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Evolution of phenotypic plasticity: where are we going now?

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The study of phenotypic plasticity has progressed significantly over the past few decades. We have moved from variation for plasticity being considered as a nuisance in evolutionary studies to it being the primary target of investigations that use an array of methods, including quantitative and molecular genetics, as well as of several approaches that model the evolution of plastic responses. Here, I consider some of the major aspects of research on phenotypic plasticity, assessing where progress has been made and where additional effort is required. I suggest that some areas of research, such the study of the quantitative genetic underpinning of plasticity, have been either settled in broad outline or superseded by new approaches and questions. Other issues, such as the costs of plasticity are currently at the forefront of research in this field, and are likely to be areas of major future development.

From nuisance to paradigm

The concept of phenotypic plasticity (the ability of a genotype to produce distinct phenotypes when exposed to different environments throughout its ontogeny; Figure 1) is at the center of the age-old question of nature versus nurture, which has occupied philosophers from Plato to Locke [1]. Yet, in modern biological literature, it has been considered a nuisance from the time of the rediscovery of Mendel's laws through to the early 1980s, when it became an integral part of our understanding of how organisms develop and interact with their environment. The problem was that many biologists retained some misconceptions about the nature of plasticity, especially its relationship with the genetics of an organism. Falconer famously entitled a classic paper 'The problem of environment and selection' [2], arguing that environmental effects were a 'problem' because they interfered with the natural and artificial selection of a given trait. One can still hear people talking of genetics versus plasticity as if plasticity were not in fact a property of the genotype.

This confusion notwithstanding, phenotypic plasticity is now acknowledged as an important concept in modern evolutionary thinking, particularly as a result of the publication of a several landmark review papers [3–7]. Here, I discuss what I consider to be some of the major issues currently shaping the study of plasticity. I do not consider the rapidly expanding field of the molecular biology of plastic responses [1,8], which is largely conceptually distinct from the matters with which I am concerned here, and which requires an indepth treatment of its own.

Genetic variation and heritability of plasticity

Even a superficial glance at the relevant literature will show hundreds of studies [1,7] reporting the finding of gene-by-environment interactions (GxE; i.e. genetic variation for plasticity), in a variety of organisms (Box 1, Figure I). Indeed, it is clear that, as a general question, this is one that has been answered: there is genetic variation in nature for plastic responses. It is now a matter of documenting specific cases of interest, when

Figure 1. An example of phenotypic plasticity: the same genotype of the weed *Arabidopsis thaliana* exposed to mechanical stimulation **(a)** or lack thereof **(b)**. Plasticity to mechanical stimulation (thigmomorphogenesis) can be an adaptive response to wind, precipitation, and/or attacks by insects. Reproduced with permission from Janet Braam and [39].

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Box 1. Plasticity, GxE and other sources of terminological confusion

Throughout this article, I have tried to keep the terms 'phenotypic plasticity' and GxE distinct, but there is widespread confusion about such a distinction, particularly because many authors use both terms to mean two distinct things, depending on whether one refers to a property of individual genotypes or of populations of genotypes (Table I).

Figure I shows the simple case of a population comprising only two genotypes. The lines represent the reaction norms (i.e. the genotype-specific environment–phenotype functions) of each genotype. Both lines have a slope in the environment–phenotype space, which means that both genotypes are plastic. The population shows GxE in the sense that there is genetic variation for the slope of the reaction norm, which would be detected by a standard analysis of variance.

So far, then, I have used 'plasticity' to indicate the property of individual reaction norms, and 'GxE' to refer to a statistical attribute of the entire population. However, 'plasticity' can also refer to a population-level attribute, in which case it is a statistical measure of how the across-genotypes phenotypic mean of a trait changes with the environment (in Figure I, this mean increases from left to right, because both reaction norms have a positive slope). Moreover, GXE can also be used at the level of a single genotype, in which case it captures the idea that phenotypes are the result of ongoing (mechanistic) interactions between genes and environments throughout the development of an organism.

A source of confusion is that there is no necessary connection between these terms when one moves from the individual to the population level of analysis. For example, GXEs are characteristic of any individual genotype during its ontogeny, but a population of genotypes might have similar individual 'developmental reaction norms,' which would not generate any statistical GxE at the population level. This is a particular example of the general problem posed by the fact that the population (statistical) level of description might not have a direct analog at the individual (mechanistic) level.

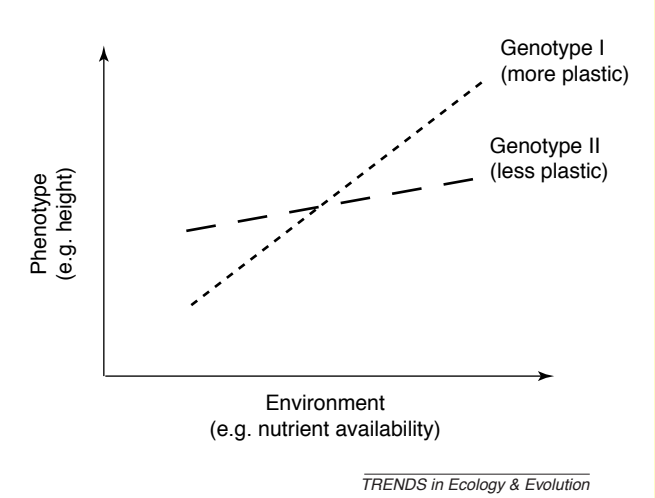

Figure I.

Table I. Key terms in plasticity research

Term	Population level meaning	Individual level meaning
Genotype	Average differences among genotypes, across environments	The actual set of genes affecting the phenotype and shaping all aspects of the norm of reaction (i.e. both its plasticity and 'height' in an environnment–phenotype space)
Plasticity	Average differences among environments, across genotypes	An attribute of the individual reaction norm, indicating that the genotype (through interactions with the environment) generates different phenotypes depending on the external conditions. The only case of zero plasticity is when the reaction norm is flat and parallel to the environment axis
Genotype x Environment	Degree of non parallelism among reaction norms (which may or may not be crossing), resulting in a statistical GxE effect	The idea that genotypes and environments interact mechanistically throughout the development of an organism to produce its phenotype

such documentation is necessary as a preliminary step toward addressing more complex questions.

As for the heritability of plasticity, Scheiner and Lyman [9] have introduced a formal way of calculating it, but such a measure has rarely been used in the literature because of logistical and conceptual reasons. Logistically, it is cumbersome to conduct experimental trials with a large enough number of genetically related families (raised in enough environments) to obtain estimates of heritability; moreover, the confidence intervals of such estimates usually span ranges that are large enough to make the effort not particularly informative. This is a well known problem for the quantification of heritability in general, but it becomes even more evident when one has to multiply the size of the experiment by several environmental conditions.

Conceptually, the problem is even more severe because of the extensive criticism that the very idea of heritability has received since the 1970s [10,11]. Indeed, it is the study of plasticity itself that has led to the realization of the limits that are intrinsic in measuring heritability: estimates of heritability of any trait can vary dramatically with the environment in which they are gathered, and there is no general rule by which to extrapolate from one environmental setting to another, including from laboratory to natural conditions [12,13]. Estimating heritabilities in individual environments might therefore not be the best use of one's time and resources.

Selection on phenotypic plasticity

The commonplace observation of genetic variation for plasticity within populations implies that it can evolve by responding to natural selection, which, in turns, suggests that adaptive phenotypic plasticity occurs in natural populations. Surprisingly, this area of research has received little attention. Again, there are good logistical reasons for avoiding studying the effects of selection on plasticity, given the large size of such experiments, as well as the difficulty of replicating ecologically plausible environmental conditions in a controlled fashion. Yet,

questions related to how (and how frequently) natural selection acts on plasticity are conceptually crucial for our understanding not only of GXE, but also of phenotypic evolution in general.

The recent literature on the question of selection and plasticity yields papers exemplifying different approaches to the problem. Huber *et al.* [14], for example, carried out a classic estimation of selection coefficients, focusing on shade avoidance traits in the plant *Impatiens capensis*. They found that there was a high degree of microenvironmental variation in selection coefficients, with the shade-avoiding phenotype only being advantageous at some microsites. Although this pattern might lead to the maintenance of phenotypic plasticity in response to changes in light quality (the cue that triggers shade avoidance), the authors found that seedling density was also a poor predictor of microenvironmental variation in directional selection. Instead, water availability appeared to have a hitherto unsuspected role, probably because the shade-avoiding phenotype is costly in dry microsites. This is the kind of study that should be more frequent in the literature, given the degree of insight that it provides into the ecological genetics of plastic responses. However, it also shows how much more messy the real world is compared with our simple expectations based on experiments carried out under controlled conditions.

A second approach to the study of selection and plasticity was taken by Fischer *et al.* [15], who carried out artificial selection on the plasticity of growth forms in *Ranunculs reptans*. This species can adopt two growth forms: 'guerrilla' plants spread rapidly by sending out propagules in all directions, and then augment the growth of those propagules that are in favorable microhabitats; 'phalanx' plants grow more slowly in a more compact pattern (in reality there is a continuum of forms between these two strategies). Fischer and collaborators selected their plants for reduced or increased 'guerrillaness' and for higher or lower plasticity of the same trait. Although they obtained significant responses to selection on guerrillaness, they did not achieve any change in its plasticity, concluding that the evolution of growth strategy in this species is possible, but by altering the height, rather than the shape, of the reaction norm (Box 1).

Another approach exploits experimental manipulation (as distinct from natural or artificial selection). Lyytinen *et al.* [16], studied the butterfly *Bicyclus anynana*, which characterized by two seasonal forms, a wet-season form with eyespots on its wings, and a dry-season form, which is spotless. The authors exposed butterflies to bird predation against the background of brown or green leaves, representing dry and wet season conditions, respectively. Their results support the idea that there is selection against the eyespots in the dry season (in favor of crypsis), alternating with selection favoring eyespots (for deflection) during the wet season. The combination of the two selective regimes might be sufficient to maintain adaptive phenotypic plasticity in eye spot formation.

These papers highlight the power of multiple experimental strategies [17], not necessarily all used by the same laboratory, combining field and controlled condition studies, as well as experimental manipulation and the collection of careful observational data. One problem might be that this field often relies on studies that are low-tech and tedious to carry out, and yet demanding high personnel costs and long periods of time, a combination that is sometimes difficult to justify to funding agencies when compared with more 'high-tech' science.

The question of costs and limits

Although there is genetic variation for plasticity in nature, and we can document that natural selection favors certain kinds of plastic response, there must also be limits to the evolution of adaptive plasticity. Other than the possibility of genetic and/or developmental constraints, the idea has been advanced that there might be several types of measurable cost to maintaining plasticity, as well as limits to the ability of an organism of being adaptively plastic [18]. Although important, the conceptual difference between costs and limits is often neglected: costs result in a decrease in fitness even when an optimal phenotype is expressed, whereas limits exist in the failure to express an optimal phenotype to begin with. Costs of plasticity were initially difficult to detect [19,20], although more recent work has found them in a variety of systems [21–23]. In some cases [23], costs are found for the same species in only some geographical areas, presumably because of local selective pressures; it is also likely that different kinds of cost are more or less difficult to demonstrate, depending on the specific evolutionary ecology of the study species [22].

According to theoretical models applicable to spatially structured populations [24], natural selection will favor reaction norms (Box 1, Figure I) that balance cost avoidance with resource acquisition; for example, the cost of maintaining a plastic response is expected to trigger the evolution of reaction norms that increase adaptation to more frequently occurring environments. The costs of producing a plastic response are presumably incurred only when a specific phenotype is generated in a given environment, which makes such costs proportional to the frequency of the environment(s) in which the target phenotype is favored. Research of costs of plasticity is still in its infancy, but is both theoretically important and empirically challenging, and should become a major area of future inquiry.

Genetic correlations as constraints?

The topic of genetic correlations as constraints often comes up in relation to phenotypic plasticity, although in two distinct contexts. However, let us recall that a genetic correlation is a correlation between any two phenotypic traits, calculated using the portion of phenotypic variance statistically associated with genetic differences among individuals.

The first context in which genetic correlations and plasticity are discussed concerns an alternative way of visualizing reaction norms by plotting genotypic means of the expression of a trait in one environment against the expression of the same trait in a second environment, yielding a cross-environments genetic correlation for that trait [25]. Although this is a convenient way of thinking of plasticity in quantitative genetic terms, it is limited by the

fact that one is forced to visualize only two environments at a time.

The second context refers to the fact that GxE can alter the magnitude, and sometimes the sign, of the genetic correlation between two traits, measured in a set of environments [26]. Genetic correlations are measured in a given environmental context, and can themselves be plastic if the environmental context is changed. There are two reasons why this is important: first, studying environmentally induced changes in genetic correlations is a good way to approach the more general problem of the context dependency of constraints and tradeoffs between characters. Second, because genetic correlations are often assumed to be constant over evolutionary time, the demonstration that they are altered within a single generation by an environmental change has obvious consequences for evolutionary quantitative genetic theory in general.

There is also increasing theoretical reason to doubt that genetic correlations are informative about constraints, and therefore useful beyond descriptive statistics in evolutionary quantitative genetics. Two papers [27,28] have demonstrated that one cannot infer the underlying genetic architecture from an observed genetic correlation, because many different underlying causal pathways can generate the same correlational pattern; thus, one can go from hypotheses about the causes to predictions of observed correlations [29], but cannot infer causes from observed correlations. This does not mean that studying genetic correlations is useless: the observed patterns might suggest causal hypotheses, which can then be tested by experimental methods. However, the debate about the proper or best use of genetic correlations in general, and their application in plasticity research in particular, is likely to continue for some time.

The genetic basis of phenotypic plasticity

An issue in the study of the evolution of plasticity that used to be prominent in the literature is the discussion of different models proposed to account for the genetic basis of plastic responses. Scheiner [7] summarizes the three main models: overdominance, pleiotropy, and epistasis. The overdominance model states that plasticity is an inverse function of heterozygosity: the more heterozygous a genotype, the less plastic it will be, because heterozygosity helps to 'buffer' environmental influences. The pleiotropic model states that plasticity originates from the fact that some genes have pleiotropic effects on a given character expressed in different environments. Finally, the epistatic model predicts that there are two separate sets of genes, one determining the height of the reaction norm (i.e. the across-environment mean), the other determining its shape (the slope, in the case of a linear reaction norm), and that these two sets interact epistatically with each other.

This is one area where questions have been superseded by the empirical research of the past decade [1,8]. From extensive studies of the molecular biology of plasticity, such as the shade avoidance response in plants, or the heat shock response [14,30,31], we know that heterozygosity (the overdominance model) has little, if anything, to do with the genetic basis of plasticity, and that both pleiotropic and epistatic effects are characteristic of any plastic response that has been extensively investigated to date. It seems fair to conclude that the problem of the genetic basis of plasticity, which has always been conceptually rather ill defined, has been dissolved into the many problems of the specific molecular underpinnings of particular kinds of plasticity.

It is helpful to reflect on a more basic question: why would we expect plasticity as a whole to have a particular kind of genetic basis? This would be similar to asking whether the expression of trait X (e.g. body size) depends either on pleiotropy, epistasis, or is affected by the degree of heterozygosity. The answer would be: all of the above; none of the three 'mechanisms' would be particularly informative, because they are all likely to underlie most complex phenotypic traits.

Mathematical modeling of the evolution of plasticity

'Modeling' plasticity also has another meaning in the literature, referring to theoretical models of how phenotypic plasticity evolves. Although some of this mathematical modeling does impinge on considerations of 'models' of the genetics underlying plastic responses discussed above, the two meanings of modeling plasticity are in fact quite independent of one another and can be discussed separately.

Scheiner [7] again provides a convenient summary of the kinds of model that have been used to study the evolution of plasticity. There are essentially three categories: optimality, quantitative genetic, and gametic. Optimality models are independent of any considerations of genetics, because they ask what sort of optimal (given whatever constraints) strategy should evolve under certain conditions (assuming the necessary genetic variation). Whereas the lack of genetics can be viewed as a limitation of optimality models, it can also be thought of as an advantage, because the goal is to explore a series of 'if-then' hypothetical scenarios about natural selection, rather than to make quantitative predictions about actual evolutionary trajectories.

Quantitative genetic models, in spite of their name, are also largely independent of the actual genetics. They treat the genetics as a 'black box,' from which the necessary 'additive' genetic variance (i.e. the genetic variation that enables a trait to respond to selection) emerges. These are statistical models and incur the same limitations as genetic correlations.

Gametic models are the only ones that incorporate any real genetics, in that they describe what happens to traits that are not only under the influence of certain evolutionary processes such as selection or migration, but that are also affected by a specified number of loci, interacting in particular fashions. Whereas the lure of gametic models is the ability to deal directly with genetic phenomena, such as pleiotropy and epistasis, the problem is that these models are, by necessity (of mathematical tractability), limited to simple genetic scenarios, and become unwieldy as one attempts to make them more realistic (simulation models only partially obviate this problem, because they

become computationally unmanageable, or do not guarantee general solutions).

An analysis of the recent literature on modeling the evolution of reaction norms shows that most researchers are currently interested in optimality models, often tied to particular evolutionary ecological situations. For example, Ergon *et al.* [32] used an optimality approach to investigate the relationship between body size and energy expenditure during winter in voles *Microtus agrestis*. The model included a tradeoff between the survival benefits of being large, and the cost that this implies in terms of foraging. Their model predicts that the voles should be smaller in environments that are more demanding, with a resulting negative correlation between daily energy expenditure and body mass. However, if the animals display phenotypic plasticity in adjusting energy intake as a function of the cost of foraging, then the model predicts a positive correlation between body mass and energy expenditure. The authors tested their model empirically, and found that energy expenditure was highest at locations where the voles were smaller, in spite of a positive correlation between the two measures within sites. They concluded that variation in size is attributable to heterogeneity in food quality or availability, rather than to adaptive plasticity in foraging activity.

Similar combinations of optimality modeling and empirical work to test theoretical predictions have been used to study, for example, resource allocation based on life-history traits in the Pacific oyster *Crassostrea gigas* [33], root architecture and resource acquisition in beans *Phaseolus vulgaris* [34], and maternal control of offspring sex in fig wasps *Otitesella* spp. in response to local population density [35]. This particular aspect of modeling plasticity is ripe for a general review that would be of use to both empirical and theoretical biologists.

Macroevolution by phenotypic plasticity?

Plasticity has also been suggested as a potentially important mechanism facilitating macroevolution [1,38]. This can happen through at least two pathways: on the one hand, plasticity can lead to the genetic assimilation of a character when a population occupies a new environment. Essentially, pre-existing variation for plasticity could enable a population to persist under new conditions, even though the population might be sub-adapted to them. Such persistence would then allow time for new genetic variation to arise (through mutations and/or recombination), and for natural selection to increase the fit to the new conditions. If the new conditions persist, selection might favor a decrease in plasticity, essentially genetically assimilating the trait(s). Such a mechanism might be operating, for example, during the well known 'lag phase' that accompanies colonization by many invasive species, before they spread in the new habitat.

On the other hand, genetically induced changes of the phenotype are accommodated by the natural plasticity of the developmental system ('phenotypic accommodation'). An example is the ability of some quadrupeds to develop a quasi-bipedal posture through a complex set of changes in their muscle–skeletal system, in response to a mutation that renders their forelimbs non functional [6,38]. Natural selection can then assimilate the novel phenotype, yielding the appearance of 'mosaic' evolution.

Future research on the macroevolutionary consequences of plasticity must document instances of both genetic assimilation and phenotypic accommodation, map them in a phylogenetic context, and devise empirical approaches to study them. This is a daunting task, both conceptually and logistically, but might represent one of the major forthcoming revolutions in our way of thinking about the appearance of evolutionary novelties.

Studying phenotypic plasticity: the next generation

It is always risky to make predictions concerning where science will go, even in the short term [36]. Nonetheless, speculating on what directions might be worth pursuing (or not) in the study of plasticity should provide some food for thought for researchers and graduate students interested in the field.

I suggest that there are some research questions that have either been ill conceived or are no longer relevant. Among these, the issue of the genetic basis of plastic responses is perhaps paradigmatic. Never a conceptually sound question to begin with, it is now superseded by research on the molecular basis of specific plasticities. Also rather troublesome is the issue of the relationship between genetic correlations and plasticity. Although there is plenty of beneficial use for quantitative genetic studies and the characterization of genetic variance–covariance matrices, we must get to grips with the fact that these are still just correlational studies. Another area of the study of plasticity that appears unlikely to provide major new insights in is the quantification of patterns of quantitative genetic variation and heritability of plasticity. This is not because of any inherent conceptual problem, but because it has been done enough to have a clear answer to the broad questions: we now know that there is abundant natural genetic variation for plastic responses.

On the positive side, there is much to be done on quantifying and understanding patterns of natural selection on plastic responses [37]. Again, we do know that there can be selection on plasticity, and it can respond to selection. However, we still know little about what ecological conditions favor stabilizing or directional selection on reaction norms, and what kinds of life history, mating system and even phylogenetic history are more or less conducive to genetic variation that can respond to such selective pressures. This is research that, although vital to our understanding of the evolutionary ecology of plasticity, is logistically cumbersome and tedious. It should, nonetheless, be pursued as vigorously as possible.

The verdict about theoretical modeling of plasticity is a mixed one. Although quantitative genetic and gametic models have produced interesting insights into the evolution of reaction norms, it seems that optimality modeling coupled with detailed empirical data is the most useful research approach. This suggestion, however, hinges on considerations of the tradeoffs among the realism, precision and generality of models and their usefulness in quantitative biology, topics that are beyond the scope of this article.

The study of GxE in the broadest sense has evolved over the past few decades from a marginal interest of a few researchers to something that cannot be avoided by any serious evolutionary ecologist: plastic responses to heterogeneous environmental conditions, far from being a nuisance, are one of the most common phenomena characterizing the living world.

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