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How organisms respond to environmental changes: from phenotypes to molecules (and vice versa)

Massimo Pigliucci

One of the most repeated truisms of evolutionary ecology is that coping with changing environmental conditions is a fundamental aspect of organic evolution. Yet, investigating how this is achieved is a daunting and at the same time fascinating problem for modern organismal biologists. The classical neodarwinian view of adaptation maintains that populations tend to evolve characteristics that make them well suited to the particular conditions experienced over a long period of time and a small spatial scale. This led to the flourishing of the so-called 'ecotype' concept of adaptation¹. At the apex of the evolutionary synthesis of the 1940s, a few researchers pointed out that things might be a bit more complicated, because organisms and their progeny experience a variety of environments during their life cycle (see, for example, the series of studies by Clausen and colleagues)². Schmalhausen³, in particular, re-examined the old concept of reaction norm (that is, the function that describes the phenotypes produced by a given genotype when exposed to a certain range of environments) and made it the central object of natural selection (even though he scarcely mentioned the term 'reaction norm' throughout his book). But Schmalhausen's work was ignored for a long time. This was in part because the English translation of his book was not very clear.

The ability of organisms to produce different phenotypes under different environmental conditions (phenotypic plasticity) has been an object of evolutionary and ecological studies since the neodarwinian synthesis. Yet, until lately, our knowledge in this field was limited to statistical approaches based on the classical tools of quantitative genetics. In recent years, however, a new dialog between organismal biologists and researchers interested in uncovering the mechanistic details of physiological and phenotypic responses has yielded several new insights. Some classic examples of phenotypic plasticity have now been traced to specific alterations in DNA transcription and RNA translation rates, and to changes in patterns of protein expression. Conversely, the explicit use of evolutionary and ecological theory is helping us to put a panoply of molecular data into a coherent historical and organismal perspective.

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However, arguably a major reason was that he came under political attack in the Soviet Union by Lysenko; this left him little time further to develop and publicize his work. Starting from the mid-1960s, and especially since the mid-1980s, however, the study of reaction norms and of phenotypic plasticity (a measure of how different the phenotypes produced in distinct environments are from each other, and therefore a property of the reaction norm) has become a centerpiece of modern evolutionary and ecological genetics^{4–8}.

The currently favored approach to the study of reaction norms and the modeling of their evolutionary trajectories is based on an extension of the concepts and tools of classical quantitative genetics⁹. That is, a group of genetically related individuals (a collection of isogenic lines, clones, half- or full-sib families) is exposed to a variety of environmental conditions (treatments), and their response is graphed as a reaction norm (i.e. as a plot of phenotype versus environment for each genotype, family or clone). A family of statistical techniques related to the analysis of variance is then used to partition the observed phenotypic variation in at least three components: (1) genotype, representing differences in the average response across environments among genotypes; (2) environment, quantifying the average effect of the treatments across genotypes; and

(3) genotype by environment interaction, gauging the genetic variation for the shape of the reaction norm existing in that particular population. This information can be easily translated into estimates of fundamental quantitative genetic parameters such as genetic correlations and heritabilities¹⁰. These estimates can then be plugged into multivariate extensions of the classical quantitative genetic equations that describe response to selection¹¹, yielding predictions of rates and directions of evolutionary change in that particular population, given certain simplifying assumptions¹².

Lately, a certain dissatisfaction with the purely statistical approach typical of classical quantitative genetics has been expressed¹³: quantitative genetic models either do not deal with the specific genetic basis of phenotypic plasticity (the so-called 'black box' approach), or they assume a very simple model of many genes that have small additive effects. One alternative that is currently being pursued is the mapping of genes affecting quantitative trait loci using a combination of techniques that bridges the gap between a purely statistical description of a population and a more mechanistic understanding of the genetic machinery underlying complex traits¹⁴. On a different front, the concept of 'plasticity genes'¹⁵ has been proposed. Because of some confusion with previous definitions, I will refer to these as 'regulatory loci that directly respond to a specific environmental stimulus by triggering a specific series of morphogenic changes', a more restricted and conceptually useful definition than previously proposed. It has been suggested that understanding the origin, evolution and function of plasticity genes must be an integral component of any satisfactory scenario of organismal evolution in response to heterogenous environments¹⁵. But what do we know about the origin, evolution and function of plasticity genes?

Two mechanistic approaches to the study of plasticity genes

There are two important and complementary approaches that one can use to dissect the genetic and physiological basis of phenotypic reactions to environmental changes. We can try to isolate mutants that abolish or interfere with a well-characterized pattern of phenotypic plasticity, or we can expose plants

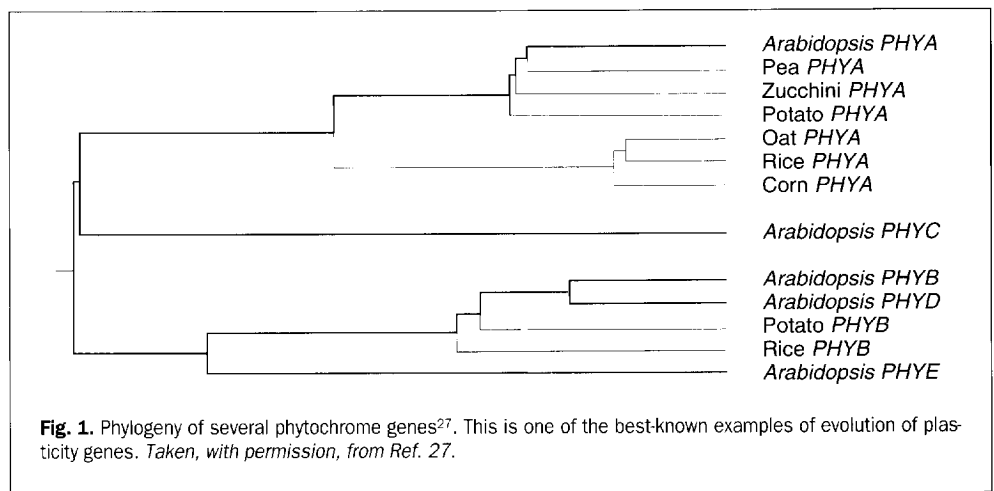


Fig. 1. Phylogeny of several phytochrome genes²⁷. This is one of the best-known examples of evolution of plasticity genes. Taken, with permission, from Ref. 27.

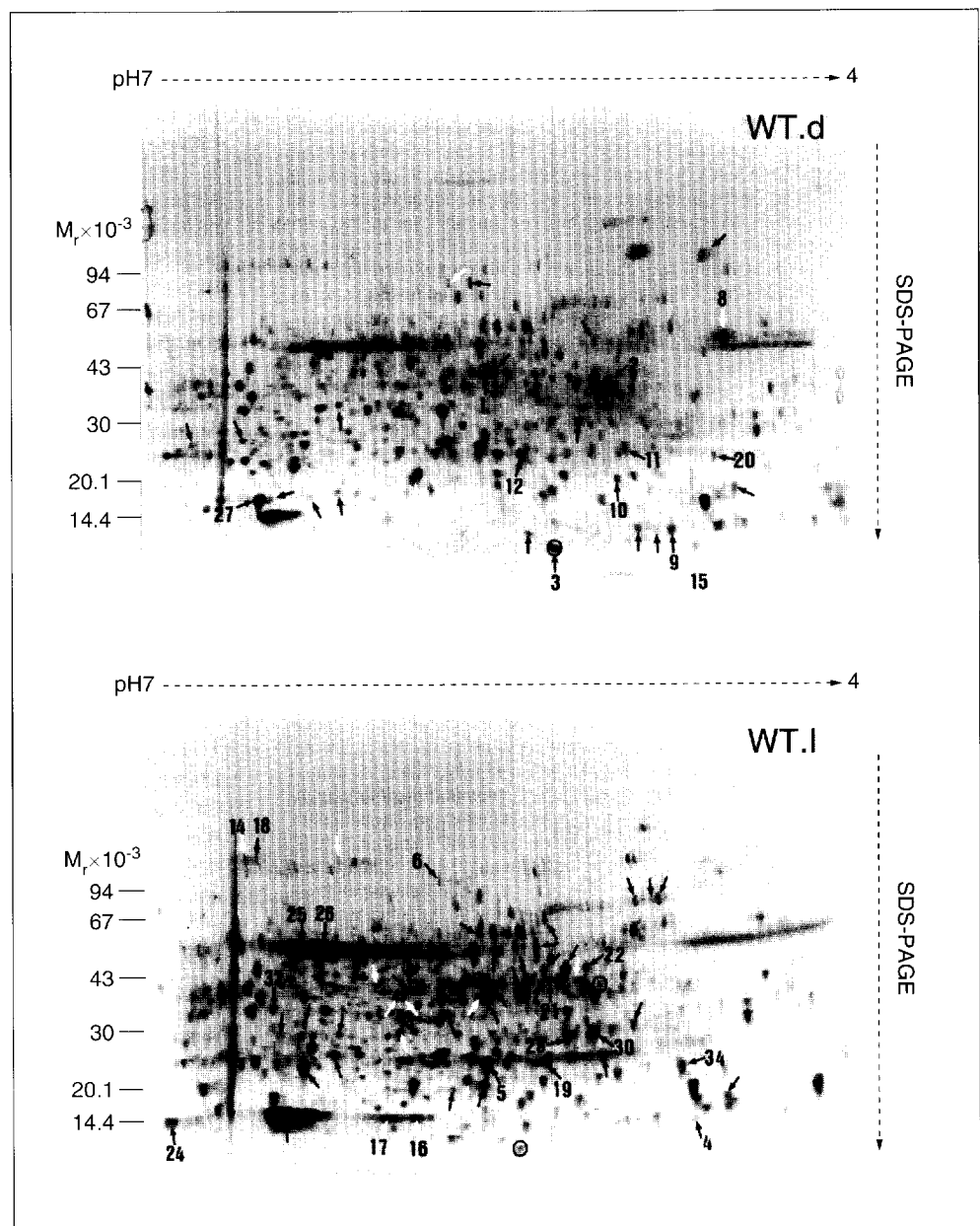


Fig. 2. Comparison of 2-D gel patterns of total proteins isolated from dark-grown wild-type plants (WT.d) and light-grown wild-type plants (WT.l) of *Arabidopsis*. Black arrows indicate spots (proteins) of higher intensity (related to protein quantity) when compared to the same spot in the other pattern. White arrows indicate spots that specifically appeared in the pattern and that were undetectable in the other growth conditions. As expected, many of the spots show significant differences when their intensities are compared between these growth conditions. Taken, with permission, from Ref. 16.

Box 1. The response to light in flowering plants

Light is one of the fundamental abiotic variables of the environment of any plant. Different aspects of light availability influence the development, physiology and morphology of a plant: the day length (photoperiodic response), the intensity and direction of irradiation (phototropism, photosynthesis), and the spectral quality (used as a way to gauge canopy density and forthcoming competition from neighbors²⁵). Phenotypic responses of flowering plants to light have been investigated since Darwin's demonstration that the tip of the growing embryo is responsible for phototropism (tracking the direction of incident light) during development. Recently, a number of studies have dealt with how plants perceive changes in light quality, and how these are translated in one of the best-known examples of adaptive phenotypic plasticity²⁶.

At the phenotype level, it is known that plants that typically colonize open gaps in the canopy, or otherwise live in habitats where shading is not a constant component of the environment, dramatically alter their architecture and phenology when exposed to abnormally low ratios of red (R) to far-red (FR) light. Under these conditions, plants tend to suppress branching, increase the rate of vertical elongation of the stem, and flower earlier. Since the lower R:FR ratio is caused by the fact that their neighbors absorb the photosynthetically active red light, while reflecting the far-red portion of the spectrum, this so-called 'shade-avoidance' response is thought to give an advantage to plants by allowing them to complete the life cycle and produce progeny before being shaded by competitors.

At the physiological level, it has been known for some time that a specific class of molecules, known as phytochromes, are characterized by a sensitivity of their molecular structure to the R:FR ratio. In particular, phytochromes can exist in two states, and the switch between them is catalyzed by the spectral quality of the incident light. The use of mutants and of probes derived from them recently enabled researchers to identify a family of at least five genes coding for phytochromes in flowering plants²⁷ (Fig. 1). The functions of only two of these, phytochromes A and B, are known to some extent, and they appear to be partially overlapping, with effects that are specific to either molecule (photoperiod for Phy-A, certain components of shade-avoidance for Phy-B) and effects that are controlled by both (germination)²⁸. Recent advances point to the interaction of phytochromes with at least some plant hormones (most notably gibberellin) to form complex light perception-transduction pathways that include several other regulatory genes²⁹ and that account for the sophisticated plasticity exhibited by plants at the phenotypic level.

or animals to different environments and screen directly for changes in their RNA or protein patterns that are specifically associated with a set of environmental conditions.

The study of mutants was the foundation of modern genetics ever since Gregor Mendel and, later, Thomas Hunt Morgan, and (in a different version involving the deliberate disruption of normal developmental processes) it has recently received renewed attention because of its usefulness as an 'entry'-level method to isolate specific genetic factors. The story of the study of genes coding for light receptors in plants is a typical example (Box 1). Two of these genes were initially found by screening for mutants exhibiting a long hypocotyl phenotype under normal light conditions (the elongated hypocotyl, or embryonic shoot, is typical of plants in the dark phase, when the seedling has to emerge from the soil). Once found, they were mapped on the genome of *Arabidopsis thaliana* by classical genetic methods. Given the small genome of *A. thaliana*, however, it is relatively easy to isolate and sequence a gene of known position. After a plasticity gene is sequenced, it is then possible to clone similar genes from several species to find possible homologs and trace back its evolutionary history (Fig. 1). An even more direct approach to mutagenesis has been developed recently. In the case of 'tagged' mutagenesis, one can insert a known sequence (the tag) in a random position in the genome, either by transformation or by using transposons. A regular screening for mutations is then performed and potentially interesting mutants are isolated. The tag sequence can then be rescued with a probe of complementary sequence and the DNA regions upstream and downstream from the tag can be explored.

One can also screen directly for differences in protein or nucleic acid expression between individuals exposed to different environmental conditions. A particularly elegant example of protein expression is the work of Santoni *et al.*¹⁶. They compared the total protein profiles obtained by two-dimensional gel electrophoresis of the same genotype of *A. thaliana* growing in the dark and in normal light conditions. They also compared the patterns of single-gene mutants with an elongated hypocotyl to that of the wild type under identical environmental conditions. In both cases, they were able to demonstrate the switching on or off of dozens of proteins, in either an environmental- or genotypic-specific way (Fig. 2). To conduct screenings for environmentally sensitive genes at the nucleic acid level, the organism is exposed to the environmental conditions of interest (e.g. water stress) and is then quickly frozen. DNA profiles of the treated and control organisms reveal portions of the genome that are specifically present only under the treatment conditions. Once the sequences of these fragments have been obtained, Northern blots can be performed in order to study the pattern of expression of the corresponding RNAs. Thus, the environmental specificity of the genes can be demonstrated, and also their timing of expression and their localization can be studied¹⁷.

Box 2. Heat-shock responses across kingdoms

Most living organisms have to deal with fluctuations in the temperature of their habitat, and these fluctuations can vary enormously in amplitude, duration and predictability. It is, therefore, not surprising that a special type of plasticity to heat shock has evolved across kingdoms (with known examples existing among bacteria, fungi, plants, invertebrates and vertebrates)^{30,31}. Phenotypically, this response is usually measured in terms of fecundity or survival, that is, it deals with plasticity for fundamental components of fitness. Typically, survival is minimal if an organism is subjected to a sudden increase in ambient temperature (acute shock). On the other hand, the ability to withstand the change increases proportionally to the time of acclimation at increasingly higher temperatures³². Also, lines of *Drosophila buzzatii* collected from natural populations that normally experience higher temperatures in their habitats show more resistance to the shock treatment³², suggesting that the plastic response has the ability to evolve within species. Another interesting observation is that the temperature that activates the response strictly depends on the specific habitat of the organism considered: for example, Antarctic fishes of the genera *Pagothenia*, *Nothotenia* and *Chionodraco* react when the temperature becomes as 'hot' as 5°C (Ref. 31).

The molecular basis of the heat-shock response is fairly well elucidated in invertebrates. In *Drosophila melanogaster*, there are at least eight major polypeptides that are synthesized in response to heat shock. The cDNA from one of these, *hsp70*, has been used as a probe to locate genes with similar characteristics in an array of organisms. At least three different promoters have been located upstream of *hsp70* in *Drosophila*. Some heat-shock proteins are constitutively expressed, while others are activated only during the acclimation period: the first type can be considered a regulatory or structural gene (depending on its specific function) with environmentally independent expression. The second class is either an example of plasticity genes (if they respond directly to the environmental signal), or it is activated by unidentified plasticity genes. The activation of the heat-shock genes is mediated in most organisms by a heat-shock factor (HSF), which acts as a transcriptional activator³³. This gene is constitutively expressed, but heat shock causes a marked elevation of the level of its mRNA. The physiological role of the heat-shock proteins is far less clear, although they are thought of as involved in stabilizing other proteins when the cell is exposed to the heat shock³¹. A role as molecular chaperons has been demonstrated for *hsp20*, *hsp60* and *hsp70* (Ref. 33).

What evidence do we have for the existence of plasticity genes?

The number of studies demonstrating the existence of genes that specifically respond to a particular type of environmental alteration by triggering a given pattern of morphogenic changes is increasing steadily. Boxes 1-4 highlight four cases in which the phenotypic reaction, the physiology and the molecular biology are particularly clear: response to light in flowering plants (controlled by phytochromes, Box 1); heat-shock response across kingdoms (hsp-family of proteins, Box 2); adaptive plasticity to sulfur limitations in cyano-

Box 3. Adaptive responses to sulfur limitation in cyanobacteria

Some of the most convincing and molecularly best-characterized examples of adaptive phenotypic plasticity are found outside the ecological and evolutionary literature. A case in point is a study by Mazel and Marliere³⁴ on strategies for coping with sulfur limitations in the habitat of cyanobacteria. Sulfur is a fundamental constituent of the lateral chains of cysteine and methionine, and so it is used in the synthesis of many fundamental polypeptides in the cell. A common problem for many prokaryotes is to find themselves in habitats poor in sulfur, and therefore they are unable to synthesize proteins at a normal rate. This is particularly problematic for cyanobacteria, since sulfur is necessary to build phycobilins, the light-harvesting molecules that allow cyanobacteria to function autotrophically.

The classical evolutionary solution to this problem would be to evolve alternative forms of the proteins involved, which do not rely on cysteine and methionine (or which, at least, require less of these two amino acids). We would, therefore, expect that mutations that substitute cysteine and methionine with sulfur-free amino acids of similar structural and biophysical properties should be favored under conditions of sulfur deprivation. This has been demonstrated to be the case, for example, in *Salmonella typhimurium*³⁴.

Mazel and Marliere³⁴, however, show what happens if the organism finds itself in an environment in which the resource is not simply scarce, but fluctuates. The cyanobacterium *Calotryx* sp. PCC 7601 has two versions of all its proteins that are rich in cysteine and methionine, in particular, phycocyanin and the proteins associated with its functional properties. The alternative set substitutes amino acids in key positions, resulting in lower requirements for sulfur. Significantly, the alternative set is activated only when sulfur is scarce in the environment. These proteins are in no way involved in the biological fixation of sulfur. Yet, the switch in pattern of protein synthesis does substantially alter the budget of intracellular sulfur. There are three phycocyanin operons in *Calotryx*: one is constitutively expressed, regardless of any environmental signal; the second one responds to the light spectral quality; and the third – the one characterized by Mazel and Marliere³⁴ – shows a dramatic increase in expression under low sulfur conditions. The five genes in the third operon are homologs of those present in the second operon, and they are arranged in the same way. The phycocyanin genes have a long evolutionary history, since they evolved by successive gene duplications from an ancestor related to the globin family. A similar mechanism for the evolution of regulatory plasticity had in fact been proposed by Smith³⁵.

bacteria (involving an alternative sulfur-independent set of phycocyanins, Box 3); and temperature-dependent sex determination in reptiles (catalyzed by the antagonistic action of aromatase and reductase on testosterone, Box 4). Other examples include: facultative metamorphosis in amphibians; heterophyly in semi-aquatic plants; the SOS response to DNA damage in *E. coli*; wing dimorphism in response to crowding or photoperiod in insects; and alterations in flowering phenology in response to temperature and photoperiod in plants.

There are two major reasons why these results have not been connected to the theory of evolution in heterogeneous environments. First, most researchers interested in the molecular and physiological basis of plasticity do not actually work in contact with population and evolutionary biologists. In fact, in most cases, the word 'plasticity' does not appear in their papers, and few or none of the fundamental theoretical and empirical papers dealing with the ecology and evolution of plasticity are cited in these reports. As a consequence, discoveries of very interesting patterns of gene expression are left in a vacuum because of the lack of a proper ecological–evolutionary framework to interpret their significance. Conversely, most population and evolutionary biologists publishing in the field are not in touch with the ample physiological and molecular literature available. Consequently, our models of the evolution of plasticity are based on a very rough understanding of the biological machinery actually involved in these processes. Overall, we

have a dramatic case of poor communication between scientific enterprises that could clearly benefit from a tighter interaction. However, this situation is beginning to change very rapidly.

What can we learn from the combination of ecological and molecular studies of phenotypic plasticity?

The nascent interaction between molecular and organismal studies of reactions to environmental heterogeneity is altering our perspective of how phenotypes evolve. First, we are gaining a more precise and biologically realistic understanding of gene–gene interactions (the classical quantitative genetic concept of epistasis). Epistasis has always been considered a 'noise' in quantitative genetic analyses of phenotypic variation, and it is routinely ignored on the grounds that it accounts for a small portion of the total genetic variance. As Cheverud and Routman¹⁸ have elegantly demonstrated, however, this is true only for what they call 'statistical' epistasis. If we consider 'physiological' epistasis, that is, the physiological rather than statistical effects attributable to gene–gene interactions at the individual (as opposed to the population) level, epistasis can make a substantial contribution to additive, dominance and interaction genetic variance components, thereby deserving a more prominent place in our descriptions of evolution¹⁸.

Second, all the available molecular and physiological evidence points to the fundamental role played by regulatory genes with major effects in phenotypic evolution. Studies of single gene mutants show that they can be responsible for dozens of changes at the protein-regulation level, thereby forming the basis for truly universal pleiotropy^{16,19}. When double and triple mutants are constructed, we can investigate directly what developmental and physiological effects can be attributable to epistasis, instead of simply deducing them from statistical analyses. Very elegant models of gene action in response to environmental cues have recently been produced using this method²⁰.

A third point is that fundamental regulatory gene functions seem to have a very ancient and highly conserved evolutionary history (Fig. 1). It is now common practice to isolate a new regulatory gene from a particular organism and then to screen gene sequence databases to find homolog sequences not only in different species of the same genus or family, but even in different kingdoms. Yet, some of the phenotypic variation within species can, surprisingly, be produced by null or defective mutants for important regulatory functions²¹. One of the consequences of these observations is the reopening of the old and still-controversial debate on the relative importance of quantitative versus major-effect genes in evolution²².

Fourth, molecular research can benefit from a better understanding of the ecology of the organism. For example, although the so-called 'shade-avoidance' response in plants is supposed to be one of the best examples of adaptive plasticity, and it is very well characterized at the mechanistic level (Box 1), in fact, the direct ecological evidence that such a response is adaptive has only become available very recently²³.

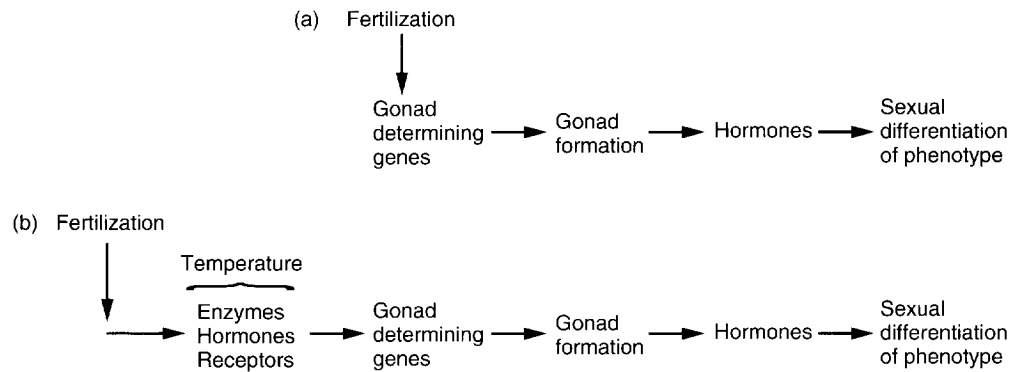
Mechanistic biology can also gain from a more organismal oriented perspective in another way. The more we dig into the details of organic reactions to environmental change, the more we realize that these reactions are much too complex for a satisfactory gene–phenotype mapping function ever to be reconstructed (notwithstanding the credo of some molecular biologists). We are beginning to understand that there is still a void between the genetic and the phenotypic levels that is populated by poorly specified 'emergent

Box 4. Temperature-dependent sex determination in reptiles

In 1966, Madeline Chamier discovered a peculiar phenomenon concerning sex determination in the lizard *Agama agama*: the sex of the offspring appeared to be determined by the incubation temperature of the eggs. Temperature-dependent sex determination (TSD) is now known to be a widespread phenomenon among reptiles³⁶. While the exact adaptive value of TSD is still being debated³⁷, its genetic and physiological basis, as well as its potential phylogenetic importance, are becoming much clearer³⁸.

The phenotypic response is obviously quite complex: it includes the functional sex of the individual, all the primary and secondary sexual morphological attributes, as well as the behavioral traits associated with being male or female. In the red-eared slider turtle (*Trachemys scripta*), a temperature below 29°C produces males almost exclusively, while values above that threshold result in females³⁸. The fundamental switch at the molecular level is associated with the effect of either aromatase or reductase on testosterone. If the temperature is female-inducing, the enzyme aromatase transforms the precursor testosterone into estradiol. This leads to a chain of events that will culminate with the development of a female individual. If the temperature is below the proper threshold, on the other hand, reductase will effectively compete with aromatase for the substrate, and will produce dihydrotestosterone, switching the biochemical and developmental pathways toward maleness³⁸. One of the consequences of the elucidation of this mechanism is the realization that, in reptiles, there is no 'default' sexual development as is the case in mammals and birds. To be a female, the individual has to follow a non-male pathway, but the opposite is also true (in mammals and birds the individual is constitutionally a female, unless the male-determining factors are present).

Crews *et al.*³⁸ also suggested that TSD is, in fact, the ancestral condition for terrestrial vertebrates from which the genotypic sex determination (GSD) typical of birds and mammals evolved. The figure below (*taken, with permission, from Ref. 38*) shows the differences between the two models. (a) GSD and (b) TSD: a simple loss or inactivation of the regulatory elements that determine temperature sensitivity (plasticity genes) would make the macroevolutionary transition possible. It would be fascinating to investigate whether or not TSD-related genes are still present in lineages of birds or mammals, and to speculate what evolutionary scenarios can account for the evolution of GSD from TSD.



properties' and 'epigenetic' effects. The challenge of the future is then for organismal (ecological and evolutionary) and mechanistic (molecular and physiological) biology to converge on the common ground of developmental biology to understand truly how genotype and environment interact to yield the complexity of phenotypes observed in nature.

Conclusions

What are the potential routes that the study of phenotypic reactions to environmental heterogeneity can take during the next few years? We now know that there is genetic variation for phenotypic plasticity in natural populations, and that this variation is both character- and environment-specific. We also know that populations of plants and animals can respond to selection, altering the pattern of their sensitivity to the external conditions. Thanks to the molecular studies of the type reviewed here, we now realize that many of these reactions are not just passive responses of the genetic-physiological machinery, but are highly specific and coordinated by an array of regulatory genes acting at different hierarchical levels. Physical environmental factors such as temperature will still directly affect cellular metabolism and enzyme kinetics, thereby eliciting what I have termed 'passive' (i.e. not regulated by the organism) plasticity. The roots of passive plasticity are also developmental, and need to be understood in order to gain a more complete picture of evolution in heterogeneous environments. However, adaptive plasticity will be – by definition – 'active', that is, mediated by a more- or less-complex genetic-developmental machinery of the type discussed throughout this article, which can only originate

as the product of evolution under natural selection.

What we do not know, or have very few concrete data about, is how adaptive plasticity evolves. How does it happen that a population of *Ranunculus flammula* is capable of producing two alternative developmental pathways for the shape of its leaves, one to be expressed under water and the other under aerial conditions, but that other populations of the same species cannot do the same? And what about other species of the same genus? Comparative studies of phenotypic plasticity are mostly absent from the literature, but are a necessary component of any satisfactory understanding of the problem.

A second new line of research is trying to link the molecular characterization of plastic responses with the mostly presumptive adaptive meanings that such responses have²³. Progress in this field will come from a more complete description of the mechanistic machinery involved, as well as from measurements of selection intensity under field and controlled condi-

tions, comparing the relative fitnesses of wild types and mutants that are deficient for the plasticity gene(s).

Yet another direction of future research certainly lies in the already mentioned interface between genetics and environments, in the epigenetic machinery that somehow translates genetic effects and environmental influences into coherent phenotypes. Here, the interaction of detailed molecular studies and new theoretical approaches based on non-linear dynamic modeling are a promising, yet not explored, avenue of research²⁴. One of the emerging concepts is that the old metaphor of genes as 'blueprints' for the organism has to be abandoned in favor of a more complex view that sees organismal properties emerging from local and limited genetic effects. Clearly the stage is set for a very exciting period of research based on a true synthesis of biological knowledge, with different levels of analysis equally contributing to a deeper understanding of what organisms are and how they arose.

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