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# Inducible defense destabilizes predator–prey dynamics: the importance of multiple predators

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Phenotypic plasticity in prey can have a dramatic impact on predator–prey dynamics, e.g. by inducible defense against temporally varying levels of predation. Previous work has overwhelmingly shown that this effect is stabilizing: inducible defenses dampen the amplitudes of population oscillations or eliminate them altogether. However, such studies have neglected scenarios where being protected against one predator increases vulnerability to another (incompatible defense). Here we develop a model for such a scenario, using two distinct prey phenotypes and two predator species. Each prey phenotype is defended against one of the predators, and vulnerable to the other. In strong contrast with previous studies on the dynamic effects of plasticity involving a single predator, we find that increasing the level of plasticity consistently destabilizes the system, as measured by the amplitude of oscillations and the coefficients of variation of both total prey and total predator biomasses. We explain this unexpected and seemingly counterintuitive result by showing that plasticity causes synchronization between the two prey phenotypes (and, through this, between the predators), thus increasing the temporal variability in biomass dynamics. These results challenge the common view that plasticity should always have a stabilizing effect on biomass dynamics: adding a single predator–prey interaction to an established model structure gives rise to a system where different mechanisms may be at play, leading to dramatically different outcomes.

Keywords: phenotypic plasticity, inducible defense, stability, synchronization, predator–prey dynamics

## Introduction

Rapid adaptation in predator–prey systems has been shown to affect and sometimes dramatically change predator–prey dynamics. Rapid evolution of defense in prey, in particular, has been extensively studied in this context (Abrams and Matsuda 1997, Abrams 2000, Yoshida et al. 2003, Jones and Ellner 2007, Becks et al. 2010). Here, the frequency of defended and undefended genotypes changes in response to changes in predation pressure (e.g. predator abundance), which may impact the nature of



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predator–prey dynamics in many different ways: changing the period length and phase relationships between predator and prey oscillations (Yoshida et al. 2003, Jones and Ellner 2007, Becks et al. 2010), generating oscillations in an otherwise stable system (Abrams and Matsuda 1997), or dampening them (Ives and Dobson 1987, Abrams and Matsuda 1997, Yoshida et al. 2007).

Rapid increase of prey defense may also result from phenotypic plasticity (Kats and Dill 1998, Tollrian and Harvell1999). Here, defensive prey phenotypes are induced by a visual, chemical, or mechanical cue correlating with predation risk, allowing prey to respond rapidly to predator presence while reducing costs associated with defense when predation risk is low. Here, as with rapid evolution, temporal variability in the frequencies of defended and undefended phenotypes may strongly impact predator–prey dynamics. However, in contrast with the variety of effects rapid evolution may have, induced defense has overwhelmingly been shown to stabilize dynamics, dampening oscillations or eliminating them altogether (Ramos-Jiliberto 2003, Vos et al. 2004, Verschoor et al. 2004, Ramos-Jiliberto et al. 2008a, b, van der Stap et al. 2009, Mougi and Kishida 2009, Cortez 2011, Yamamichi et al. 2011). Direct comparisons between rapid evolution and phenotypic plasticity have consistently shown that plasticity is more stabilizing than evolution (Cortez 2011, Yamamichi et al. 2011), and that other changes in the nature of dynamics (e.g. in phase relationships) are impossible with plasticity (Cortez 2011).

In the theoretical studies cited above, defense is assumed to be subject to a tradeoff: the defended phenotype either has a lower growth or feeding rate (Ramos-Jiliberto 2003, Ramos-Jiliberto et al. 2008b, Yamamichi et al. 2011), or increased respiration or mortality (Vos et al. 2004, Ramos-Jiliberto et al. 2008a, b, Mougi and Kishida 2009). While the assumption of a tradeoff is in itself highly realistic, there are many alternative ways that defense may be costly (Strauss et al. 2002), including an increased susceptibility to pathogens or to other predators. For example, plants with a strong induced defense against spider mites suffered no growth or allocation costs, but turned out to be more susceptible to attack by a specialist beetle (Agrawal et al. 1999). Induced defense in wild radish decreased grazing by rabbits, but increased damage by specialist herbivores (Agrawal and Sherriffs 2001). In aquatic communities, *Daphnia* choosing darker habitats to avoid visually hunting predators were shown to become more susceptible to parasites (Decaestecker et al. 2002). As another example, copepod migration to deeper waters during daytime allows them to escape fish predation, but makes them vulnerable to predation by *Chaoboros* larvae (Neill 1990, 1992). Similarly, many bacteria have the ability to form biofilms, allowing them to escape from predation by suspension-feeding protozoa (Matz and Kjelleberg 2005, Justice et al. 2008), while simultaneously making them vulnerable to surface-feeding grazers such as amoebae (Parry 2004, Weitere et al. 2005).

In such incompatible-defense scenarios (Fig. 1a), defense is context-dependent: which phenotype is currently defended depends on which predator is most abundant, potentially resulting in very different effects on population and community dynamics. However, this has not received much attention from a theoretical viewpoint. Ramos-Jiliberto et al. (2008a) specifically extended models with simple predator–prey pairs to include a second predator, but did not study any scenario with incompatible defense, i.e. where defending against one predator increases vulnerability to another.

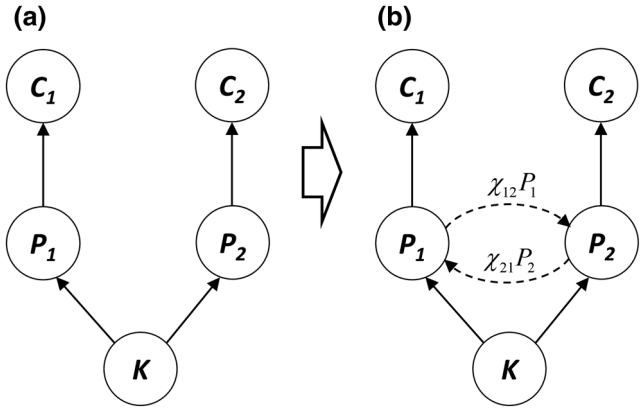


Figure 1. Schematic illustration of the food web analyzed in this study. (a) a two-predator model with non-plastic prey, where prey compete over a shared carrying capacity $K$, and each prey is consumed by a specialist predator ($C_1$ and $C_2$). (b) the model analyzed here in detail: a two-predator system with phenotypically plastic exchange between the prey types $P_1$ and $P_2$.

Here we develop a new one-prey / two-predators model to study this scenario. Phenotypic plasticity in prey allows switching between two distinct phenotypes, where each phenotype is defended against one of the predators and vulnerable to the other (Fig. 1b). Our goal is to study how the degree of switching, measured by the exchange rate between prey phenotypes, affects the variability of community dynamics, where the variability is quantified by the coefficient of variation within each trophic level (prey and predator). To make sure our results are robust and generalizable, we study two different exchange functions describing inducible defense, as well as non-adaptive (diffusive) exchange. Across all these exchange scenarios, we consistently find that increased levels of plasticity (i.e. an increase in the exchange rate between the phenotypes) lowers the stability of the system: the amplitude of oscillations and the coefficient of variation both increase with the degree of plasticity in the prey. We explain this result by showing that the amount of plastic exchange affects synchronization between the two prey phenotypes. Low levels of plasticity result in compensatory dynamics in the prey: the two phenotypes cycle in antiphase with one another, thus buffering and dampening oscillations in total prey biomass. In contrast, high levels of plasticity synchronize the dynamics of the two prey, increasing the temporal variability in total biomass on both the prey and the predator level. These results challenge the common view that plasticity should always have a stabilizing effect on biomass dynamics.

## Methods

### Model description

We consider a simple food web model where a single prey can switch between two phenotypes; each prey phenotype is consumed exclusively by a single specialized predator (Fig. 1b). Because the prey are phenotypes of the same species, they compete directly for e.g. space or nutrients. This system is represented by the following equations:

$$\begin{aligned}\frac{dP_i}{dt} &= r\left(1-\frac{P_1+P_2}{K}\right)P_i - \chi_{ij}P_i + \chi_{ji}P_j - \frac{aP_iC_i}{1+haP_i} \quad i,j\in 1,2;\ i\neq j \\ \frac{dC_i}{dt} &= \left(\frac{\varepsilon aP_i}{1+haP_i} - d\right)C_i\end{aligned} \tag{1}$$

where $P_i$ represents the biomass of prey phenotype $i$, and $C_i$ the biomass of the predator feeding on prey $P_i$. Prey grow logistically, sharing the carrying capacity $K$. Grazing by predators follows a Holling type II functional response, where $a$ and $h$ denote the attack rates and handling times, respectively. $\varepsilon$ represents the conversion efficiency of captured prey biomass into predator biomass, and $d$ the predator mortality rate. For simplicity, we assume symmetry between the food chains, i.e. the two prey and two predator types have identical parameter values (Table 1).

### Exchange rates

Exchange between the two prey phenotypes $P_1$ and $P_2$ is expressed by the exchange rates $\chi_{12}$ and $\chi_{21}$ (exchange from $P_1$ to $P_2$ and vice versa). Switching between prey phenotypes is generally regulated by cues for predation risk. Such cues often directly correspond to predator density, e.g. concentration of kairomones in the water (Kats and Dill 1998, Tollrian and Harvell 1999). Accordingly, most models on inducible defense use switching functions based on predator density (Ramos-Jiliberto 2003, Vos et al. 2004, van der Stap et al. 2009, Yamamichi et al. 2011). However, predation risk is often a function of prey density as well as predator density (Peacor 2003), and it has recently been discovered that many species use cues for both their own density and that of their predators to regulate switching behaviour (Peacor 2003, Van Buskirk et al. 2011, Tollrian et al. 2015). To capture all realistic scenarios, we model both exchange based on predator density and exchange based on predation risk ('predator-avoidant' and 'fitness-dependent' exchange). Additionally, we add a non-adaptive scenario where exchange occurs at a constant per capita rate (i.e. through diffusion); even though this does not strictly fall under inducible defense, it serves as a useful baseline case.

Table 1. Parameters and their standard values of the analyzed model. The system is parameterized for a bacteria–protozoan system (Seiler et al. 2017).

| Parameter | Description | Units | Value |
|---|---|---|---|
| $r$ | prey growth rate | day$^{-1}$ | 0.5 |
| $K$ | carrying capacity | mg C l$^{-1}$ | 1–4 |
| $a$ | attack rate | day$^{-1}$(mg C l$^{-1}$)$^{-1}$ | 5 |
| $h$ | handling time | day | 1 |
| $\varepsilon$ | conversion efficiency | – | 0.33 |
| $d$ | predator mortality | day$^{-1}$ | 0.1 |
| $\chi_{max}$ | maximum exchange rate | day$^{-1}$ | 0.001–1 |

#### *Non-adaptive exchange*

For non-adaptive plasticity, the per capita exchange rates $\chi_{ij}$ are constant. To make this scenario directly comparable to predator-avoidant and fitness-dependent exchange, we set $\chi_{12}$ and $\chi_{21} = 0.5\cdot\chi_{max}$, where $\chi_{max}$ is the maximum exchange rate (Fig. 2).

#### *Predator-avoidant exchange*

In this scenario, prey use a cue directly related to predator abundance (e.g. kairomone concentration) to regulate exchange (Vos et al. 2004). The per capita exchange rate of each prey type increases with the biomass of the predator that it is vulnerable to, assuming a sigmoid curve:

$$\chi_{ij} = \chi_{max}\frac{1}{1+e^{b(C^*-C_i)}} \tag{2}$$

Here, $b$ and $C^*$ determine the steepness and the inflection point of the curve, respectively (Fig. 2a). Increasing $b$ increases the sensitivity of the response to predator presence; the non-adaptive case thus corresponds to $b=0$. To ensure that the inflection points $C^*$ have meaningful values across different parameter combinations, we account for the expected range of predator biomasses the prey will encounter. We thus assume $b$ to be a function of a shape parameter $\alpha$ and the maximum predator biomass $C_{max}$ (Fig. 2a):

$$b = \frac{\alpha}{C_{max}} \tag{3}$$

To determine $C_{max}$ for each set of simulations, we recorded the maximum predator biomass of both predator species during simulations with $b=0$ and either a very low ($\chi_{max}=10^{-3}$) or very high ($\chi_{max}=10$) exchange rate. $C_{max}$ was set at the highest recorded predator biomass; the inflection point $C^*$ was set at $0.5\cdot C_{max}$.

#### *Fitness-dependent exchange*

Because other factors besides predator density may play a role in whether or not switching is adaptive (Peacor 2003, Mougi and Kishida 2009, Mougi 2012, Tollrian et al. 2015), we model a third scenario where exchange rates are a function of fitness differences (Mougi and Kishida 2009). Fitness of each phenotype is here defined as its net per capita growth rate, i.e. the difference between per capita growth and predation rates:

$$F_i = r\left(1-\frac{P_1+P_2}{K}\right) - \frac{aC_i}{1+haP_i} \tag{4}$$

Because we assume that the prey phenotypes have the same $r$ and $K$, the per capita growth rates of the prey phenotypes

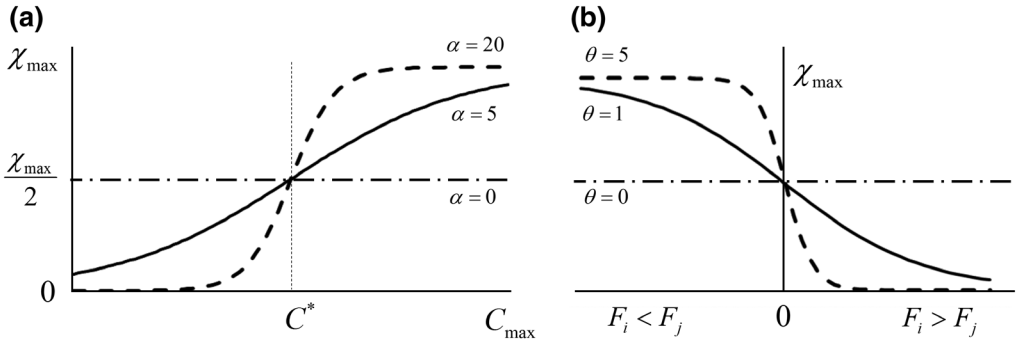


Figure 2. (a) Response functions for predator-avoidant exchange $\chi_{ij}$ for different values of the shape parameter $\alpha$. The inflection point $C^*$ of the response curve is set at $0.5 \cdot C_{max}$; the dash-dotted line denotes non-adaptive exchange ($\chi_{ij} = \chi_{ji} = 0.5\chi_{max}$). (b) Response functions for fitness-dependent exchange $\chi_{ij}$: outgoing exchange is high when the fitness of phenotype $i$ $F_i$ is low compared to the alternative phenotype $j$ $F_j$.

are equal. Thus, in this case, the difference in their fitness reduces to the difference in per capita predation rates:

$$\Delta F_i = \left(F_i - F_j\right) = \frac{aC_j}{1 + haP_j} - \frac{aC_i}{1 + haP_i} \quad (5)$$

The exchange rate is then calculated as a sigmoid function of the fitness difference (Abrams 2007, Mougi and Kishida 2009):

$$\chi_{ij} = \chi_{max} \frac{1}{1 + e^{\theta \cdot \Delta F_i}} \quad (6)$$

where $\theta$ is the shape parameter determining the sensitivity to fitness differences, equivalent to $\alpha$ in the predator-avoidant exchange scenario (Fig. 2b). While this scenario is conceptually similar to predator-avoidant exchange, the two scenarios differ slightly in the resulting temporal patterns of exchange rates (Supplementary material Appendix 1 Fig. A1).

### Simulations

In all simulations we focused on the impact of increasing the maximum exchange rate $\chi_{max}$. We also varied systematically the prey carrying capacity $K$, which is expected to have a strong impact on stability, and the parameters determining the sensitivity of exchange: α for predator-avoidant exchange, and θ for fitness-dependent exchange. Standard values for the other parameters are listed in Table 1. Each simulation was run for 30 000 time steps. A simulated time series of the last 10 000 time steps was used for all calculations.

### Analysis of results

For each simulation run, we calculated the mean biomasses and standard deviations of the dynamics of each prey phenotype and predator separately, and of the total biomass dynamics on both trophic levels ($P_1 + P_2$, $C_1 + C_2$). To measure the overall stability of the food web, we used the coefficient of variation (i.e. the standard deviation of the dynamics, divided by the mean biomass) of the total prey biomass ($P_1 + P_2$) and total predator biomass ($C_1 + C_2$) dynamics (Tilman et al. 1998, Cottingham et al. 2001).

Finally, as a measure of synchronization within each trophic level, we calculated the Pearson correlation coefficients between the net per capita prey growth rates (for synchronization on the prey level) and predator growth rates (for synchronization on the predator level) over the last 10 000 time steps of the simulation (Bjørnstad et al. 1999).

### Data deposition

Data available from the Dryad Digital Repository: <http://dx.doi.org/10.5061/dryad.d8fj8s3> (van Velzen et al. 2018).

## Results

To disentangle the effects of different exchange mechanisms from the effects of increasing the maximum degree of plasticity (i.e. the maximum exchange rate $\chi_{max}$), we first describe the impact of $\chi_{max}$ in the non-adaptive (diffusive) exchange model. These results are then used as a benchmark for comparing the dynamics of adaptive (predator-avoidant and fitness-dependent) exchange.

### Impact of increasing the exchange rate, non-adaptive exchange

Keeping all other parameters constant, changing the exchange rate has a major impact on the dynamics of the food web. Depending on the amount of exchange, three types of dynamics are found (Fig. 3; regions I–III in Fig. 4).

#### *Low exchange rates (region I)*

For low values of $\chi_{max}$, there are compensatory dynamics between the prey phenotypes, and thus also between the predators (Fig. 3a, upper panel). Because each phenotype is high when the other is low and vice versa, changes in phenotype biomasses partly cancel each other out; oscillations in the total prey and total predator dynamics are smaller than those in the individual phenotypes (Fig. 3a, lower panel).

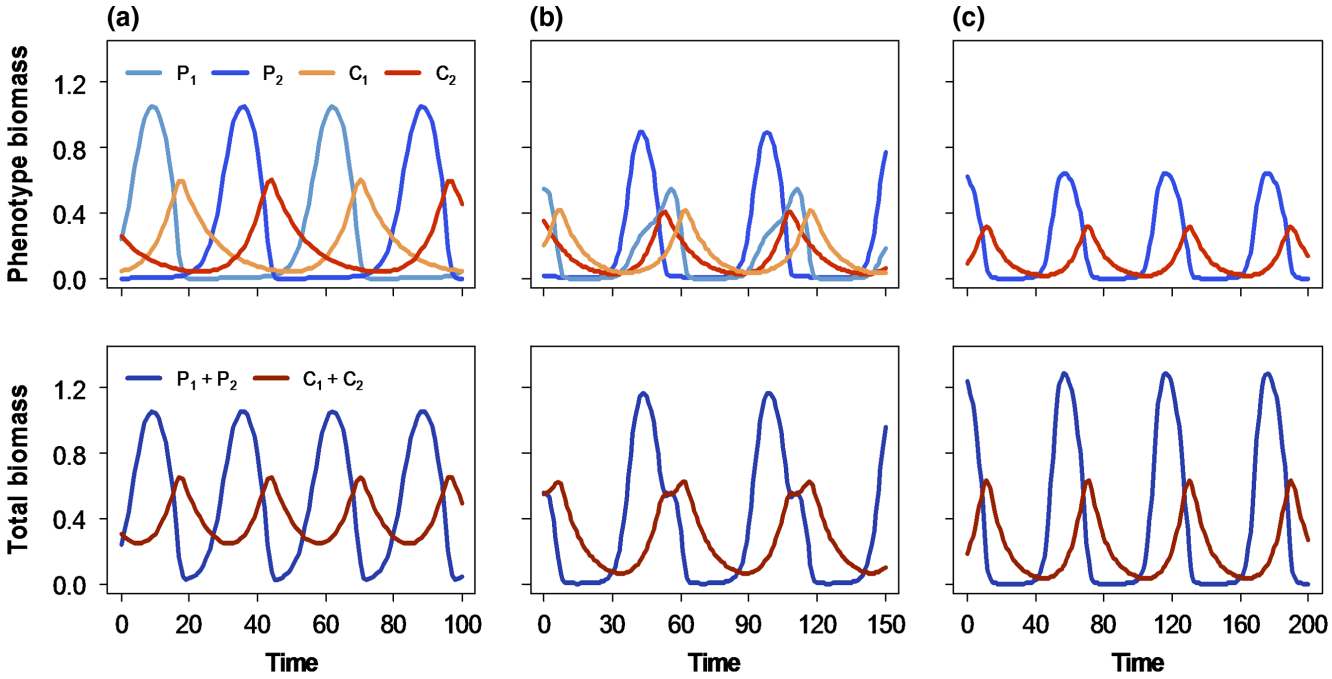


Figure 3. Examples of simulation runs with non-adaptive exchange, corresponding to (a–c) in Fig. 5. (a) low exchange rate ($\chi_{max}=0.005$); (b) intermediate exchange rate ($\chi_{max}=0.05$); (c) high exchange rate ($\chi_{max}=0.5$). Note that in (c), prey phenotypes $P_1$ and $P_2$ and predators $C_1$ and $C_2$ have identical biomasses. $K=1.5$; all other parameters can be found in Table 1.

In other words, the dynamics of each trophic level as a whole are more stable than the dynamics of the individual phenotypes or species within each trophic level.

In addition to the stabilizing effect of the compensatory dynamics, increasing the exchange rate in this region strongly dampens the oscillations in the individual prey phenotypes; predator oscillations are similarly dampened, though only to a lesser degree (Fig. 4, upper left panel). This causes oscillations in total prey and total predator biomass to become dampened as well (Fig. 4, upper right panel). The amplitudes of all oscillations in this region are smaller than those under no exchange at all (Fig. 4, upper panels, region X).

This dampening effect of exchange on the dynamics affects both the mean biomasses and the standard deviations (Fig. 4, middle panels), and through this thus also the coefficients of variation which are used as the measure for overall stability (Fig. 4, lower panels). The standard deviations in prey dynamics decrease with increasing exchange, both in the individual phenotypes (Fig. 4, middle left panel) and in total prey biomass (Fig. 4, middle right panel). At the same time, however, the mean prey biomass declines equally (Fig. 4, middle panels), so that the coefficient of variation remains nearly constant, both within this region and when compared to the scenario with no exchange (Fig. 4, lower panels). In contrast, while the standard deviations in predator dynamics change little, the mean predator biomasses increase with increasing exchange (Fig. 4, middle panels), resulting in a lower coefficient of variation (Fig. 4, lower panels). Thus, while exchange has little to no impact on the stability of prey dynamics in this region, predator dynamics are stabilized; but this stabilization is almost entirely driven by an increase in predator biomass, not by a decrease in the standard deviation.

***Intermediate exchange rates (region II)***

When the exchange rate increases further, the dynamics between the prey become asymmetric (Fig. 3b, top panel). While these dynamics are still compensatory, with maxima of one prey coinciding with low values of the other (Fig. 3b, top panel), the differences in amplitudes cause oscillations in total biomass to become only partly dampened (Fig. 3b). Thus, even though the variability in the individual phenotypes stays nearly constant (Fig. 4, lower left panel), the variability in total prey and predator biomasses increases strongly as the dampening within trophic levels is reduced (Fig. 4, lower right panel).

***High exchange rates (region III)***

At high exchange rates, the dynamics within trophic levels become completely synchronized (Fig. 3c, top panel). Because the maxima and minima of the two phenotypes occur simultaneously, there is no longer any dampening: the coefficient of variation of the total prey and predator biomasses are as high as those of the individual phenotypes (Fig. 4, bottom right panel). The mean biomass on both trophic levels is low (Fig. 4, middle panels).

When we vary both $\chi_{max}$ and the prey carrying capacity $K$, we find that high exchange rates invariably result in synchronization within trophic levels (Fig. 5, bottom panels); and moreover, that the correlations within each trophic level correspond directly to their stability. Negative correlations are

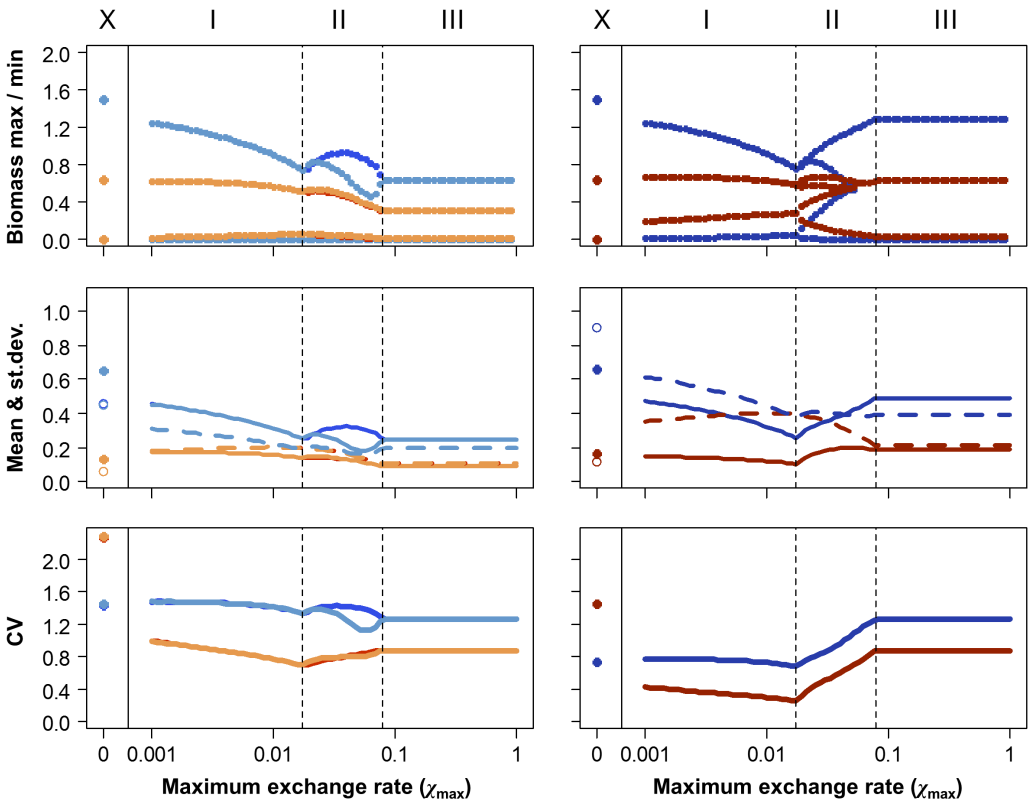


Figure 4. Bifurcation plots of the predator–prey dynamics, mean biomasses and standard deviations, and coefficients of variation (CV) of the dynamics. The very left (region X) shows the comparison with a non-plastic model ($\chi_{max}=0$). Top panels: maxima and minima of biomass dynamics. Middle panels: mean biomass (dashed lines in regions I–III; open symbols in region X) and standard deviations of the dynamics (solid lines in regions I–III; filled symbols in region X). Lower panels: CV (standard deviation divided by the mean). Left panels show the dynamics of individual prey phenotypes (blue) and predators (orange) are shown; right panels the dynamics of total prey (dark blue) and total predators (dark red). Three types of dynamics are found: symmetric compensatory dynamics (region X and region I, see example shown in Fig. 3a); asymmetric compensatory dynamics (region II, see Fig. 3b); and synchronized dynamics (region III, see Fig. 3c).

consistently associated with low CV (i.e. high stability), while positive correlations result in a high CV (Fig. 5, middle and bottom panels). Exchange-induced synchronization again consistently reduces the mean prey and especially the mean predator biomass (Fig. 5, top panels).

### Effect of inducible defenses: adaptive exchange

Under the two adaptive exchange scenarios, predator-avoidant and fitness-dependent exchange, the pattern described above for varying $K$ and $\chi_{max}$ remains the same (Supplementary material Appendix 2, Fig. A2.1, A2.3). Compensatory dynamics, associated with negative correlations, are found under low exchange rates, while high exchange rates result in strongly positive correlations indicating synchronization; and this synchronization has a strong negative impact on stability (Supplementary material Appendix 2, Fig. A2.1, A2.3). Synchronization in both adaptive exchange scenarios typically occurs at higher values for $\chi_{max}$ compared to non-adaptive exchange, especially under predator-avoidant exchange (compare Supplementary material Appendix 2 Fig. A2.1, A2.3 with Fig. 5). Increasing the adaptive value of exchange (i.e. the steepness of the response curves, θ and α; Fig. 2) enhances this pattern (Fig. 6 and 7, lower panels).

For intermediate exchange rates, more complex dynamics may occur: chaotic oscillations (Fig. 8a; Supplementary material Appendix 2 Fig. A2.2, region IIa; Fig. A2.5, region II) and complex but regular oscillations (Fig. 8b; Supplementary material Appendix 2 Fig. A2.2, region IIb) are both possible in this parameter range. Steeper response functions increase the range over which such dynamics are found, especially for predator-avoidant exchange (Fig. 6). Just as the asymmetric oscillations found under non-adaptive exchange (Fig. 3b), the dynamics within trophic levels here still largely appear compensatory (Fig. 8a–b), but the asymmetry in the amplitudes of oscillations causes the dampening within trophic levels to be incomplete, and the variability in these regions is relatively high (Fig. 6– 7, 8a–b, Supplementary material Appendix 2 Fig. A2.2, A2.5).

Finally, under fitness-dependent exchange, a combination of high exchange and a steep response curve can generate asymmetry in the synchronized oscillations (Fig. 7, Fig. 8c, Supplementary material Appendix 2 Fig. A2.5, region IIIb). This results in a slightly lower coefficient of variation

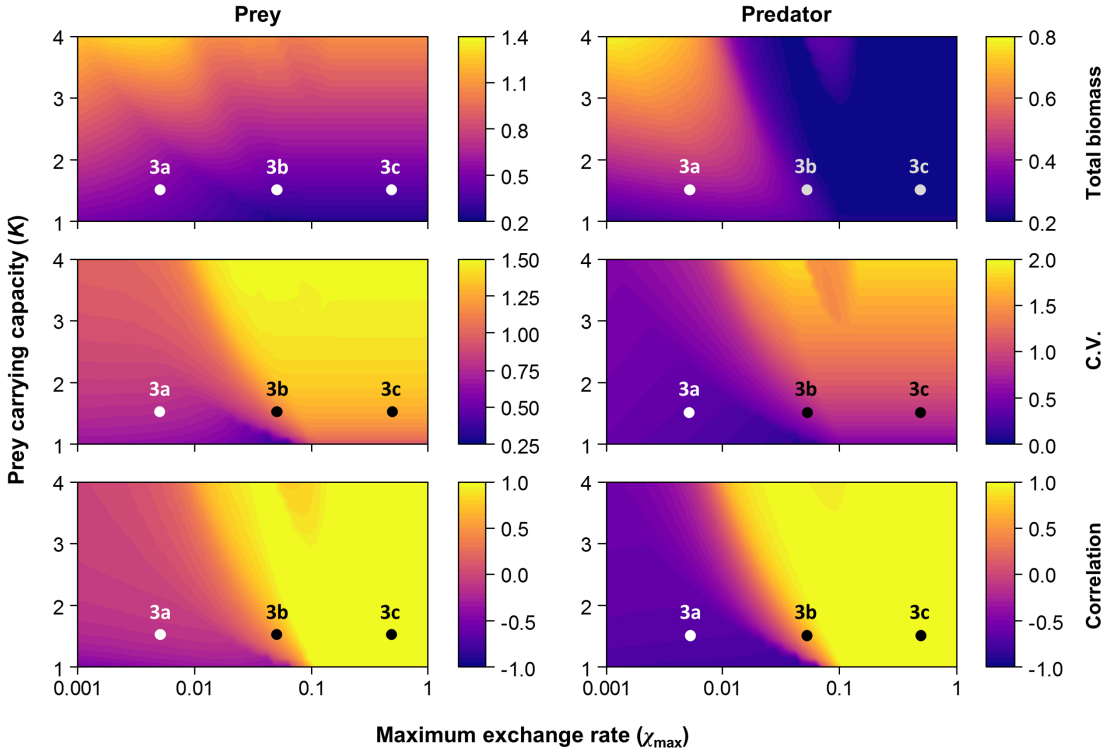


Figure 5. Impact of non-adaptive (diffusive) exchange on total prey and total predator biomass (top), variability in the total biomass on both trophic levels measured by the coefficient of variation (middle), and synchronization between the dynamics of the two phenotypes on both trophic levels, measured by the Pearson correlation between their net growth rates (bottom). Note the different colour scales used for the different panels. For parameter values, see Table 1. 3a, 3b and 3c indicate the parameter combinations used for the time series plots in Fig. 3.

(Fig. 7, Supplementary material Appendix 2 Fig, A2.5), but because the dynamics remain synchronized, the effect on stability is small.

## Discussion

Rapid changes in the level of defense displayed by prey may change population dynamics, resulting in a feedback between ecological and evolutionary dynamics (Yoshida et al. 2003, Schoener 2011). Moreover, it is not only the presence of rapid changes, but the mechanism driving them, that determines the outcome of such feedbacks: there are major differences between the effects of rapid evolution and phenotypic plasticity (Cortez 2011, Yamamichi et al. 2011, Mougi 2012). While rapid evolution can have an array of potential effects on dynamics, previous theoretical work has shown the impact of inducible defense to be much more uniform, consistently pointing towards a stabilizing effect on predator–prey dynamics (Ramos-Jiliberto 2003, Vos et al. 2004, Verschoor et al. 2004, Ramos-Jiliberto et al. 2008a, b, van der Stap et al. 2009). Some minor exceptions to this have been observed: Edelstein-Keshet and Rausher (1989) found that inducible defense can destabilize, but only when predators are subjected to an Allee effect, and even then only under a restricted parameter range. Similarly, some specific combinations of the level of prey competition and the severity and type of costs associated with defense was shown to destabilize dynamics (Ramos-Jiliberto 2003), but this was again an exception to an overall pattern where inducible defense was stabilizing. Finally, Kopp and Gabriel (2006) found that inducible defense may drive cycles in an otherwise stable discrete-time predator–prey model, if the population level of plasticity is very high and defense is very strong.

In this study, we extended an inducible defense model with a single prey and predator pair (Ramos-Jiliberto 2003, Vos et al. 2004, van der Stap et al. 2009, Cortez 2011, Yamamichi et al. 2011) to a two-predator system, where defense against one predator results in vulnerability to another (Strauss et al. 2002, Decaestecker et al. 2002, Weitere et al. 2005). While not uncommon in nature, this scenario has previously received very little theoretical attention. The closest is a study on the dynamic effects of reciprocal phenotypic plasticity (defense in prey and offense in predators; Mougi and Kishida 2009). They showed that a model with incompatible defense is less likely to be stabilizing than a classic "arms race" scenario, where offensive predators feed mostly on undefended prey. However, apart from this, they gave the incompatible defense model no further attention.

Here we studied such a scenario in detail, and find a far more striking result: in strong contrast to previous studies, we consistently show that inducible defense has a destabilizing

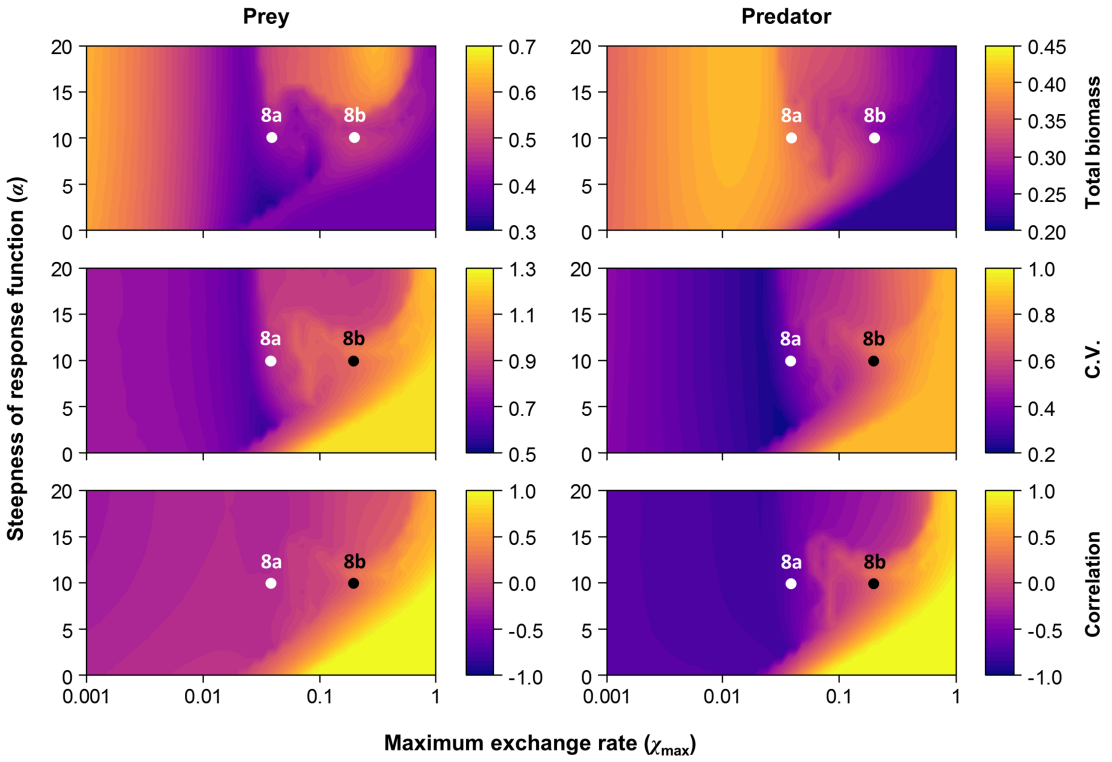


Figure 6. Impact of predator-avoidant exchange on total prey and total predator biomass (top), variability in the total biomass on both trophic levels (middle), and synchronization between the dynamics of the two phenotypes on both trophic levels (bottom). $K = 1.5$; all other parameter values can be found in Table 1. 8a and 8b indicate the parameter combinations used for the time series plots in Fig. 8a–b.

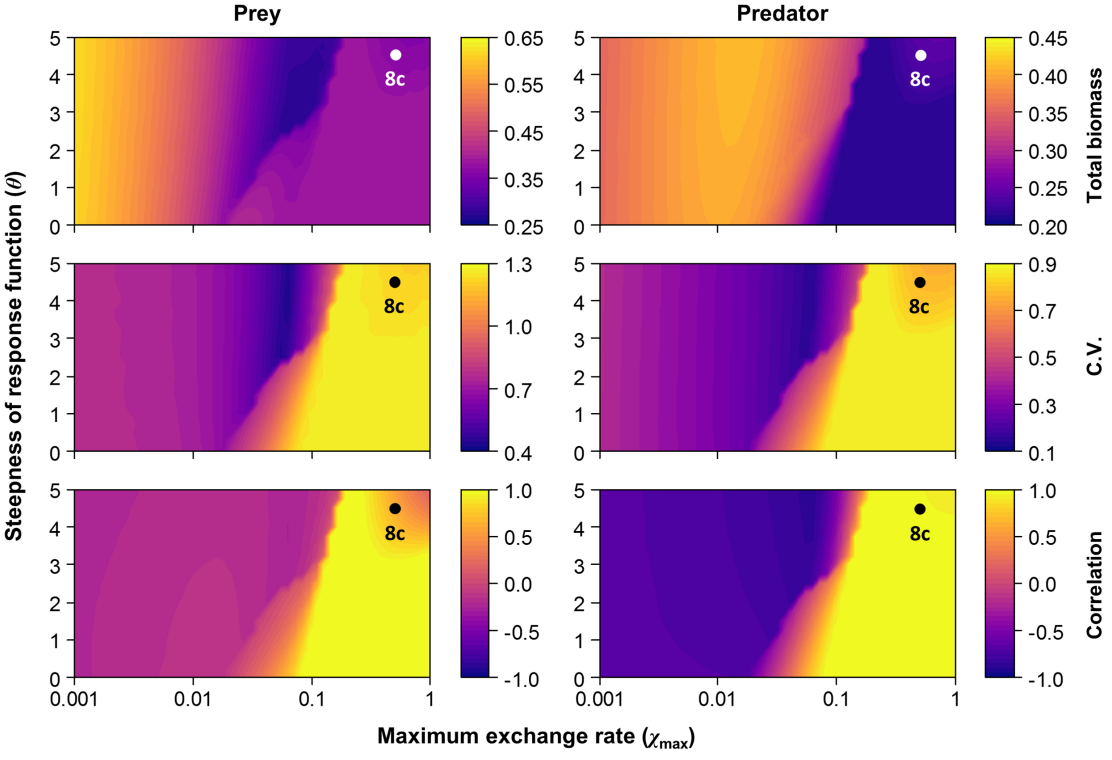


Figure 7. Impact of fitness-dependent exchange on total prey and total predator biomass (top), variability in the total biomass on both trophic levels (middle), and synchronization between the dynamics of the two phenotypes on both trophic levels (bottom). $K = 1.5$; all other parameter values can be found in Table 1. 8c indicate the parameter combinations used for the time series plots in Fig. 8c.

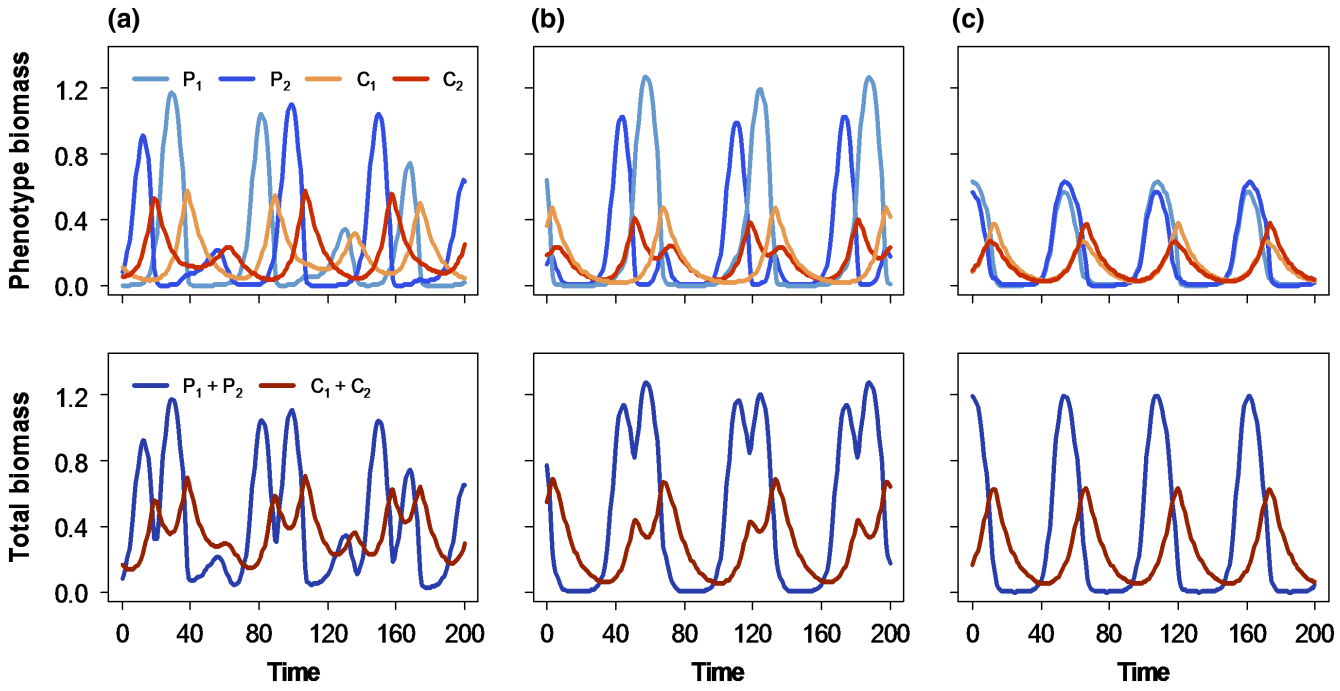


Figure 8. Examples of simulation runs with more complex dynamics not found under non-adaptive exchange. (a–b): dynamics under predator-avoidant exchange, corresponding to 8a and 8b in Fig. 6. (a): chaotic dynamics, $\chi_{max} = 0.04$; (b): complex regular oscillations, $\chi_{max} = 0.2$. (c): dynamics under fitness-dependent exchange, corresponding to 8c in Fig. 7: asymmetric synchronized oscillations, $\chi_{max} = 0.5$.

effect on community dynamics. This result is found for non-adaptive (diffusive) and both types of adaptive (predator-avoidant and fitness-dependent) exchange, and is caused by the strong synchronizing effect of exchange. This synchronization has been demonstrated previously by Vandermeer (2004, 2006), who studied the dynamic effects of coupling between two predator–prey food chains. Coupling through competition between the prey over a shared resource ('resource coupling') results in compensatory dynamics between the prey, and thus also between the predators. In contrast, coupling through niche overlap in the predators ('consumer coupling', i.e. both predators feed to some extent on both prey) results in synchronized prey dynamics. In our model, both types of coupling are present: direct competition over a shared carrying capacity results in resource coupling, while consumer coupling arises indirectly through plasticity in the prey. While the predators in our model are completely specialized and only feed directly on one prey phenotype, indirect feeding on the non-preferred phenotypes is generated by exchange: in the process of avoiding the other predator, a fraction of the non-preferred prey will switch to become the preferred prey. The relative strength of these two types of coupling determines whether prey dynamics are compensatory or synchronized: at low exchange, consumer coupling is very weak and resource coupling dominates, driving compensatory dynamics between the prey phenotypes. Conversely, at high exchange rates, consumer coupling becomes the dominating force, synchronizing the prey and resulting in high-amplitude oscillations in total biomass; in particular, the minima of both prey and predator biomass can fall very low. This explanation holds regardless of the mechanism regulating exchange, with only minor quantitative differences between exchange scenarios. For example, both predator-avoidant and fitness-dependent exchange generally lead to weaker consumer coupling, particularly when the response is very sensitive (Fig. 6, 7), increasing the degree of exchange required to cause synchronization; but the general synchronization pattern remains the same.

Our results are in strong agreement with previous studies on the impact of synchronization on stability. In the dynamics of larger communities, asynchronous or compensatory dynamics between different species at the same trophic level lead to a dampening of total biomass dynamics, resulting in a positive impact on stability (Tilman et al. 1997, 1998, Cottingham et al. 2001). In contrast, when population oscillations are synchronized, total community variability increases and thus stability decreases (Gonzalez and Loreau 2009).

While high exchange rates have a negative impact on stability, our results show that intermediate exchange rates may be stabilizing compared to low exchange rates or no exchange, as long as they remain low enough to prevent synchronization. This positive effect on stability is caused by a net flow from the more abundant to the less abundant prey phenotype, dampening their oscillations. This result is similar to the effects of prey or predator migration between patches in metacommunity models: while small amounts of migration between asynchronous patches generally have a stabilizing effect on metapopulation dynamics, higher migration rates tend to synchronize dynamics across patches,

increasing regional variability and the risk of extinctions (Heino et al. 1997, Gouhier et al. 2010, Hauzy et al. 2010). Our model structure is to some extent analogous to such patch-migration models. However, a critical distinction is that in metacommunity models, prey populations in different patches do not directly compete with one another, while in our model the two prey phenotypes remain a single prey population and continue to compete over the same resources, promoting compensatory dynamics (Vandermeer 2004). This explains why, in metacommunity models, the level of migration necessary to synchronize dynamics is typically very low (Jansen 1999, 2001, Hauzy et al. 2010), which stands in some contrast to our results. In metacommunity models, consumer coupling arises from the same mechanism as in our model; but resource coupling is entirely absent, allowing synchronization to occur even for low migration between patches.

Our results further show that high exchange rates consistently result in decreased total predator biomass. This is true even for the non-adaptive exchange scenario, indicating that this is not driven by active predator avoidance, but is caused at least in part by synchronization within trophic levels. Somewhat more surprisingly, high exchange rates also often result in lowered prey biomass, even in the fitness-dependent exchange scenario (Fig. 7) where exchange can only result in higher net prey growth rates. High levels of phenotypic plasticity, thus, do not necessarily have a positive effect on the prey; instead, synchronization at both prey and predator trophic levels has multiple negative effects. First, because prey compete over a shared carrying capacity, compensatory dynamics allow each prey phenotype to take strong advantage of the available resources when its competitor is at low biomass; conversely, synchronization increases competition, leaving only half of the resources for either prey phenotype. Second, synchronization of the two predators means that prey can no longer escape high predation through switching to the other phenotype. The benefit of exchange is thus lost through synchronization, leaving only the cost (increased competition), resulting in lowered prey biomass. Thus, a high degree of plasticity not only has a negative impact on stability, but it is not necessarily beneficial to the prey itself.

Drawing conclusions on the impact of plasticity on stability requires using an appropriate measure for stability. Following numerous previous studies on the diversity–stability relationship (Tilman et al. 1997, 1998, Cottingham et al. 2001, Schindler et al. 2010), we used the coefficient of variation (CV) as the measure of temporal variability in prey and predator dynamics. As an alternative to this, a stability index is sometimes used, calculated as the mean divided by the standard deviation (Gouhier et al. 2010, Tilman et al. 2006). As this is the inverse of the CV, it conveys the same information. Similarly, previous studies showing a stabilizing effect of phenotypic plasticity also focus on measuring stability by the presence of cycles or the temporal variability of dynamics (Ramos-Jiliberto 2003, Verschoor et al. 2004, Cortez 2011, Yamamichi et al. 2011). However, many other ways of conceptualizing and measuring stability exist, such as the degree of long-term species persistence, or the sensitivity to environmental or other disturbances (Grimm and Wissel 1997). It is entirely possible, even likely, that phenotypic plasticity has a positive effect on stability when measured by such alternatives, for example by preventing extinctions (Vos et al. 2004, van der Stap et al. 2009).

For our analysis we made the simplifying assumptions that both predators are completely specialized on a single prey phenotype. This means that defense against one of the predators is perfect, as would be the case in e.g. biofilm-forming bacteria. While a strong assumption, it does not affect our conclusions. Due to the absence of direct cross-feeding, any degree of consumer coupling in our simulations is generated by exchange between prey phenotypes. When defense is imperfect, the strength of consumer coupling increases, which causes the prey to synchronize at even lower exchange rates (results given in Supplementary material Appendix 3 Fig. A3.1–A3.3), but the overall pattern remains the same.

While theoretical analysis of predator–prey dynamics often focuses on single prey and predator pairs, such interactions are in natural systems always embedded in a more complex food web or community. Results from simple models are not necessarily valid to extrapolate to such complex communities. Extending simple models to include e.g. an additional trophic interaction or an additional trophic level can have a profound impact on dynamics and model predictions (Vos et al. 2004, Ramos-Jiliberto et al. 2008a, Ellner and Becks 2011, Hiltunen et al. 2014). Here we show a strong example of such an effect: adding a single feeding interaction, by adding a second specialized predator, drastically changes the predicted effect of plasticity on community dynamics, and on community stability in particular. Moreover, the mechanism responsible for the degree of dynamic stability in our model (i.e. exchange-driven synchronization between prey phenotypes) is absent in models without a second predator species, explaining why our model predicts such dramatically different outcomes. This finding highlights the importance of taking the ecological context into account, especially when applying model predictions to natural systems. Experimental tests have confirmed the stabilizing effect of inducible defense in systems with a single predator (Verschoor et al. 2004, van der Stap et al. 2009). Whether our model predictions hold up for a two-predator system with incompatible defense, such as biofilm-forming bacteria (Weitere et al. 2005, Seiler et al. 2017), will be an important avenue for future experimental study.

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Supplementary material (available online as Appendix oik-04868 at <www.oikosjournal.org/appendix/oik-04868>). Appendix 1–3.