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E-Note

Environmental Change, Phenotypic Plasticity, and Genetic Compensation

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abstract: When a species encounters novel environmental conditions, some phenotypic characters may develop differently than in the ancestral environment. Most environmental perturbations of development are likely to reduce fitness, and thus selection would usually be expected to favor genetic changes that restore the ancestral phenotype. I propose the term "genetic compensation" to refer to this form of adaptive evolution. Genetic compensation is a subset of genetic accommodation and the reverse of genetic assimilation. When genetic compensation has occurred along a spatial environmental gradient, the mean trait values of populations in different environments may be more similar in the field than when representatives of the same populations are raised in a common environment (i.e., countergradient variation). If compensation is complete, genetic divergence between populations may be cryptic, that is, not detectable in the field. Here I apply the concept of genetic compensation to three examples involving carotenoid-based sexual coloration and then use these and other examples to discuss the concept in a broader context. I show that genetic compensation may lead to a cryptic form of reproductive isolation between populations evolving in different environments, may explain some puzzling cases in which heritable traits exposed to strong directional selection fail to show the expected evolutionary response, and may complicate efforts to monitor populations for signs of environmental deterioration.

Keywords: countergradient variation, genetic assimilation, genetic accommodation, cryptic evolution, environmental gradient, canalization.

When a species encounters novel environmental conditions, as can occur, for example, during range expansion, climate change, habitat degradation, or host plant shifts, some phenotypic characters may develop differently than in the ancestral environment. It has long been recognized that environmental perturbations of development have the potential to facilitate adaptive evolution and enable populations to reach new adaptive peaks (Baldwin 1902; Waddington 1953; Mouseau et al. 2000; Price et al. 2003; West-Eberhard 2003). As with genetic mutations, however, most environmental perturbations of development are likely to reduce fitness, at least initially. Natural selection would usually be expected to favor genetic changes that restore the ancestral phenotype or compensate for the environmental change in other ways (Levins 1968; Conover and Schultz 1995). Compensatory evolutionary responses to environmental change have not gone unnoticed by evolutionary biologists, but the phenomenon deserves broader attention than it has received.

"Genetic accommodation" is a general term for gene frequency changes caused by selection in response to environmentally (or genetically) induced changes in the phenotype (West-Eberhard 2003). When environmentally induced changes in the phenotype increase fitness, they may become genetically assimilated (Waddington 1942, 1953, 1961). Genetic assimilation is a form of genetic accommodation in which environmentally induced phenotypes gradually become canalized and develop in the absence of the triggering environmental stimulus (reviewed in Pigliucci and Murrena 2003; West-Eberhard 2003). For example, genetic assimilation of environmentally induced changes in leg length may have occurred during the adaptive radiation of Caribbean *Anolis* lizards (Losos et al. 2000).

Conversely, when persistent environmentally induced changes in the phenotype reduce fitness, the reverse of genetic assimilation may occur. To my knowledge, there is no established general term for this process. I propose the term "genetic compensation," which can be defined simply as the reverse of genetic assimilation or, equivalently, as a form of genetic accommodation in which ancestral phenotypes are restored in the presence of a phenotype-altering environmental stimulus.

Genetic compensation shifts the mean of a trait back toward the ancestral state without necessarily narrowing

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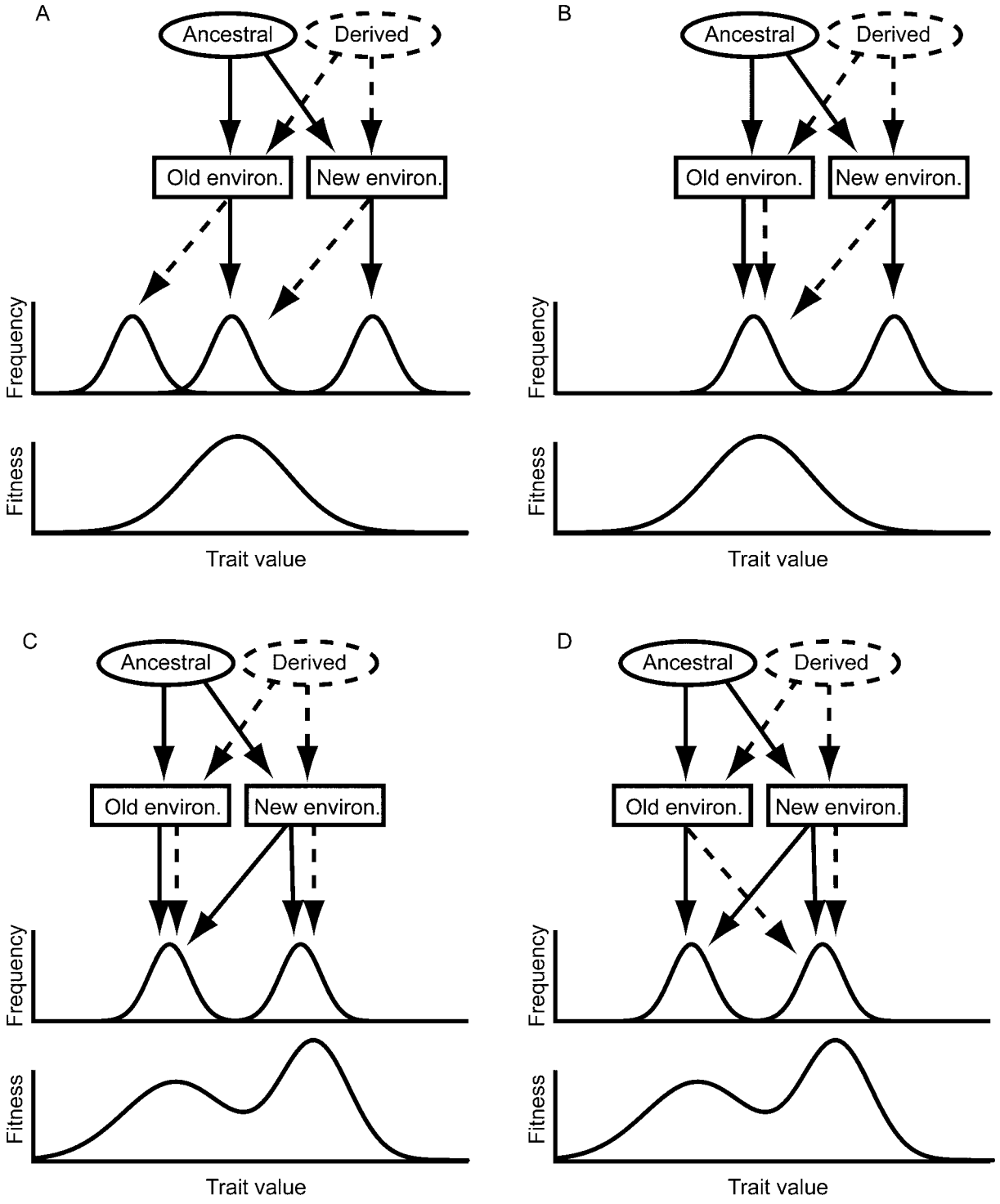

Figure 1: Schematic comparison of genetic compensation without canalization (*A*), genetic compensation with canalization (*B*), partial genetic assimilation (*C*), and full genetic assimilation (*D*). The top graphs show the phenotype distribution under different developmental scenarios, and the bottom graphs show the fitness landscape for a trait of interest. Solid arrow paths show how the ancestral population develops in the old and new environments. Dashed arrow paths show how development proceeds in the derived population after multiple generations of selection. Under genetic compensation (*A*, *B*), the ancestral population develops suboptimally in the new environment. Selection favors genetic changes that shift the mean trait value of the population back toward the fitness peak, thereby restoring the ancestral phenotype. In *A*, individuals in the derived

the norm of reaction to the environmental factor (fig. 1). Thus, unlike genetic assimilation, genetic compensation need not cause increased canalization. Canalization refers to an evolved reduction in developmental flexibility that buffers development of an adaptive phenotype against environmental or genetic perturbations (Waddington 1942; Scharloo 1991; Gibson and Wagner 2000; Rutherford 2000; West-Eberhard 2003). Although genetic compensation reverses the effects of environmental perturbations of development, it does not necessarily lead to increased canalization. Instead, the environmental factor may be incorporated as a normal determinant of trait development. If so, the phenotype would remain sensitive to future changes in the environmental factor, not buffered against environmental change. Genetic assimilation, in contrast, leads to canalization of what began as an environmentally induced developmental pathway. Once genetic assimilation is complete, the environmental factor is no longer required to induce development to proceed down the same pathway (fig. 1). Genetic compensation and genetic assimilation are opposites only in terms of their effect on the fate of novel, environmentally induced phenotypes. Both processes involve natural selection and the accumulation of genetic changes that alter the norm of reaction to an environmental factor.

When genetic compensation has occurred along a spatial environmental gradient, the mean trait values of populations in different environments may be more similar in the field than when representatives of the same populations are raised in a common environment. This pattern is known as countergradient variation (Levins 1968). Most documented examples of countergradient variation involve the effects of altitude, latitude, or temperature gradients on growth or development rates (reviewed in Conover and Schultz 1995; see also Arendt and Wilson 1999; Arnett and Gotelli 1999; Laugen et al. 2003; Skelly 2004). For example, in nature, larvae of the frog *Rana sylvatica* grow more rapidly at lowland sites than at high-elevation sites, but when larvae from the different sites are raised under identical conditions in the laboratory, the mountain larvae grow more rapidly than the lowland larvae (Berven 1982*a*, 1982*b*). In this case, the genetic difference in growth rate, likely caused by past selection for rapid growth in the relatively short mountain growing season, partially masks the influence of the environmental (temperature) gradient. Genetic compensation need not always produce a spatial countergradient pattern, however, because environmental change can occur without a spatial component. For example, climate change could trigger compensatory genetic responses across a species' range.

Genetic compensation differs from other forms of local adaptation in that the same mean trait values may be favored in different environments. If compensation is complete, genetic divergence between populations may be cryptic, that is, not detectable in the field. If genetic compensation is incomplete because of evolutionary time lag or developmental or genetic constraints, the phenotypic differences between populations observed in a common (e.g., laboratory) environment may be in the opposite direction from the phenotypic differences observed in the field (as in Berven's study of *R. sylvatica*). Below I apply the concept of genetic compensation to three examples involving carotenoid-based sexual coloration and then use these and other examples to discuss the concept in a broader context.

Genetic Compensation and Sexual Coloration

Carotenoids are red, orange, and yellow pigments that animals cannot synthesize de novo and usually obtain from food (Goodwin 1984). The amounts and types of carotenoids available in the diet can directly limit the coloration of animals that rely on these pigments (Olson and Owens 1998). Species vary in carotenoid assimilation efficiency, in the pattern of carotenoid deposition in the integument (skin, cuticle, feathers, etc.), in the ability to convert one type of carotenoid into others with different spectral properties, and in their reliance on synthetic pigments (e.g., pteridines). The first example that I review below involves countergradient variation in carotenoid assimilation efficiency, the second involves genetic changes in carotenoid metabolism and deposition that may have followed the appearance of an evolutionarily novel carotenoid in the diet, and the third involves compensatory genetic changes in pteridine production along an environmental gradient in carotenoid availability.

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population are well adapted to the new environment but develop suboptimally in the old environment; the level of canalization remains unchanged relative to the ancestral condition. In *B*, individuals in the derived population develop the ancestral phenotype in both environments, and thus canalization has increased relative to the ancestral condition. Under genetic assimilation (*C*, *D*), the new environment causes some individuals in the ancestral population to develop novel, high-fitness trait values. Selection favors genetic changes that increase the probability of producing the novel trait. In *C*, individuals in the derived population reliably produce the novel trait in the new environment but still produce the suboptimal ancestral phenotype in the old environment. In *D*, individuals in the derived population produce the novel trait in both environments. Development is canalized relative to the ancestral condition in both *C* and *D* but more so in *D*. Note that in *A* and *B*, the ancestral phenotype is favored in both environments while in *C* and *D* the novel phenotype is favored in both environments.

Example 1

Pacific salmon (*Oncorhynchus nerka*) occur in two reproductively isolated morphs: anadromous sockeye, which mature in the Pacific Ocean and return to lakes and rivers to spawn, and nonanadromous kokanee, which remain in freshwater lakes throughout their lives (Craig and Foote 2001 and references therein). Kokanee are thought to have evolved repeatedly from "residuals," a term used for sockeye that fail to migrate to the ocean. At sexual maturity, sockeye and kokanee both display intensely red carotenoid-based breeding coloration, but this similarity in coloration masks an important difference between the environments in which the two morphs develop. Carotenoid availability for salmon is probably much lower in the oligotrophic lakes inhabited by kokanee than in the ocean, where sockeye normally develop (Craig and Foote 2001). Residuals, the ancestral form of kokanee, are largely green at sexual maturity as a result of developing in the low-carotenoid lacustrine environment. Red breeding coloration has repeatedly re-evolved in kokanee, through additive genetic changes in carotenoid assimilation efficiency; kokanee are three times more efficient at assimilating ingested carotenoids than are sockeye (Craig and Foote 2001).

In short, it appears that the environmentally induced change in the phenotype (red to green) that occurs when Pacific salmon become established in freshwater lakes has been reversed repeatedly by genetic compensation, perhaps in response to a hue-based mate preference (Foote et al. 2004).

Example 2

Male western tanagers (*Piranga ludoviciana*) produce scarlet head feathers by blending the deep red carotenoid rhodoxanthin, which the birds obtain directly from food, with metabolically produced yellow carotenoids (canary-xanthophylls). Other species in this genus, including the closely related scarlet tanager (*Piranga olivacea*), produce red carotenoids through metabolic conversion of yellow dietary carotenoids (Hudon 1991). Hudon (1991) proposed that the use of rhodoxanthin by western tanagers is a derived condition, possibly resulting from the inclusion of a new food item in the diet. Rhodoxanthin use may be adaptive in the sense that the metabolic cost of producing scarlet coloration is reduced by using a red dietary carotenoid, compared with the putative ancestral condition of converting yellow carotenoids into red carotenoids (Hudon 1991; Price et al. 2003). Initially, however, consumption of rhodoxanthin probably shifted the coloration of this species away from the optimum phenotype, in terms of both hue and color pattern. When bird species that do not encounter rhodoxanthin in their natural diet consume this pigment, it shows up in any feathers that bear carotenoids (Völker 1955; Mulvihill et al. 1996). For example, the yellow tail tip and belly feathers of cedar waxwings (*Bombycilla cedrorum*) turn orange where these birds consume the rhodoxanthin-rich berries of an introduced honeysuckle (*Lonicera morrowi*; Hudon and Brush 1989; Mulvihill et al. 1996; Witmer 1996). In the western tanager, however, rhodoxanthin is restricted to the head feathers although yellow carotenoids are deposited elsewhere in the plumage. This implies that genetic changes in follicular selectivity or metabolism occurred after this species first began consuming rhodoxanthin (Hudon 1991). Western tanagers appear to have compensated genetically for the use of rhodoxanthin in at least two other ways as well. First, to consistently produce a scarlet hue in the head feathers, production and deposition of yellow canary-xanthophylls must be matched to the availability of rhodoxanthin in the environment because the hue depends on the ratio of the two types of pigments (J. Hudon, personal communication). Second, feather structure and melanin pigmentation of this species differ from those of congeners in ways that compensate for the different spectral properties of the pigments (Hudon 1991).

Example 3

The orange spots of male guppies (*Poecilia reticulata*) contain two types of pigments, yellow carotenoids (mostly tunaxanthins) and red pteridines (drosopterins). Because tunaxanthins and drosopterins have different spectral properties (fig. 2*A*), the ratio of the two types of pigments affects the shape of the orange spot reflectance spectrum (fig. 2*B*) and, presumably, the hue perceived by females (Grether et al. 2001*a*, 2005). The primary source of carotenoids for guppies in nature is unicellular algae, the availability of which is largely a function of the openness of the forest canopy (i.e., light availability), which in turn is largely a function of stream width (Grether et al. 1999, 2001*b*). Orange spot carotenoid content varies with carotenoid availability (Grether et al. 1999), but the ratio of the two types of pigments in the orange spots is roughly conserved across streams (fig. 2*C*; Grether et al. 2001*a*). Laboratory common environment experiments revealed that variation in the hue of the orange spots is reduced in the field as a direct consequence of genetic differentiation between populations in drosopterin production (fig. 2*D*; Grether et al. 2005). Thus, genetic differences between populations in drosopterin production mask the effect of the carotenoid availability gradient on the hue of the or-

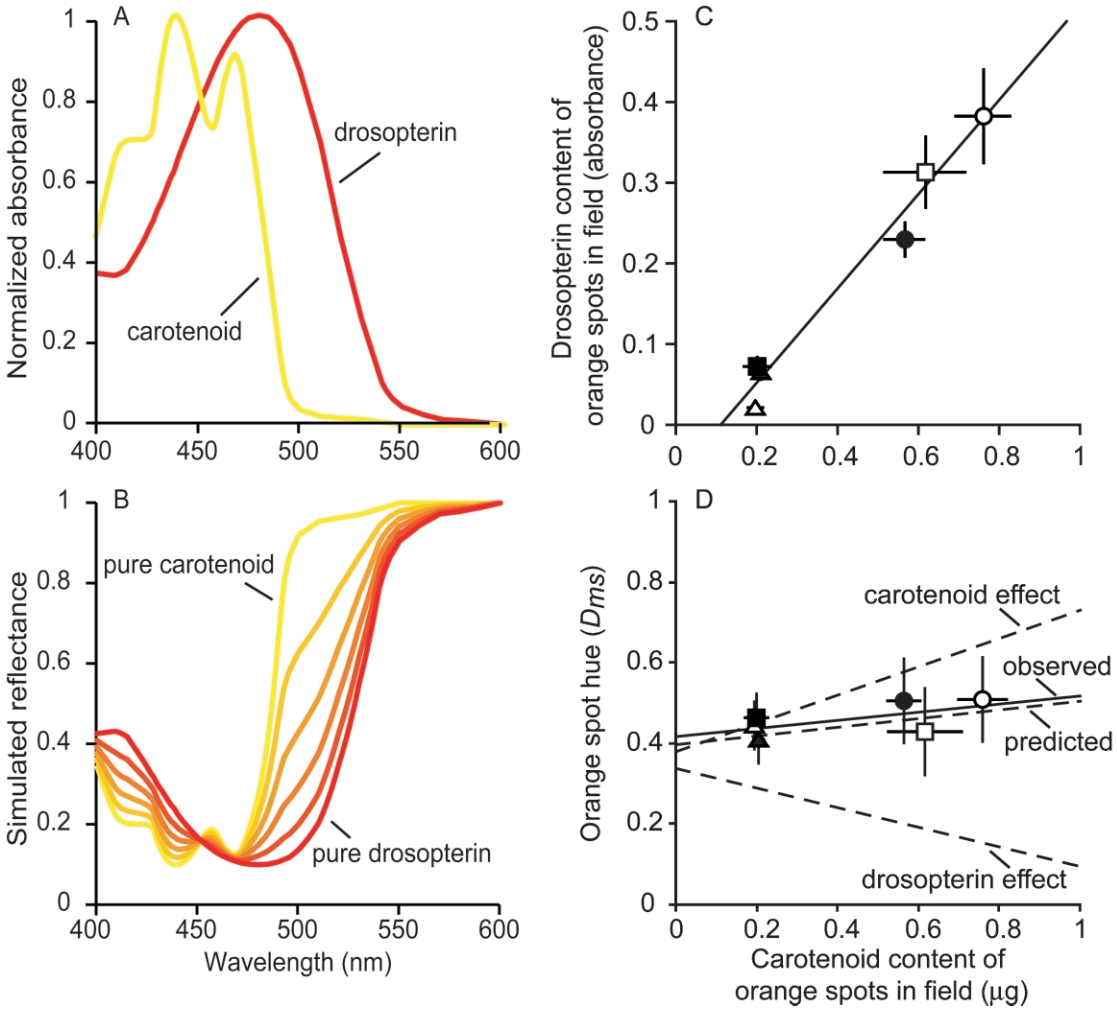

Figure 2: Evidence for genetic compensation in the sexual coloration of guppies. *A*, Normalized absorbance spectra of the two types of pigments (carotenoids and drosopterins) in the orange spots of male guppies. *B*, Simulated reflectance spectra for different carotenoid : drosopterin ratios. *C*, Drosopterin content of the orange spots co-varies positively with the carotenoid content across populations in the field. *D*, Measure of orange spot hue based on photoreceptor contrasts (D_{ms}) is conserved across populations in the field because of the counterbalancing effects of drosopterins and carotenoids. The solid line in *D* represents the least squares regression through the observed population means; the upper and lower dashed lines represent the effect of each pigment separately on D_{ms}; the middle dashed line represents the predicted values of D_{ms} given the observed orange spot pigment content means (see Grether et al. 2005 for further details). Plotted points represent population means ($\pm$SE). *A–C* were adapted from Grether et al. (2001*a*), and *D* was adapted from Grether et al. (2005).

ange spots. A logical, but as yet unverified, explanation for these results is that female guppies prefer males with a particular orange hue.

As in Pacific salmon, the low-carotenoid-availability form of guppies has apparently evolved repeatedly from the high-carotenoid-availability form, as these fish have colonized (and recolonized) the headwaters of different river drainages (carotenoid availability is lowest in small headwater streams; Grether et al. 1999, 2001*b*).

Although the taxonomic scales in these examples differ, in each case genetic changes can be inferred to have compensated for the effect of changes in an environmental factor (carotenoid availability) on the phenotype (color). These inferences were possible because taxa representing the putative ancestral and derived conditions were available for comparison. As with genetic assimilation, most examples of genetic compensation are probably lost in time. Moreover, because genetic compensation is a cryptic

process (i.e., it masks phenotypic differences between populations and species), it is inherently difficult to detect.

Genetic Compensation versus Genetic Assimilation

Genetic compensation and genetic assimilation are both caused by natural selection and involve genetic responses to environmental perturbations of trait development, but they have opposite effects on the development of the affected trait. Perhaps the most easily made distinction is that genetic assimilation leads to a change in the phenotype, relative to the ancestral condition, while genetic compensation reduces or eliminates phenotypic change (fig. 1). In the past, the term "genetic assimilation" has occasionally been used to refer to both processes, but I think this is confusing and inconsistent with the original meaning of the term. For example, the evolutionary scenario described above for the western tanager has been presented as an example of genetic assimilation (Price et al. 2003). Waddington (1961, p. 289), however, defined genetic assimilation as "a process by which a phenotypic character, which initially is produced only in response to some environmental influence, becomes, through a process of selection, taken over by the genotype, so that it is found *even in the absence of the environmental influence which had at first been necessary*" (emphasis added). The western tanager example does not fit this definition because, according to the evolutionary scenario proposed by Hudon (1991), rhodoxanthin was not needed to produce scarlet coloration initially but became necessary after selection restored scarlet coloration in the presence of rhodoxanthin. This is the reverse of the process Waddington described. Likewise in guppies and salmon, the environmental influence—geographic variation in carotenoid availability—has become necessary to maintain hue constancy across populations. When fish from different populations are raised on a single level of carotenoids in the laboratory, hue constancy is disrupted (Craig and Foote 2001; Grether et al. 2005).

In short, genetic compensation causes phenotypic stasis on an evolutionary timescale but can leave development open to environmental perturbation, while genetic assimilation causes phenotypic change on an evolutionary timescale but canalizes development against environmental perturbation. Both processes belong in the larger category of environmentally (as opposed to genetically) induced forms of genetic accommodation (West-Eberhard 2003).

Cryptic Reproductive Isolation

Genetic compensation has the potential to lead to a cryptic form of reproductive isolation between populations evolving in different environments. Consider, for example, that a low-drosopterin male guppy who dispersed downstream over a barrier waterfall as a juvenile, from a low-carotenoid-availability site to a high-carotenoid-availability site, would develop orange spots with an abnormally high carotenoid : drosopterin ratio. Upstream male migrants, on the other hand, would develop abnormally low carotenoid : drosopterin ratios. If female guppies indeed prefer males with orange spots of the normal hue, male interpopulation migrants would suffer a mating disadvantage and so would their hybrid male offspring. This form of incipient reproductive isolation could not be detected, however, with standard laboratory mate choice tests. To be valid, mate choice tests would have to be carried out using true interpopulation migrants or fish raised on diets designed to precisely match the carotenoid availability that a migrant would encounter.

In Pacific salmon, nonanadromous hybrids between sockeye and kokanee occur at low frequency in lakes where the two morphs spawn sympatrically (Craig et al. 2005). The hybrids are viable and fertile but have carotenoid assimilation rates intermediate between the two parental morphs and develop green coloration at maturity (Craig et al. 2005). Mate choice tests suggest that the abnormal coloration of the hybrids substantially reduces their mating success with both parental morphs (Foote et al. 2004). Had genetic compensation not occurred in kokanee, they would still develop green coloration at maturity and presumably would not discriminate against hybrids on the basis of color on the spawning grounds. Thus, this may be an example of genetic compensation promoting population divergence in sympatry (i.e., reinforcement).

The soapberry bug *Jadera haematoloma* provides another example of how genetic compensation could contribute to reproductive isolation. In Florida, soapberry bugs are found in two geographically adjacent races, the putative ancestral race, which uses a native plant (*Cardiospermum corindum*) as its host, and a derived race, which uses an introduced plant (*Koelreuteria elegans*) as its host (Carroll et al. 2001 and references therein). The derived race has diverged from the ancestral race in morphology, but time from hatching to adult metamorphosis (development time) appears to have changed very little. When bugs from either race were reared on the other race's host plant, however, development time increased significantly (fig. 3). Carroll et al. (2001) inferred that the derived race initially suffered reduced performance on the introduced host but eventually evolved back to the ancestral development time through genetic changes in physiology (i.e., genetic compensation) and that these (and other) genetic changes reduced the performance of the derived race on the native host. Therefore, immatures from eggs laid by a female on the "wrong" host plant would develop abnormally slowly, reducing the probability that the off-

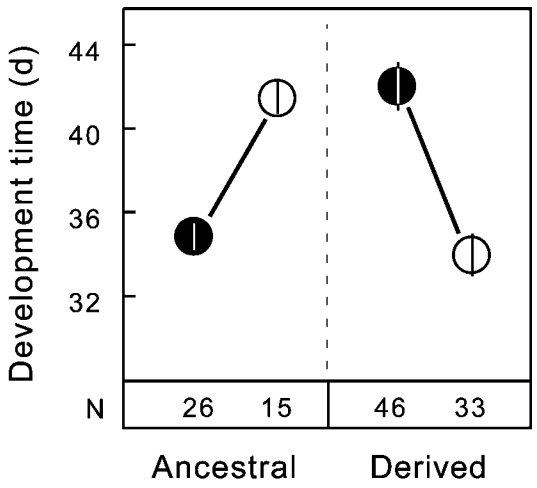

Figure 3: Evidence for genetic compensation in the development time of soapberry bugs. The plotted values are means (±SE) from cross-rearing experiments in which individuals from the putative ancestral and derived races were reared from hatching to adult metamorphosis on seeds of the native host plant (*filled circles*) or on those of the introduced host (*open circles*). Adapted with permission from Carroll et al. (2001).

spring would survive to mate and reproduce with members of the other race.

These examples differ from the usual scenario of local adaptation reducing gene flow between populations in that the phenotypic traits of interest (guppy orange spot hue, salmon nuptial coloration, soapberry bug development time) may have the same optima in the different environments and interpopulation migrants would develop phenotypes that are suboptimal in both environments. In the usual scenario of local adaptation reducing gene flow, different trait values are optimal in different environments, and interpopulation migrants develop phenotypes suited to the environment of their origin.

Evolutionary Stasis and Biological Monitoring

Genetic compensation may help explain some puzzling cases in which heritable traits exposed to strong directional selection fail to show the expected evolutionary response (Price et al. 1988; Alatalo et al. 1990; Cooke et al. 1990; Hosken 2001). For example, Merilä et al. (2001) showed that the mean relative body weight at fledging of collared flycatchers (*Ficedula albicollis*) declined from 1980 to 2000 despite a strong positive selection differential and significant heritability for this trait. Quantitative genetic analyses showed that breeding values for relative body weight were increasing over the same time period but not rapidly enough to compensate for environmental deterioration in the study area. Merilä et al. (2001, p. 76) interpreted this as an example of a "genetic response to selection … [being] masked by opposing changes in the environment." An alternative explanation, however, is that the change in the environment was being masked by opposing selection on relative body weight (i.e., genetic compensation). The wording used by Merilä et al. (2001) implies that the association between directional selection on relative body weight and environmental deterioration was coincidental. Under this hypothesis, the mean relative body weight of the birds would have increased during the study if the environment had not been deteriorating. By contrast, under the genetic compensation hypothesis, directional selection on relative body weight was caused by environmental deterioration, and thus the mean relative body weight of the birds would not have changed if the environment had remained static.

The collared flycatcher example illustrates that genetic compensation could complicate efforts to monitor populations for signs of environmental deterioration. If the traits monitored (e.g., condition indices, growth rates, hormone levels) are heritable, mean trait values may not decline as rapidly as the rate of environmental deterioration.

Conclusion

In her landmark book, *Developmental Plasticity and Evolution*, West-Eberhard (2003) discusses genetic accommodation at length and clearly intends this term to encompass the process that I call genetic compensation (p. 154). However, no examples of genetic compensation are given in the book, and countergradient variation is not discussed. I mention these omissions not to disparage West-Eberhard's contribution but rather to highlight the fact that genetic compensation, by any name, is a neglected topic. As illustrated by the examples above, the concept of genetic compensation can provide valuable insights into how trait development evolves to make particular environmental and particular genetic inputs essential to produce a particular end result. Genetic assimilation undoubtedly has greater potential to promote the evolution of novel traits than does genetic compensation. Nevertheless, given that most novel phenotypes are likely to have low Darwinian fitness, genetic compensation may be far more common than genetic assimilation. Although genetic compensation causes phenotypic stasis in the short term, the underlying genetic changes may reduce gene flow between populations in different environments. Thus, genetic compensation has the potential to facilitate further local adaptation and population divergence. Moreover, genetic compensation provides a mechanism for different developmental pathways to the same phenotype to evolve under natural selection within a single species. In repeated cycles of environmental change and genetic compensation,

the developmental pathway to a given phenotype may diverge even between populations subject to the same environmental fluctuations because different mutations and allele combinations are likely to arise in different populations. Finally, genetic compensation may be of some practical importance; understanding how organisms adapt to deleterious environmental perturbations of development may be pertinent to predicting and monitoring the consequences of environmental change.

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