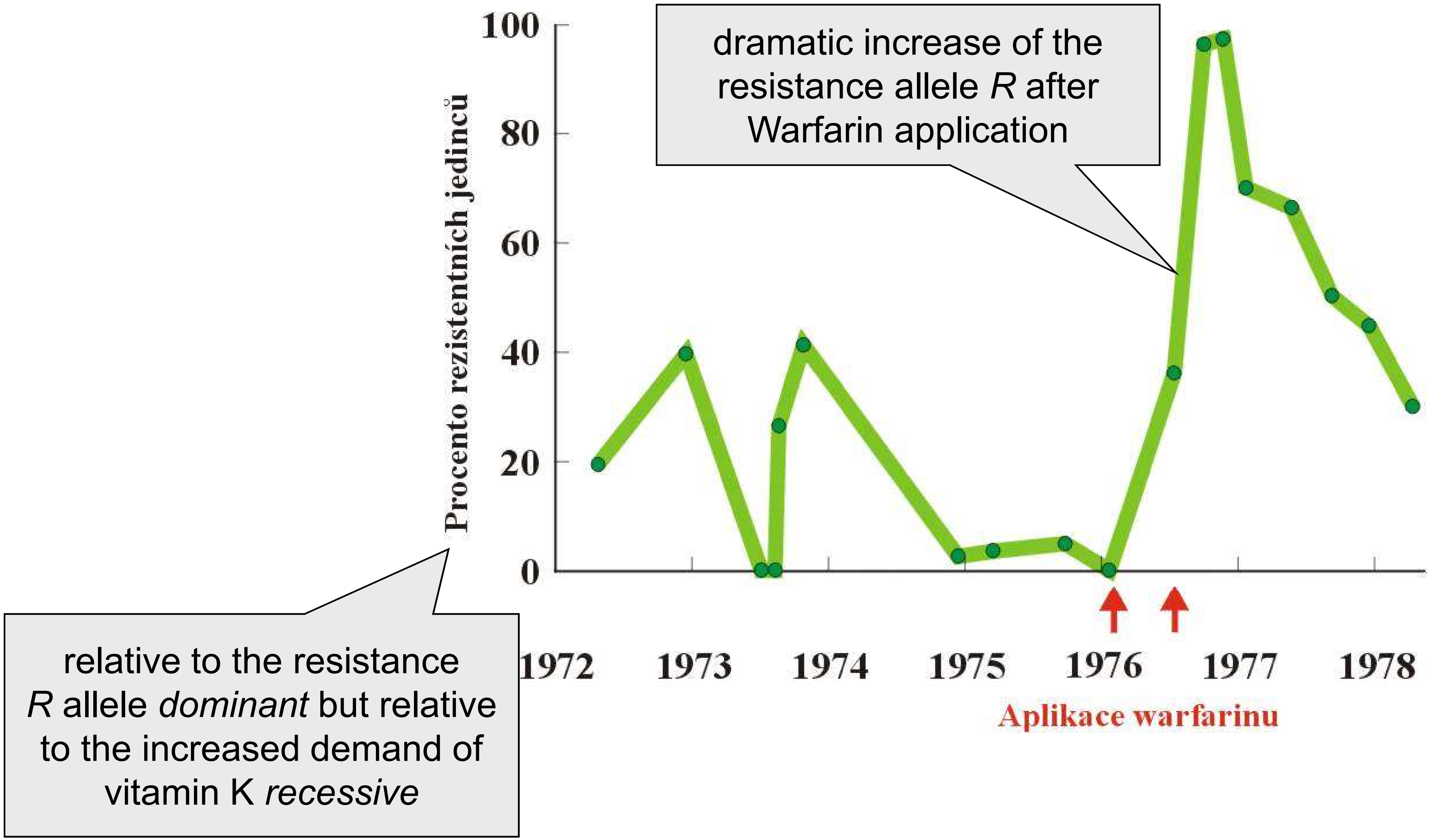
5. Resistance

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



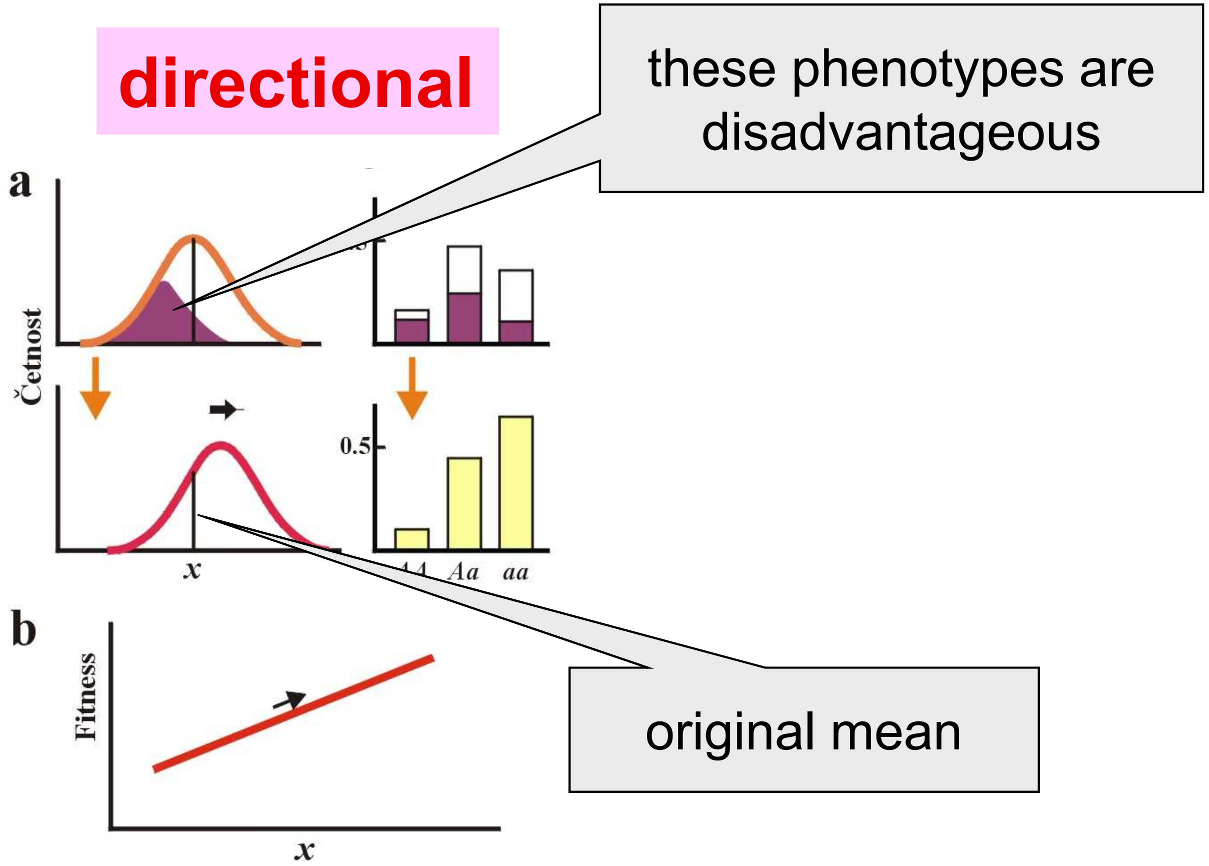
eg.: Warfarin resistance in rats:

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



Relationship between phenotype and fitness: basic selection regimes

directional



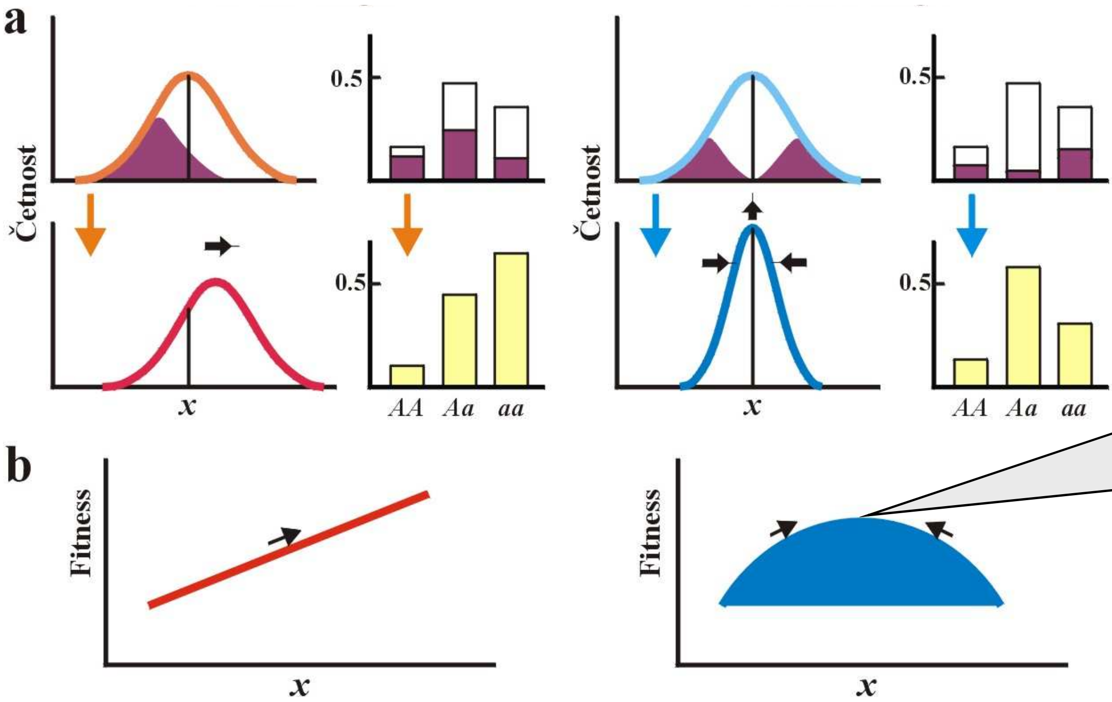
consistent change of environment

shift of the mean

variance unchanged

Relationship between phenotype and fitness: basic selection regimes

directional | **stabilizing**



highest fitness in individuals with intermediate (mean) phenotypes

consistent change of environment

shift of the mean

variance unchanged

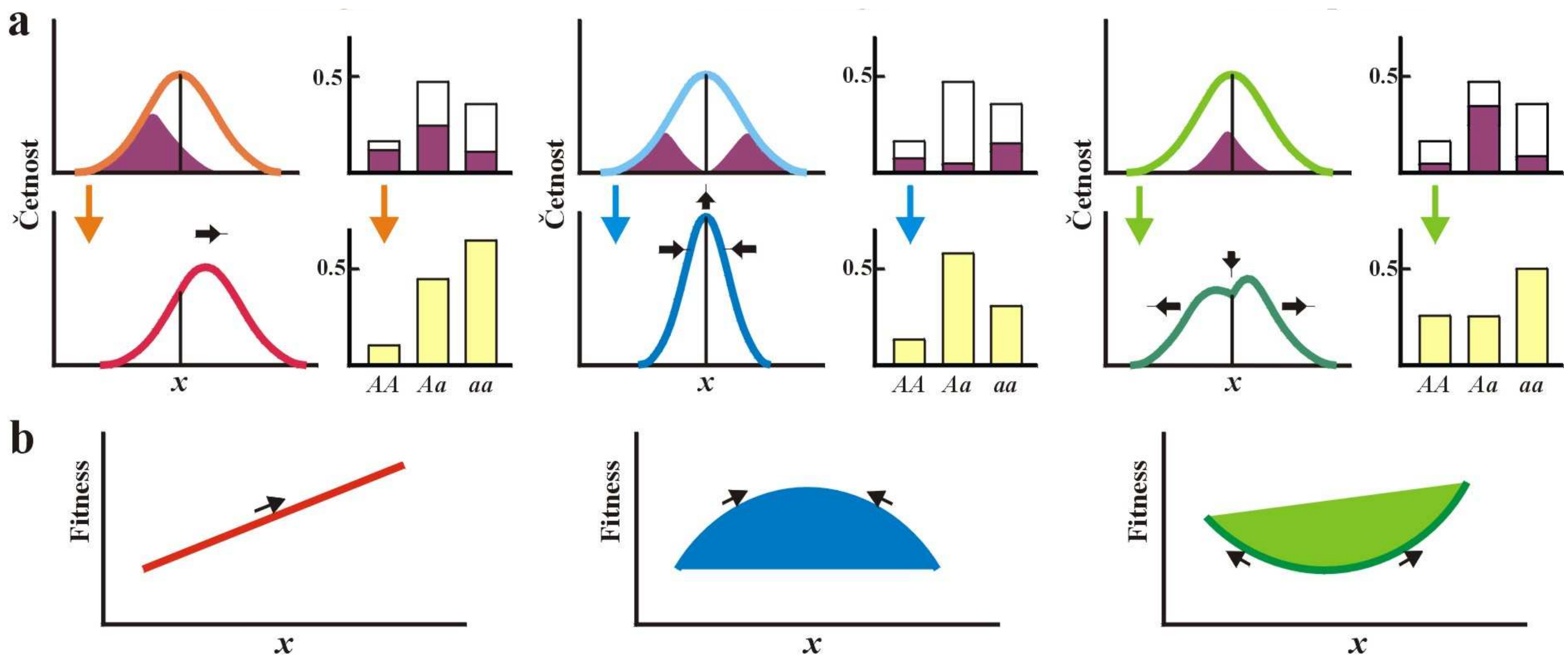
stable environment

mean unchanged

lower variance

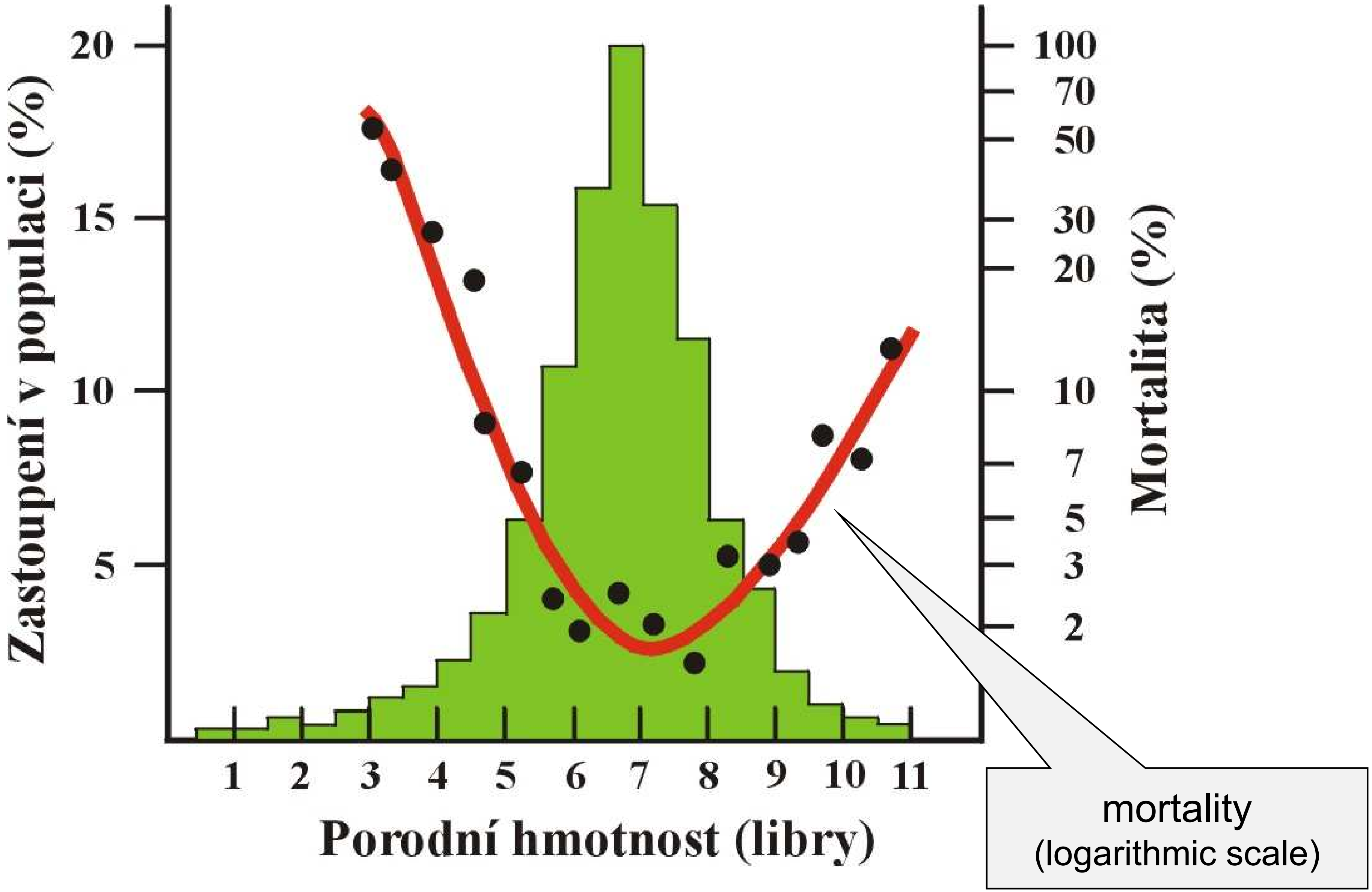
Relationship between phenotype and fitness: basic selection regimes

directional | stabilizing | disruptive



directional	stabilizing	disruptive
consistent change of environment	stable environment	heterogenous environment
shift of the mean	mean unchanged	intermediate phenotypes disadvantageous
variance unchanged	lower variance	higher variance

stabilizing selection – birth weight in humans



Equilibrium between selection and mutation

recurrent emergence of a deleterious mutation × elimination by selection

equilibrium

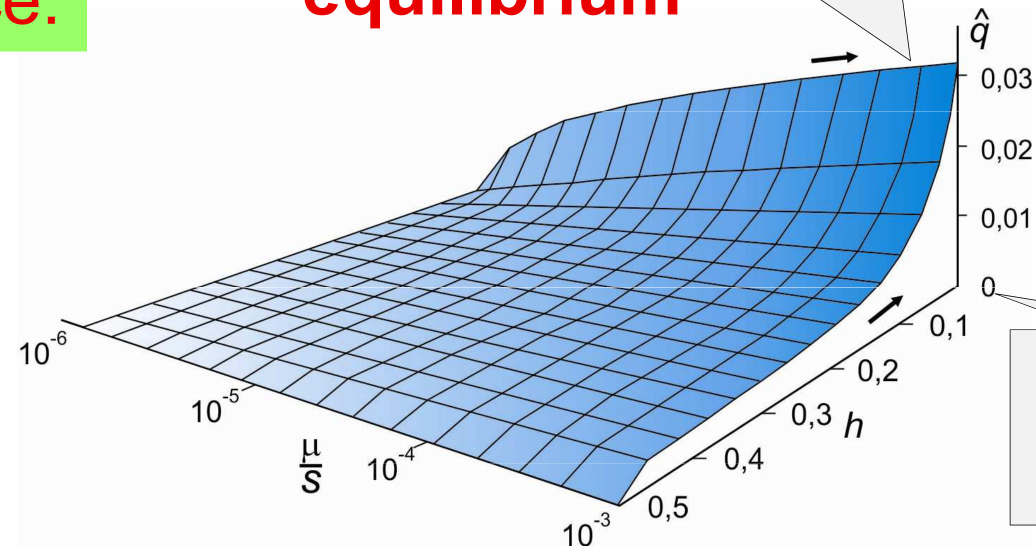
dominance:

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

recessivity:

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

weaker selection ⇒ higher frequency



complete recessivity (h = 0) ⇒ higher frequency

Muller-Haldane principle:

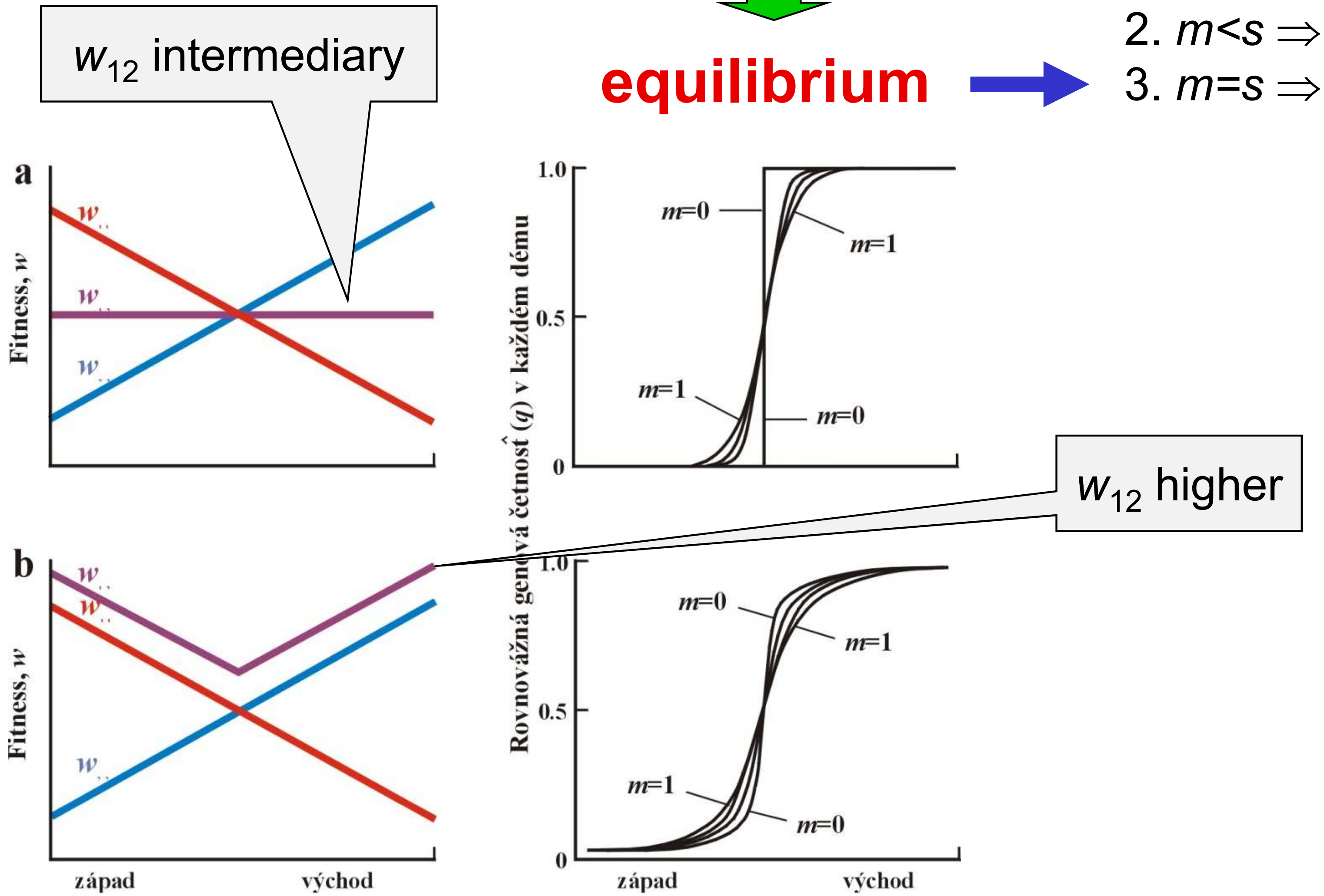
Regardless of dominance/recessivity of a deleterious mutation, its impact on decreasing fitness is <u>independent of the level of its harmfulness</u>.

Equilibrium between selection and gene flow

repeated „influx“ od a deleterious allele × elimination by selection

equilibrium

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

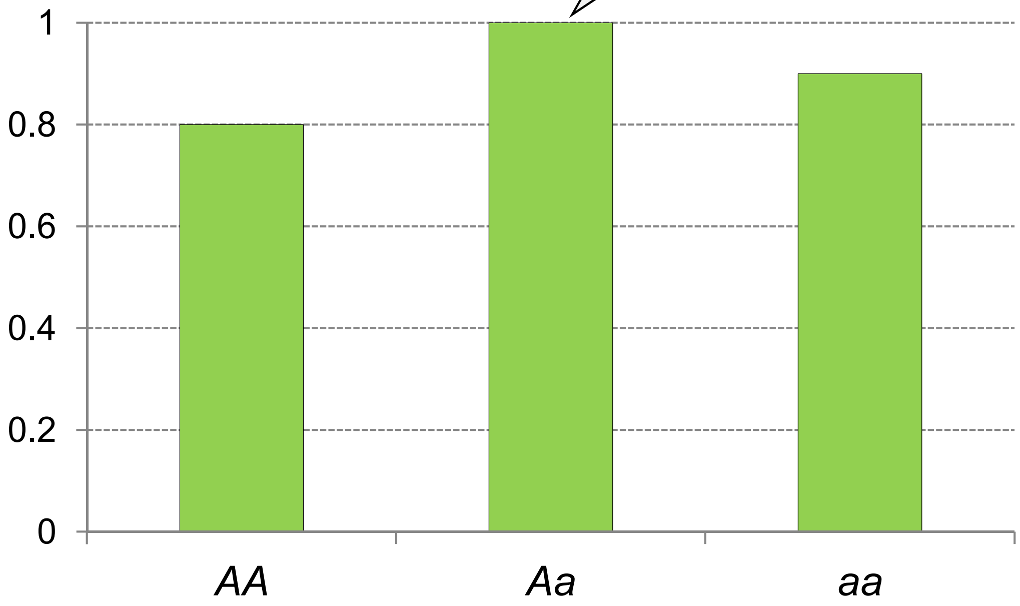


Balancing selection

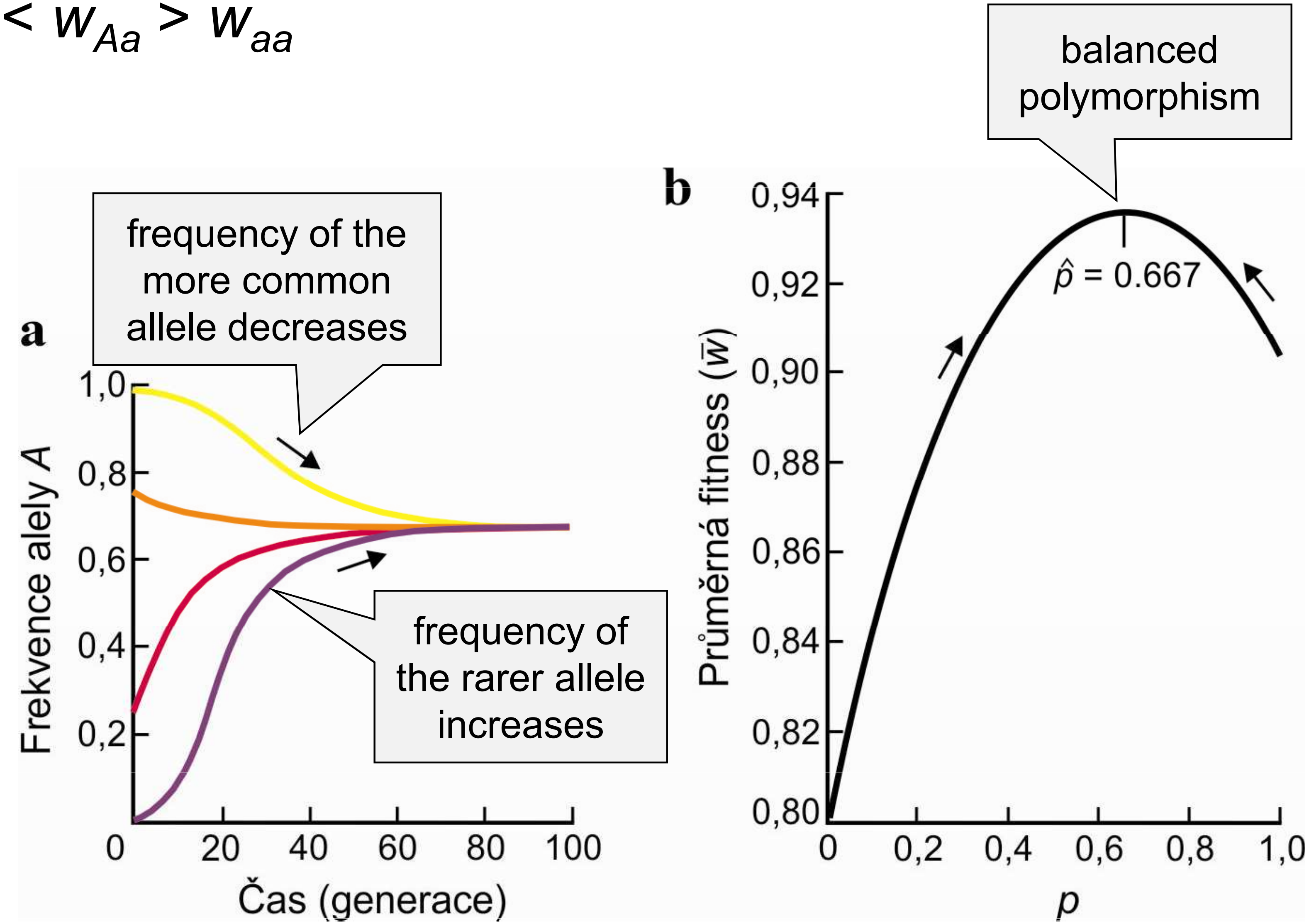
1. Selective advantage of heterozygotes = overdominance

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

heterozygote fitness is higher than *w* of homozygotes

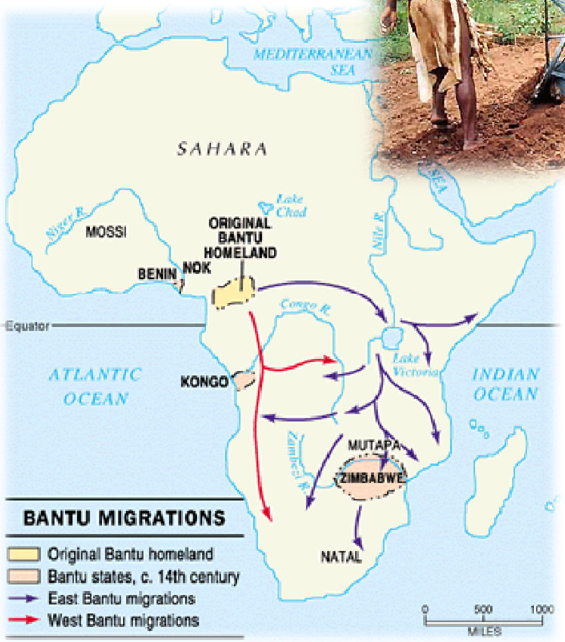


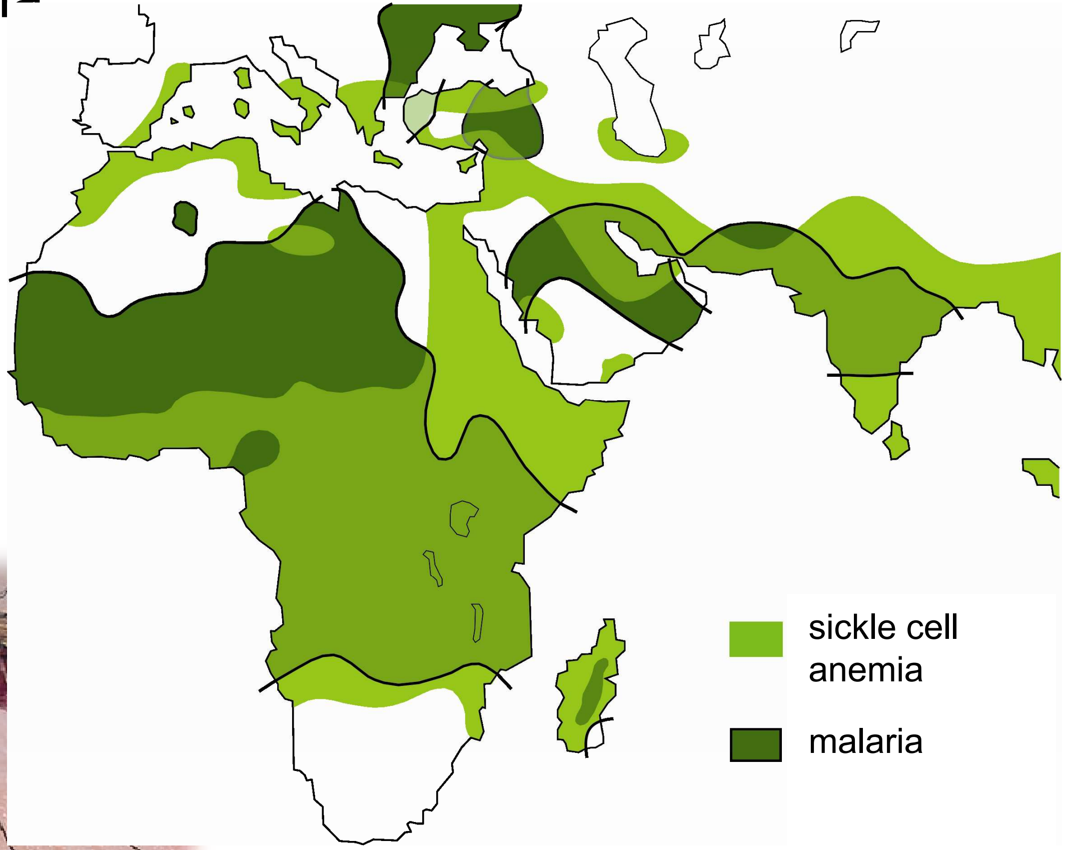
$w_{AA} < w_{Aa} > w_{aa}$



Selection maintains balanced polymorphism

Eg.: sickle cell anemia and malari





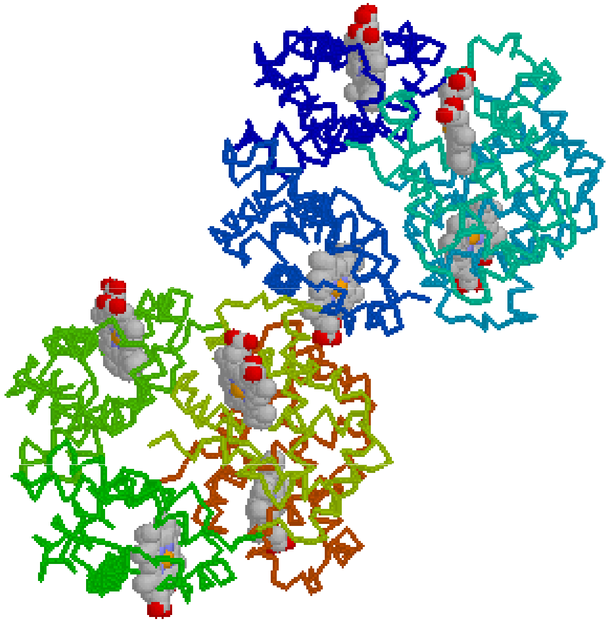
~ 2000 years ago expansion of Bantu peoples

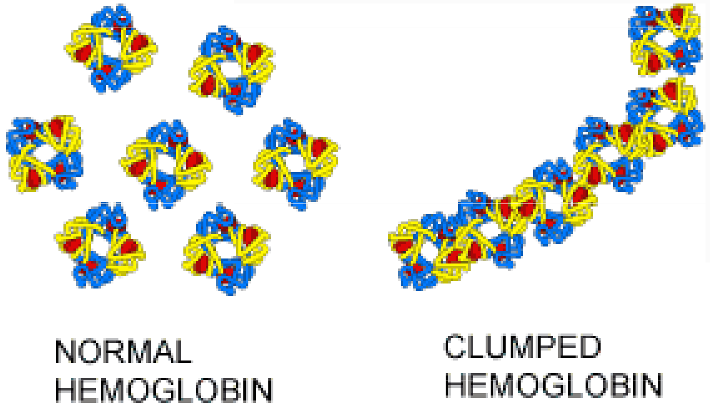
burning off savannas and forests, increase of population density → suitable environment for *Anopheles* mosquitos (*A. gambiae*), the host of *Plasmodium falciparum*

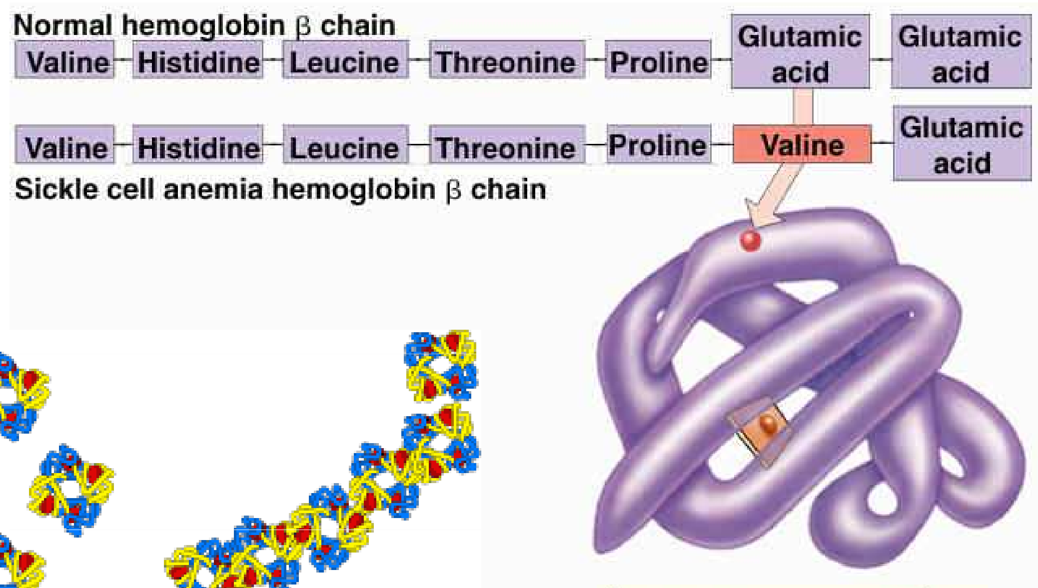
⇒ malaria

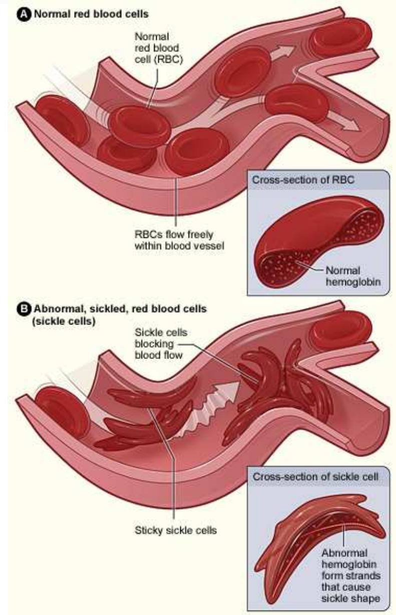
Sickle cell anemia and malaria:

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







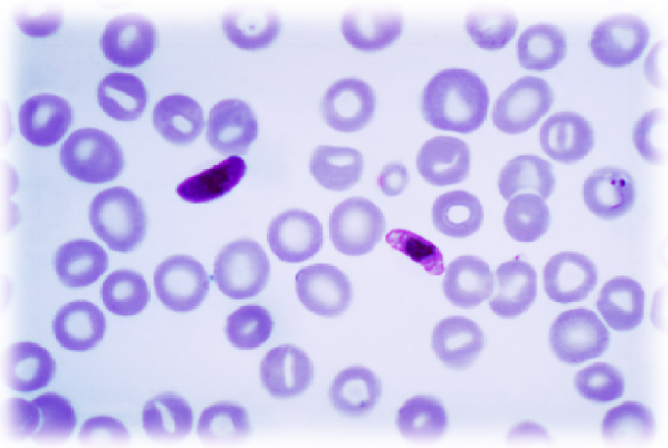


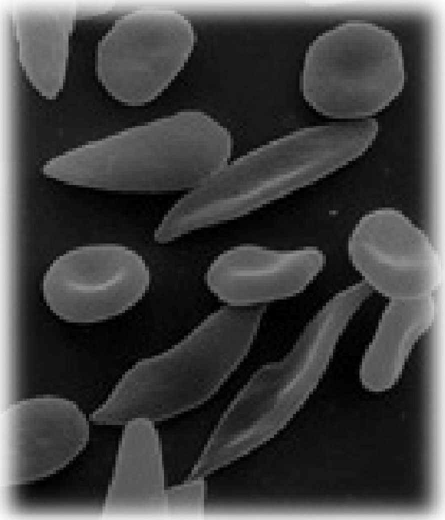
at low O_2 concentrations → production of elongated crystals

⇒ anemia

AS – only transmission of anemia, *SS* – strong anemia sickle-cell red blood cell invaded by *Plasmodium* is rapidly breaking ⇒ the parasite cannot reproduce and multiply ⇒ resistance

→ heterozygote advantage





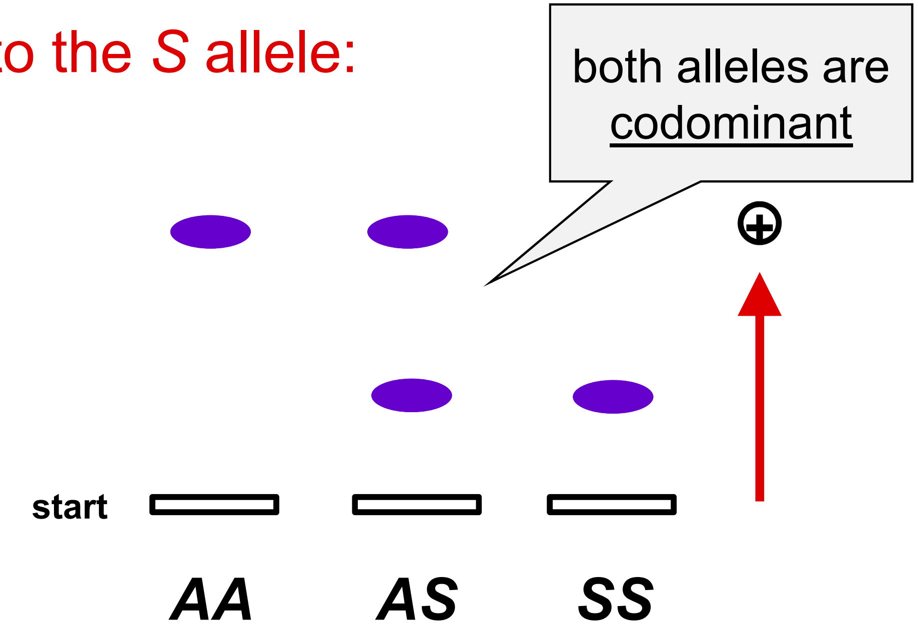
Relative fitness of genotypes related to sickle cell anemia:

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
AS	Malarial resistance	1.00	1.00
SS	Hemolytic anemia	0.20	0.20

Phenotypes related to the *S* allele:

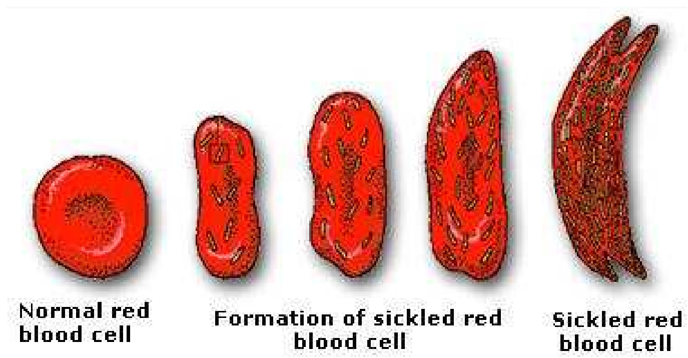
1. Electrophoretic mobility



normal erythrocyte — ***AA***

sickled erythrocyte — ***AS***

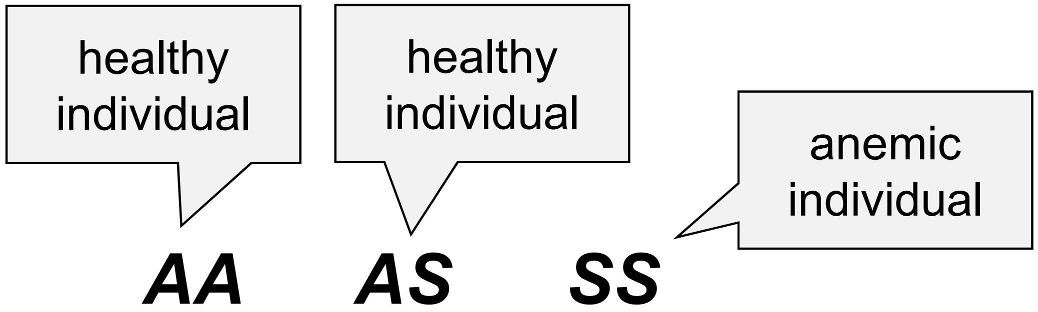
sickled erythrocyte — ***SS***



2. Sickling

sickling in *SS* and *AS* individuals ⇒ with respect to deformation *S* dominant

Phenotypes related to the *S* allele:



3. Anemia

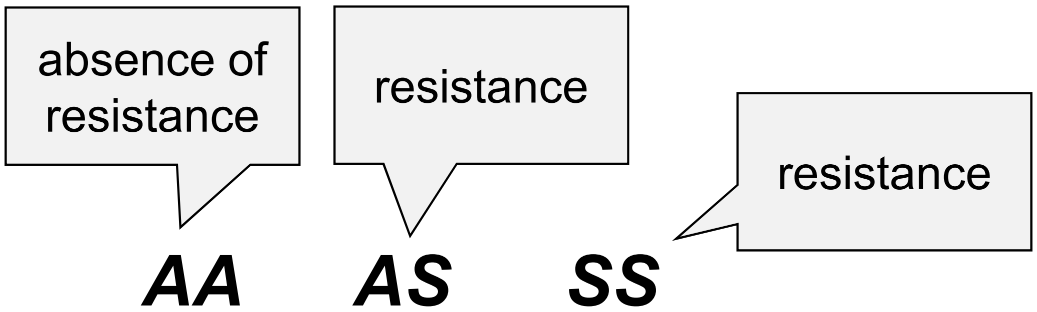
in *SS* individuals longer chains ⇒ stronger deformation of red blood cells ⇒ more fatal impacts on the organism: erythrocyte rupture (anemia), clogging of capillaries etc.

clinical syndromes only in *SS* ⇒ *S* allele <u>recessive</u>

Phenotypes related to the *S* allele:

4. Resistance to malaria

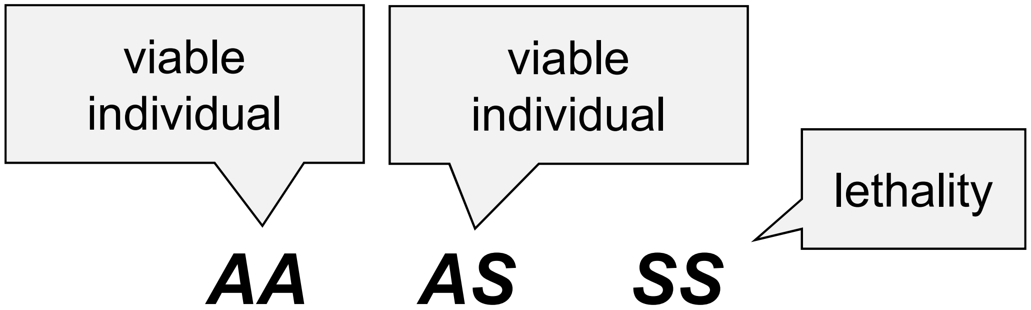
with respect to resistance the *S* allele <u>dominant</u>



Phenotypes related to the *S* allele:

5. Phenotype of health (viability)

nonmalarial environment: *S* <u>recessive</u>



malarial environment: *SS* – strong anemia; *AA* – malaria; *AS* – no anemia, weak malaria ⇒ *S* is <u>overdominant</u>

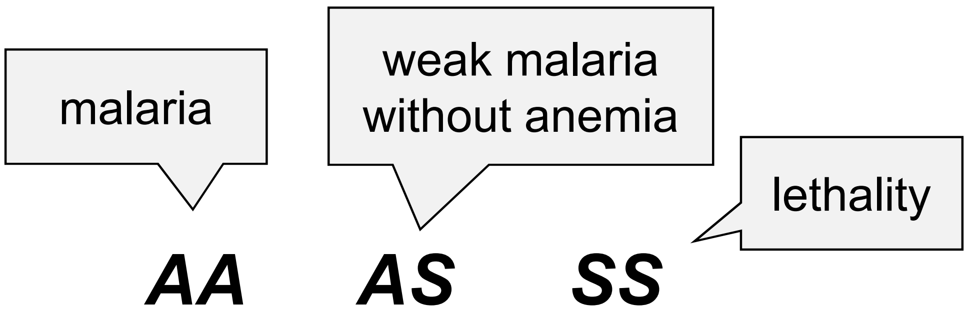


Table 11.1. Phenotypic Attributes and Relative Fitnesses (Viabilities) of Six Genotypes Formed by *A*, *S*, and *C* Alleles at *β-Hb* Locus in Humans in Wet, Tropical Africa

Genotype	Phenotypic Attributes	Fitness in Nonmalarial Environment	Fitness in Malarial Environment
AA	Malarial susceptibility	1.00	0.89
AS	Malarial resistance	1.00	1.00
SS	Hemolytic anemia	0.20	0.20
AC	Malarial susceptibility	1.00	0.89
SC	Hemolytic anemia	0.71	0.70
CC	Malarial resistance	1.00	1.31

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:

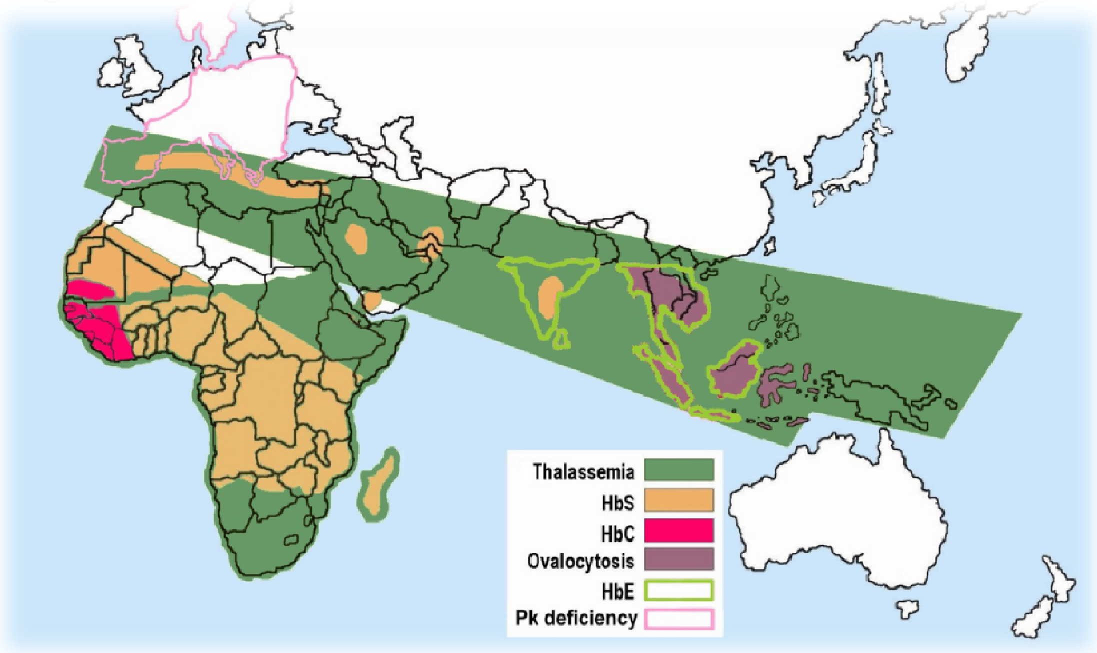
possible genotypes: w_{AC} = 0,89; w_{SC} = 0,70

w_{AS} = 1,00 ⇒ selection acts against beneficial allele!

Although *C* higly 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.

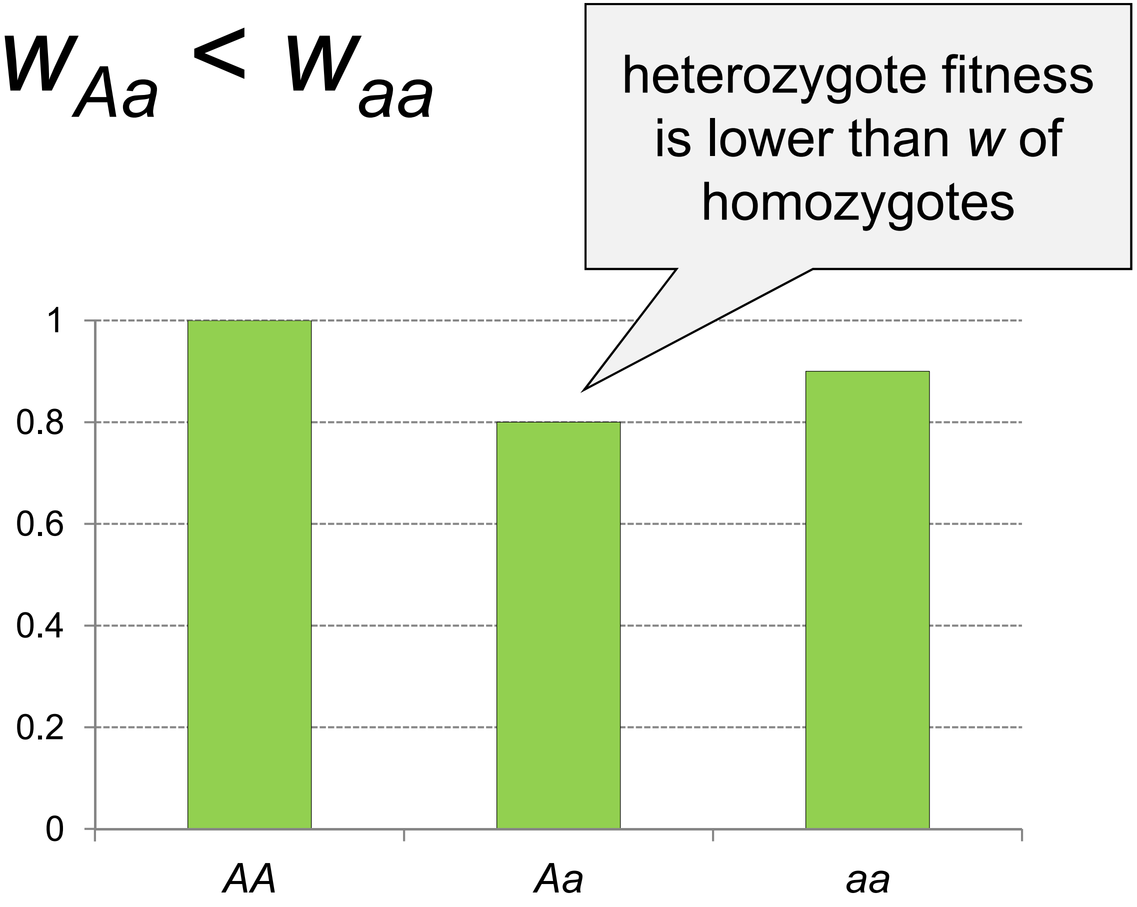


*) glucose-6-phosphate dehydrogenase
**) pyruvate kinase

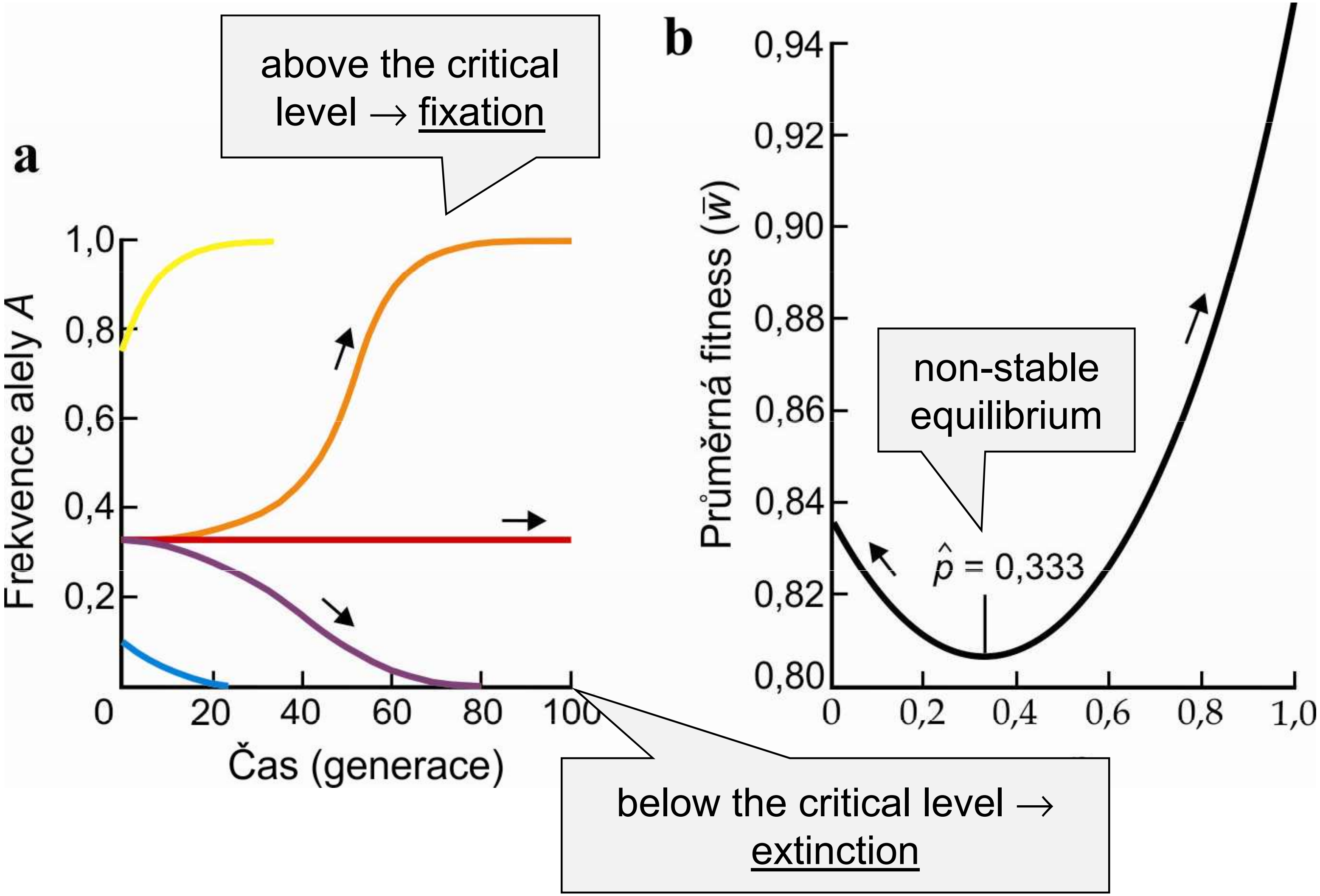
However, selection in favour of heterozygotes is not widespread in nature

Alternative equilibrium: selection against heterozygotes (underdominance)

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



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



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

2. Selection in heterogeneous environment

environmental variation:

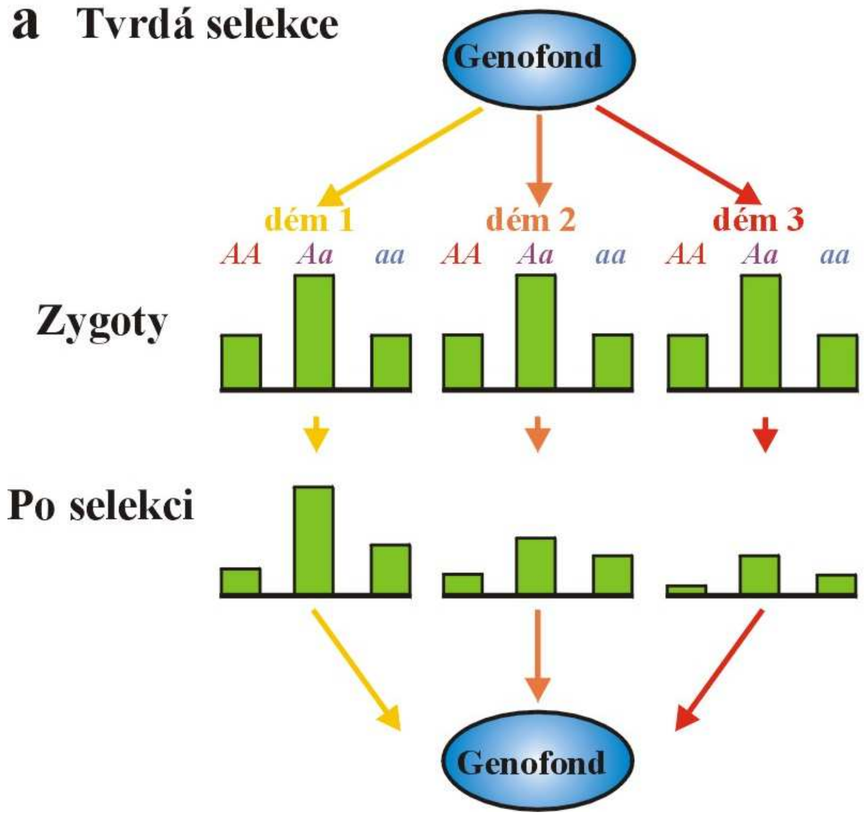
- spatial
- temporal

- coarse-grained: single environment throughout lifetime
- fine-grained: environmental heterogeneity throughout lifetime

selection: soft
hard

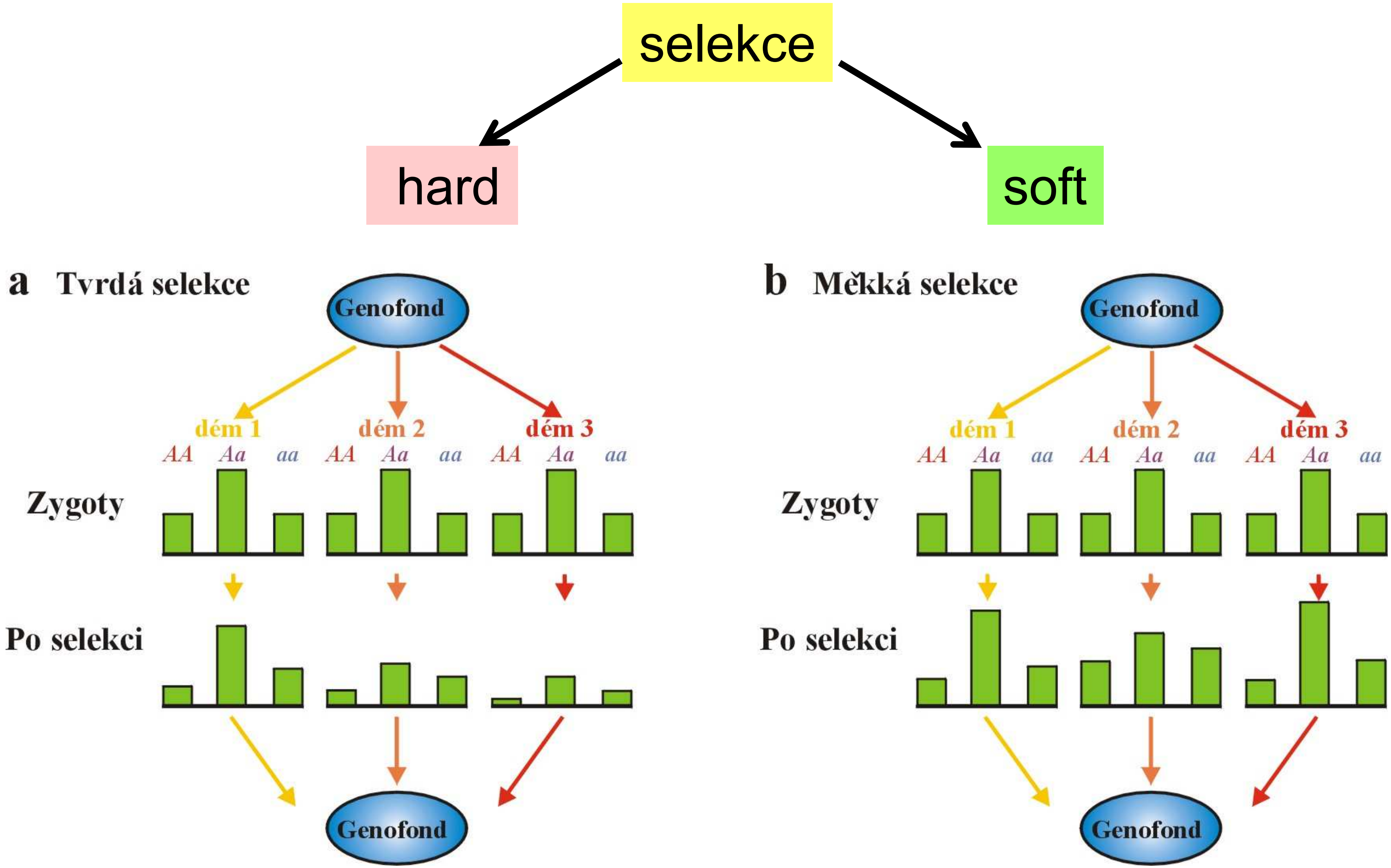
selection

hard



Silene vulgaris ssp. *humilis*

Minuartia verna



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

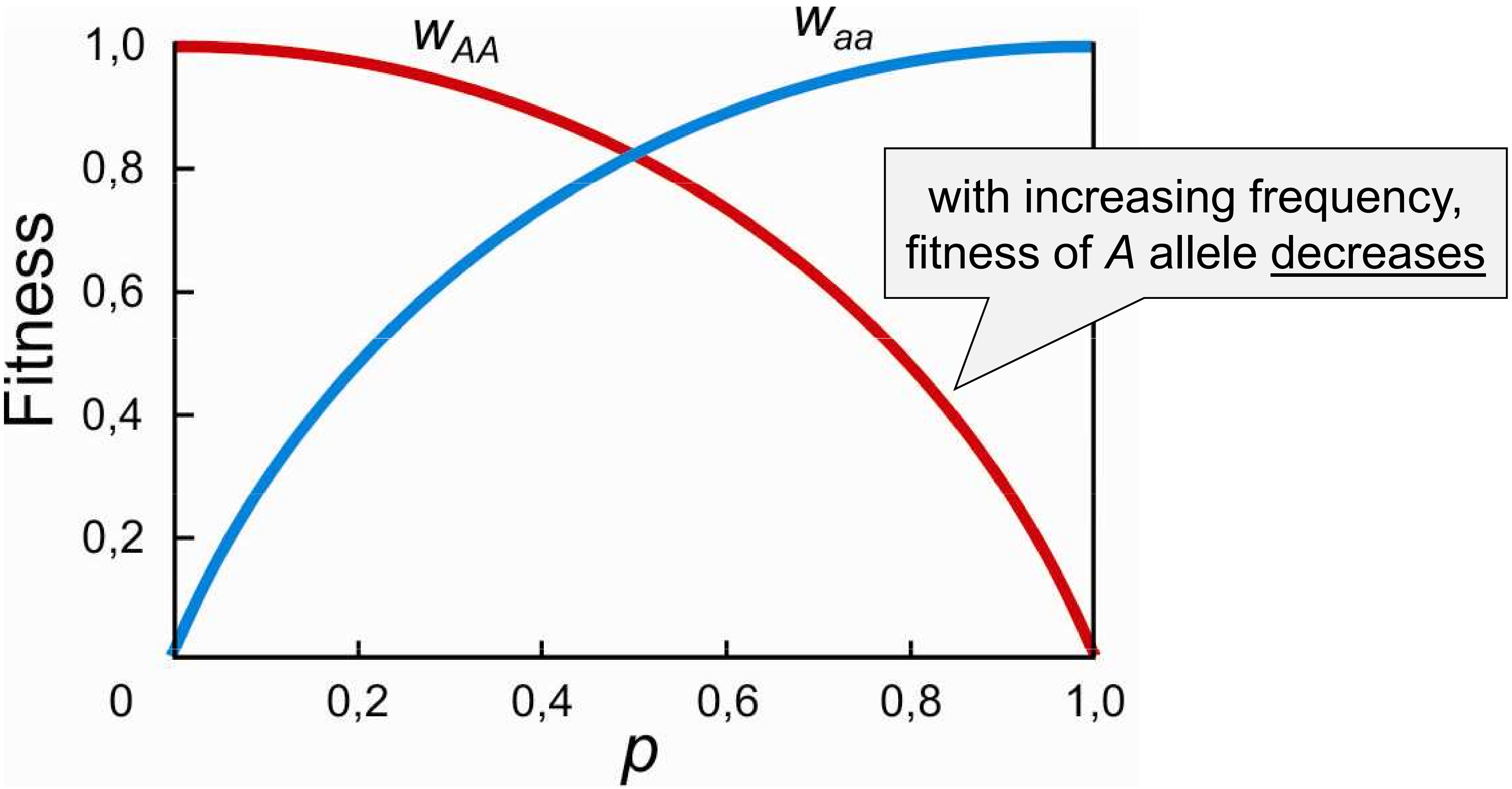
3. Antagonistic selection

different sexes

different ontogenetic stages

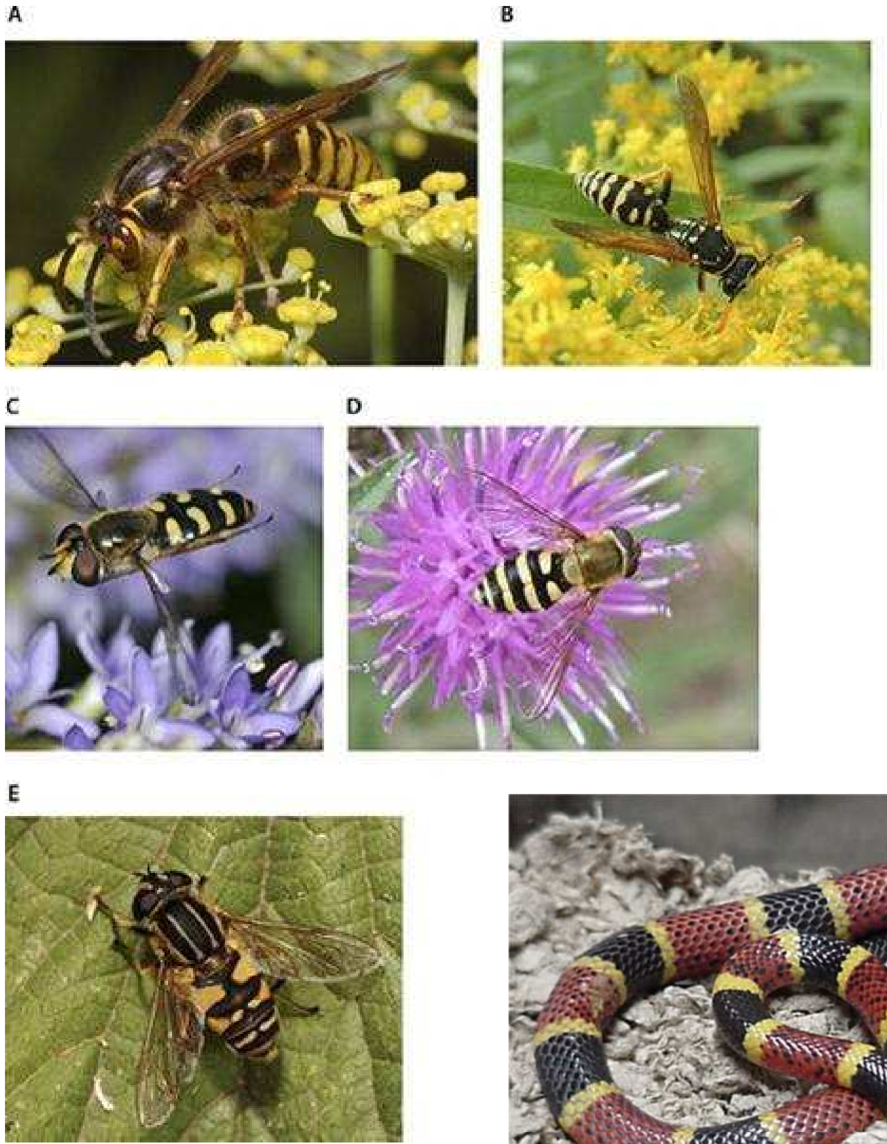
gametic $\times$ zygotic phase

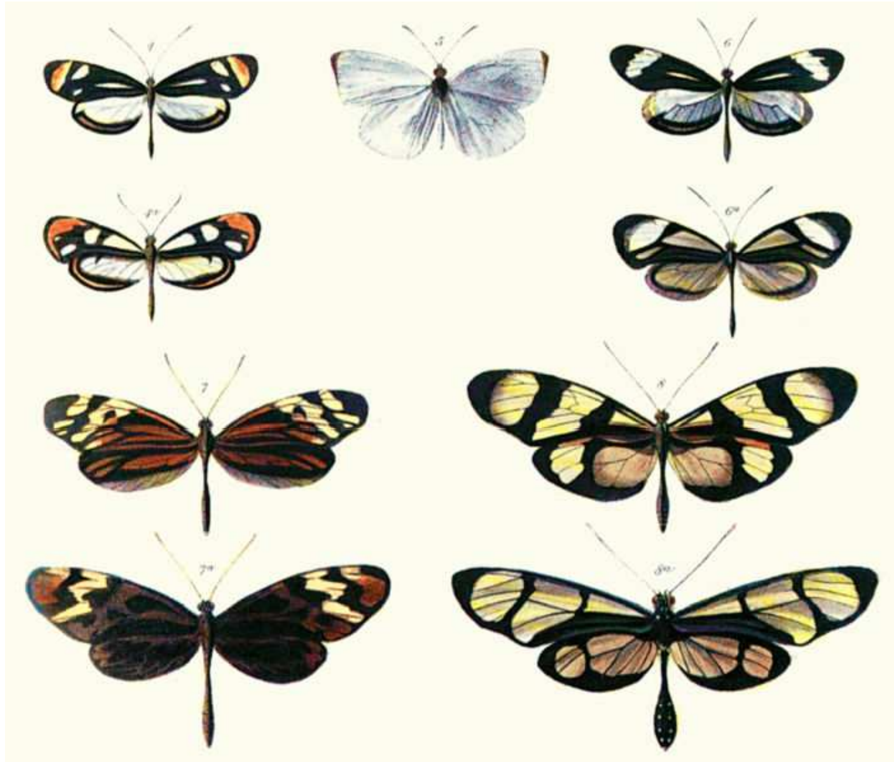
4. Frequency-dependent selection
I. Negative



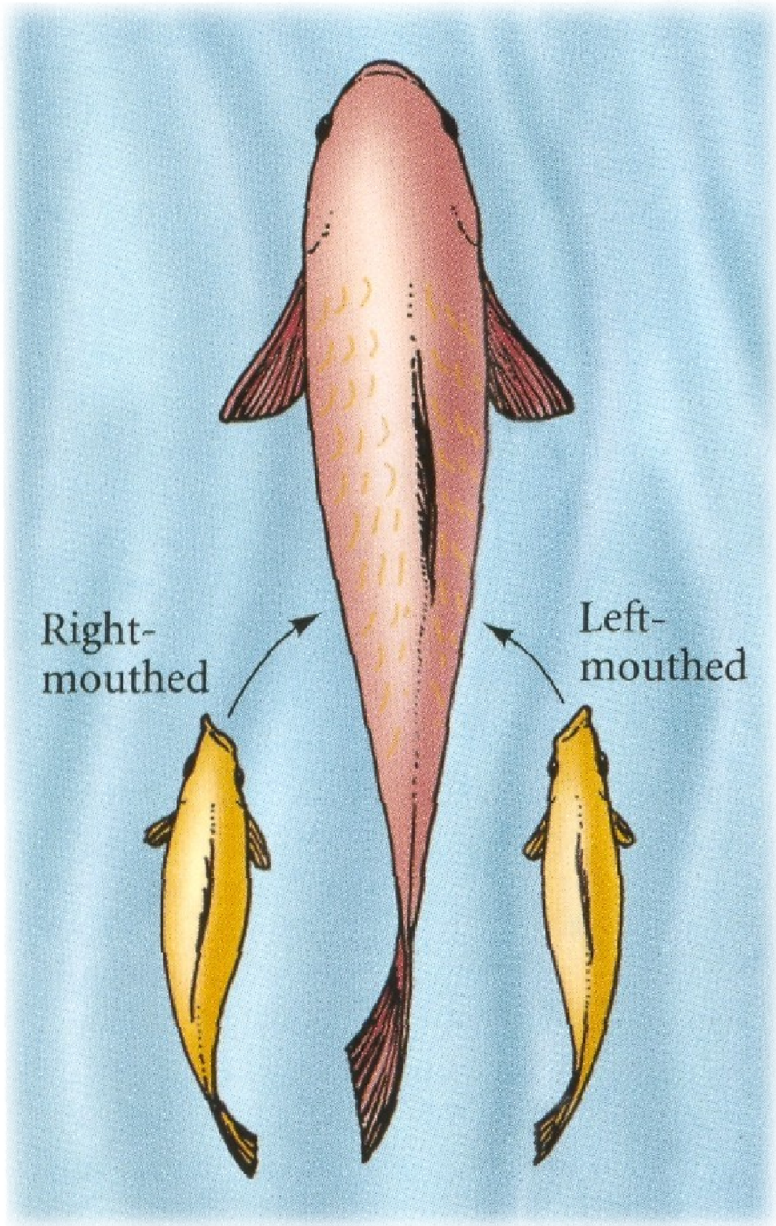
Eg.: Batesian mimicry

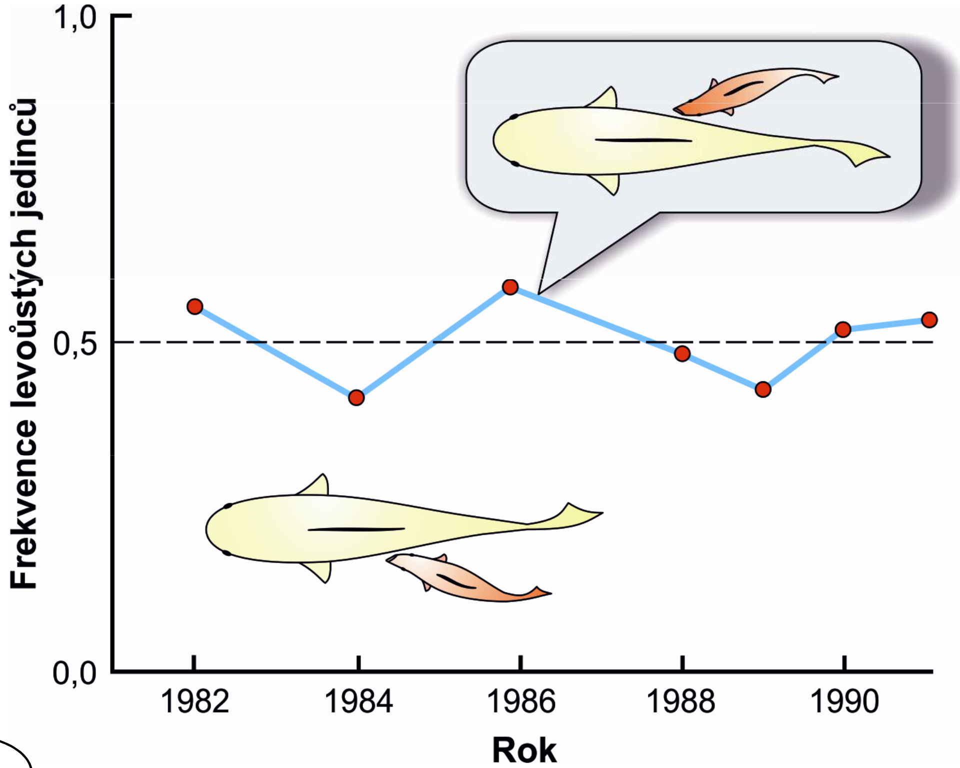
[in this case it is rather density-dependent selection]

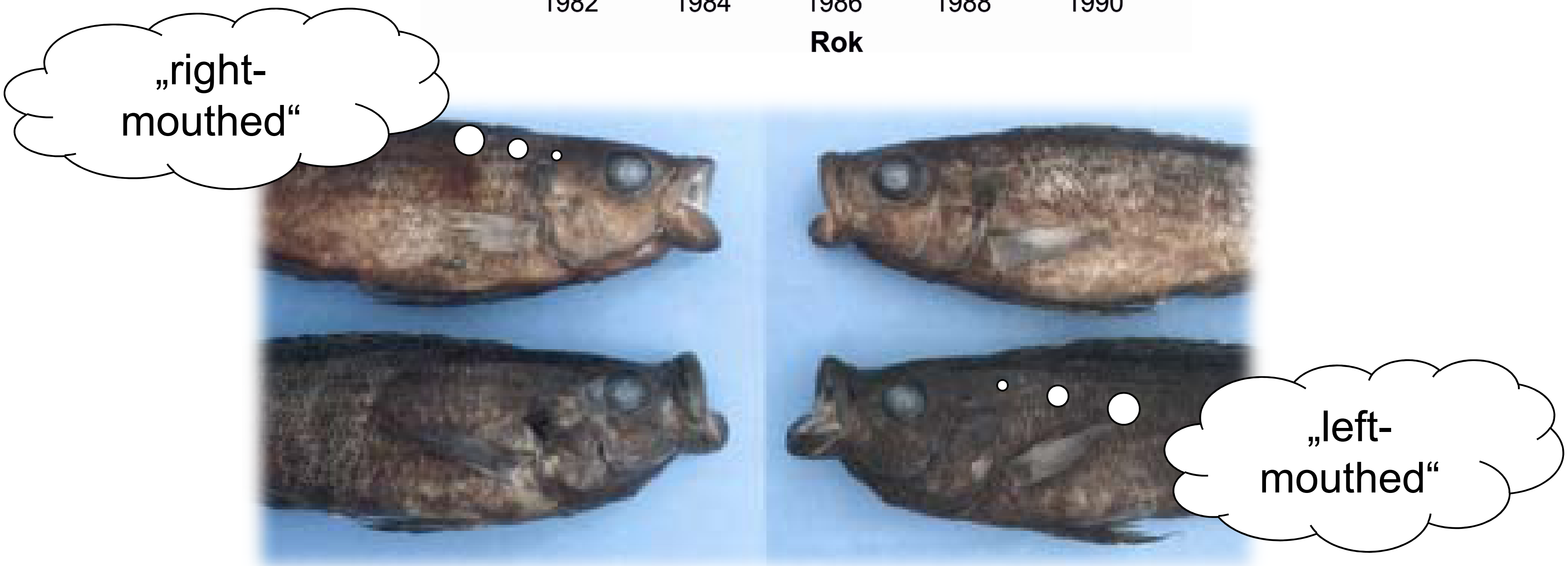




Eg.: cichlid *Perissodus microlepis* (Tanganyika)




1,0
0,5
0,0
Frekvence levoústých jedinců
1982
1984
1986
1988
1990
Rok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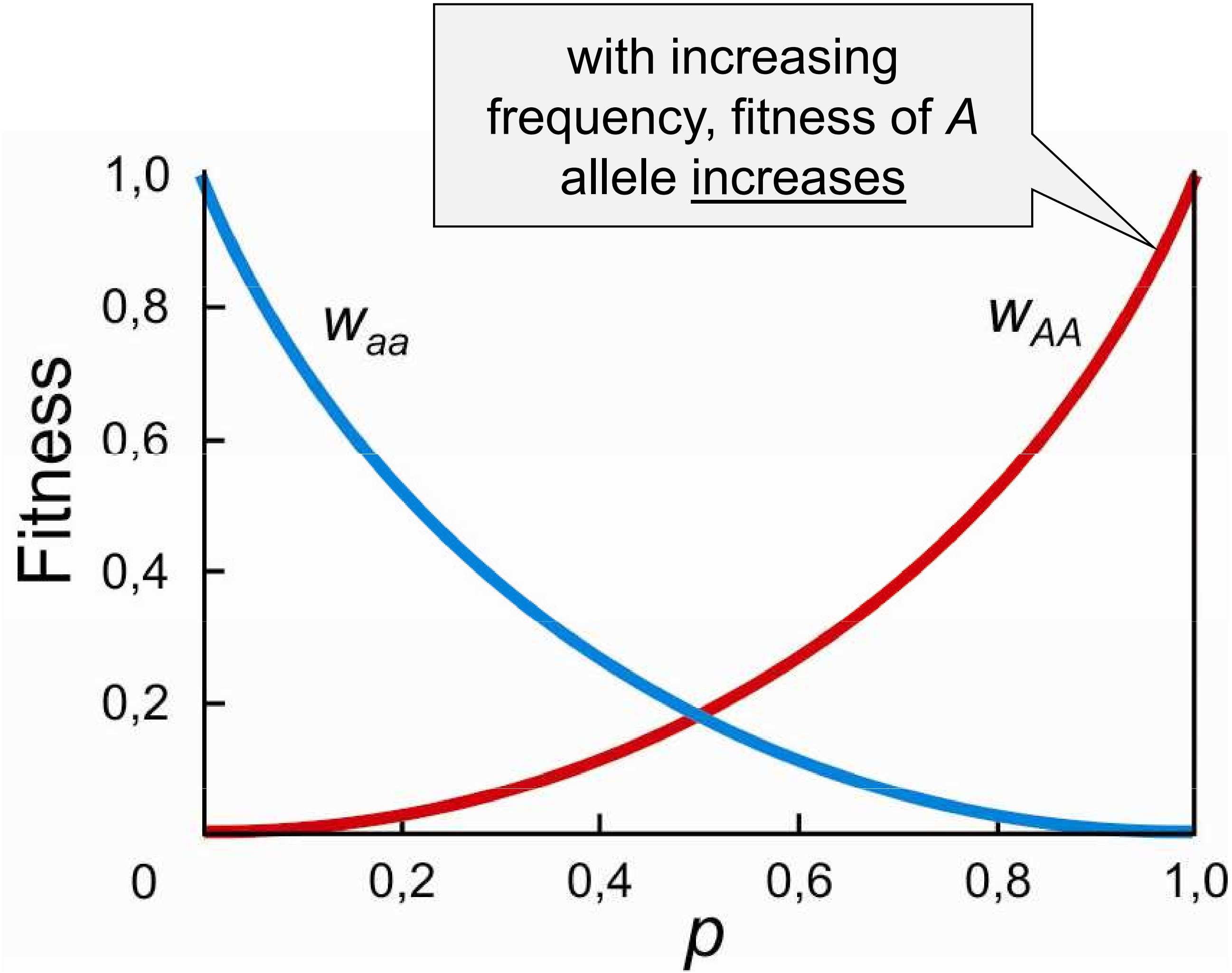
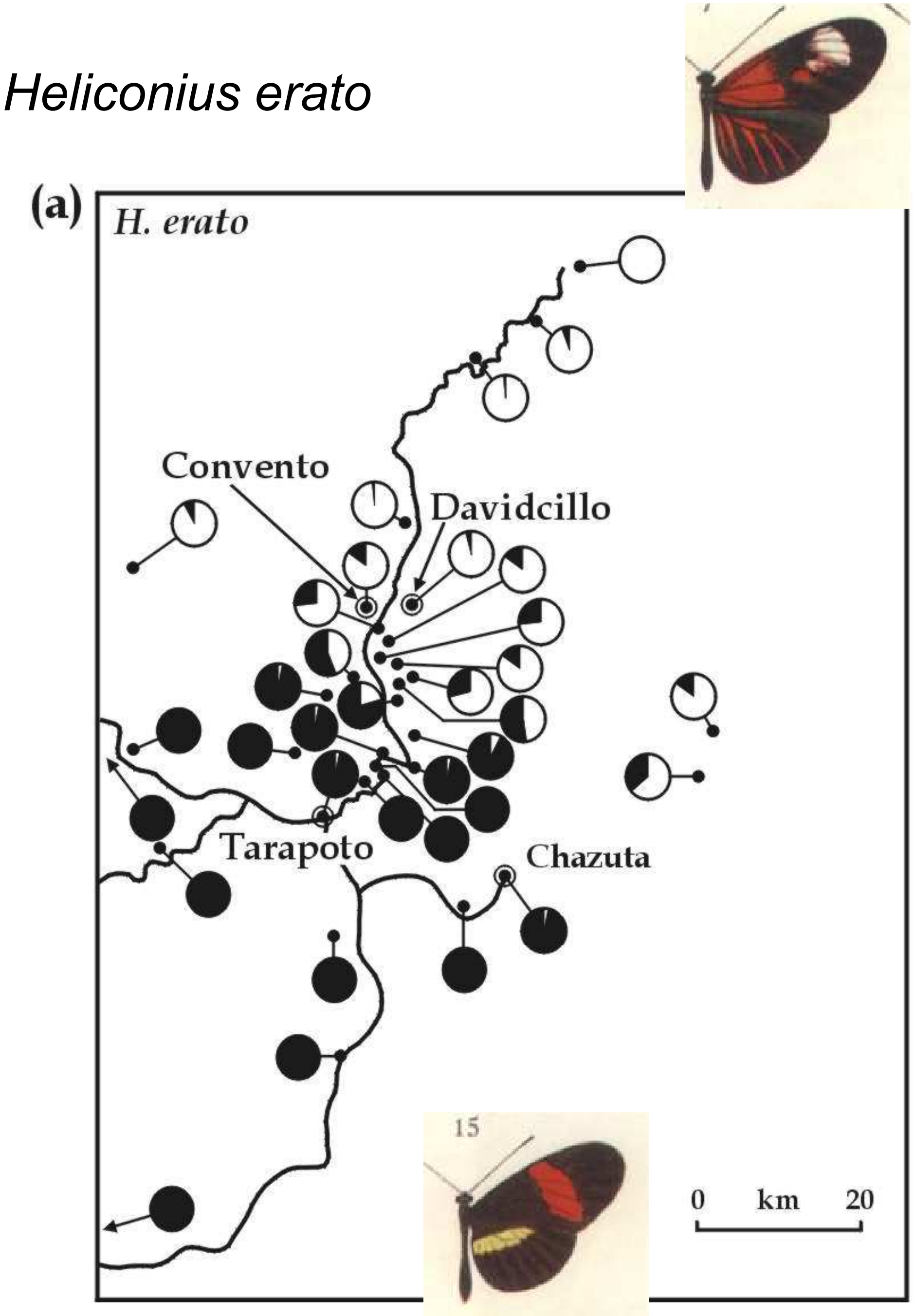
4. Frequency-dependent selection
II. Positive

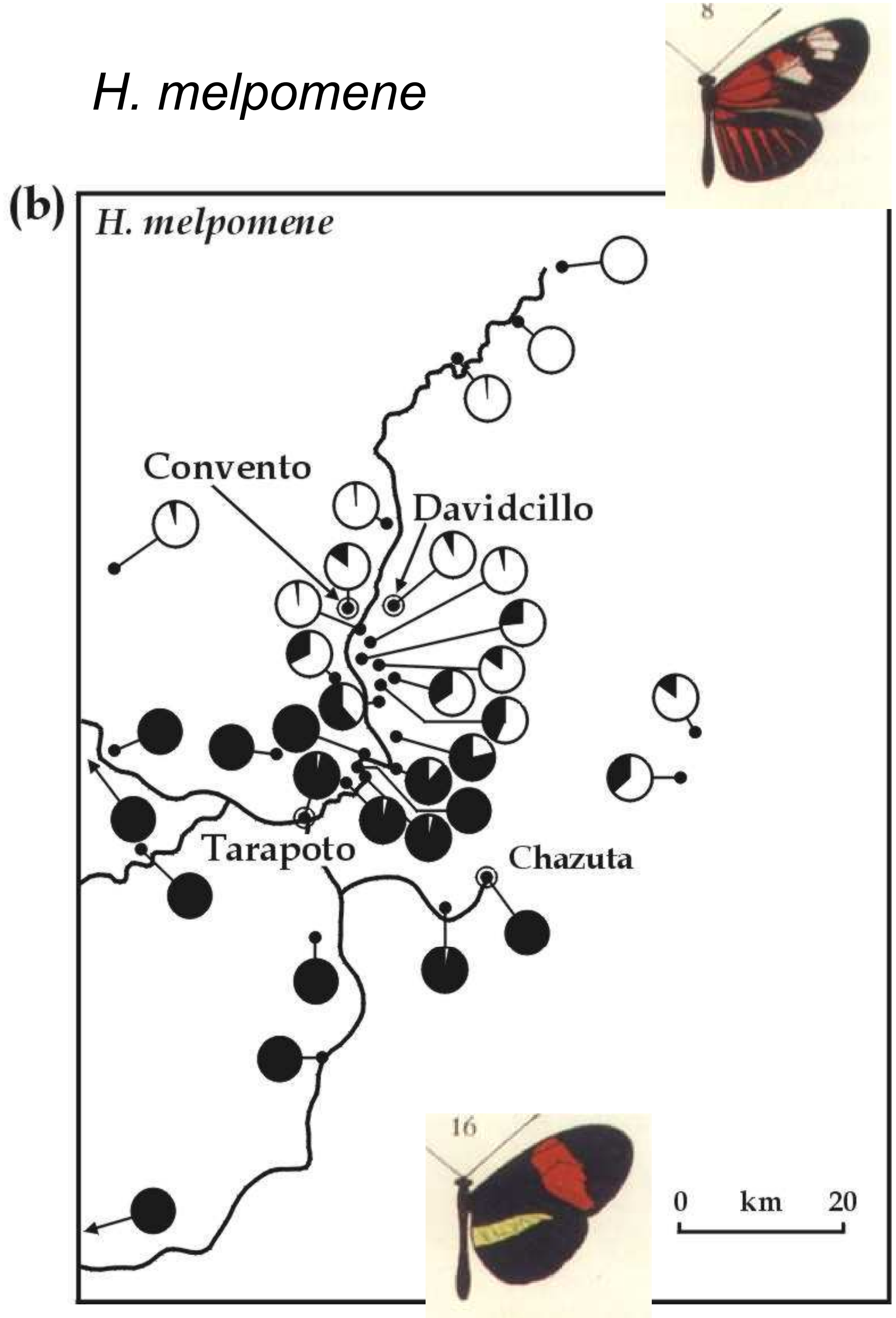


Müllerian mimicry:

Heliconius erato

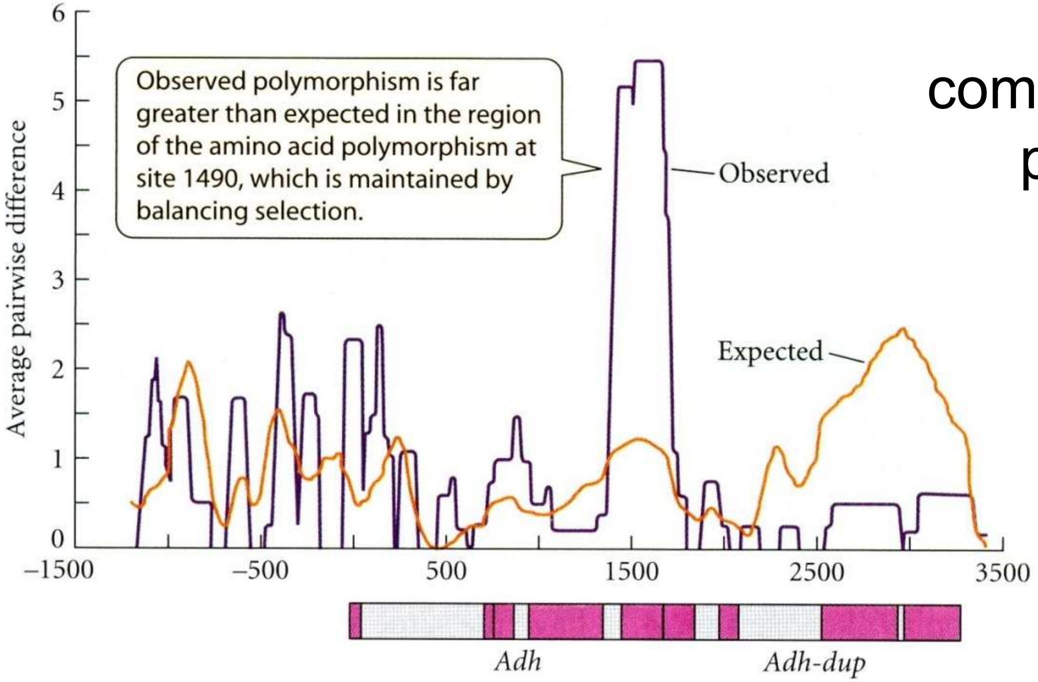


H. melpomene



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