Overcoming maladaptive plasticity through plastic compensation

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Abstract Most species evolve within fluctuating environments, and have developed adaptations to meet the challenges posed by environmental heterogeneity. One such adaptation is phenotypic plasticity, or the ability of a single genotype to produce multiple environmentally-induced phenotypes. Yet, not all plasticity is adaptive. Despite the renewed interest in adaptive phenotypic plasticity and its consequences for evolution, much less is known about maladaptive plasticity. However, maladaptive plasticity is likely an important driver of phenotypic similarity among populations living in different environments. This paper traces four strategies for overcoming maladaptive plasticity that result in phenotypic similarity, two of which involve genetic changes (standing genetic variation, genetic compensation) and two of which do not (standing epigenetic variation, plastic compensation). Plastic compensation is defined as adaptive plasticity overcoming maladaptive plasticity. In particular, plastic compensation may increase the likelihood of genetic compensation by facilitating population persistence. We provide key terms to disentangle these aspects of phenotypic plasticity and introduce examples to reinforce the potential importance of plastic compensation for understanding evolutionary change [Current Zoology 59 (4): 526–536, 2013].

Keywords Homeostasis, Canalization, Ecological speciation, Standing genetic variation, Genetic compensation, Countergradient variation

Evolution often occurs within fluctuating environments, with species developing adaptations to meet the challenges posed by these different environmental conditions. One such adaptation is phenotypic plasticity, or the ability of a single genotype to produce multiple environmentally-induced phenotypic traits (for this and other definitions see Box 1). The past decade has seen a re-emergence of interest in the adaptive nature and evolutionary consequences of plasticity. For instance, theoretical and empirical work has demonstrated that individuals that can plastically adjust their phenotype to meet the demands of a new environment are likely to be more effective colonizers of new environments than non-plastic individuals [Plasticity-Mediated Population Persistence (PMPP), Baldwin, 1896; Yeh and Price, 2004; Pavey et al., 2010; Thibert-Plante and Hendry, 2010; Hahn et al., 2012]. Recent conceptual and empirical work has generated significant interest in the role of adaptive plasticity in facilitating adaptive phenotypic divergence, ecological speciation, and adaptive radiation (Agrawal, 2001; West-Eberhard, 2003; Ghalambor et al., 2007; Wund et al., 2008; Lande, 2009; Pfenning et al., 2010). Not all plasticity is adaptive, however. The evolutionary significance of maladaptive plasticity has received comparatively little attention (Ghalambor et al., 2007; Fitzpatrick, 2012), but such attention has resulted in insights about the causes of cryptic evolution (Conover and Schultz, 1995; Grether, 2005). Maladaptive plasticity can result in phenotypic divergence between populations residing in different environments, but can also lead to genetic or plastic strategies to reduce this phenotypic divergence. Here we review the causes of phenotypic similarity, despite initial phenotypic divergence via maladaptive plasticity, and present the concept and significance of plastic compensation, defined as adaptive plasticity overcoming maladaptive plasticity.

1 Maladaptive Plasticity

Maladaptive plasticity, also referred to in the literature as non-adaptive plasticity, can be defined as the plastic response to changes in the environment, such that there is increased divergence between the expressed trait and the phenotypic optimum. From an evolutionary perspective, maladaptive plasticity can be defined as occurring when alleles that confer plasticity are less likely to spread through the population than alleles that confer reduced plasticity. Maladaptive plasticity is typically considered for a set of environments with identical
Box 1  Definitions

**Compensated phenotype:** In plastic compensation, the phenotype that expresses maladaptive plasticity in the absence of a compensating phenotype. In the presence of a compensating phenotype, the compensated phenotype is restored to its original state and does not express or temporarily expresses maladaptive plasticity.

**Compensating phenotype:** In plastic compensation, any phenotype that expresses adaptive plasticity such that maladaptive plasticity in the compensated phenotype is not expressed.

**Epigenetic assimilation:** Occurs when epigenetic changes result in the loss of phenotypic plasticity across several generations. Thus a trait is expressed even in the absence of the environment that originally induced it. Plasticity may be regained in subsequent generations through epigenetic modifications.

**Genetic assimilation:** Occurs when mutations result in the loss of phenotypic plasticity. Thus a trait is expressed even in the absence of the environment that originally induced it.

**Genetic divergence:** Significant differences in the statistical properties (mean, variance) of the nucleotide composition of two or more populations, for a single locus. Throughout this paper, genetic divergence is relative to the locus of interest, and is not meant to imply divergence throughout the genome.

**Genetic similarity:** A lack of significant difference for the average nucleotide composition of two or more populations, for a single locus. Throughout this paper, genetic similarity is used to suggest an absence of genetic change in explaining phenotypic divergence.

**Impacted phenotype:** In plastic compensation, the phenotype that experiences the cost of overcoming maladaptive plasticity in the compensated phenotype.

**Phenotype:** Any observable component in the make-up of some group of organisms, that ignores particularities that distinguish individuals. Under this definition, a phenotype could be the product of genetic, epigenetic, or environmental influences or their interactions. Subcellular, molecular and extended phenotypes are included. This definition makes no statement about the evolutionary history or present function of the phenotype. Therefore a phenotype can be so-called even if not subject to selection. The particular variations of a phenotype are called a *trait*.

**Phenotypic accommodation:** The plastic adjustment of (often numerous) phenotypes to accommodate a new phenotypic trait produced through genetic or environmental perturbations. Plastic compensation is the subset of phenotypic accommodation that focuses on overcoming phenotypic differences generated by maladaptive plasticity, rather than overcoming phenotypic differences generated by genetic perturbations, or enhancing phenotypic differences generated by environmental or genetic perturbations.

**Phenotypic divergence:** Significant differences in the statistical properties (mean, variance) of a phenotype between two populations, that could be due to genetic, epigenetic, or environmental differences.

**Phenotypic plasticity:** The production of alternative traits for a single phenotype by a single genotype, caused by different environmental inputs.

**Phenotypic similarity:** A lack of significant differences in the statistical properties (mean or variance) of a phenotype between two populations. Phenotypic similarity, as emphasized in this paper, could be due to genetic, epigenetic, or environmental similarity, genetic divergence, or phenotypic plasticity in some other phenotype.

**Plastic compensation:** The maintenance of a single trait for some compensated phenotype across environments, due to adaptive plasticity in some other phenotype. Without the expression of this adaptive plasticity, maladaptive plasticity in the compensated phenotype would be visible.

**Trait (phenotypic trait):** The particular expression of a phenotype that differentiates individuals, due to genetic, epigenetic, or environmental differences.

Phenotypic optima, such that in the original environment the optimum is attained (the “ancestral” environment), whereas in the novel environment the optimum is not attained due to plasticity. For instance, the optimal fruit mass for a plant may not differ between a high nutrient and low nutrient environment, yet the low nutrient environment may induce smaller fruit sizes due to energy constraints. Alternatively, the optimum in a novel environment may differ from the ancestral optimum, with maladaptive plasticity moving the organism away from, rather than towards, this new optimum. Other types of maladaptive plasticity are possible (Fig. 1). In this paper, we define maladaptive plasticity in the first sense, as expressed when the across-environment optimum remains the same. That is, this paper focuses on the production of similar phenotypes in populations experiencing different environments, despite initial phenotypic divergence driven by maladaptive plasticity.

The evolutionary consequences of maladaptive plasticity remain poorly understood, in part because maladaptive plasticity is not predicted to result in the same evolutionary innovation that may be generated by adaptive plasticity. Whereas adaptive plasticity may increase the likelihood that a population will successfully colonize a new environment (PMPP), maladaptive plasticity should decrease the likelihood of colonization (Plasticity-Mediated Population Extinction – PMPE) (Ghalambor et al., 2007). PMPE may therefore be an important
Fig. 1  Some representative types of maladaptive plasticity are shown
For clarity, in boxes (A) and (B) fitness is not shown, although in each case fitness will be lower in the novel derived environment than in the ancestral environment. The phenotypic optimum in each environment is represented by open circles. Phenotypic values are arbitrary. Assume that fitness is relative to some non-plastic genotype. Maladaptive plasticity can occur when: (A) and (B) The environment moves the phenotype away from its phenotypic optimum. C. Adaptive plasticity (solid line) gives the organism a high fitness (dashed line) across environments. However, if some other environmental factor changes, or if the environmental cue is only reliable under certain circumstances, the phenotypic optimum could change (star) resulting in a usually adaptive response that has become maladaptive with reduced fitness (dash-dot line). Plastic individuals would then be at a disadvantage (see Langerhans and DeWitt 2002; Scoville and Pfrender 2010 for examples). (A) is the form of maladaptive plasticity represented in this paper.

contributor in preventing the production of adaptive phenotypic variation within a species. Furthermore, whereas adaptive plasticity forms the basis of the flexible stem model of adaptive evolution (West-Eberhard, 2003; Wund et al., 2008), in which phenotypic divergence between two populations predates genetic divergence but comes under genetic control (genetic assimilation), thereby maintaining phenotypic divergence even in a common garden, maladaptive plasticity should result in mechanisms to reduce phenotypic divergence if it does not result in extinction. Four ways in which populations can overcome maladaptive plasticity, and their evolutionary consequences, will be discussed below.

2  Overcoming Maladaptive Plasticity: Standing Genetic Variation

Recent investigations into the plasticity of individual genotypes suggest that many populations harbour standing genetic variation (SGV) (Knies et al., 2006; Barrett and Schluter, 2008) for reaction norms (Cheplick, 1995; Ellers et al., 2008; Grishkevich et al., 2012). Sometimes this genetic variation can be observed in natural populations under regularly experienced fluctuating environments – that is, genotype-by-environment interactions are regularly observed in nature. Sometimes, however, individuals exhibit little variation among reaction norms in the wild, and new environmental conditions are required to unmask any SGV that may exist. This cryptic genetic variation (CGV) (Gibson and Dworkin, 2004; Schlichting, 2008; Hayden et al., 2011; McGuigan et al., 2011) may be expressed due to a breakdown in the mechanisms that suppress the influence of genetic variation on the phenotype, or to the accumulation of neutral mutations in unexpressed regions of the reaction norm that are induced under the new environmental conditions. In the initial stages of colonizing a new environment, individuals may express maladaptive plasticity, but the degree of maladaptive plasticity that they express may vary with the genotype due to the presence of SGV. Selection may then remove plastic genotypes, with the net result being a colonized population that exhibits little or no maladaptive plasticity (Fig. 2A,B). Although phenotypically similar, the ancestral and derived populations will be genetically distinct at these loci, with the derived population exhibiting less genetic heterogeneity than the ancestral population. Populations may therefore overcome maladaptive plasticity through reductions in genetic variation, resulting in reduced evolutionary potential at these particular loci.

3  Overcoming Maladaptive Plasticity: Standing Epigenetic Variation

Reaction norm variation may exist in a population for reasons other than standing genetic variation (Frankenhuis and Giudice, 2012; Zhang et al., 2012). For instance, the “plasticity of plasticity” may result in genetically identical individuals with different environmental sensitivities. Frankenhuis and Panchanathan (2011) hypothesized that individuals may modify their phenotype after a period of environmental sampling through exploratory behavior. A homogeneous environment could result in the irreversible plastic production of some trait that enhances fitness in that environment; once developed, the individual would no longer retain the capacity for plasticity. A heterogeneous environment could lead to a delay in the production of a specialist phenotype, resulting in individuals that retain their plasticity later into development. If the environ-
ment were to vary spatially and/or temporally, and individuals were to stochastically sample their environment, genetically identical individuals from the same age class could vary in their degree of plasticity – those that by chance sampled an apparently homogeneous environment would be non-plastic, while those that by chance sampled a heterogeneous environment would retain plasticity. Although Frankenhuis and Panchanathan (2011) did not discuss the possibility of maladaptive plasticity, their hypothesis could be the plastic alternative to standing genetic variation. If the environment were to change but the phenotypic optimum were to remain the same, individuals that had lost their ability to be plastic could be at an advantage over those whose development was still plastic, as this novel environment could induce maladaptive plasticity in plastic individuals. Thus in the absence of genetic variation, there could be selection for a subset of individuals whose developmental trajectories, driven by epigenetic changes, were no longer plastic. Just as a population may contain standing genetic variation for plasticity, it could also carry standing epigenetic variation (SEV) for plasticity (e.g. Wong et al., 2005; Johannes et al., 2009; Schrey et al., 2012), with SEV being caused by variation in individuals’ past experiences of their environment. A population exposed to a novel environment may therefore be phenotypically similar to its ancestral population, but have reduced variation in SEV for the loci involved.

4 Overcoming Maladaptive Plasticity: Genetic Compensation

Individuals may successfully colonize a new environment despite maladaptive plasticity, if selection against maladaptive plasticity is not too strong. For instance, stunted plants growing in a low-nutrient environment may produce fewer seeds than their ancestral counterparts, but if some seedlings survive to reproduce the population may nevertheless persist. Under such conditions, in which phenotypic divergence between ancestral and derived populations is maintained via maladaptive plasticity, Grether (2005) hypothesized, based on the concept of countergradient variation (Conover and Schultz, 1995), that selection may work to restore the ancestral phenotypic trait by favouring any mutations that reduce between-population phenotypic variation. Grether called this genetic compensation, whereby selection on mutated genotypes compensates for the maladaptive plasticity expressed in the novel environment. The result of genetic compensation is cryptic evolution (Fig. 2c, d). Whereas the flexible stem model of adaptive divergence begins with phenotypic divergence, which is then fixed through genetic divergence to maintain phenotypic divergence, genetic compensation begins with phenotypic divergence which is then reduced via genetic divergence. A key test of genetic compensation involves raising phenotypically similar populations under derived conditions. The ancestral population should express maladaptive plasticity, producing phenotypic divergence. Grether (2005) maintained that the opposite, however, was not necessarily true: derived populations under ancestral conditions could maintain their phenotypic trait or exhibit phenotypic divergence via plasticity. Thus, genetic compen-
sation produces phenotypic similarity in different environments, but may generate new forms of phenotypic divergence when both populations are raised in a common, non-derived environment.

There is empirical evidence for genetic compensation in nature. Kokanee salmon *Oncorhynchus nerka*, landlocked anadromous fish that spend their entire life cycles in fresh water, evolved from “residual” sockeye salmon that failed to return to the ocean. During the breeding season male sockeye and kokanee both exhibit a bright red coloration that is under strong sexual selection. This breeding coloration is a consequence of the carotenoids in their diet. Due to the reduced availability of carotenoids in freshwater lakes, residual sockeye males fail to turn red, displaying their non-reproductive green coloration even during the breeding season. How did kokanee re-evolve red breeding coloration? Sexual selection (Foote et al., 2004) in kokanee favoured mechanisms for increased efficiency in the assimilation of carotenoids (Craig and Foote, 2001), thereby restoring the red phenotypic trait. The genetic nature of these changes has been inferred from the fact that hybrids have intermediate carotenoid assimilation rates when compared to either parent (Craig et al., 2005).

Genetic compensation results in phenotypically similar, genetically divergent populations that have evolved unique mechanisms for producing the ancestral trait despite maladaptive plasticity. Maladaptive plasticity, therefore, can be an important factor in the evolution of adaptive genetic divergence, although the extent to which genetic compensation occurs in nature is currently not known (but see Storz et al., 2010; Deere et al., 2012). Maladaptive plasticity is rarely incorporated into models of adaptive evolution, and as such hypotheses regarding the likelihood of genetic compensation under different evolutionary scenarios remain to be developed. For instance, under what scenarios could maladaptive plasticity lead to the evolution of a new trait, rather than genetic compensation to restore the original trait?

5 Overcoming Maladaptive Plasticity: Plastic Compensation

Many organisms have mechanisms for buffering against the effects of environmental perturbations. This has been long known and oft-studied, going under a variety of names and processes (e.g., homeostasis, Bradshaw-Sultan Effect, tolerance, compensation, etc.: Strauss and Agrawal, 1999; Pigliucci, 2001; Ali et al., 2003; Carroll, 2007; O’Brien, 2011). These processes have not been discussed in the context of the evolutionary consequences of maladaptive plasticity (Ghalambor et al., 2007; Fitzpatrick, 2012), likely because they are multitudinous, or perhaps because maladaptive plasticity is not considered when maladaptive plasticity cannot be observed. Whatever the reason, these disparate means of buffering against environmental perturbations almost, if not always, involve adaptive plasticity. For convenience, we define any process by which plastic changes in one phenotype overcome maladaptive plasticity in another phenotype as “plastic compensation,” differentiating it from genetic compensation (Fig. 3).

Plastic compensation can most easily be defined as adaptive plasticity overcoming maladaptive plasticity. At least three phenotypes are involved in plastic compensation: (1) the compensated phenotype, that should express maladaptive plasticity in the new environment, but which may not due to (2) the compensating phenotype, that expresses adaptive plasticity in the new environment such that the compensated phenotype is restored to its ancestral state, thereby limiting the expression of maladaptive plasticity in the compensated phenotype; and (3) the impacted phenotype that experiences Fig. 3 The effects of (A) plastic compensation and (B) a lack of plastic compensation on (C) fitness Phenotype values are arbitrary. A. A compensated phenotype (dashed line) maintains its phenotypic optimum (open circles) under novel conditions in the derived environment. The compensating phenotype (dotted line) plastically adjusts to its phenotypic optimum (stars) in the derived environment. Plasticity in the compensating phenotype causes non-plasticity in the compensated phenotype (arrow). B. The consequence of not having a functional compensating phenotype is maladaptive plasticity in the compensated phenotype (arrow). C. Plastic compensation maintains fitness between the two environments (dashed-dotted line) while a lack of plastic compensation results in fitness reduction (fine dashed line). Plastic compensation likely does not perfectly maintain fitness due to the cost of plastic compensation (plasticity in the impacted phenotype, not shown), but the loss in fitness is still a gain relative to the loss of fitness without plastic compensation.
the cost of plasticity generated by the compensating phenotype (Fig. 3). The result of plastic compensation, like genetic compensation, is phenotypic similarity between ancestral and derived populations for the compensated phenotype. However, plastic compensation differs from genetic compensation in several ways: (1) Plastic compensation does not require genetic divergence for phenotypic similarity to be ensured. (2) Plastic compensation can, but does not have to, occur immediately upon colonization of the novel environment. That is, maladaptive plasticity may never be seen. (3) Plastic compensation usually results in phenotypic differentiation between populations for the compensating and impacted phenotypes. (4) Plastic compensation occurs without evolutionary change (but could lead to epigenetic inheritance of the compensating phenotype). Genetic compensation and plastic compensation, although distinct, are not mutually exclusive. If plastic compensation does not perfectly overcome maladaptive plasticity, genetic compensation can occur. Similarly, genetic compensation may include the evolution of compensating phenotypes.

One possible objection to plastic compensation is that it includes every instance of adaptive plasticity, but this is not the case. For instance, Daphnia produce body armour in the presence of fish kairomones as an adaptation to predation. The production of this body armour involves a suite of transcriptionally plastic genes (Tollrian and Leese, 2012). Neither this molecular plasticity, nor its macrophenotypic effect on morphology, fits the definition of a compensating phenotype, since these plastic changes drive phenotypic divergence rather than phenotypic similarity. Furthermore, any individual that was prevented from expressing this transcriptional or morphological plasticity in the presence of predators would not express maladaptive plasticity, but would instead be phenotypically identical to individuals in a predator-free environment. Maladaptive plasticity is not overcome by the production of body armour, and so plastic compensation does not apply in this situation.

Plastic compensation can be defined as a subtype of phenotypic accommodation. Phenotypic accommodation is defined as the adaptive adjustment of numerous phenotypes to genetically- or environmentally-induced changes in some other phenotype during development (West-Eberhard, 2003, 2005). This inherent plastic responsiveness of organisms permits considerable coordinated phenotypic change to occur without genetic change. Not every example of phenotypic accommodation, however, is plastic compensation. Slijper’s two-legged goat (Slijper, 1942a, b), which learned to hop on its hindlimbs, developed kangaroo-like changes to its musculature and skeletal structure in response to behavioural changes, without requiring novel genetic input. Such an example does not fulfill the definition of plastic compensation, since (1) the limb defect was not an example of maladaptive plasticity, and (2) this novel genetic input compelled the production of phenotypic novelty, but this phenotypic novelty did not in turn create phenotypic similarity. Behavioural plasticity in the goat drove phenotypic accommodation in a suite of plastic phenotypes; but the absence of behavioural plasticity would have led to regular muscle and skeletal development. Although West-Eberhard envisioned phenotypic accommodation in terms of phenotypic changes that produce adaptive phenotypic novelty, plastic compensation could be conceptualized as the subset of phenotypic accommodation cases in which maladaptive plasticity is overcome by adaptive plasticity, and is thus not observed. Plastic compensation is to phenotypic accommodation as Grether’s genetic compensation (2005) is to West-Eberhard’s (2003) genetic accommodation. It is the very breadth of phenotypic accommodation that has likely prevented its recognition in maladaptive plasticity literature; plastic compensation was so defined as to bring into focus the importance that phenotypic accommodation should have for those who study maladaptive plasticity.

6 Types of Plastic Compensation

There are likely numerous types of plastic compensation, but a few possible ways in which plastic compensation could be expressed are discussed below. First, a population split into ancestral and novel environments could remain phenotypically similar for the compensated phenotype, due to behavioural, morphological, physiological, or molecular plasticity in some compensating phenotype. Without plasticity in the compensating phenotype, maladaptive plasticity in the compensated phenotype would be visible. Second, there could be a time lag between the induction of maladaptive plasticity and restoration of the ancestral trait. This time lag could be due to (1) a time lag between the induction of maladaptive plasticity in the compensated phenotype and adaptive plasticity in the compensating phenotype; (2) the simultaneous induction of maladaptive and adaptive plasticity, but a time lag before the effects of the compensating phenotype on the compensated phenotype become visible; (3) a time lag between generations, in which maladaptive plasticity is expressed in the colo-
nists but is absent in their offspring. Third, the compensated phenotype may not be restored until the environmental conditions that induced maladaptive plasticity are removed. Finally, the compensated and compensating phenotypes may reside in different individuals of the same or different species. For instance, plasticity in parents could overcome maladaptive plasticity in offspring, or plasticity in one mutualist could overcome maladaptive plasticity in its partner species. If a population with a mutualist species exhibited phenotypic similarity in two environments, but in the absence of the mutualist species developed maladaptive phenotypic divergence, the mutualist species would be a good candidate for the producer of the compensating phenotype.

An example of each type of plastic compensation will be given below.

**Examples of plastic compensation**

Examples of plastic compensation are likely as numerous and diverse as the organisms that produce them. However, a few important historic and modern examples can illustrate the importance of plastic compensation for overcoming the effects of maladaptive plasticity.

1) **Transcriptional plasticity in sea urchins** – Projected increases to ocean acidity via rising carbon dioxide levels are predicted to pose a problem for organisms that rely on calcium carbonate production (Doney et al., 2009). Experimental manipulations have shown a wide range of echinoderm developmental responses to acidity, from disrupted development at low pHs (Reuter et al., 2010) to a developmental insensitivity to pH (Byrne et al., 2010). What accounts for these vastly different responses among echinoderms? In one experiment, eggs and larvae of the sea urchin *Paracentrotus lividus* were reared at six different pHs, ranging from their native pH of 8.1 to a projected future pH of 7. The urchins were insensitive to pH for a suite of morphological features up to a pH of 7.2, after which significant morphological changes occurred. This remarkable lack of morphological plasticity was due to an increase in transcript production for genes associated with biomineralization. At a pH of 7, however, transcriptional regulation broke down and transcript levels returned to their high pH state. Consistent with a plastic compensation hypothesis, this break-down in differential gene expression resulted in the greatest measured disturbance to the size of the post-oral and antero-lateral arms, and to the ratio of post-oral arms to somatic rod lengths (Martin et al., 2011). Although this experiment did not measure fitness, such morphological plasticity can be reasonably inferred to be maladaptive, due to the increased transcription associated with maintaining this phenotype up until extremely unnatural acidities. Thus researchers demonstrated a compensated phenotype (morphology) and a compensating phenotype (transcript abundance). No impacted phenotype was measured. A related experiment on a species of brittlestar did measure an impacted phenotype, with decreasing pH resulting in reduced muscle mass, due to energy reallocation towards biomineralization (Wood et al., 2008). The next step would be to test whether those urchin species that are developmentally sensitive to pH lack the corresponding physiological and transcriptional plasticities found in insensitive urchin species.

2) **Parental allocation of reproductive energy** – As seen in the above example, organisms may overcome maladaptive plasticity through the interaction of a “hierarchy of plasticities” (Bradshaw, 1965). This has been referred to as the Bradshaw-Sultan Effect (Pigliucci, 2001) after a cross-generational form of plastic compensation in *Persicaria maculosa* (at the time called *Polygonum persicaria*) (Sultan, 1995, 1996). Plants reared in stressful low light and non-stressful high light conditions produced offspring with similar biomass. This non-plasticity in offspring biomass seemed to be related to non-plasticity in fruit mass. The stressed parents, it was discovered, were preferentially allocating energy away from the outer pericarp and investing it in the fruit. Plasticity in energy allocation thereby allowed offspring biomass non-plasticity. In this example there are two related compensated phenotypes (fruit mass and offspring biomass), a compensating phenotype (unmeasured phenotypes responsible for energy allocation), and an impacted phenotype (pericarp mass). Had energy not been allocated away from pericarp production, fruit mass would have decreased and offspring growth and survival would have been impaired.

3) **Compensatory growth in fishes** – Growth rates and size-at-age are important aspects of fish fitness, influencing their abilities to migrate, overwinter, catch prey, and produce offspring. Given the trade-offs between predation and food availability that govern fish growth, population-level differences in growth rates are often cited as evidence for local adaptation (Garcia de Leaniz et al., 2007). Growth is highly plastic, and can be suppressed by low temperature, low food availability, high density, and other environmental stressors. Any event that forces an organism to grow below its adaptive growth rate would likely impose a fitness cost, and thus there should be selection on strategies for plastically
overcoming periods of growth depression. One such strategy is compensatory growth. Compensatory growth, which is triggered after release from an environmental stressor, is defined as an increase in growth rate relative to routine growth, such that individuals catch up to the size or condition that they would have been had their growth rate not been suppressed (Ali et al., 2003). For instance, threespine stickleback raised at 10°C and then transferred to 6°C waters had slower growth rates than fish kept at 10°C, and thus grew to be significantly smaller. However, upon release to 10°C waters, these growth-depressed fish grew rapidly, attaining the size of 10°C fish after twelve weeks. That is, despite having access to the same resources as 10°C fish, compensating fish were able to increase growth rates above their normal 10°C growth rate, and catch-up to the size they would have been had their growth not been initially depressed (Lee et al., 2012). This compensatory growth, although it overcame the maladaptive effects of low temperatures, came with the cost of a decreased lifespan (Lee et al., 2013). Presumably the gains achieved by attaining a larger size make up for this cost. The exact mechanisms of compensatory growth in stickleback are unknown, but in other fishes have been shown to include behavioural plasticity (hyperphagia), physiological plasticity (increased food conversion efficiency), and molecular plasticity (increase in ghrelin production) (Ali et al., 2003; Terova et al., 2008). In this example, there is a compensated phenotype (size/length/condition-at-age), likely several underlying compensating phenotypes (e.g. hyperphagia, ghrelin production) that produce compensatory growth, and at least one impacted phenotype (lifespan). Note that in this case maladaptive plasticity is not overcome in the stressful environment, but is overcome by plastic compensation only after a return to non-stressful conditions.

7 Identifying Plastic Compensation

Because maladaptive plasticity may not be observed during plastic compensation, and because non-plastic phenotypes are likely underreported in the literature, it has been difficult to establish a link between plasticity in one phenotype and a lack of plasticity in another. That said, specific observations may indicate plastic compensation. First, if a population is resistant to an environmental stressor, increasing the stress until maladaptive plasticity is expressed could reveal plastic compensation. Plasticity would have to be monitored for some other phenotype, particularly transcript or protein abundance. If the expression of maladaptive plasticity coincides with an abrupt decrease in plasticity in some other trait, this could be strong evidence for plastic compensation. Similarly, comparing plasticity for a suite of phenotypes in populations that are sensitive versus populations that are insensitive to the environment could reveal plastic compensation.

The steps to identifying plastic compensation are as follows: (1) Identify a resistance to maladaptive plasticity for some important trait, for some novel environment. (2) Identify plasticity in a phenotype that should be associated with the production or maintenance of the putatively compensated phenotype. (3) Establish a causal link between the two, by identifying individuals that vary in plasticity for the presumably compensating phenotype, by generating mutants that cannot express plasticity for the presumably compensating phenotype, or by testing the organism under unnaturally stressful conditions. Assess the impact this has on the compensated phenotype. If maladaptive plasticity is triggered by a decrease in plasticity in the compensating phenotype, this is a good candidate for plastic compensation.

8 Plastic Compensation and Evolution

Plastic compensation likely has important effects on the adaptive divergence of populations (Fig. 4). By overcoming maladaptive plasticity, the compensating phenotype permits PMPP, which is the first step in ecological speciation (Thibert-Plante and Hendry, 2010). Plastic compensation may furthermore facilitate genetic divergence in that, if plastic compensation does not completely overcome maladaptive plasticity, genetic compensation could work to enhance phenotypic similarity via genetic changes to the production of the compensated or compensating phenotypes (Fig. 4). Indeed, imperfect plastic compensation could make genetic compensation more likely, by increasing the probability of colonization. However, cryptic evolution could be limited if plastic compensation perfectly restores the compensated phenotype. Finally, it is important to keep in mind that the compensating phenotype is adaptively plastic and results in adaptive phenotypic divergence between populations in different environments without any corresponding genetic change. Populations in the ancestral environment and the derived environment express different portions of the compensating phenotype’s reaction norm. Theory suggests that, in the absence of environmental change, the particular trait expressed by each population could become fixed, such that it no longer expresses plasticity (Waddington 1953).
Fig. 4  The evolutionary consequences of plastic compensation

Phenotypic values are arbitrary. Phenotypic optima are represented by open circles. **A.** A novel environment may induce maladaptive plasticity (dashed line) such that the likelihood of persisting in the environment is virtually zero (fitness as dash-dot line). This is Plasticity-Mediated Population Extinction (PMPE). **B.** Even slight plasticity in a compensating phenotype (dotted line) may be enough to reduce (arrow) maladaptive plasticity in the compensated phenotype (solid dashed line), such that the negative effect on fitness is reduced (dash-dot line). The population may then be able to persist because of imperfect plastic compensation (Plasticity-Mediated Population Persistence, PMPP). Genetic compensation may then restore the compensated phenotype to its ancestral state (solid line).

This fixation could occur via genetic change (genetic assimilation) or epigenetic inheritance (epigenetic assimilation) (Sollars et al., 2003; Ruden et al., 2005; Stern et al., 2012). Once fixed, the compensating phenotype would become robust (canalized) to environmental change for at least several generations. This could, like genetic compensation, lead to countergradient variation, in that assimilation of the compensating phenotype could lead to phenotypic divergence for the compensated phenotype when ancestral and derived populations are raised in a common garden.

9  Conclusion

Maladaptive plasticity is an important, and underappreciated, means by which organisms respond to environmental change. If maladaptive plasticity does not result in PMPE, it could have important evolutionary implications, depending on the means by which it is overcome. Maladaptive plasticity could be overcome through genetic responses (SGV, genetic compensation) or their equivalent plastic responses (SEV through exploratory behavior, plastic compensation), resulting in phenotypically similar populations residing in different environments.

Phenotypic similarity is common for populations residing in different environments, but this does not mean that interesting processes are not at work. Standing genetic variation could result in selection against maladaptively plastic genotypes, resulting in a derived population with reduced genetic variation at the involved loci. Exploratory behavior could result in genetically identical individuals expressing variation in reaction norms, with selection favoring the subset of individuals that sampled homogeneous environments early in development and thus became resistant to environmental change. Genetic compensation could result in the evolution of novel genetic pathways to restore the ancestral trait, with populations being phenotypically similar in their respective environments, but phenotypically divergent in a common environment. Finally, phenotypic similarity could be caused by plastic compensation, where adaptive plasticity in the compensating phenotype overcomes maladaptive plasticity in the compensated phenotype. This could result in populations that differ phenotypically for the compensating phenotype, but for which maladaptive plasticity cannot be observed. Although adaptive plasticity has received the majority of attention, maladaptive plasticity and strategies for overcoming it are important to consider as we seek to understand the link between the genotype, phenotype, and fitness under natural environmental conditions.

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