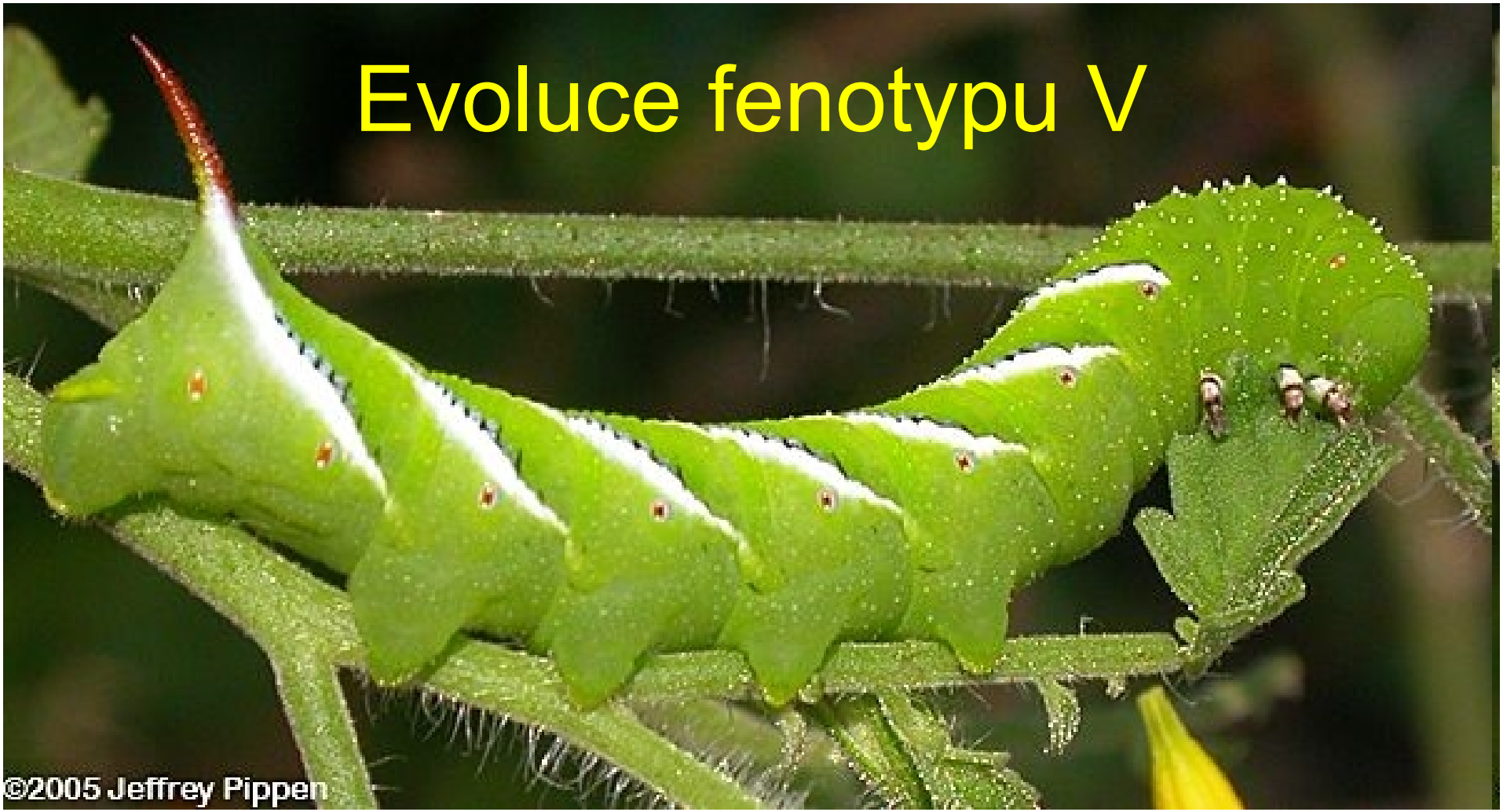
Evoluce fenotypu V



Existence trade-offs záleží na proximátních mechanismech ovlivňujících znaky

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B

Phil. Trans. R. Soc. B (2010) **365**, 567–575
doi:10.1098/rstb.2009.0249

Conflicting processes in the evolution of body size and development time

H. Frederik Nijhout[1,*], Derek A. Roff[2] and Goggy Davidowitz[3]

$$\text{Final size} = w5 * \exp\left(\left(k * \text{ICG} + \ln 5.33 - \frac{0.8}{W_0}\right) * \left(1 - 0.073 * \text{ICG}\right)\right), \quad (2.1)$$

where W_0 is the initial weight of the instar and k is the growth exponent, which is calculated from the growth rate (GR) during the third day of the instar as follows:

$$k = 0.15 * \exp(-0.65 * W_0) * \text{GR} + 0.27. \quad (2.2)$$

The duration of the instar (development time) is given by

$$\text{Duration of instar} = \frac{\ln(5.33 - 0.8/W_0)}{k} + \text{ICG}. \quad (2.3)$$

We note that these equations do not include the photoperiodic gating (i.e. as written, they stop after the second checkpoint). We calculate the gating numerically by knowing when the larva starts growing to the nearest hour and calculate whether equation (2.3) predicts a time inside a gate; if it does not then we add the appropriate time interval to the ICG term in equations (2.1) and (2.2).

The model thus requires only three easily measurable inputs, which we call the *underlying factors*. These are (i) the growth rate, (ii) the initial weight and (iii) the ICG. The critical weight (CW) is related to the initial weight of the instar by the linear function $\text{CW} = 5.33 * W_0 - 0.8$ (Nijhout *et al.* 2006). In the figures used in this paper, we show body size and development time as functions of the CW. The model accurately predicts individual final weights and development times for the entire physiologically relevant range of the three underlying factors, which are growth rate = 1–4 g d^{-1}, ICG = 16–96 h and CW = 3–9 g. The mathematical model is implemented in MatLab (The Math Works, Natick, MA, USA), and the visualizations of the multivariate data as well as the calculations of the gradients were done in Amira (Mercury Computer Systems, Chelmsford, MA, USA).

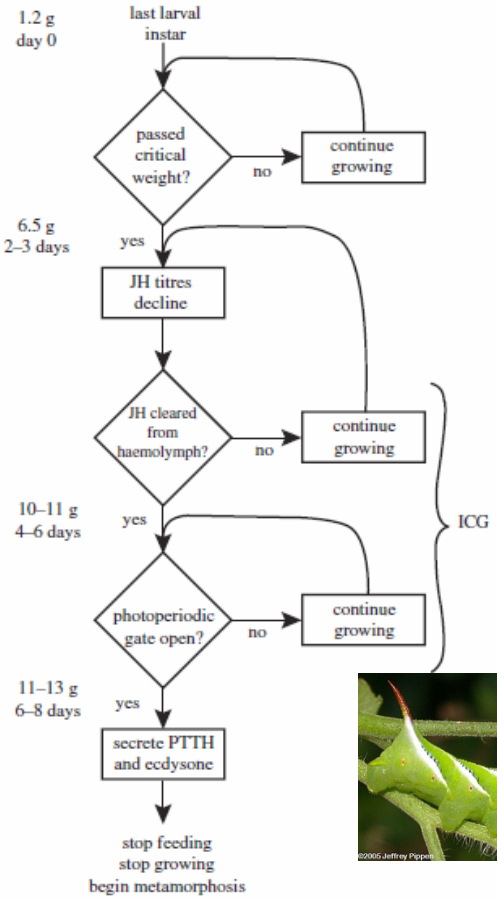
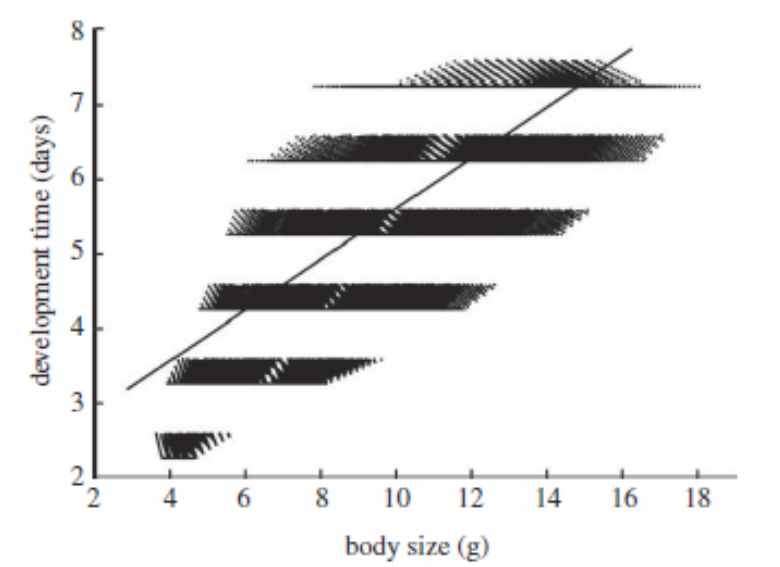


Figure 1. Logic diagram of the mathematical model for body size and development time in the final (fifth) larval instar of *M. sexta*. Diamonds represent checkpoints. Approximate masses and times for the wild type at each stage are indicated. ICG, interval to cessation of growth; JH, juvenile hormone; PTTH, prothoracicotropic hormone.

Parameters of body size and developmental time:

- the growth rate
- the initial weight
- the ICG

Celkový vztah mezi dobou vývinu a velikostí predikovaný modelem:



Existují oblasti, kde vztah není pozitivní:

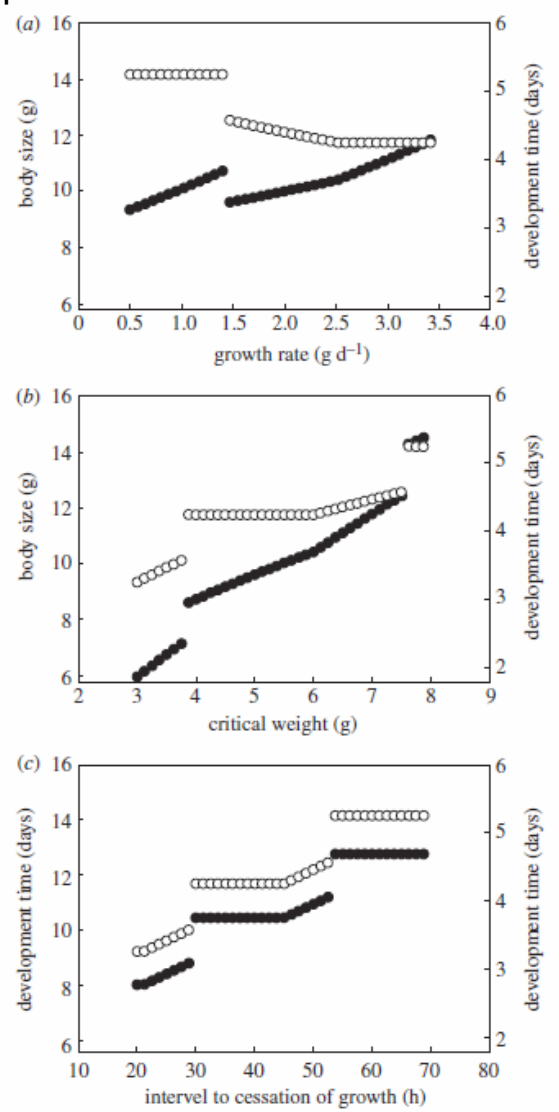


Figure 4. Body size and development time as functions of either the growth rate (*a*), critical weight (*b*), or ICG (*c*), when the other factors are held constant. The constant values were growth rate, 2.5 g d^{-1}; critical weight, 6 g; and ICG, 48 h. Filled circles, body size; open circles, development time.

Fenotypové krajiny pro velikost a dobu vývoje se značně liší:

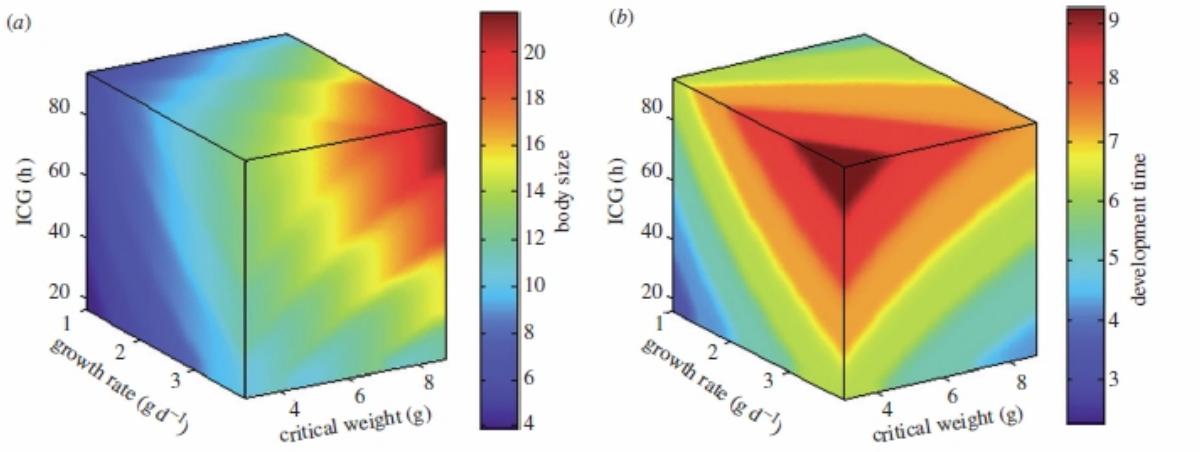


Figure 5. Phenotypic landscapes of body size (*a*) and development time (*b*) as functions of the growth rate, critical weight and ICG. Orientation of axes is the same for the two landscapes. Body size (g) and development time (days) are indicated by colour scales.

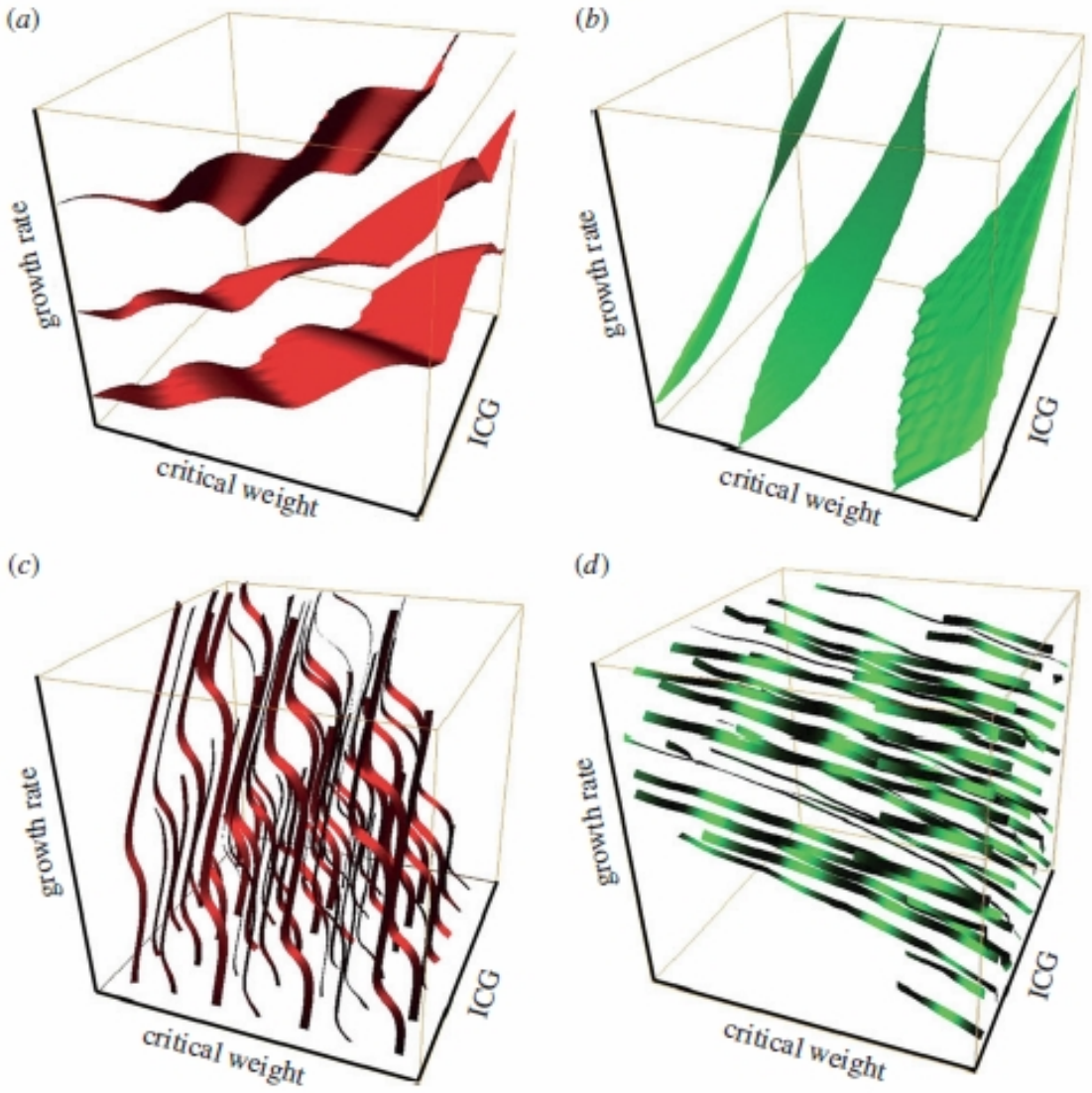
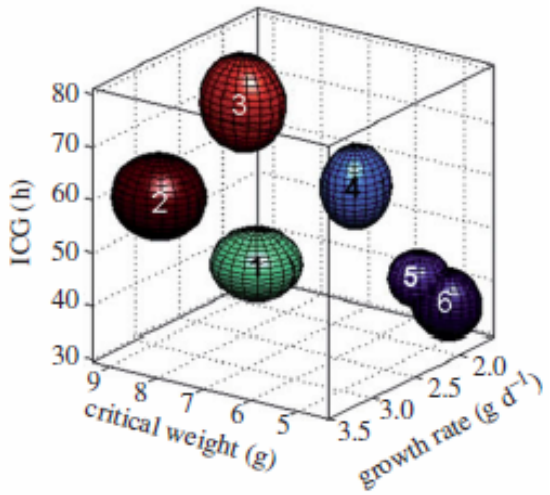
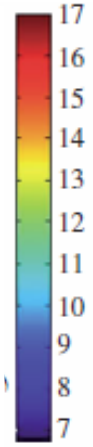


Figure 7. Selected isosurfaces for (*a*) body size (red) and (*b*) development time (green). Orientation of the phenotypic landscape is the same in all panels. Isosurfaces are combinations of parameter values that produce the same phenotype. The gradients of the phenotypic landscapes for (*c*) body size and (*d*) development time. The gradients are shown as ribbons that follow the steepest internal slopes of the phenotypic landscapes. The gradients are orthogonal to the isosurfaces.

Parametry modelu jsou závislé na podmínkách prostředí, ale i na genotypu





Mutant s recesivní alelou ovlivňující titry juvenilního hormonu

Figure 8. Stereo-pair showing distributions of six strains of *M. sexta* within the phenotypic landscape of body size. Strain 1 is the ancestor of strains 2–5, which were derived by selection for increased or decreased body size and development time. Strain 6 is the black larval mutant. The dimensions of the axes of the spheroids correspond to standard deviations around the mean for each of the underlying factors. Colour of the spheroids indicates body size.

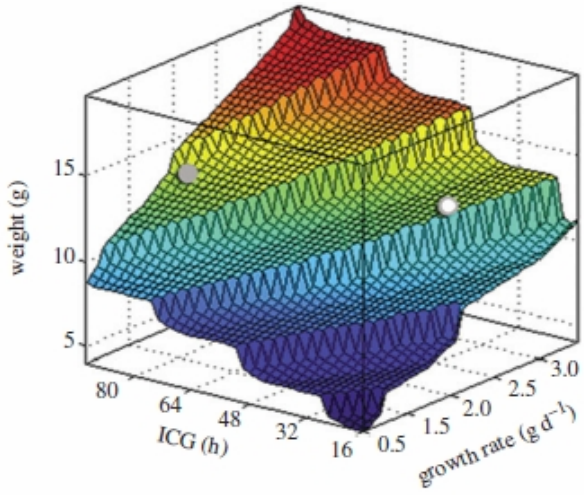
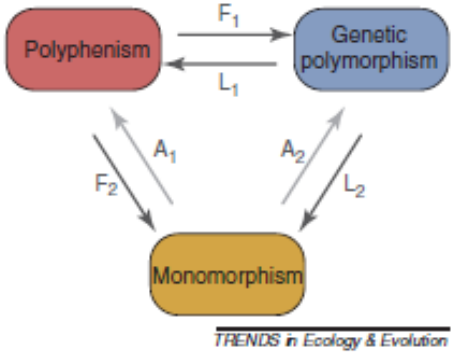


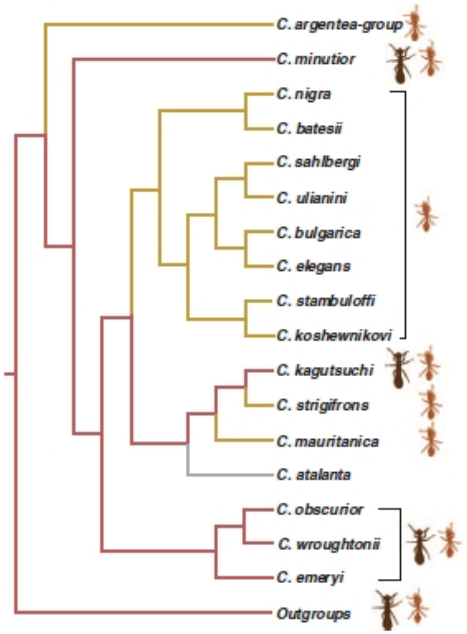
Figure 6. Phenotypic landscape for body size when critical weight is held constant at 7 g. The two circles indicate phenotypes at two different temperatures: 30°C (on the right) and 20°C (left).

Fenotypový polymorfismus je někdy spojen s genetickým polymorfismem, jindy s fenotypovou plasticitou (polyfénie)

Genes as leaders and followers in evolution

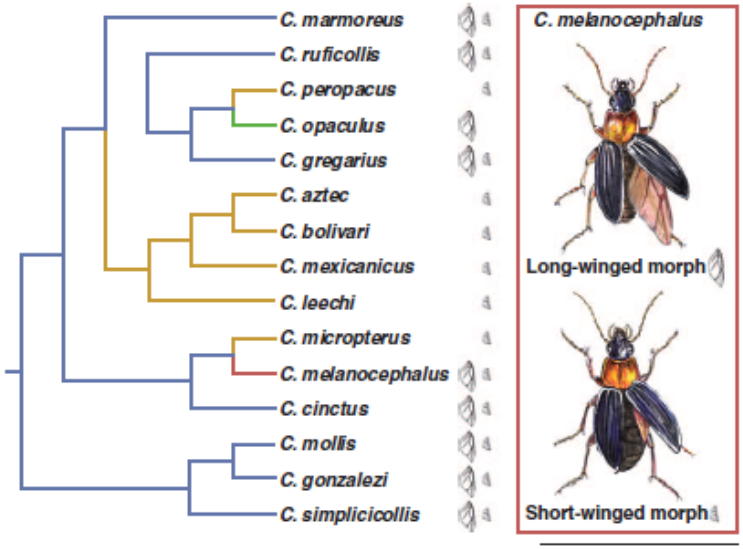
Tanja Schwander[1,3] and Olof Leimar[2,3]



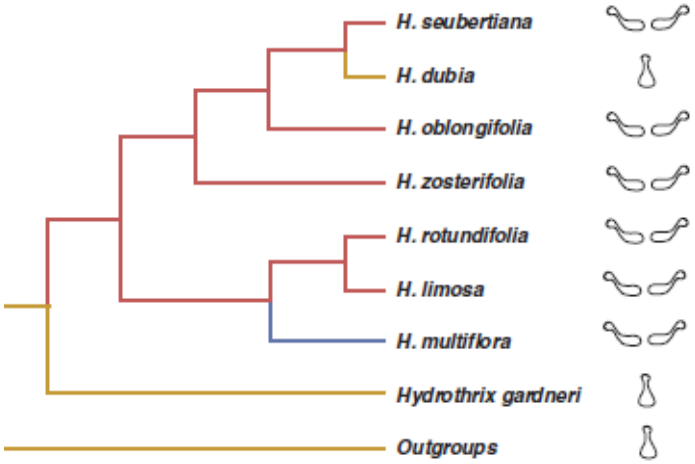


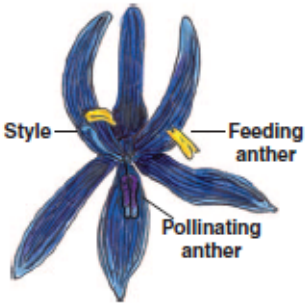
C. obscurior males

polyfénie ➡ monomorfismus (genetická asimilace)



genetický polymorfismus ➡ monomorfismus a polyfénie





Left-styled flower

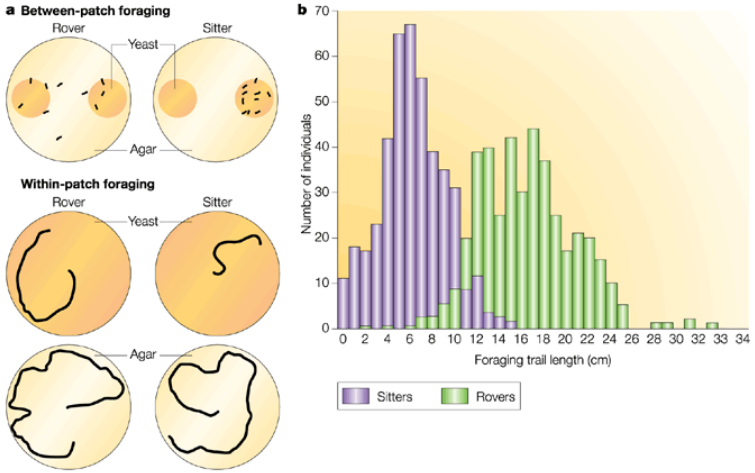
Right-styled flower

polyfénie ➡ monomorfismus a genetický polymorfismus

Fenotypový polymorfismus je někdy spojen s genetickým polymorfismem, jindy s fenotypovou plasticitou (polyfénie)

Maintaining a behaviour polymorphism by frequency-dependent selection on a single gene

Mark J. Fitzpatrick[1], Elah Feder[2], Locke Rowe[2] & Marla B. Sokolowski[1]



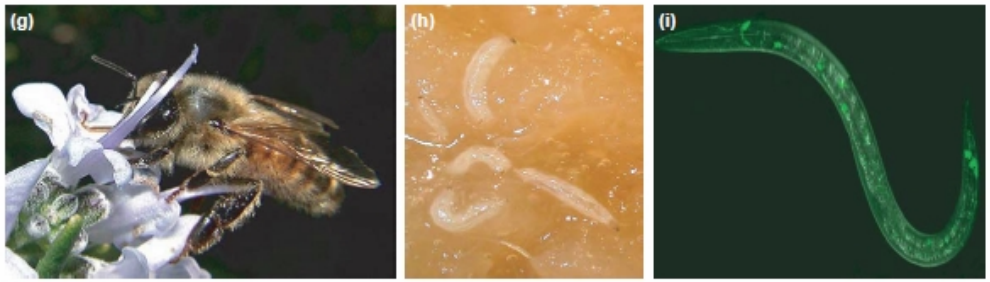
Nature Reviews | Genetics

gen *foraging* (*for*): guanosine 3´,5´-monophosphate-dependent protein kinase

Review TRENDS in Ecology and Evolution Vol.20 No.2 February 2005

Candidate genes for behavioural ecology

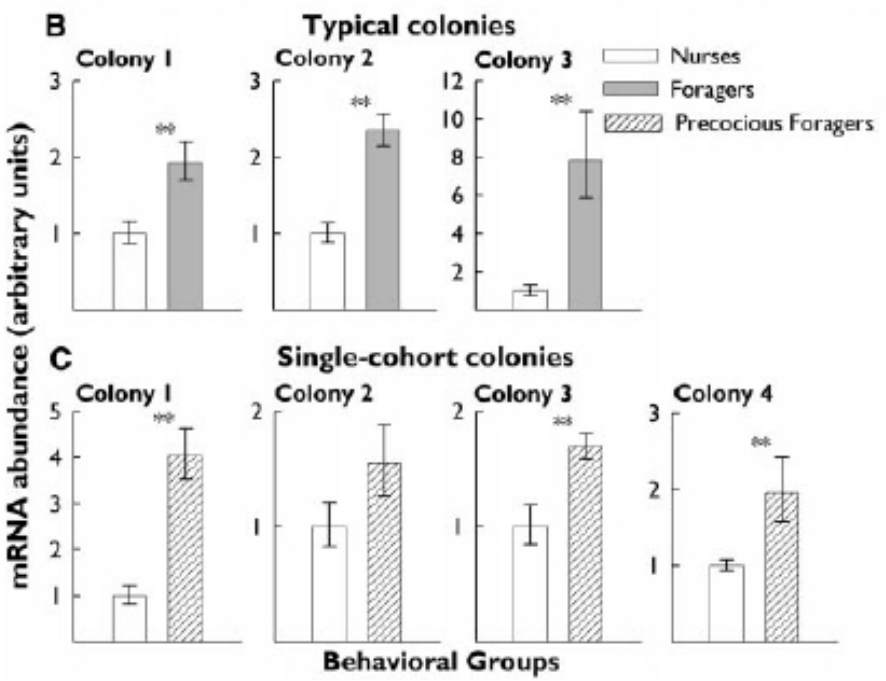
Mark J. Fitzpatrick[1], Yehuda Ben-Shahar[2], Hans M. Smid[3], Louise E.M. Vet[3,4], Gene E. Robinson[5] and Marla B. Sokolowski[1]



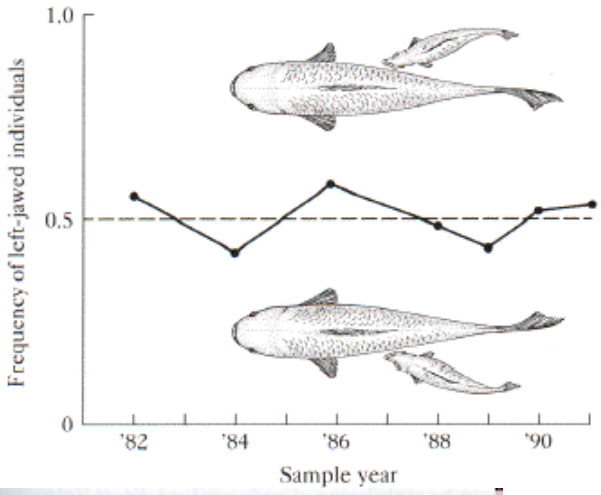
Influence of Gene Action Across Different Time Scales on Behavior

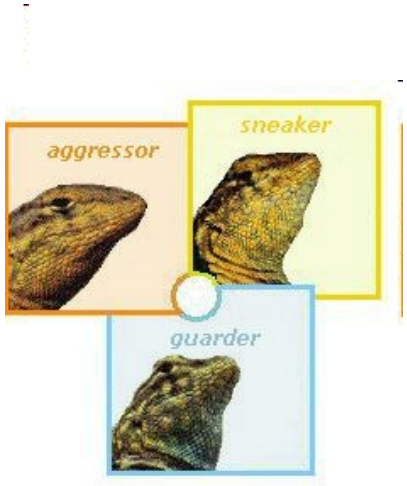
Y. Ben-Shahar,[1] A. Robichon,[3] M. B. Sokolowski,[4] G. E. Robinson[1,2]*

SCIENCE VOL 296 26 APRIL 2002



Fenotypový polymorfismus je určován genetickým polymorfismus spíš pod frekvenčně-závislou selekcí, ale fenotypovou plasticitou (polyfénie) spíš pod cyklickou selekcí





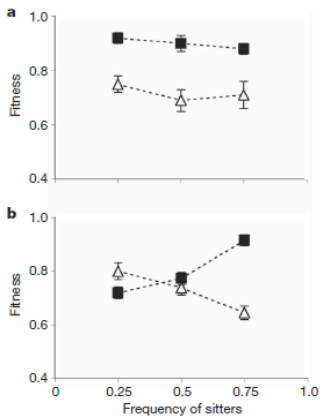


Figure 1 | The effects of frequency and nutrient level on rover and sitter fitness. The *x* axis, plotted as the frequency of sitters, is inversely proportional to the frequency of rovers. **a**, **b**, Rover (for^R, squares) and sitter (for^s, triangles) morphs were reared together under a range of frequencies (3:1, 1:1, 1:3) and on either higher (**a**) or lower (**b**) nutrient abundance media. To facilitate counts, rovers were marked with GFP. Fitness was estimated using the proportion that survived to pupation (mean ± s.e.m.). Sample sizes were 20 (**a**) and 40 (**b**) vials per treatment.

vs.

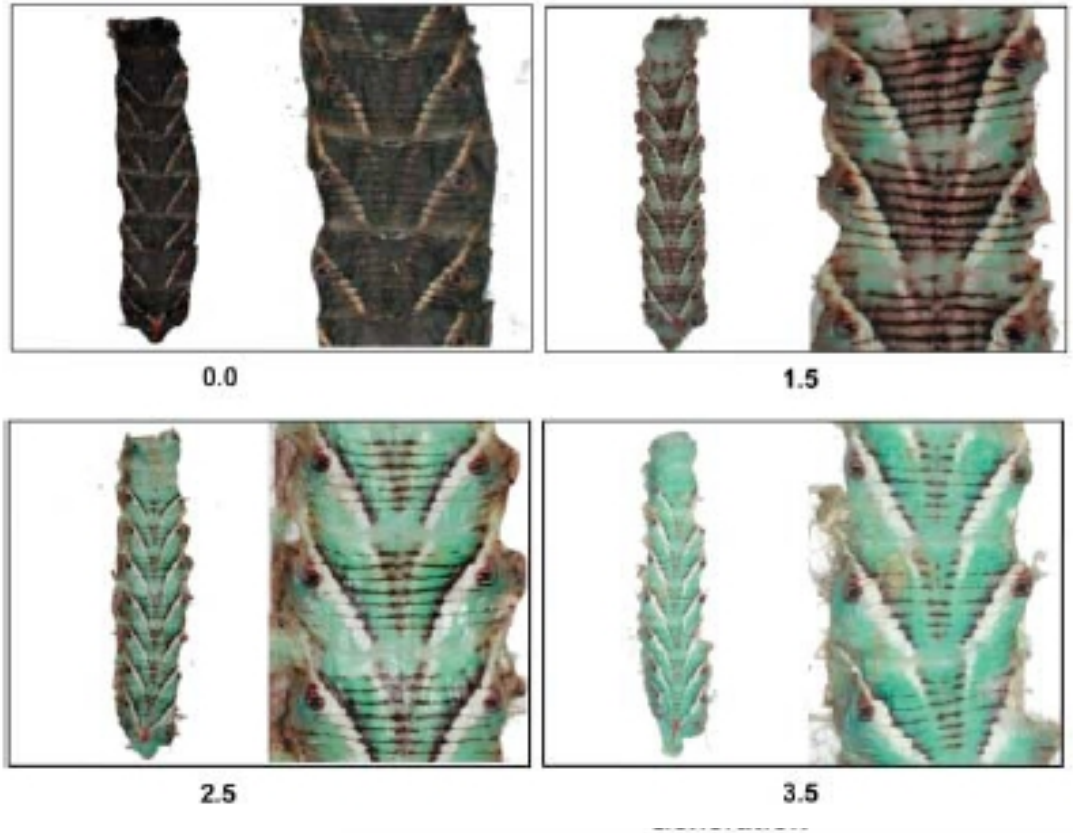
Možný scénář evoluce polyfénie

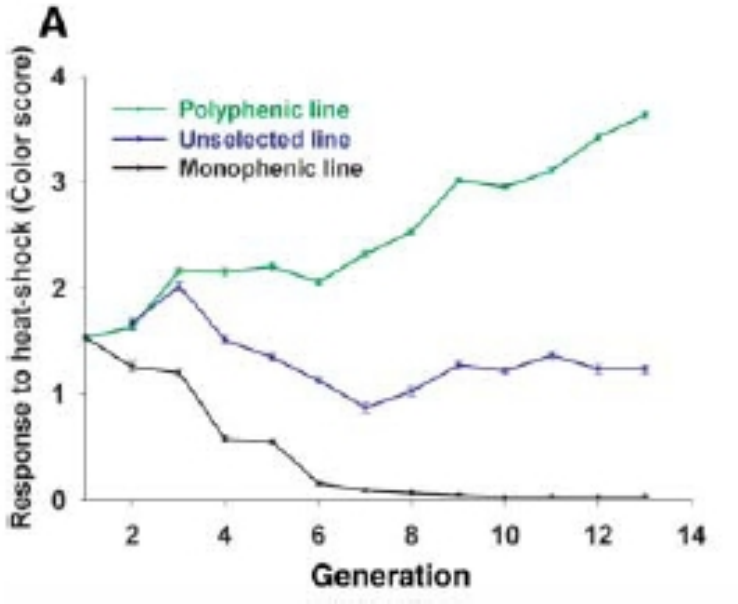
Evolution of a Polyphenism by Genetic Accommodation

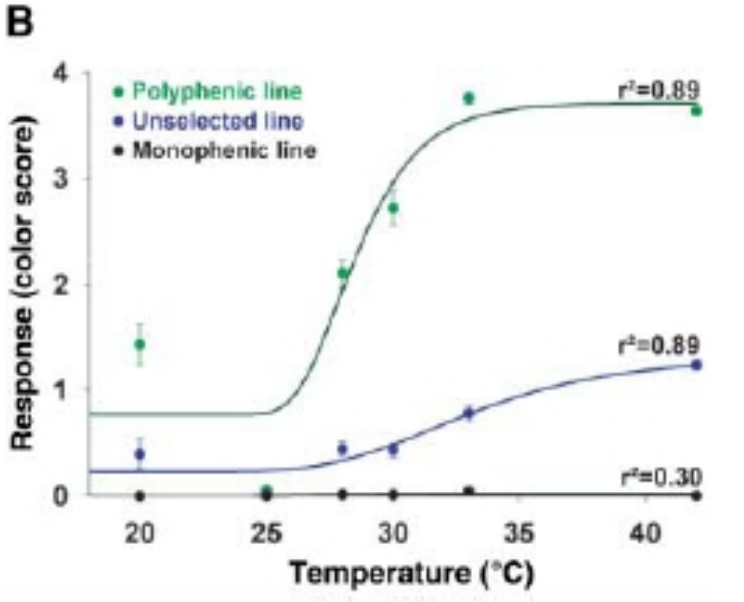
Yuichiro Suzuki* and H. Frederik Nijhout

3 FEBRUARY 2006 VOL 311 **SCIENCE**

Heat-shocked *black* mutant

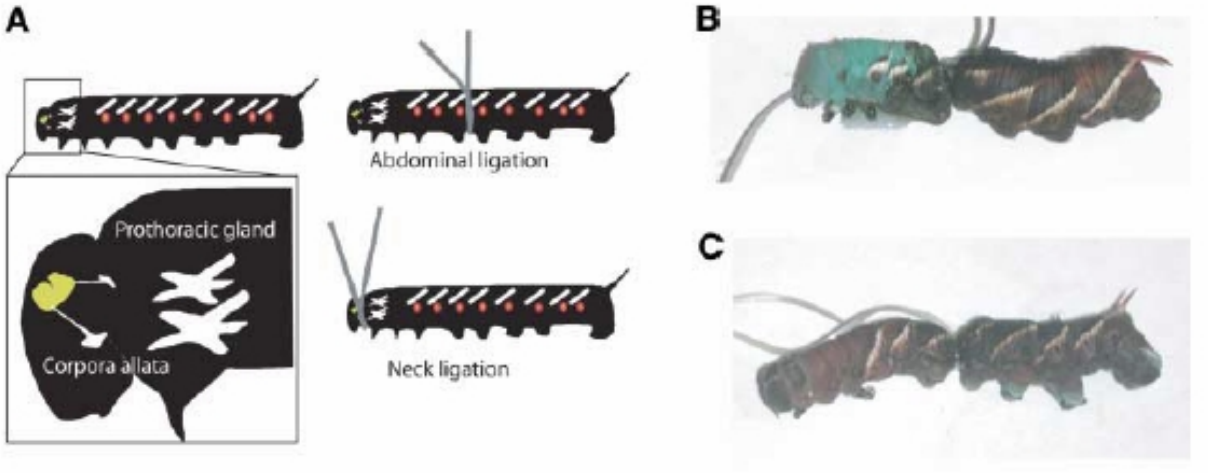




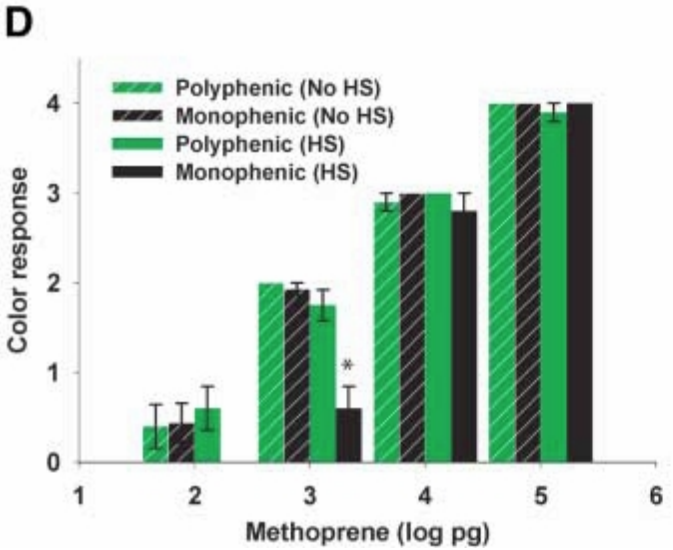


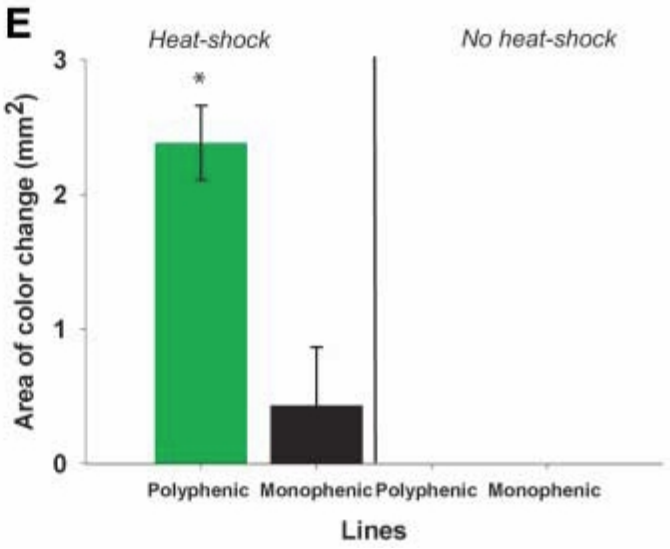
Možný scénář evoluce polyfénie

mozek a corpora allata – produkce např. JH
prothorakální žlázy v thoraxu – produkce např. ekdysonu

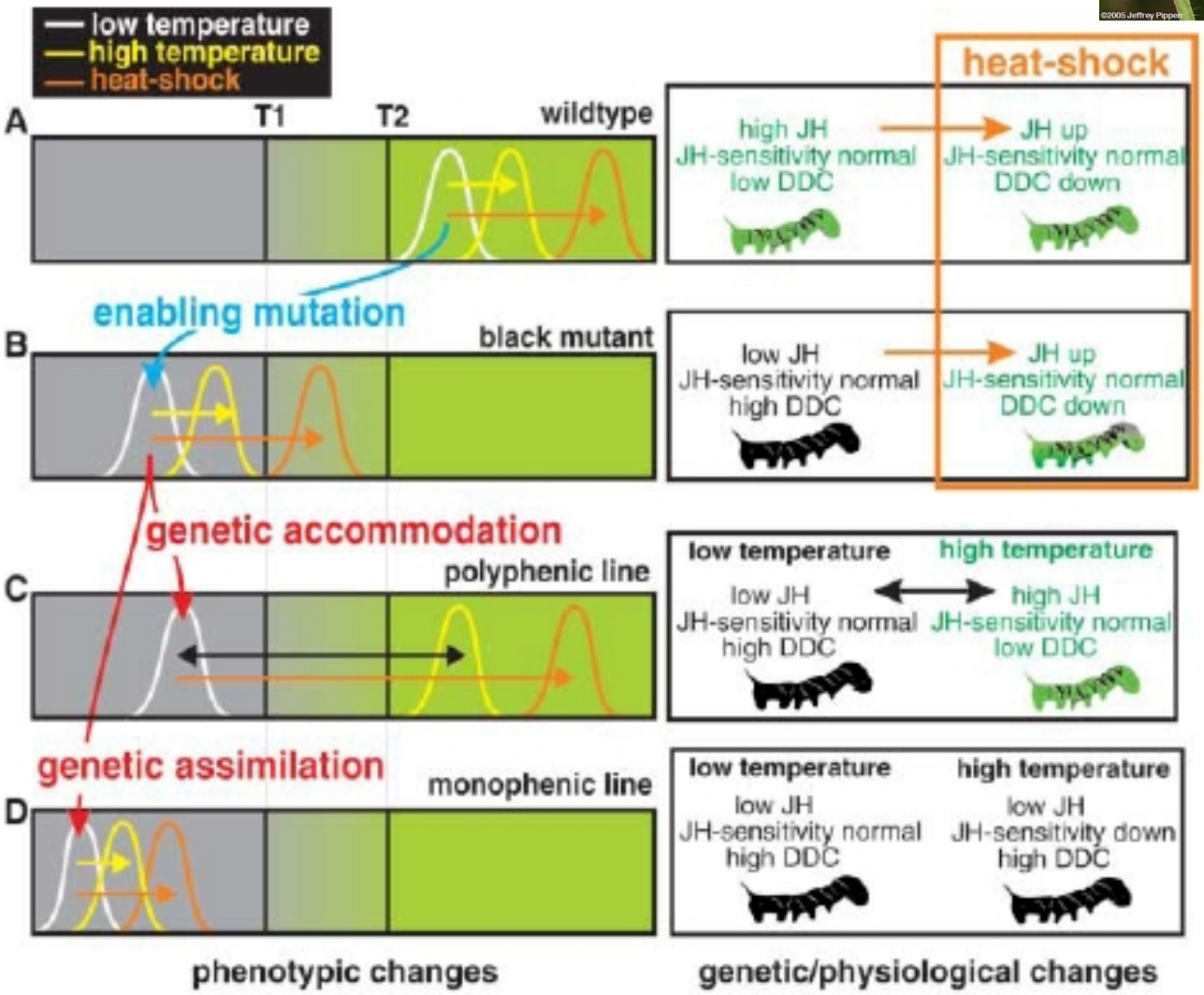


změna barvy je pravděpodobně spojena se změnou v metabolismu JH, ale s jakou:
- sekrece/degradace JH?
- sensitivita na JH?
- molekulární interakce níže v kaskádě spuštěné JH?





Možný scénář evoluce polyfénie



DDC: Dopa-dekarboxyláza, enzym konvertující dopa na dopamin v dráze syntetizující melanin

Fenotypová plasticita je pravděpodobně ancestrální, teprve později vznikla regulace (kanalizace)

Epigenetic mechanisms - conditional, non-programmed determinants of individual development, of which the most important are (1) interactions of cell metabolism with the physicochemical environment within and external to the organism, (2) interactions of tissue masses with the physical environment on the basis of physical laws inherent to condensed materials, and (3) interactions among tissues themselves, according to an evolving set of rules.

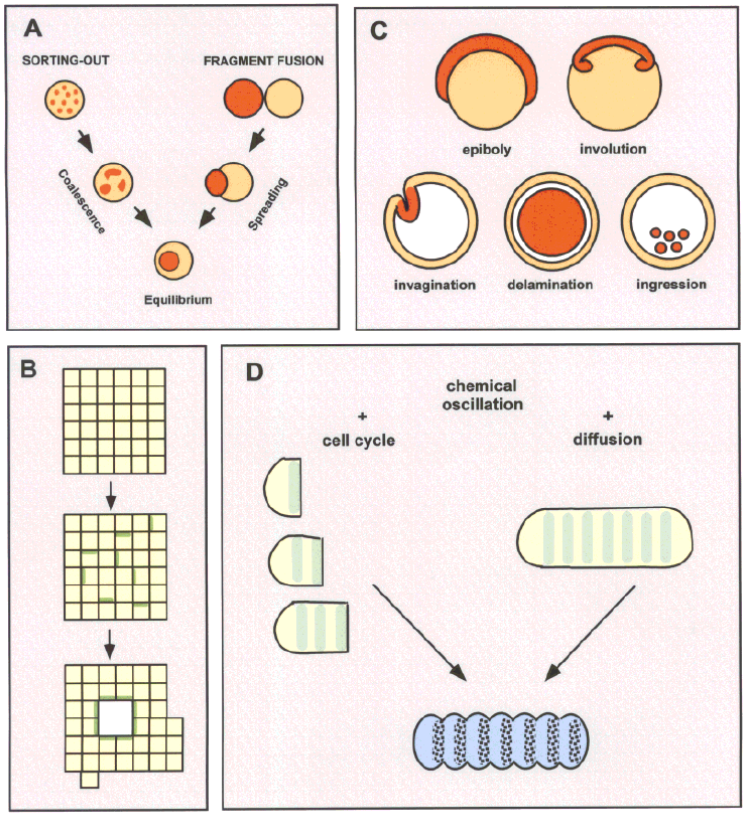


Figure 1.

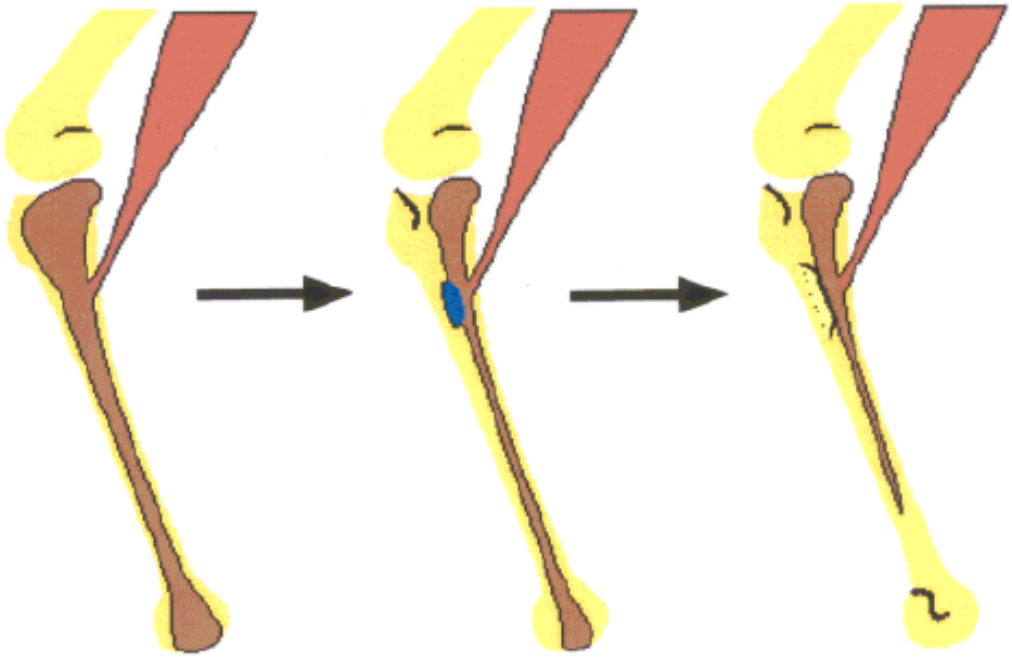
Epigenetic Mechanisms of Character Origination

STUART A. NEWMAN[1]* AND GERD B. MÜLLER[2,3]

JOURNAL OF EXPERIMENTAL ZOOLOGY (MOL DEV EVOL) 288:304–317 (2000)

Fig. 1. Generic processes in tissue morphogenesis. **A**: Schematic representation of the behavior of intermixed cells and corresponding tissue fragments in the case where the two cell populations are differentially adhesive. The cell mixture will sort out as the more adhesive cells establish more stable bonds with one another than with cells of the other population. Random motion leads to the formation of cohesive islands of these cells, and these will ultimately coalesce into a separate tissue phase, or compartment. The equilibrium configuration of the cell mixture is identical to that which would be formed by fusion and spreading of fragments of tissue consisting of the same differentially adhesive cell populations. **B**: Schematic view of formation of a lumen or internal cavity by differential adhesion in an epithelioid tissue consisting of polarized cells. In the original state (top) the cells are uniformly adhesive, and make contacts around their entire peripheries. Upon expression of an anti-adhesive protein (green) in a polarized fashion in a random subpopulation of cells (center), and random movement of the cells throughout the mass, bonds between adhesive surfaces are energetically favored over those between adhesive and nonadhesive surfaces, resulting in lumen formation (bottom). **C**: Schematic cross-sectional views of the five main types of gastrulation. In each case a new population of cells differentiates from a solid or hollow embryo and assumes a position that would be attained by a similarly situated differentially adhesive population. **D**: Schematic representation of two modes of tissue segmentation that can arise when the tissue's cells contain a biochemical circuit that generates a chemical oscillation or "molecular clock," and the oscillating species directly or indirectly regulates the strength or specificity of cell adhesivity. In the mechanism shown on the left, the periodic change in cell adhesivity occurs in a growth zone in which the cell cycle has a different period from the regulatory oscillator; as a result, bands of tissue are sequentially generated with alternating cohesive properties. In the mechanism shown on the right, one or more of the biochemical species can diffuse, leading to a set of standing waves of concentration of the regulatory molecule by a reaction-diffusion mechanism. This leads to the simultaneous formation of bands of tissue with alternating cohesive properties. See Newman, '93 for additional details. (A, with changes, from Steinberg, '98; B after Newman and Tomasek, '96)

Důležitost mechanických stimulů a integrace epigenetických procesů do vývinu



Origin of the “fibular crest” in archosauran hindlimbs by mechanical regulation of mesenchymal morphogenesis. Progressive evolutionary reduction of the fibula increases the mechanical load on the connective tissue between the tibia (yellow) and the fibula (brown), exerted by the pulling action of the iliofibularis muscle (red). A stress-dependent cartilage (blue) forms in response and becomes later incorporated into the ossifying tibia to form a prominent crest (stippled), a homologue shared by theropod dinosaurs and carinate birds. This tight fixation of the proximal fibula permits its further distal reduction in avian limbs.

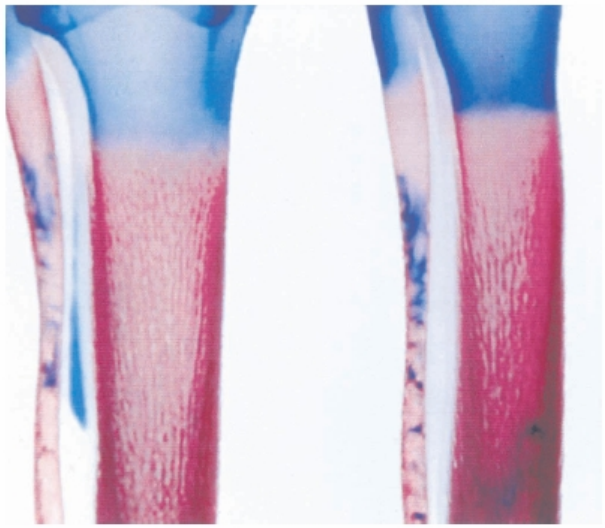
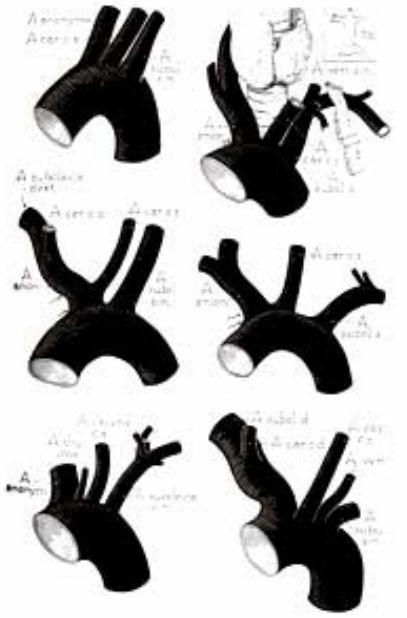
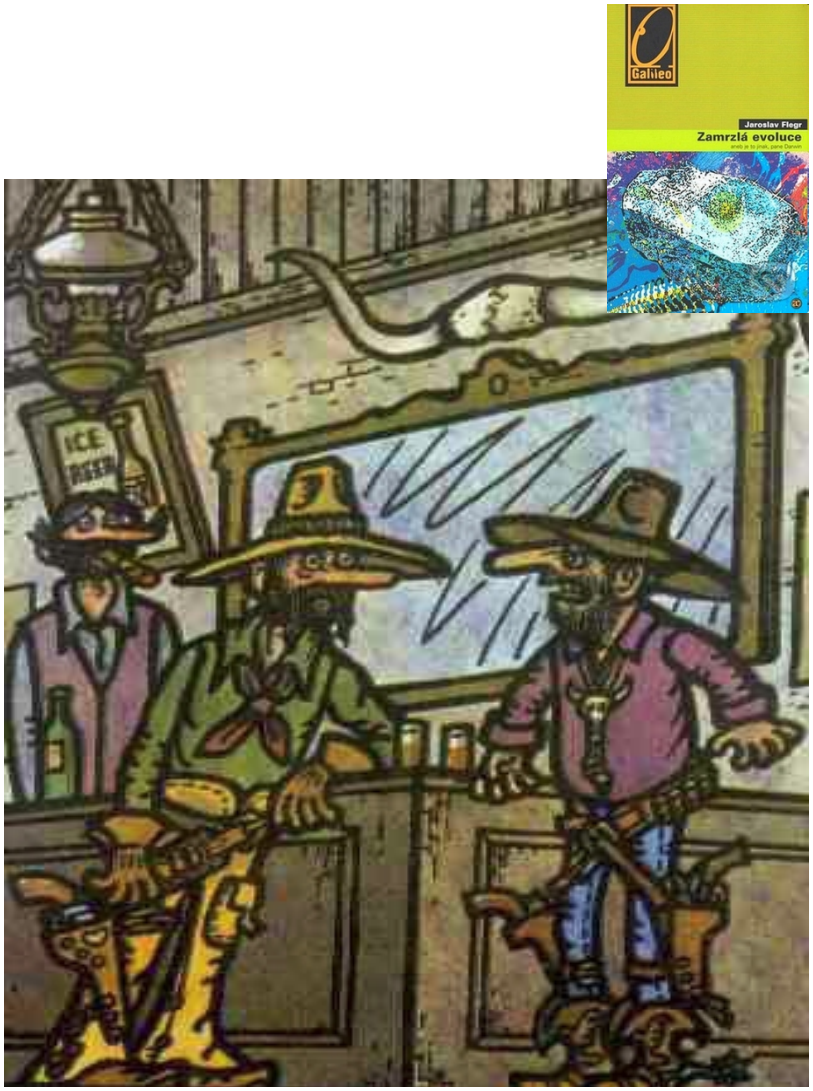


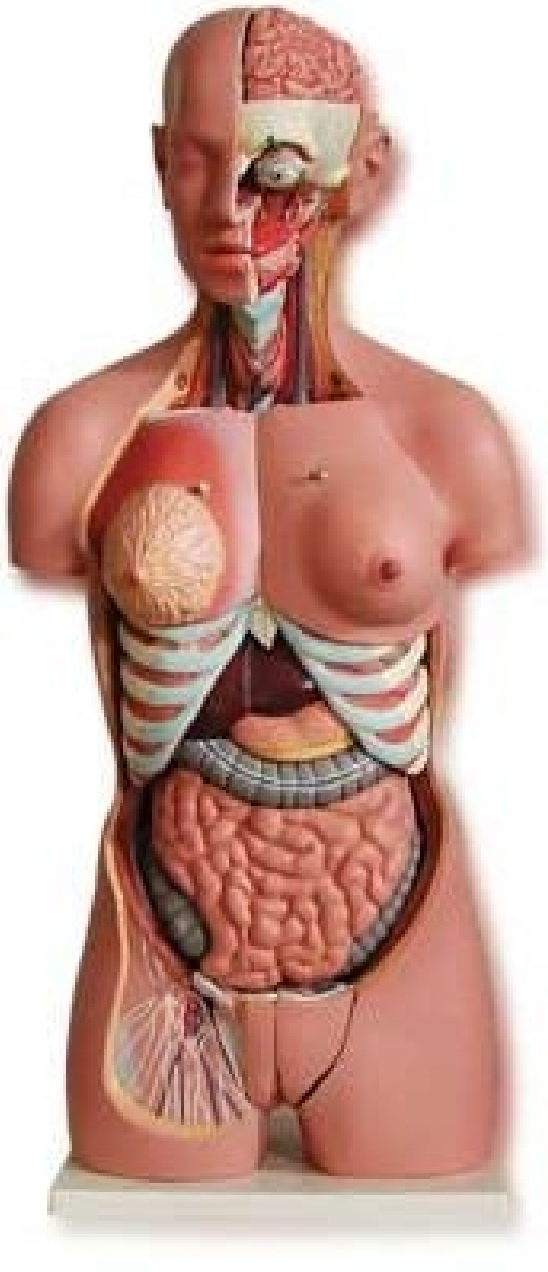
Fig. 5. Complete aplasia of the crest cartilage in a paralyzed chick embryo. Left, control.



Může být mutant s radikálně pozměněným fenotypem životaschopný?



Tak mi laskavě vysvětli, k čemu jsou mi dobrý kolty proklatě nízko, když mám krátký ruce!



Důležitým faktorem prostředí může být velikost vajíčka

EVOLUTION & DEVELOPMENT 11:6, 728–739 (2009)
DOI: 10.1111/j.1525-142X.2009.00380.x

Evolutionary and experimental change in egg volume, heterochrony of larval body and juvenile rudiment, and evolutionary reversibility in pluteus form

Douglas F. Bertram,[a] Nicole E. Phillips,[b] and Richard R. Strathmann[c,*]

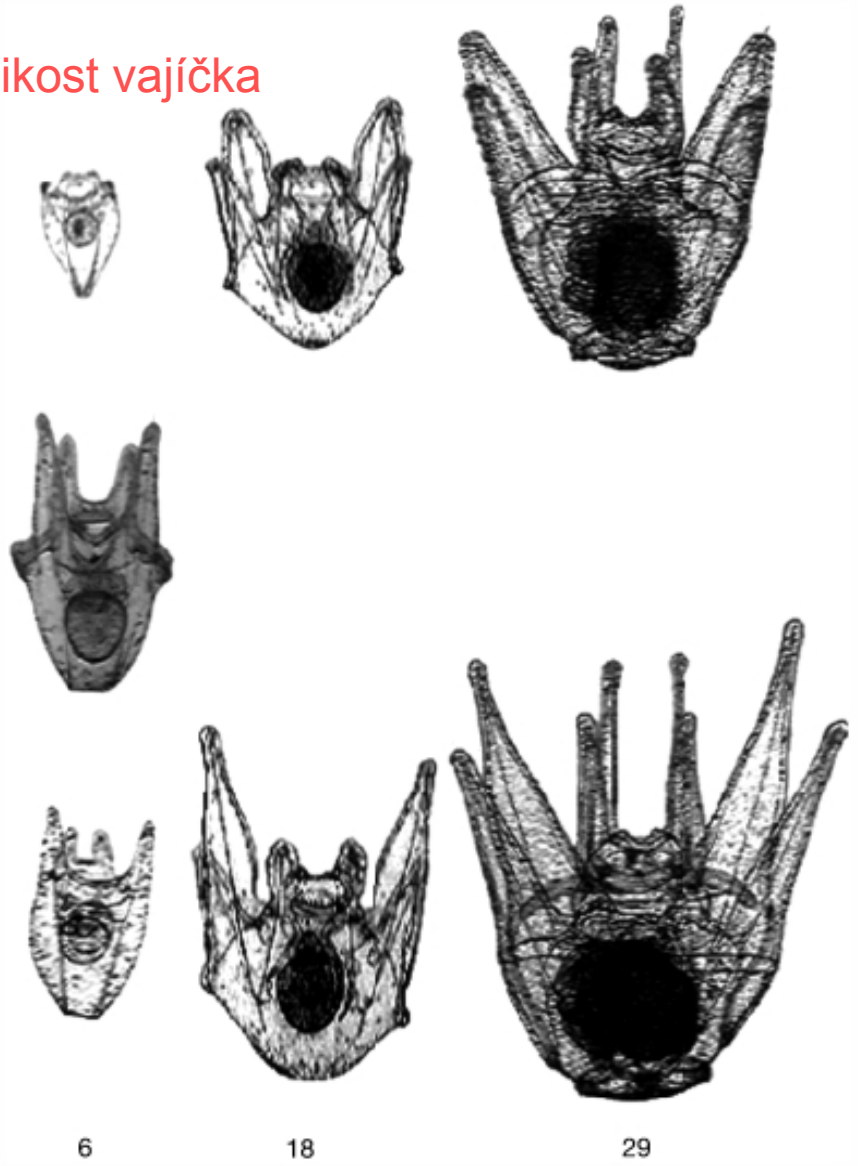


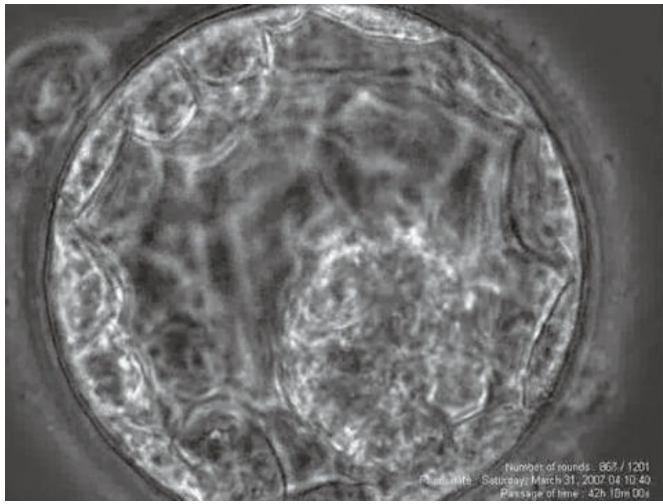
Fig. 8. Larvae of *Strongylocentrotus purpuratus* from single eggs (top row) and two fused eggs (bottom row) at 6, 18, and 29 days of age. Larva of *Strongylocentrotus droebachiensis* from a single egg (middle row) at 6 days of age.

… či prostředí v děloze

An Effect of the Uterine Environment upon Skeletal Morphology in the Mouse

ANNE MCLAREN & DONALD MICHIE

Nature **181**, 1147 - 1148 (19 April 1958);
doi:10.1038/1811147a0



Je fenotypová plasticita v daném znaku adaptivní?

- někdy ano (polyfénie)
- někdy neutrální
- někdy maladaptivní

- náklady a omezení adaptivní fenotypové plasticity:

Box 1. Nine potential costs and limits of phenotypic plasticity

Costs of plasticity

- *Maintenance costs*: Energetic costs of the sensory and regulatory mechanisms of plasticity.
- *Production costs*: The production cost of inducible structures has been viewed by some as a cost of plasticity. Other authors disagree because production costs are also paid by fixed genotypes. In some cases, the production costs that plastic genotypes pay will exceed those paid by fixed genotypes; the excess is a true cost of plasticity.
- *Information acquisition cost*: The process of acquiring information about the environment may be risky, involve energy for sampling, or reduce foraging or mating efficiency.
- *Developmental instability*: Phenotypic imprecision may be inherent for environmentally contingent development. Imprecision can result in reduced fitness under stabilizing selection.
- *Genetic costs*: (1) Linkage – genes promoting plasticity may be linked with genes conferring low fitness. (2) Pleiotropy – plasticity genes may have negative pleiotropic effects on traits other than the plastic trait. (3) Epistasis – regulatory loci producing plasticity may modify expression of other genes.

Limits to the benefit of plasticity

- *Information reliability limit*: Plastic organisms can produce maladapted phenotypes when they are wrong about the environment, or, when correct initially but the environment changes.
- *Lag-time limit*: A plastic strategy must invoke development to alter phenotypes. The lag-time between an environmental change and a phenotypic response can reduce fitness.
- *Developmental range limit*: Fixed development may be more capable of producing adaptive, extreme phenotypes than facultative development.
- *Epiphenotype problem*: Plastic add-on phenotypes may be ineffective compared with the same phenotypic element that is integrated during early development.

Costs and limits of phenotypic plasticity

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Toward a population genetic framework of developmental evolution: the costs, limits, and consequences of phenotypic plasticity

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Shrnutí

- Změny navozené prostředím (fenotypová plasticita) mohou být podobné jako změny genetické
- Genetický polymorfismus vedoucí k polymorfismu ve fenotypu se patrně udržuje jinými evolučními mechanismy než adaptivní fenotypová plasticita
- Polyfénie se často mohla vyvinout díky genetické akomodaci
- Fenotypová plasticita je univerzální jev, může být přímým důsledkem změn ve fyzikálně-chemických podmínkách prostředí
- Ne každá fenotypová plasticita je adaptivní
- Omezení a limity jsou důležitým faktorem evoluce adaptivní fenotypové plasticity