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What is Phenotypic Plasticity and Why is it Important?

Douglas W. Whitman[1] **and Anurag A. Agrawal**[2]

[1]*Department of Biological Sciences, Illinois State University, Normal, IL, USA 61790-4120, E-mail: dwwhitm@ilstu.edu*

[2]*Department of Ecology and Evolutionary Biology, Cornell University, Ithaca, New York, USA 14853, E-mail: agrawal@cornell.edu*

Abstract

Phenotypic plasticity, the capacity of a single genotype to exhibit variable phenotypes in different environments, is common in insects and is often highly adaptive. Here we review terminology, conceptual issues, and insect plasticity research, including variance partitioning, reaction norms, physiological mechanisms, adaptive value, and evolution. All plasticity is physiological, but can manifest as changes in biochemistry, physiology, morphology, behavior, or life history. Phenotypic plasticity can be passive, anticipatory, instantaneous, delayed, continuous, discrete, permanent, reversible, beneficial, harmful, adaptive or non-adaptive, and generational. Virtually any abiotic or biotic factor can serve to induce plasticity, and resulting changes vary from harmful susceptibilities to highly integrated and adaptive alternative phenotypes. Numerous physiological mechanisms accomplish plasticity, including transcription, translation, enzyme, and hormonal regulation, producing local or systemic responses. The timing, specificity, and speed of plastic responses are critical to their adaptive value. Understanding plasticity requires knowing the environment, physiological mechanisms, and fitness outcomes. Plasticity is thought to be evolutionarily favored under specific conditions, yet many theoretical predictions about benefits, costs, and selection on plasticity remain untested. The ecological consequences of plasticity range from simple environmental susceptibilities to mediating interspecific interactions, and extend to structuring of ecological communities, often through indirect effects. Phenotypic plasticity, through its ecological effects, can facilitate evolutionary change and speciation. Plasticity is important because it is an encompassing model to understand life on earth, it can increase fitness, generate novelty, and facilitate evolution, it structures ecological communities, and it has numerous practical applications. As such, all biologists should understand phenotypic plasticity.

Introduction

A young caterpillar feeds on oak flowers and develops into a stunning mimic of an oak catkin (Fig 1b.). A second caterpillar from the same egg batch feeds on leaves and becomes a twig mimic (see Chapter 4, this volume). In response to low-quality, fibrous food, a grasshopper develops larger mandibles and mandibular muscles (Thompson 1992), and another develops a larger gut (Yang and Joern 1994). A different grasshopper alters the number of chemosensilla on its antennae in response to the number of plant chemicals it encounters (Chapman and Lee 1991, Rogers and Simpson 1997). In a nearby aphid colony, females are busy adjusting the future morphology and behavior of their offspring in response to predator threats. When ant bodyguards are absent, females rapidly produce soldier offspring (Shingleton and Foster 2000), and produce winged offspring when predators invade the colony (Weisser et al. 1999). Close by, a gravid fly, unable to locate her normal host plant, deposits her eggs on a novel host. Surprisingly, the larvae survive on the new host, and chemically imprint on it before dispersing as adults. The flies subsequently orient to the novel plant to mate and oviposit, instead of their ancestral plant (Feder et al. 1994, see Chapter 18, this book). In the same tree, a caterpillar bites into a leaf. A plant sensory mechanism detects the caterpillar saliva and signals the entire plant to begin synthesis of anti-herbivore toxins and the release of volatile pheromones. The latter dissipate to neighboring plants, alerting them to the presence of herbivores, and stimulating them to synthesize their own chemical defenses. But, the plant's clever counter-ploys do not go unchallenged; in response to increasing plant toxins, the caterpillar synthesizes detoxifying gut enzymes, effectively negating the plant's chemical escalation (see Chapter 7). On the ground below, a *Drosophila*

Fig. 1 Morphological phenotypic plasticity in insects. (a, b) Discrete seasonal polyphenisms in *Nemoria arizonaria* caterpillars (fam. Geometridae). Summer brood feeds on oak leaves and resembles an oak twig. Spring brood feeds on and resembles oak catkins. Photos: E. Greene (Greene 1989). (c) Wet-season (left) and dry-season (right) *Precis octavia* (fam. Nymphalidae) butterflies, from Africa (McLeod 2007). Photos courtesy of F. Nijhout. (d) Many insects alter body color in response to rearing temperature: *Romalea microptera* grasshoppers (fam. Romaleidae) from south Florida reared at $35^{o}C$ (top) and at $25^{o}C$ (bottom). (e) Harlequin bugs, *Murgantia histrionica* (fam. Pentatomidae). Black and yellow individuals were reared at 22 and $30^{o}C$, respectively. (f) Nutrition strongly influences insect body size. *Taeniopoda eques* grasshoppers (fam. Romaleidae), from the Chihuahuan Desert in SE Arizona, showing plasticity in body size to nutrition. Males on left; females on right. Bottom four individuals from site that received ample rains and had lush vegetation; top four individuals from a site 15-km distant

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that received poor rains and had poor vegetation. In previous years, rain, vegetation, and grasshopper size patterns were reversed at these two sites (d-f: Whitman, unpubl.).

maggot, feeding inside a sun-exposed fruit, responds to near-lethal temperatures by mounting a full-fledged biochemical counter-response. Rapid transcription and translation floods the cells with protective heat-shock proteins that stabilize thermal-labile proteins, preventing death. In a nearby shaded sibling, no heat shock proteins are produced (Chapter 17). Across the meadow, a different insect is trapped on a poor-quality host. This inadequate diet profoundly alters her life history and fecundity by reducing her development and growth rates, body size (Figs. 1f, 4i), number of ovarioles, and clutch size and egg size, which, in turn, alters the life history and fecundity of her offspring (Chapter 11). A beetle larva, sensing its fungal competitor, accelerates its development (Roder et al. 2008). As fall turns to winter, the adult, exposed to short day lengths, radically switches its behavior and physiology. It stops feeding, burrows into the soil, changes color, dramatically lowers its metabolism, and fortifies its tissues with cyroprotectants, enabling survival at frigid temperatures. Its sibling, kept in long-day conditions, exhibits none of these changes and is killed by mildly cold temperatures (Chapter 16).

The above insects share a singular commonality: in each case, an individual has changed its morphology, physiology, behavior, or life history in response to changing environmental conditions. Such phenotypic plasticity is universal among living things and derived from the fact that environments vary. These environmental changes, be they temporal, spatial, abiotic, or biotic, are challenging because they can destabilize homeostasis and development, and disrupt the match between an organism's phenotype and the environment, thereby lowering fitness. Organisms counter environmental variation with their own adaptive variation of two types: between- and within-generation variation (Meyers and Bull 2002, DeWitt and Langerhans 2004). The former is mostly genetic and can result in adaptive change within a population. Between-generation variation has been the primary focus of evolutionary biologists and is based on natural selection acting on heritable variation caused by mutation, recombination, genetic drift, etc. In contrast, within-generation variation is almost always non-genetic, occurs in individuals, and is frequently adaptive, because it allows individuals to adjust to environmental variation in real time.

Interest in phenotypic plasticity has grown exponentially in the last 20 years, igniting an explosion of literature. Most of the ideas expressed in this chapter are derived from the following excellent reviews, and readers should consult these sources for a more comprehensive understanding of plasticity: Bradshaw 1965, Scheiner 1993, Nylin and Gotthard 1998, Schlichting and Pigliucci 1998, Tollrian and Harvell 1999, Agrawal 2001,

2005, Pigliucci 2001, Zera and Harshman 2001, Schlichting and Smith 2002, Nijhout 2003a, West Eberhard 2003, Benard 2004, Dewitt and Scheiner 2004, Ohgushi 2005, Emlen et al. 2007, Shingleton et al. 2007, Sultan 2007.

What is Phenotypic Plasticity?

The concept of phenotypic plasticity is deceptively simple. Numerous authors have defined phenotypic plasticity (Box 1), and, at face value, these definitions seem fairly similar. However, the devil is in the details, and we consider these details, below. For our purposes, we define phenotypic plasticity as the capacity of a single genotype to exhibit a range of phenotypes in response to variation in the environment (Fordyce 2006).

Box 1   Definitions of Phenotypic Plasticity

- Plasticity is shown by a genotype when its expression is able to be altered by environmental influence . . . it does not have any implications concerning the adaptation value of the change occurring . . . (Bradshaw 1965).
- A change in the expressed phenotype of a genotype as a function of the environment *or* when an individual's phenotype is influenced by its environment (Scheiner 1993).
- The capacity of an organism to develop any of several phenotypic states, depending on the environment; usually this capacity is supposed to be adaptive (Futuyma 1998).
- The ability of an organism to express different phenotypes depending on the environment (Agrawal 2001).
- The property of a given genotype to produce different phenotypes in response to distinct environmental conditions (Pigliucci 2001).
- Any change in an organism's characteristics in response to an environmental signal (Schlichting & Smith 2002).
- Condition-sensitive development *or* the ability of an organism to react to an environmental input with a change in form, state, movement, or rate of activity (West-Eberhard 2003).
- Environment-dependent phenotype expression *or* the environmentally sensitive production of alternative phenotypes by given genotypes (Dewitt & Scheiner 2004).
- The expression of different phenotypes in a single genotype when subjected to different environments (Ananthakrishnan & Whitman 2005).
- Variation, under environmental influence, in the phenotype associated with a genotype (Freeman & Herron 2007).
- Environmental sensitivity for a trait (Various authors).

Variance Partitioning

Phenotypic plasticity represents measureable variation, and as such can often be expressed and analyzed by Analysis of Variance (ANOVA) (Pigliucci 2001). A statistical measure of variation is variance, which quantifies the deviation of values around a mean. The variance of a phenotypic trait can be partitioned as follows:

$$V_P = V_G + V_E + V_{G \times E} + V_{\text{error}}$$

Where:

V_P = Total phenotypic variance for a trait

V_G = Genetic variance (proportion of phenotypic variation attributable to genes)

V_E = Environmental variance (proportion of variation caused by the environment)

$V_{G \times E}$ = Genotype × environment interaction (Genetic variation for phenotypic plasticity)

V_{error} = Unexplained variance, including developmental noise, measurement error, etc.

ANOVA can partition phenotypic variation into the above components. However, these terms, especially the expression of genetic variance, are often further divided into component parts (Debat and David 2001, Piersma and Drent 2003). Thus, experimental designs with some form of genetic structure (i.e., using clones, half-sibling families, multiple populations, etc.) and environmental treatments are extremely powerful for studying phenotypic plasticity. Nonetheless, genetic structure is not required for the study of plasticity. A simple design of several individuals of a species, randomly assigned to different environments, can often yield a robust estimate of plasticity. Here, V_G and $V_{G \times E}$ are unknown, but V_P can still be partitioned into what is explained by V_E (i.e., phenotypic plasticity) and all other sources of phenotypic variation. $V_{G \times E}$ is an important term because it shows that different genotypes express different plastic responses. Such genetic variance in plasticity allows plasticity to evolve.

Graphic Representation: Reaction Norms

Phenotypic plasticity can be visualized by the use of reaction norms, which plot values for a specific phenotypic trait across two or more environments or treatments (Schlichting and Pigliucci 1998, Sarkar 2004). Figure 2 shows hypothetical reaction norms, for a specific trait (in this case, let's say horn length), for five genotypes in a population. Each genotype expresses a

different mean value for horn length in Environment 1 (V_G). However, when subjected to a new environment, most genotypes alter their horn length. In this case, when comparing the grand means (the triangles) in each environment, we see that horn length generally increases in Environment 2 (V_E). However, each genotype exhibits a different reaction norm (i.e., a different response to environment, or different slopes in Figure 2). Genotype 4 shows no plasticity for this particular trait: mean horn length remains the same in both environments. In contrast, Genotype 3 shows extreme phenotypic plasticity for mean horn length, growing long horns in Environment 2. Alternatively, for Genotype 1, mean horn length decreases in Environment 2. The fact that each Genotype shows a different response (non-parallel reaction norms) represents genotype × environment interaction ($V_{G \times E}$), indicating genetic variation in plasticity itself, upon which natural selection can act to alter the shape and variance of the species' reaction norm. Figure 3 shows real reaction norms from real animals; additional examples can be found throughout this book. Note that when multiple environments or continuous environmental gradients are included, reaction norms may be highly curvilinear or discontinuous (Roff 1996, Emlen and Nijhout 2000, David et al. 2004). One problem with both variance partitioning and reaction norms is that they do not explain the evolution, underlying mechanisms, or consequence of phenotypic plasticity
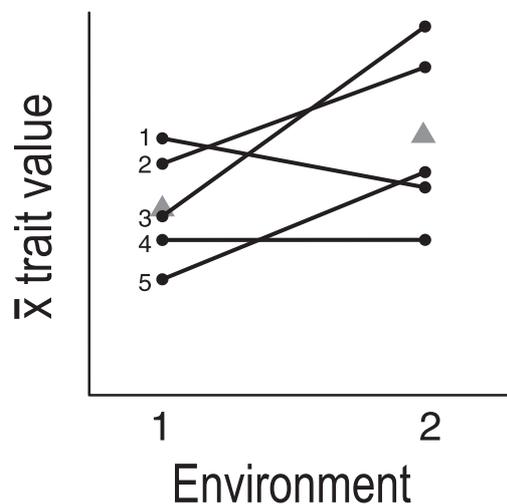

Fig. 2 Hypothetical reaction norms for five genotypes in one population. Triangles show mean population trait value at two different environments. See text for explanation.
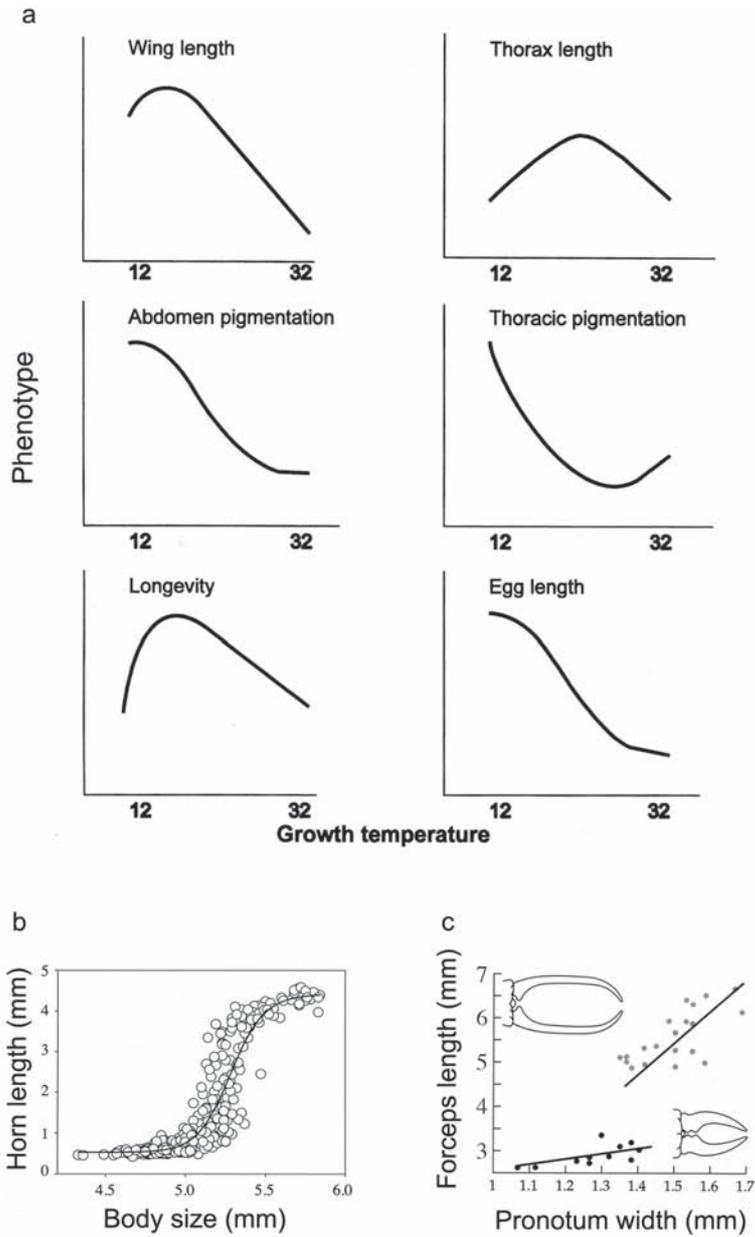
Fig. 3 Reaction norms from insects, showing the great diversity in phenotypic plasticity response. (a) Reaction norms for various traits in *Drosophila* in response to growth temperature (David et al. 2004; by permission of Oxford University Press, Inc.). (b) Sigmoid allometry for

— they simply depict their variable and heritable natures (Nijhout 2003a, Frankino and Raff 2004).

Characterizing Phenotypic Plasticity

Scientists agree that phenotypic plasticity concerns environmentally induced changes to phenotypes (see Box 1). Also, most consider discrete morphological polyphenisms (Figs. 1a-d, 4a,b,k,l, 5) as good examples of this concept. However, environments can influence phenotypes in diverse and complicated ways, and it is among these varied effects that opinions about plasticity begin to diverge. Below, we discuss some of the complexities and controversies surrounding phenotypic plasticity.

What Kinds of Traits Exhibit Phenotypic Plasticity?

Virtually any trait can show phenotypic plasticity. The concept was first applied to morphological traits (Woltereck 1909, Schlichting and Pigliucci 1998), and some authors still link phenotypic plasticity to morphology. However, it is clear that organisms can also alter biochemistry, physiology, behavior, and life history in response to the environment (B. Agarwala 2007, see Chapters 5, 11, 12, this book), and such changes are now generally accepted as phenotypic plasticity. Hence, such diverse phenomena as heat shock reaction (Chapter 17), acclimatizations (Chapter 16), diapause, immunology, learning and imprinting (Chapter 18), host-plant switching (Chapter 18), enzyme induction, predator-induced defense (Chapters 7, 8), maternal effects (Chapter 19), homeostasis (Chapter 15), mate choice and hybridization (Pfennig 2007), dispersal (Chapter 14), environmentally induced transcription and translation, and general stress responses are now often analyzed under the rubric of phenotypic plasticity. However, because virtually all phenotypic traits result from underlying biochemical-physiological processes, virtually all phenotypic plasticity represents (or results from) altered physiology.

Many authors view plasticity as a developmental process (Cronk 2005), and even ontogeny can be considered a continuous reaction norm of the

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horn length in male *Onthophagus taurus* beetles in response to body size, which is largely determined by larval nutrition (after Moczek et al. 2004, see Chpt.3). (c) Allometry for nutrition-influenced forceps length in *Eluanon bipartitus* male earwigs, showing two discrete morphs with no intermediaries (Tompkins & Simmons 1996, Schlichting & Piglucci 1998, Tomkins 1999).

entire genotype (Schlichting and Smith 2002), with time as the "environmental" variable. Others link phenotypic plasticity to environmental induction of gene or allele expression (e.g., Czesak et al. 2006). However, these all represent biochemical-physiological processes. Development is particularly susceptible to deviating perturbations, with manifold downstream consequences, and this is why plasticity theory is closely tied to development (see Chapters 3, 4, 12, 13, 14).

Organisms are complex networks of interacting systems. As such, altered environments induce not single, but manifold changes, altering suites of independent and interconnected traits that range across multiple levels of biological organization (Relyea 2004a, Gorur et al. 2005, de Kroon et al. 2005, Chapters 2, 14). An example is locust polyphenism, in which solitary and gregarious phenotypes differ in behavior, morphology, food selection, body color, gene expression, neuro-, endocrine, and nutritional physiology, metabolism, immune responses, pheromone production, reproduction, and longevity (Simpson et al. 2005, Song 2005, see Chapters 5, 6, this book). A developmental evolutionary challenge is the integration of numerous plasticities into a functioning individual of high fitness (Pigliucci and Preston 2004, Shingleton et al. 2007).

Fig. 4   Morphological phenotypic plasticity in insects. (a) Soldier vs. worker in *Reticulitermes flavipes* termites (Klausnitzer 1987; Courtesy: Edition Leipzig). (b) Non-estivating (left) and estivating (right) nymphs of *Periphyllus granulatus* aphids (Hille Ris Lambers 1966. Reprinted, with permission, from *Annual Review of Entomology*, Vol. 11 (1966) by Annual Reviews, www.anualreviews.org. (c) Soldier (top) and non-soldier *Pseudoregma alexanderi* aphids. Reprinted from: Minks & Harrewijn 1987, courtesy of Elseiver Ltd. See Stern & Foster 1996, Shibau et al. 2003, 2004). (d) Oedymerous (left) and gynaecoid (right) male *Tiarothrips subramanii* thrips (Ananthakrishnan 2005). (e) Small and large male *Phoxothrips pugilator* thrips (Haga & Okajima 1975, Mound 2005). (f) Polyphenism in *Forficula auricularia* male earwig cerci (Carpenter 1899, Tomkins & Simmons 1996, Tomkins 1999). (g) Horned and hornless *Onthophagus taurus* dung beetles. (h) Polyphenism in male *Cladognathus giraffe* stag beetles (Otte & Stayman 1979). (i) Male *Brentus anchorago* weevils from Costa Rica exhibit enormous plasticity in body length (7 to 49 mm) (Johnson 1982; Courtesy of John Wiley & Sons Ltd). (j) Heads of small and large male *Mecynothrips kraussi* thrips (Palmer & Mound 1978). (k) Phenotypic plasticity to host in a trichogrammatid egg parasitoid, *Trichogramma semblidis*: small winged male (left) from moth eggs. Large wingless male (right) from alder fly eggs (Salt 1937). (l) Dispersing and non-dispersing forms of male *Pseudidarnes minerva* fig wasps (Cook et al. 1997; Courtesy of Royal Society of London. See also Pienaar & Greef 2003a,b).
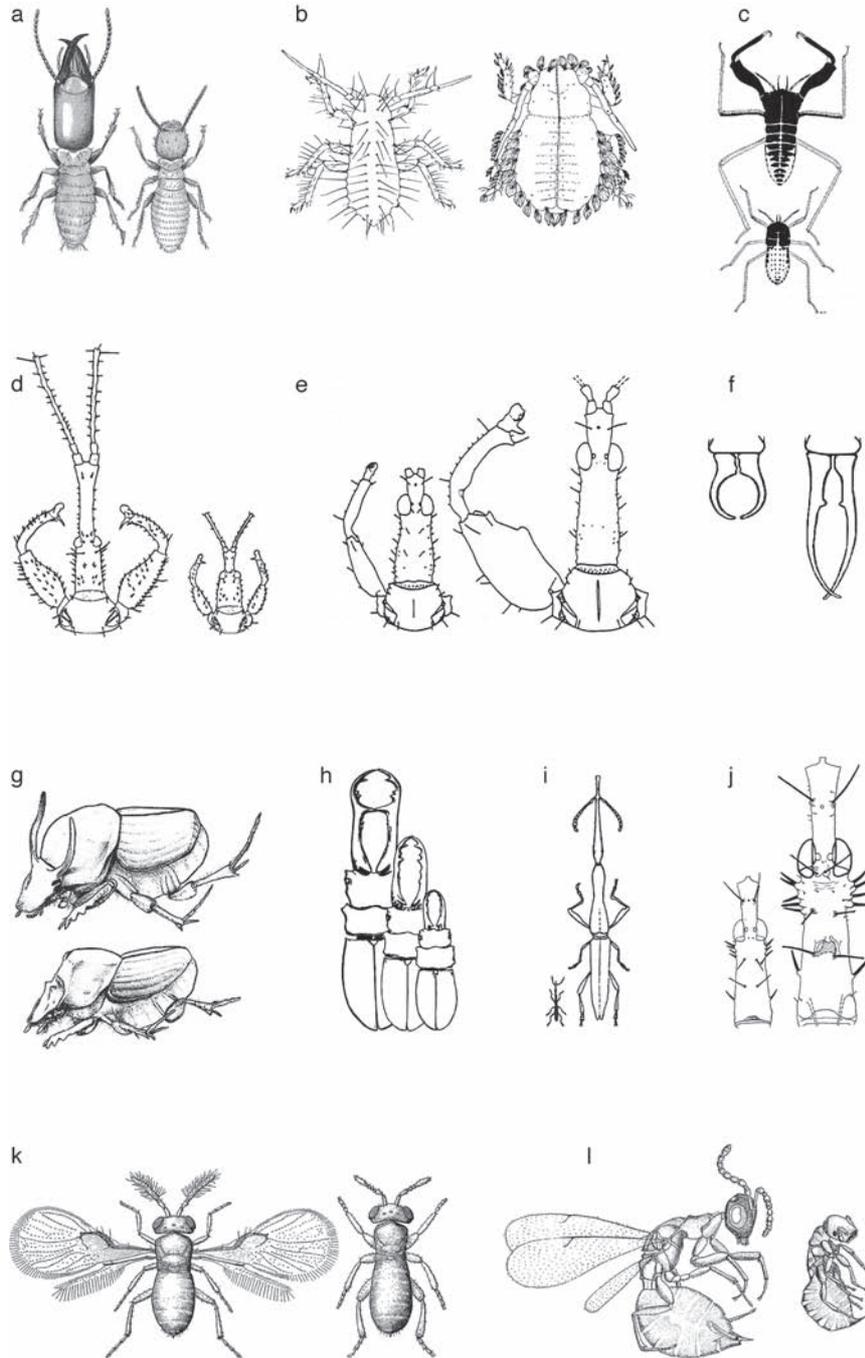

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Fig. 4

Phenotypic Modulation vs. Developmental Conversions

Phenotypic plasticities range from graded, continuous responses (phenotypic modulation), to discrete switches in phenotype with no intermediate forms (developmental conversion or threshold traits) (Roff 1996, Windig et al. 2004) (see Glossary). The former are sometimes assumed to be non-adaptive, reversible "susceptibilities," and produce continuous linear or curvilinear reaction norms. Examples include nutrition and temperature effects on growth rate and body size (Figs. 1f, 3a). Developmental conversions are sometimes assumed to be beneficial, permanent adaptations, and produce discontinuous or sigmoid reaction norms. Examples include discrete polyphenisms (Figs. 1a-c, 3c, 4a, b, k, l, 5). Most plasticities fall somewhere between these extremes. Importantly, a plastic trait may be erroneously designated a developmental conversion due to improper sampling or failure to expose experimental organisms to intermediate environments (Fig. 7e) (Nijhout 2003a). In addition, a continuous, graded process may underlie a discontinuous plasticity, such as when a trait responds to a gradual change in an underlying hormone concentration, via a threshold mechanism (Roff 1996, Nijhout, 2003a). Note that developmental conversions can be alternatives (such as in social castes) or sequential, as in sequential sex change (Munday et al. 2006).

Cues

Phenotypic plasticity can be initiated by either environmental stimuli or cues. The former are often environmental factors such as temperature or oxygen level that directly disrupt homeostasis or development in non-adaptive ways. In contrast, organisms can evolve mechanisms to sense and adaptively respond to certain cues that predict environmental change (Nijhout 2003a). Hence, cues are generally considered to be specific environmental signals that predict environmental change, and induce adaptive plasticities. Cues tend to be non-harmful stimuli (i.e., photoperiod or a predator-released chemical) that do not harm the individual directly, whereas stimuli, themselves, are often harmful selective agents (toxin, high temperature). However, the division between these two is blurred, and the same environmental factor, such as temperature, can simultaneously initiate a highly adaptive plastic response and harmful physiological disruption. In general, organisms should evolve mechanisms to detect and respond to environmental stimuli or signals that accurately predict future environmental conditions. Hence, stress factors and correlated predictive signals should evolve into cues.
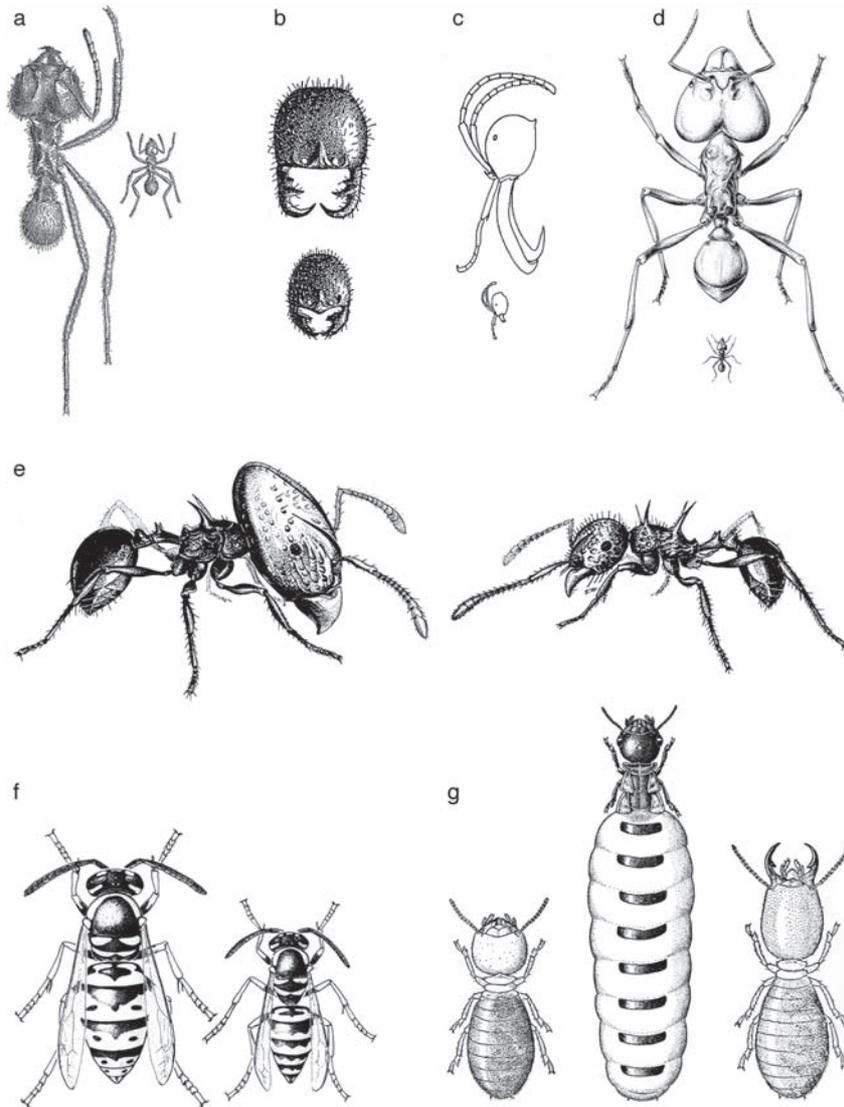
Fig. 5   (a) Soldier (left) and small worker caste (right) in *Atta texana* leafcutter ants (Wheeler 1910). (b) Heads of soldier vs. small worker castes in *Cheliomyrmex nortoni* driver ants (Wheeler 1910). a and b courtesy Columbia University Press. (c) Heads of large soldier (top) and small worker (bottom) of *Eciton burchelli* army ants (after Schneirla & Topoff 1971). (d) Caste polyphenism in *Atta laevigata* leafcutter ants. (e) Minor and major workers of an *Acanthomyrmex* species from the Celebes (d & e drawn by Turid Hölldobler; Oster & Wilson 1978); courtesy of Princeton University Press. (f) *Vespula maculifrons* queen and worker illustrated by S. Landry (Evans & West-Eberhard 1970). Courtesy University Michigan Press. (g) Worker (left) queen (middle) and soldier (right) of *Amitermes hastatus* (after Skaife 1954).

Both stimuli and cues can originate internally or externally. For example, initial hatchling size, growth rates, nutrient titers, or pathogen presence may serve as internal cues that determine alternative developmental outcomes (Nijhout 2003a,b, Mirth and Riddiford 2007, Shingleton et al. 2007). Virtually any factor can serve as a stimulus or cue to initiate a plastic response, and can be received via any sensory modality [chemical, visual, thermal, mechanical (tactile, acoustic), electrical, etc.] (see other chapters, this book). In *Diacamma* ants, queens induce young adults to become workers by chewing off their vestigial wings (Peeters and Higashi 1986, Baratte et al. 2006). Water force cues development rate in stonefly nymphs (Franken et al. 2008) and penis length in barnacles (Neufeld and Palmer 2008).

Specific vs. General Plasticities

Some plastic responses are highly specific in either requisite stimuli or response. For example, some plants possess receptor proteins that detect only their most common natural enemy (Zhao et al. 2005). Such specificity is seen in corn plants that increase defense in response to saliva from young, but not old armyworm caterpillars, perhaps because the plastic defense is only effective against young caterpillars (Takabayahshi et al. 1995). Elm trees produce volatiles attractive to egg parasitoids, in response to oviposition by its primary beetle herbivore, but not to beetle feeding (Meiners and Hilker 2000). Following fires, some grasshoppers will respond to altered light quality by adaptively changing their body color to black (see Uvarov 1966). Other grasshopper species fail to respond to light, but change color specifically in response to temperature, humidity, food, or crowding, or to some combination of these cues (Rowell 1971, Tanaka 2004, Chapters 5, 6).

Other elicitors and responses are more general, such as temperature (Chapters 12, 16, 17) and nutrition (Chapters 3, 10, 11, 19) which can influence nearly every aspect of an animal's phenotype and ecology. In some aphids, alate production is induced by any combination of photoperiod, crowding, nutrition, or presence of natural enemies (B. Agrawal 2008). Learning is a general form of plasticity that can respond to manifold environmental stimuli, and produce a great variety of plastic responses (Kukas 2004, Chapter 18). Likewise, growth and development rates are plastic to innumerable environmental factors (Chapter 10). Complex plasticities, such as locust polyphenism and life history plasticities, represent composites of numerous underlying plastic traits (Song 2005). The species characteristics and environmental factors that favor the evolution of general vs. specific cues and responses, and the underlying physiological and ecological constraints that shape these responses are currently unknown.

Adaptive Plasticity

Many examples of phenotypic plasticity are clearly adaptive (i.e., beneficial as the result of past selection), such as some immune responses, antipredator defenses, acclimatizations, diapause, life-history shifts, dispersals, etc. (West-Eberhard 2003, Lyytinen et al. 2004, Schmid-Hempel 2005). Other plasticities are non-adaptive. These include many susceptibilities to abiotic factors, and manipulations of hosts by parasites and pathogens (Hurd and Lane 1998, Roy et al. 2006, Kenyon and Hunter 2007, Poinar and Yanoviak 2008). For example, some leaf miners and gall makers induce maladaptive resource allocations and leaf retention in host plants (Prichard and James 1984, Oishi and Sato 2007). However, the environment can influence phenotypes in complex ways, and it is often difficult to determine whether or not altered phenotypes are beneficial or adaptive (van Kleunen and Fischer 2005, Pigliucci 2005, see Chapters 7, 10). Plasticities are under conflicting selective pressures (Sih 2004) and carry numerous costs and tradeoffs (DeWitt et al. 1998, Fordyce 2001, Chapters 3, 7, 10, 11, 12, 14), and some have argued that it is nearly impossible to ever know their total cost/benefit ratios. First is that a great many traits may be altered by a single environmental factor, and not all of these changes may be recognized or studied, including their numerous and complex physiological and environmental interactions and consequences (Relyea 2004a, Agrawal 2005). A specific altered trait may be highly beneficial in one context, but overwhelmingly detrimental in another. For example, plastic production of large spines or heavy armor in a prey (Fig. 6b) in response to the presence of predators may aid antipredator defense, but reduce feeding, migration, mating, fecundity, etc. (Roff 1996). Hence, the benefit of any phenotype is relative to a specific time and place and presence or absence of interacting individuals (Nykänen and Koricheva 2004, Thompson 2005). To understand adaptive plasticity, one must consider benefits and costs of plastic phenotypes in several environments.

Genetic and environmental correlations are themselves plastic to the environment (Piggliucci 2005). A particular plastic response may be highly advantageous in one season and detrimental in the the next. Indeed, a specific plastic response might be evolutionarily favored, and thus maintained in a population even if its expression produces great fitness benefits only once every 10 years; i.e., uncommon, periodic events may drive some evolution. In most years, a researcher would have little chance of observing an uncommon, but powerful selective event (e.g., Stephen 2005). Also, cost/benefit analysis should continue into the next generation, because of parental effects (Agrawal 2001, Mondor et al. 2005). Most cost/
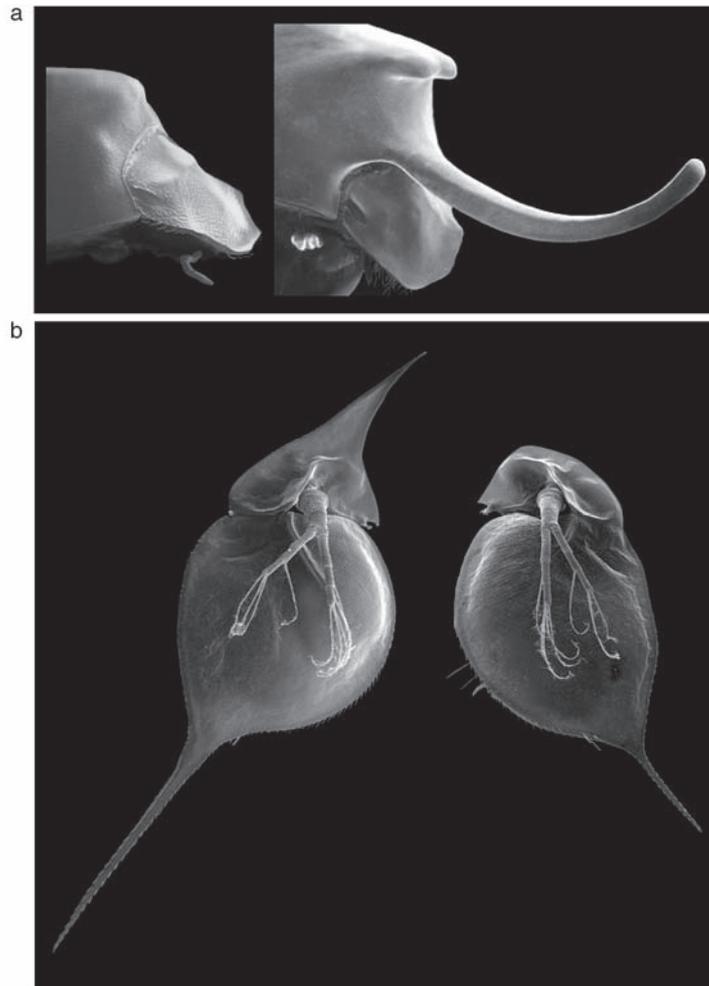
Fig. 6 (a) Horn polyphenism in *Onthophagus nigriventris*. Courtesy of D. Emlen. See Emlen et al. 2006, 2007. (b) Predator-induced plasticity in *Daphnia lumholtzi*. Left individual was exposed to fish-predator chemicals, right individual was not. The long spines reduce predation (Agrawal 2001).

benefit studies are conducted in the laboratory, greenhouse, or outdoor cages, and may not accurately reflect the realities of nature. Finally, "adaptive" implies past selection, but a population's history is often opaque (Doughty and Reznick 2004). Hence, it is difficult to know the adaptive value of phenotypic plasticity, and, for that reason, "adaptiveness" cannot

be the criterion for judging if an environmentally altered trait represents plasticity (van Klunen and Fischer 2005).

There is another reason for not restricting phenotypic plasticity to only "adaptive" traits. This is because, whether adaptive or not, all environment-induced changes to phenotypes are similar in that they place those individuals into a different selective regime, with potential fitness and evolutionary consequences. Indeed, the evolution of many adaptive plasticities, such as diapause, alternative morphologies, mating and life history strategies in small individuals, and even sociality, may have been stimulated by detrimental plastic responses to harmful factors such as low temperature or poor nutrition (West-Eberhard 2003, Emlen et al. 2006, Chapter 3).

Of course, we should continue to test the Beneficial Plasticity Hypothesis, and to evaluate both the specific and overall value of plasticity (Wilson and Franklin 2002, Doughty and Reznick 2004, van Kleunen and Fischer 2005, Chapter 10). Two features that often imply adaptation are anticipatory and active plasticities (see below).

Anticipatory vs. Responsive Phenotypic Plasticity

Some plastic responses are anticipatory, in that individuals initiate phenotypic change before the appearance of a harmful (or beneficial) environmental factor. Examples include diapause induction before the onset of winter, and detoxification induction in caterpillars. Some plants exhibit defense plasticity whereby caterpillar feeding induces the production of the plant hormones jasmonate and salicylate, which, in turn, triggers synthesis of anti-herbivore toxins (Chapter 7). Amazingly, *Helicoverpa zea* caterpillars have deciphered the plant's chemical signaling, and apparently monitor the plant's hormone concentration, which allows them to preempt poisoning. Consumption of the plant hormones activates four genes in the caterpillar that code for cytochrome P450 detoxifying enzymes, preparing it for the oncoming plant defensive onslaught (Li et al. 2002). Other plastic responses are non-anticipatory and are only triggered after the appearance of the new environment. Anticipatory and responsive plasticities are sometimes termed cued plasticity and direct plasticity, respectively (West-Eberhard 2003).

Because non-anticipatory plasticity may allow damage before the individual has a chance to change, we would expect direct plasticity to evolve into cued (anticipatory) plasticity, when possible. Likewise, if a particular trait's differential expression is strongly associated with fitness, we would expect organisms to evolve to respond to multiple predictive cues, as in the

case of aphids, which produce winged, dispersing phenotypes in response to photoperiod, crowding, nutrition, and densities of both natural enemies and ant body guards (B. Agarwala 2007). A requirement for anticipatory plasticity is that the cue must reliably predict the environmental change (Karban et al. 1999). Consistent abiotic cues of seasonal change (i.e., photoperiod) are perhaps the most reliable cues favoring the evolution of anticipatory plasticity (Bradshaw and Holzapfel 2007).

Active vs. Passive Phenotypic Plasticity and Susceptibilities

Many environment-induced phenotypic changes are active in that the response involves multiple regulatory genes and processes acting at different hierarchies to produce a complex, coordinated change. Good examples are discussed throughout this book, and include locust polyphenisms (Chapter 5) and environmentally induced diapause, which are plastic, highly integrated responses, involving behavior, physiology, morphology, and life history, regulated by specific genes and feedback mechanisms, and complex coordinated physiological-endocrine processes. As previously noted, diapause plasticity is also often anticipatory, in that the insect responds to environmental cues that predict future stressful environmental changes. Active, anticipatory phenotypic plasticities provide strong circumstantial support for adaptive plasticity.

In contrast to active plasticity, other environmentally induced phenotypic alterations appear to be simple susceptibilities to physical or chemical environmental stresses. Toxins, poor nutrition, and extreme temperatures, pH, O_2 levels, and osmolarities can directly alter chemical, enzymatic, cellular, and developmental process, producing passive (not regulated by the organism) changes to the phenotype. Small size resulting from poor nutrition is perhaps the classic example of passive plasticity. Nonetheless, most forms of plasticity likely contain active and passive components, and distinguishing them can be difficult. Even for poor nutrition-induced small size, one could make the argument that smaller individuals are more fit than larger individuals, given the current environment. Active and passive plasticity can act simultaneously on the same trait in the same individual, and can be in similar or different directions (van Kleunen and Fischer 2005). Both types represent phenotypic plasticity.

Period of Responsiveness

Species vary greatly as to when in their development they can respond to environmental change. Some species remain responsive throughout much

of their lives. In others, developmental processes create specific windows when plasticity is possible. This is particularly true in arthropods because of their discrete life stages (i.e., metamorphosis) and their external skeleton, which is not amenable to change after scleritization (Frankino and Raff 2004). Hence, for insects, phenotypic plasticity in external morphology must be initiated before molting (Shingleton et al. 2007). Many species have evolved precise temporal windows of responsiveness, and if they do not receive the appropriate environmental stimuli during that critical period, plasticity does not occur. Examples are some butterfly polyphenisms (Nijhout 1991, 2003a) and insects that imprint on their host or habitat immediately following adult eclosion (Davis and Stamps 2004, Chapter 18).

Speed of Induction

If one includes behavior (Sih 2004) and transgenerational plasticity (Mousseau and Dingle 1991, Mousseau and Fox 1998), then the speed of plastic responses ranges from instantaneous to generational. Phenotypic change should match environmental change, and too long of a lag time can be maladaptive. Thus, a key is again the match between the response, no matter what the time scale, to the environment experienced: rapidly changing environments should select for rapid plastic response, and slowly changing environments for graded or slowed responses. The former would require behavioral or physiological phenotypic plasticity, and the latter could be met with slower acting developmental plasticity, including altered morphologies or life histories.

Reversibility

Plastic traits vary in their permanency. In general, behavioral and physiological traits are rapidly reversible within individuals, whereas morphology and life history can be permanent. A great many traits fall somewhere in-between. It is difficult to shed morphologies, but even more so in insects because of their hardened exoskeleton, and because they are relatively short lived, and thus have little time or need to reverse phenotypes. However, some insect morphological plasticities are reversible. Adult Thysanura and krill (Crustaceae) molt to a smaller size in response to poor nutrition (Marinovic and Mangel 1999, Piersma and Drent 2003). *Kosciuscola tristis* grasshoppers can repeatedly alter their body color in as little as 1 hr (Key and Day 1954a,b). Conversely, induced plasticities in plants and humans can last for years (Tollrian and Harvell 1999), or across generations (Mousseau and Fox 1998, Agrawal et al. 1999, Bateson et al. 2004).

Physiological Mechanisms underlying Phenotypic Plasticity

Adaptive phenotypic plasticity is accomplished via a vast diversity of mechanisms, involving virtually all physiological levels and systems. Detailed understanding of biochemical pathways and mechanisms exists for pathogen-induced plasticity in vertebrates and plants (Frost 1999, Defranco 2007, Chapter 7), beetle horns (Emlen et al. 2007, Chapter 3), butterfly polyphenisms (Chapter 9), body size and allometry (Emlen and Allen 2004, Shingleton et al. 2007, Chapters 10, 13), wing polyphenisms (Chapter 14), some acclimations (Chapter 16), stress proteins (Chapter 17), and social castes (Page and Amdam 2007). For adaptive, coordinated phenotypic plasticity, the process involves cue recognition, stimulus transduction, and complex effector systems (Schlichting and Smith 2002, Windig et al. 2004). Cues can originate from in- or outside the individual. In some cases environmental cues are specific and are detected by specialized sense organs or mechanisms designed primarily for that purpose. In other cases, eliciting cues are more generic and are received by the general sense organs, such as eyes or mechanoreceptors. For example, tactile stimulation of sensory hairs on the legs of locusts triggers behavioral phase change in response to high density (Chapter 5). Translated signals may be sent to specific tissues and used immediately, or stored for later induction. Phenotype alteration can be accomplished from a single, unchanging genome via any combination of transcription, translation, enzyme, hormone, and morphogen regulation, morphogenesis, apoptosis, and neural control, with appropriate regulation and feedbacks between subcomponents of the overall process (Miura 2005, Amdam 2007, Emlen et al. 2007, Wolschin and Amdam 2007, Zhou et al. 2007, Chapter 7). Between cue reception and production of the ultimate phenotype, may lie dozens of steps, influenced by hundreds of genes and untold environmental/ physiological factors. This, in part, is what makes understanding of both genetic and physiological control of plasticity so difficult. In insects, environmental factors can directly turn on or off genes (Ellers et al. 2008, Chapters 7, 16, 17) or hormones (Emlen and Nijhout 2000), and hormones induce differential gene expression and development (Nijhout 1994, Evans and Wheeler 1999). Hormones lie at the base of virtually all insect developmental conversions (discrete polyphenisms) (Nijhout 1994, 1999, 2003a, Chapters 9, 13, 14), and small evolutionary changes in thresholds or timing of hormone release or sensitivity periods of specific tissues produce different reaction norms in different taxa (Emlen et al. 2007, Shingleton et al. 2007, Chapters 13, 14). In insects, parts of the endocrine and nervous

systems are one-and-the-same, which probably aids transduction of environmental cues into physiological responses (Nijhout 2003a).

Selection may act anywhere along this chain. However, reaction norm evolution is often accomplished by altering timing of physiological mechanisms that control developmental switches (Moczek and Nijhout 2003, Chapter 13). Brief and disparate sensitivity periods for different tissues, such as various imaginal disks, allow independent, compartmentalized regulation and evolution of traits, fostering great diversity of plastic responses (Emlen et al. 2007, Chapter 13).

Inability to elucidate the physiological mechanisms underlying phenotypic plasticity greatly hampered past plasticity research, and a complete understanding of plasticity will require knowing its physiology (Ricklefs and Wiekelski 2002, Frankino and Raff 2004, Lessells 2008, Chapters 11, 13, 14). However, multiple approaches for physiological understanding are now available, and modern molecular tools are stimulating rapid progress (Frankino and Raff 2004, Cossins et al. 2006, Emlen et al. 2006, Shiu and Borevitz 2008). For example, microarrays provide for the simultaneous monitoring of the expression of thousands of genes during induction and expression of phenotypic plasticity, and, when coupled with knockouts and other technologies, will allow identification of the specific genes and pathways responsible for adaptive responses.

In social insects, caste determination in different species ranges from environmental to genetic (O'Donnell 1998, Miura 2004, 2005, Hayashi et al. 2007, Hunt 2007, Whitfield 2007). Caste ratios are often determined by positive and negative feedback mechanisms, whereby increasing numbers of one caste feed back to reduce production of that caste, often accomplished via pheromones or nutrition (Shibao et al. 2004).

Physiological Homeostasis is Phenotypic Plasticity

Rapid, short-term physiological homeostasis such as regulation of blood pH and osmolarity represents phenotypic plasticity. Somewhat counter intuitively, homeostasis is derived from monitoring internal and external conditions, and manipulating physiology, i.e, keeping some aspect of the phenotype constant by altering enzyme activity or other physiological or behavioral parameters, in response to a varying environment (Chapters 13, 15). Some traditional homeostatic mechanisms and phenotypic plasticities share similar physiological mechanisms (Chapter 16). Physiological changes, be they rapid and short-term, or delayed and long-term, represent altered phenotypes to altered environments, and, as such, have the potential

to produce the same evolutionary effect – increased fitness for those genotypes that can show the beneficial plasticity. Hence, phenotypic plasticity can not be defined by velocity and reversibility of responses.

Phenotypic Plasticity vs. Canalization

Phenotypic plasticity is often considered the opposite of canalization. However, reaction norms can be canalized (Scheiner 1993). In addition, to hold one trait constant in the face of a changing environment often requires change (plasticity) in another trait. For example, some insects exhibit canalized egg size, and when confronted with poor nutrition or end of season, such insects maintain egg size, but express plasticity in clutch size or oocyte development rates (Chapter 11). In other species, clutch size or oocyte development may be canalized (Stearns 1992, Nylin and Gotthard 1998, Fox and Czesak 2000). Given trade-offs, and that particular traits can evolve to be plastic or canalized, the evolutionary outcome is presumably based on the relative advantages of different strategies in different habitats. Furthermore, what at first may appear to be a non-adaptive passive response (for example, lowered clutch size under poor nutrition), may in fact be an evolved plastic response to maintain egg size, oocyte development rate, or female survival. As mentioned above, physiological homeostasis also requires an underlying plasticity. As such, canalizations in physiology, life-history, and development are often accomplished via phenotypic plasticity.

Why is Phenotypic Plasticity Important?

Phenotypic plasticity is important because it expands the existing "genocentric" evolutionary theory, producing an encompassing paradigm to explain life on earth. Plasticity was once considered "noise" but is now widely recognized as potentially adaptive under a wide array of circumstances. As with any major shift in scientific thinking, phenotypic plasticity engenders new ideas, causing us to ask new questions and test hypotheses that would not otherwise be examined, leading us to productive new scientific insights.

Phenotypic plasticity is a counterbalance to mutation-driven evolution — It is not surprising that during the first half of the 20th Century, scientists, flushed with excitement about Mendelian genetics, viewed evolution primarily as a mutational process. However, this bias largely ignored an important reality of evolution – that natural selection selects not among genotypes, but among

phenotypes. Thus, the phenotype, and variation among phenotypes, plays a major role in evolution. And, because the environment in which an individual develops determines its phenotype, the environment also assumes a greater role in evolution, and may, in fact, produce more viable phenotypic variation than do mutations (West-Eberhard 2003, 2005). This is because mutations are not only rare, but usually deleterious. In contrast, a single environmental factor may alter the phenotypes of an entire population, providing natural selection with access to perhaps thousands of environmentally altered individuals, as opposed to a single mutant individual. In addition, mutations generally arise randomly with no correlation to specific environments, whereas new environmentally induced phenotypes are both directional and highly correlated with the specific new environment, allowing new environments to immediately produce and select among new phenotypes (Badyaev 2005). Altered environments may influence a diversity of traits that are not genetically linked, and hence may rearrange phenotypes in novel ways unavailable to single mutations. Unlike most mutations, a developmental rearrangement is likely to include both the altered trait and its background regulation (West-Eberhard 2005). And, because the inducing environmental factor may recur year after year, the new phenotype will recur often. Recurrence of a novel phenotype among large numbers of individuals that differ in numerous genetic, phenotypic, and environmental characteristics provides a fertile substrate for selection to act. Indeed, selection cannot act on a trait, if that trait is not exposed (i.e., the trait must be expressed in the phenotype). By producing novel combinations of phenotypic traits, the environment creates new raw products for selection. This process is believed to lead to adaptive phenotypic plasticity that we see today, and even to the generation of new species (West-Eberhard 2003, Schlichting 2004, Fordyce 2006, but see deJong 2005).

Under traditional evolutionary theory, the environment acts after phenotypic variation is produced, and plays a *single* role: selecting among genetically produced variation. With phenotypic plasticity, the environment plays a *dual* role in evolution: it both creates phenotypic variation and selects among that variation. This is a major change in how we view evolution. As such, environmentally induced phenotypic variation comes to assume a more important (perhaps dominant) position in evolutionary theory (West-Eberhard 2003). Similarly, theories of how organisms adapt to environmental heterogeneity previously emphasized between-generation adaptation by populations. In contrast, phenotypic plasticity emphasizes how individuals adapt within their lifetimes. Merging within- and between-

generation, individual and population adaptation produces a more comprehensive theoretical framework of adaptive variation to environmental heterogeneity (Pigliucci 2001, Meyers and Bull 2002, West-Eberhard 2003), and may contribute to a new grand unifying theory of biology (Pigliucci 2007).

Including phenotypic plasticity produces a better model — As suggested above, the inclusion of phenotypic plasticity can result in a better model than mutation-allelic substitution alone in explaining the production of organismal diversity. For example, the initial evolution of warning color (aposematism), starting as a rare mutation is problematic because conspicuous prey should be quickly found and removed by predators (Lindström et al. 2001). In contrast, evolution of aposematism is easily explained by phenotypic plasticity (Sword 2002). Likewise, for development, phenotypic plasticity explains the evolution of allometry and exaggerated morphologies (Emlen and Nijhout 2000, Shingleton et al. 2007). For physiology, phenotypic plasticity explains adaptive, beneficial plasticities such as acclimation (Chapter 16), and response to exercise (Swallow et al. 2005), quite well. In ecology, it aids our understanding of life-history variation (Beckerman et al. 2002), population dynamics (Haukioja 1990, Gardner and Agrawal 2002), community structure (Werner and Peacor 2003, Agrawal 2005), and modeling of ecological and evolutionary processes (DeAngelis and Mooij 2005). Phenotypic plasticity also helps explain some sexual selection (Qvarnström and Price 2001), alternative mating strategies (Pfennig 2007, Chapter 3), and evolution of sociality (West-Eberhard 2003, Page and Amdam 2007).

Phenotypic plasticity elevates the importance of stress — Viewing evolution through environment-induced phenotypic plasticity elevates stress as a major ecological and evolutionary concept (Badyaev 2005, Roelofs et al. 2007). Environmentally induced stress is a constant reality for most individuals. How do organisms respond to stress and what are the physiological, ecological, and evolutionary consequences of stress? Are there commonalities among the responses to osmotic, thermal, temporal, nutritional, social, predator, and competitive stresses? The response of individuals to environmental stress may have stimulated the evolution of stress proteins, homeostasis, acclimation, canalization, immune response, learning, and the numerous phenotypic plasticities noted throughout this book (Gabriel 2005, Emlen et al. 2006). Furthermore, there are remarkable consequences of understanding stress plasticity in the context of the ecology

and environmental impacts of interactions between humans and the environment (e.g., Relyea 2003a, Relyea and Hoverman 2006).

Phenotypic plasticity merges genes and environment — Phenotypic plasticity also addresses the nature vs. nurture controversy, because it merges these two polar concepts to reduce this exaggerated dichotomy. Under phenotypic plasticity, nature cannot be separated from nurture. Even at the lowest developmental level (transcription), gene activity is influenced by the surrounding internal and external environment. Environment influence on phenotype only increases down the epigenetic cascade of development. Even gamete genes are encased in a cytoplasmic environment that was presumably influenced by the parental environment, and continues to be influenced by current environmental conditions. Epigenetic inheritance further blurs genes and environment (Jablonka and Lamb 2005). Hence, genes and gene activities can never be separated from direct environmental influence, and most traits represent a gene-by-environment interaction. This realization elevates the role of environment in gene expression and development, and the role of development in evolution, and is partially responsible for the recent surge in evolutionary developmental biology (evo-devo) (Brakefield and French 2006, Sultan 2007).

Phenotypic plasticity alters environments and structures communities — Recent studies support the importance of phenotypic plasticity in shaping communities. An example is spittlebug-induced plasticity in growth form in willows, which subsequently alters the abundance of more than 30 willow-associated insects (Nakamura and Ohgushi 2003, Ohgushi 2005). An important realization is that impacts in communities need not be regulated by competition and predation in the classic sense of these factors shaping resource availability or the densities of organisms (Inbar et al. 2004, Schmitz et al. 2004, Van Zandt and Agrawal 2004, Agrawal 2005, Miner et al. 2005, Fordyce 2006, Schmitz 2006, Ohgushi et al. 2007, Chapter 7).

Phenotypic plasticity has practical importance — It aids systematics and taxonomy by helping to correct erroneous synonyms. Indeed, highly plastic genotypes have often been considered different species (Schlichting and Pigliucci 1998). This is especially problematic in entomology, where environmentally induced phenotypes are confused as distinct species (Uvarov 1966, Greene 1989, Mound 2005). For example, more than 20 divergent phenotyes of the thrips *Ecacanthothrip tibialis* were previously assigned species status (Ananthakrishnan 1969). Inaccurate species

identification or failure to recognize phenotypic plasticity can hamper basic research, disease diagnosis and medical and agricultural pest control.

Phenotypic plasticity may help us forecast establishment and spread of invasive species (Peacor et al. 2006, Richards et al. 2006, Muth and Pigliucci 2007, Slabber et al. 2007), aid conservation (Beckerman et al. 2002, Davis and Stamps 2004), and help us understand the consequence of environmental disruption (Bradshaw and Holzapfel 2006, 2007, Hendry et al. 2008). Differential plastic responses among interacting species may alter ecosystem interactions (Visser et al. 2006). Plasticity may aid environmental monitoring (Ellison and Gotelli 2002, DeCoen et al. 2006, Lee et al. 2006). For example, herbivore attack often induces a plastic defense response in plants, including the release of novel volatile compounds (Chapter 7). Different plant taxa generally release different volatile blends. As such, scientists could monitor community stress levels by analyzing the local atmosphere (DeMoraes et al. 2004). In industry, phenotypic plasticity is already aiding bioprospecting, as companies expose species to extreme environments or specific elicitors, to induce synthesis of novel bioactive substances (Poulev et al. 2003, Li and Barz 2005). In the future, induction of plastic biochemical pathways in plants or tissue cultures will be used to produce any number of commercially useful substances (Al-Tawaha et al. 2005, de Jeong and Park 2005, Zhao et al. 2005).

In agriculture, phenotypic plasticity helps us to understand variation in crop performance vis-à-vis herbivores, pathogens, anthropogenic inputs, and seasonal and spatial variability (Karban et al. 2004, Agrawal 2005). Phenotypic plasticity in pests, crop plants, or natural enemies can influence pest control (Bean et al. 2007, Luczynski et al. 2007, Pereira et al. 2007, Chapters 6, 7, 8), and the evolution of resistance (Adler and Karban 1994), and might be employed to our benefit (Davis and Stamps 2004). Once we understand the biochemical pathways and regulatory genes controlling induced defenses in crop plants, we can manipulate them, use genetic engineering to increase those beneficial responses, or transfer the capability to produce beneficial plasticities to other species (Kliebenstein et al. 2002, Edreva 2004, Agrawal 2005, Kappers 2005, Von Rad et al. 2005, Dana et al. 2006). Fisheries and animal husbandry also benefit from understanding phenotypic plasticity (de Jong and Bijma 2002, Collier et al. 2006).

Knowledge of how humans respond to stress, disease, carcinogens and drugs will continue to aid Medicine (Bateson et al. 2004, Nadeau and Topol 2006, Swynghedauw 2006, Calderwood et al. 2007). Likewise, phenotypic plasticity in human performance in response to exercise, altitude, nutrition,

temperature, space travel, etc, are of great interest (Flück 2006, Geurts et al. 2006, Asea and DeMaio 2007, Radakovic et al. 2007). Phenotypic plasticity influences racial disparity in IQ (Flynn 1987, Hernstein and Murray 1994, Pigliucci 2001), and confounds anthropology (Collard and Lycett 2008).

Benefits of Phenotypic Plasticity

Phenotypic plasticity is beneficial when it allows an individual to alter its phenotype to adaptively match a changing environment (DeWitt and Langerhans 2004). Plasticity can also be a beneficial self-reinforcing process (a self-induced adaptive plasticity) such as when voluntary exercise increases heart and muscle mass, which increases exercise ability (Swallow et al. 2005), or when sampling a new, but toxic food induces detoxifying enzymes which then allows the individual to switch to that new food. Because it can increase fitness in multiple environments, phenotypic plasticity widens niche breadth and geographic range, and may aid dispersal and colonization (Price et al. 2003, Schlichting 2004, Pigliucci et al. 2006, Chapters 16, 19), and evolutionary transitions (Aubret et al. 2007). Plastic species should be able to survive ecological catastrophes and avoid extinctions, not only because of their presumed broader geographic ranges, but because they already live in and have adapted to different habitats and express different phenotypes (Schlichting 2004). For interacting species, possession of phenotypic plasticity may retard coevolution in an antagonistic species, because of a "moving target effect" (Alder and Karban 1994, Agrawal 2001), and it may prevent competitive exclusion (Pijanowska et al. 2007).

The greatest benefit of phenotypic plasticity may be that it generates adaptive genetic change (see below), an essential long-term strategy for evolutionary persistence. Plasticity may foster adaptive evolution by allowing genotypes to jump maladaptive valleys to reach fitness peaks in adaptive landscapes (Price 2006). It may also protect hidden genetic diversity from elimination, allowing that stored diversity to be exposed under specific conditions (Schlichting 2004, Suzuki and Nijhout 2006). Indeed, maintenance of genetic variation is so essential to life that costly mechanisms to achieve it (recombination and sexual reproduction) are nearly universal. Phenotypic plasticity may serve a similar role by both shielding genetic diversity, and by producing organic novelty that can then be incorporated into the genome via genetic assimilation (see Box 2). By maintaining a capacity for plasticity, heredity may provide for modification of its own machinery (Baldwin 1896).

> **Box 2** Genetic Assimilation
>
> Genetic assimilation (**GA**) is a process by which an environmentally induced trait comes, after selection, to be constitutively expressed. Conrad H. Waddington proposed the idea in 1942, and then went on to demonstrate it experimentally, twice, using *D. melanogaster*. In the first case, he applied heat shock to fly pupae to induce a new adult phenotype with a reduced cross vein. After 14 generations of artificial selection under heat shock for expression of the plastic trait, some flies produced the veinless condition without heat shock (Waddington 1952, 1953a,b). In the second case, Waddington exposed fly eggs to ether to induce a novel phenotyopic abnormality, "bithorax," in the adult. After 29 generations of selection, the flies produced the bithorax phenotype in the absence of ether, and this new phenotype was heritable (Waddington 1956, 1961). In a third case, Waddington induced large anal papillae by exposing fly larvae to high salt levels. After 21 generations, the maggots expressed both large papillae and greater plasticity in low salt media (Waddington 1959).
>
> Waddington (1953a) proposed that selection had altered the regulation of trait expression, such that the thresholds for expressing these traits were lowered to the point that the traits were expressed in all environments (Fig. 8). Examples from nature might include fixation of extrafloral nectar production in *Acacia* (Heil et al. 2004), and fixation of aposematism (Sword 2002). Suzuki & Nijhout (2006) showed GA of body color in the lab.
>
> GA is an important idea because it suggests that acquired, phenotypic-plastic traits can become genetically fixed (Schmalhausen 1949). Hence, environmental induction can initiate evolutionary change (Pigliucci & Murren 2003). Furthermore, because the bithorax condition (above) created a second pair of wings, it mimics macroevolution, and thus suggests that macroevolutionary jumps might occur via genetic assimilation. GA in one trait might favor plasticity evolution in other traits, because as one trait becomes invariable to environmental conditions, it may increase conditional expression or selection pressure for plasticity in another (Jablonka & Lamb 2005). GA, its occurrence in nature, and its role in evolution are controversial subjects (de Jong 2005, Pigliucci et al. 2006, Crispo 2007), in part because of its similarity to Lamarckian evolution, the inheritance of environmentally acquired traits. However, GA is assumed to proceed via traditional Mendelian and Darwinian processes (see main text).

Plasticity Evolution

Phenotypic plasticity is probably ancestral, in the sense that environments have always changed and all living things are susceptible to abiotic and biotic factors (Nijhout 2003a). Although plasticity is not required to be beneficial or to have undergone adaptive evolution, it often has. One hypothetical pathway for the evolution of adaptive plasticity is through

susceptibility. In this scenario, an environmental variable disrupts physiological homeostasis and development, creating new traits and new trait values, and rearranging phenotypes to produce novel trait combinations (Eshel and Matessi 1998). Most organisms contain large amounts of "non-functional" genetic material in their genomes. These genes are normally repressed, via genetic canalization. However, extreme environmental or biochemical conditions may disrupt such inhibition, allowing expression of these hidden genes (releasing hidden phenotypes), while reducing expression of others, leading to novel phenotypes. Although some such changes are beneficial, most are probably not. Recurrence and selection would then presumably adjust the regulation of gene expression and select for gene combinations that produced either increased canalization or adaptive plasticities (Nijhout 2003a). Plasticities to diet, disease, and abiotic factors may have evolved this way.

There are many other hypothetical routes for the evolution of adaptive phenotypic plasticity. For example, plasticity could evolve as an exaptation, when a previously existing plasticity comes to serve a new function, is induced by a different cue, or is shifted in its expression (e.g., when a biochemical plasticity evolves to produce a morphological plasticity or when plasticity in overall body size is co-opted for a single structure) (Emlen et al. 2006). For example, eusociality in wasps may have evolved from diapause or nutritional plasticity (Page and Amdam 2007, Hunt et al. 2007). Likewise, plasticity could evolve after hybridization of two populations, each of which has evolved different fixed phenotypes, if expression of the different phenotypes in the new hybrid population now becomes environmentally controlled. In this case, the hybrid population already possessed the capacity to produce both phenotypes; all that is required is to link differential production to environment. Epigenetic processes (McCaffery et al.1998, Brodie and Agrawal 2001, Kirschner and Gerhart 2005), and extraneous sources of hormones may have influenced plasticity evolution (Chapter 20). A non-adaptive plasticity could evolve via genetic correlation with other traits under strong selection (Scheiner 1993).

A key to understanding how phenotypic plasticity can evolve is the concept of interchangeability (Fig. 8). Most traits are both genetic and environmentally influenced (Roundtree and Nijhout 1994, Bradshaw and Holzapfel 2001). An example is the pigment melanin, which is the end product of well-known enzyme chains (Baraboy 1999, Sugumaran 2002, Ito 2003). The sequence instructions for these enzymes are coded by DNA and are heritable (True 2003). But in many animals, melanin production and deposition are also environmentally influenced, whereby colder
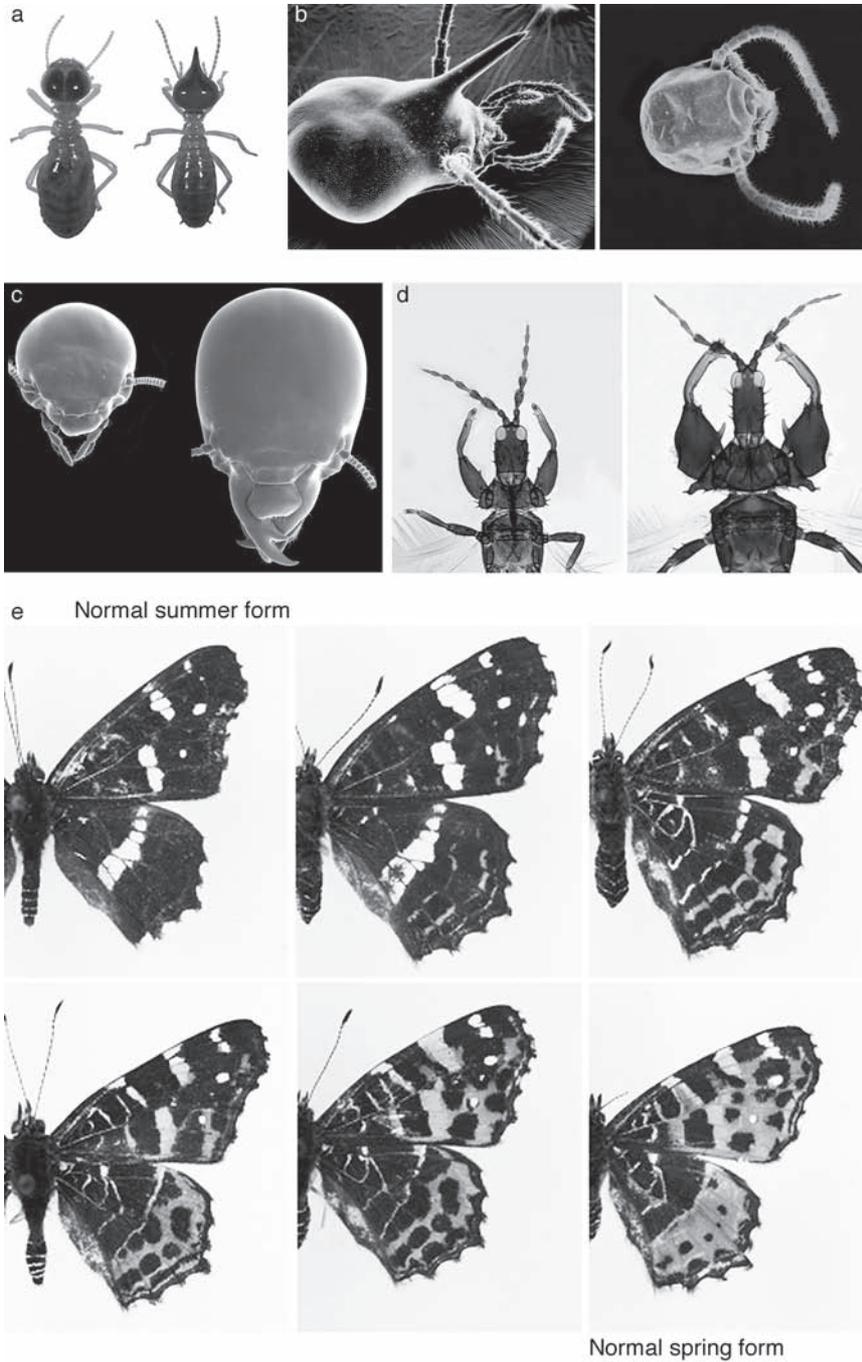
a

b

c

d

e   Normal summer form

Normal spring form

temperatures increase melanin and thus darken the body (Figs. 1d,e). This benefits individuals via solar heating, which counters the negative effects of cold temperatures (May 1984, Heinrich 1993). Hence, melanin production is both genetic and environmentally controlled, and this control is evolutionarily interchangeable: when there is genetic variation for degree of environmental influence, natural selection can select for either increased or decreased environmental sensitivity (West-Eberhard 2003, Suzuki and Nijhout 2006). Elimination of all flexibility produces a genetically fixed trait. Regulation of many traits is easily altered by adjusting response thresholds, enzyme saturation kinetics, timing of endocrine or development events or sensitivity periods of target tissues to hormones and morphogens (Meiklojohn and Hartl 2002, Moczek and Nijhout 2003a,b, Chapter 13). Hence, selection can easily slide trait regulation anywhere between total genetic and seemingly total environmental control (Fig. 8). The evolution of new phenotypes does not require the evolution of new gene complexes, but only the repatterning of existing genetic architecture and epigenetc interaction (Schlichting and Pigliucci 1998, Suzuki and Nijhout 2006, Emlen et al. 2007). Subsequent evolutionary loss of flexibility can permanently canalize the trait. Hence, a plastic trait can become subsumed into the genome as a canalized trait. An example may be extrafloral nectar (**EFN**) production in *Acacia* plants (Heil et al. 2004). In this genus, herbivore leaf damage induces the plant hormone jasmonic acid (**JA**) which induces EFN production, which attracts carnivorous plant bodyguards, which attack the herbivores. EFN inducibility is ancestral. But, in some *Acacia* species that have obligate ant bodyguards, the response to JA has evolved to such a low threshold, that individuals always produce EFN, in response to low, endogenous levels of JA. Hence, a plastic trait has been converted to a canalized trait via adjustments to the regulation of trait expression.

Fig. 7 Contd. ...

Fig. 7   (a) Worker and nasute-soldier of *Nasutitermes takasagoensis* termites from Japan (Hojo et al. 2004). Photo by Masaru Hojo. (b) Head of soldier (left) and minor worker (right) of *Hospitalitermes medioflavus* termites (Miura & Matsumoto 1995, 2000, Miura et al. 1998). (c) Head of worker (left) and soldier (right) of *Hodotermopsis sjostedti* termite (Miura 2005). (d) Large and small males of *Ecacanthothrips tibialis* thrips (Mound 2005). (e) Only two discrete forms of the nymphalid butterfly *Araschnia levana* are found in nature: the summer form (top left) and the spring form (bottom right). However, in the laboratory, intermediate phenotypes can be produced by subjecting individuals to intermediate environments or timed ecdysone injections, documenting that a continuous reaction norm lies at the base of this seasonal, diphenic polyphenism (Nijhout 2003a).
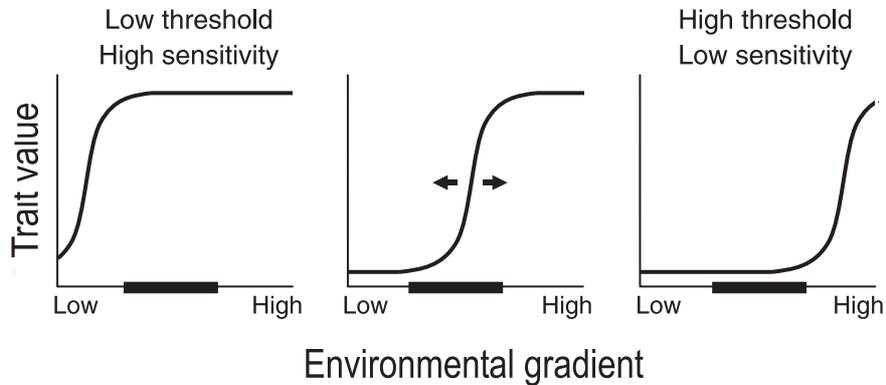
Low threshold
High sensitivity

High threshold
Low sensitivity

Trait value

Low High   Low High   Low High

Environmental gradient

Fig. 8 Interchangeability between genetic and environmental control of a trait over evolutionary time. For each graph, the center (bold) of the horizontal axis denotes the normal range of values for the given environmental factor. Seldom encountered extreme conditions are shown by thin lines. In the right graph, the trait has a low value in virtually all normal environments and is thus considered a genetic trait. In the left graph, the trait has a high value in nearly all environments and is also considered genetically fixed. In the middle graph, trait value flips between high and low, depending on the environment, and hence is considered an environmentally determined trait.

Interchangability explains the phenomenon of phenocopies, which are environmentally induced phenotypes that resemble genetically determined ones (Figs. 1d,e) (Goldschmidt 1935, West-Eberhard 2003). Exposing a species to extreme conditions can elicit hidden phenocopies (Suzuki and Nijhout 2006, Otaki 2007, 2008).

When Should Phenotypic Plasticity Evolve and What Form Will it Take?

If environments were unchanging, then fixed phenotypes would be favored. But, because environments are constantly changing, plasticity is often favored. Indeed, the only way for an *individual* to adapt to a changing environment is by changing its phenotype. A plastic individual can achieve high fitness in two or more environments, whereas a fixed-phenotype specialist that is highly adapted to only one environment would be less fit in a different environment. Likewise, a fixed-phenotype generalist would presumably have only moderate fitness in all environments.

In general, phenotypic plasticity should be favored when it produces higher fitness than a fixed strategy across all environments (Berrigan and Scheiner 2004). A reaction norm (RN) is a trait of the genotype, and like all

traits, a RN should evolve given directional selection on heritable additive genetic variance for plasticity (Doughty and Reznick 2004). Reaction norms respond to both artificial (Scheiner 2002, Suzuki and Nijhout 2006, Chapter 21) and natural selection (Hairston et al. 2001, Scheiner 2002, Bradshaw and Holzapfel 2006, Parsons and Robinson 2006), showing that plasticity can evolve. Note that plasticity evolution can be reversed (Chapter 21), and a flat RN (canalization or no plasticity) might evolve if it produced the highest fitness.

Theoreticians, modelers, and empiricist have proposed and examined numerous factors that favor or restrict plasticity evolution, alter reaction norms, or select for one type of plasticity over another (de Jong and Behera 2002, Scheiner 2002, Sultan and Spencer 2002, Berrigan and Scheiner 2004, David et al. 2004, de Jong 2005, Van Kleunen and Fischer 2005, Gabriel 2005, Gabriel et al. 2005, Garland and Kelly 2006, Chapters 15, 21), and these factors divide roughly into environmental factors, genetic (population and species) factors, and gene x environment interaction factors.

Environmental characteristics — Researchers interested in plasticity have examined a wide range of environmental factors including temporal vs. spatial heterogeneity, fine- vs. coarse-grain environments, predictability, speed, pattern, and permanency of change, number of selective factors, intensity of selection, frequency and strength of selection in alternative habitats, and reliability of cues that predict or signal environmental change (Chapter 7). By definition, phenotypic plasticity is a response to temporal or spatial environmental variation, and high variation should favor its expression and evolution Scheiner 1993, Sultan and Spencer 2004). Models and empirical studies suggest that plasticity should be more likely to evolve in temporal vs. spatial heterogeneity (Moran 1992), when cues are reliable (Karban et al.1999, DeWitt and Langerhans 2004), and in response to selective agents that slowly harm individuals, such as disease, cold, desiccation, etc., vs. those that act instantly with no warning, such as a tornado (Järemo et al. 1999, Sultan and Spencer 2002, Garland and Kelly 2006). Different environmental factors should select for different plasticities (Relyea 2003b, 2004b Boege and Marquis 2005). Speed of induction should correspond with speed of environmental change, and this may determine the type of plasticity that evolves (Van Buskirk 2002). When environmental changes are permanent, plastic change should be permanent (Relyea 2003c). Transgenerational plasticity should evolve when parent's environment predicts that of the offspring (Galloway 2005). When environmental variation is great and random, but cues are unreliable, then plasticity will

not be favored and individuals should employ bet hedging strategies (Seger and Brockmann 1987, DeWitt and Langerhans 2004).

Genetic (population & taxon) characteristics — see de Jong and Behera 2002, DeWitt and Scheiner 2004. Phylogenetic constraints clearly prohibit certain plasticities in certain taxa. For example, plants are limited in behavioral plasticity, and in arthropods, molting, metamorphosis, and exoskeletons may preclude or favor certain plasticities. Plastic responses should change with ontogeny (Relyea 2003c), and decline with age, because of diminished developmental capability or because of impending senescence (Frechette et al. 2004). Some suggest that restricted gene flow favors plasticity evolution (Karban and Nagasaka 2004, Van Buskirk and Arioli 2005), and others opine that migration and panmixis favor plasticity (Tufto 2000, Sultan and Spencer 2002, Zhang 2006). K-strategists should be plastic, because they are long-lived, and thus encounter more temporal variability, and, with low fecundity, cannot afford to lose a single offspring. In contrast, large size and ample reserves in K-strategists may buffer environmental variation, obviating the need for plasticity. Short-lived r-strategists should have little need for plasticity, and can afford to lose some of their many offspring through bet-hedging. In contrast, plasticity may be favored in r-strategists because of their high rates of dispersal into new habitats. Polygenic quantitative traits should be more plastic than single locus traits (Roff 1996). Some models suggest that heterozygosity inhibits plasticity, because heterozygosity buffers environmental effects (see Scheiner 1993), but others disagree (Pigliucci 2005). If individuals cannot exhibit a reaction norm but groups can, then that reaction norm could feasibly evolve via group selection (Via et al. 1995, Sih 2004).

Gene x environment interaction factors — Plasticity evolution is presumably influenced by how individuals interact with the environment, including relative fitness benefits of plastic change in different environments, ecological tradeoffs, inclusive fitness, relative lengths of lifetime vs. stress period, dispersal range vs patch size, ecological feedbacks (i.e., when altered phenotypes alter the environment, which then alters selection on plasticity), including reciprocal plasticity interactions between genomes.

The type of plasticity that evolves should hinge on the ratio between generation time and environment fluctuation time (Gabriel and Lynch 1992, Schlichting and Pigliucci 1998, Meyers et al. 2005). Rapid, reversible behavioral and physiological plasticity should evolve when the environment rapidly switches back and forth and when life-span is much

longer than environmental change (Gabriel 2005, Gabriel et al. 2005). With slower environmental change, morphological and life history plasticities should evolve, including once per lifetime developmental conversions. Longer cycle environmental fluctuations might select for transgenerational plasticities. Plasticity itself may or may not have high costs (Padilla and Adolph 1996, Van Tienderen 1997, Gabriel 2005), and therefore a plastic trait should be more likely to evolve when there are weak genetic or ecological correlations with other traits that are under selection in a different direction (de Jong 2005, Garland and Kelly 2006). However, plasticity evolution should increase when it produces large benefits, and when there is a positive correlation with other favorable traits, such as in plants when increased plastic antiherbivore defense aids pollination, allelopathy or disease resistance. Phylogenetic constraints on performance tradeoffs may prohibit certain plasticities in certain taxa. A correlation between habitat selection and trait plasticity should favor evolution of plasticity (Scheiner 1993). Selection can act directly on the shape of the reaction norm (Harshman et al. 1991, Scheiner and Lyman 1991, Scheiner 2002). However, directional selection on the mean constitutive value of a trait can also increase plasticity of that trait (alter the reaction norm), in the direction of selection (Swallow et al. 2005, Garland and Kelly 2006). Some suggest that traits highly correlated with fitness should have low plasticity (Schlichting and Smith 2002), and a flat reaction norm may be highly adaptive. However, some traits strongly linked to fitness, such as antipredator defenses and seasonal adaptations are often highly plastic (Chapters 4-9 & 16). In species with wide geographic ranges, different populations exhibit different, adaptive plasticities (Winterhalter and Mousseau 2007).

In sum, plasticity evolution is favored by environmental variation, strong differential selection in alternative environments, cues that accurately signal environmental change, high fitness benefits and low costs to plasticity, and heritable genetic variance for plasticity (Berrigan and Scheiner 2004).

A seldom discussed concept is that mutations lie at the base of phenotypic plasticity. This is because all trait expression is embedded in a particular genetic background. Different genotypes produce different reaction norms – one consequence of underlying genetic variation. The ultimate origin of such genetic variation is mutation. Hence, phenotypic plasticity is a consequence of mutational evolution. However, there is a fundamental difference between traditional mutational evolution and evolution via phenotypic plasticity. The mutations that produce specific plastic responses may remain hidden from the phenotype (or at least to that specific trait state) for millions of years.

It is only when the environment exposes that trait that selection on that trait state can begin. In general, mutations have little evolutionary impact until they are exposed in the phenotype.

There has been confusion regarding the relation between genetic variability for a specific plastic trait vs. overall genetic variability, and the occurrence vs. the evolvability of phenotypic plasticity. Phenotypic plasticity does not require genetic variability. For example, (except for developmental noise) all individuals of a genetically identical clone would exhibit the same phenotypic plasticity to the same altered environment. In this case, lack of genetic variability in all traits would preclude selection on both the reaction norm and associated traits (genetic accommodation). Thus, excepting for new mutations, this genotype could not evolve. In the case of background genetic variation, but no genetic variation for the plastic trait, the reaction norm could not evolve, but the new phenotype could evolve to be more fit via genetic accommodation. Hence, an invariant and initially detrimental plastic response could, over evolutionary time come to be imbedded in a highly fit phenotype. If genetic variation existed for both the plastic trait and most other traits, then the reaction norm, background traits, and fitness could evolve, to produce a highly intetgrated and adaptive plasticity.

In insects, evolution of plasticity is aided by their modularity and metamorphosis. For holometabolous insects in particular, future adult structures such as wings and legs derive from small clumps of cells (imaginal disks) that persist through immature development and are only activated via hormones during the pupal stage. Differences in timing of induction and in response of different imaginal disks allow independent expression and evolution of the resulting organs (Nijhout 2003a, Emlen et al. 2007).

Plasticity as a Factor in Evolution

Scientists debate whether or not phenotypic plasticity speeds or retards evolution (Chapter 21). Some suggest that plasticity shields traits from evolution because selection chooses among phenotypes (Huey et al. 2003, Price et al. 2003, de Jong 2005). Individual adaptation may preclude genetic selection. An example might be when a plastic behavior such as solar basking, microhabitat shift, or seasonal migration moderates body temperature, preempting selection for fur, melanin or thermal-adapted enzymes. Others suggest that plasticity stimulates evolutionary diversification by generating novelty (West-Eberhard 2003, Schlichting

2004), and/or via genetic accommodation. An example is dung beetles, where plasticity in body size may have subsequently stimulated evolution of testis size and alternative mating tactics (Simmons et al. 2007). Phenotypic plasticity may act as an evolutionary capacitor to shield genetic variation from elimination, only to release it under extreme environmental conditions (Masel 2005, Feder 2007). Price (2006) argues that phenotypic plasticity can either retard or accelerate rates of evolution, based on relative fitness of the new phenotype. If an environmentally induced plastic change has high fitness, then there should be little subsequent selection on either the plastic trait or associated traits (no genetic change), as long as the population is exposed to both environments. If the plastic change is highly detrimental, then selection should act on genes to reduce the plastic response or compensate in other ways (Nijhout 2003a, Grether 2005). If the plasticity is slightly or moderately favorable, then subsequent selection should produce genetic change that alters the reaction norm and associated traits to bring the genome to an adaptive peak.

How Phenotypic Plasticity Contributes to Speciation

A remarkable claim is that phenotypic plasticity stimulates evolution and contributes to speciation. But how can environment-induced changes to the phenotypes of individuals influence evolution? Isn't this Lamarckian, the assimilation of environmentally acquired traits into the genome? Surprisingly, phenotypic plasticity theory suggests that environment-induced changes to individuals can become absorbed into the genome (Jablonka et al. 1998, ten Cate 2000), but via traditional Medelian processes (West-Eberhard 2003, Schlichting 2004, Pigliucci et al. 2006). There are different hypothetical pathways for this to occur (e.g., Grether 2005, Rodríguez et al. 2007), but one possible pathway would be the following:

(1) *Trait origin via phenotypic plasticity* - the production of an environment-induced alteration of the phenotype. This could be passive, and be detrimental, neutral, or beneficial with regard to fitness.

(2) *Phenotypic accommodation*, whereby the individual accommodates the changed phenotype by adaptively altering additional phenotypic traits, such as physiology, behavior, or morphology (West-Eberhard 2003). Such accommodation increases survival of the new phenotype in the new environment, allowing reproduction. A hypothetical example is when the environment (such as a new food or infection by a non-lethal microorganism) induces a darker body color, which increases diurnal heating, thus limiting diurnal foraging for a

normally diurnal insect. An individual might accommodate this new phenotype by altering its foraging pattern to forage during the cooler night. Note that in this hypothetical example, an environmental factor has exposed a new trait (dark body color) that was not previously present. Expression of this novel trait (a different phenotype) places the population in a new selective regime (greater susceptibility to sunlight, nocturnal foraging, different predators, etc.).

(3) *Genetic accommodation* — Assuming population genetic variation in most traits, the recurrence of this particular environmental induction (dark color) in numerous individuals and generations would allow this novel phenotype to be tested repeatedly in the new environment and among a vast assortment of genetic variants. Over time, this would allow natural selection to select for alleles and gene combinations that improve regulation, form and side effects of the novel trait and its genetic background, increasing survival and fitness of individuals expressing the new environmentally induced traits (dark color and nocturnal foraging). Examples of genetic accommodation in this hypothetical case might be better nocturnal eyesight or longer antennae for non-visual sensing. Genetic accommodation can shift the overall fitness value of the environmentally induced phenotype, moving it from detrimental to beneficial.

(4) *Adjustment of the capacity and shape of the reaction norm via the Baldwin Effect* (see Box 3) — Here, natural selection alters the frequency of genes and gene combinations that influence the expression of the plasticity – genes that do not produce the optimal plastic response are eliminated.

(5) *Genetic assimilation* — An evolutionary mechanism by which environmentally induced (facultative) traits become genetically fixed (obligatory) (Box 2; Fig. 8). In our hypothetical case, the population would evolve to always express dark body color, under virtually all conditions and in all environments.

(6) *Speciation* — Phenotypic plasticity and some combination of steps 2-5 (above) produces differences that increase assortative mating or otherwise restrict gene flow. Continual natural selection, genetic drift, and mutation of the population increase habitat, mating, and genetic divergence from adjacent populations, leading to eventual speciation.

In the above scenario, the environment exposes a new trait, which eventually becomes genetically fixed in a population. Moreover, an environmentally induced change to the phenotype sends a population down a different evolutionary pathway, leading to speciation. Note that

evolutionary divergence begins not with genetic change, but with environmentally induced change to the phenotype. Reproductive isolation does not initiate speciation. The process is Mendelian and Darwinian, because it relies on preexisting genetic variation and traditional natural selection, and may be as important as mutation for producing Earth's diversity (Schlichting and Pigliucci 1998, West-Eberhard 2003).

A real life example of how phenotypic plasticity might lead to evolutionary divergence is the famous case of sympatric diversification in *Rhagoletis pomonella* fruit flies. Adults of this New World species oviposit

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Box 3 The Baldwin Effect

A proposed process by which traits acquired during an individual's lifetime influence subsequent evolution of those acquired traits.

James Baldwin was an American psychologist and evolutionary theorist who was interested in the development and evolution of cognition. He proposed that a beneficial learned behavior acquired during an individual's lifetime contributed to the fitness of that individual, and thus favored the evolution of increased capacity to acquire or perform that behavior in the population (Baldwin 1896): *"The most plastic individuals will be preserved to do the advantageous things for which their variations show them to be the most fit, and the next generation will show an emphasis of just this direction in its variations."* Here, phenotypic plasticity (a switch in behavior) of an individual during its life is a factor in evolution. Although Baldwin was primarily concerned with learning, his ideas could be applied to any number of plastic traits that are acquired by individuals in response to variable environments, such as increased muscle mass, melanization, calluses, and O_2 capacity in individuals exposed to exercise, sunlight, dermal irritation, or high altitudes, respectively. Natural selection should select for greater or lesser capacity for such plastic responses, depending on their contribution to fitness.

Baldwin's ideas are controversial (see Nortman 2003, Webber & Depew 2003, Crispo 2007), but important for a number of reasons. He was one of the first to recognize plasticity in individuals and to link such plasticity to evolution. He suggested that the expression of environmentally induced traits (the shape of the reaction norm) can evolve. His theory also approached Lamarckism in that acquired beneficial traits, induced by the environment, could (in some cases) become more genetically determined. Baldwin (1896) suggested that his process was an example of, *". . . heredity providing for the modification of its own machinery. Heredity not only leaves the future free for modifications, it also provides a method of life in operation of which modifications are bound to come."* The Baldwin Effect might explain sympatric speciation, instinct, fixation of learned songs in birds, host plant preferences in insects, and numerous other phenomena (see Webber & Depew 2003), and was recently demonstrated by Suzuki & Nijhout (2006).

into fruits, which the resulting maggots consume. After the colonization of North America by Europeans, some populations of *R. pomonella* jumped from native hawthorn fruits to introduced apple, and then to introduced cherry (Bush 1975, Prokopy et al. 1988, Feder et al. 1994), and this may have occurred via phenotypic plasticity (Chapter 18). Hawthorn fruit flies prefer hawthorn fruits. However, naïve adults that experience apple alter their phenotype (via behavioral phenotypic plasticity) to prefer apple (Prokopy et al. 1988), leading to habitat-specific mating and ovipostion (Feder et al. 1994). Recurrence in the new environment (apple or cherry), allowed natural selection to alter allele frequencies to fit the new habitat. For example, each fruit species ripens at a different time of year, and each host-population has evolved to emerge at the appropriate time (Bush 1975). The different populations also evolved different allele frequencies for thermally adapted enzymes that match the respective temperatures of their fruits (Feder et al. 1997, Filchak et al. 2000). In this case, phenotypic plasticity has apparently initiated a rapid evolutionary divergence in *R. pomonella*, which now exists as distinct races (Feder et al. 1994, Dambrowski et al. 2005). See also Bolnick and Fitzpatrick (2007).

Note that a well-established plasticity is a bridge to speciation. Indeed, a highly integrated and adapted developmental conversion is already ideally suited to stand on its own as an independent species.

Can Stress Induce Adaptive Mutations in Individuals? Is Genetic Variability a Plastic Trait?

Stress (poor nutrition, xenobiotics, radiation, extreme temperatures, etc.) can alter genes in individuals (Badyaev 2005). The best examples are the non-heritable, but adaptive mutations forming the mammalian immune response (Frost 1999). Stress also increases mutation rates in bacteria (Kidwell and Lisch 2001, Bjedov et al. 2003, Saint-Ruf and Matic 2006), often by induction of DNA mutases (Radman 1999). Although these mutations are non-directional, and mostly harmful, some are beneficial (Bjedov et al. 2003). *Escherichia coli* can switch phenotypes from high- to low-mutagenesis forms depending on environmental stress levels. This ability (hypermutation) varies among strains and is thus under genetic control (Bjedov et al. 2003). This is not restricted to bacteria. Individual flax plants mutate when nutritionally stressed, altering both their and their progeny's morphologies (Marx 1984, Cullis 1987, 1988). Hence, organisms may have evolved mechanisms to increase both genetic and phenotypic variation when exposed to harmful environments, and thus the probability of generating adaptive variants. However, even if the new mutations were not

immediately adaptive, environmentally induced mutagenesis creates more raw product upon which selection can act. Moreover, the mutations occur at exactly the time when increased variation is needed – i.e., during profound environmental change. Because stress alters the phenotype, these new mutations are already imbedded in a plastic response. Presumably, both mutagenic capability/susceptibility and phenotypic sensitivity to mutation evolve and may be more common in populations or species that have evolved in highly variable environments (Bjedov et al. 2003, Meyers et al. 2005, Jones et al. 2007, Landry et al. 2007).

An example of this phenomenon in insects might be locusts, which display phenotypic plasticity in response to population density (Chapters 5, 6). The low-density phenotype is sedentary, whereas the high-density, gregarious phenotype typically migrates hundreds of kilometers to new environments. It is interesting that in the gregarious morph, recombination increases during meiosis, perhaps as an adaptation to increase genetic variability prior to dispersal into new, unknown environments (Nolte 1974).

Any number of mechanisms could increase genetic variation in individuals following environmental change (Mckenzie and Rosenberg 2001, Badyaev 2005), including stress-induced transposable elements (Kidwell and Lisch 2001), or lowered immunity, which fosters mutation-inducing viruses. Finally, genomes often contain large amounts of presumably neutral, non-functional genes. This largely unstudied and unknown genetic "dark matter" may represent a vast storehouse of potential genetic alternatives, waiting to be employed when changing environmental conditions necessitate altered phenotypes (Eshel and Matessi 1998). In general, genetic (V_G) and mutational (V_M) variability varies among environments and genotypes (Schlichting 2004, Landry et al. 2007).

Phenotypic Plasticity in Outcomes, Including Niche Construction

Although ecological and behavioral outcomes have not traditionally been considered phenotypic plastic traits (Thompson 1988), they are a direct extension of the phenotype, vary with genotype, and are greatly influenced by environmental conditions, and, hence, should be considered as plastic traits. Thus, competitive outcomes such as dominance and territory size, performance outcomes, such as mating success, fecundity, distance dispersed, and resources harvested, and survival under alternative environmental conditions can be plastic (Gorur et al. 2005, Engqvist 2008).

One outcome of the phenotype is niche construction, which refers to an organism's ability to alter the environment so as to influence or produce a

niche (Day et al. 2003, Odling-Smee et al. 2003). Examples include structures produced by habitat architects, such as gallers, beavers, and nest-building social insects (Inbar et al. 2004). Habitat architects and ecosystem engineers may create new habitats, which feedback to dramatically induce phenotypic changes in those same individuals. For example, nest construction in social insects may influence any number of phenotypic traits of nest builders, such as body size, development rate, caste, fecundity, time spent in defense vs. foraging, etc. (Hölldobler and Wilson 1990). Likewise, by constructing reefs, corals alter local wave force and turbulence, temperature, oxygen and light levels, and associated biota, including predators, pathogens, and prey, and conspecific densities. Hence, there can be a continuous, reciprocal interplay between phenotype and environment. Altered environments induce phenotypic changes in individuals, and altered individuals may alter their environment in a continuous phenotypic plasticity-environment feedback loop.

Reciprocal Phenotypic Plasticity among Interacting Individuals

Individuals respond to changing environments by phenotypic plasticity, but what happens when a changing environment includes other individuals that are themselves changing? Phenotypic change in one individual may induce a change in a second individual, which induces further change in the first, in a continuous, reciprocal, phenotypic plasticity loop (Agrawal 2001, Fordyce 2006, Mooney and Agrawal 2007). Such interactions occur both within and between species, and can be mutualistic, antagonistic, or commensal. Conspecific examples are seen in certain social wasps and ants in which physical aggression between individuals determines queen vs. non-queen developmental trajectories, including differences in morphology, pheromone release, fecundity, and life history (Premnath et al. 1996, Heinze 2004). Here, an individual's phenotype changes over time in response to phenotype changes in an antagonist. Heterospecific cases include mutualistic reciprocal interactions between reward-producing herbivores or plants and their insect bodyguards (Huxley and Cutler 1991, Whitman 1994, Chapter 7). For example, *Piper cenocladum* plants produce more food bodies when bodyguard ants are present, which induces increased residency, feeding, and guarding by attendant *Pheidole bicornis* ants, which, in turn, presumably induces more food body production (Risch and Rickson 1981). Antgonistic reciprocal phenotypic plasticity can occur between predators and prey (Chapter 7). For example, some mussels increase shell thickness and muscle strength in

response to crab presence, and some mussel-eating crabs produce larger claws in response to thick muscle shells (Smith and Palmer 1994, Reimer and Tedengren 1996, Leonard et al. 1999).

Reciprocal phenotypic plasticity requires the addition of a new term to the equation for phenotype variance, because now the environment includes the genes and plastic responses of one or more interacting organisms (Strauss and Irwin 2004, Wolf et al. 2004). Plotting the phenotype of one genotype vs. either density or phenotype of the interacting genotype provides a traditional reaction norm. Plotting the changing phenotypes of two interacting genotypes over time produces a plastic interaction norm (Thompson 1988, Agrawal 2001). Evolution of such plastic interaction norms could lead to runaway phenotypes, whereby each population continues to evolve ever greater capacity for phenotypic plasticity (more sensitive or extreme reaction norms) (Adler and Grünbaum 1999, Agrawal 2001). Such "plasticity coevolution" might have resulted in the phenomenal ploy-counter ploy interactions that we see among some antagonists (Chapter 7). Plasticity coevolution should be common among symbiotic species.

Transgenerational Effects on Offspring Phenotype

Parents can influence offspring phenotypes through a variety of non-genetic processes (Bernardo 1996, Mousseau and Fox 1998). Parental effects can derive from the nutritional status of the parent, or from more elaborate environmental effects generated by the habitat or the parent itself (Stelgenga and Fischer 2007). Females may adaptively vary size, quality, and diapause state of eggs (Fox and Czesak 2000, Chapters 11, 19), and allocate resources to offspring based on mate quality (Sheldon 2000). Egg size may determine plastic capacity of larvae (McAlister 2007). Parents also produce galls, nests, and habitats in which populations densities of interacting species have been radically altered. Galls, nests (Hölldobler and Wilson 1990) and altered habitats can persist for generations, serving as an ecological inheritance (Day et al. 2003). Lemon ants produce devil's gardens that can contain monocultures of 350 trees and last for 800 years (Frederickson et al. 2005). Parents can also pass culture to offspring (Sherry and Galef 1990, Grant and Grant 1996), and cultural divergence can lead to genetic divergence (ten Cate 2000, Slabbekoorn and Smith 2002).

In locusts, phase state is passed to the next generation by a water-soluble factor placed in the foam of the egg pod (Miller et al. 2008, Chapter 5). Mothers also adaptively control offspring sex and morphology (Pienaar and

Greef 2003b). In *Orthophagus* dung beetles, fathers influence son's mating strategy (Hunt and Summons 2000, Chapter 3). Large males help females to produce larger dung balls, which produce larger male offspring with horns. Only horned males fight for females; small males sneak copulations. In other insects, food- or habitat-imprinting causes epigenetic inheritance of habit preference, with numerous phenotypic plasticity consequences for offspring (Bernardo 1996, Davis and Stamps 2004). Repeated cycles of habitat-induction or imprinting in successive generations allow habitat-specific genetic adaptation (Davis and Stamps 2004).

Females also bequeath offspring with specific detrimental (O'Neill et al. 1997, Boucias and Pendland 1998) or beneficial (Baumann 2005) microorganisms, such as mutualistic symbionts, which alter host phenotype in numerous ways, such as increasing fitness under cold conditions (Dunbar et al. 2007). Mothers may choose to pass or not pass endosymbionts, depending on local conditions (e.g., Stern and Foster 1996). Changes induced by symbiotic microorganisms may drive genetic divergence (Wade 2001, Flor et al. 2007, Riegler and O'Neill 2007).

Conclusion

Phenotypic plasticity theory suggests that existing paradigms may be incomplete. It shifts our thinking, because it requires that virtually all biological fields consider individual flexibility. In developmental biology, ontogeny is no longer fixed, but is a flexible process subject to environmental input. We can not assume that differences in organisms are genetic, because the genotype does not determine a set phenotype, but a range of possible phenotypes. In systematics, species morphology is not static, but can vary in time and space. In physiology, canalization is often accomplished via underlying plasticity, and performance is conditional on present, past, and parent's environments. In ecology, phenotypic plasticity tells us that environmental interactions are much more complex and dynamic than previously imagined, and that plasticity can lead to altered community structure. Individuals can rapidly and adaptively alter their (and their offspring's) relationships with the environment, with profound fitness and ecosystem consequences. This process is reciprocal when altered environments, including interacting individuals, feedback to induce additional plasticity in individuals. In evolution, plasticity theory suggests that phenotypic plasticity generates novelty, and hence stimulates adaptation and speciation. As West-Eberhard (2003) notes, individual adaptation can lead to population adaptation, and genes are useful

followers, not leaders in evolutionary change. Indeed, the developmental reaction norm may be the main object of selection on phenotypes (Dobzhansky 1951, Schlichting and Pigliucci 1998).

Phenotypic plasticity makes us realize that genes and environment are forever intimately linked. Biological existence is an iterative reciprocal process between genes, individuals, and environment. Genes provide a menu of developmental possibilities and phenotypes, but the environment determines the phenotypic outcome. The environment subsequently selects among altered individuals to alter population gene frequencies, which determine how future individuals will respond to environmental variability.

Glossary of Terms Used in Phenotypic Plasticity
(see Chapter 2, this Volume for additional discussion)

Baldwin effect – Stabilizing selection on the shape of a reaction norm.

Canalization – The operation of internal factors during development, physiology, or behavior, that reduces the influence of environmental stimuli to produce one outcome (Waddington 1940, 1942). Environmental canalization—The production of a single phenotype despite environmental variability (see Debat & David 2001).

Coevolution – Reciprocal genetic change in species engaged in an interaction.

Developmental conversion – An adaptive, discrete, and (normally) permanent phenotypic plasticity, usually with no intermediate forms. Thought to be produced via a developmental switch (Smith-Gill 1983).

Developmental plasticity – Irreversible phenotypic plasticity resulting from environmental influence on development of an individual (Piersma & Drent 2003).

Developmental switch – A threshold mechanism that produces a discrete phenotype (polyphenism), e.g., worker vs. queen determination in honeybees (Levins 1968).

Epigenetic – Development and interactions of products and processes downstream of primary gene products.

Epistasis – The effects of two or more genes on a single trait.

Genetic accommodation – A change in gene frequencies due to selection on the regulation, form, or associated effects of a novel trait (West-Eberhard 2003).

Genetic assimilation – When environmentally induced phenotypic variation becomes constitutively produced (no longer requires the environmental stimulus to be induced).

Genotype – The genes possessed by an individual, including all the genes or a specific defined subset of the total genome.

Homeostasis – Maintenance of an equilibrium state by some self-regulating capacity of an individual (see Debat & David 2001).

Lamarckian evolution – The theory that traits induced in individuals by the environment during their lifetime could be inherited by their offspring, thus causing evolution. This idea was discredited because it could not be demonstrated and lacked a mechanism by which an environmentally altered phenotypic trait could alter gametes. Nonetheless Lamarck had two very important contributions that often get lost in the discredit of "inheritance of acquired characteristics": 1) the recognition of phenotypic plasticity as an important fitness enhancing strategy within a generation, and 2) the many non-genetic parental environment effects that do influence the phenotype of offspring.

Life-cycle staging – Cyclic, reversible phenotypic plasticity in a long-lived individual in response to predictable seasonal changes, such as winter color change in fur of sub-Arctic birds and mammals.

Phenotype – The manifestation of an organism including its morphological, physiological, behavioral, and life history traits, exclusive of genetic composition, but inclusive of genetic expression.

Phenotypic accommodation – The immediate adaptive adjustments of an individual to the appearance of a new trait, without genetic change (see West-Eberhard 2003).

Phenotypic flexibility – Phenotypic plasticity that is reversible within individuals (Piersma & Drent 2003), e.g., gain or loss of muscle mass with exercise.

Phenotypic integration – The coordination in the expression of individual traits in response to environmental variation**.**

Phenotypic modulation – A continuous, often passive and reversible, non-adaptive phenotypic plasticity (Smith-Gill 1983).

Pleiotropy – When one gene influences multiple traits.

Polymorphism (genetic) – The existence of two or more genotypes in a population. Genetic polymorphism can lead to divergent phenotypes (see West-Eberhard 2003).

Polymorphism (phenotypic) – The existence of two or more (often discrete) morphological forms in a population, caused by either genetic polymorphism or phenotypic plasticity, but generally not ontogeny. Some authors include any variable phenotypic trait including allozymes,

physiology, behavior, life history, etc. This definition of polymorphism is no longer widely used.

Polyphenism – Phenotypic variation a single genotype, due to phenotypic plasticity or development. Includes morphological, physiological, behavioral, and life history variation (see Mayr 1963, West-Eberhard 2003). Some authors (Nijhout 2003a, Piersma & Drent 2003) restrict polyphenism to discrete, adaptive, or irreversible alternative morphologies (see Chapters 2 & 13, this Volume).

Reaction norm (= norm of reaction) – The set of phenotypes expressed by a genotype when maintained under different environments. Usually illustrated as a line graph plotting phenotypes vs. environment for different genotypes. Some authors restrict reaction norms to continuous plasticities (e.g., Nijhout 2003a).

Threshold trait – A trait that exists in two or more discrete states (phenotypes), determined by a threshold level of an underlying continuously variable morphogen, such as hormone titer (Roff 1996).

References

Adler, F. R. and D. Grünbaum. 1999. Evolution of forager responses to inducible defenses. pp. 257–285 *in* R. Tollrian and C. Harvell (eds.). *The Ecology and Evolution of Inducible Defenses*. Princeton Univ. Press, Princeton, NJ.

Adler, F. R. and R. Karban. 1994. Defended fortresses or moving targets? Another model of inducible defenses inspired by military metaphors. Am. Nat. 144: 813–832.

Agrawal, A. A. 2001. Phenotypic plasticity in the interactions and evolution of species. Science 294: 321–325.

Agrawal, A. A. 2001. Transgenerational consequences of plant responses to herbivory: an adaptive maternal effect? Am. Nat. 157: 555–569.

Agrawal, A. A. 2005. Future directions in the study of induced plant responses to herbivory. Entomol. Exp. Appl. 115: 97–105.

Agrawal, A A., C. Laforsch and R. Tollrian. 1999. Transgenerational induction of defenses in animals and plants. Nature 401: 60–63.

Agrawal, B. K. 2007. Phenotypic plasticity in aphids (Homoptera: Insecta): components of variation and causative factors. Curr. Sci. 93: 308–313.

Al-Tawaha, A. M., P. Seguin, D. L. Smith and C. Beaulieu. 2005. Biotic elicitors as a means of increasing isoflavone concentration of soybean seeds. An. Appl. Biol. 146: 303–310.

Amdam, G. V., K. A. Nilsen, K. Norberg, M. K. Fondrk and K. Hartfelder. 2007. Variation in endocrine signaling underlies variation in social life history. Am. Nat. 170: 37–46.

Ananthakrishnan, T. N. 1969. Indian Thysanoptera. CSIR Zoological Monograph 1: 102–117.

Ananthakrishnan, T. N. 2005. Perspectives and dimensions of phenotypic plasticity in insects. pp. 1-23 *in* T. N. Ananthakrishnan and D. W. Whitman (eds.). *Insect Phenotypic Plasticity: Diversity of Response*s. Science Publishers, Enfield, NH.

Ananthakrishnan, T. N. and D. W. Whitman (eds.). 2005. *Insect Phenotypic Plasticity: Diversity of Response*s. Science Publishers, Enfield, NH.

Asea, A. A. and A. DeMaio (eds.). 2007. *Heat Shock Proteins: Potent Mediators of Inflammation and Immunity.* Springer, New York.

Aubret, F., X. Bonnet, R. Shine. 2007. The role of adaptive plasticity in a major evolutionary transition: early aquatic experience affects locomotor performance of terrestrial snakes. Functional ecology 21: 1154–1161.

Badyaev, A. V. 2005. Stress-induced variation in evolution: from behavioural plasticity to genetic assimilation. Proc. R. Soc. B 272: 877–886.

Baldwin, J. M. 1896. A New Factor in Evolution. Am. Nat. 30: 441–451, 536–553.

Baraboy, V. A. 1999. Melanin: the structure, biosynthesis, biological functions. Ukrainskii Biokhimicheskii Zhurnal 71: 5–14.

Baratte, S., M. Cobb, C. Peeters. 2006. Reproductive conflicts and mutilations in queenless *Diacamma* ants. Anim. Behav. 72: 305–311.

Bateson, P., D. Barker, T. Clutton-Brock, D. Deb, B. D'Udlna, R. A. Foley, P. Gluckman, K. Godfrey, T. Kirkwood, M. M. Lahr, J. McNamara, N. B. Metcalfe, P. Monaghan, H. G. Spencer and S. E. Sultan. 2004. Developmental plasticity and human health. Nature 430: 419–421.

Baumann, P. 2005. Biology of bacteriocyte-associated endosymbionts of plant sap-sucking insects. Annu. Rev. Microbiol. 59: 155–189.

Bean, D. W., T. L. Dudley and J. C. Keller. 2007. Seasonal timing of diapause induction limits the effective range of *Diorhabda elongata deserticola* (Coleoptera: Chrysomelidae) as a biological control agent for tamarisk (*Tamarisk* spp.). Environ. Entomol. 36: 15–25.

Beckerman, A., T. G. Benton, E. Ranta, V. Kaitala and P. Lundberg. 2002. Population dynamic consequences of delayed life-history effects. Trends Ecol. Evol. 17: 263–269.

Benard, M. F. 2004. Predator-induced phenotypic plasticity in organisms with complex life histories. Annu. Rev. Ecol. Evol. Syst. 35: 651–673.

Bernardo, J. 1996. Maternal effects in animal ecology. Am. Zool. 36: 83–105.

Berrigan, D. and S. M. Scheiner. 2004. Modeling the evolution of phenotypic plasticity. pp. 82-97 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic Plasticity*. Oxford Univ. Press, Oxford.

Bjedov, I., O. Tenaillon, B. Gérard, V. Souza, E. Denamur, M. Radman, F. Taddel and I. Matic. 2003. Stress-induced mutagenesis in bacteria. Science 300: 1404–1409.

Boege, K. and R. J. Marquis. 2005. Facing herbivory as you grow up: the ontogeny of resistance in plants. Trends Ecol. Evol. 20: 441–448.

Bolnick, D. I. and B. M. Fitzpatrick. 2007. Sympatric speciation: models and empirical evidence. Annu. Rev. Ecol. Evol. Syst. 38: 459–487.

Boucias, D. G. and J. C. Pendland. 1998. *Principles of Insect Pathology*. Klewer, Boston.

Bradshaw, A. D. 1965. Evolutionary significance of phenotypic plasticity in plants. Advances in Genetics. 13: 115–155.

Bradshaw, W. E. and C. M. Holzapfel. 2001. Genetic shift in photoperiodic response correlated with global warming. Proc. Nat. Acad. Sci. 98: 14509–14511.

Bradshaw, W. E. and C. M. Holzapfel. 2006. Evolutionary response to rapid climate change. Science 312: 1477–1478.

Bradshaw, W. E. and C. M. Holzapfel. 2007. Evolution of animal photoperiodism. Annu. Rev. Ecol. Evol. Syst. 38: 1–25.

Brakefield, P. M. and V. French. 2006. Evo-devo focus issue. Heredity 97: 137–138.

Brodie III, E. D. and A. F. Agawal. 2001. Maternal effects and the evolution of aposematic signals. Proc. Natl. Acad. Sci. USA. 98: 7884–7887.

Bush, G. L. 1975. Sympatric speciation in phytophagous parasitic insects. pp. 187-206 *in* P. Price (ed.). *Evolutionary Strategies of Parasitic Insects and Mites*. Plenum, New York.

Calderwood, S. K., M. Y. Sherman and D. R. Ciocca (eds.). 2007. *Heat Shock Proteins in Cancer*. Springer, New York.

Carpenter, G. H. 1899. *Insects their Structure & Life*. J. M. Dent & Co., London.

ten Cate, C. 2000. How learning mechanisms might affect evolutionary processes. Trends Evol. Ecol. 15: 179–181.

Chapman, R. F. and J. C. Lee. 1991. Environmental effects on numbers of peripheral chemoreceptors on the antennae of a grasshopper. Chem. Senses 16: 607–616.

Collard, M. and S. J. Lycett. 2008. Does phenotypic plasticity confound attempts to identify hominin fossil species? Folia Primatologia 79: 111-122.

Collier, R. J., G. E. Dahl and M. J. VanBaale. 2006. Major advances associated with environmental effects on dairy cattle. J. Dairy Sci. 89: 1244–1253.

Cook, J. M., S. G. Compton, E. A. Herre and S. A. West. 1997. Alternative mating tactics and extreme male dimorphism in fig wasps. Proc. R. Soc. Lond. B 264: 747–754.

Cossins, A., J. Fraser, M. Hughes and A. Gracey. 2006. Post-genomic approaches to understanding the mechanisms of environmentally induced phenotypic plasticity. J. Exp. Biol. 209: 2328–2336.

Crispo, E. 2007. The Baldwin effect and genetic assimilation: revisiting two mechanisms of evolutionary change mediated by phenotypic plasticity. Evolution 61: 2469–2479.

Cronk, Q. C. B. 2005. Plant eco-devo: the potential of popular as a model organism. New Phytol. 166: 39–48.

Cullis, C. A. 1987. The generation of somatic and heritable variation in response to stress. Am. Nat. 130 (supplement): S62–S73.

Cullis, C. A. 1988. Control of variation in higher plants. pp. 49-62 *in* M-W. Ho and S. Fox (eds.). *Evolutionary Processes and Metaphors*. Wiley, Chichester, NY.

Czesak, M. E., C. W. Fox and J. B. Wolf. 2006. Experimental evolution of phenotypic plasticity: how predictive are cross-environment genetic correlations? Am. Nat. 168: 323–335.

Dambroski, H. R., C. Linn, S. H. Berlocher, A. A. Forbes, W. Roelofs and J. L. Feder. 2005. The genetic basis for fruit odor discrimination in *Rhagoletis* flies and its significance for sympatric host shifts. Evolution 59: 1953–1964.

Dana, M. D., J. A. Pintor-Toro and B. Cubero. 2006. Transgenic tobacco plants overexpressing chitinases of fungal origin show enhanced resistance to biotic and abiotic stress agents. Plant Physiol. 142: 722–730.

David, J. R., P. Gibert and B. Moreteau. 2004. Evolution of reaction norms. pp. 50-63 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic Plasticity: Functional and Conceptual Approaches*. Oxford University Press, Oxford.

Davis, J. M. and J. A. Stamps. 2004. The effect of natal experience on habitat preferences. Trends Ecol. Evol. 19: 411–416.

Day, R. L., K. N. Laland and J. Odling-Smee. 2003. Rethinking adaptation: the niche-construction perspective. Perspectives in Biology and Medicine 46: 80–95.

DeAngelis, D. L and W. M. Mooij. 2005. Individual-based modeling of ecological and evolutionary processes. Annu. Rev. Ecol. Evol. Syst. 36: 147–168.

Debat, V. and P. David. 2001. Mapping phenotypes: canalization, plasticity and developmental stability. Trends Ecol. Evol. 16: 555–561.

De Coen, W., J. Robbens and C. Janssen. 2006. Ecological impact assessment of metallurgic effluents using in situ biomarker assays. Environmental Pollution. 141: 283–294.

Defranco, A. L., R. M. Locksley and M. Robertson. 2007. *Immunity*. New Science Press, London.

De Moraes, C. M., J. C. Schultz, M. C. Mescher and J. H. Tumlinson. 2004. Induced plant signaling and its implications for environmental sensing. J. Toxicol. Environ. Health 67: 819–834.

DeWitt, T. J. and R. B. Langerhans. 2004. Integrated solutions to environmental heterogeneity. pp. 98-111 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic Plasticity: Functional and Conceptual Approaches.* Oxford University Press, Oxford.

DeWitt, T. J. and S. M. Scheiner (eds.). 2004. *Phenotypic Plasticity: Functional and Conceptual Approaches*. Oxford Univ. Press, Oxford.

DeWitt, T. J. and S. M. Scheiner. 2004. Phenotypic variation from single genotypes: a primer. pp. 1-9 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic plasticity: Functional and Conceptual Approaches*. Oxford University Press. Oxford.

DeWitt, T. J., A. Sih and D. S. Wilson. 1998. Costs and limits of phenotypic plasticity. Trends Ecol. Evol. 13: 77–81.

Dobzansky, T. 1951. *Genetics and the Origin of Species*. 3rd edition. Columbia Univ. Press, New York.

Doughty, P. and D. N. Reznick. 2004. Patterns and analysis of adaptive phenotypic plasticity in animals. pp. 126-150 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic Plasticity: Functional and Conceptual Approaches*. Oxford Univ. Press, Oxford.

Dukas, R. 2004. Evolutionary biology of animal cognition. Annu. Rev. Ecol. Syst. 35: 347–374.

Dunbar, H. E., A. C. C. Wilson, N. R. Ferguson and N. A. Moran. 2007. Aphid thermal tolerance is governed by a point mutation in bacterial symbionts. PLoS Biol. 5: 1006–1015.

Edreva, A. 2004. A novel strategy for plant protection: induced resistance. Cell and Molecular Biology 3: 61–69

Ellers, J., J. Marien, G. Driessen and N. M. Van Straalen. 2008. Temperature-induced gene expression associated with different thermal norms for growth rate. J. Exp. Zool. B. 310: 137-147.

Ellison, A. M. and N. J. Gotelli. 2002. Nitrogen availability alters the expression of carnivory in the northern pitcher plant, *Sarracenia purpurea*. Proc. Natl. Acad. Sci. USA. 99: 4409–4412.

Emlen, D. J. and C. E. Allen. 2004. Genotype to phenotype: Physiological control of trait size and scaling in insects. Integr. Comp. Biol. 43: 617–634.

Emlen, D. J. and H. F. Nijhout. 2000. The development and evolution of exaggerated morphologies in insects. Annu. Rev. Entomol. 45: 661–708.

Emlen, D. J., Q. Szafran, L. S. Corley and I. Dworkin. 2006. Insulin signaling and limb-patterning: candidate pathways for the origin and evolutionary diversification of beetle 'horns.' Heredity 97: 179–191.

Emlen, D. J., L. C. Lavine and B. Ewen-Campen. 2007. On the origin and evolutionary diversification of beetle horns. Proc. Natl. Acad. Sci. USA 104: 8661–8668.

Engqvist, L. 2008. Genetic variance and genotype reaction norms in response to larval food manipulation for a trait important in scorpionfly sperm competition. Functional Ecology 22: 127–133.

Eshel, I. and C. Matessi. 1998. Canalization, genetic assimilation and preadaptation: a quantitative genetic model. Genetics 149: 2119–2133.

Evans, H. E. and M. J. West-Eberhard. 1970. *The Wasps*. University of Michigan Press, Ann Arbor.

Evans, J. D. and D. E. Wheeler. 1999. Differential gene expression between developing queens and workers in the honey bee, *Apis mellifera*. Proc. Natl. Acad. Sci. USA. 96: 5575–5580.

Feder, J. L., S. B. Opp, B. Wlazlo, K. Reynolds, W. Go and S. Spisak. 1994. Host fidelity is an effective premating barrier between sympatric races of the apple maggot fly, *Rhagoletis pomonella*. Proc. Nat. Acad. Sci. USA. 91: 7990–7994.

Feder, J. L., J. B. Roethele, B. Wlazlo and S. H. Berlocher. 1997. Selective maintenance of allozyme differences among sympatric host races of the apple maggot fly. Proc. Nat. Acad. Sci. USA. 94: 11417–11421.

Feder, M. E. 2007. Evolvability of physiological and biochemical traits: evolutionary mechanisms including and beyond single-nucleotide mutation. J. Exp. Biol. 210: 1653–1660.

Filchak, K. E., J. B. Roethele and J. L. Feder. 2000. Natural selection and sympatric divergence in the apple maggot *Rhagoletis pomonella*. Nature 407: 739–742.

Flor, M., P. Hammerstein and A. Telschow. 2007. *Wolbachia*-induced unidirectional cytoplasmic incompatibility and the stability of infection polymorphism in parapatric host populations. J. Evol. Biol. 20: 696–706.

Flück, M. 2006. Functional, structural and molecular plasticity of mammalian skeletal muscle in response to exercise stimuli. J. Exp. Biol. 209: 2239–2248.

Flynn, J. R. 1987. Massive IQ gains in 14 nations: what IQ tests really measure. Psychol. Bull. 101: 171–1191.

Fordyce, J. A. 2001. The lethal plant defense paradox remains: inducible host-plant aristolochic acids and the growth and defense of the pipevine swallowtail. Entomol. Exp. Appl. 100: 339–346.

Fordyce, J. A. 2006. The evolutionary consequences of ecological interactions mediated through phenotypic plasticity. J. Exp. Biol. 209: 2377–2383.

Fox, C. W. and M. E. Czesak. 2000. Evolutionary ecology of progeny size in arthropods. Annu. Rev. Entomol. 45: 341–369.

Franken, R. J., J. J. Gardeniers, J. A. Beijer and E. T. Peeters. 2008. Variation in stonefly (*Nemoura cinerea* Retzius) growth and development in response to hydraulic and substrate conditions. J. N. Am. Benthol. Soc. 27: 176-185.

Frankino, W. A. and R. A. Raff. 2004. Evolutionary importance and pattern of phenotypic plasticity. pp. 64-97 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic plasticity: Functional and Conceptual Approaches*. Oxford University Press. Oxford.

Fréchette, B., A. F. G. Dixon, C. Alauzet, and J-L. Hemptinne. 2004. Age and experience influence patch assessment for oviposition by an insect predator. Ecol. Entomol. 29: 578–583.

Frederickson, M. E., M. J. Greene and D. Gordon. 2005. 'Devil's gardens' bedevilled by ants. Nature 437: 495–496.

Freeman, S. and J. C. Herron. 2007. *Evolutionary Analysis*. Prentice Hall, Upper Saddle River, NJ.

Frost, S. D. W. 1999. The immune system as an inducible defense. pp. 104-126 *in* R. Tollrian and C. D. Harvell (eds.). *The Ecology and Evolution of Inducible Defenses*. Princeton University Press, Princeton, NJ.

Futuyma, D. J. 1998. *Evolutionary Biology*. Sinauer, Sunderland, MA.

Gabriel, W. 2005. How stress selects for reversible phenotypic plasticity. J. Evol. Biol 18: 873–883.

Gabriel, W. and M. Lynch. 1992. The selective advantage of reaction norms for environment tolerance. J. Evol. Biol. 5: 41–59.

Gabriel, W., B. Luttbeg, A. Sih and R. Tollrian. 2005. Environmental tolerance, heterogeneity, and the evolution of reversible plastic responses. Am. Nat. 166: 339–353.

Galloway, L. F. 2005. Maternal effects provide phenotypic adaption to local environmental conditions. New Phytol. 166: 93–100.

Gardner, S. N. and A. A. Agrawal. 2002. Induced plant defence and the evolution of counter-defences in herbivores. Evol. Ecol. Res. 4: 1131–1151.

Garland, T. Jr. and S. A. Kelly. 2006. Phenotypic plasticity and experimental evolution. J. Exp. Biol. 209: 2344–2361.

Geurts C. L. M., G. G. Sleivert and S. S. Cheung. 2006. Local cold acclimation during exercise and its effect on neuromuscular function of the hand. Physiologie Appliquee, Nutrition et Metabolisme. 31: 717–725.

Goldschmidt, R. 1935. Gen und Ausseneigenschaft, Z. Induktive Abstammungs Verbunglehre 69: 38–131.

Gorur, G., C. Lomonaco and A. Mackenzie. 2005. Phenotypic plasticity in host-plant specialization in *Aphis fabae*. Ecol. Entomol. 30: 657–664.

Grant, B. R. and P. R. Grant. 1996. Cultural inheritance of song and its role in the evolution of Darwin's finches. Evolution 50: 2471–2487.

Greene, E. 1989. A diet-induced developmental polymorphism in a caterpillar. Science 243: 643–646.

Grether, G. F. 2005. Environmental change, phenotypic plasticity, and genetic compensation. Am. Nat. 166: E115–E123.

Haga, K. and S. Okajima. 1975. Redescription and status of the genus *Phoxothrips* Karny (Thysanoptera). Kontyu. Tokyo. 42: 375–384.

Hairston, N. G. Jr., C. L. Holtmeier, W. Lampert, L. J. Weider, D. M. Post, J. M. Fischer, C. E. Cáceres, J. A. Fox, and U. Gaedke. 2001. Natural selection for grazer resistance to toxic cyanobacteria. Evolution 55: 2203–2214.

Haukioja, E. 1990. On the role of plant defences in the fluctuations of herbivore populations. Oikos 35: 202–213.

Hayashi, Y., N. Lo, H. Miyata and O. Kitade. 2007. Sex-linked genetic influence on caste determination in a termite. Science 318: 985–987.

Heil, M., S. Greiner, H. Meimberg, R. Krüger, J-L. Noyer, G. Heubl, K. E. Linsenmair and W. Boland. 2004. Evolutionary change from induced to constitutive expression of an indirect plant resistance. Nature 430: 205–208.

Heinrich, B. 1993. The Hot-Blooded Insects. Harvard University Press, Cambridge.

Heinze, J. 2004. Reproductive conflict in insect societies. Advances in the Study of Behavior. 34: 1–57.

Hendry, A. P., T. J. Farrugia, M. T. Kinnison. 2008. Human influences on rates of phenotypic change in wild animal populations. Molecular Ecology 17: 20–29.

Herrnstein, R. J. and C. Murray. 1994. The Bell Curve. Free Press Paperbacks, New York.

Hille Ris Lambers, D. 1966. Polymorphism in Aphididae. Annu. Rev. Entomol. 11: 47–78.

Hojo, M., S. Koshikawa, T. Matsumoto and T. Miura. 2004. Developmental pathways and plasticity of neuter castes in *Nasutitermes takasagoensis* (Isoptera: Termitidae). Sociobiology 44: 433–441.

Hölldobler, B. and E. O. Wilson. 1990. *The Ants*. Harvard University Press, Cambridge, MA.

Huey, R. B., P. E. Hertz and B. Sinervo. 2003. Behavioral drive versus behavioral inertia in evolution: a null model approach. Am. Nat. 161: 357–366.

Hunt, J. H. 2007. *The Evolution of Social Wasps*. Oxford University Press, New York.

Hunt, J. and L. W. Simmons. 2000. Maternal and paternal effects on offspring phenotype in the dung beetle *Onthophagus taurus*. Evolution 54: 936–941.

Hunt, J. H., B. J. Kensinger, J. A. Kossuth, M. T. Henshaw, K. Norberg, F. Wolschin and G. V. Amdam. 2007. A diapause pathway underlies the gyne phenotype in *Polistes* wasps, revealing an evolutionary route to caste-containing insect societies. Proc. Nat. Acad. Sci. 104: 14020–14025.

Hurd, H. and R. Lane (eds.). 1998. Parasite-insect interactions: reciprocal manipulation. Parasitology 116 (Supplement): 1–128.

Huxley, C. R. and D. F. Cutler. 1991. *Ant-Plant Interactions*. Oxford University Press, Offord.

Inbar, M., M. Wink and D. Wool. 2004. The evolution of host plant manipulation by insects: molecular and ecological evidence from gall-forming aphids on *Pistacia*. Mol. Phylogenet. Evol. 32: 504–511.

Ito, S. 2003. A chemist's view of melanogenesis. Pigment Cell Research 16: 230–236.

Jablonka, E., M. Lamb and E. Avital. 1998.'Lamarckian' mechanisms in Darwinian evolution. Trends Ecol. Evol. 13: 206–210.

Jablonka, E. and M. J. Lamb. 2005. Evolution in Four Dimensions: Genetic, Epigenetic, Behavioral, and Symbolic Variation in the History of Life. MIT Press, Cambridge, MA.

Järemo J., J. Tuomi and P. Nilsson. 1999. Adaptive status of localized and systemic defense response in plants. pp. 33-44 in Tollrian R and Harvell CD (eds.). The Ecology and Evolution of Inducible Defenses. Princeton University Press, Princeton, NJ.

Johnson, L. K. 1982. Sexual selection in a brentid weevil. Evolution 36: 251–262.

Jones, A. G., S. J. Arnold and R. Bürger. 2007. The mutation matrix and the evolution of evolvability. Evolution 61: 727–745.

de Jong, G. 2005. Evolution of phenotypic plasticity: patterns of plasticity and the emergence of ecotypes. New Phytol. 166: 101–118.

de Jong, G. and N. Behera. 2002. The influence of life-history differences on the evolution of reaction norms. Evol. Ecol. Res. 4: 1–25.

de Jong G. and P. Bijma. 2002. Selection and phenotypic plasticity in evolutionary biology and animal breeding. Livest. Prod. Sci. 78: 195–214.

de Jeong, G. T. and D. H. Park. 2005. Enhancement of growth and secondary metabolite biosynthesis: effect of elicitors derived from plants and insects. Biotechnol. Bioprocess Eng. 10: 73–77.

Kappers, I. F., A. Aharoni, T. W. J. M. van Herpen, L. L. P. Luckerhoff, M. Dicke and H. J. Bouwmeester. 2005. Genetic engineering of terpenoid metabolism attracts bodyguards to *Arabidopsis*. Science 309: 2070–2072, 2067.

Karban, R. and K. Nagasaka. 2004. Are defenses of wild radish populations well matched with variability and predictability of herbivory? Evol. Ecol. 18: 283–301.

Karban, R., A. A. Agrawal, J. S. Thaler and L. S. Adler. 1999. Induced plant responses and information content about risk of herbivory. Trends Ecol. Evol. 14: 443–447.

Karban R., M. Huntzinger and A. C. McCall. 2004. The specificity of eavesdropping on sagebrush by other plants. Ecology 85: 1846–1852.

Kenyon, S. G. and M. S. Hunter. 2007. Manipulation of oviposition choice of the parasitoid wasp, *Encarsia pergandiella*, by the endosymbiotic bacterium *Cardinium*. J. Evol. Biol. 20: 707–716.

Key, K. H. L. and M. F. Day. 1954a. A temperature-controlled physiological colour response in the grasshopper, *Kosciuscola tristis* Sjöst. (Orthoptera, Acrididae). Australian J. Zool. 2: 309–339.

Key, K. H. L. and M. F. Day.1954b. The physiological mechanism of colour change in the grasshopper, *Kosciuscola tristis* Sjöst. (Orthoptera, Acrididae). Australian J. Zool. 2: 340–363.

Kidwell, M. G. and D. R. Lisch. 2001. Perspective: transposable elements, parasitic DNA, and genome evolution. Evolution 55: 1–24.

Kirschner, M. W. and J. C. Gerhart. 2005. *The Plausibility of Life: Resolving Darwin's Dilemma*. Yale University Press, New Haven.

Klausnitzer, B. 1987. *Insects their Biology and Cultural History*. Universe Books/Edition Leipzig, NY.

van Kleunen, M. and M. Fischer. 2005. Constraints on the evolution of adaptive phenotypic plasticity in plants. New Phytol. 166: 49–60.

Kliebenstein, D. J., A. Figuth and T. Mitchell-Olds. 2002. Genetic architecture of plastic methyl jasmonate responses in *Arabidopsis thaliana*. Genetics 161: 1685–1696.

de Kroon, H., H. Huber, J. Stuefer and J. M. van Groenendael. 2005. A modular concept of phenotypic plasticity in plants. New Phytol. 166: 73–82.

Landry, C. R., B. Lemos, S. A. Rifkin, W. J. Dickinson and D. L. Hartl. 2007. Genetic properties influencing the evolvability of gene expression. Science 317: 118–121.

Lee, S. M., S. B. Lee, C. H. Park and J. Choi. 2006. Expression of heat shock protein and hemoglobin genes in *Chrionomus tentans* (Diptera, Chironomidae) larvae exposed to various environmental pollutants: A potential biomarker of freshwater monitoring. Chemosphere. 65: 1074–1081.

Leonard, G. H., M. D. Bertness and P. O. Yund. 1999. Crab predation, waterborne cues, and inducible defenses in the blue mussel, *Mytilus edulis*. Ecology 80: 1–14.

Lessells, C. M. 2008. Neuroendocrine control of life histories: what do we need to know to understand the evolution of phenotypic plasticity? Phil. Trans. R. Soc. Lond. B 363: 1589-1598.

Levins, R. 1968. Evolution in Changing Environments. Princeton University Press, Princeton.

Li, W. W. and W. Barz. 2005. Biotechnological production of two new 8,4'-oxynorneolignans by elicitation of *Echinacea purpurea* cell cultures. Tetrahedron Lett. 46: 2973–2977.

Li, X., M. A. Schuler and M. R. Berenbaum. 2002. Jasmonate and salicylate induce expression of herbivore cytochrome P450 genes. Nature 419: 712–715.

Lindström, L., R. V. Alatalo, A. Lyytinen and J. Mappes. 2001. Strong antiapostatic selection against novel rare aposematic prey. Proc. Natl. Acad. Sci. USA 98: 9181–9184.

Luczynski A, J. P. Nyrop and A. Shi. 2007. Influence of cold storage on pupal development and mortality during storage and on post-storage performance of *Encarsia formosa* and *Eretmocerus eremicus* (Hymenoptera : Aphelinidae). Biological Control. 40: 107–117.

Lyytinen, A., P. M. Brakefield, L. Lindström and J. Mappes. 2004. Does predation maintain eyespot plasticity in *Bicyclus anynana*? Proc. R. Soc. Lond. B 271: 279–283.

Marinovic, B. and M. Mangel. 1999. Krill can shrink as an ecological adaptation to temporarily unfavourable environments. Ecol. Lett. 2: 338–343.

Marx, J. L. 1984. Instability in plants and the ghost of Lamarck. Science 224: 1415–1416.

Masel, J. 2005. Evolutionary capacitance may be favored by natural selection. Genetics 170: 1359–1371.

May, M. L. 1984. Thermoregulation. pp. 507-552 *in* G. Kerkut and L. Gilbert (eds.). *Comprehensive Insect Physiology Biochemistry and Pharmacology*, Vo. 4. Pergamon Press, Oxford.

Mayr, E. 1963. *Animal Species and Evolution*. Belknap Press, Cambridge, MA.

McAlister, J. S. 2007. Egg size and the evolution of phenotypic plasticity in larvae of the echinoid genus *Strongylocentrotus*. Exp. Marine Biol. Ecol. 352: 306–316.

McCaffery, A. R., S. J. Simpson, M. S. Islam, P. Roessingh. 1998. A gregarizing factor present in the egg pod foam of the desert locust *Schistocerca gregaria*. J. Exp. Biol. 201: 347–363.

McKenzie, G. J. and S. M. Rosenberg. 2001. Adaptive mutations, mutator DNA polymerases and genetic change strategies of pathogens. Current Opinion in Microbiology 4: 586–594.

McLeod, L. 2007. Further investigations of the effect of low temperatures on the phenotype of the adults of *Precis octavia* (Cramer) (Lepidoptera: Nymphalidae). Metamorphosis 18: 48–55.

Meiklejohn, C. D. and D. L. Hartl. 2002. A single mode of canalization. Trends Ecol. Evol. 17: 468–473.

Meiners, T. and M. Hilker. 2000. Induction of plant synomones by oviposition of a phytophagous insect. J. Chem. Ecol. 26: 221–232.

Meyers, L. A. and J. J. Bull. 2002. Fighting change with change: adaptive variation in an uncertain world. Trends Ecol. Evo. 17: 551–557.

Meyers, L. A., F. D. Ancel and M. Lachmann. 2005. Evolution of genetic potential. PLoS Comput. Biol. 1: 236–243.

Miller, G. A., M. S. Islam, T. D. Claridge, T. Dodgson and S. J. Simpson. 2008. Swarm formation in the desert locust *Schistocerca gregaria*: isolation and NMR analysis of the primary maternal gregarizing agent. J. Exp. Biol. 211: 370-376.

Miner, B. G., S. E. Sultan, S. G. Morgan, D. K. Padilla and R. A. Relyea. 2005. Ecological consequences of phenotypic plasticity. Trends Ecol. Evol. 20: 685–692.

Minks, A. K. and P. Harrewijn. 1987. *Aphids their Biology, Natural Enemies and Control*. Elsevier, Amsterdam.

Mirth, C. K. and L. M. Riddiford. 2007. Size assessment and growth control: how adult size is determined in insects. BioEssays 29: 344–355.

Miura, T. 2004. Proximate mechanisms and the evolution of caste polyphenism in social insects: From sociality to genes. Ecol. Res. 19: 141–148.

Miura, T. 2005. Developmental regulation of caste-specific characters in social-insect polyphenism. Evolution & development 7: 122–129.

Miura, T. and T. Matsumoto. 1995. Worker polymorphism and division of labor in the foraging behavior of the black marching termite *Hospitalitermes medioflavus*, on Borneo Island. Naturwissenschaften 82: 564–567.

Miura, T. and T. Matsumoto. 2000. Soldier morphogenesis in a nasute termite: discovery of a disk-like structure forming a soldier nasus. Proc. R. Soc. Lond. B 267: 1185–1189.

Miura, T., Y. Roisin and T. Matsumoto. 1998. Developmental pathways and polyethism of neuter castes in the processional nasute termite *Hospitalitermes medioflavus* (Isoptera: Termitidae). Zoo. Sci. 15: 843–848.

Moczek, A. P. and F. H. Nijhout. 2003. Rapid evolution of a polyphenic threshold. Evol. Dev. 5: 259–268.

Mondor, E. B., J. A. Rosenheim and J. F. Addicott. 2005. Predator-induced transgenerational phenotypic plasticity in the cotton aphid. Oecologia 142: 104–108.

Mooney, K. A. and A. A. Agrawal. 2007. Phenotypic plasticity. pp. 43-57 *in* K. J. Tilmon (ed.). *The evolutionary biology of herbivorous insects: Specialization, speciation, and radiation*. University of California Press, Berkeley, CA.

Moran, N. A. 1992. The evolutionary maintenance of alternative phenotypes. Am. Nat. 139: 971–989.

Mound, L. A. 2005. Fighting, flight and fecundity: Behavioural determinants of Thysanoptera structural diversity. pp. 81-105 *in* T. N. Ananthakrishnan and D. W. Whitman (eds.). *Insect Phenotypic Plasticity.* Science Publishers, Enfield, NH, USA.

Mousseau, T. A. and H. Dingle. 1991. Maternal effects in insect life histories. Annu. Rev. Entomol. 36: 511–534.

Mousseau, T. A. and C. W. Fox (eds.). 1998. *Maternal Effects as Adaptation.* Oxford University Press, Oxford.

Munday, P. L., P. M. Buston and R. R. Warner. 2006. Diversity and flexibility of sex-change strategies in animals. Trends Ecol. Evol. 21: 89–95.

Muth, N. Z. and M. Pigliucci. 2007. Implementation of a novel framework for assessing species plasticity in biological invasions: responses of *Centaurea* and *Crepis* to phosphorus and water availability. Ecology 95: 1001–1013.

Nadeau, J. H. and E. J. Topol. 2006. The genetics of health. Nat. Genet. 38: 1095–1098.

Nakamura, M. and T. Ohgushi. 2003. Positive and negative effects of leaf shelters on herbivorous insects: linking multiple herbivore species on a willow. Oecologia 136: 445–449.

Neufeld, C. J. and A. R. Palmer. 2008. Precisely proportioned: intertidal barnacles alter penis form to suit costal wave action. Phil. Trans. R. Soc. Lond. B. 275: 1081-1087.

Nijhout, H. F. 1991. *The Development and Evolution of Butterfly Wing Patterns*. Smithsonian Institution Press, Washington, D.C.

Nijhout, H. F. 1994. *Insect Hormones*. Princeton Univ. Press, Princeton.

Nijhout, H. F. 1999. Control mechanisms of polyphenic development in insects. BioScience 49: 181–192.

Nijhout, H. F. 2003a. The development and evolution of adaptive polyphenisms. Evolution & Development 5: 9–18.

Nijhout, H. F. 2003b. The control of body size in insects. Developmental Biology 261: 1–9.

Nolte, D. J. 1974. The gregarization of locusts. Biol. Rev. Cambridge Philos. Soc. 49: 1–14.

Nortman, D. 2003. The evolution of phenotypic plasticity through the Baldwin Effect. Noesis 6: 1–13.

Nunney, L. and W. Cheung. 1997. The effect of temperature on body size and fecundity in female *Drosophila melanogaster*: evidence for adaptive plasticity. Evolution 51: 1529–1535.

Nykänen, H. and J. Koricheva. 2004. Damage-induced changes in woody plants and their effects on insect herbivore performance: a meta-analysis. Oikos 104: 247–268.

Nylin, S. and K. Gotthard. 1998. Plasticity in life-history traits. Annu. Rev. Entomol. 43: 63–93.

Odling-Smee, F. J., K. N. Laland and M. W. Feldman. 2003. Niche construction: the neglected process in evolution. Monographs in Population Biology 37. Princeton University Press, Princeton.

O'Donnell, S. 1998. Reproductive caste determination in eusocial wasps (Hymenoptera: Vespidae). Annu. Rev. Entomol. 43: 323–346.

Ohgushi T. 2005. Indirect interaction webs: herbivore-induced effects through trait change in plants. Annu. Rev. Ecol. Evol. Syst. 36: 81–105.

Ohgushi, T., T. P. Craig and P. Price (eds.). 2007. *Ecological Communities: Plant Mediation in Indirect Interaction Webs.* Cambridge University Press, Cambridge.

Oishi, M. and H. Sato. 2007. Inhibition of premature leaf abscission by a leafminer and its adaptive significance. Environ. Entomol. 36: 1504–1511.

O'Neill, S. L., A. A. Hoffmann and J. H. Werren (eds.). 1997. Influential Passengers: Inherited Microorganisms and Arthropod Reproduction. Oxford University Press, New York.

Oster, G. F. and E. O. Wilson. 1978. *Caste and Ecology in the Social Insects.* Princeton University Press, New Jersey.

Otaki, J. M. 2007. Stress-induced color-pattern modifications and the evolution of the painted lady butterflies *Vanessa cardui* and *Vanessa kershawi.* Zoological Science 24: 811–819.

Otaki, J. M. 2008. Phenotypic plasticity of wing color patterns revealed by temperature and chemical applications in a nymphalid butterfly *Vanessa indica.* 2008. J. Thermal Biol. 33: 128-139.

Otte, D. and K. Stayman. 1979. Beetle horns: some patterns in functional morphology. pp. 159-292 *in* M. Blum and N. Blum (eds.). *Sexual Selection and Reproductive Competition in Insects.* Academic Press, New York.

Padilla, D. K. and S. C. Adolph. 1996. Plastic inducible morphologies are not always adaptive: the importance of time delays in a stochastic environment. Evol. Ecol. 10: 105–117.

Page, R. E. Jr. and G. V. Amdam. 2007. The making of a social insect: developmental architectures of social design. BioEssays 29: 334–343.

Palmer, J. M. and L. A. Mound. 1978. Nine genera of fungus-feeding *Phlaeothripidae* (Thysanoptera) from the Oriental Region. Bull. British Mus. (Nat. History). Ent. 37: 153–215.

Parsons, K. J. and B. W. Robinson. 2006. Replicated evolution of integrated plastic responses during early adaptive divergence. Evolution 60: 801–813.

Peacor, S. D., S. Allesina, R. L. Riolo and M. Pascual. 2006. Phenotypic plasticity opposes species invasions by altering fitness surface. PLoS Biology 4(11) e372: 1–21.

Peeters, C. and S. Higashi. 1989. Reproductive dominance controlled by mutualition in the queenless ant *Diacamma australe.* Naturwissenschaften 76: 177–180.

Pereira R, N. Silva, C. Quintal, R. Abreu, J. Andrade and L. Dantas. 2007. Effect of acclimation to outdoor conditions on the sexual performance of mass-produced medflies (Diptera : Tephritidae). Florida Entomologist. 90: 171–174.

Pfenning, K. S. 2007. Facultative mate choice drives adaptive hybridization. Science 318: 965–967.

Pienaar, J. and J. M. Greeff. 2003a. Different male morphs of *Otitesella pseudoserrata* fig wasps have equal fitness but are not determined by different alleles. Ecology Letters 6: 286–289.

Pienaar, J. and J. M. Greeff. 2003b. Maternal control of offspring sex and male morphology in the *Otitesella* fig wasps. J. Evol. Biol. 16: 244–253.

Piersma, T. and J. Drent. 2003. Phenotypic flexibility and the evolution of organismal design. Trends in Ecology and Evolution 18: 228–233.

Pigliucci, M. 2001. Phenotypic Plasticity: Beyond Nature and Nurture. Johns Hopkins University Press, Baltimore, MD.

Pigliucci, M. 2005. Evolution of phenotypic plasticity: where are we going now? Trends Ecol. Evol. 20: 481–486.

Pigliucci, M. 2007. Do we need an extended evolutionary synthesis? Evolution 61: 2743–2749.

Pigliucci, M. and C. J. Murren. 2003. Genetic assimilation and a possible evolutionary paradox: can macroevolution sometimes be so fast as to pass us by? Evolution 57: 1455–1464.

Pigliucci, M. and K. Preston. 2004. Phenotypic Integration. Oxford University Press, Oxford.

Pigliucci, M., C. J. Murren and C. D. Schlichting. 2006. Phenotypic plasticity and evolution by genetic assimilation. J. Exp. Biol. 209: 2362–2367.

Pijanowska, J., P. Bernatowicz and J. Fronk. 2007. Phenotypic plasticity within *Daphnia longispina* complex: differences between parental and hybrid clones. Polish J. Ecol. 55: 761–769.

Poinar, G. and S. P. Yanoviak. 2008. *Myrmeconema neotropicum* n. g. sp., a new tetradonematid nematode parasitizing South American populations of *Cephalotes atratus* (Hymenoptera: Formicidae), with the discovery of an apparent parasite-induced host morhp. Systematic Parasitolgy 69: 145–153.

Poulev, A., J. M. O'Neal, S. Logendra, R. B. Pouleva, V. Timeva, A. S. Garvey, D. Gleba, I. S. Jenkins, B. T. Halpern, R. Kneer, G. M. Cragg and I. Raskin. 2003. Elicitation, a new window into plant chemodiversity and phytochemical drug discovery. J. Med. Chem. 46: 2542–2547.

Premnath, S. A. Sinha and R. Gadagkar. 1996. Dominance relationship in the establishment of reproductive division of labour in a primitively eusocial wasp (*Ropalidia marginata*). Behav. Ecol. Sociobiol. 39: 125–132.

Price, T. D. 2006. Phenotypic plasticity, sexual selection and the evolution of color patterns. J. Exp. Biol. 209: 2368–2376.

Price, T. D., A. Qvarnstöm and D. E. Irwin. 2003. The role of phenotypic plasticity in driving genetic evolution. Proc. R. Soc. B 270: 1433–1440.

Prichard, I. M. and R. James. 1984. Leaf mines: their effect on leaf longevity. Oecologia 64: 132–139.

Prokopy, R. J., S. R. Diehl and S. S. Cooley. 1988. Behavioral evidence for host races in *Rhagoletis pomonella* flies. Oecologia 76: 138–147.

Qvarnström, A. and T. D. Price. 2001. Maternal effects, paternal effects and sexual selection. Trends Ecol. Evol. 16: 95–100.

Radakovic SS, Maric J, Surbatovic M, Radjen S, Stefanova E, Stankovic N and Filipovic N 2007. Effects of acclimation on cognitive performance in soldiers during exertional heat stress. Military Medicine. 172: 133–136.

Radman, M. 1999. Enzymes of evolutionary change. Nature 401: 866–869.

Reimer, O. and M. Tedengren. 1996. Phenotypical improvement of morphological defences in the mussel *Mytilus edulis* induced by exposure to the predator *Asterias rubens*. Oikos 75: 383–390.

Relyea, R. A. 2003a. Predator cues and pesticides: a double dose of danger for amphibians. Ecol. Appl. 13: 1515–1521.

Relyea, R. A. 2003b. How prey respond to combined predators: a review and an empirical test. Ecology 84: 1827–1839.

Relyea, R. A. 2003c. Predators come and predators go: the reversibility of predator-induced traits. Ecology 84: 1840–1848.

Relyea, R. A. 2004a. Integrating phenotypic plasticity when death is on the line. pp. 176-190 *in* M. Pigliucci and K. Preston (eds.). *Phenotypic Integration*. Oxford University Press, Oxford.

Relyea, R. A. 2004b. Fine-tuned phenotypes: tadpole plasticity under 16 combinations of predators and competitors. Ecology 85: 172–179.

Releya, R. A. and J. Hoverman. 2006. Assesing the ecology in exotoxicology: a review and synthesis in freshwater systems. Ecol. Let. 9: 1157–1171.

Richards, C. L., O. Bossdorf, N. Z. Muth, J. Gurevitch and M. Pigliucci. 2006. Jack of all trades, master of some? On the role of phenotypic plasticity in plant invasions. Ecol. Lett. 9: 981–993.

Ricklefs, R. E. and M. Wikelski. 2002. The physiology/life-history nexus. Trends Ecol. Evol. 17: 462–468.

Riegler, M. and S. L. O'Neill. 2007. Evolutionary dynamics of insect symbiont associations. Trends Ecol. Evol. 22: 625–627.

Risch, S. J. and F. R. Rickson. 1981. Mutualism in which ants must be present before plants produce food bodies. Nature 291: 149–150.

Roder, G., M. Rahier and R. E. Naisbit. 2008. Counter-intuitive developmental plasticity induced by host quality. Phil. Trans. R. Soc. Lond. B. 275: 879-885.

Rodríguez, R. L., L. M. Sullivan, R. L. Snyder and R. B. Cocroft. 2007. Host shifts and the beginning of signal divergence. Evolution 62: 12–20.

Roelofs, D., M. G. M. Aarts, H. Schat and N. M. Van Straalen. 2007. Functional ecological genomics to demonstrate general and specific responses to abiotic stress. Funct. Ecol. 21: 1–12.

Roff, D. A. 1996. The evolution of threshold traits in animals. Quarterly Rev. Biol. 71: 3–35.

Rogers, S. M. and S. J. Simpson. 1997. Experience-dependent changes in the number of chemosensory sensilla on the mouthparts and antennae of *Locusta migratoria*. J. Exp. Biol. 200: 2313–2321.

Rountree, D. B. and H. F. Nijhout. 1994. Tenetic control of a seasonal morph in *Precis coenia* (Lepidoptera: Nymphalidae). J. Insect Physiol. 41: 1141–1145.

Rowell, C. H. F. 1971. The variable coloration of the Acridoid grasshoppers. Adv. Insect Physiol. 8: 145–198.

Roy, H. E., D. C. Steinkraus, J. Eilenberg, A. E. Hajek and J. K. Pell. 2006. Bizarre interactions and endgames: entomopathogenic fungi and their arthropod hosts. Annu. Rev. Entomol. 51: 331–357.

Saint-Ruf, C. and I. Matic. 2006. Environmental tuning of mutation rates. Environ. Microbiol. 8: 193–199.

Salt, G. 1937. The egg-parasite of *Sialis lutaria*: a study of the influence of the host upon a dimorphic parasite. Parasitology 29: 539–553.

Sarkar, S. 2004. From the *Reaktionsnorm* to the evolution of adaptive plasticity a historical sketch, 1909-1999. pp. 10-30 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic plasticity: Functional and Conceptual Approaches*. Oxford University Press. Oxford.

Scheiner, S. M. 1993. Genetics and evolution of phenotypic plasticity. Annu. Rev. Ecol. Syst. 24: 35–68.

Scheiner, S. M. 2002. Selection experiments and the study of phenotypic plasticity. J. Evol. Biol. 15: 889–898.

Scheiner, S. M. and R. F. Lyman. 1991. The genetics of phenotypic plasticity. II. responses to selection. J. Evol. Biol. 4: 23–50.

Schlichting, C. D. 2004. The role of phenotypic plasticity in diversification. pp. 191-200 *in* T. DeWitt and S. Scheiner (eds.). Phenotypic plasticity: functional and conceptual approaches. Oxford Univ. Press, Oxford.

Schlichting, C. D. and H. Smith. 2002. Phenotypic plasticity: linking molecular mechanisms with evolutionary outcomes. Evol. Ecol. 16: 189–211.

Schlichting, C. D. and M. Pigliucci. 1998. Phenotypic Evolution a Reaction Norm Perspective. Sinauer Associates, Sunderland, MA.

Schmalhausen, I. I. 1949. Factors of Evolution. Blakiston, Philadelphia.

Schmid-Hempel, P. 2005. Evolutionary ecology of insect immune defenses. 2005. Annu. Rev. Entomol. 50: 529–551.

Schmitz, O. J. 2006. Predators have large effects on ecosystem properties by changing plant diversity, not plant biomass. Ecology 87: 1432–1437.

Schmitz, O. J., V. Krivan and O. Ovadia. 2004. Trophic cascades: the primacy of trait-mediated indirect interactions. Ecol. Lett. 7: 153–163.

Schneirla, T. C. and H. R. Topoff. 1971. *Army Ants A Study in Social Organization.* W. H. Freeman and Company, San Francisco.

Seger, J. and H. J. Brockmann. 1987. What is bet-hedging? pp. 182-211 *in* P. Harvey and L. Partridge (eds.). Oxford Surveys in Evolutionary Biology 4. Oxford University Press, Oxford.

Sheldon, B. C. 2000. Differential allocation: tests, mechanisms and implications. Trends Ecol. Evol. 15: 397–402.

Sherry, D. F. and B. G. Galef. 1990. Social learning without imitation: more about milk bottle opening by birds. Anim. Behav. 40: 987–989.

Shibao, H., M. Kutsukake and T. Fukatsu. 2003. Density triggers soldier production in a social aphid. Proc. R. Soc. Lond. B (Suppl.) (Biology Letters). 03BL0217: S1-S4.

Shibao, H., M. Kutsukake and T. Fukatsu. 2004. Density-dependent induction and suppression of soldier differentiation in an aphid social system. J. Insect Physiol. 50: 995–1000.

Shingleton, A. W. and W. A. Foster. 2000. Ant tending influences soldier production in a social aphid. Proc. R. Soc. Lond. B 267: 1863–1868.

Shingleton, A. W., W. A. Frankino, T. Flatt, H. F. Nijhout and D. J. Emlen. 2007. Size and shape: the developmental regulation of static allometry in insects. BioEssays 29: 536–548.

Shiu, S. H. and J. O. Borevitz. 2008. The next generation of microarray research: applications in evolutionary and ecological genomics. Heredity 100: 141–149.

Sih, A. 2004. A behavioral ecological view of phenotypic plasticity. pp. 112-125 *in* T. DeWitt and S. Scheiner (eds.). *Phenotypic Plasticity*. Oxford University Press, Oxford.

Simmons, L. W., D. J. Emlen and J. L. Tomkins. 2007. Sperm competition games between sneaks and guards: a comparative analysis using dimorphic male beetles. Evolution 61: 2684–2692.

Simpson, S. J., G. A. Sword and A. De Loof. 2005. Advances, controversies and consensus in locust phase polyphenism research. J. Orthopt. Res. 14: 213–222.

Skaife, S. H. 1954. The black-mound termite of the Cape, *Amitermes atlanticus* Fuller. Trans. R. Soc. S. Afr. 34: 251–272.

Slabbekoorn, H. and T. B. Smith. 2002. Bird song, ecology and speciation. Philos. Trans. R. Soc. Lond. B 357: 493–503.

Slabber S, M. R. Worland, H. P. Leinaas and S. L. Chown. 2007. Acclimation effects on thermal tolerances of springtails from sub-Antarctic Marion Island: Indigenous and invasive species. J. Insect Physiol. 53: 113–125.

Smith, L. D. and A. R. Palmer. 1994. Effects of manipulated diet on size and performance of brachyuran crab claws. Science 264: 710–712.

Smith-Gill, S. J. 1983. Developmental plasticity: developmental conversion versus phenotypic modulation. Am Zool. 23: 47–55.

Song, H. 2005. Phylogenetic perspectives on the evolution of locust phase polyphenism. J. Orthopt. Res. 14: 235–245.

Stearns, S. C. 1992. *The Evolution of Life Histories*. Oxford Univ. Press, Oxford.

Stelgenga, M. J. and K. Fischer. 2007. Within- and between-generation effects of temperature on life-history traits in a butterfly. J. Thermal Biol. 32: 396–405.

Stephen, W. P. 2005. A serendipitous observation of a massive kill of the soil nesting bee, *Nomia melanderi* Cockerell (Hymenoptera: Apoidea: Halictidae). Pan-Pac. Entomol. 81: 181–183.

Stern, D. L. and W. A. Foster. 1996. The evolution of soldiers in aphids. Biol. Rev. 71: 27–76.

Strauss, S. Y. and R. E. Irwin. 2004. Ecological and evolutionary consequences of multispecies plant-animal interactions. Annu. Rev. Ecol. Evol. Syst. 35: 435–466.

Sugumaran, M. 2002. Comparative biochemistry of eumelanogenesis and the protective roles of phenoloxidase and melanin in insects. Pigm. Cell Res. 15: 2–9.

Sultan S. E. 2005. An emerging focus on plant ecological development. New Phytol. 166: 1–5.

Sultan S. E. 2007. Development in context: the timely emergence of eco-devo. Trends Eco. Evol. 22: 575–582.

Sultan S. E. and H. G. Spencer. 2002. Metapopulation structure favors plasticity over local adaptation. Am. Nat. 160: 271–283.

Suzuki, Y. and F. Nijhout. 2006. Evolution of a polyphenism by genetic accommodation. Science 311: 650–652.

Swallow, J. G., J. S. Rhodes and T. Garland, Jr. 2005. Phenotypic and evolutionary plasticity of organ masses in response to voluntary exercise in house mice. Integr. Comp. Biol. 45: 426–437.

Sword, G. A. 2002. A role for phenotypic plasticity in the evolution of aposematism. Proc. R. Soc. Lond. B. 269: 1639–1644.

Swynghedauw, B. 2006. Phenotypic plasticity of adult myocardium. Molecular mechanisms. J. Exp. Biol. 209: 2320–2327.

Takabayashi, J., S. Takahashi, M. Dicke and M. A. Posthumus. 1995. Developmental stage of herbivore *Pseudaletia separate* affects production of herbivore-induced synomone by corn plants. J. Chem. Ecol. 21: 273–287.

Tanaka, S. 2004. Environmental control of body-color polyphenism in the American Grasshopper, *Schistocerca americana*. Ann. Entomol. Soc. Am. 97: 293–301.

Thompson, D. B. 1992. Consumption rates and the evolution of diet-induced plasticity in the head morphology of *Melanoplus femurrubrum* (Orthoptera: Acrididae). Oecologia 89: 204–213.

Thompson, J. N. 1988. Variation in interspecific interactions. Annu. Rev. Ecol. Syst. 19: 65–87.

Thompson, J. N. 2005. The Geographic Mosaic of Coevolution. University of Chicago Press, Chicago.

Tollrian, R. and C. D. Harvell (Eds). 1999. The Ecology and Evolution of Inducible Defenses. Princeton, NJ: Princeton University Press.

Tomkins, J. L. 1999. Environmental and genetic determinants of the male forceps length dimorphism in the European earwig *Forficula auricularia* L. Behav. Ecol. Sociobiol. 47: 1–8.

Tomkhins, J. L. and L. W. Simmons. 1996. Dimorphisms and fluctuating asymmetry in the forceps of male earwigs. J. Evol. Biol. 9: 753–770.

True, J. R. 2003. Insect melanism: the molecules matter. Trends Ecol. Evol. 18: 640–647.

Tufto, J. 2000. The evolution of plasticity and nonplastic spatial and temporal adaptations in the presence of imperfect environmental cues. Am. Nat. 156: 121–130.

Uvarov, B. 1966. Grasshoppers and Locusts. Vol. 1. Cambridge University Press, Cambridge.

Van Buskirk, J. 2002. A comparative test of the adaptive plasticity hypothesis: relationships between habitat and phenotype in anuran larvae. Am. Nat. 160: 87–102.

Van Buskirk, J. and M. Arioli. 2005. Habitat specialization and adaptive phenotypic divergence of anuran populations. J. Evol. Biol. 18: 596–608.

van Kleunen, M. and M. Fischer. 2005. Constraints on the evolution of adaptive phenotypic plasticity in plants. New Phytol. 166: 49–60.

Van Tienderen, P. H. 1997. Generalists, specialists, and the evolution of phenotypic plasticity in sympatric populations of distinct species. Evolution 51: 1372–1380.

Van Zandt, P. A. and A. A. Agrawal. 2004. Community-wide impacts of herbivore induced plant responses in milkweed (*Asclepias syriaca*). Ecology 85: 2616–2629.

Via, S., R. Gomulkiewicz, G. de Jong, S. M. Scheiner, C. D. Schlichting and P. H. Van Tienderen. 1995. Adaptive phenotypic plasticity: consensus and controversy. Trends Ecol. Evol. 10: 212–217.

Visser, M. E., L. J. M. Holleman and P. Gienapp. 2006. Shifts in caterpillar biomass phenology due to climate change and its impact on the breeding biology of an insectivorous bird. Oecologia 147: 164–172.

Von Rad, U., M. J. Mueller and J. Durner. 2005. Evaluation of natural and synthetic stimulants of plant immunity by microarray technology. New Phytol. 165: 191–202.

Waddington, C. H. 1940. *Organisers and Genes.* Cambridge University Press, Cambridge.

Waddington, C. H. 1942. Canalisation of development and the inheritance of acquired characters. Nature 150: 563–565.

Waddington, C. H. 1952. Selection for the genetic basis of an acquired character. Nature 169: 278.

Waddington, C. H. 1953a Genetic assimilation of an acquired character. Evolution 7: 118–126.

Waddington, C. H. 1953b. The "Baldwin Effect," "genetic assimilation" and "homeostasis." Evolution 7: 386–387.

Waddington, C. H. 1956. Genetic assimilation of the *bithorax* phenotype. Evolution 7: 1–13.

Waddington, C. H. 1959. Canalisation of development and genetic assimilation of acquired characters. Nature 183: 1654–1655.

Waddington, C. H. 1961. Genetic assimilation. Adv. Genet. 10: 257–290.

Wade, M. J. 2001. Infectious speciation. Nature 409: 675.

Webber, B. H. and D. J. Depew. 2003. Evolution and Learning: the Baldwin Effect Reconsidered. MIT Press, Cambridge.

Werner, E. E. and S. D. Peacor. 2003. A review of trait-mediated indirect interactions in ecological communities. Ecology 84: 1083–1100.

West-Eberhard, M. J. 1969. The social biology of polistine wasps. Misc. Publ. Mus. Zool., Univ. Michigan No. 140: 1–101.

West-Eberhard, M. J. 2003. Developmental Plasticity and Evolution. Oxford University Press, Oxford.

West-Eberhard, M. J. 2005. Developmental plasticity and the origin of species differences. Proc. Nat. Acad. Sci. USA. 102: 6543–6549.

Wheeler, W. M. 1910. *Ants*. Columbia University Press, New York.

Whitfield, J. 2007. Who's the queen? Ask the genes. Science318: 910–911.

Whitman, D. W. 1994. Plant bodyguards: mutualistic interactions between plants and the third trophic level. Pp. 207-248 *in* T. N. Ananthakrishnan (ed.). *Functional Dynamics of Phytophagous Insects*. Oxford & IBH Publishing, New Delhi.

Wilson, R. S. and C. E. Franklin. 2002. Testing the beneficial acclimation hypothesis. Trends Ecol. Evol. 17: 66–70.

Windig, J. J., C. G. F. De Kovel and G. De Jong. 2004. Genetics and mechanics of plasticity. Pp. 31-97 *in* T. J. DeWitt and S. M. Scheiner (eds.). *Phenotypic plasticity: Functional and Conceptual Approaches*. Oxford University Press, Oxford.

Winterhalter, W. E. and T. A. Mousseau. 2007. Patterns of phenotypic and genetic variation for the plasticity of diapause incidence. Evolution 61: 1520–1531.

Wolf, J. B., E. D. Brondie and M. J. Wade. 2004. The genotype-environment interaction and evolution when the environment contains genes. pp. 173-190 *in* T. DeWitt and S. Scheiner (eds.). Phenotypic Plasticity. Oxford University Press, Oxford.

Wolschin, F. and G. V. Amdam. 2007. Plasticity and robustness of protein patterns during reversible development in the honey bee (*Apis mellifera*). Analytical & Bioanalytical Chemistry 389: 1095–1100.

Woltereck, R. 1909. Weitere experimentelle Untersuchungen über Artveränderung, speziell über das Wesen quantitativer Artunterschiede bei Daphniden. Verh. D. Tsch. Zool. Ges. 1909: 110–172.

Yang, Y. and A. Joern. 1994. Gut size changes in relation to variable food quality and body size in grasshoppers. Funct. Ecol. 8: 36–45.

Zera, A. J. and L. G. Harshman. 2001. The physiology of life-history trade-offs in animals. Annu. Rev. Ecol. Syst. 32: 95–126.

Zhang, X. S. 2006. The phenotypic variance within plastic traits under migration-mutation-selection balance. Evolution 60: 1125–1136.

Zhao, J., L. C. Davis and R. Verpoorte. 2005. Elicitor signal transduction leading to production of plant secondary metabolites. Biotechnology Advances 23: 283–333.

Zhou X, M. R Tarver and M. E. Scharf. 2007. Hexamerin-based regulation of juvenile hormone-dependent gene expression underlies phenotypic plasticity in a social insect. Development 134: 601–610.