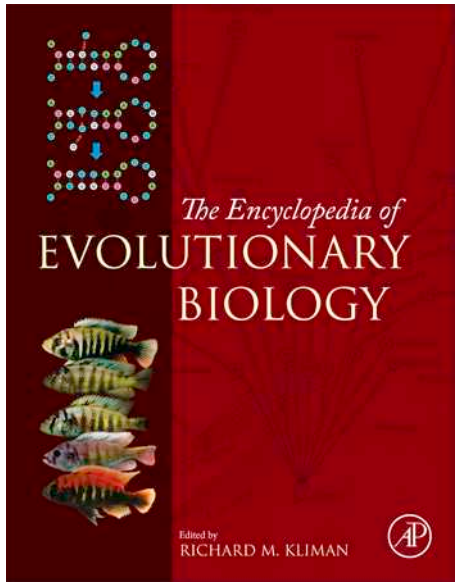

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Ecological Evolutionary Developmental Biology

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Glossary

Canalization The situation that arises when a trait is 'fixed' or expressed constitutively regardless of normal changes in the environment.

Cryptic genetic variation Variation that is expressed only under atypical conditions.

Developmental plasticity See 'phenotypic plasticity.'

Differential gene expression The situation that arises when different genes are activated, or expressed, in different cell types or organisms; nearly always accompanies 'phenotypic plasticity.'

Ecomorphs (morphs) Alternative phenotypes induced by the environment that occupy different ecological niches.

Enhancer It is involved in 'differential gene expression'; the region of a gene (i.e., a sequence of DNA) that binds 'transcription factors'; the resulting complex interacts with the gene's 'promoter,' thereby activating the gene and beginning transcription.

Epigenetics Any mechanism of development that generates phenotypic variation without altering the base-pair nucleotide sequence of DNA; phenotypic variation that involves changes in the expression of genes rather than their sequence.

Epigenetic inheritance Heritable phenotypes that are not encoded by the genome.

Genes as followers The hypothesis that the most important role of genetic mutations in evolution may be to contribute not so much to the origin of phenotypic novelties as to the pool of genetic variation that makes 'genetic accommodation' possible.

Genetic accommodation A mechanism of evolution wherein a novel phenotype, generated by either a mutation or environmental change, is refined into an adaptive phenotype through quantitative genetic changes; can result in either increased or decreased environmental sensitivity of an induced phenotype; when environmentally induced phenotypes lose their environmental sensitivity, they undergo 'genetic assimilation.'

Genetic assimilation An extreme form of 'genetic accommodation' that occurs when environmentally induced phenotypes lose their environmental sensitivity over evolutionary time and become 'canalized' or expressed constitutively.

Lamarckianism The hypothesis, attributable to the French naturalist Jean-Baptiste Lamarck (1744–1829), which holds that an organism can pass on characteristics that it acquired during its lifetime to its offspring.

Maternal effects The situation that arises when a female's phenotype influences its offspring's phenotype, independent of the direct effects of the female's coding sequences on its offspring's phenotype.

Phenotypic plasticity The ability of an individual organism to change its phenotype in direct response to stimuli or inputs from the environment (often used synonymously with 'developmental plasticity').

Promoter The region of a gene (i.e., a sequence of DNA) that binds the enzyme RNA polymerase, thereby activating the gene and beginning transcription (see also 'enhancer' and 'transcription factor').

Reaction norm A graphical representation of the sensitivity of a group of organisms of the same genotype to some specific environmental variable.

Transcription factor A protein that binds to a specific DNA sequence, thereby activating the gene and beginning transcription (see also 'promoter' and 'enhancer').

Introduction

Ecological evolutionary developmental biology ('eco-evo-devo') seeks to understand how interactions between an organism's genome and environment shape its development, and how such environmentally responsive development, in turn, impacts ecology and evolution (Sultan, 2007; Gilbert and Epel, 2009). This focus on environmentally contingent development is a departure from how developmental and evolutionary biology have been studied in the past. For example, until relatively recently, developmental biology has focused on a few species ('model organisms') reared in uniform environments. This approach has fostered the view that environmentally contingent development is rare or unimportant. Similarly, evolutionary biology has traditionally regarded environmental responsiveness as simply

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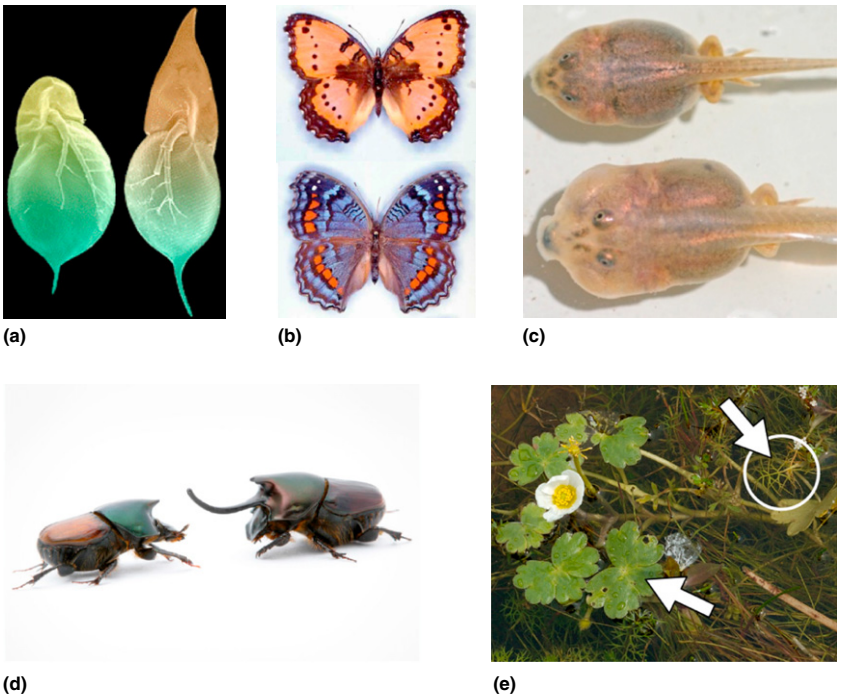

Figure 1 Examples of environmentally contingent development (i.e., 'phenotypic plasticity'). (a) Normal (left) and predator-induced (right) morphs of water fleas, *Daphnia cucullata*; (b) wet-season (top) and dry-season (bottom) gaudy commodore butterflies, *Precis octavia*; (c) omnivore (top) and carnivore-morph (bottom) spadefoot toad tadpoles, *Spea multiplicata*; (d) small-horned (left) and large-horned (right) dung beetles, *Onthophagus nigriventris*; (e) broad, aerial leaves and narrow, submerged leaves (circled) on the same water crowfoot plant, *Ranunculus aquatilis*. Reproduced with permission from Pfennig, D.W., Wund, M.A., Snell-Rood, E.C., *et al.*, 2010. Phenotypic plasticity's impacts on diversification and speciation. Trends in Ecology and Evolution 25, 459–467.

developmental 'noise' that has no long-term evolutionary consequences (Orr, 1999). Indeed, the architects of the modern synthesis of evolutionary biology explicitly cho se not to incorporate environmentally responsive development into the field's conceptual framework, partly as a reaction against Lamarckianism (reviewed in Pigliucci, 2007).

Yet, it has become increasingly apparent that an organism's environment can profoundly alter its phenotype (**Figure 1**). Moreover, it has even been suggested that such environmentally initiated phenotypic change might often precede, and even facilitate, genetic evolution and therefore plays a key role in generating biodiversity. Additionally, a growing body of evidence has revealed that environmentally induced phenotypic change can be transmitted between generations, which challenges our basic assumptions of how inheritance works (reviewed in Jablonka and Lamb, 2010; see also below).

This article explores these issues in greater detail. As described below ongoing research in eco-evo-devo promises to provide fresh insights into the evolutionary process.

Phenotypic Plasticity

For much of the twentieth century, genes were the dominant paradigm for explaining biodiversity. Yet, scholars have long suspected that genes alone do not determine which traits organisms produce (Bonduriansky, 2012). Indeed, it is now abundantly clear that the environment is a normal and necessary agent in phenotype production (Schlichting and Pigliucci, 1998; Gilbert and Epel, 2009). Nowhere is this point more clearly illustrated than by the widespread occurrence of phenotypic plasticity (Nijhout, 2003; West-Eberhard, 2003).

'Phenotypic plasticity' (often used synonymously with 'developmental plasticity') is the ability of an individual organism to change its phenotype in direct response to stimuli or inputs from the environment (sensu West-Eberhard, 2003). As it turns out, many (perhaps all) organisms can alter their phenotype in response to a diverse array of environmental factors, such as temperature, nutrition, light, pressure, or gravity, and the presence of predators, parasites, or competitors (Gilbert and Epel, 2009). For instance, temperature can influence phenotype production, because nearly all enzyme activity is temperature-dependent (**Figure 1(b)**); food often contains potent chemical signals that induce phenotypic change (**Figure 1(d)**); light can stimulate plants to produce different-shaped leaves and shoots (**Figure 1(e)**); and pressure can cause muscle and bone to grow differently. Moreover, predators can release chemicals that induce defenses in their prey (**Figure 1(a)**) and competitors can cause stress (which releases stress hormones) and alter resource abundance (and thereby an individual's nutritional state), which can trigger an alternative phenotype (**Figure 1(c)**). Regardless of the precise environmental factor, a feature common to all the above is that phenotypic plasticity is nearly always accompanied by changes in gene expression (Gilbert and Epel, 2009; e.g., see **Figure 2**).

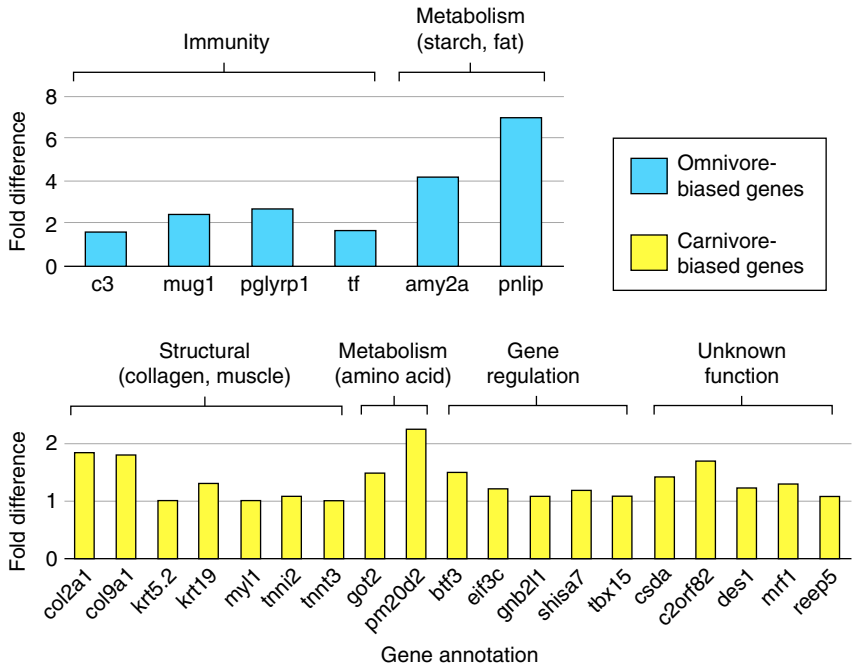

Figure 2 Differential gene expression in alternative, environmentally induced, larval ecomorphs of spadefoot toads (genus *Spea*). Depending on their diet, spadefoot tadpoles develop into either an omnivore morph, which is a dietary generalist, or a distinctive, large-headed carnivore morph, which specializes on shrimp and other tadpoles (see **Figure 1(c)**). Shown are fold differences (a measure of the change in expression level of a particular gene) for each of six genes expressed at significantly higher levels in omnivores than in carnivores and 19 genes expressed at significantly higher levels in carnivores than in omnivores; above each gene is its putative function (where known). Data from Leichty, A.R., Pfennig, D.W., Jones, C.D., Pfennig, K.S., 2012. Relaxed genetic constraint is ancestral to the evolution of phenotypic plasticity. Intergrative and Comparative Biology 52, 16–30.

Considerable effort has gone into identifying the molecular mechanisms of differential gene expression and, thus, of phenotypic plasticity (Gilbert and Epel, 2009). One model (based on studies of how different cells in a multicellular organism that share the same genome can assume different shapes/functions) is that differential gene expression is mediated by the differential binding of proteins called 'transcription factors' to a gene's enhancer region. To understand how this process works, consider that for a gene to be activated (specifically, for RNA polymerase to attach to a gene's promoter and begin transcription), RNA polymerase must be held in place on the promoter site. Transcription factors play a key role in stabilizing RNA polymerase on the gene's promoter. Importantly, different transcription factors bind different enhancers and thereby alter expression of different genes. Moreover, which transcription factors are present in any particular cell can be influenced by signals from outside the cell (e.g., in the organism's external environment). Thus, one proposed mechanism of phenotypic plasticity is that the neuroendocrine system transduces sensory information from the external environment by recruiting different transcription factors, thereby activating different genes (Nijhout, 2003) which, in turn, produce different gene products (e.g., see **Figure 2**) and ultimately, different phenotypes (**Figure 1**).

Other mechanisms can alter phenotype production, however. For instance, gene expression changes can be induced directly, as when bacteria in an animal's gut induce changes in the expression of intestinal genes (Gilbert and Epel, 2009). Future research is needed to identify additional molecular mechanisms of phenotypic plasticity.

Phenotypic Plasticity and Evolution

Although phenotypic plasticity is commonplace, its evolutionary significance remains controversial (Pfennig *et al.*, 2010; Moczek *et al.*, 2011). On the one hand, evolutionary biologists have long held that plasticity has no relevance for the evolutionary process other than to perhaps impede it by dampening the effects of selection (reviewed in Schlichting, 2004). On the other hand, several prominent, early evolutionists, such as Weismann, Goldschmidt, Schmalhausen, and Waddington, maintained that phenotypic plasticity plays a central role in the origins of new traits and phenotypic differences between species (Jablonka and Lamb, 1995).

Indeed, for over a century, researchers have hypothesized that environmentally induced phenotypic change might facilitate genetic evolution and thereby fuel the origins of new, ecologically relevant traits (Baldwin, 1902; Schmalhausen, 1949 [1986]; Waddington, 1953; West-Eberhard, 2003). Although various mechanisms have been proposed (dubbed the 'Baldwin effect,' 'genetic assimilation,' 'stabilizing selection,' and 'genetic accommodation'), all such mechanisms assume that: (1) environmentally induced phenotypes evolve first; and (2) selection favors those phenotypes (whether induced or not) that are the most adaptive (West-Eberhard, 2003).

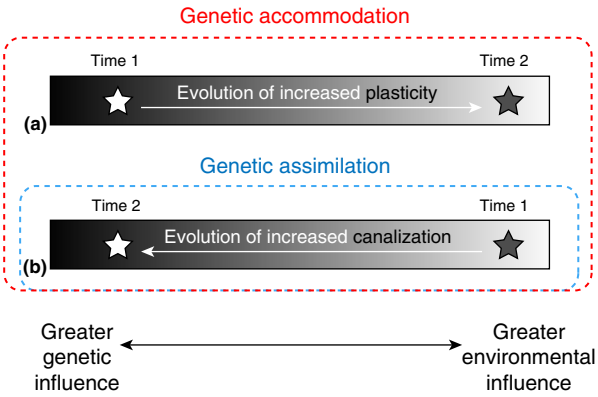

Figure 3 A diagram illustrating the distinction between genetic accommodation and genetic assimilation. Genetic accommodation is any adaptive genetic change in the regulation and form of a phenotype. For example, a trait may evolve either (a) increased or (b) decreased environmental sensitivity (i.e., phenotypic plasticity). The complete loss of phenotypic plasticity (i.e., increased canalization) is an extreme form of genetic accommodation known as genetic assimilation.

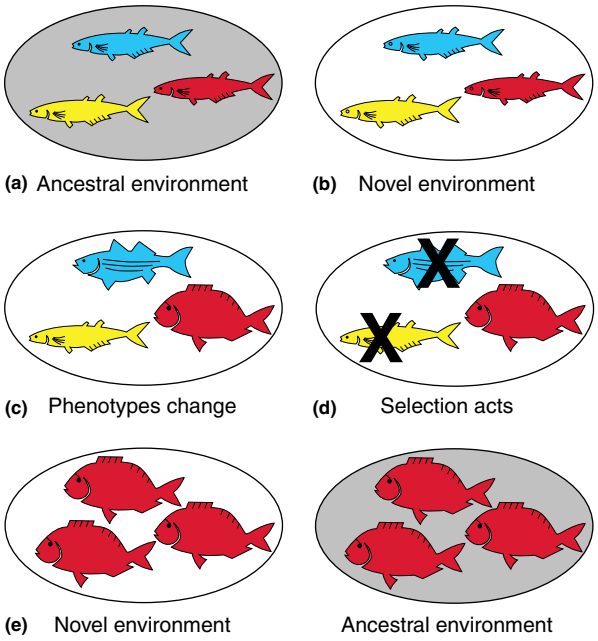

Figure 4 Phenotypic plasticity, along with genetic accommodation/assimilation, may facilitate the evolution of a new, canalized trait through the following steps (here, different fish shapes are alternative ecomorphs; different colors are different genotypes). (a) A genetically variable population (b) experiences a novel environment (indicated here as a change from a shaded to an unshaded background). (c) Consequently, novel phenotypes are induced by the environment, but different genotypes respond differently. (d) Selection disfavors those genotypes that produce maladaptive phenotypes in the new environment (indicated here by an 'X'). (e) Such selection may result in the evolution of a novel, canalized trait (e.g., a novel ecomorph) that is expressed regardless of the environment (i.e., even if the environment changes back to the original, ancestral state).

According to one prominent version of this theory (West-Eberhard, 2003), when selection acts on quantitative genetic variation regulating the expression of an initially environmentally induced trait, it can promote the evolution of either increased or decreased plasticity through an evolutionary process known as 'genetic accommodation' (Figure 3). If the affected trait evolves decreased plasticity to the point of becoming constitutively expressed, 'genetic assimilation' occurs (sensu Waddington, 1953; Figure 3). The outcome of genetic assimilation is a novel, canalized trait (i.e., a trait that is 'fixed,' or expressed constitutively regardless of normal changes in the environment).

To illustrate this process, imagine a population that experiences a novel environment (Figure 4). Individuals in such populations often cope with stress associated with unfamiliar environments by producing new phenotypes through phenotypic plasticity (Badyaev, 2005). If such an induced trait improves fitness, then selection will favor alleles or genotypes that enhance the trait's expression (different genotypes within the same population often vary in the degree to which they respond to any particular environmental stimulus; Gupta and Lewontin, 1982). As these alleles/genotypes accumulate in the population, individuals may express the trait without the original environmental stimulus; i.e., genetic assimilation occurs. In this way, a novel canalized trait emerges from a trait that was originally environmentally induced.

With genetic accommodation/assimilation, the origin of a complex new trait need not require the evolution of new genes. Instead, selection acts on existing genes and converts a plastic trait into a canalized trait via an evolutionary adjustment in the regulation of trait expression (West-Eberhard, 2003). A plausible mechanism whereby such an adjustment could occur is through changes in gene expression (Gilbert and Epel, 2009; Renn and Schumer, 2013; see Figure 5). Recall from above that gene expression is typically environmentally sensitive, and that changes in gene expression often (possibly, always) underlie phenotypic plasticity. If selection is persistent and coarse-grained (i.e., individuals encounter only one selective environment), then induced differences in gene expression may evolve to become less environmentally responsive until they become fixed (e.g., via allelic substitutions at regulatory loci; Grishkevich and Yanai, 2013).

Although genetic accommodation can occur whether a novel trait is mutationally or environmentally induced, environmentally triggered novelties likely have greater evolutionary potential than mutationally induced ones for at least two reasons. First, changes in the environment often impact many individuals simultaneously (e.g., consider how exposure to sudden cold temperatures or a new competitor or predator can affect a population). By contrast, a new mutation initially affects only one individual (and its immediate descendants). The widespread impact of environmental change enables a newly induced trait to be tested in diverse genetic backgrounds, thereby increasing the chances that genetic accommodation will occur (see above). Second, an environmentally triggered novelty is automatically associated with a particular environmental situation – the one that induced it. Therefore, such traits are more subject to consistent selection and

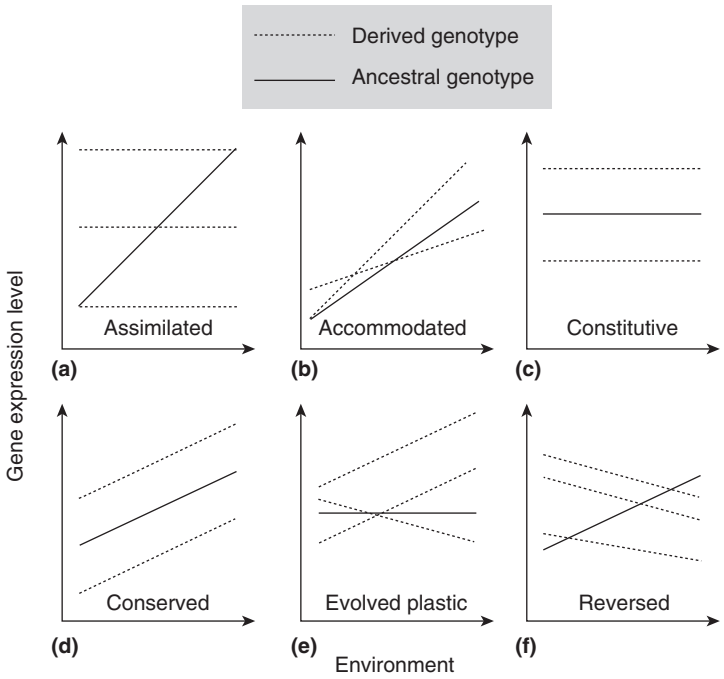

Figure 5 Alternative patterns by which gene expression might evolve. Each panel depicts the 'reaction norms' of ancestral and derived genotypes (a reaction norm is a graphical representation of the sensitivity of a group of organisms of the same genotype to some specific environmental variable). Redrawn, with permission from Renn, S.C.P., Schumer, M.E., 2013. Genetic accommodation and behavioural evolution: Insights from genomic studies. Animal Behaviour 85, 1012–1022.

directional modification than are mutationally induced novelties, for which expression is more likely to be random with respect to the environment.

Phenotypic plasticity also promotes the accumulation of 'cryptic genetic variation' – variation that is expressed only under atypical conditions (Paaby and Rockman, 2014) – that makes genetic accommodation possible. Phenotypic plasticity facilitates the build up of such variation, both because the effects of novel genetic variants are buffered by compensatory plastic responses (Moczek, 2008), and because environment-specific genes experience relaxed selection in the non-inducing environment (Lahti *et al.*, 2009; Leichty *et al.*, 2012). This accumulation of cryptic genetic variation can lead to further phenotypic novelty when it is revealed through a subsequent change in the environment or genome. Such unmasking of standing genetic variation may play an important role in fueling evolutionary changes (Moczek *et al.*, 2011).

Thus, the most important role of genetic mutations in evolution may be to contribute not so much to the origin of phenotypic novelties as to the pool of genetic variation that makes genetic accommodation possible. Or, as West-Eberhard (2003, p. 158) put it, genes may be 'followers, not necessarily leaders, in phenotypic evolution.'

Although laboratory studies have demonstrated that genetic accommodation can occur (Waddington, 1953; Rutherford and Lindquist, 1998; Suzuki and Nijhout, 2006), relatively little is known about whether this process is responsible for ecologically and evolutionarily relevant traits in natural populations (but see Schlichting and Wund, 2014). Generally, if plasticity has preceded – and facilitated – the evolution of a particular novel trait, then there should be evidence that: (1) ancestral species express the trait only conditionally; (2) novel environments uncover cryptic genetic variation; and (3) trait expression has been refined in derived species. A number of examples appear to satisfy these conditions (**Table 1**). Moreover, a recent meta-analysis has uncovered several convincing cases in which genes appear to be 'followers' in the origins of novel traits (Schwander and Leimar, 2011). Additionally, there is evidence that genetic accommodation might even play a role in speciation: there are numerous examples in which a formerly plastic trait has undergone canalization (i.e., lost its plasticity) in a particular lineage, and such shifts are typically accompanied by speciation (Schwander and Leimar, 2011). Yet, additional tests from natural populations are needed to evaluate plasticity's role (if any) in speciation.

Phenotypic Plasticity and Ecology

Phenotypic plasticity also has important implications for ecology, which, in turn, have additional evolutionary consequences. Recall from above that an organism's phenotype is shaped by its ecology. Yet, the reciprocal is also true: an organism's ecological interactions – and thus the selective regimes that it experiences – can be influenced by its developmental responses.

For instance, the individuals of many species respond adaptively to interspecific competition by facultatively modifying their resource-use traits through phenotypic plasticity (Pfennig and Pfennig, 2012). Species that can alter their phenotype in this way may persist in the face of novel

Table 1 Possible examples of genetic accommodation in natural populations of animals. For possible examples in plants, see Schlichting and Wund (2014)

Organism	*Trait(s) undergoing adaptive evolution*	*Reference(s)*
Water fleas (*Daphnia melanica*)	Pigmentation	Scoville and Pfrender (2010)
Stickleback fish (*Gasterosteus aculeatus*)	Benthic and limnetic ecomorphs	Wund *et al.* (2008)
Stickleback fish (*Gasterosteus aculeatus*)	Growth rate	Robinson (2013)
Spadefoot toads	Adult body/leg length and larval duration	Gomez-Mestre and Buchholz (2006)
Spadefoot toads (genus *Spea*)	Carnivore and omnivore ecomorphs	Ledón-Rettig *et al.* (2008); Pfennig and Martin (2010)
Australian tiger snakes (*Notechis scutatus*)	Head size	Aubret and Shine (2009)
House finches (*Carpodacus mexicanus*)	Sex determination and sex-specific resource allocation	Badyaev (2009)

competitive interactions because they can immediately (i.e., within a single generation) switch to a selectively favored phenotype. In the absence of such plasticity, species may be driven locally extinct through competitive exclusion (Pfennig and Pfennig, 2012). Thus, environmentally responsive development can reduce extinction risk, thereby influencing the composition of ecological communities (similar arguments can be made regarding the ability to respond rapidly to any sort of change in an organism's biotic or abiotic environment). Of course, populations that do not go extinct should ultimately be more likely to diversify. This may explain, at least in part, why clades in which conspicuous phenotypic plasticity (e.g., **Figure 1(c)**) has evolved are more species rich than sister clades lacking such plasticity (Pfennig and McGee, 2010).

Epigenetic Inheritance and Evolution

Lastly, research in eco-evo-devo is changing our view of inheritance, and indeed, our very definition of evolution. Because inheritance is a prerequisite for evolution to occur, clarifying how inheritance works has long been a goal of evolutionary biology (Bonduriansky and Day, 2009). Generally, most evolutionary biologists assume that inheritance occurs exclusively through alterations in the base-pair nucleotide sequence of genes; by contrast, phenotypic changes induced directly by the environment are assumed to be incapable of being inherited and to therefore play no role in mediating evolutionary change (Futuyma, 2013). Indeed, evolution is typically defined as change in a population's genes (Futuyma, 2013).

Yet, a growing body of evidence has conclusively shown that various extra- (or epi-)genetic factors that influence phenotype production – and that were initially induced directly by changes in the organism's environment – can be transmitted from one generation to the next (Jablonka and Raz, 2009). Such transmissible 'epigenetic' changes (Gilbert and Epel, 2009) thereby constitute a form of inheritance – distinct from that based on changes in DNA base-pair sequence – known as 'transgenerational epigenetic inheritance' (Jablonka and Lamb, 2010).

Transgenerational epigenetic inheritance is underlain by two main mechanisms. The first involves 'chromatin marking,' which occurs when small chemical groups are added to the DNA strand. For instance, for RNA polymerase to attach to a gene's promoter and begin transcription, the DNA strand must be unwound and untangled from the proteins that surround it (this DNA/protein complex is the 'chromatin'). The addition of a methyl group (CH_3) condenses the chromatin more tightly (Gilbert and Epel, 2009), which can prevent RNA polymerase from finding the promoter, thereby inactivating the affected gene. Importantly, the addition of a methyl group to a DNA strand can be triggered by a change in the organism's environment (Gilbert and Epel, 2009, pp. 43–46). Once a gene has been inactivated in this manner, the gene's inactivated state can be inherited when special enzymes – DNA methyltransferases – recognize a methylated sequence on the parent strand and then methylate the same region on the newly synthesized daughter strand (Gilbert and Epel, 2009).

Thus, the altered state of activation of a gene – as well as any changes to the phenotype – can thereby be transmitted across generations in the absence of any changes in base-pair nucleotide sequence of the DNA. Although the degree to which such epigenetic marks mediate the transmission of ecologically relevant traits across generations is unclear, there are some compelling examples (reviewed in Jablonka and Raz, 2009). Moreover, because chromatin marking mediates inheritance of cellular epigenetic variants (e.g., different cell types within a multicellular organism), the potential for transgenerational epigenetic inheritance is therefore present in all multicellular organisms (Maynard Smith and Szathmáry, 1995).

The second major mechanism of transgenerational epigenetic inheritance involves soma-to-soma transmission of epigenetically based variations (Jablonka and Lamb, 2010). Soma-to-soma transmission encompasses many different processes, all of which entail reconstructing the parental phenotype during somatic development in successive generations without direct involvement of the germline. Three main (non-mutually exclusive) processes mediate such soma-to-soma transmission.

First, phenotypes can be inherited via a 'maternal effect.' Maternal effects arise when a female's phenotype influences its offspring's phenotype, independent of the direct effects of the female's coding sequences on its offspring's phenotype. For instance, in many animals, larger females produce larger young, simply because they produce larger eggs, more milk, and/or have larger wombs. Subsequently, the large daughters of these large females may perpetuate the trend of producing

large offspring (Jablonka and Lamb, 2010). Likewise, females of many species differentially endow their seeds, eggs, or offspring with acquired information or materials (e.g., RNA transcripts, cytoplasm, and hormones) that can influence their offspring's phenotype (Mousseau and Fox, 1998). These effects may endure for many generations, even persisting long after the original environmental stimulus that created the maternal effect disappears (Kirkpatrick and Lande, 1989). Moreover, such maternal effects have been shown to mediate the inheritance of divergent phenotypes in natural populations, often in a manner indistinguishable from a phenotypic shift stemming from a change in DNA base-pair nucleotide sequence (e.g., Badyaev *et al.*, 2002; Pfennig and Martin, 2009).

Second, soma-to-soma transmission of epigenetic variants can occur via transmission of symbionts (smaller organisms that live inside a host organism as parasites or mutualists). Many symbionts alter their host's phenotype, and are transmitted primarily or even exclusively from parent to offspring (Werren *et al.*, 2008). Differential transmission of symbionts may mediate phenotypic divergence between populations and possibly even speciation, especially when different symbionts are incompatible within the same host (Brucker and Bordenstein, 2013).

Third, soma-to-soma transmission of epigenetic variants can occur via learning. For example, in animals, feeding (Papaj and Prokopy, 1989), mating (Crews *et al.*, 2007), and habitat (Slagsvold *et al.*, 2013) preferences can be transmitted from parent to offspring exclusively through learning. The transmission of fitness-enhancing information via learning may play an important role in both ecology and evolution by facilitating speciation and reducing extinction risk (Beltman *et al.*, 2004; Verzijden *et al.*, 2012).

Note, however, that the long-term stability of epigenetically based variations – whether mediated by chromatin marks or soma-to-soma transmission – remains uncertain. Unlike with DNA replication – where accuracy is largely insensitive to the environment – the stability of epigenetic changes depends on an organism's current environment; a change in the environment can modify, and even reverse, epigenetically based variations (Jablonka and Lamb, 2010). Yet, even if most epigenetic variants are found to last only a few generations, epigenetic inheritance mechanisms may still be important if they decrease the chances of extinction and/or increase the likelihood of genetic changes (Pfennig and Servedio, 2013).

Concluding Remarks

Eco-evo-devo is an emerging field that seeks to understand the causes and consequences of a common feature of development – its tendency to be responsive to changes in an individual's environment. Research in this field has at least three important implications for evolutionary biology. First, as described above, an understanding of the interconnectedness between an organism's environment and its developmental responses can illuminate how the environment not only selects among diverse phenotypes, but how it also creates those phenotypes in the first place (essentially, the environment can dictate both the 'survival' as well as 'arrival of the fittest'; Gilbert and Epel, 2009). Second, this phenotypic plasticity might play a critical, and often underappreciated, role in initiating evolutionary innovation and diversification. Finally, such phenotypic change induced by the environment can form the basis of an alternative inheritance system, which might – by itself – mediate evolutionary change. This shift in emphasis on environmentally initiated phenotypic change in evolution should not be seen as a threat to modern evolutionary theory (Orr, 1999); rather, it represents an opportunity to expand the theory (Pigliucci, 2007; Pfennig *et al.*, 2010).

Additional studies are required, however, especially those utilizing new model organisms (Collins *et al.*, 2007). Ideally, these new models would include related groups of species whose phylogenetic relationships are resolved; whose ecology is well known; that experience diverse ecological (and thus, selective) regimes; that display different levels of phenotypic plasticity; and that are amenable to experimental manipulation (e.g., Emlen, 2000; Ledón-Rettig and Pfennig, 2011). Using such new model organisms, greater effort is especially needed to ascertain: (1) the conditions and frequency with which phenotypic plasticity facilitates, rather than impedes, evolution; (2) the degree to which phenotypic plasticity impacts large-scale (i.e., macroevolutionary) change; and (3) the long-term stability and efficacy of epigenetically based variations in mediating evolution. Answers to these and other questions in eco-evo-devo research promise to continue to provide fresh insights into the evolutionary process.

See also: Developmental Plasticity and Phenotypic Evolution. Epigenetic Inheritance. Epigenetics and Genome Evolution. Genotype-by-Environment Interaction. Maternal Effects. Robustness and Evolvability in Molecular Evolution. Waddington's Epigenetic Landscape, History of

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