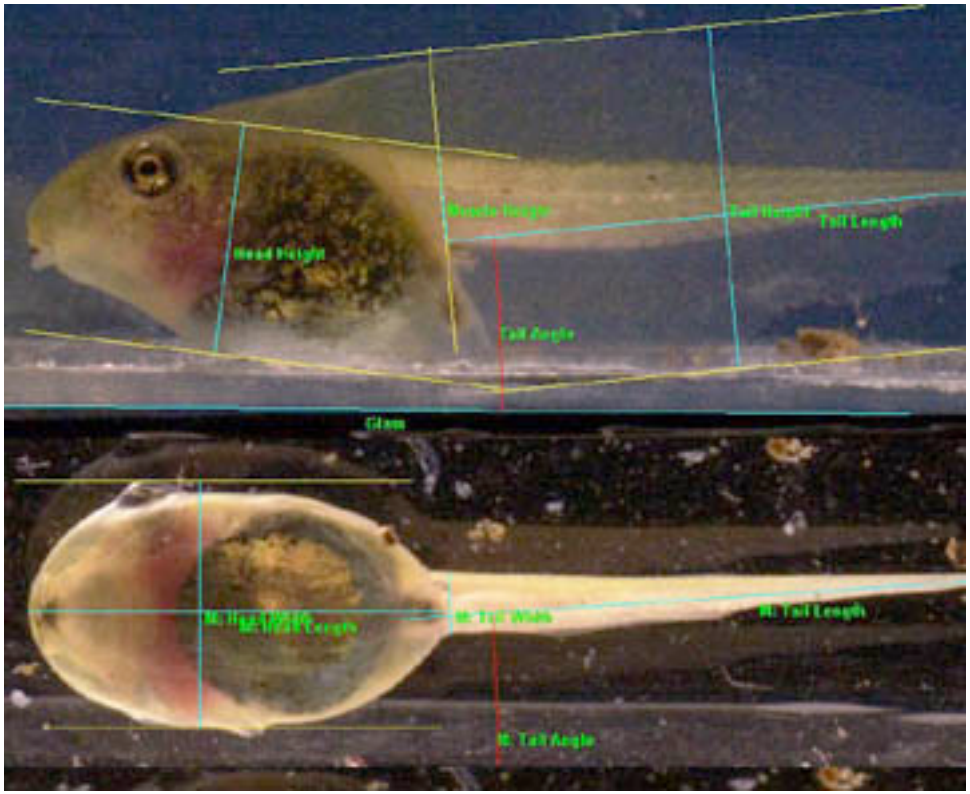
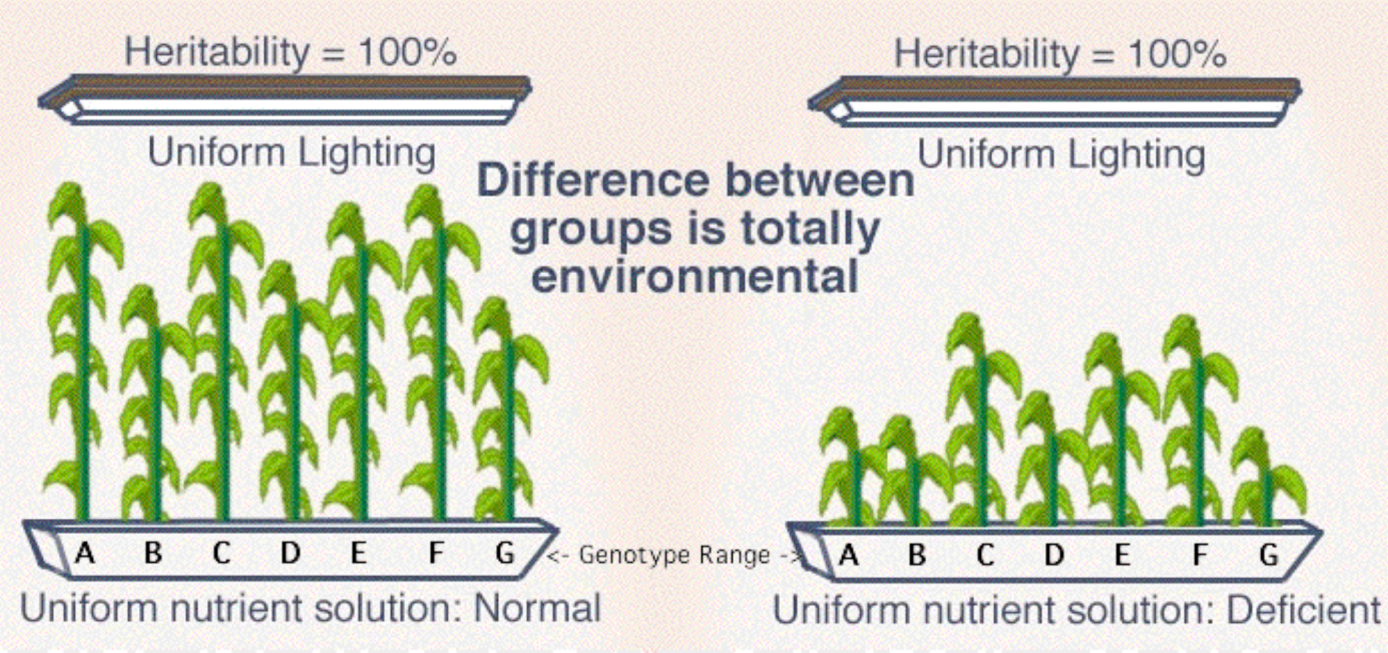
PLASTICIDAD FENOTIPICA



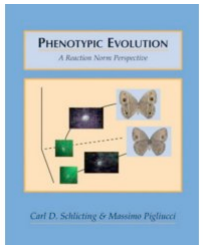
Phenotypic plasticity

The environmentally sensisity production of alternative phenotypes by given genotypes.

Environment-dependent phenotype expression



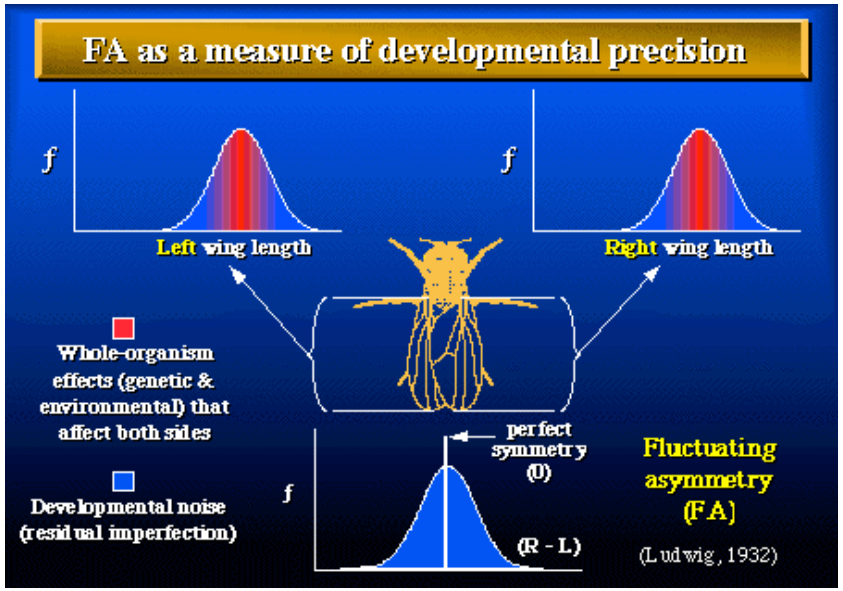
Schilling y Pigliucci 2004. Phenotypic evolution: a reaction norm perspective

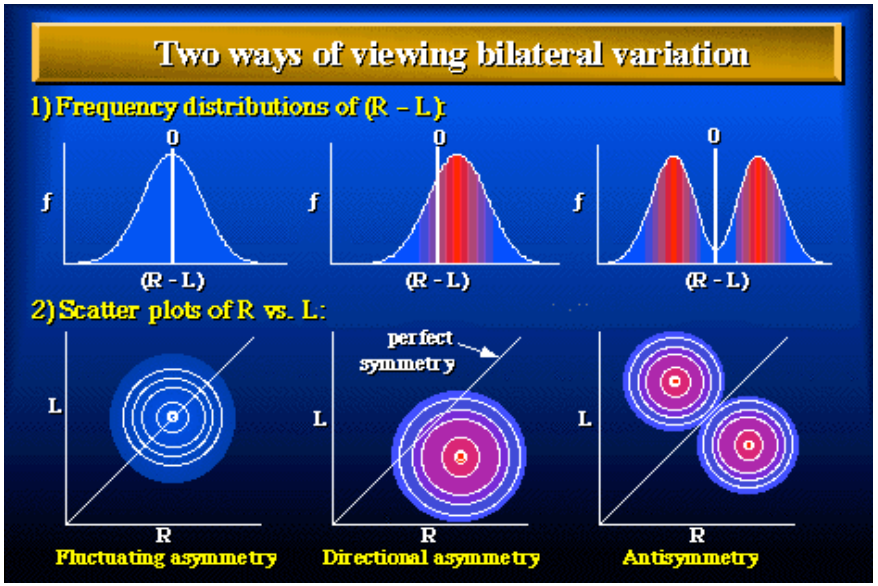


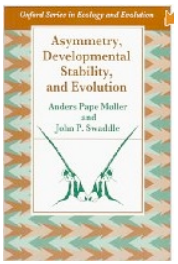
Phenotypic plasticity vs. Developmental noise

Developmental noise are random fluctuations arising during development and that alter the phenotypic product of development.

Fluctuatingasymmetry: Deviation from perfect bilateral symmetry caused by environmental stresses, developmental instability and genetic problems during development







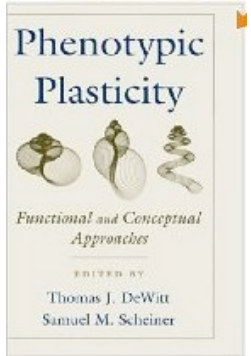
Moller & Swaddle 1998. *Asymmetry, Developmental Stability, and Evolution*.OUP

Plasticidad fenotípica

Table 1. Mutually exclusive definitions of the four most commonly historically used categories of phenotypic plasticity[a,b]

Plasticity category	Phenotypic change is reversible	Variability occurs within a single individual	Phenotypic change is seasonally cyclic[b]
Developmental plasticity	No	No	No
Polyphenism[c]	No	No	Yes
Phenotypic flexibility	Yes	Yes	No
Life-cycle staging[c]	Yes	Yes	Yes

[a]Phenotypic plasticity itself indicates the general capacity for change or transformation within genotypes in response to different environmental conditions.
[b]Previous workers might have used less restrictive definitions.
[c]Can be regarded as a subcategory of developmental plasticity; life-cycle staging is a subcategory of phenotypic flexibility.



DeWitt y Scheiner 2004. Phenotypic plasticity: functional and conceptual approaches. OUP

Developmental plasticity: *Gasterosteus aculeatus*

Polyphenism: *Pontia spp.*

Summer

Spring

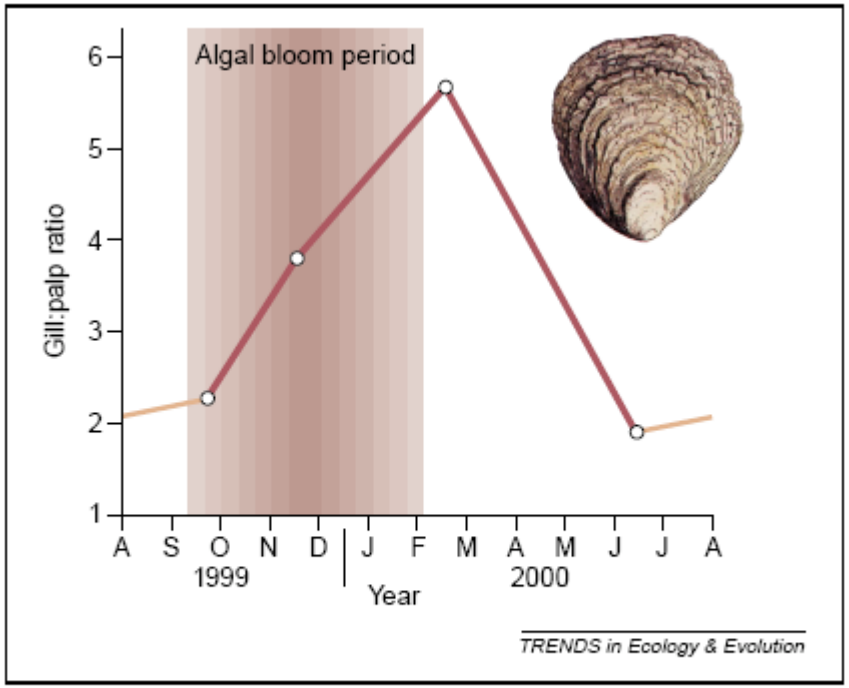


Fig. 2. Seasonal cycle of the gill:palp ratio in Pacific oysters *Crassostrea gigas* in eastern Australia in relation to the occurrence of blooms of their phytoplankton food (shaded area). Based on data from [36].

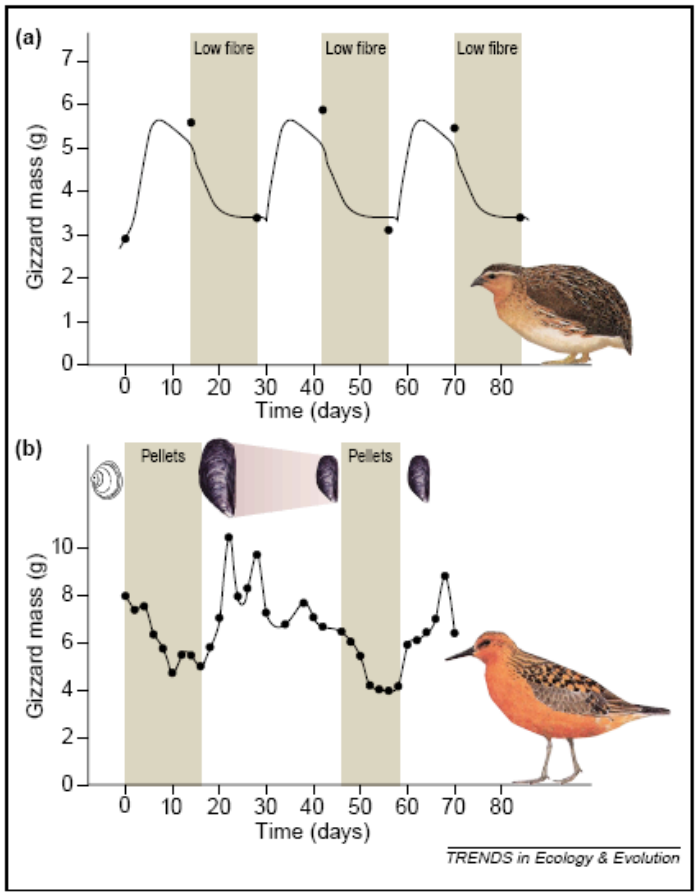


Fig. 3. Reversible size changes in the gizzards of adult Japanese quail *Coturnix japonica* (a) and red knots *Calidris canutus* (b). The quail were given a diet of alternately low or high nondigestible fibre content (3% and 45%, respectively). Within 14 days, they showed a doubling of the size of the gizzard. Red knots are specialized molluscivore shorebirds with strong muscular gizzards, which they use to crush ingested hard-shelled prey. With a change in diet from medium–small mussels *Mytilus edulis* ingested whole (the smallest size classes are easiest to crush) to a diet of soft food-pellets, gizzard mass halved within the first eight days following the diet shift. Shifts from a pellet to a mussel diet induced doublings of gizzard mass within even shorter periods of time. The data for quail were based on the dissection of samples on the day of the diet switch, whereas, in red knots, gizzard size of individuals was monitored with the use of ultrasonography. In quail, the precise time course of size changes was also studied with the use of ultrasonography, but only in one group. The pattern found in the latter group is repeated in (a) to illustrate the probable time course of the changes. Based on data from [41] and [42], respectively.

Phenotypic plasticity & Quantitative Genetics

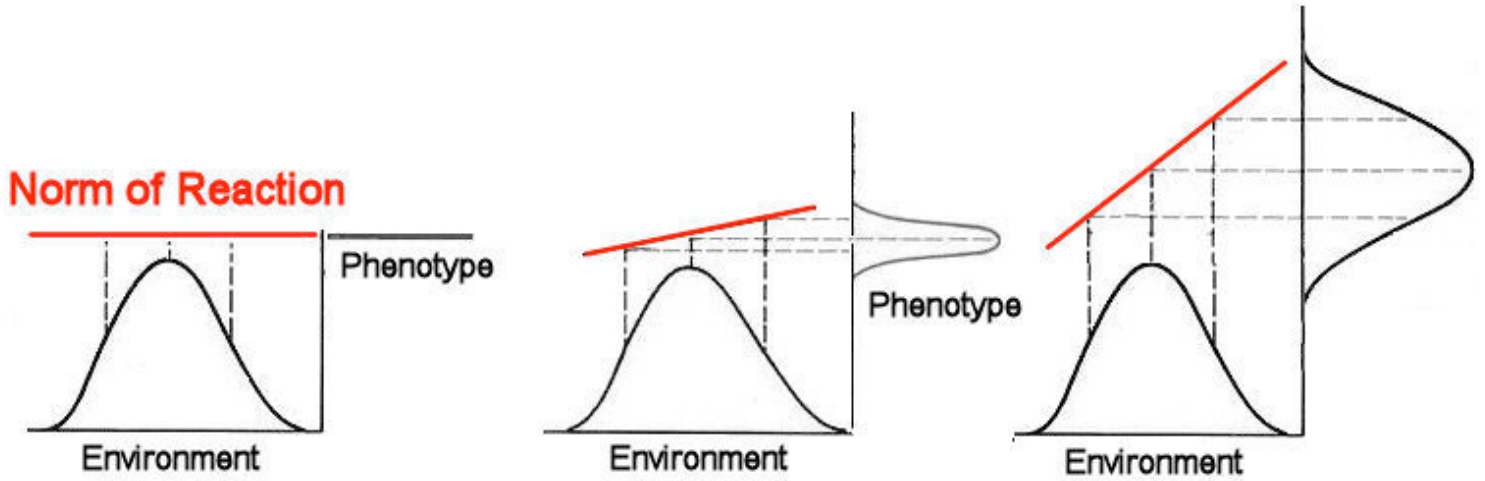
Table 1. The interpretation of canalization, phenotypic plasticity and developmental stability in a quantitative genetic context

	Partition of phenotypic variance[a]	Genetic mechanisms	Effect on variance
Genetic canalization	$V_P = V_A + V_{NA} + V_E$	Epistatic modifiers (genetic-canalizing genes)	Negative on V_A
Environmental canalization	$V_P = V_A + V_{NA} + V_E$	Environment-canalizing genes	Negative on V_E
Phenotypic plasticity	$V_P = V_A + V_{NA} + V_{\mu E} + V_{ME}$	Gene expression dependent on macroenvironment	Positive on V_{ME}
Developmental stability	$V_P = V_{Ind} + V_{within}$	Genes controlling homogeneous development of homologous body parts	Negative on V_{within}

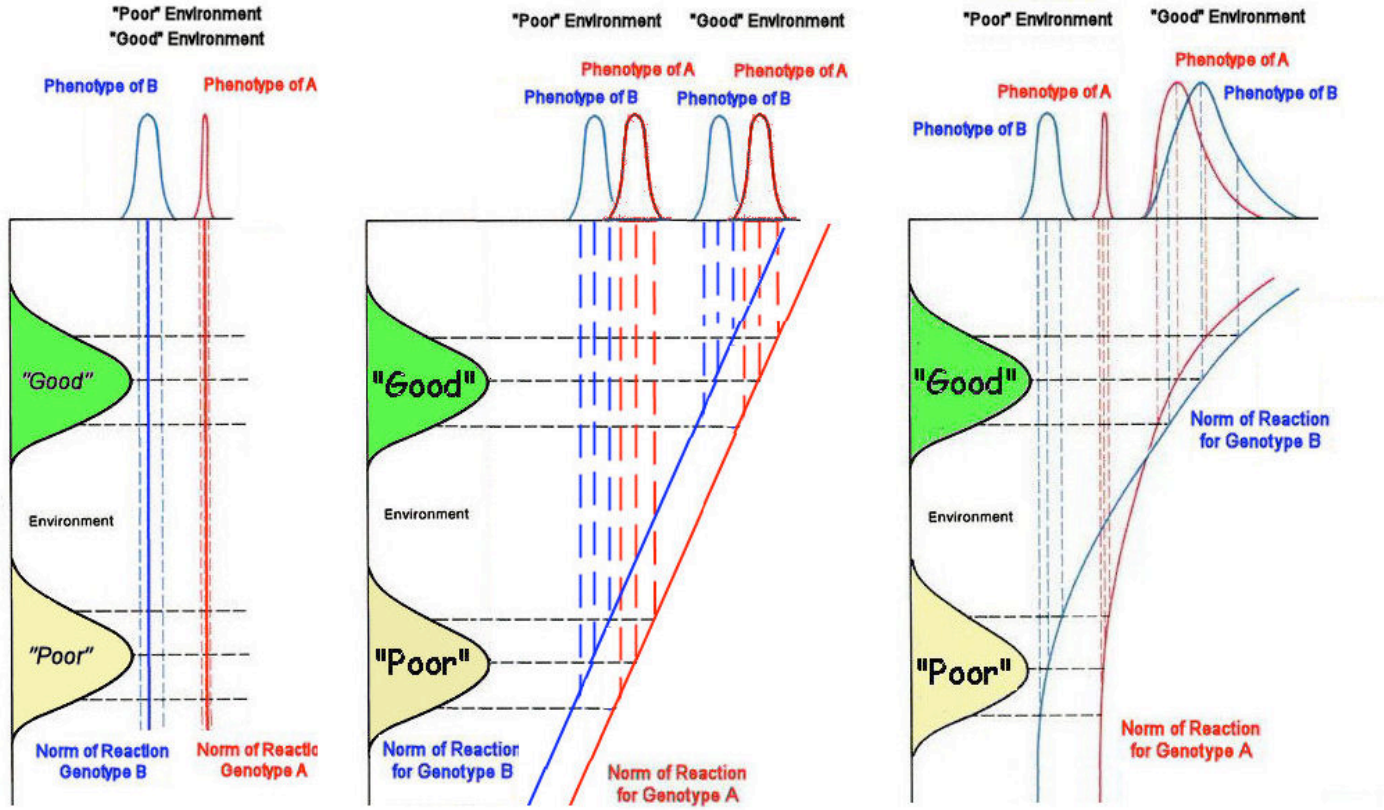
[a] V_P, phenotypic variance; V_A, additive genetic variance; V_{NA}, nonadditive genetic variance; V_E, environmental variance; $V_{\mu E}$, microenvironmental variance; V_{ME}, macroenvironmental variance; V_{Ind}, among-individual variance; V_{within}, within-individual variance.

Norm of reaction

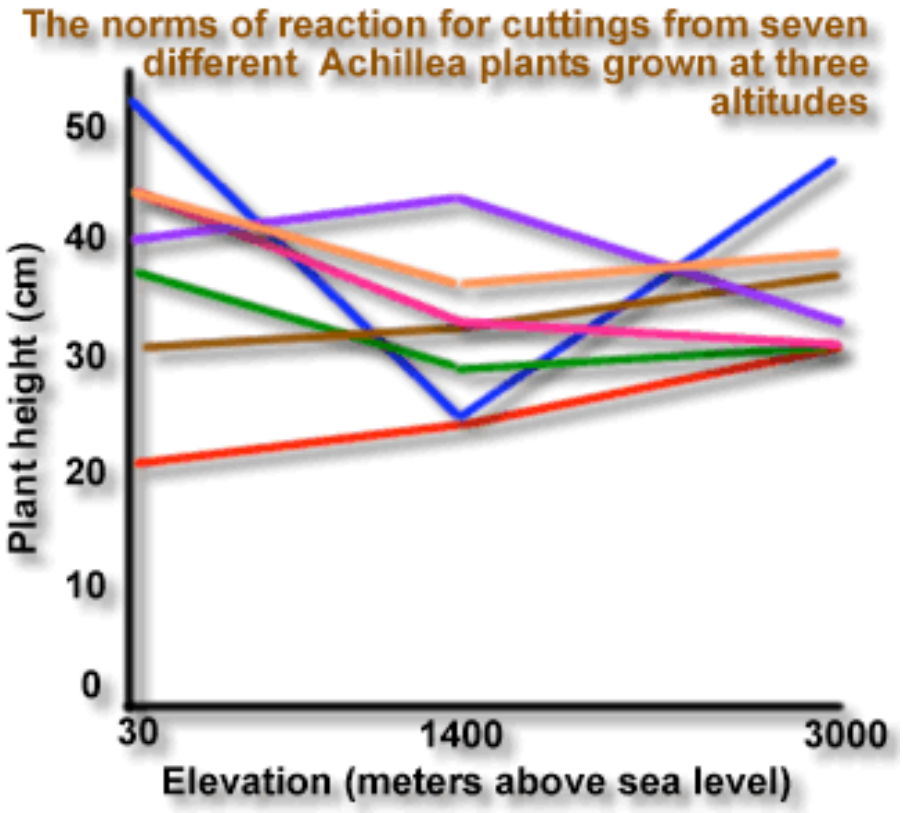
es una función específica del genotipo que relaciona a cada fenotipo con el ambiente en el que ha sido producido



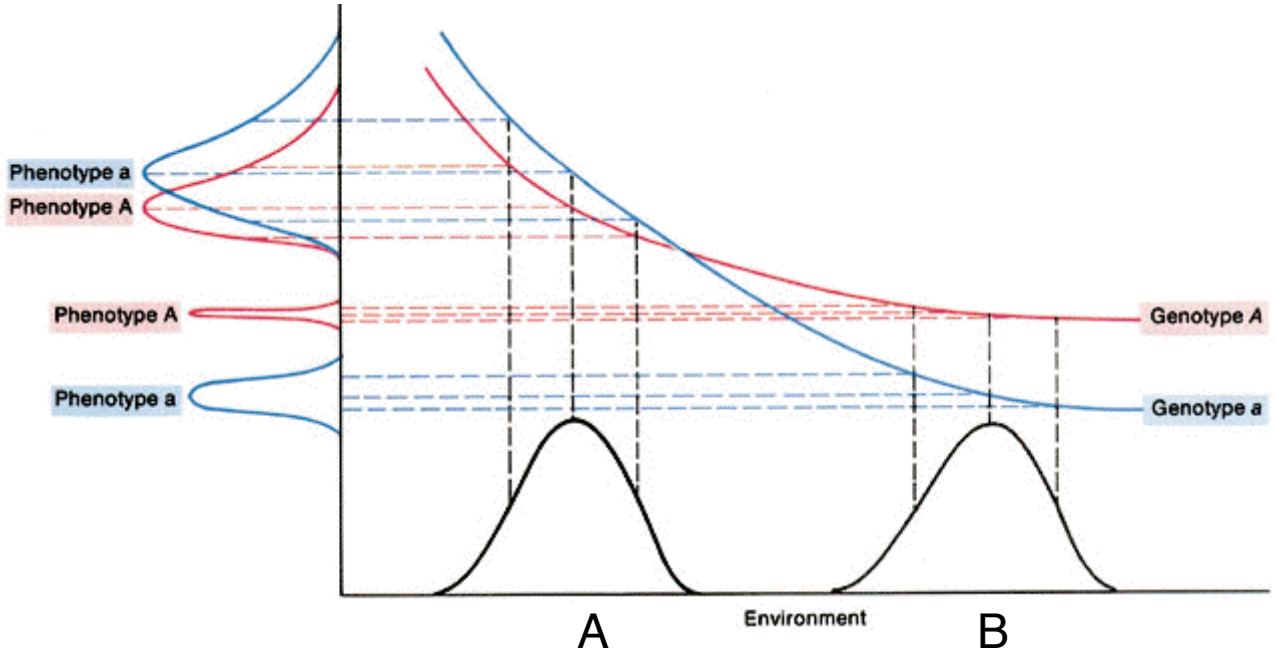
Norm of reaction



Norm of reaction

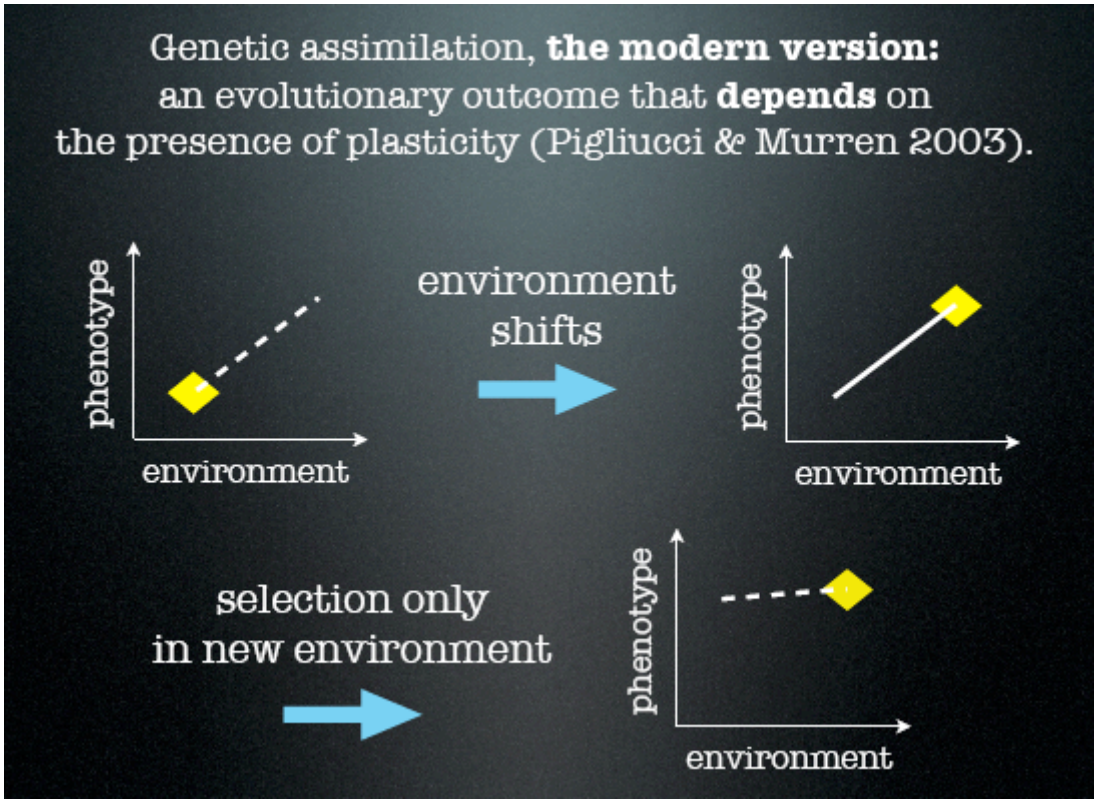


Relación heredabilidad-plasticidad



$h^2_B >> h^2_A$

Genetic assimilation via phenotypic plasticity



Phenotypic plasticity, provides the intermediate step for the process of genetic assimilation, as illustrated in this sequence of diagrams. The initial reaction norm (top left) is such that when the organism is exposed to a new environment (top right) it produces a different/novel phenotype without any genetic change. Eventually, natural selection will “stabilize” the new phenotype, if adaptive under the new conditions (the phenotype becomes “assimilated”), and the organism might even loose its plasticity (bottom) due to genetic drift, or even because of selection, if there is a cost to maintain a plastic reaction norm when the old environment is no longer ecologically relevant.

Adaptive phenotypic plasticity

Aquella Plasticidad fenotípica mantenida por selección natural

Requisitos para que ocurra PF adaptativa:

1) Interacción genotipo-ambiente

2) Un genotipo plástico funciona mejor que uno no plástico en todas las condiciones

3) El fitness promedio de un fenotipo plástico es mayor que el de un fenotipo no plástico

Adaptive phenotypic plasticity

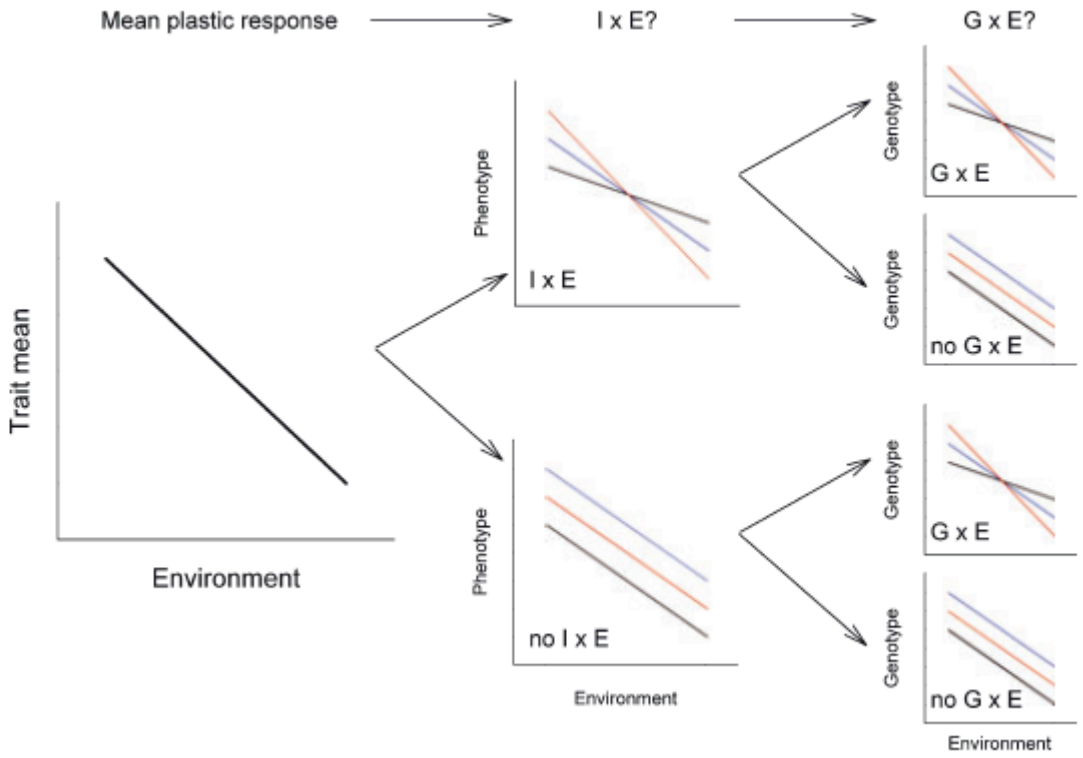


Fig. 1 Three different levels of analysis are involved in understanding individual plasticity in labile life history traits. Cross-sectional studies examine population-level responses to the environment (left most graph), which can be underpinned by variation between individuals in their plastic response to the environment (I × E) or not. The presence or absence of I × E can itself be accompanied by underlying variation in plasticity at the additive genetic level (G × E) or not.

Adaptive phenotypic plasticity

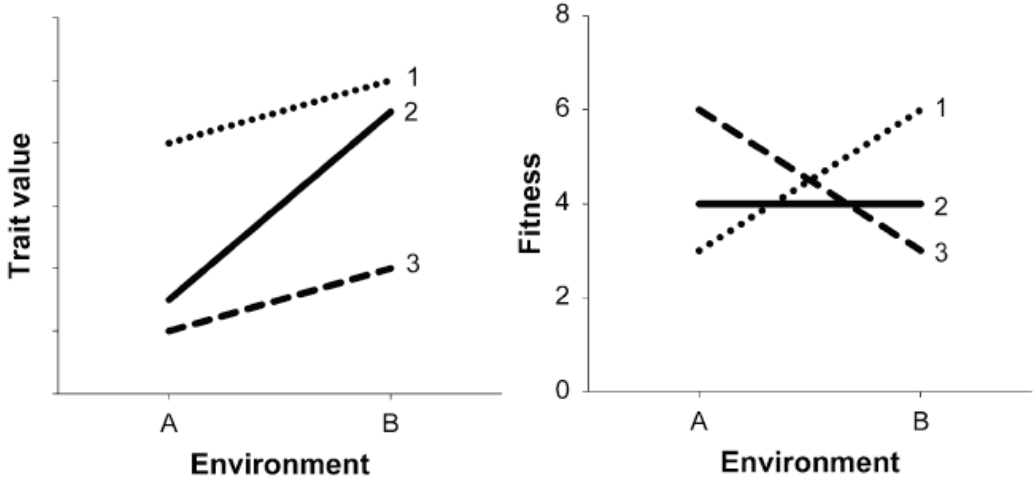


Fig. 2 Illustration of a situation where selection pressures on a trait in two different environments are opposing suggesting that plasticity would be beneficial but where overall there is no selection for phenotypic plasticity in this trait. Summed over both environments the fitness of the plastic genotype 2 (fitness = 8) is lower than the one of the less plastic genotypes 1 and 3 (fitness = 9). This might be a consequence of nonlinear relations between the trait and fitness or costs of plasticity.

Adaptive phenotypic plasticity

PF adaptativa evoluciona cuando:

1) Ambiente heterogéneos

2) Señales ambientales fácilmente interpretables

3) Ausencia de “super-fenotipos” que ganan en todos los ambientes

4) Bajo costo de la plasticidad

Costs of phenotypic 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.

De Witt et al. 1998: TREE 13: 77-79

Ecological costs. External ecological factors constraining phenotypic plasticity.

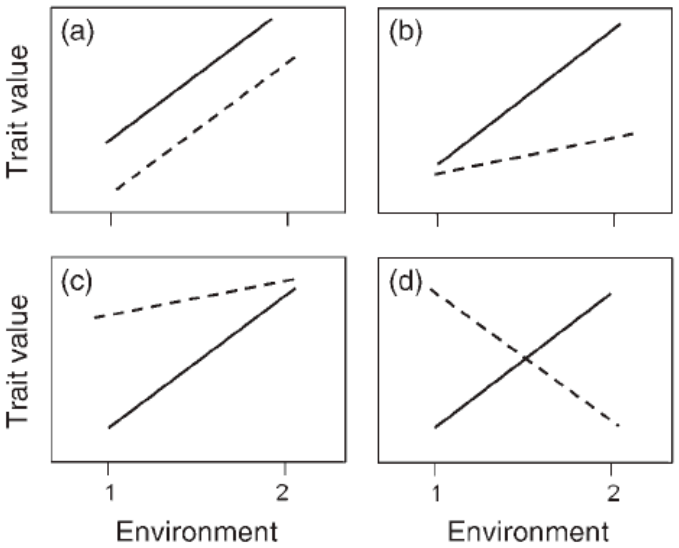


Fig. 4 Scenarios of constraints on plasticity imposed by biotic factors (herbivory). The panels show the reaction norms in the absence (continuous line) and presence (dashed line) of herbivores. It is assumed that the continuous line depicts the functionally optimal response of the plant to the environment (i.e. the phenotype of greatest fitness in each environment). In panel a there is no effect of herbivory on the slope of the reaction norm (i.e. on plasticity) but its elevation is changed, causing a slight departure from the optimal phenotype in each environment. In panels b and c there is a significant reduction in the slope of the reaction norm caused by herbivores. Whereas in b reduced plasticity results in a maladaptive phenotype in environment 2, in c reduced plasticity causes a maladaptive phenotype in environment 1. In d there is a change in the direction, but not in the slope, of the reaction norm, causing a maladaptive phenotype in both environments. If environments 1 and 2 are equally stressful, then the examples shown in b and c are similar in terms of plant fitness losses. If environment 2 is more stressful than environment 1, then b depicts a scenario of greater fitness losses than c. The most injurious case is shown in d.

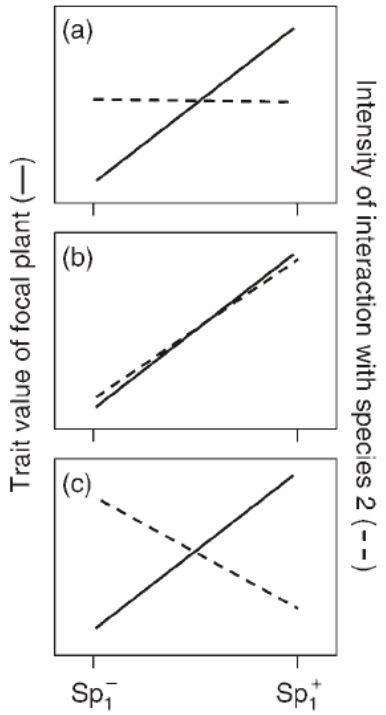


Fig. 5 Potential outcomes of the effect of one species (species 1) on the value of a trait of a focal plant (continuous line) and on its indirect, trait-mediated effect on a second species (dashed lines, representing intensity of interactions between focal plant and species 2; abundance of species 2 could be a valid surrogate of intensity of interaction in certain situations). For the sake of simplicity, the main assumption of this model is that trait value is a sole function of the presence of species 1. (a) No cost of phenotypic plasticity because changes in trait value induced by species 1 have no effect on species 2; (b) phenotypic plasticity is beneficial if species 2 is mutualist and costly if species 2 is antagonist; and (c) phenotypic plasticity is costly if species 2 is mutualist and positive if species 2 is antagonist. The shift from a situation depicted in one panel to that depicted in another one can take place as the result of a change in the identity of the interacting species, the abiotic conditions, or both.