# Environmental Tolerance, Heterogeneity, and the Evolution of Reversible Plastic Responses

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abstract: Phenotypic plasticity is a key factor for the success of organisms in heterogeneous environments. Although many forms of phenotypic plasticity can be induced and retracted repeatedly, few extant models have analyzed conditions for the evolution of reversible plasticity. We present a general model of reversible plasticity to examine how plastic shifts in the mode and breadth of environmental tolerance functions (that determine relative fitness) depend on time lags in response to environmental change, the pattern of individual exposure to inducing and noninducing environments, and the quality of available information about the environment. We couched the model in terms of prey-induced responses to variable predation regimes. With longer response lags relative to the rate of environmental change, the modes of tolerance functions in both the presence or absence of predators converge on a generalist strategy that lies intermediate between the optimal functions for the two environments in the absence of response lags. Incomplete information about the level of predation risk in inducing environments causes prey to have broader tolerance functions even at the cost of reduced maximal fitness. We give a detailed analysis of how these factors and interactions among them select for joint patterns of mode and breadth plasticity.

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Phenotypic plasticity is the ability of a genotype to change its phenotype in response to different environmental conditions (Bradshaw 1965). Although originally rooted in evolutionary biology, phenotypic plasticity is a broad, interdisciplinary topic, which lies at the intersection between most of today's important biological disciplines (reviewed in Pigliucci 2001 and DeWitt and Scheiner 2004). Phenotypic plasticity occurs in traits ranging from morphology to physiology and behavior and can be observed in nearly all classes of organisms (reviewed in Tollrian and Harvell 1999*a*). Understanding the selective advantage and the limits of plasticity (i.e., why and how individual organisms respond to environmental change) is of critical importance for numerous issues in ecology and evolution (Pigliucci 2001; Abrams and Matusda 2004; Vos et al. 2004).

Types of phenotypic plasticity can differ a great deal in their relative speed and reversibility of change (see Tollrian and Dodson 1999). At one extreme, some plastic changes might be relatively slow and irreversible. For example, in systems with discrete polyphenisms (e.g., alternative morphotypes or castes), a single genotype can develop very different morphologies depending on the environment during development (e.g., Greene 1989). Once set during development, these basic morphotypes show little if any further plasticity. At the other extreme, behavioral changes are thought to be very rapid and infinitely reversible.

Most plastic traits, however, fall somewhere in between the two extremes of being irreversible or being instantaneously reversible. For example, even though morphological defenses are typically thought to be irreversible (e.g., bent morphs in barnacles; Lively 1986), in some cases they can be reversed during subsequent periods of growth. Cladocerans can reverse helmets, spines, or neckteeth with subsequent molts (Tollrian 1993). With cell division, ciliates can reverse an induced winged morph back to a typical morphed cell (Kuhlmann and Heckmann 1985; Kuhlmann et al. 1999). Finally, predator-induced changes

in body or tail depth in fish or amphibian larvae are reversible with further growth (Brönmark and Miner 1992; Brönmark et al. 1999; Relyea 2003).

Likewise, while behavioral responses are often rapid and highly reversible, they can lag behind environmental changes or have less than infinite reversibility. Time lags between environmental changes and when individuals respond behaviorally have long been noted (Tinbergen 1960; Getty and Krebs 1985; Sih 1992). The primary explanation for these lags has been that individuals receive incomplete information about their environment, and thus it takes time for them to detect environmental changes. In addition, the reversibility of behavioral responses can be reduced by earlier experiences that set behavioral tendencies (behavioral syndromes; Sih et al. 2004). For example, experiences with danger can cause prey to be fearful (Boissy 1995) and thus relatively inactive even at future times when predators are absent. These behavioral carryovers can reflect relatively fixed physiological or morphological traits that are set by early behaviors. For example, if prey reduce their activity and hide in refuge for long periods, they might develop low metabolic rates or morphologies that make them less well adapted for high activity later in life.

Interestingly, although patterns of plasticity appear to show a full range of degree of reversibility and speed of response, models of plasticity fall into two main camps that consider the extremes of speed and inducibility of response (Sih 2004). The adaptive plasticity (AP) approach examines irreversible, relatively slow responses to environmental variation (Gabriel and Lynch 1992; Scheiner 1993; Via et al. 1995), whereas the behavioral ecology (BE) approach looks primarily at rapid behavioral responses to varying environments (Stephens 1987; Lima 1998). The BE approach in some cases has incorporated response lags due to incomplete information (McNamara and Houston 1980; Stephens and Krebs 1986; Luttbeg and Schmitz 2000). However, these models have focused on the timing of behaviors rather than how time lags affect what behaviors individuals use. These different scenarios of reversibility and the speed of responses have been addressed using fundamentally different modeling approaches (quantitative genetics for AP vs. optimality theory for BE) that can thus produce very different predictions about plasticity.

Given that phenotypic plasticity can show significant response lags and that induced traits can be reversible, we clearly need theory that addresses reversible plasticity with response lags (Piersma and Drent 2003). Not only would such theory apply to many organisms that do not fit existing theory, but also it should provide a bridge between the fields of behavioral ecology and adaptive phenotypic plasticity. Two previous models addressed reversible plasticity with response lags. Padilla and Adolph (1996) showed that a plastic strategy does poorly in comparison to fixed strategies if the time lag between the environment changing and the individual responding is long relative to the rate of environmental change. However, the comparison was only between two specialist strategies optimized for either of two environments and a generalist strategy that switches between these specialist strategies. Gabriel (1999) assumed that reversible plasticity in trait values results in fitness effects that can be described as reversible plastic changes in the mode and breadth of an environmental tolerance function induced by the environment. (Using a Gaussian fitness function, mean and standard deviation correspond to mode and breadth.) In this model, under a broad range of conditions, reversible plasticity was favored over both no plasticity and irreversible plasticity. In this model, however, the noninduced values of mode and breadth were not allowed to evolve but were kept fixed to the values of nonplastic genotypes. Under these conditions, plasticity in the breadth of adaptation seemed to be of minor importance. Gabriel (1999) did not consider temporal variation of the inducing environment nor the reliability of the inducing cue, two components that might be quite relevant for the understanding of the evolution of reversible plasticity.

Here, we present three major extensions of the model of Gabriel (1999). First, the noninduced values of mode and breadth are no longer fixed to the values of nonplastic genotypes, which allows concerted evolution of induced and noninduced values of mode and breadth. Second, temporal variation of the inducing environment is explicitly modeled. Third, the reliability of the inducing cue is considered for the two extreme cases of incomplete and complete information.

With this extended model, we explore how response lags, patterns of environmental variation, and information available to organisms affect optimal patterns of reversible plasticity in terms of both the mean and breadth of tolerance functions. Our model should be suitable for all kinds of reversible plasticity. However, to keep our discussion from being too abstract, we focus on prey responses to variations in predation risk, particularly on inducible defenses. Inducible defenses are widespread, with examples from unicellular organisms to vertebrates (Tollrian and Harvell 1999*a*; Relyea 2001). Predation risk is the varying environmental state, and its impact on fitness is described by a tolerance function. Prey exhibit a reversible plasticity where they can switch their phenotypes back and forth in response to the changes in predation risk. We examine how time lags between changes in predation risk and the responses of prey affect the optimal mode and breadth of their tolerance functions. We outline and describe the results of a model of optimal patterns of reversible prey plasticity, summarize our main predictions,

and discuss the implications of our model for theoretical and empirical studies of plasticity.

## Reversible Plasticity: Model Concept and Assumptions

Consider first a simple, general scenario where predation risk is the environmental gradient $\phi$ with two main states: a noninducing state and an inducing state. The noninducing state is when too few predators are present to evoke a response from prey, and the inducing state is when enough predators are present to evoke a response. We define the state of the noninducing environment as 0. (Note that $\phi = 0$ does not mean that the predation risk is 0, but it is low compared with induced states. Therefore, the tolerance function is defined also for negative values $\phi$ on this scale.) As predator density increases and decreases, prey exhibit reversible plasticity with induced and noninduced phenotypes. Because there is a time lag before prey can attain their full plastic response, the match between prey phenotype and the environmental state follows a sequence with four conditions labeled as periods a, b, c, and d (see fig. 16.2 in Gabriel 1999). Setting the total time budget to 1, period a is when predators are rare and prey exhibit the appropriate phenotype for the noninducing environmental state. Period b occurs after predator abundance has increased (the inducing environmental state) but before prey have shifted to their induced phenotype. Period c follows with the inducing environmental state and prey having shifted to their induced phenotype. Finally, period d is when predators are rare again (the noninducing environmental state), but prey have not yet returned to their noninduced state. After period d, prey returns to the conditions of period a. Prey fitness for a given genotype is the multiplicative product of its fitnesses from the four periods weighted by their relative durations. Prey fitness in each of the four periods depends on a complex mix of phenotypic traits (behavior, size, shape, other morphological characters, etc.) and how they match various environmental selection pressures. Rather than attempt to explicitly model these plastic traits, we combine their effects on fitness into Gaussian environmental tolerance curves that plot a given phenotype's fitness on an environmental gradient. In our model, the plastic responses of prey are translated into changes in their environmental tolerance curves.

The model not only is suitable for the simple scenario of two environmental states outlined above but also will handle a continuum of environmental states. For example, if predation risk changes from instant to instant, the induced phenotype might also be altered to best fit the current environment. During its lifetime, an organism might express a variety of phenotypes depending on various environmental cues. How well a phenotype is adapted to various environmental settings is described by a unimodal fitness function on an environmental gradient. After a proper scale transformation of the environmental gradient, this function is a Gaussian curve so that its mode ( = mean) and breadth ( = SD) are sufficient to determine the fitness function. Lynch and Gabriel (1987) termed this the environmental tolerance function. The mode is the environmental state that the organism is best adapted to (which may or may not be the environment that it is found in). A small value of the breadth (i.e., a narrow fitness function) represents a more specialized strategy, while a broad fitness function is more generalized. Both the mode and the breadth of the environmental tolerance curves are reversibly plastic.

Lynch and Gabriel (1987) thought in their model on environmental tolerance primarily about abiotic factors as environmental gradients. Additional considerations arise when the environmental gradient is a biotic factor. For example, in predator-prey systems, the evolution of inducible defenses might select for changes in behavior and morphology of the predator (Kopp and Tollrian 2003). Our model is not suitable to describe such coevolutionary processes that would modify the predation risk. The predictions of the model are valid only on a timescale for which evolutionary changes of predators can be neglected. The predator behavior might be highly complex, and there might be feedback from prey to predator; for example, predators' behavior might depend on the amount of inducible defense present. The model can handle such a system as long as this complex behavior is calculable, and results in a change in prey fitness can then be expressed by a unimodal function. We consider the environmental state variable, $\phi$, to be a reliable measure of predation risk that depends on predator density. If prey plasticity feeds back to affect future predator density, then the model could be extended to consider population dynamics. Here, we do not include this feedback; thus, our model is most suitable for situations where predator density is determined largely by external factors.

In this article, we extend the model of Gabriel (1999) by including variation in the environmental state and by considering the quality of information available to prey. Gabriel (1999) started from a nonplastic genotype that is adapted only to a predator-free environment and asked for the advantage of reversible plasticity under predation risk. The genotypes were allowed to plastically express phenotypes that are adapted to an inducing environment, but the noninduced phenotype was kept identical to the nonplastic genotype. In contrast to Gabriel (1999), we start from a nonplastic genotype that is constitutively defended by being adapted to the mean environment including predation risk. Further, we also optimize the noninduced values of plastic genotypes. Therefore, noninduced values of

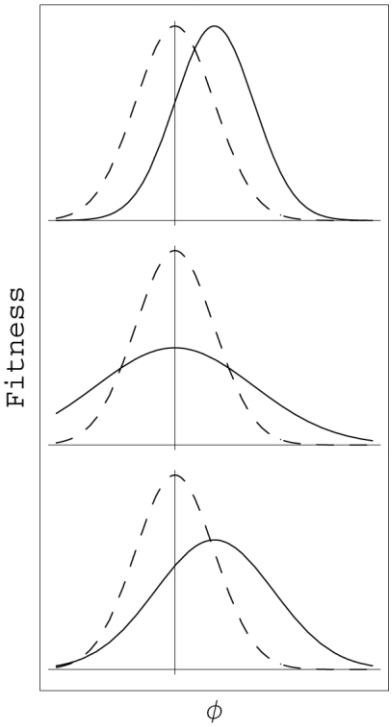


**Figure 1:** Tolerance curves that differ in the mode and the breadth of adaptation. *Top*, plasticity in the mode. *Middle*, plasticity in the breadth of adaptation. *Bottom*, plasticity in mode and breadth. *Solid line*, plastic genotype. *Dashed line*, nonplastic genotype and noninduced plastic genotype.

mode and breadth of plastic genotypes can and will differ from the values of nonplastic genotypes.

To keep our model as simple as possible, we avoid complications arising from treating mode and breadth of tolerance curves with all the details of quantitative traits (e.g., we do not account for developmental noise). Further, we do not consider explicitly any kind of spatial heterogeneity.

During period a, when predators are rare and prey are in the noninduced state, prey fitness is defined by the value of its noninduced fitness function at $\phi = 0$. During period c, when predators are abundant and prey are in the induced state, prey fitness is the value of its induced fitness function at $\phi$. The interesting conditions are where prey are poorly adapted to their environments because of response lags. In period b, prey suffer the lowered fitness of being in the noninduced state when predation risk is high ($\phi$), and in period d, prey suffer the fitness cost of being in the induced state when predation risk is low ($\phi = 0$).

As in Gabriel (1999), we assume a specialist/generalist trade-off, that is, a broad fitness function comes at a cost of reduced maximum fitness at the mode. This trade-off is quantified without introduction of an extra parameter by assuming that the total area under the fitness curve is a constant (see Lynch and Gabriel 1987). Thus, an increase in the breadth of the fitness curve requires a reduction in the height of the fitness curve.

The degree to which individuals are mismatched with their environment depends on the environmental variation and the information prey receive about the environmental changes. We consider two scenarios: first, predation risk varies and prey have complete, accurate information about predation risk; second, predation risk is variable, but prey have information only about the long-term level of predation risk in inducing environments (incomplete information).

Finally, in order to better understand the relative importance of plastic shifts in the mode and breadth of tolerance curves, we examined the net benefits associated with two forms of reversible plasticity (see fig. 1): first, shifts in only the mode of the environmental tolerance functions, holding the breadths constant (m-plasticity); second, plasticity in both the mode and breadth of tolerance functions (m-b-plasticity).

## What Do Shifts in the Mode and Breadth of Fitness Functions Mean?

To understand our model framework and results, it is necessary to have a clear understanding of the meaning of shifts in the mode and breadth of environmental tolerance functions. As noted above, the mode of a fitness function is the environmental state that the organism is best adapted to, and the breadth of the function is the degree to which the individual is specialized (narrow breadth) or generalized (large breadth) in its range of adaptation. For narrow tolerance functions, a mode shift represents a shift from one specialized adaptation to another, while an increase in breadth represents a shift from a specialist to a generalist strategy (see fig. 1).

Mode shifts occur when conflicting traits are favored in different environments. In the context of antipredator traits, mode shifts might be associated with responses to different types of predators. For example, *Daphnia* show opposite, adaptive plastic changes in life history in response to vertebrate versus invertebrate predators (Stibor 1992). *Daphnia* respond to chemical cues from fish, which hunt visually and select larger daphnids, by shifting their energy allocation from growth to reproduction, thus reaching maturity earlier at a smaller size. They additionally produce more but smaller offspring, which again reach maturity at a smaller size (Lampert 1993). In contrast, invertebrate predators, such as larvae of the phantom midge *Chaoborus*, select smaller daphnids. In response to *Chaoborus*, *Daphnia* shift their life history toward growth.

They thus reach maturity later at a larger size and produce fewer but larger offspring, which again reach maturity at a larger size. Each set of plastic life-history changes increases daphnid fitness in the presence of one type of predator while reducing their fitness in the presence of the other (Taylor and Gabriel 1992, 1993).

An increase in the breadth of environmental tolerance functions without a mode shift occurs when increased plasticity enhances fitness in the induced environment but the organism still enjoys its highest fitness in the original noninducing environment. This might be common when the environmental gradient is simply the magnitude of predation risk and especially when the corresponding phenotypic shift has a limited defensive effect. Although prey-induced defenses result in reduced predation rates (relative to no antipredator plasticity), prey usually still have higher fitness in the absence of predators than in the presence of predators because of costs associated with the defense formation and because the defense reduces but does not eliminate mortality caused by predation. For example, the formation of morphological defenses like helmets or neckteeth in *Daphnia* offers protection when predators are present but does not switch the mode of the fitness function. Behavioral examples include sensitization to predator cues that increase alertness to predators in *Daphnia* (Pijanowska 1994), which increases their ability to escape predators (Brewer et al. 1999) at the cost of a higher number of unnecessary escape responses.

Alternatively, plasticity in the breadth of an environmental tolerance function can be visualized as a risk-spreading strategy. For example, if prey are threatened by contrasting predation regimes that select for either small or large prey, and prey receive incomplete cues about predation risk that give no information about whether large or small prey are currently at higher risk, then an adaptive strategy that broadens the tolerance function would be to produce a high variance in offspring size.

Many plastic responses may be composed of shifts in both the mode and breadth of environmental tolerance functions. In addition, the effect of a given plastic response on changes in mode and breadth can depend on other selective forces outside of the main environmental gradient. For example, the type of shift in a tolerance function can depend on the intensity of competition (Relyea 2002). In the absence of strong competition, induced prey responses to enhanced predation risk can cause a shift in breadth, not a shift in the mode; that is, even when prey exhibit their induced phenotype, prey still probably have higher fitness in the absence rather than the presence of predators. If, however, competition is particularly intense in the predator-free environment (Menge and Sutherland 1976; Sih et al. 1985), then prey that display the predator-induced phenotype might have a reduced fitness in the predator-free environment or, alternatively, an increased fitness in the predator environment because of exclusion of noninduced competitors. For example, in a predator-free environment, prey might face strong competition that requires high feeding activity, whereas in the presence of predators, prey must reduce their activity to hide from predators (Werner and Anholt 1993; Anholt and Werner 1995). Each activity level would result in poor performance in the other environment. If the best environment for noninduced prey is the predator-free one, while for induced prey, their best environment is one with predators, the overall shift would likely be a combined shift in mode and breadth. This could even be a pure shift in the mode if competition would narrow the breadths of the tolerance curves to be equal in the presence versus absence of predators.

## Methods and Notations

We use the following procedure to find optimal modes and breadths of tolerance functions. For a given scenario, we first calculate fitness as it depends on free parameters. We obtain the optimal values of these parameters by maximizing the long-term geometric mean fitness of a given genotype. For free parameters, we use the noninduced and induced values of the mode and breadth of the tolerance function. Fitness depends on the following fixed parameters: time delays for response, relative length of the inducing period, the value of the inducing environmental state (e.g., current predation risk), and its temporal variance. We calculate fitness for the following genotypes: nonplastic, plastic in the mode only, and plastic in mode and breadth. The fitness for plastic genotypes depends also on the reliability of the inducing environmental cue. In our example of predator-prey systems, how much information prey have about predation risk affects the modes and breadths of tolerance functions. Complete and incomplete information are analyzed as extremes. Incomplete information means that prey know when there is enhanced predation risk but cannot estimate the amount of predation risk. Details of the derivation of the formulas and additional formulas are given in the appendix in the online edition of the *American Naturalist*.

All variables and indexes used are listed in table 1. We will use the indexes "m," "mb," and "f" to distinguish the kinds of plasticity; complete or incomplete information is indicated by "c" or "i." To discriminate the noninduced and induced values, we use as indexes "n" and "p." The period where a plastically shifted phenotype is selectively advantageous is denoted as $t_s$; $t_s$ is the period where the predator is present and is composed of the period where the defense is building ($t_b$) and the period where the defense is completely formed ($t_c$). The period needed to per-

**Table 1:** Variables and indexes

| Variable or index | Definition |
|---|---|
| $b$ | Breadth of tolerance function |
| c | Complete information |
| $CV_\phi$ | Coefficient of variation of $\phi$ |
| f | Fixed, nonplastic |
| i | Incomplete information |
| $m$ | Mode of tolerance function |
| m | m-plastic: mode plastic |
| mb | m-b-plastic: mode and breadth plastic |
| n | Noninduced |
| p | Induced |
| $t_r$ | Reaction time |
| $t_s$ | Stress time |
| $w$ | Relative fitness |
| $\phi$ | Environmental state |
| $\bar{\phi}$ | Arithmetic mean of $\phi$ |
| $\phi_{geo}$ | Geometric mean of $\phi$ |

form the reversible plastic shifts is denoted as reaction time $t_r$ and is the sum of the time periods b and d. For the analysis, the simplifying assumption $t_b = t_d = t_r/2$ is made in order to reduce variables.

From these definitions of $t_r$ and $t_s$ follows the obvious constraint $t_r < 2t_s$ because for larger $t_r$, a plastic shift would become disadvantageous. The time periods $t_r$ and $t_s$ are measured relative to a total time budget of 1. Besides its obvious interpretation for a single cycle of predator appearance, the contribution of time intervals to fitness (geometric mean fitness; see appendix) imply that $t_s$ and $t_r$ also give the relative time averaged over the whole life span.

In the following, we will use $\bar{\phi}$ and $\sigma_\phi^2$ as mean and variance of $\phi$ when predators are present and $CV_\phi = \sigma_\phi/\bar{\phi}$ as the corresponding coefficient of variation. This means that for calculating mean and variance, we exclude all values $\phi = 0$ and its small variations not caused by the event or cue under investigation.

## Results

### *The Mode and Breadth of Nonplastic Genotypes*

The optimal mode of the tolerance curve of a nonplastic genotype is

$$m_f = t_s\bar{\phi}, \tag{1}$$

which is equal to the average environment, given our assumption that the safer environment ($\phi = 0$) has a duration of $1 - t_s$. The breadth of this optimal nonplastic tolerance curve is

$$b_f = \sqrt{(\bar{\phi})^2}\sqrt{t_s(CV_\phi^2 + 1 - t_s)}. \tag{2}$$

The dependence of $b_f$ on the proportion of time that predators are present is shown in figure 2 for various coefficients of variation of $\phi$.

### *The Induced and Noninduced Modes of Plastic Genotypes and Their Dependence on Time Lag*

For plastic genotypes, the mode of the noninduced tolerance curve is

$$m_{m,n} = m_{mb,n} = \frac{t_r}{2(1 - t_s)}\bar{\phi}. \tag{3}$$

(Note that $t_s > 0$ is assumed.) Therefore, the mode of the noninduced tolerance curves increases as either $t_r$ or $t_s$ increases (fig. 3; table 2) and is independent of whether individuals have complete or incomplete information on predation risk when predators are present.

In contrast, the mode of the induced tolerance curve depends on whether prey have incomplete or complete information on $\phi$:

$$m_{m,i,p} = m_{mb,i,p} = \left(1 - \frac{t_r}{2t_s}\right)\bar{\phi},$$

$$m_{m,c,p} = m_{mb,c,p} = \left(1 - \frac{t_r}{2t_s}\right)\phi. \tag{4}$$

Prey that have incomplete information set their induced tolerance curves on the basis of their best estimate of predation risk—the long-term average risk $\bar{\phi}$—while prey with complete information set their induced tolerance curves on the basis of the actual level of predation risk, $\phi$. Both of these are affected by the ratio of the time lag ($t_r/2$) versus the duration of the riskier environment ($t_s$; table 2). Parallel to the results for the noninduced tolerance curve, a larger time lag relative to the duration of the risky period pulls the mode for the induced tolerance curve closer to the predator-free state.

As time lag $t_r$ increases, noninduced ($m_n$) and induced ($m_p$) mode values converge as they move away from the state of the environment for which they are intended and toward the state of the other environment (fig. 3). Which of the modes moves quickest toward an intermediate value depends on the relative durations of safer and riskier environments. When $t_s < 0.5$, safer environments have longer durations than riskier environments, and with increasing $t_r$, the ratio of $t_r/t_s$ increases more rapidly than the ratio of $t_r/(1 - t_s)$. Thus, $m_p$, which depends on $4t_r/t_s$, changes more rapidly than $m_n$, which depends on $t_r/(1 - t_s)$. The

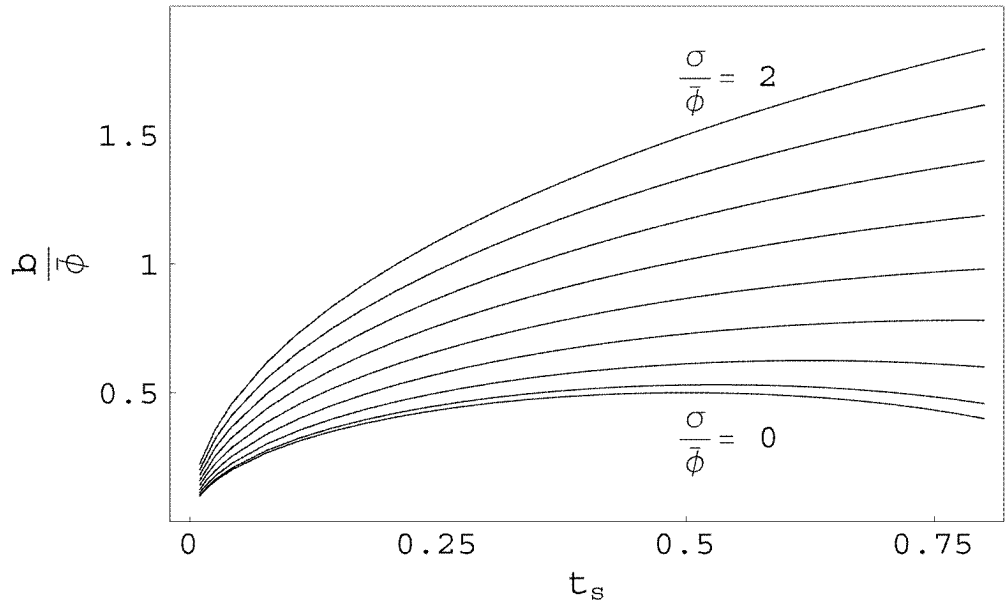


**Figure 2:** Optimal breadth of adaptation for nonplastic genotypes. The breadth $b$ of adaptation increases with $\phi$ according to equation (2) ($\phi > 0$ is assumed; otherwise $|\phi|$ has to be taken). The dependence of $b/\bar{\phi}$ on $t_s$ is shown for various coefficients of variation ($CV_\phi = \sigma_\phi/\bar{\phi} = 0, 0.25, 0.5, \ldots, 2$).

opposite holds true for $t_s > 0.5$. It is worthwhile to note that the optimal mode values do not depend on the amount of environmental variation ($CV_\phi$) and that breadth plasticity in addition to plasticity in the mode does not change the optimal values.

### *The Induced and Noninduced Breadth Values of Plastic Genotypes*

In contrast to the mode values, it can be intuitively expected that optimal breadth depends on the variability of the environment. Further, we expect that optimal breadth varies with the kind of plasticity and with the completeness of information.

For genotypes that are plastic only in the mode (m-plastic), induced and noninduced phenotypes have the same optimal breadth, but they differ for complete (c) and incomplete (i) information:

$$b_{m,i} = \sqrt{(\bar{\phi})^2}\sqrt{t_s CV_\phi^2 + t_r - \frac{t_r^2}{4(1 - t_s)t_s}}, \tag{5}$$

$$b_{m,c} = \sqrt{(\bar{\phi})^2}\sqrt{t_r(1 + CV_\phi^2) - \frac{t_r^2[1 + CV_\phi^2(1 - t_s)]}{4(1 - t_s)t_s}}. \tag{6}$$

These formulas imply that the breadth of m-plastic genotypes converges to the breadth of nonplastic genotypes with increasing environmental variation ($b_{m,i} \to b_f$ for $CV_\phi \to \infty$). Further, for small environmental variation, the completeness of information becomes irrelevant ($b_{m,i} \to b_{m,c}$ for $CV_\phi \to 0$).

For genotypes that are plastic in both their mode and breadth (m-b-plastic), we have to calculate optimal breadth values for the noninduced and the induced phenotype. The noninduced breadth is

$$b_{mb,i,n} = b_{mb,c,n} = \sqrt{(\bar{\phi})^2}\frac{\sqrt{t_r[2(1 - t_s)(CV_\phi^2 + 1) - t_r]}}{2(1 - t_s)}, \tag{7}$$

given $t_r < 2(1 - t_s)$. This breadth is independent of the completeness information but depends on the mean value of the inducing environment ($\bar{\phi}$). The breadth is greater if $\bar{\phi}$ (predation risk) is higher (see also table 2). The breadth of the induced tolerance curve depends on whether individuals have complete or incomplete information about the value of $\phi$:

$$b_{mb,i,p} = \sqrt{(\bar{\phi})^2}\frac{\sqrt{(2t_s - t_r)(2t_s CV_\phi^2 + t_r)}}{2t_s}, \tag{8}$$

$$b_{mb,c,p} = \sqrt{\phi^2}\frac{\sqrt{(2t_s - t_r)t_r}}{2t_s}. \tag{9}$$

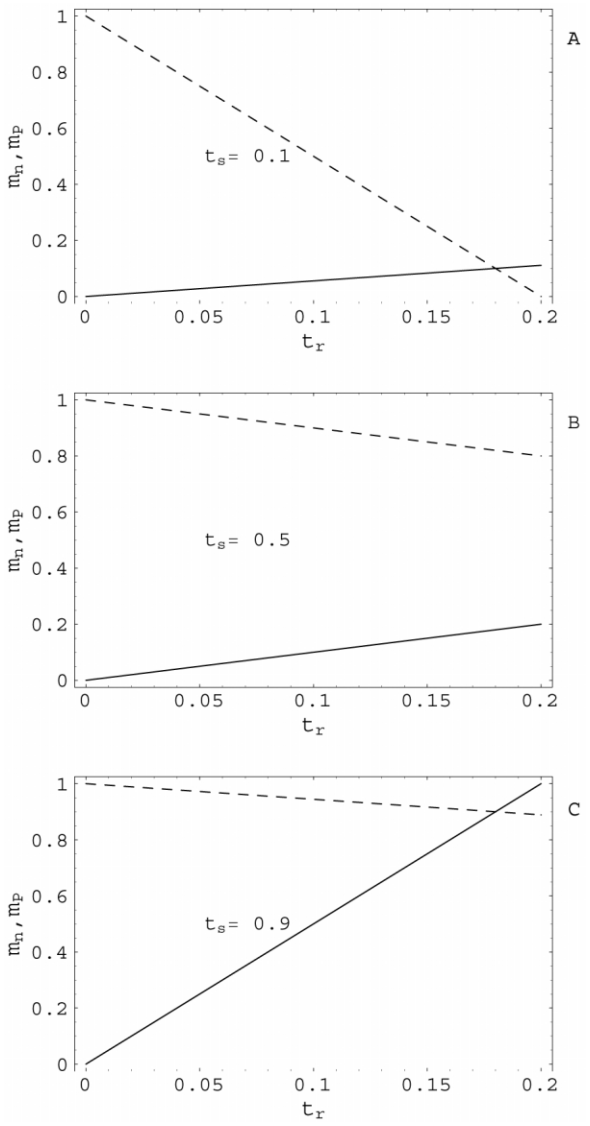


**Figure 3:** Modes of noninduced (*solid line*) and induced (*dashed line*) plastic tolerance curves as lag times ($t_r$) are varied; $\bar{\phi} = 1$ for incomplete information and $\phi = 1$ for complete information. *A*, $t_s = 0.1$. *B*, $t_s = 0.5$. *C*, $t_s = 0.9$. (Note that $m_n$ and $m_p$ are independent of $CV_\phi$.)

The breadth of the induced tolerance curve increases as the (mean) difference between noninducing and inducing environmental states increases. Incomplete information enlarges the breadth of the tolerance curve further depending on $CV_\phi$, a measure of uncertainty about the value of $\phi$.

### *Effects of Time Lags, Environmental Variance, and Completeness of Information on Tolerance Curves*

Overall, then, how prey respond to the presence versus absence of predators depends on the ratio of $t_r/t_s$, on whether prey have complete or incomplete information about $\phi$, and, in the case of incomplete information, on the coefficient of variation in $\phi$. When time lags are small relative to the duration of the risky period (i.e., when the ratio of $t_r/t_s$ is small) and prey have either complete information about the value of $\phi$ or incomplete information with a low coefficient of variation for $\phi$, then prey should set the modes of their two tolerance curves close to the two environmental states (0 and $\phi$) and the breadths of their tolerance curves should be relatively narrow (figs. 4, 5*D*). This is similar to what would be predicted by models without time lags. In this case, prey are relying primarily on a pair of specialized defenses that are each optimal for a narrow set of predator regimes.

If $t_r/t_s$ is small, prey have incomplete information about $\phi$, and $\phi$ has a high coefficient of variation, then again prey set the modes of their two tolerance curves to closely match the two environmental states (0 and $\phi$); however, in that situation, they increase the breadth of their tolerance curves, particularly the plastic tolerance curve (figs. 4, 5*B*). Because these prey have incomplete knowledge of the value of $\phi$ during riskier times, they compensate by increasing the breadth of their tolerance curve to cover a greater range of $\phi$. In this case, individuals are still using a specialized defense in the sense that their modes are far apart, but they generalize their response to changing environments by expanding the breadth of their tolerance curves.

As $t_r/t_s$ increases, the cost of using the wrong tolerance curve during lag periods increases. As a result, regardless of whether prey have complete or incomplete information about predation risk, the modes of the two tolerance curves converge (i.e., $m_p - m_n$ decreases; figs. 4, 5*A*, 5*C*). In this sense, these prey are reducing their specialized defenses since the modes of their tolerance curves are becoming less different between environments. Instead, individuals are compensating for increased lag times by using generalized defenses (i.e., converging modes and increased breadths).

### *Fitness Benefits of Mode and Breadth Plasticity*

With the optimal values for mode and breadth, we can now calculate the fitness of the different genotypes to examine how plasticity in the mode and the breadth affects prey fitness with either complete or incomplete information. Fitness for nonplastic individuals is

$$w_f = \frac{1}{\sqrt{2e\pi(\bar{\phi})^2}\sqrt{(1 + CV_\phi^2 - t_s)t_s}}. \quad (10)$$

For plastic genotypes, we obtain four different fitness func-

tions depending on the kind of plasticity and completeness of information:

$$w_{\mathrm{m,i}} = \sqrt{\frac{2}{e\pi(\bar{\phi})^2}}\sqrt{\frac{(1-t_{\mathrm{s}})t_{\mathrm{s}}}{4(1-t_{\mathrm{s}})(t_{\mathrm{r}}t_{\mathrm{s}}+t_{\mathrm{s}}^2\mathrm{CV}_{\phi}^2)-t_{\mathrm{r}}^2}}, \tag{11}$$

$$w_{\mathrm{m,c}} = \sqrt{\frac{2}{e\pi(\bar{\phi})^2}}\sqrt{\frac{(1-t_{\mathrm{s}})t_{\mathrm{s}}}{t_{\mathrm{r}}[4(1-t_{\mathrm{s}})t_{\mathrm{s}}+\mathrm{CV}_{\phi}^2(1-t_{\mathrm{s}})(4t_{\mathrm{s}}-t_{\mathrm{r}})-t_{\mathrm{r}}]}}, \tag{12}$$

$$w_{\mathrm{mb,i}} = \frac{\sqrt{\frac{2}{e\pi}}\left(\frac{1}{\sqrt{(\bar{\phi})^2}}\right)(1-t_{\mathrm{s}})^{1-t_{\mathrm{s}}}t_{\mathrm{s}}^{t_{\mathrm{s}}}}{\sqrt{t_{\mathrm{r}}^{1-t_{\mathrm{s}}}[2-t_{\mathrm{r}}+2\mathrm{CV}_{\phi}^2(1-t_{\mathrm{s}})-2t_{\mathrm{s}}]^{1-t_{\mathrm{s}}}(2t_{\mathrm{s}}-t_{\mathrm{r}})^{t_{\mathrm{s}}}(t_{\mathrm{r}}+2\mathrm{CV}_{\phi}^2t_{\mathrm{s}})^{t_{\mathrm{s}}}}}, \tag{13}$$

$$w_{\mathrm{mb,c}} = \frac{\sqrt{\frac{2}{e\pi}}\left(\frac{1}{\sqrt{(\bar{\phi})^2}}\right)^{1-t_{\mathrm{s}}}\left(\frac{1}{\phi_{\mathrm{geo}}}\right)^{t_{\mathrm{s}}}(1-t_{\mathrm{s}})^{1-t_{\mathrm{s}}}t_{\mathrm{s}}^{t_{\mathrm{s}}}}{\sqrt{t_{\mathrm{r}}[2-t_{\mathrm{r}}+2\mathrm{CV}_{\phi}^2(1-t_{\mathrm{s}})-2t_{\mathrm{s}}]^{1-t_{\mathrm{s}}}(2t_{\mathrm{s}}-t_{\mathrm{r}})^{t_{\mathrm{s}}}}}. \tag{14}$$

For the definition of $\phi_{\mathrm{geo}}$, see equation (A10) in the online edition of the *American Naturalist*.

All fitness functions show the same dependence on $\bar{\phi}$ (after substituting $\phi_{\mathrm{geo}}$ by a function of $\bar{\phi}$; see appendix). Therefore, ratios of the above fitness functions do not depend on $\bar{\phi}$. This facilitates the analysis of the fitness advantage of reversible plasticity.

The fitness benefits of plasticity in the mode and/or breadth for complete and incomplete information are shown in the ratio ($w_{\mathrm{m...,...}}/w_{\mathrm{f}}$) of fitnesses for plastic genotypes and nonplastic genotypes (fig. 6). Plasticity in the mode increases the fitness of prey in comparison to no plasticity, and plasticity in the mode and breadth increases fitness in comparison to mode plasticity alone. These fitness benefits depend on the coefficient of variation in $\phi$ (table 2). If prey have complete information, the benefit of mode plasticity over no plasticity is unchanged or only increases slightly as the coefficient of variation in $\phi$ increases. However, if prey have incomplete information about the level of $\phi$, then the fitness benefit of mode plasticity relative to no plasticity declines sharply as the coefficient of variation in $\phi$ increases.

The fitness benefits of plasticity also depend on $t_{\mathrm{r}}$ and $t_{\mathrm{s}}$. As the lag time ($t_{\mathrm{r}}$) decreases, prey can have tolerance curves that are more specifically adapted to their present environment rather than the environment during the lag time period. Therefore, as $t_{\mathrm{r}}$ decreases, both mode plasticity and mode and breadth plasticity have larger positive effects on prey fitness.

**Table 2:** Responses of tolerance curves to changing parameter values with mode and breadth plasticity

| Changes in parameters | $m_{\mathrm{n}}$ | $b_{\mathrm{n}}$ | $m_{\mathrm{p}}$ | $b_{\mathrm{p}}$ |
|---|---|---|---|---|
| $\uparrow t_{\mathrm{r}}$ | ↑ | ↑↓ | ↓ | ↑↓ |
| $\uparrow t_{\mathrm{s}}$ | ↑ | ↑ | ↑ | ↑↓ |
| $\uparrow \phi$ | ↑ | ↑ | ↑ | ↑ |
| $\uparrow \mathrm{CV}_{\phi}$ | No effect | ↑ | No effect | ↑ |
| Incomplete information[a] | No effect | No effect | ↑↓ | ↑ |

[a] Incomplete information refers to how the modes and breadths of tolerance curves change when prey go from having complete information about $\phi$ to having incomplete information about $\phi$.

## Discussion

It is obvious that reversible phenotypic plasticity can be advantageous if an organism is exposed to variation in selection regimes during its lifetime. It might be, however, surprising how large the corresponding selective advantage can be. The model predictions are valid in a quite general context, but to avoid a too abstract discussion, the first part of the discussion will focus on antipredator ecology. We will discuss how response lags and the pattern and predictability of variation in predation regimes should influence patterns of prey plasticity with regard to both the mode and breadth of their environmental tolerance functions. In the second part of the discussion, we will concentrate on future directions of research, for example, how inherent limitations of the current model might be overcome in order to improve the applicability to more specific systems or to even more general questions.

### *Response Lags, Incomplete Information, and Antipredator Ecology*

If prey can respond instantaneously to changing environments and have complete information about the current state of their environment, then they should ignore other potential environmental states until those states occur; that is, they should ignore predators until predators arrive, and they should not account for a world without predators until predators actually leave. We find that when prey have no response lag (i.e., $t_{\mathrm{r}} = 0$) and prey either have complete information or have incomplete information but $\mathrm{CV}_{\phi} = 0$, they should show high plasticity with their fitness modes, $m$, matched perfectly to the environmental optima, 0 when predators are absent and $\phi$ when predators are present, and minimal breadths in their tolerance functions. That is, they switch between one specialist strategy that ideally matches the no predator situation (e.g., full feeding activity in an open habitat) to another specialist strategy that ideally matches an observed predation regime (e.g., the optimal degree of reduced activity and increased refuge use).

Individuals often have response lags and incomplete information about the current state of their environment (Stephens 1987; Sih 1992). We have shown that these limitations should cause prey to respond to anticipated situations that they are not currently experiencing. Response

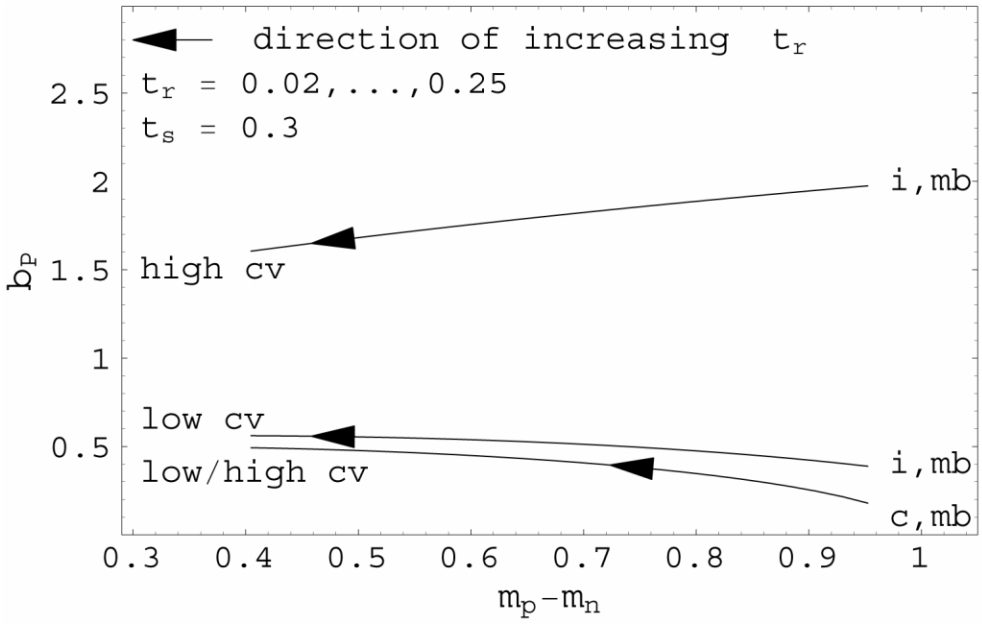


**Figure 4:** Influence of $t_r$ (lag time), $CV_\phi$ (coefficient of variation in $\phi$), and information status (complete, incomplete) on the difference between induced and noninduced plastic modes ($m_p - m_n$) and on the size of plastic breadth. The three lines represent the modes and breadths of tolerance curves as $t_r$ is varied from 0.02 to 0.25 from right to left along the lines, $t_s = 0.3$. *Top line*, incomplete information with high $CV_\phi$ (=2.0). *Middle line*, incomplete information with low $CV_\phi$ (=0.35). *Bottom line*, complete information with high or low $CV_\phi$ (=2.0 or 0.35).

lags, in particular, can be very costly. If prey continue to be highly active outside of a refuge even when predators are present, prey can obviously suffer high mortality (Sih et al. 1988; Sih 1992). Conversely, if prey show long lags before resuming activity after predators have left an area, they can suffer significant costs in terms of lost feeding or mating opportunities (Sih 1992). Our model shows that to reduce these costs, in each situation (i.e., predators absent or present) prey should shift their modal strategy, *m*, toward the other situation and broaden their breadth of tolerance, *b*. Both of these shifts reduce fitness in the current situation but increase the prey's ability to cope with the new environment during the lag phase.

The degree of shift in *m* toward intermediate values reflects the trade-off between the benefit of being better at coping with new conditions during the lag phase (before prey show their full plastic response) versus the cost of reduced adaptation to current conditions. Increases in the lag time increase the benefit of preadapting to the other environment (fig. 5). As a result, when $t_r$ is large, prey should show relatively little shift in their fitness modes as predators come and go (i.e., as $t_r$ increases, $m_n$ and $m_p$ converge). If predators are important ($t_s$ approaches 1) and lag times are relatively long, prey should adopt a specialized strategy that accounts for predators even when predators are not present; for example, prey should stay in safe flocks or maintain an intermediate degree of a morphological defense even when predators are absent. Alternatively, if predators are only occasionally present ($t_s$ approaches 0) and lag times are relatively long, then prey should exhibit only a weak specialized response to predators. In either situation, they can partially compensate for having the wrong specialized traits in one environment by increasing their reliance on a generalized strategy that increases their breadth of tolerance.

Given that response lags should be of critical importance, we clearly need more data on response lags. Developmental constraints presumably set a minimum value for response times for a given type of character; for example, morphological characters might usually have longer response lags than behavior simply because morphologies take more time to build. However, theory and a few empirical examples suggest that relative response lags can also be shaped by information constraints and selection. Detecting an increase in predation risk in many situations may be quicker than detecting a decrease in predation risk. A single observation that a predator is present should immediately outweigh any prior estimates of low predation risk, while each observation that predators are not present only decreases the estimated probability that predators are present (Mangel and Clark 1983). As a result, response lags should exhibit an asymmetry: prey should respond

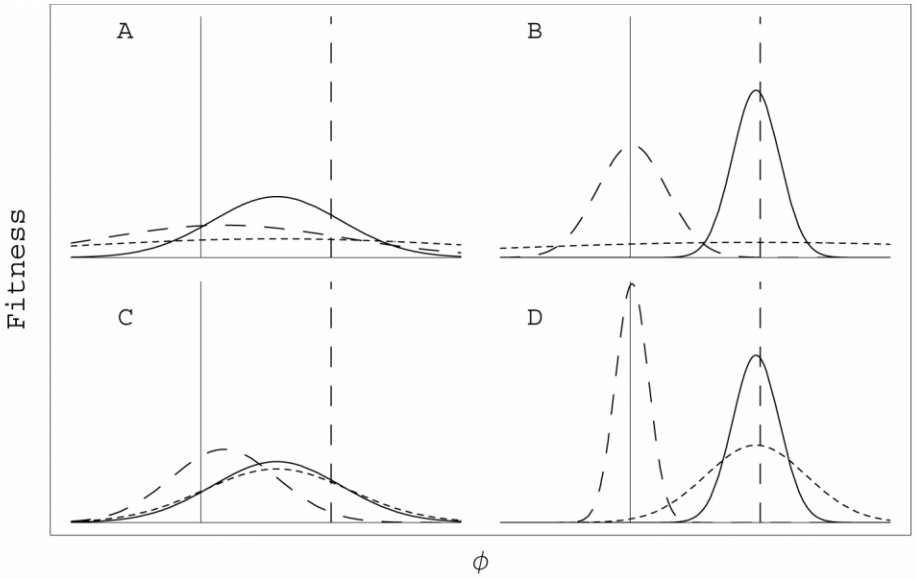


**Figure 5:** Tolerance curves corresponding to the end points of the three lines in figure 4 ($t_s = 0.3$). *A*, $t_r = 0.25$, $CV_\phi = 2.0$. *B*, $t_r = 0.02$, $CV_\phi = 2.0$. *C*, $t_r = 0.25$, $CV_\phi = 0.35$. *D*, $t_r = 0.02$, $CV_\phi = 0.35$. *Dashed line*, noninduced tolerance curve. *Dotted line*, plastic tolerance curve with incomplete information. *Solid line*, plastic tolerance curve with complete information. *Solid vertical line*, $\phi = 0$. *Dashed vertical line*, $\phi = \bar{\phi}$.

rapidly to predator arrival but exhibit long recovery lags after predators leave. Furthermore, an asymmetric response lag would be selected for in situations where once predators arrived, they might stay in the vicinity, and consequently a return is very likely. Relative response lags should also depend on the type of cue used to detect predators. Chemical cues linger longer after predators leave than visual cues. Thus, prey that rely on chemical cues might exhibit longer recovery lags after predators leave.

The proportion of time that prey spend in the presence versus absence of predators should have important effects on optimal prey strategies; that is, the temporal pattern of predation risk matters. In general, we predict that when $t_r$ is large, prey should show relatively few specialized responses to rare situations. If predators are frequently present, then prey should retain specialized traits that keep them safe from predation even when predators are absent. If predators are rarely present, prey should show little specialized response to predators but should rely instead on a generalized response (i.e., a response that is only adequately effective at reducing risk but that allows them to maintain at least adequate feeding rates during the lag recovery period after predators leave).

One other recent model examined effects of temporal variation in predation risk on optimal antipredator behavior (Lima and Bednekoff 1999). They predicted that prey should show their strongest antipredator responses when predators are rare (i.e., during rare pulses of risk), whereas if predators are frequently present, prey might have to show relatively weak response to predators. The differences between our predictions reflect different assumed scenarios. Their results are based on the assumption that prey require a threshold energy need that must be met to survive (which we did not explicitly incorporate in our model), while we assumed response lags (which they ignored). Thus, in their model, prey can afford to become totally inactive during rare predator encounters but have to stay moderately active if predators are permanently present.

Effects of the temporal pattern of risk on antipredator responses have been largely ignored in antipredator ecology; that is, models and experiments on antipredator adaptations have typically examined prey responses to predator presence versus absence without reference to the proportion of time that predators are present. If $t_s$ is important, then investigators should attempt to measure $t_s$ in nature and either account for it in their experimental designs or manipulate it as an experimental treatment (Lima and Bednekoff 1999; Sih and McCarthy 2002).

Finally, incomplete information also had major effects on predicted antipredator responses (fig. 4). In particular, if predation risk when predators are present is highly variable (large $CV_\phi$) and prey have information only on the mean risk, then prey should show a very broad tolerance curve in the presence of predators (fig. 4). That is, prey

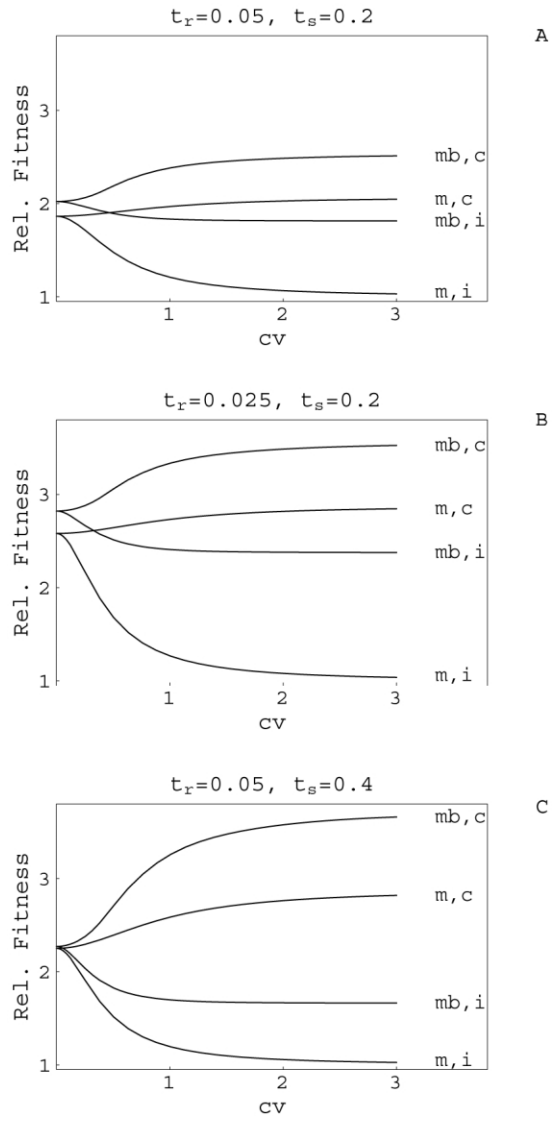


**Figure 6:** Fitness advantage of plastic genotypes. Relative fitnesses are given as ratios of the fitnesses of a plastic genotype to a nonplastic genotype. Relative fitness of mode plasticity and mode and breadth plasticity with complete or incomplete information are plotted versus $CV_\phi$, the coefficient of variation in $\phi$. *A*, $t_r = 0.05$, $t_s = 0.2$. *B*, $t_r = 0.025$, $t_s = 0.2$. *C*, $t_r = 0.05$, $t_s = 0.4$. Definitions: *mb*, *c*, mode and breadth plasticity with complete information; *mb*, *i*, mode and breadth plasticity with incomplete information; *m*, *c*, mode plasticity with complete information; *m*, *i*, mode plasticity with incomplete information.

should treat predation as a yes/no phenomenon and respond with a generalized response that is adequately effective regardless of exact predation pressure.

### *Future Directions*

For this model, we have made a number of simplifying assumptions that deserve further examination. We treated response lags, the duration and pattern of predation risk, and the quality of information prey have about predation risk as parameters that influence prey patterns of plasticity. Each of these, however, could be variables that prey influence in adaptive ways.

The duration of response lags ($t_r$) can vary adaptively in response to variations in the quality of information and the costs of errors in plasticity (Sih 1992; Tollrian and Harvell 1999*b*). Further, these response lags could be adaptively asymmetric, with prey more quickly switching to their induced form than to their noninduced form because of the asymmetry in the cost of incorrectly assessing the current state of their environment or because of differences in how hard it is to detect the absence versus the presence of predators. Likewise, patterns of the duration of predation risk ($t_s$) can be determined not only by predator movements but also by prey habitat selection (Sih et al. 1988; Lima 1998), and information about predation regimes depends on prey sampling and learning. Future two-tiered models could blend how selection shapes these variables (prey response lags, habitat selection, assessment of predation risk) that, in turn, affect adaptive reversible plasticity.

We did not explicitly implement possible costs of plasticity in this model. (In many cases, the costs of plasticity should be negligible compared with the indirect fitness cost of being in a suboptimal state.) If the costs of plasticity are an additional constant factor, independent of the amount of expressed plasticity (as would be the case for the general costs of sustaining the biochemical machinery for reversible plasticity), then the model results for the optimal mode and breadth of tolerance curves would remain unchanged. However, if a substantial part of the cost of plasticity depends on the amount of plastic change (i.e., if costs are a function of $\phi$), then the mode and breadth of tolerance curves might be changed.

Furthermore, we did not include complications arising from possible costs and constraints of defense formations (DeWitt et al. 1998; Tollrian and Harvell 1999*b*). One benefit of plasticity (compared with having constitutively fixed traits) is that the costs of maintaining a trait can be avoided during periods when the trait is not needed. On the other hand, constraints might prevent the inducible formation of traits of the same magnitude as constitutively fixed traits. Thus, constitutively fixed traits might provide a higher maximal benefit, that is, defensive value in our scenario.

In this model, we have assumed that individuals experience both the noninducing and inducing environments; thus, we examined the fitnesses of nonplastic and plastic genotypes summed over these environments. However, in some cases, individuals or lineages can avoid environments through diapause or migration. If individuals

can effectively avoid environments, then the tolerance curves of these individuals should become specialized to match only the environments that they are going to experience. Further modeling is needed to see how this avenue to specialization would affect the evolution of reversible plasticity.

We focused our description on prey responses to predators but still in general fitness terms. To generate more directly testable predictions on specific antipredator traits (e.g., refuge use, vigilance, life-history shifts, induced morphologies), we need explicit functions relating prey traits to fitness (including both risk and other fitness needs) as a function of environmental risk gradients. Because prey often show multiple responses to predators (e.g., all of the responses listed above; Endler 1995; DeWitt et al. 1998; Tollrian and Dodson 1999) that differ in benefits, costs, and response lags, future models should address how multiple responses should be integrated into an overall adaptive response to a variable risk regime. All these complications might lead to the coexistence of genotypes with different strategies, as it is frequently found in natural communities (De Meester et al. 1995)

Our model addressed patterns of plasticity in terms of general fitness functions on general environmental gradients. Thus, our results should apply to any trait that exhibits reversible plasticity in response to environmental change. The model equations can be solved without the simplifying assumption of symmetric response delays (equations and solutions are given in Gabriel 2005), and such an extended version of the model should be used if there is large asymmetry in response delays. Besides predator-prey interaction, the model could be fruitfully applied to plasticity in plants (for a recent review, see Dudley 2004), but for some questions it might be necessary to extend the model by including spatial variation.

Our model predicts that any organism that is exposed to a regularly occurring stress that typically is shorter than their average lifespan will be strongly selected to have reversible plastic traits. Also, we have shown that the fitness effects of plastic traits can be described by tolerance functions with reversible plastic modes and breadths. Thus, this model can be used for studying the causes of individual niche variation (recently reviewed by Bolnick et al. 2003), with the breadth of an individual's niche being reversible. Using the concept of environmental tolerance functions, Lynch and Gabriel (1987) introduced the first model that could predict the evolution of niche width as it depends on environmental variability. Gabriel and Lynch (1992) studied the effects of irreversible plasticity on niche width but found that changes in the modes of environmental tolerance functions greatly outweigh changes in the breadths of the functions. However, we have shown that there are large fitness advantages for plasticity in the breadth of environmental tolerance functions when that plasticity is reversible. Thus, we expect that reversible plasticity will have significant impacts on the evolution of niches, but further extensions of the current model, including an explicit implementation of spatial heterogeneity, will be needed to test this idea.

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