LETTER

Comparing the qualitatively different effects rapidly evolving and rapidly induced defences have on predator–prey interactions

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Abstract
Interspecific interactions depend not only on the population densities of the interacting species, but on their phenotypes as well. Phenotypic variation can be plastic or heritable and both mechanisms can drive phenotypic change at rates comparable to or faster than those of ecological dynamics (e.g. changes in population abundances or spatial distributions). In this study, we compare the effects rapidly induced and rapidly evolving defences have on community dynamics by considering the fast phenotypic change limit using fast-slow systems theory. Our approach allows us to study phenotypically plastic and evolving systems with one overarching theory, thus capturing the effects rapidly induced defences have on ecological dynamics and how those effects differ from the effects of evolving defences. Our results show that rapidly induced defences tend to stabilize community dynamics and that some behaviours observed in rapidly evolving systems cannot be produced by phenotypic plasticity.

Keywords
Inducible defence, phenotypic plasticity, predator–prey, rapid evolution.

INTRODUCTION

Interactions between trophic levels are known to be influenced not only by population densities but also by the behavioural, life history and morphological traits of the interacting individuals. This interplay between populations, mediated by ecologically important traits (trait-mediated interactions: Bolker et al. 2003), is expected to have important consequences at the individual, population and community levels (Lima 1998; Agrawal 2001; Bolker et al. 2003; Werner & Peacor 2003; Miner et al. 2005; Preisser et al. 2005; Berg & Ellers 2010).

It is also increasingly recognized that changes in ecologically important species’ traits can occur fast enough to affect interspecific interactions while they are taking place (Tuda 1998; Werner & Peacor 2003; Agrawal et al. 2007). Rapid phenotypic changes in populations can arise through trait evolution at ecological rates (eco-evolutionary dynamics: Fussmann et al. 2007; Ginzton & Hairston 2007) or plasticity within individuals (Tollrian & Harvell 1999). For example, trait evolution has been shown to occur in fewer than six generations in birds (Grant & Grant 2002), crustaceans (Hairson & Dillon 1990), fish (Conover & Munch 2002) and reptiles (Sinervo et al. 2000). Similarly, induced predatory responses have been observed after timespans ranging from a few hours (Green & Ryan 1972; Haukoja 1980; Kuhlmann & Heckmann 1985; Kusch 1993) to a few weeks (Agrawal et al. 1999; Tollrian & Harvell 1999; Relyea & Auld 2004).

The shared rapidity in response exhibited in these systems suggests that rapid evolution and rapidly induced defences could have similar effects on community level dynamics. Despite this similarity, most theoretical work investigating the effects of rapidly evolving and plastic traits has either progressed independently or removed the distinction between the two adaptive processes by assuming that one model applies to either system (e.g. Abrams et al. 1993). Eco-evolutionary studies have shown that rapidly evolving species’ traits can drive complex population dynamics, some of which are unobservable in evolutionarily fixed systems (Yoshida et al. 2007; Jones et al. 2009) and cannot be captured in phenotypically plastic models (Shertzer et al. 2002). Furthermore, rapid evolution has the potential to stabilize or destabilize population dynamics (Abrams 1992; Abrams & Matsuda 1997; Cortez & Ellner 2010). In contrast, many theoretical studies of inducible defence systems suggest that plasticity stabilizes population dynamics (Vos et al. 2004b; Ramos-Jiliberto et al. 2007, 2008; Serizawa et al. 2008). This conclusion is not supported universally (Ramos-Jiliberto 2003; Ramos-Jiliberto & Garay-Narváez 2007), but even systems destabilized by inducible defences do not exhibit the full spectrum of dynamics observed in rapidly evolving systems. For example, antiphase (half-period lag) oscillations are observed in eco-evolutionary models (Fig. 1c), but plastic models only exhibit oscillations with quarter-period or less lags (Fig. 1a).

In total, current theory suggests that the two means of adaptive change, although both occurring at ecological rates, do not have the same effects on community dynamics. But due to the specificity of the models and the independent routes through which they have been investigated, it is difficult to compare how evolving and inducible defences affect community level dynamics and to determine why differences are observed. A general theory that both encompasses and differentiates rapidly evolving and phenotypically plastic communities would aid in determining how differences and similarities between these systems arise.

To begin to develop such a theory, we focus on predator–prey systems and consider the limit where phenotypic change occurs faster than changes in population density. This approach follows from fast-slow systems theory (an area known as singular perturbation theory, Arnold et al. 1995) and was used previously to study how rapidly evolving traits affect population dynamics in predator–prey systems.
In the following sections, we explore how phenotypic plasticity and evolution can be studied under one unifying theory. Our analysis shows that rapidly induced defences stabilize community dynamics and synchronize population oscillations in predator–prey systems. Furthermore, inducible defences cannot generate many types of cyclic dynamics observed in evolving systems. Thus, some kinds of population oscillations are indicative of rapidly evolving defences. Finally, we address how the fast induction limit yields insight into systems where the rates of adaptive change are comparable to those of ecological processes.

**MATERIALS AND METHODS**

**Predator–prey model with phenotypic plasticity**

We begin with a general predator–prey model where prey can be in one of two classes, \( x_1 \) or \( x_2 \), with phenotypes \( a_1 < a_2 \), respectively. We assume that smaller values of the trait yield decreased susceptibility to predation at a fitness cost, thus creating a trade-off. We also assume that individuals switch classes depending on the current predator density, \( y \), and more prey becoming defended as predator density increases. For example, the change could be driven by an environmental cue. Individuals switch to class \( i \) at rate \( \epsilon P_i(y) \), where \( P_i(y) = 1 + \epsilon \) is a positive constant. Thus, if the predator density was fixed at a value \( y \), a fraction \( P_i(y) \) of the total prey population would be defended. If \( P_i(y) \) and \( P_2(y) \) are both non-zero for a particular value of \( y \), then some individuals are maladaptively switching to a less fit phenotype. When maladaptive switching is rare, \( P_1(y) \) and \( P_2(y) \) will be near 0 or 1 for all values of \( y \), and individuals will switch phenotypes at a threshold predator density. In simulations, we consider more gradual switches as some experimental data suggest this is the case (Gilbert & Waage 1967; Kusch 1993; Buskirk & Arioli 2002), but our analytical results do not differ.

Under the above assumptions, our model is

\[
\begin{align*}
\frac{dx_1}{dt} &= F_1(x_1, x_2) - G_1(x_1, x_2, y) + \epsilon^{-1}x_2P_1(y) - \epsilon^{-1}x_1P_2(y) \\
\frac{dx_2}{dt} &= F_2(x_1, x_2) - G_2(x_1, x_2, y) - \epsilon^{-1}x_2P_1(y) + \epsilon^{-1}x_1P_2(y) \\
\frac{dy}{dt} &= H_i(x_1, x_2, y) + H_2(x_1, x_2, y) - D(y) \\
\end{align*}
\]

\( F_i \) is the growth rate of class \( i \) in the absence of predation, \( G_i \) is the predation rate on class \( i \), \( H_i \) is the product of the prey to predator conversion and predation rate on class \( i \), and \( D \) is the death rate of the predator population. We assume that \( D \) is increasing in \( y \) and assume \( G_i \) and \( H_i \) are increasing in \( x_i \) and \( y \). Typically, the functions \( F_i, G_i, H_i \) and \( D \) are written in terms of per capita growth rates, e.g. \( F_i = xifi(x_i, y, y) \). We use the more general functions to simplify notation. The positive constant \( \epsilon^{-1} \) represents the phenotypic switching rate of the prey. As we assume that induced changes in phenotype occur rapidly, \( \epsilon \) will be very small, making \( \epsilon^{-1} \) very large.

Particular examples of system (1) include Shertzer et al. (2002) and Vos et al. (2004a,b).

As seen in Supporting Information Appendix S1, system (1) is approximated by the following predator–prey model with an average phenotype \( \bar{x} \).

\[
\begin{align*}
\frac{dx}{dt} &= F(x, \bar{x}) - G(x, y, \bar{x}) \\
\frac{dy}{dt} &= H(x, y, \bar{x}) - D(y) \\
\frac{d\bar{x}}{dt} &= \epsilon A(\bar{x}) V \left[ \frac{\partial}{\partial \bar{x}} \left[ \frac{1}{x} \frac{dx}{dt} \right] \right] + (\bar{x} - \bar{x})P_1(y) - (\bar{x} - \bar{x})P_2(y) \\
\end{align*}
\]

Here, our new state variables are \( x = x_1 + x_2 \), the total prey population density, and \( \bar{x} = (x_1x_1 + x_2x_2)/x \), the average trait of the prey population. \( F(x, \bar{x}) = F_1(x_1, x_2) + F_2(x_1, x_2) \) represents the growth rate of the total prey population, where we use the reverse transformations \( x_1 = \frac{a_1 - a_2}{a_2 - a_1} \bar{x} \) and \( x_2 = \frac{a_2 - a_1}{a_2 - a_1} \bar{x} \) to evaluate \( F_\bar{x} \). \( G = G_1 + G_2 \) and \( H = H_1 + H_2 \) are similarly defined. \( A(\bar{x}) V \) represents the population variance of the trait (see Appendix S1).
The population dynamics of systems (1) and (2) are exactly the same. Thus, differences between the two systems arise only in the trait equation. As the error introduced by our approximation is multiplied by the small positive constant $\epsilon$ (see Appendix S1a), in the limit where phenotypic change is fast, the error is negligible and the two systems have the same dynamics.

The last two terms of the $dx/dt$ equation in system (2) represent changes in the mean trait value due to phenotypic plasticity (i.e. direct transfer between classes). The first term is the individual fitness gradient, which represents changes in the mean trait value due to births and deaths in each prey class. This term is multiplied by the small constant $\epsilon$ and consequently, as expected from our initial assumptions, changes in the mean phenotype are dominated by phenotypic plasticity, not the birth and death of individuals. The $\epsilon$ multiplying the $dx/dt$ in the trait equation creates a separation of time scales between the trait and population dynamics. Mathematically, this ensures that the mean trait value changes faster than the population densities.

### Predator–prey model with fast evolution

We compare system (2) with a predator–prey system where the mean prey trait, $x$, evolves at a rate much faster than the rate of the ecological dynamics. This model has been studied in Cortez & Ellner (2010) and we only briefly introduce it here. The model is

$$
\frac{dx}{dt} = F(x, y, x) - G(x, y, x)
$$

$$
\frac{dy}{dt} = H(x, y, x) - D(y),
$$

$$
\frac{d\mathbf{a}}{dt} = A(\mathbf{a})V \frac{\partial}{\partial \mathbf{a}} \left[ \frac{1}{2} \frac{dx}{dt} \right]
$$

where all terms are interpreted as in system (2).

The trait dynamics of system (3) follow from the quantitative genetics approach derived in Lande (1982) and Abrams et al. (1993). The mean trait changes in the direction of increasing fitness, determined by the fitness gradient $\frac{\partial}{\partial \mathbf{a}} \left( \frac{1}{2} \frac{dx}{dt} \right)$. Thus, trait evolution is driven by differences in birth and death rates of different phenotypes, not environmental cues.

### Model generality

The above models have been derived under particular restrictive assumptions. For example, system (1) assumes individuals can switch between discrete trait values after birth and system (3) assumes a continuous trait. Here, we address how our models apply more generally to systems with plastic or heritable adaptation.

The phenotype of a plastic individual can either be determined at birth (e.g. Daphnia helmet size, Agrawal et al. 1999) or switched after birth (e.g. tadpole gut and tail length, Relyea & Auld 2004). Model (1) corresponds to a switch-after-birth scenario. We address a switch-before-birth model in Appendix S5. System (1) also implicitly assumes that offspring are born expressing their parent’s phenotype. The consequences of relaxing this assumption are addressed in Appendix S1(b). In the fast induction limit, these differences in mechanism have a small effect and the dynamics exhibited in all cases are qualitatively the same. In addition, in the fast phenotypic change limit, all of the models are approximated by system (2) with negligible error (see Appendices S1b and S5). Thus, system (2) captures the dynamics of any plastic system with discrete phenotypic classes. We focus on the switch-after-birth scenario to simplify the comparison of inducible and evolved defence systems.

System (2) is also a general model for continuous plastic traits. In this setting, an individual can have any trait value between $x_1$ and $x_2$, and the trait equation describes the rate at which individuals switch directly to the optimal trait value, $x_2P_2(y) - x_1P_1(y)$. The optimal trait value increases with predator density and the functions $P_i(y)$ define how fast individuals switch phenotypes. The dual nature of system (2) allows one to study continuous and discrete plastic traits with a single model and interpret properties and results accordingly. For example, the derivative of $P_i(y)$, $dP_i(y)/dy$, measures how maladaptively switching is. Smaller values of $dP_i(y)/dy$ yield large ranges of predator abundance over which maladaptively switching occurs for discrete traits and slower switching to the optimal trait value for continuous traits.

The continuous trait evolutionary model (3) also approximates a system where multiple prey clonal types are present (i.e. discrete trait values, Abrams & Matsuda 1997). In this scenario, evolution occurs as allele frequencies in the prey population change. Consider Fig. 1(b,d). The prey population oscillates between being almost completely defended and almost completely undefended. A clonal system where selection favours extreme phenotypes would behave similarly.

In total, systems (2) and (3) capture the dynamics exhibited by predator–prey systems with continuous and discrete plastic and evolutionary defences. The key difference between the two systems lies in the trait dynamics, $dx/dt$. In the fast evolution system (3), the mean trait changes due to fitness differences between phenotypes via the fitness gradient. Thus, trait evolution is tied directly to differences in the birth and death rates of different phenotypes. In contrast, individual births and deaths have a negligible effect on the trait dynamics of system (2) as individuals rapidly switch to the optimal phenotype. Consequently, changes in plastic traits are driven by the response functions $P(y)$, not the fitness gradient.

### RESULTS

#### Fast-slow dynamics in systems (2) and (3)

When $\epsilon$ is small and positive, solutions to systems (2) and (3) spend nearly all of their time near an object called the critical manifold and the rest jumping between different pieces of the critical manifold. The critical manifold, $C$, of system (2) is a two-dimensional surface whose points satisfy $dx/dt = 0$ when $\epsilon = 0$, $C = \{(x, y, x): (x_2 - x) P_2(y) - (x - x_1) P_1(y) = 0\}$. Equivalently, $C$ is given by the equation $x = x_2P_2(y) + x_1P_1(y)$. An example of a typical critical manifold for an inducible defence system is given in Fig. 2(a).

Solutions stay near a piece of the critical manifold or jump from it depending on the stability of the fast dynamics near that piece. For system (2), this stability is determined by an eigenvalue of the fast dynamics, $Q_f = \frac{\partial}{\partial \mathbf{a}} \left( \frac{1}{2} \frac{dx}{dt} \right)$. The constant negative sign of $Q_f$ has two important consequences for the behaviour of system (2). First, solutions to system (2) will always approach the critical manifold and never jump away from it. Fig. 2(a) demonstrates this behaviour. Initially, the solution quickly runs towards the critical manifold with the values of $x$ and $y$ remaining nearly constant. Once near the critical manifold, the solution behaves as if it were on the critical manifold and remains near it for all time.
the defining equation for the critical manifold, $\alpha = a_1 P_1(y) + a_2 P_2(y)$, into the first two lines of system (2). The stability of this new system is then compared with the stability of a system with a fixed defence (see Appendix S3 for details).

We consider two types of stability. The first deals with whether the coexisting predator and prey tend to equilibrium or oscillate when fixed defences are replaced with inducible defences. As shown in Appendix S3, the effect phenotypic plasticity has on this kind of stability is determined by the quantity $(x_2 - x_1) dP_1 / dy$, i.e. the product of the trait range and the extent to which maladaptive switching occurs. This quantity is zero when $P_1(y) = 0$ or $P_2(y) = 0$, and positive otherwise. $(x_2 - x_1) dP_1 / dy$ is zero when the equilibrium density of the predators is so large that all prey are defended or so small such that all prey are undefended. In these two cases, plasticity does not affect the stability of the ecological dynamics, even when cycles around the equilibrium are large enough to induce a response in the prey.

The quantity $(x_2 - x_1) dP_1 / dy$ is greater than zero when at equilibrium there is a mix of discrete strategies (maladaptive switching) or an intermediate value of the trait is optimal (continuous trait). In these cases, phenotypic plasticity always dampens and decreases the amplitude of predator–prey oscillations, potentially eliminating them (Fig. 3). Note that because $dP_1 / dy$ decreases with increased maladaptive switching, the dampening effect is smaller as switching becomes more maladaptive. In all cases, our results imply that rapidly induced defences cannot cause population oscillations. Thus, cyclic dynamics are due solely to unstable ecological dynamics.

The second type of stability addresses whether replacing fixed defences with inducible defences could result in predator extinction. The effect plasticity has on species coexistence depends on the quantity $(x_2 - x_1) dP_1 / dy$ (see Appendix S3 for details). As above, when $P_1(y) = 0$ or $P_2(y) = 0$, all individuals are defended or undefended at equilibrium and plasticity neither promotes nor inhibits extinction.

When $(x_2 - x_1) dP_1 / dy$ is greater than zero, plasticity may promote or inhibit coexistence. If the populations oscillate when the defence is fixed, then $\frac{dy}{dt}$ is typically negative and inducible defences promote predator extinction. If the ecological dynamics tend to equilibrium when the defence is fixed, then $\frac{dy}{dt}$ is typically negative and inducible defences tend to inhibit predator extinction. As switching becomes more maladaptive, these effects decrease.

In total, plasticity has no effect when all individuals are defended or undefended. If maladaptive switching occurs (discrete trait) or

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**Figure 2** Comparison of (a, b) predator–prey oscillations in an inducible defence system and (c, d) cryptic oscillations in an evolved defence system. (a, c) Trajectory (black) and critical manifold (grey sheets) in phase space. Double arrow indicates fast motion towards or away from the critical manifold and single arrows indicate slow motion near the critical manifold. (b, d) Predator (solid black), prey (dashed black), and trait mean (grey dash-dot) time series for the trajectory. In (c, d), populations are scaled to 0–1 to emphasize that predator oscillations are driven by fluctuations in the prey rather than oscillations in prey abundance. In particular, the variation in the prey abundance is so small, compared to typical measurement errors and small variability in population processes, that in most cases it would be undetectable.

**Figure 3** An example of inducible defences stabilizing population oscillations. (a) Predator (solid black) and prey (dashed black) populations cycle when the defence (dashed-dot grey) is fixed. (b) Population and trait dynamics tend to equilibrium with an inducible defence.
intermediate defence levels are optimal (continuous trait), then plastic traits cannot destabilize stable ecological systems, they dampen or eliminate oscillations, and they increase the chance for predator extinction in unstable systems. The magnitude of these effects diminishes as switching becomes more maladaptive.

**Population phase lags**

In purely ecological predator–prey systems, predator oscillations lag behind prey oscillations by less than a quarter of the period (Bulmer 1975). Previous work has shown that fast predator evolution does not change the lag (Cortez & Ellner 2010). In contrast, fast prey evolution increases or decreases the lag depending on whether the trait yields accelerating or diminishing gains as predator abundance increases; see Cortez & Ellner (2010) for details. Here, we discuss how plasticity affects the phase relations of predator–prey oscillations.

As shown in Appendix S4, the effect inducible defences have on predator–prey phase lags is determined by the sign of product \( a_2 - a_1 \) \( H_y dP_1/dy \). The term \( H_y > 0 \) represents how much the trait affects predator growth. As the product is always negative or zero, inducible defences only decrease the lag between predator and prey oscillations. Thus, plasticity synchronizes oscillations, preventing oscillations where the lag is greater than a quarter of a period. Fig. 4 compares the effects of plasticity and evolution on phase relations. While rapidly induced defences only decrease the lag (Fig. 4b), rapid evolution can also increase the lag and yield oscillations where the lag is greater than a quarter of the period (Fig. 4d).

These results suggest that oscillations where the lag is greater than a quarter of the period or cryptic oscillations are not possible in phenotypically plastic systems. Such oscillations are observed in system (3) either through changes in phase relations described above or through mechanisms that require \( Q_h \) from section 'Fast-slow dynamics in systems (2) and (3)' to be positive. This qualitative difference in population dynamics implies that experimental data exhibiting such oscillations are likely due to rapid evolution, not plasticity.

**DISCUSSION**

In this study, we considered the limit where prey can change their phenotype instantaneously to gain insight into the effects rapidly induced defences have on community dynamics and to determine how those effects differ from the effects of evolutionary processes. Our results show that rapidly induced defences promote stability in predator–prey systems and that rapidly induced defences cannot produce some dynamics observed in rapidly evolving systems.

The stabilizing effect of inducible defences has been observed in previous studies of predator–prey and other higher order trophic systems (Underwood 1999; Vos et al. 2004a,b; Ramos-Jiliberto et al. 2007), but this trend does not hold universally (Ramos-Jiliberto 2003; Ramos-Jiliberto & Garay-Narváez 2007). When induction of the defence is fast, our analysis shows that inducible defences always promote stability in predator–prey systems. This conclusion is independent of when the phenotypic change occurs during the life history of an organism, i.e. switch-before-birth (Appendix S5) or switch-after-birth [system (2) and Appendix S1b]. The forms of the trait equations in system (2), system (A10) of Appendix S1(b), and system (E3) of Appendix S5 suggest why this is and why stabilization is not observed in all models when the plastic response is not nearly instantaneous.

In the fast induction limit, the trait dynamics of all three models have the same governing equation. In particular, the trait equation becomes \( 0 = (a_2 - a_1)P_2(y) - (a - a_1)P_1(y) \) when \( \epsilon = 0 \). This arises because the assumption that an individual can switch phenotypes almost instantaneously after birth [system (2) and system (A10) of Appendix S1] and the assumption that the turnover of individuals in the prey population is fast relative to changes in population size (Appendix S5) are equivalent mathematically. Thus, in the limit where the mean value of the trait changes faster than population abundances, we expect the effects of phenotypic plasticity to be independent of when the defence is induced during the life history of an organism.

But when the rate of phenotypic change is comparable to rates of ecological change, these differences in timing do matter. In system (2), births and deaths of individuals within each prey class have a minimal effect on the rate at which the mean trait value changes when phenotypic change is fast (\( \epsilon \) is small). However, as the separation of time scales between the ecological and adaptive processes becomes less pronounced (\( \epsilon \) approaches 1), gains and losses within each class will influence the rate of change of the mean trait value more. Similarly for continuous traits, as plastic responses become slower, selection against individuals with suboptimal trait values will become more important. As prey recruitment and predation affect the trait dynamics of systems (2), (A10) and (E3) differently, we should expect to see life history-based differences in the effects of phenotypic plasticity on community dynamics.

In particular for system (2), as the rates of adaptive and ecological processes become comparable, we can expect inducible defences to be
destabilizing in some cases. Evolutionary traits have the potential to be destabilizing (Abrams & Matsuda 1997; Cortez & Ellner 2010) and terms similar to those that govern the trait dynamics in evolutionary models are also present in our phenotypically plastic models [e.g. the gradient terms multiplied by $\epsilon$ in the trait equation of system (2)]. Similarly, while rapidly induced defences only synchronize predator–prey oscillations, rapidly evolving traits can also desynchronize cycles and increase the lag between the predator and prey oscillations when costs for defence are small (Cortez & Ellner 2010). Weak costs for inducible defences have been observed in many systems (Buskirk & Steiner 2009), suggesting that inducible defences may desynchronize population dynamics when the rate of induction is comparable to the rates of ecological processes. These conclusions emphasize an advantage of considering fast adaptation limit. While the fast induction limit does not capture all of the effects plastic changes have on population dynamics, it does illuminate how and when additional effects can arise.

The above suggests that without a separation of time scales, evolution and plasticity should have similar effects. However, some dynamics observed in evolutionary predator–prey systems cannot be observed in inducible defence models. Evolution has been shown experimentally and theoretically to yield antiphase or cryptic oscillations in predator–prey systems (Yoshida et al. 2007; Jones et al. 2009; Cortez & Ellner 2010). Our results show that such dynamics are indicative of trait evolution, not plasticity. Fig. 5 demonstrates why these types of oscillations cannot arise in rapidly induced defence systems.

When plastic phenotypic changes are optimal, individuals always have the phenotype with the greatest fitness. Thus, the trait always converges to the highest peak in the fitness landscape (open circles and triangular arrow in Fig. 5). In contrast, evolution is driven by the slope of the fitness curve, i.e. the fitness gradient, and consequently, evolution cannot cross a fitness valley to reach a higher peak. Often, evolutionary and plastic trait dynamics will behave similarly and selection will drive the trait in the direction of increasing fitness and arrive at the highest fitness peak (Fig. 5a and right v-backed arrow of Fig. 5b). Antiphase and cryptic oscillations arise in eco-evolutionary models when the trait dynamics differ.

In continuous trait systems, antiphase and cryptic oscillations occur via bistability when two local fitness maxima of the trait are separated by a fitness minimum (Fig. 5b). In such situations, selection drives the trait in the direction of increasing fitness, but depending on which side of the valley it started on, the trait may arrive at a local, but not global, fitness maximum (left v-backed arrow of Fig. 5b). As plasticity always chooses the most fit trait value, bistability cannot arise with a plastic continuous trait.

In evolutionary systems with a discrete trait (e.g. clonal species), antiphase and cryptic oscillations arise because changes in the mean value of the trait are directly driven by births and deaths within each phenotypic class. This creates a lag between when the direction of selection switches and when a corresponding measurable change in the trait occurs. As discrete plastic traits also respond to environmental stimuli, plastic trait dynamics are decoupled from the birth and death processes within each class. This reduces the lag and prevents antiphase and cryptic oscillations.

The above conclusions do depend on the type of trait and may not hold for all plastic traits. Some studies of learned behavioural traits where individuals can learn from other conspecifics have used models that are nearly identical to the evolutionary trait model (3) (e.g. Abrams & Matsuda 2004). As fitness valleys can arise in such systems and the trait dynamics depend on the fitness gradient, plastic behavioural traits may be able to generate the complex dynamics seen in evolutionary models.

The theory presented in this study can also be applied to phenotypically plastic predator traits [e.g. inducible offenses (Agrawal 2001) or foraging theory (Křivan 2007)]. Rapidly induced offenses that depend on prey density in two species systems stabilize predator–prey oscillations (Appendix A6). Stabilization has been observed in many optimal foraging systems (Yamauchi & Yamamura 2005; Křivan 2007; Uchida et al. 2007), but destabilization is also possible (Abrams 1992). Our results suggest that these different predictions can be understood using similar analysis and may be due to the rate of induction and whether the adaptive process depends on the fitness gradient. Our approach may also be fruitful in understanding community dynamics in systems where both inducible defences and inducible offenses are present (e.g. Feng et al. 2009; Kishida et al. 2009) and how they differ from eco-coevolutionary dynamics.

This work demonstrates that the mechanisms through which adaptive changes arise play an important role in determining what effects they have on community dynamics. The fast phenotypic change limit and the general theory presented here allow us to capture the different effects heritable and plastic adaptation have on ecological systems. In particular, rapidly induced defences tend to stabilize population dynamics and they cannot generate some of the dynamics observed in rapidly evolving systems. Thus, this work begins to unify the theory of adaptive change and to determine how the effects of adaptive change depend on the underlying driving process.

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Appendix S5 An alternative inducible defence model.
Appendix S6 Inducible predator traits.

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