As of phenotypic plasticity has been a controversial subject. Daw [1] proposed the existence of genes controlling plasticity was that plasticity is controlled by a set of genes distinct from those underlying variation in the across-environment. He based this conclusion on the elementary observation from quantitative genetics experiments - the plasticity and the ability to selection, and/or yield quite distinct heritabilities characterized by a lower heritability than the trait mean.

I conclude that the two aspects of that trait are indeed genetic simple and apparently innocuous statement has been the source of controversy that has finally erupted in the mid-90s, the beginning of Daw's original paper.

It was fired by Via [46] with an article in which plasticity is the by-product of selection occurring in distinction to before evolve as a character in its own right. The importance of this belief was highlighted and Pigliucci [35], as well as from Scheiner and Scheiner, particularly strong in their conclusion that phenotypic plasticity in circumstances - evolve as a trait independent of a particular form of a genotype, including and especially the activity. Via was referring to the within-environment me
Two reaction norms in an environment-phenotype phenotype interaction yields very low phenotypic values in the environment toward the left end of the diagram, and very high phenotypic values toward the right end of the diagram. Since Genotype 1 produces the heritability of phenotypic plasticity will be very high (addition for plasticity). However, given that the means of the two genotype are very similar (the mean is similar to the average of high + low), the heritability ("right") of the reaction norm will be close to zero.
with the bath's water. Heterophylly has historically been viewed as a phenotypic plasticity to be investigated, and if we start with a simple definition of plasticity along the lines of the ability of a population to exhibit different phenotypes in response to distinct environments, we can expand from the clearest case to a family of similar yet more complex scenarios. First of all, what if instead of a single individual we consider a group of individuals to experience different environments (Fig. 2)? This can be seen as an extension of kin selection, or as a bet-hedging strategy. It is clear that evolutionary biologists should account for these processes [8,20], and plasticity may increase inclusive fitness. An example is provided by the evolution of plasticity in the eyespots on the wings of butterflies of the genus [8]. These insects sport two seasonal forms (which are characterized by a showy phenotype during the winter and a cryptic one during the less active (dry) season. The two forms seem to be radically different: attracting the attention of predators in the first case, helping to avoid the rare predators in the second case. The molecular biology of this system is being worked out and specific genes respond to reliable environmental cues such as changes in temperature and photoperiod, leading to the appropriate developmental pathway. Now, these butterflies display "plasticity", since the progeny of one form experiences many of the same changes (i.e., there is temporal fine grained environmental plasticity), therefore makes sense to conclude that this is indeed a phenotypic plasticity of currently useful and of derived by a high degree
Two basic ecological situations leading to the targeting directly phenotypic plasticity or to the other ecotype. In (a) the progeny of the plant in the experience one of two environments with a given probability. If such progeny is able to grow well on both environments the inclusive fitness of the mother plant. This evolution of an appropriate plastic response. New involved here perceive the environment as fine-grained, there may be other kinds of environmental patches of the mother plant can only lend on one type is therefore perceived as coarse-grained, and the mother plant will increase as a result of the (ecotypic) specialization. Any plasticity evolving in result of occasionally mixing distinct ecotypes, and entirely a by-product of selection.
ance. Similarly, the debate on the evolution of plasticity raises the general evolutionary ecological question of when or how plasticity (by evolving locally adapted ecotypes) vs. generalists (by evolution of phenotypic plasticity or behavioral flexibility). This is linked to yet another major question in evolutionary biology: How are traits maintained in natural populations? It has been suggested that "buffer" the action of selection [16]; adaptive plasticity may be part of the paradox of natural populations well adapted to environmental conditions capable of expressing significant heritable genetic variation.

The basis of phenotypic plasticity and why it does not evolve

Plasticity genes, that is of genes whose function is primarily in response (and that evolved for that purpose), originally lines of evidence for the fact that plasticity is somewhat common. The arguments to this effect made above are so far reasonable and certainly amenable to modeling. The published to date which demonstrate selection on fitness, given the experimental difficulties of carrying out selection in mice and I had originally in mind while writing this was a compelling demonstration that some types of direct selection. The reasoning goes like this: if a population only as environmental receptors and consequence, lying two or more developmental pathways, then their function is limited to any particular environment, but only...
The curves take one or another form. In fact, Schlich has argued that this must be the preliminary step for them all, even those that eventually come to rely on molecular signals to regulate and fine-tune the response.

Molecular biologists might cry foul and make the argument that the different name (plasticity genes) gives a phenomenon that is different. But, namely the existence of environmental receptors, is it not the classic regulatory element of the cell? It is perhaps the oldest known case of "plasticity gene"? Yes!

Our discussion on plasticity genes is not to give new names but to bring together two areas of research so far entirely separate: the study of environmental receptors on one hand, and the study of organismal responses to environmental changes on the other.

We have known about the Lac operon for decades, we have known about the link between molecular and evolutionary concepts, but it is in this link between molecular and evolutionary biology that is the concept of plasticity genes.

Of plasticity genes and related concepts

To resolve any residual confusion, I would like to offer a definition along with the above discussion, as well as outline the other types of any particular plastic response.

Plasticity genes. These are environmental receptors whose function in a developmental signal and trigger a cascade of other genetic events, eventually leading down one of a number of alternative pathways. (Fig. 2). Notice that this definition...
In example of plasticity genes. (Right) The so-called shade avoidance response in plants is a mechanism that allows a plant to grow if the light conditions are not appropriate (Left). Plants will often stop growing and opening the cotyledons when the ambient light intensity is low. (Left) A class of photoreceptors known as phytochromes are involved in controlling the shade avoidance response. The mechanism of action is as simple as it is effective: if the plant is shaded for a period of time, the ratio between the red and the far red wavelengths (normally about 1:1 under sunlight) goes down (Left).
Experimental study of plasticity genes (from PLoS press in J Evol Biol). The shade avoidance response of Arabidopsis thaliana can be dissected by inducing point mutations or deletion the functionality of one or more photoreceptors (phytochromes in this case). The phenotypic plasticity then compared to the one displayed by the mutants to determine the specificity of the candidate genes' action and their interaction with their environment.
...dependently of growth rates. In the same fast,
ject a plastic response (e.g., shade avoidance in plants,
Schmitt, in press) regardless of growth rates. There-
ypic plasticity as a genetically unique feature of an o

mother of all explanations

ence a possible explanation for the "historical" root-
esy. Via's argument that phenotypic plasticity emerged
 within environments [46,47] is very likely the result of
 stem she worked with [43,44], and the quantitative
 ade applied to modeling the evolution of phenotypic
 al work was originally on two taxa of phytophagous
 one of two plant hosts, although occasionally they c
 status of these entities is still under discussion, an
 ocal ecotypes of the same species, subspecies, or ev
omm.). Whatever the case, Via studied the reaction to fitness, such as rates of development, when each
 the observation was that each fly was doing better
alternative one. This plasticity for fitness is clearly n
ness is bound to be), and it obviously is the by-pro-
ments: each taxon clearly evolved means to exploit
host, and simply found life difficult on the other h
the environment is coarse from the point of view of
idual and very likely its progeny experience only
tensioned framework of quantitative genetic theory. But many functions connecting a series of experimental points are never real continuous functions, which in some cases can cause the "inverse reification" problem. In the case of plant's response, an infinite number of possible environments, so the machinery of the plant is designed to deal with the whole route rather than just one point along it (incidentally, continuous environmental variation is the nature's genetic framework, as demonstrated by Kirkpatrick, [18] on infinite dimensional modeling). The inferential validity of different approaches does not tell us anything more than the rationale of a reaction norm: only a detailed knowledge of the direct or indirect our biological thinking.

I'll pardon me for the clearly out of proportion part. Such like the one originating the much broader dispute over evolution happening by mass selection on many genes versus universal pleiotropy [56]. The two rivals started with different terms and theoretical assumptions in mind, and came to something, almost independently of what the other school was (empirical evidence was suggesting).

"Pluralistics"

The impression that the plasticity genes debate is sometimes reported to me at informal gatherings. The debate was Via's original question [46,47]: is plasticity genetic? There can be no amount of rhetoric that may obscure the plasticity either in the context of reducing variation, the
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