REVIEW

Modifying effects of phenotypic plasticity on interactions among natural selection, adaptation and gene flow

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Abstract

Divergent natural selection, adaptive divergence and gene flow may interact in a number of ways. Recent studies have focused on the balance between selection and gene flow in natural populations, and empirical work has shown that gene flow can constrain adaptive divergence, and that divergent selection can constrain gene flow. A caveat is that phenotypic diversification may be under the direct influence of environmental factors (i.e. it may be due to phenotypic plasticity), in addition to partial genetic influence. In this case, *phenotypic* divergence may occur between populations despite high gene flow that imposes a constraint on *genetic* divergence. Plasticity may dampen the effects of natural selection by allowing individuals to rapidly adapt *phenotypically* to new conditions, thus slowing adaptive genetic divergence. On the other hand, plasticity may promote future adaptive divergence by allowing populations to persist in novel environments. Plasticity may promote gene flow between selective regimes by allowing dispersers to adapt to alternate conditions, or high gene flow may result in the selection for increased plasticity. Here I expand frameworks for understanding relationships among selection, adaptation and gene flow to include the effects of phenotypic plasticity in natural populations, and highlight its importance in evolutionary diversification.

Introduction

Several factors may influence adaptive phenotypic divergence among populations, and three of these include: (i) divergent natural selection (Schluter, 2000), (ii) gene flow between selective environments (Garant *et al.*, 2007) and (iii) phenotypic plasticity (Pigliucci, 2001). Relationships among natural selection, adaptive divergence and gene flow have been considered both theoretically and empirically in numerous studies (e.g. García-Ramos & Kirkpatrick, 1997; Hendry *et al.*, 2001; Hendry & Taylor, 2004; Nosil & Crespi, 2004), yet the interplay among these factors and *plasticity* is rarely, if ever, considered. However, plasticity can be an important

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factor in evolutionary diversification. Modifying effects of plasticity on adaptation and gene flow may be either positive or negative, depending on the nuances of specific systems. The aim of the current paper is two-fold. First, I provide a conceptual basis for understanding how phenotypic plasticity influences adaptive divergence and gene flow, and conversely how adaptive divergence and gene flow influence phenotypic plasticity, in natural systems. Second, I highlight how phenotypic plasticity can affect inferences made about adaptive divergence in natural systems. My goal is to provide a framework for understanding interactions among selection, gene flow and plasticity in studies of adaptive evolutionary divergence.

Phenotypic plasticity may play important roles in the relationships among divergent selection, adaptive divergence and gene flow. First, plasticity and adaptive genetic divergence may interact, irrespective of gene flow. For example, plasticity can either promote future genetic

change or retard genetic change (Price *et al.*, 2003; Ghalambor *et al.*, 2007; Paenke *et al.*, 2007), depending on whether costs/limits are associated with the plastic response. Second, plasticity may allow for increased gene flow between selective environments, by allowing individuals to phenotypically adapt to alternate environments (e.g. see Crispo & Chapman, 2008). On the other hand, gene flow may have positive effects on plasticity, if selection for increased plasticity occurs in high gene flow scenarios (Sultan & Spencer, 2002), which should be the case if the most plastic individuals are most likely to survive under a wide range of selective pressures. Third, plasticity may alter an apparent relationship between divergent selection and gene flow by driving phenotypic divergence among environments, beyond that which is under genetic influence, making it appear that gene flow imposes little or no constraint on adaptive divergence. Recent studies have documented that adaptive divergence can occur in the face of high gene flow (e.g. Emelianov *et al.*, 2004; Jordan *et al.*, 2005; Hemmer-Hansen *et al.*, 2007; Kotlik *et al.*, 2008; Niemiller *et al.*, 2008), but the relative influences of genetic change versus direct environmental effects on phenotypic divergence are often not known. Thus adaptive genetic divergence may be under a constraint imposed by gene flow, despite high levels of environmentally induced (plastic) phenotypic divergence.

A few points require attention. First, I here use the term 'adaptive divergence' to refer specifically to adaptive *genetic* divergence, but any type of phenotypic change (genetic or environmentally induced) could be considered adaptive. Second, I refer primarily to plasticity that is in the adaptive direction (i.e. plasticity that increases mean fitness across environments, or increases fitness in newly colonized environments), but maladaptive plasticity may instead occur. A plastic response that is not in the adaptive direction requires a different set of predictions (discussed below). Third, I refer to phenotypic plasticity versus local adaptation, but plasticity may actually be locally adaptive if populations evolve in heterogeneous environments with variation in selective pressures. Fourth, plasticity may be either labile or

developmental. The former refers to phenotypic changes that may occur throughout the lifespan of an individual in response to environmental change, and may be reversible (e.g. many behavioural and physiological traits). The latter refers to phenotypic changes that occur during development, and once the phenotype is established it cannot be altered or reversed (e.g. some morphological traits). The former may have more profound effects on adaptive divergence and gene flow because individuals are able to rapidly adapt to new selective pressures. Yet, developmental plasticity may also have an effect if a few individuals of the parental generation were able to penetrate the selective barrier (i.e. were able to survive and reproduce despite being less well-adapted than resident individuals). Selection against migrant genotypes would then be diminished in the F_1 generation. However, the presence of this selective barrier would depend on the developmental stage at which individuals disperse, as well as the developmental stage at which selection takes place (discussed below). The scenarios described throughout the present paper could refer to either developmental or labile plasticity, unless otherwise specified.

Plasticity and adaptive divergence

A traditional view of adaptive phenotypic plasticity is that it retards adaptive genetic divergence by buffering individuals from the action of negative selection (Wright, 1931; reviewed by Schlichting & Pigliucci, 1998, pp. 66–68; West-Eberhard, 2003, p. 178). Alternatively, local genetic adaptation could evolve in constant environments, preventing the need for plastic, environmentally induced adaptive responses (arrow 1 in Fig. 1; Table 1). Plastic and genetic variation could thus be considered two unique ways of adapting to local environmental conditions. Yet, plasticity has potential costs/limits, hence the reason why we observe genetically divergent forms rather than 'Darwinian monsters' (Pigliucci, 2001, p. 174) that are able to phenotypically adapt to any environment through plastic responses (see below). It would be more beneficial for organisms to be plastic than
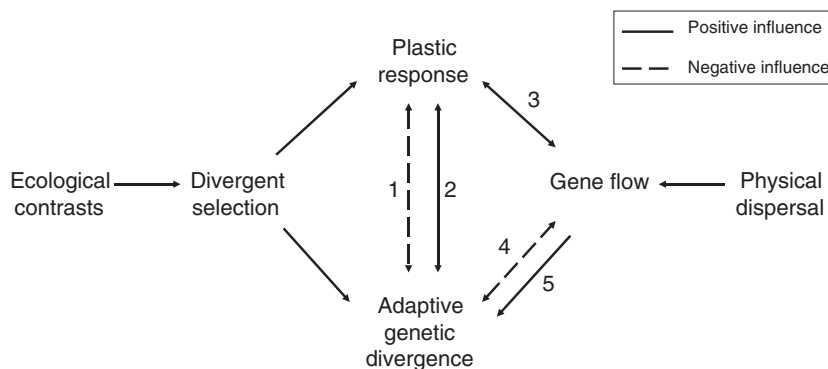

Fig. 1 Potential interrelationships among divergent selection, adaptive divergence, adaptive phenotypic plasticity and gene flow.

Table 1 Interrelationships among adaptive genetic divergence, phenotypic plasticity and gene flow.

Causal factor	Affected factor	Type of influence	Arrow in Fig. 1	Mechanism
PP	AD	−	1	PP buffers individuals from the negative action of natural selection
AD	PP	−	1	Local genetic adaptation favoured over PP due to costs/limits associated with PP
PP	AD	+	2	PP permits colonization of novel environments
AD	PP	+	2	Genetic isolation of populations allows for the evolution of locally adaptive PP
GF	PP	+	3	Meta-population structure promotes the evolution of PP to allow for phenotypic adaptation to alternate environmental conditions
PP	GF	+	3	High levels of PP allow for increased phenotypic adaptation to alternate environments after dispersal
GF	AD	−	4	Genotypes adapted to alternate environmental conditions hinder local adaptation
AD	GF	−	4	Divergent selection reduces GF between environments because resident individuals/genotypes have higher fitness than dispersing individuals/genotypes
GF	AD	+	5	GF between selective environments promotes the evolution of reinforcement

AD, adaptive genetic divergence; PP, phenotypic plasticity; GF, gene flow.

to genetically adapt to one local environment when (i) environments are spatially or temporally heterogeneous, (ii) environmental variation is predictable and (iii) the plastic response occurs as rapidly as the environmental change (reviewed by Alpert & Simms, 2002).

An alternative view is that plasticity permits colonization and persistence in novel environments, thus increasing the potential for future adaptive genetic divergence (arrow 2 in Fig. 1; Table 1; reviewed by Price et al., 2003; Crispo, 2007; Ghalambor et al., 2007). Adaptation could occur more rapidly via the plastic response than via natural selection, because plasticity allows an entire population, or group individuals, to adapt simultaneously, whereas natural selection works upon standing genetic variation among individuals or new mutations that may arise. A body of evidence suggests that plasticity

may promote adaptive divergence in numerous systems, based largely on studies showing that plasticity results in adaptive phenotypic change, often followed by genetic changes in the direction of the plastic response (i.e. cogradient variation; Conover & Schultz, 1995; Byars et al., 2007). For example, when spadefoot toad tadpoles (*Spea multiplicata* and *Spea bombifrons*) were raised at various densities, *S. multiplicata* became more omnivorous as the density of *S. bombifrons* increased (the latter being a more effective predator; Pfennig & Murphy, 2002). In addition, omnivory in *S. multiplicata* was positively associated with the presumed abundance of *S. bombifrons* in their native ponds, when raised under identical conditions, indicating a genetic component, in addition to the plastic component, of diet variation. Similarly, when sympatric morphs of Arctic charr (*Salvelinus alpinus*) were raised in a common garden environment in the laboratory, a considerable portion of variation in head morphology (related to trophic differences) between wild-caught and lab-raised fish was attributable to the rearing environment (wild or laboratory), but genetic differences occurred between the morphs raised in a common environment (Adams & Huntingford, 2004). These examples suggest that plasticity may drive initial phenotypic divergence in some cases, and that genetic changes may follow in the direction of the plastic response (Pigliucci & Murren, 2003; Price et al., 2003; West-Eberhard, 2003; Crispo, 2007; Ghalambor et al., 2007). However, it cannot be conclusively known in the above examples which came first, the plastic or genetic responses to selection. Long-term studies of recently established populations, or artificial selection experiments in the laboratory, may better address the question of whether plasticity can set the stage for future genetic change.

Once adaptive divergence has occurred, it may result in a subsequent reduction in plasticity via genetic assimilation (Waddington, 1953a,b, 1956). Genetic assimilation is a process whereby a phenotype that was once induced by an environmental cue becomes canalized, so that it is expressed even when the environmental cue is removed (reviewed by West-Eberhard, 2003; Crispo, 2007). Plasticity could be lost due to neutral processes (i.e. drift) if populations are located in homogeneous environments (i.e. plasticity not beneficial) among which low gene flow occurs (see below). Adaptive genetic divergence would be *favoured* over plasticity if the plastic response was costly to maintain – these may include, as examples, costs associated with the maintenance of mechanisms for sensing changes in the environment, the production of alternate phenotypes, and epistatic or pleiotropic effects (reviewed by DeWitt et al., 1998; see also Pigliucci, 2001, pp. 174–180). Plasticity may also be limited such that a lag time exists between sensing the environmental cue and production of the appropriate phenotype, or if the optimal phenotype falls outside the range of phenotypes produced via

the plastic response (DeWitt *et al.*, 1998; see Fig. 8 in Crispo & Chapman, 2008). In situations in which plasticity is costly or limited, we would expect that the plastic response may be lost in favour of genetic adaptation.

Alternatively, adaptive population divergence may promote the evolution of locally adaptive plasticity (arrow 2 in Fig. 1; Table 1). This would occur if different plastic responses are adaptive under different environmental conditions (e.g. see Donohue *et al.*, 2000; Edelaar *et al.*, 2005; Kishida *et al.*, 2007), but initial genetic divergence in other traits is required to isolate populations reproductively so that adaptive divergence of plasticity may be permitted. Implicit in the prediction that adaptive divergence in plasticity should evolve is the assumption that plasticity has a heritable component. Wolterek (1909) proposed that it was the reaction norm (i.e. plasticity, or more specifically, the slope of the relationship between phenotype and environment) that was under selection rather than the individual phenotype, and Bradshaw (1965) was the first to propose that plasticity may have a genetic component and be heritable. Although this has long been a contentious issue (reviewed by Via *et al.*, 1995), recent research has shown that plasticity can indeed be selected upon and inherited in at least some cases. For example, plasticity in the timing of reproduction is heritable in great tits (*Parus major*; Nussey *et al.*, 2005). Seasonal plasticity in body mass is heritable in bighorn sheep (*Ovis canadensis*), and the most plastic individuals have the greatest reproductive success (Pelletier *et al.*, 2007). Selection for plasticity would require that standing genetic variation for plasticity is present in the population. Although debate has revolved around whether selection can act directly on plasticity, or whether plasticity is indirectly selected for through selection on other traits (see Scheiner & Lyman, 1989; Schlichting & Pigliucci, 1993; Via, 1993a, b), the end product would be similar – plasticity would increase in scenarios in which the plastic response enhances adaptation to a new environment.

Plasticity and gene flow

High gene flow between selective environments may actually favour the evolution of increased phenotypic plasticity over adaptive genetic divergence between populations (arrow 3 in Fig. 1; Table 1). This effect would be most apparent if gene flow was multidirectional, i.e. in cases with meta-population structure where populations experience spatial environmental heterogeneity (Sultan & Spencer, 2002). Plasticity could allow individuals to phenotypically adapt to new conditions within one or two generations, and thus would allow them to persist in novel environments. In very high gene flow scenarios, we would predict that a jack-of-all-trades strategy would be the most likely to evolve, where the phenotypic response to the environment maximizes fitness in an *average* environment, as opposed to a master-of-some strategy where fitness is maximized in only one specific environment (Richards *et al.*, 2006). If gene flow was unidirectional, we would only expect increased plasticity in the recipient population – because no gene flow from divergent environments occurs into the donor population, the donor population may be permitted to genetically adapt to its local environment without negative effects of gene flow. Yet, the most plastic individuals in the donor population (assuming variability in plasticity, i.e. gene-by-environment interactions) may be able to *phenotypically* adapt within one generation (if plasticity was labile) to a new environment after dispersal. Developmental plasticity may also be selected for if a few dispersing individuals were able to penetrate the selective barrier, because the environment would induce an adaptive response in the offspring of these dispersers.

Conceivably, if individuals are highly plastic with respect to traits that are adaptive in alternate forms under different environmental conditions, increased gene flow between selective environments may be permitted (arrow 3 in Fig. 1; Table 1). If individuals are able to phenotypically adapt to new conditions through an environmentally induced response, dispersing individuals should be less likely to be selected against (i.e. die before reproducing, or produce maladaptive offspring). Divergent natural selection among environments would thus be diminished. This phenomenon would be most likely to occur if plasticity was labile, but could also be apparent if plasticity was developmental (see above). Developmental plasticity may allow offspring of dispersers to phenotypically adapt; however, divergent selection must be weak enough to allow some adult individuals to penetrate the selective barrier (assuming selection acts on adults). The relative effects of labile versus developmental plasticity would depend on the developmental stage at which selection and/or dispersal take place – if either occurs at an early developmental stage, the effects of labile and developmental plasticity may be similar. If selection occurs on juveniles, adult dispersers would bypass the selective barrier. If dispersal occurs early in life, plasticity would allow dispersers to adapt to conditions in the new environment.

In support of the hypothesis that plasticity should allow for increased gene flow, numerous studies have shown that invasive species, and newly colonized populations, are more plastic than their native counterparts (reviewed by Daehler, 2003; Richards *et al.*, 2006; see also, for example: Yeh & Price, 2004; Geng *et al.*, 2006), suggesting that plasticity may have facilitated colonization of the newly occupied areas. Phenotypic plasticity may have facilitated gene flow between dissolved oxygen regimes in an African cichlid fish (*Pseudocrenilabrus multicolor*), found in high

abundance in free-flowing rivers and open lakes, and in oxygen-scarce swamps that occur in parapatry with the high-oxygen environments (Crispo & Chapman, 2008). Dissolved oxygen concentration should pose selective pressure on gill structures; yet, plasticity in gill structures has been documented (Chapman *et al.*, 2000; L. J. Chapman, J. Albert & F. Galis, unpublished manuscript). Population genetic structure is not related to dissolved oxygen regime in this species, indicating that genes flow freely between selective environments, and this gene flow could be facilitated by plasticity of the gills (Crispo & Chapman, 2008). However, it cannot be determined conclusively in the above examples whether plasticity increases gene flow or whether gene flow increases plasticity (see Richards *et al.*, 2006; the issue of inferring causality is discussed below).

Plasticity and the gene flow-selection balance

I have thus far described how phenotypic plasticity can influence both adaptive divergence and gene flow and vice versa. Now I turn to the problem of how plasticity can confound expected relationships between adaptive genetic divergence and gene flow. Theory predicts (e.g. García-Ramos & Kirkpatrick, 1997; Hendry *et al.*, 2001; Lenormand, 2002), and empirical evidence shows (e.g. King & Lawson, 1995; Storfer *et al.*, 1999; Moore *et al.*, 2007), that gene flow between selective environments can constrain adaptive divergence (arrow 4 in Fig. 1; Table 1). Yet, if plasticity occurs, and if plasticity is in the adaptive direction (i.e. cogradient variation), phenotypic divergence between environments may result despite little or no adaptive genetic divergence. In this case, it may appear that high gene flow has little or no consequence for adaptive divergence; yet, *genetic* components of trait divergence may be under stronger constraint than the overall phenotypic patterns would suggest. Attempts to distinguish between genetic and environmental components of trait divergence in natural populations show that divergence in most traits has both a genetic and environmental component (e.g. Chapman *et al.*, 2000; Pfennig & Murphy, 2002; Adams & Huntingford, 2004; Byars *et al.*, 2007; Jiménez-Ambriz *et al.*, 2007).

In the absence of plasticity, divergent selection may decrease gene flow between selective environments, because well-adapted residents and their alleles will be favoured over maladapted dispersers and dispersing alleles (arrow 4 in Fig. 1; Table 1; i.e. ecological speciation; Schluter, 2001; Nosil *et al.*, 2005). However, plasticity may permit increased gene flow between selective environments, because individuals are able to adapt to the new environment within one or two generations (as noted above). Plasticity thus may have one of two effects: (1) plasticity may increase gene flow, which in turn decreases adaptive divergence (arrows 3 and 4 in Fig. 1; Table 1), or (2) plasticity may underlie phenotypic divergence, making it appear that divergent selection is not acting to reduce gene flow between selective environments; yet selection may be acting against dispersers if plasticity is costly (i.e. local genetic adaptation favoured over plasticity) and/or selection may act against non-plastic dispersing individuals (assuming variability in plasticity in the donor population). These points differ in that (1) details negative effects of plasticity on adaptive genetic divergence, whereas (2) details how plasticity would make ecological speciation (i.e. a reduction in gene flow between selective environments) undetectable if it was occurring.

To control for direct environmental influence on the phenotype, common garden experiments should be used in studies attempting to make inferences about adaptive divergence among populations. When doing so, it is imperative to use rearing conditions that are comparable to natural conditions experienced by the populations; this is because traits may have environmental thresholds, or switch points, above and below which different phenotypes are expressed (reviewed by Roff, 1996; West-Eberhard, 2003). In other words, phenotypes may not be represented by linear reaction norms along environmental gradients, so extrapolation may be misleading. The best way to circumvent this problem would be to rear individuals from multiple populations, using a split brood design (to control for the genetic background), under multiple treatments, each treatment representing a condition experienced by one of the populations. Alternately, one could perform reciprocal transplant experiments in the field (Kawecki & Ebert, 2004). Maternal effects related to the dam's rearing environment could also influence offspring phenotype, and thus F_2 or later generations could be used to remove these effects if they are expected to be strong. Adaptive divergence and gene flow can be compared after controlling for direct environmental effects in this way.

I have thus far discussed the negative effects of gene flow on adaptive divergence. Yet potential benefits of gene flow to adaptation are becoming more widely recognized (reviewed by Garant *et al.*, 2007). These benefits may include (1) allowing for increased evolutionary potential in changing environments via the spread of advantageous alleles, (2) a reduction of inbreeding depression and maintenance of peripheral populations and (3) increased potential for the evolution of reinforcement (reviewed by Garant *et al.*, 2007). Of these points, (3) would be most likely to specifically increase adaptive divergence between populations (arrow 5 in Fig. 1; Table 1), and not just local adaptation. Yet an increase in adaptive potential caused by gene flow [point (1) above] could also be achieved through an adaptive plastic response, thus reducing any potential benefits of gene flow in that case.

Additional considerations

Maladaptive plasticity

So far I have referred only to plastic responses that are adaptive. Yet in certain cases, plasticity may oppose the direction of adaptive genetic change among populations; this negative covariance between the environmentally induced and genetically based responses is termed 'countergradient variation' (Conover & Schultz, 1995). This response is most likely to occur when an environment is of poor quality (e.g. nutrient deprived, high in toxins), such that individuals not genetically adapted to these poor conditions are not able to develop adequately (but see Röder *et al.*, 2008). For this type of response, a new set of predictions for the relationship among plasticity, selection and gene flow is needed. We expect that (i) genetic adaptation should occur to compensate for the maladaptive plastic response (i.e. genetic compensation; Grether, 2005), and (ii) gene flow should be reduced between selective environments, because well-adapted residents would be favoured over dispersers exhibiting maladaptive plasticity (Fig. 2).

An example of countergradient variation was demonstrated in the plant *Thlaspi caerulescens* (Jiménez-Ambriz *et al.*, 2007). Two different ecotypes occur in the wild, one adapted to soils with high heavy metal pollution. In the field, no differences in most morphological and life-history traits occur between ecotypes. In a lab-rearing experiment, however, ecotypes responded differently to changes in zinc concentration in the soil. The nonmetal tolerant plants responded poorly to high zinc concentrations – some of the effects included poor growth, smaller rosettes, delayed flowering, shorter flowering season, and smaller and fewer seeds (Jiménez-Ambriz *et al.*, 2007). Plants adapted to soils with high metal concentrations did not respond differently to high and low zinc concentrations in the laboratory. This result suggests that despite higher plasticity in the nonmetal tolerant plants, gene flow from these populations towards the metal tolerant populations should actually be selected *against*, because well-adapted residents would be favoured over dispersers
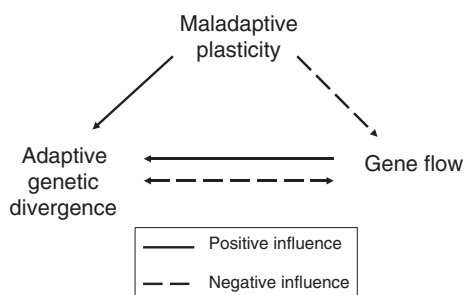
displaying maladaptive plasticity. However, no evidence was found for decreased neutral gene flow between environments (Jiménez-Ambriz *et al.*, 2007). To the best of my knowledge, this is the only study documenting countergradient variation for which gene flow was estimated both between divergent populations and among populations experiencing similar selective pressures –more studies are thus needed to determine if these results are typical.

The above example documents a type of genotype-by-environment (G × E; or more specifically, ecotype-by-environment) interaction, i.e. the slope of the reaction norm differs among genotypes. Plasticity may be adaptive in the face of gene flow if the slopes of reaction norms of two populations occur in the same direction (e.g. Fig. 3a, b) – the plastic response brings the phenotype closer to the optimum in the alternate environment. If the slopes of the reaction norms were in opposite directions, but the rank order of the phenotypes remained the same in each environment (Fig. 3c), plasticity would be beneficial only for genotypes moving in one direction (from environments 1 to 2 in Fig. 3c) – movement in the opposite direction brings the phenotype further from the optimum. If the slopes were in the opposite direction, but the *rank order* of phenotypes differed between environments (Fig. 3d), a genotype may have a less optimal phenotype after the plastic response to the new environment than had it not been plastic. Thus G × E interactions may represent maladaptive plasticity if gene flow occurs between selective environments, and may actually promote a reduction in the gene flow between environments. This would occur if populations from high-quality environments have low fitness in
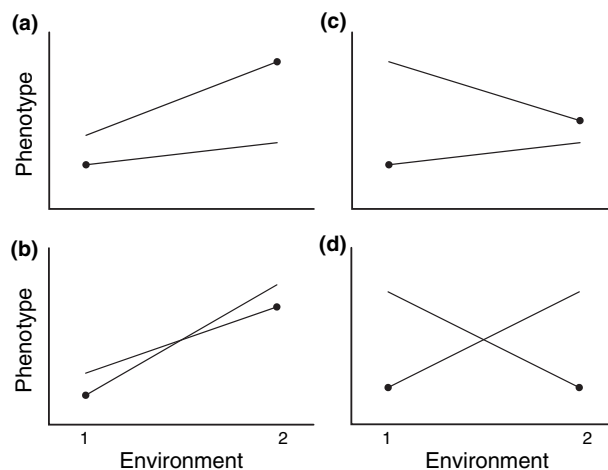
Fig. 3 Reaction norms for two genotypes, one adapted to environment 1 and the other adapted to environment 2. The optimal phenotypic value in each environment is indicated by a dot. Four types of genotype-by-environment interactions are depicted (see text).

Fig. 2 Potential interrelationships among adaptive divergence, maladaptive plasticity and gene flow.

poor-quality environments in relation to individuals adapted to the low-quality environments (as above).

Inferring causality

How can we infer the direction of causality for the bidirectional arrows presented in Fig. 1? Significant correlations between (i) plasticity and adaptive divergence, (ii) plasticity and gene flow and (iii) adaptive divergence and gene flow would suggest that one variable influences the other, or they both influence each other, but tells us nothing of the causal pathway, unless other variables (such as factors posing selective pressures and physical barriers to dispersal) are also considered. Yet, understanding causal pathways may be important when making management decisions or when addressing evolutionary questions. Suggestions for how causality inferences can be made for adaptive divergence and gene flow have already been proposed (Räsänen & Hendry, 2008). For example, a negative relationship between gene flow and environmental dissimilarity may suggest that adaptive divergence constrains gene flow (Table 2; Räsänen & Hendry, 2008). On the other hand, a positive relationship between adaptive divergence and geographical distance or physical barriers to dispersal, while controlling for environmental variation, may suggest that gene flow (regulated by the effects of geographical distance or barriers) constrains adaptive divergence (Table 2; Räsänen & Hendry, 2008). Yet no causality inferences have been made regarding phenotypic plasticity.

Can we make causality inferences regarding interactions between plasticity and adaptive divergence in the absence of gene flow? A positive relationship between adaptive genetic divergence and environmental dissimilarity (i.e. divergent natural selection) may suggest that adaptive divergence should slow the evolution of plasticity (Table 2). We can infer this direction of causality because a correlation between adaptive genetic divergence and environmental dissimilarity would suggest that the environment is acting on the phenotype via natural selection – thus plasticity might not be necessary for allowing individuals to adapt to environmental conditions, or at least plasticity is less important than if adaptive genetic divergence was not apparent. It remains possible, however, that plasticity still occurs and that adaptive divergence may actually be weaker than if plasticity was not present. In this case, plastic change, permitting phenotypic adaptation to novel environments, may have been the precursor for adaptive genetic divergence, although this cannot be determined from point-in-time observations.

In situations in which gene flow occurs between environments, inferences of causality for adaptive divergence and plasticity may be easier. A positive relationship between the degree of plasticity (slope of reaction norm) and gene flow may suggest that plasticity diminishes adaptive divergence through the action of gene flow, or that gene flow diminishes adaptive divergence through the action of plasticity (Table 2). Detecting costs associated with plasticity may suggest that local genetic adaptation would be preferable over plasticity (DeWitt et al., 1998). Alternatively, a positive relationship between plasticity and within-site environmental heterogeneity would suggest that plasticity may actually be locally adaptive (Table 2; e.g. Day et al., 1994; Gianoli & González-Teuber, 2005; Richter-Boix et al., 2006; Lind & Johansson, 2007; reviewed by Alpert & Simms, 2002).

How can we determine if gene flow is promoting phenotypic plasticity, or if plasticity is promoting gene flow? Gene flow should be regulated by geographical features, such as physical distance and barriers to dispersal; yet, plasticity is not expected to be directly influenced by these features. Thus a negative relationship between plasticity (as above) and geographical distance/barriers may suggest that gene flow (regulated by these geographical features) promotes plasticity (Table 2). Alternatively, a positive relationship between plasticity and within-site spatial/temporal environmental heterogeneity (suggesting that plasticity is adaptive; see references above) may suggest that plasticity instead promotes gene flow (Table 2).

I have discussed here ways in which causal pathways between pairs of factors (adaptive divergence, plasticity and gene flow) can be inferred. In most scenarios, it is probable that all three factors modify one another simultaneously. The above interpretations can, however, allow us to infer which directional pathway acts most strongly in a given natural context. Future research

Table 2 Correlations between pairwise factors and the inferred causal pathways among adaptive genetic divergence, phenotypic plasticity and gene flow.

Correlated factors	Type of correlation	Predicted direction of causality
GF and environmental dissimilarity	–	AD (directly related to environmental divergence) constrains GF
AD and barriers to dispersal	+	GF (negatively related to barriers) constrains AD
AD and environmental dissimilarity	+	AD constrains PP
PP and GF	+	PP (acting through GF) constrains AD, or GF (acting through PP) constrains AD
PP and environmental heterogeneity	+	PP = local adaptation; PP promotes gene flow
PP and barriers to dispersal	–	GF (negatively related to barriers) promotes PP

AD, adaptive genetic divergence; PP, phenotypic plasticity; GF, gene flow.

attempting to make causality inferences regarding adaptive divergence, plasticity and gene flow could examine correlations not only among these factors, but also between these factors and spatial/temporal environmental heterogeneity and/or the potential for dispersal (Table 2), and could also examine potential fitness costs associated with plasticity.

Conclusions

I have here provided a framework for understanding how phenotypic plasticity, adaptive divergence and gene flow interact among natural populations, and discussed how plasticity is implicated in studies of gene flow and divergent natural selection. I have highlighted that plasticity can be an important factor in the evolution of diversification; the effects may be either positive or negative, and depend on the nuances of the specific system. First, plasticity can allow individuals to rapidly adapt to new environmental conditions, thereby reducing the strength of divergent selection between populations. Alternatively, plasticity can push populations up new adaptive peaks and promote further diversification. Second, it can promote gene flow between selective environments, which in turn could have positive or negative effects on adaptive divergence. Thus it is important to consider the combined effects of selection, gene flow and plasticity on adaptive divergence in natural systems under the framework presented here.

Acknowledgments

Glossary

Adaptive genetic divergence – Phenotypic divergence between populations as a result of divergent natural selection. Based on genetic changes rather than on environmental induction (and hence is detectable through common garden and reciprocal transplant experiments).

Cogradient variation – Genetic and environmentally induced phenotypic changes that occur in the same direction among populations.

Countergradient variation – Genetic and environmentally induced phenotypic changes that occur in opposing directions among populations.

Developmental plasticity – Environmentally induced change that occurs during the development of an individual and is fixed/irreversible.

Genotype-by-environment interaction – Slopes of reaction norms that differ among genotypes.

Genetic assimilation – A process whereby a phenotype that was once environmentally induced becomes canalized (results in a reduction in the slope of a reaction norm).

Labile plasticity – Environmentally induced change that can occur at any time during the life of an individual and can be reversible.

Local adaptation – Genetic change due to natural selection occurring within a population.

Maladaptive plasticity – Environmentally induced phenotypic change that either decreases fitness in one particular environment, or decreases the average fitness of an individual/genotype across environments.

Phenotypic plasticity – An environmentally induced change in the phenotype, usually expressed as a property of a genotype. Plasticity itself can be heritable and thus evolves.

Reaction norm – Describes the phenotypes produced by a single genotype along an environmental gradient.

Reinforcement – The evolution of mating preferences in response to selection against hybrid offspring between differentiated populations.

Selective barrier – Divergent natural selection that occurs between environments, preventing or reducing the movement of genotypes between these environments.

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