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14 Phenotypic Plasticity

Massimo Pigliucci

The concept of phenotypic plasticity has just turned 100 (Woltereck 1909), and yet it is common both in the published literature and especially in the halls of scientific meetings to hear professional biologists make pronouncements that betray their lack of understanding of what plasticity actually is. By far the most common misunderstanding is that plasticity is simply a fancy word to indicate the old "environmental component" of a phenotype (Falconer 1952), and that therefore it still makes sense to think in terms of genetics versus plasticity. Next in line among the misconceptions about plasticity is the idea that one can tell whether an organism is "more" or "less" plastic than another one, across the board. In fact, ever since Woltereck coined the term "reaction norm," it should have been clear that plasticity is a property of a genotype, and that it is specific to particular traits within a given range of environments. Before we come to a discussion of the possible roles of phenotypic plasticity as a causal factor in evolution, therefore, it may be appropriate to review the basics.

A norm of reaction is a genotype-specific function that relates the range of environments experienced during ontogeny to the range of phenotypes that the particular genotype produces in that range of environments (figure 14.1). A population of reaction norms, therefore, is characterized by three general properties (Pigliucci 2001): there can be genetic variation for the focal trait across the environments being considered, which means that some norms of reaction are positioned higher than others on the diagram; there can be overall plasticity (i.e., across genotypes) if on average the reaction norms show a nonzero slope; and there can be genotype-by-environment interaction if the slopes of distinct reaction norms are significantly different from each other.

From a reaction norm perspective, therefore, it simply makes no sense to attempt to separate genetic from environmental effects, because the

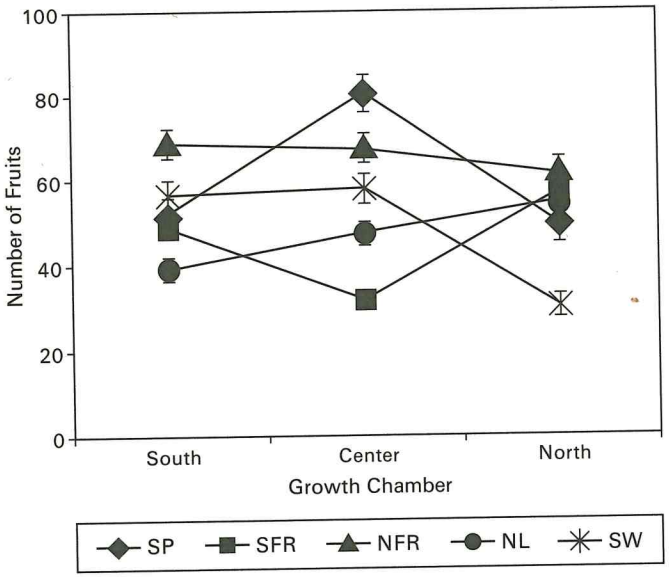

Figure 14.1
A simple set of norms of reaction from a plasticity experiment conducted on the model plant system *Arabidopsis thaliana*. The horizontal axis represents the environment (in this case the simulated photoperiods of northern, central, and southern latitudes in Europe), and the vertical axis is a measure of phenotype. Each line represents a distinct genotype's reaction norm. The diagram illustrates the properties of genetic variation (different heights of the various norms), environmental variation (when individual norms have a slope different from zero) and genotype-by-environment interaction (when different norms are not parallel to each other). Symbols to the right indicate the provenances of individual populations: SP = Spain; SFR = Southern France; NFR = Northern France; NL = Netherlands; SW = Sweden (Data from Josh Banta, Pigliucci Lab)

"genetic," "environmental," and interaction variances are all properties of the specific genotype–environment combinations that are characteristic of a given population of organisms. To use a metaphor first introduced by Richard Lewontin, if you were building a house with bricks and lime, it would make little sense to estimate the different contributions of the two components by weighing them: the house is made of the specific pattern of brick-and-lime layering, though of course if you really insisted, you could count the bricks and weigh the lime; it just would not tell you much of interest. This has strong implications for the concept of heritability, which Lewontin (1974)—on the basis of a reaction norm perspective—showed to be entirely dependent on the specific combination of genotypes and environments one is considering: change the environment, and the "genetic" variance in the population might,

counterintuitively, change; alter the frequency of different genotypes and, perhaps even more counterintuitively, the "environmental" variance is altered as well. Since heritability is by definition the ratio of genetic to total (i.e., including environmental) variance, one can easily see how heritability is anything but a fixed feature of a population, and even less so of a species.

While norms of reaction are usually depicted as continuous functions of the environment, the phenomenon of phenotypic plasticity is more general, and some of its most spectacular manifestations are the well-known "polyphenisms" that characterize plant and animal species. For instance, different larval diets produce male horned beetles with or without the horn (Moczek 2006), a trait that dramatically influences their ability to compete for mates. In plants, one of the earliest examples of adaptive phenotypic plasticity ever studied is a type of polyphenism: heterophylly in semiaquatic plants is an instance of developmental plasticity in which the organism produces different types of leaves depending on whether it finds itself below or above water (Cook and Johnson 1968; Wells and Pigliucci 2000; see figure 14.2), the advantage being that each type of leaf is best suited—both morphologically and physiologically—to its own environment.

Phenotypic plasticity is now the paradigmatic way of thinking about gene–environment interactions (the so-called nature–nurture problem), and one of the best studied biological phenomena in the evolutionary literature, with knowledge steadily advancing about its genetic–molecular underpinnings (Schlichting and Smith 2002; Suzuki and Nijhout 2008), ecological role (Callahan and Pigliucci 2002; Nussey et al. 2007), and evolution (Pigliucci et al. 2003; Paenke et al. 2007). In the rest of this chapter I will explore how evolutionary biologists are now using the concept of plasticity to expand the horizons of the Modern Synthesis of the 1930s and 1940s (Mayr and Provine 1980), and what role plasticity may play in the shaping of an Extended Evolutionary Synthesis.

Plasticity, Buffering, and Capacitance

measure of ability of a pop to produce the same phenotype regardless of its env. or genotype is robustness

For a long time in the literature the idea of phenotypic plasticity has been linked to those of homeostasis, canalization, and buffering (Flatt 2005). Although plasticity is often portrayed as the opposite of canalization, it is easy to see why this cannot strictly be the case: a canalized phenotype is one that is reliably produced by the developmental system;

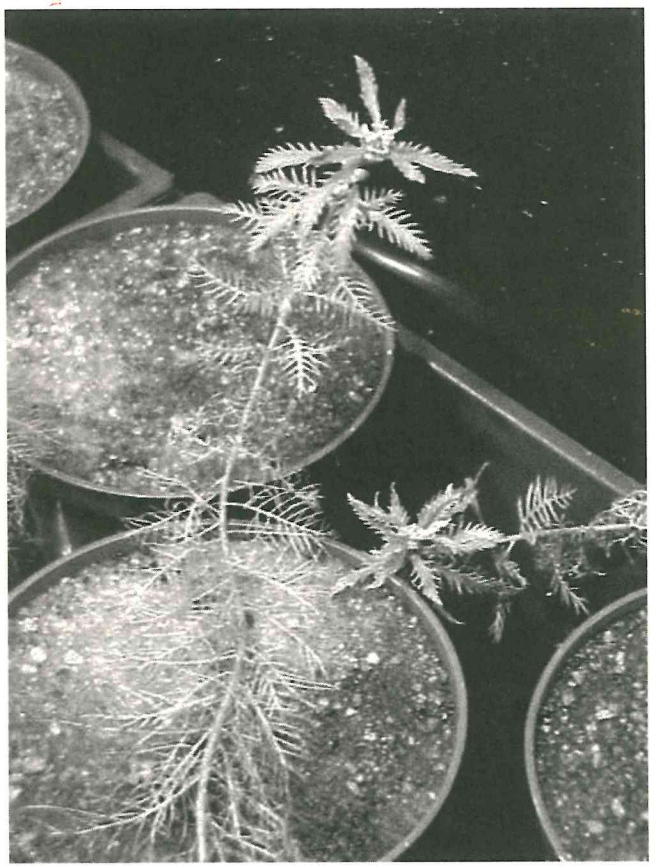

Figure 14.2
An example of adaptive plasticity in plants, the polyphenism known as heterophylly (the ability to produce distinctly shaped leaves depending on whether the stem of the plant is below or above water). The species photographed here is *Proserpinaca palustris*. (Photo by Carolyn Wells, Pigliucci Lab)

but a reaction norm can also be a reproducible (set of) phenotype(s), given a particular genotype and range of environments. In other words, there is no contradiction in speaking of canalized plastic norms of reaction; hence, canalization is not the opposite of plasticity. On the other hand, there does seem to be an inverse relationship between plasticity and (environmental) homeostasis, if one understands homeostasis as the maintenance of a given phenotype regardless of (external) conditions. Finally, "buffering" is a generic term for whatever mechanism leads to genetic or environmental homeostasis.

plasticity ↔ homeostasis.

To summarize the differences and relationships among these biological phenomena, then:

- Phenotypic plasticity is a trait- and environment-specific property of the genotype which may or may not be advantageous and may or may not be the result of adaptive evolution (i.e., natural selection).
- Canalization is a property of the developmental system that allows it to reliably reproduce the same phenotype under the same set of conditions. Plasticity can be canalized, and canalization is usually thought of as a derived condition, resulting from natural selection.
- Homeostasis comes in two flavors: environmental and genetic. Either way, it means that the developmental system is resilient to changes in the external or internal environment. A plastic reaction norm is not (environmentally) homeostatic, by definition. Homeostasis is also usually thought of as a derived evolutionary outcome, brought about by natural selection.
- Buffering refers to the range of mechanisms that cause homeostasis, which means that a breakdown in buffering may lead to (probably nonadaptive) phenotypic plasticity.

Several authors have argued that nonadaptive plasticity must be considered a default attribute of biological systems, because a variety of environmental factors, such as temperature, pH, and others, alter the functionality of biomolecules by default, simply as a result of standard biochemistry. As Newman et al. (2006: 290) put it: "Much morphological plasticity reflects the influence of external physico-chemical parameters on any material system and is therefore an inherent, inevitable property of organisms." According to Nijhout (2003: 9): "Temperature, nutrition, photoperiod, and so on . . . affect the underlying chemical and metabolic processes of development directly, without the intervention of a specially evolved mechanism . . . phenotypic plasticity is obtained gratis, as a by-product of the physics and chemistry of development." A typical example is represented by the well-known temperature–activity curves that are characteristic of enzymes: while the specific shapes of these curves vary with the protein being studied, enzymes simply cannot avoid having an optimal (and usually narrow) range of temperatures, flanked by temperatures at which they can still function, but suboptimally, and finally by temperatures at which they have no detectable biological activity. This kind of phenomenon helps us make sense of the above-mentioned idea that environmental homeostasis, where it exists, must be an evolutionarily derived state of things.

biochemical constraint

By the same token, continuous reaction norms like those depicted in figure 14.1 may not necessarily be adaptive, but simply the inevitable result of a developmental system exposed to different environmental conditions. On the other hand, more structured plastic responses, such as the polyphenisms mentioned earlier, are usually thought of as the result of natural selection because their sharply distinct phenotypes are often associated with clearly advantageous functions. The obvious inference is that polyphenisms evolve from preexisting, continuous, reaction norms.

It is the interplay between inherent plasticity and the necessity to maintain functionality over a range of environments that links the evolution of phenotypic plasticity to that of buffering mechanisms, as was first glimpsed by Waddington (1942, 1961) with his discussion of what at the time were still termed "acquired characters." (Waddington did not use the reaction norm terminology or framework, although today we can see that his famous mechanism of genetic assimilation is really an example of selection on the shape of an organism's reaction norm. I will return to this point later in the chapter.) Waddington was interested in the generation of novel phenotypes, using an environmental stimulus simply as a trigger. The end product of his process of genetic assimilation was a "canalized" trait that would no longer require the environmental trigger. Though Waddington was successful in demonstrating by experiments on Drosophila that his hypothetical mechanism could in fact work, the standing criticism of that body of work has always been that genetic assimilation had not been demonstrated to occur under natural conditions (of course the fact that few people bothered to look for it should have significantly weakened that objection, but somehow failed to do so).

The situation changed recently with the onset of research on so-called "capacitors" of phenotypic evolution, an approach that in a sense turned Waddington's interest on its head, focusing on how the disruption of a buffering system may yield an explosion of phenotypic forms and a surge in phenotypic plasticity, thereby providing new raw material for natural selection to work on (Rutherford and Lindquist 1998; Queitsch et al. 2002). This body of work—ironically, still conducted by means of experimental protocols on model systems—has prompted a new appreciation of Waddington's ideas, now cast in the modern language of reaction norms and capacitance. It is an entirely open question whether and to what extent these phenomena are relevant to organismic evolution, but there are at least compelling arguments based on computer models that natural selection may in fact favor capacitance (Masel 2005), and therefore plasticity, as an intermittently major player in the generation of phenotypic novelties.

Whether one looks at the problem of the generation of novel phenotypic variation from the point of view of genetic assimilation or from that of capacitance—which, I am arguing, are in fact two sides of the same coin—phenotypic plasticity emerges as a key player, either in allowing the initial steps leading to assimilation or in providing the raw material for renewed evolution after the disruption of a buffering system. In either case, the idea is that phenotypic evolution may occur surprisingly fast, within the span of a few generations. This has led Murren and myself (Pigliucci and Murren 2003) to suggest one counterintuitive reason why it may be difficult to find natural examples of genetic assimilation (other than the already mentioned fact that few people have been looking for them): the telltale signs may be gone from a natural population so quickly as to induce the investigator to think that all that is going on is standard selection on genetic variants, just as prescribed by the theoretical framework of the Modern Synthesis. There are ways around this problem, as discussed in Pigliucci and Murren (2003), once one knows what to look for, but this issue will come back a fortiori when we consider West-Eberhard's ideas about phenotypic and genetic accommodation below.

The possibility that plasticity may facilitate the evolution of fast phenotypic changes has consequences in at least two areas of evolutionary biology: the study of invasive species and the question of speciation. Richards et al. (2006) have pointed out that phenotypic plasticity may allow a potential invader to establish populations at low demographic levels, essentially buying time until the standard genetic variation–selection mechanism kicks in and allows the invader to fine-tune its adaptation to the novel environment. This would neatly account for an oft-observed pattern characterizing biological invasions in which the invader colonizes multiple locations at low population densities and then survives at those densities for a "lag time" that may last decades. Suddenly one or more of the established populations then experiences an aggressive bout of growth, and the devastating part of the invasion begins. This is precisely the pattern that a "plasticity first" hypothesis would predict, and there is an increasing interest in the invasive biology community in empirically testing this role of phenotypic plasticity in ongoing demographic changes of alien species.

As for the link between plasticity and speciation, here is what Levin (2004: 808) has to say: "An ecological shift most often involves the

occupation of novel habitats in the physical and genetic vicinity of the source population . . . plasticity buys populations time to adapt, in that they may persist across generations without genetic alteration. . . . Long-term population survival of the newly founded populations is conditional on genetic refinement." This is precisely the idea I have articulated a few lines above in the context of invasions, which Levin independently applies to the possibility of ecological speciation. This mode of speciation has received increasing attention recently (Fournier and Giraud 2008; Rasanen and Hendry 2008), but to my knowledge little has yet been done to integrate a reaction norm perspective into the empirical study of speciation driven by natural selection.

The Mechanics of Plasticity: Development, Genetics, and Epigenetics

In order to understand how deep a role phenotypic plasticity plays in the restructuring of evolutionary theory, one has to consider issues related to the mechanics of plasticity in terms of molecular basis (genes, proteins, and hormones involved), as well as of epigenetic effects and development more broadly construed. Let me start with the latter.

If plasticity, especially inherent (i.e., not necessarily adaptive) plasticity, is about anything, it is about the direct influence of the environment on the developing phenotype. As Moore (2003) pointed out, the mechanical environment in particular (meaning whatever mechanical forces may be applied to the organism during development) can play four distinct functions: it can (a) act as a selective environment, essentially discriminating between developmental processes that do and do not work for a particular type of organism; (b) provide cues for the developmental processes themselves; (c) be itself modified by the organism; or (d) alter the morphogenetic process by means of the inherent plasticity of the developing organism. Examples of the latter possibility in particular are easy to find, and are beginning to play an increasingly important role in our understanding of the evolution of adaptive organismal forms. Just think of the shapes of colonies of scleractinian corals, which are directly molded by water currents, or of the instantaneous transition to bipedalism in some mammals effected by developmental defects in the forelimbs—the so-called "bipedal goat effect." Figure 14.3 shows a less well known, but not for that less spectacular, case involving the model system Arabidopsis thaliana. Genotypes of this plant react dramatically to exposure to mechanical stimulation, which in nature can be generated by both abiotic and biotic factors (wind, rain, snow, trampling, or herbivory, to

Figure 14.3
A spectacular example of mechanically induced plasticity in the model system *Arabidopsis thaliana*. The two plants have identical genotype, but the one on the left has been exposed to gentle mechanical stimulation throughout its growth (in nature this may be caused by abiotic factors such as wind and rain, or by biotic ones such as herbivory). Notice not just the very different sizes of the two organisms, but also the distinct branching architectures. (Photo by Janet Braam, used with permission)

mention some). The genetic basis of this response has been investigated (Braam 2005), but—as is often the case—we are far behind in terms of understanding its ecology and evolution. Nonetheless, it is tempting to see simple plasticity to mechanical stimuli as the vehicle through which specialized alpine ecotypes of a variety of plant species may have evolved all over the world (Alokam et al. 2002).

Newman et al. (2006) go much further and actually propose an elaborate hypothesis that places plasticity to mechanical (and chemical) stimulation at the center of the origin of nothing less than the variety of animal body plans. These authors identify two key cell properties, differential adhesion and cell polarity, as well as four types of patterning mechanisms: diffusion gradients, sedimentation gradients, chemical oscillation, and reaction–diffusion. They then show how different combinations of these elements—all mediated by inherent physical–chemical plasticity—can generate the fundamental body plans found in the animal kingdom. The point, of course, is not that genes had nothing to do with the evolution of developmental processes, but rather that the initial facilitation may have come through nongenetic mechanisms which would lead the way, so to speak, in a manner analogous to what I have discussed above in the cases of invasions and ecological speciation.

Moving from development to genetics, one simply cannot write about the mechanistic bases of plasticity without asking whether there are genes "for" it. Famously, Via (1993) answered in the negative, suggesting that plasticity evolves as a by-product of selection within environments. I have elsewhere (Schlichting and Pigliucci 1995) made the argument that Via is fundamentally mistaken here, apparently not realizing that the two possibilities are certainly not mutually exclusive, and that there are plenty of examples in the literature of genes that cannot possibly be conceived as being for anything other than adaptive phenotypic plasticity, for instance, light-sensitive phytochromes in plants (Ballaré 1999). Whether a gene can reasonably be considered "for" a trait is actually a complex question that philosophers of science have debated for some time, but it seems clear that if anything satisfies the rather stringent set of requirements laid out by Kaplan and Pigliucci (2001), then genes underlying known adaptive plasticity syndromes such as the shade avoidance response in plants certainly qualify. The important point is that in order to make a case for a gene being "for" something, one has to know quite a bit about both the molecular–developmental biology of the system and its ecology–evolution. Well-studied instances of adaptive

plasticity provide precisely this intellectually satisfying conjunction of molecular and organismal biological research.

Of course the mechanistic bases of phenotypic plasticity are not limited to genes, and in fact here, too, studying plasticity is providing increasingly convincing examples of how to do so-called "integrative biology" while at the same time highlighting the limitations of the Modern Synthesis's simplistic view of genes-to-phenotypes mapping. The functionally flexible hormonal systems of both plants and animals (though the two do work very differently) have offered a starting point from which to understand how environmental signals are translated, interpreted, and reacted to by the organism (Friml and Sauer 2008). Nijhout (2003: 9) concluded that "the development of alternative phenotypes in reaction norms and polyphenisms can be caused by especially evolved mechanisms that are regulated by variation in the patterns of hormone secretion," while according to Badyaev (2005: 880), "phenotypic assimilation of the appropriate stress response is . . . facilitated by a common involvement of neural and endocrine pathways of the stress response in other organismal functions." Finally, Crespi and Denver (2005: 50) commented that "the neuroendocrine stress axis represents a phylogenetically ancient signaling system that allows the fetus or larva to match its rate of development to the prevailing environmental conditions."

More recently, yet another layer of investigation of the mechanisms of phenotypic plasticity has been brought into the discussion, with the possibility that epigenetic processes such as methylation patterns and interference RNAs may be involved in mediating plastic responses (Bossdorf et al. 2008; Jablonka and Raz 2008). Epigenetic inheritance has long been suspected to play some role in the connection between environmental stimuli and heritable phenotypic responses, as in the classic case of floral characteristics in flax (Cullis 1986). But it has been only recently, with the conceptual articulation of multiple "dimensions" of the evolutionarily relevant inheritance systems (Jablonka and Lamb 2005), that we begin to see the coalescence of a coherent theoretical picture encompassing the entire spectrum of phenotypically plastic responses, from their mechanistic generation to their inheritance, to their evolutionary ecology. Much more work needs to be done, of course, but we now have in place a sufficient number of pieces of the puzzle to turn to an examination of the most complete and current model of how plasticity plays a potentially major role in the evolution of phenotypic novelties (see chapter 12 in this volume), a role that simply could not have figured in the conceptual arsenal of the Modern Synthesis.

Phenotypic and Genetic Accommodation

The current buzzword in discussions of the macroevolutionary implications of phenotypic plasticity is "accommodation," a term introduced by West-Eberhard (2003) and much discussed since (Crispo 2007). West-Eberhard's ideas clearly are historically related to those of a series of scholars, chief among them James Marc Baldwin, Conrad Hal Waddington, and Ivan Ivanovich Schmalhausen. In this section I will briefly summarize these historical antecedents and then present a discussion of the modern sense of phenotypic and genotypic accommodation. I suggest that contemporary writers both be aware of the historical precedents (and accordingly recognize intellectual priorities) and adopt West-Eberhard's more modern and compact terminology.

Baldwin (1861–1934) wrote a historical paper on what is now known as the "Baldwin effect" (a term introduced by Simpson in 1953 to criticize it), titled "A New Factor in Evolution" (Baldwin 1896). In it, he presented ideas similar to those developed independently by Morgan (1896) and Osborn (1896), aimed at explaining the role of behavior—and of what today we would call phenotypic plasticity—in evolution. The discussion was framed in the then still relevant context of the possibility of Lamarckian processes, with which Darwin himself had flirted. Baldwin's idea was that behavior can affect the action of natural selection, in some instances facilitating it. The result would be something that would look like acquired inheritance, but that in fact was due to this additional "factor" that simply interacted with, but did not invalidate, the role of selection (Baldwin was no Lamarckian). It is actually difficult to read Baldwin unambiguously, because he was writing in a pre–Mendelian world (Mendel's work had been published, but was yet to be broadly acknowledged). Nonetheless, the Baldwin effect has been explored more recently in works dealing with the interaction between learning and evolution (e.g., Hinton and Nowlan 1987; Mayley 1996). The most sensible modern interpretation of Baldwin is that phenotypic plasticity can facilitate evolution by natural selection, depending on the particular combination of shape of the reaction norms and of the selection pressures in a given population of organisms: in particular, if some of the reaction norms happen to produce a viable (if suboptimal) phenotype in a novel environment, then those genotypes will have a chance to survive, and the population, to establish itself. After that, as discussed above, natural selection will fine-tune the reaction norm by its standard filtering of existing and novel genetic variation.

Waddington's (1905–1975) conceptual and experimental work on related subject matters spanned several decades (Waddington 1942, 1961), and it is a bit easier to frame in modern terms because it was published during the genetic–molecular revolution, as well as largely after the Modern Synthesis itself—although, interestingly, Waddington, too, framed his ideas in terms of a Darwinian explanation of alleged "acquired" characteristics, against Lamarckism. We have already briefly examined Waddington's idea of genetic assimilation, which is closely related (though not identical) to the Baldwin effect. As in the Baldwin effect, it is preexisting variation for plasticity that makes it possible for a fraction of the population to produce a novel phenotype in response to an environmental stimulus. In contrast to the cases of interest to Baldwin, however, Waddington focused on the evolution of a newly canalized trait, which would eventually be stabilized regardless of the continued presence of certain environmental circumstances (evolution of environmental homeostasis). In this sense, although Waddington actually showed experimentally that his mechanism could work, genetic assimilation is probably of less broad interest than the original Baldwin effect.

Schmalhausen (1884–1963), despite having done his work contemporaneously with Waddington's early production, is more obscure and difficult to read—partly because we have to rely on Dobzhansky's translation from the original Russian, and partly because he was more isolated from mainstream science and developed his own terminology. Regardless, his process of "stabilizing selection" (Schmalhausen 1949) should not be confused with what we mean today by that term (i.e., with selection for maintenance of the current population mean, having the result of lowering the population's variance for the trait under selection). Rather, stabilizing selection sensu Schmalhausen is closely related (but, again, not identical) to both genetic assimilation and the Baldwin effect, especially the latter. Schmalhausen envisioned a process by which a shift in environmental conditions would trigger selection for a new "norm" (of reaction), essentially describing a mode of evolution of phenotypic plasticity. Eventually, the new norm is "stabilized" if the environmental shift persists (differing in this from genetic assimilation), producing a population adapted to the novel conditions. In a sense, Schmalhausen's ideas can be interpreted as a generalization of the Baldwin effect that applies to any plastic trait, not just to behavioral ones.

West-Eberhard (2003) has updated the concepts and terminology accumulated by Baldwin, Waddington, Schmalhausen, and a number of

others throughout the twentieth century, and essentially distilled it into two key phenomena: phenotypic accommodation and genetic accommodation. In her words:

> Phenotypic accommodation is the adaptive mutual adjustment, without genetic change, among variable aspects of the phenotype, following a novel or unusual [external or internal] input during development. (West-Eberhard 2005a: 610)

> Genetic accommodation is simply quantitative genetic change in the frequency of genes that affect the regulation or form of a new trait. (emphasis added; West-Eberhard 2005b: 6547)

Phenotypic accommodation, therefore, is a direct consequence of the inherent plasticity of developmental systems, and—in West-Eberhard's definition—is related to the concepts of environmental and genetic homeostasis. Genetic accommodation, as should be clear from the above quote, simply refers to the standard mechanism of genetically enabled evolutionary change envisioned by the Modern Synthesis, applied to the specific case of phenotypic novelties; the reason for the introduction of a new term here is simply to provide a unified model of evolutionary change under the broad rubric of "accommodation."

Indeed, West-Eberhard goes on to present what she sees as a four-step recipe for evolutionary change in general:

1. A novel input affects one (in the case of mutation) or several (in the case of an environmental change) individuals in a population.
2. Because of inherent developmental plasticity, we observe phenotypic accommodation of the novel input; consequently, a novel phenotype emerges.
3. The initial spread of the novel phenotype may be rapid (if it is due to an environmental effect) or slow (if it is the result of genetic input).
4. If the novel phenotype is advantageous, natural selection "fixes" it by stabilizing its appearance through an alteration of the genetic architecture; genetic accommodation has occurred.

There are two key conceptual points in this list that need to be appreciated. First, the fact that phenotypic change always begins with the plastic reaction of the developmental system to a genetic or environmental perturbation. Of course, there is no claim that such plastic response will be adaptive (in the sense of advantageous to the organism under current conditions), but there is in fact a good chance that it will be adaptive because—the idea is—developmental systems have been selected in the

past to maintain functionality in the face of a broad range of perturbations. Moreover, the "novel" environment (if the change is environmental in nature) will often not be entirely novel at all, but will be some variant of the sort of environment that has been common through the history of the species in question. This makes it even more likely that the existing developmental norm of reaction will produce at least a suboptimal phenotype.

Second, notice that—if the novel stimulus is environmental—there will likely be several developmental systems that will respond in similar fashions, because the corresponding reaction norms will be similar. This is crucial, because it may lead not only to the appearance, but also to the prevalence (or at least the non-rarity), of the new phenotype in the population. This will in turn facilitate the process of genetic accommodation that follows, because several individuals simultaneously will provide the raw material for selection to act on.

By far the most famous example of phenotypic accommodation is the so-called bipedal goat effect mentioned earlier. It refers to the fact that mammals' (and perhaps other organisms') developmental plasticity allows animals that are born with nonfunctional forelimbs to adjust their muscular and skeletal system and adopt a bipedal posture. The first recorded case is that of a goat with such condition, studied in detail by Slijper (1942), but similar cases are common in dogs and even in primates. Figure 14.4 shows the case of a bipedal macaque found in an Israeli zoo; the animal had suffered from a severe and life-threatening infection that had paralyzed its upper limbs. It is hard to stare at pictures like this one and not think that perhaps this sort of phenotypic plasticity is what first opened the way to the evolution of bipedalism in hominids. This is, of course, speculation, but surely a very tempting one.

More generally, and on more solid developmental and evolutionary ground, some authors have called attention to the role of bone morphogenetic proteins in structuring the inherent plasticity of vertebrate developmental systems. These proteins are involved in the shaping of spectacular phenotypes such as the turtle's carapace, bat wings, the spotted hyena's sagittal crest (from chewing food), the jaws of cichlid fish (in response to diet), and the shape of bird bills (e.g., in Darwin's finches, again in response to diet) (Young and Badyaev 2007). Most of these examples, presumably, are of genetic accommodation, because the original plastic response has been stabilized (in the sense of Schmalhausen) in the currently existing populations, and it has a clearly adaptive meaning. The difficult task, of course, is to uncover convincing examples

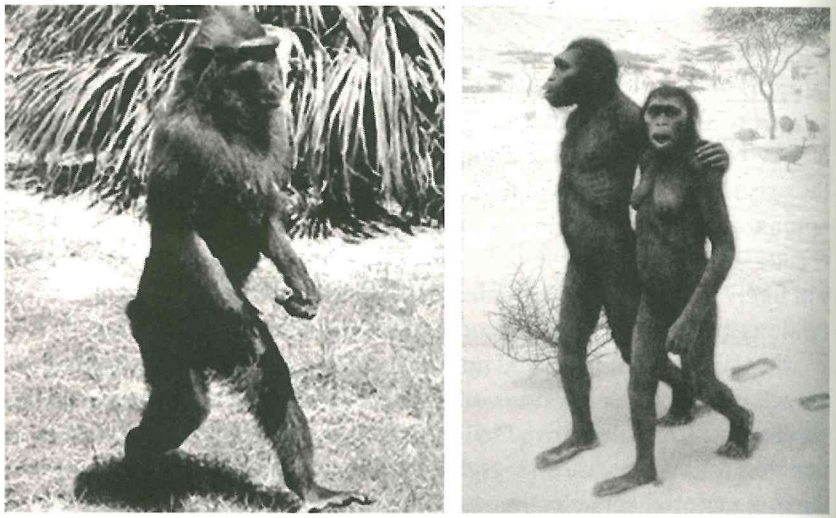

Figure 14.4
(Left) Phenotypic accommodation in a macaque suffering from a crippling disease that made its upper limbs nonfunctional from early on in development, and led—through the inherent plasticity of the developmental system—to a bipedal posture. (right) Reconstruction of two australopithecines, likely members of the line of ancestry that led to humans. Could they have started their evolution toward bipedalism by a similar process of phenotypic accommodation? (Original attribution of left photo unknown, various copies on Internet; right drawing, Wikipedia Commons)

of transition from phenotypic to genetic accommodation and, moreover, to show that the phenomenon is sufficiently common to be evolutionarily relevant. Studies of this type are beginning to appear in the literature (Gomez-Mestre and Buchholz 2006; Suzuki and Nijhout 2008), though it is too early to draw broad conclusions on their generality.

Consequences for an Extended Evolutionary Synthesis

What are the consequences of contemporary views on phenotypic plasticity for the possibility of expanding the Modern Synthesis into an Extended Evolutionary Synthesis? There are at least six that should help provide a blueprint for future research on both the theoretical and the empirical fronts:

1. As West-Eberhard (2003) pointed out, genes could come to be seen as "followers" rather than leaders in the evolutionary process, a change that may have little impact on, say, research in molecular genetics, but that would represent a major conceptual shift in evolutionary theory. As

West-Eberhard (2005b: 6547) puts it: "I consider genes followers, not leaders, in adaptive evolution.... We forget that ... environmental factors constitute powerful inducers and essential raw materials whose geographically variable states can induce developmental novelties as populations colonize new areas." This, of course, is true if the Baldwin effect, genetic assimilation, and related phenomena are in fact frequent enough in nature.

2. Phenotypic–genetic accommodation could come to be considered a major explanation behind the well-known phenomenon of mosaic evolution. The latter's textbook definition is along the following lines: "Evolution of different characters at different rates within a lineage is called mosaic evolution.... It says that an organism evolves not as a whole, but piecemeal" (Futuyma 1998). Except that if the "two-legged goat" effect and similar phenomena are frequent, we would have the appearance of mosaic evolution in the fossil record, even though most of the changes would have occurred simultaneously, made possible by the inherent phenotypic plasticity of developmental systems.

3. Phenotypic plasticity should also be seriously considered as a candidate mechanism for another well-known evolutionary phenomenon, pre-adaptation. Futuyma's (1998) definition of the term is "Possession of the necessary properties to permit a shift into a new niche or habitat. A structure is pre-adapted for a new function if it can assume that function without evolutionary modification." But the concept of pre-adaptation of a structure to a new function may appear rather spooky if there is no further elaboration about exactly how such pre-adaptation comes about. As I mentioned earlier, however, this is clarified once we realize that most new environments are in fact correlated to historical environments, and that therefore the variation for phenotypic plasticity existing in a given population is not altogether unlikely to include reaction norms that will work in at least a suboptimal fashion in the new environment (or for the new "function"). This is what Baldwin called "organic selection."

4. Another consequence stressed by West-Eberhard (2005a: 611) is the pre-eminent role that behavior plays in directing evolutionary change: "Behavior is, of course, a common mediator of normal skeletal and muscle development because it is especially flexible in response to environmental contingencies. It follows that behavior must often be an important mechanism in the origins of novel morphological traits. So we have to list behavior and its neuro-endocrinological underpinnings, alongside genomic changes, as among the primary developmental causes

of morphological novelty." This is, of course, what Baldwin was interested in to begin with. The point can be further broadened to all life forms if we consider phenotypic plasticity as a generalized equivalent of behavior (as is in fact done by several authors: Mayley 1996; Novoplansky 2002; Paenke et al. 2007).

5. Phenotypic plasticity should also be considered as a major player in the process of niche construction (Odling-Smee et al. 2003; Okasha 2005; Laland and Sterelny 2006), which, of course, is itself still a somewhat controversial concept (but see chapter 8 in this volume). According to Stamps (2003): "animals frequently select their own environments, or modify their environments through their own actions," which is an obvious reason why plasticity (in this case in the form of behavior) is important in this context. However, as Jablonka (2007) put it: "because it is difficult to recognize the role that persistent environmental or developmental inputs play in ontogeny, their effects are usually attributed to genetic inputs," which means that there is a built-in tendency by biologists who work within the framework of the Modern Synthesis to simply attribute phenotypic change to genes without further consideration of the developmental and epigenetic alternatives (but see chapter 7 in this volume).

6. Finally, phenotypic plasticity probably plays a hitherto largely ignored part in one of the fundamental phenomena of the biological world: speciation. Again Jablonka (2007): "Heritable nongenetic variations may initiate population divergence and lead to speciation. Gottlieb interpreted the well-known case of sympatric speciation in apple maggots (Rhagoletis pomonella) in these terms." And, along similar lines, West-Eberhard (2005b): "Geneticists may end up describing the results of speciation rather than its causes." Then again, it is still hard enough to get some biologists to take seriously even the possibility of sympatric speciation (Coyne and Orr 2004), despite documented examples in a variety of organisms (e.g., Doebeli and Dieckmann 2000; Fournier and Giraud 2008).

All of the above directly implies, it seems to me, specific steps within a research program on phenotypic plasticity and its macroevolutionary impact. This work would cover three major areas: empirical research in organismal biology, empirical research in mechanistic biology, and theoretical/conceptual investigations.

With respect to empirical research in organismal biology, we need further—and better characterized—examples of genetic accommodation. This may not be easy, because, as I mentioned above, the process may be too quick for evolutionary biologists to be able to detect, if they

do not know what they are looking for. Awareness of the stages of the process, of course, is crucial. As Darwin put it long ago while discussing the relationship between theory and "data": "How odd it is that anyone should not see that all observation must be for or against some view if it is to be of any service!" (from a letter to Henry Fawcett). This should be remembered by anyone who dismisses genetic accommodation (or sympatric speciation, or epigenetic inheritance, or niche construction) on the simple basis that "we do not have compelling data in its favor." Compelling data, as Darwin understood, do not simply emerge from a collection of facts about the world. No new theory has ever declared itself from beneath a heap of facts.

Similarly, we need more examples of phenotypic accommodation in order to better assess its short-term ecological impact and long-term evolutionary relevance. This, however, is actually simpler to achieve, as the literature on phenotypic plasticity is very rich in potential examples, and the empirical and analytical methods to study the phenomenon are well established (Pigliucci 2001). Also, there needs to be more integration of research programs on phenotypic plasticity, on the one hand, and on behavioral ecology and genetics, on the other. While students of behavior are certainly aware of phenotypic plasticity, and vice versa for researchers in the plasticity field, good examples of how the two relate to each other are still surprisingly uncommon in the literature (but see Charmantier et al. 2008).

Empirical research on the mechanistic aspects of phenotypic plasticity has, of course, been carried out for several years, and it has reached a good level of sophistication, especially when conducted on so-called model systems (e.g., Feng et al. 2008). Still, two areas of particular attention for future studies concern the role of hormones and of epigenetic inheritance systems as intermediaries between the genetic level and the actual deployment of developmentally plastic responses by the organism. Research on the role of hormones in evolutionarily relevant plasticity (e.g., Emlen et al. 2007) is comparatively much more advanced than research on heritable epigenetics, a phenomenon still largely, and I think erroneously, regarded as irrelevant to evolutionary questions (but see Bossdorf et al. 2008; Jablonka and Raz in press). The difference between the two is that we know beyond reasonable doubt that hormones (both in plants and in animals) do play a crucial role in the deployment of plastic responses, whereas the role and frequency of heritable epigenetic effects are still much more debatable, since so comparatively few examples are well characterized. Nonetheless, this may change quite rapidly

as molecular techniques become readily available for population-level screenings of epigenetic markers and, again, as conceptual awareness pushes more researchers in that direction.

As far as the theoretical evolutionary biology of macroevolutionary plasticity is concerned, several lines of inquiry can be pursued. Perhaps one of the most neglected so far has been the bridge to some interesting literature in computational science, where the concepts of learning and plasticity (broadly defined) have been explored in terms of their consequences for the evolution of artificial life systems, neural networks, and genetic algorithms (e.g., Mills and Watson 2005; Wiles et al. 2005). There is much to be gained for evolutionary biologists from a less occasional interaction with theoretical scientists in computational and cognitive sciences, as this will make it possible both to generalize the concepts arising from within each discipline, and to adopt theoretical and computational methodologies that may help biologists think outside of the box imposed by standard population–quantitative genetic models.

In turn, population and quantitative genetics, which are the theoretical–mathematical backbone of the Modern Synthesis, need to be reevaluated in light of a broader concept of what modeling in biology means (Laubichler and Müller 2007) and how it is to be pursued. Population genetics is notoriously limited in its analytic treatments to a small number of loci/alleles and to overly simplifying assumptions, without which its problems rapidly become mathematically intractable. Quantitative genetics itself, which was originally developed to address (statistically, as opposed to analytically) precisely the sort of complex problems that are outside the scope of population genetics, is beginning to show signs of reaching its own limits in terms of generality and applicability to biologically relevant questions (Pigliucci and Kaplan 2006). Indeed, referring to the limited usefulness of quantitative genetic models of adaptive landscapes (see chapter 3 of this volume), Gavrilets (1997) concluded that a model's predictive ability—the gold standard in disciplines such as physics—is not necessarily its most important contribution to science, as models in biology are more often useful as metaphors and tools to sharpen one's thinking about a given problem. Modeling in computational science and complexity theory (Toquenaga and Wade 1996) is inherently of a different kind from the approaches that have been standard in evolutionary biology for the past century or so, which may explain why today many biologists still consider results from complexity theory as "too vague and metaphorical" to be biologically informative. That may need to change in the near future.

As I mentioned above, taking a macroevolutionary role of phenotypic plasticity seriously also broadens or reopens the discussion on rather controversial topics such as niche construction and sympatric speciation. The two cases are, of course, distinct in a variety of ways. Niche construction is something that most biologists think probably does happen, and the question is how it should be explicitly incorporated into theoretical treatments of evolution, and what such incorporation might accomplish. The case of sympatric speciation is different, since a number of biologists still think it is either impossible or, in any case, a very rare event with no broad consequences for evolutionary theory. Coyne and Orr's (2004) book, for instance, treats allopatric speciation as the null hypothesis (and reinterprets the available evidence accordingly) on the ground that it is much more likely a priori. This is not the place for a discussion of the role and limitations of null models in biology (but see Pigliucci and Kaplan 2006; Sober 2008), but at the very least the possibility of additional mechanisms leading to speciation, such as the phenotypic–genotypic accommodation sequence proposed by West-Eberhard, should sound a note of caution about premature dismissal of alternative speciation models.

Phenotypic plasticity as a concept and an area of study has had a convoluted history since its inception with Woltereck (1909). Although the idea is contemporaneous with the formal recognition of the distinction between genotype and phenotype (indeed, it arguably precedes it, in the work of Baldwin, Morgan, and Osborn), it was neglected and considered a nuisance by evolutionary biologists until the latter part of the 20th century. Nowadays it is a concept that most practicing biologists recognize as important, and yet with which many still wrestle in terms of what it means for evolutionary studies. If at least part of what I have outlined in this chapter comes to fruition, in the way of development of ideas and empirical research programs, phenotypic plasticity will significantly expand the way we think organic evolution takes place.

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15 Evolution of Evolvability

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Over the last half century evolutionary biology has been a highly active and successful field of biological research with an increasing number of journals and online outlets supporting a rapidly growing scientific literature. Many of these publication organs have citation rates equal to or higher than many established journals in molecular biology, and thus it should not come as a surprise that our knowledge of evolution is rapidly expanding. The current understanding of evolution is much different, of greater reach and depth than either that of Darwin or that of the architects of the "Evolutionary Synthesis" from the 1950s. The fact that evolutionary biology has extended beyond that of the original Evolutionary Synthesis is therefore obvious. Of course there are different ways by which a science can expand and change. Rapid expansion of the factual knowledge base is one way that science makes progress, and another is change in the conceptual makeup of a discipline. When Massimo Pigliucci recently asked in the journal *Evolution* "Do we need an extended evolutionary synthesis?" he certainly had the second mode of change in mind (i.e., the question of whether the way we explain and understand evolution has changed or should change, given what we know [Pigliucci 2007]). Here we want to discuss one specific aspect of evolutionary biology that represents a break from the research tradition of the synthesis: namely, research on evolvability and its evolution. This subject is still considered by some prominent biologists as suspect (Sniegowski and Murphy 2006; Lynch 2007). Nevertheless, a PubMed search with the keyword "evolvability" yielded 236 papers on September 4, 2008; some of them refer to evolvability in a generic sense, but a large fraction also talk about evolution of evolvability. We think that the idea of evolution of evolvability is not as radical a break from the tradition of population genetics theory as some population geneticists may think. The neglect of evolution of evolvability by the research program of the synthesis