

When Predators Help Prey Adapt and Persist in a Changing Environment

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ABSTRACT: To persist in a changing world, populations must adapt. The ability to adapt is influenced by interactions with other species, such as predators. Recent experiments and theory suggest that selective pressures arising from predation may help prey adapt phenotypically to changing environments, but how this influences persistence remains unclear. In particular, it has not yet been shown whether predator-induced adaptation can outweigh predator-imposed reductions in population size, allowing prey to persist when they would otherwise go extinct. Here we examine if (and if so, how) predation can enhance the ability of prey to persist in a directionally changing environment. To do so, we extend a single-species quantitative-genetics framework that predicts rates of environmental change beyond which populations go extinct. While we assume predation decreases prey density, we find that predators can indeed help prey persist if they sufficiently increase prey adaptedness (decrease phenotypic lag). We show two ways this can occur: (1) the selective push, in which predators consume maladapted individuals and thus add selection that pushes the mean prey trait toward its optimum; and (2) the evolutionary hydra effect, when predation reduces prey density and thereby increases prey birthrate, allowing more selective events per unit time and effectively reducing generation time. We also discuss how our results apply more broadly to sources of mortality beyond predation.

Keywords: adaptation, evolutionary rescue, extinction, coevolution, quantitative genetics, trophic interactions.

Introduction

Populations are increasingly facing directional environmental shifts caused by climate change and other anthropological impacts (reviewed in Parmesan and Yohe 2003; Davis et al. 2005; Parmesan 2006; Visser 2008; Lavergne et al. 2010; Hoffmann and Sgrò 2011). For example, migrating birds face earlier springs (Møller et al. 2008), diapausing insects face later winter onsets (e.g., Bradshaw and Holzapfel 2001), and ecto-

therms face warming temperatures (e.g., Huey et al. 2009; Thomas et al. 2012). Dispersal and plasticity can help maintain adaptedness and prolong persistence (Holt 1990; Gienapp et al. 2008), but long-term survival ultimately requires genetic adaptation (Visser 2008; Gienapp et al. 2013).

The ability of a population to adapt to environmental change commonly depends on interactions with other species (Holt 1990; Harrington et al. 1999; Tylianakis et al. 2008; Gilman et al. 2010; Lavergne et al. 2010; Van der Putten et al. 2010; Lawrence et al. 2012). One ubiquitous interaction is predation, which can impose strong demographic and selective effects (Dawkins and Krebs 1979; Abrams 2000; Reznick et al. 2008). Because predators tend to reduce prey population sizes (Holt et al. 2008; Salo et al. 2010), thus imposing demographic costs, they can increase the probability of prey extinction following environmental change (Schoener et al. 2001). However, predators can also induce adaptive phenotypic evolution. For example, Tseng and O'Connor (2015) have shown that *Daphnia pulex*, which tend to have smaller body sizes in warm water, evolve to smaller sizes and achieve higher population growth rates when reared in warm water with predators than without. This suggests that predators can sometimes help prey adapt phenotypically in changing environments. However, increased phenotypic adaptation does not necessarily result in increased persistence. If predators are to help maintain biodiversity in the face of environmental change, it must be shown that predator-induced adaptive phenotypic evolution can outweigh predation's demographic cost and allow prey to persist in environments in which they otherwise could not. The results have obvious implications for the fate and conservation priority of populations with and without predators (e.g., continental vs. island, endemic vs. invasive) as well as the conservation practice of removing predators of threatened populations (Reynolds and Tapper 1996; Smith et al. 2010).

There is a small but growing number of theoretical studies on adaptation to gradual, directional environmental change that incorporate interactions between species (de Mazan-

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court et al. 2008; Johansson 2008; Jones 2008; Norberg et al. 2012; Mellard et al. 2015). Only two of these studies assessed predator-prey interactions. Jones (2008) used simulations to study, among other things, a predator-prey system where the predator trait evolves to match the prey trait and the prey trait evolves both to match the changing environmental optimum and escape the predator. His main finding was that predation can sometimes increase the persistence time of the prey, presumably when the predator removes prey that are maladapted to the abiotic environment. Mellard et al. (2015) studied an explicit resource-plant-herbivore trophic chain using a primarily numerical approach to ask how the addition of an herbivore affects plant persistence in a warming environment. They analytically clarified the conditions required for the predator to hasten prey evolution but found in simulations that, regardless of the effect on evolutionary rates, herbivory almost never aided plant persistence. In addition, Northfield and Ives (2013), who did not explicitly model a gradually changing environment, analytically demonstrated that conflicting interactions, such as predation, tended to dampen the direct effect of small environmental changes on the equilibrium densities of interacting species, potentially facilitating persistence. In their model, coevolution facilitates persistence through negative feedback loops. For example, as the predator nears extinction, the prey invests less in defense, making it easier for the predator to consume prey and rebound in abundance. Thus, up to this point, while it is clear that predators can theoretically help prey adapt in changing environments, the effect of predators on prey persistence remains unresolved.

Here we clarify the effect of predators on the ability of prey to persist in a gradually changing environment within a well-established quantitative-genetic framework (Pease et al. 1989; Lynch et al. 1991; Lynch and Lande 1993; Bürger and Lynch 1995, 1997; Lande and Shannon 1996; Chevin et al. 2010), which can be applied to both laboratory (Willi and Hoffmann 2009) and wild (Gienapp et al. 2013) populations. The framework allows analytical predictions of population densities and mean trait values, as well as the critical rate of environmental change, here defined as the minimum rate of environmental change that causes an equilibrium population size of zero. Under this framework, predators affect prey persistence if they alter the prey's critical rate of environmental change.

While there are many ways for predators to hinder prey persistence (most obviously by reducing prey population size), we find that predators can help prey persist (1) when predators prefer maladapted prey and thus push the mean prey trait closer to its moving optimum (selective push) and (2) when the potentially nonselective mortality induced by predators increases prey birthrates and thus increases the rate of selective events, accelerating prey evolution (evolutionary hydra effect).

Methods and Results

A General Model of Generalist Predation

Let prey individuals with trait value z , in a prey population of density N , be born at a per capita rate of $b(z, N)$ and die with predator-independent per capita mortality rate $m(z, N)$. Additionally, let a population of predators kill prey individuals at a per prey rate of $kf(z, N)$, where k is the overall predation intensity and f includes the potential dependence on prey trait value, which describes direct selective effects (e.g., increased consumption of slow, large, or maladapted individuals), as well as the potential dependence of predation on prey density, which allows, for example, a type II or type III functional response. We do not explicitly model predator density and trait values here (but see the "Specialist Predation" section). Instead, we assume the predator is a generalist that consumes a sufficient number of other prey species such that its density and trait values are relatively constant and the predation rate depends only on the density and trait values of the focal prey. Alternatively, constant predator trait values and density could result from the predator having a much longer generation time than the prey or from predator migration from nearby habitats. For example, with a type II functional response with handling time h and attack rate $a(z)$, we have $kf(z, N) = a(z)[1 + ha(z)N]^{-1}P$, where predator density P is approximately constant over the timescale of interest. Although we wish to interpret $kf(z, N)$ as generalist predation here, it could arise from any source of mortality whose per capita rate does not vary too much in time with factors other than prey trait value and population density. Under these conditions, the rate of change in prey density is approximately

$$\begin{aligned} \frac{dN}{dt} &= N \int_{-\infty}^{\infty} [b(z, N) - m(z, N) - kf(z, N)]p(z)dz, \\ &= N[\bar{b}(\bar{z}, N) - \bar{m}(\bar{z}, N) - \bar{k}\bar{f}(\bar{z}, N)], \end{aligned} \quad (1)$$

where $p(z)$ is the probability distribution of trait values in the prey population, \bar{b} , \bar{m} , and $\bar{k}\bar{f}$ are the population mean per capita birth, nonpredator mortality, and predation rates, respectively, and \bar{z} is the population mean trait value.

When phenotypic variance is small, the rate of evolution in the mean trait value is roughly

$$\frac{d\bar{z}}{dt} = V_A \frac{\partial}{\partial \bar{z}} \left(\frac{1}{N} \frac{dN}{dt} \right) \propto \frac{\partial \bar{b}}{\partial \bar{z}} - \frac{\partial \bar{m}}{\partial \bar{z}} - k \frac{\partial \bar{f}}{\partial \bar{z}} \quad (2)$$

(Lande 1976), where $V_A > 0$ is the additive genetic variance, which determines the strength of the response to selection. Predators could potentially affect prey adaptation by influencing prey variance, V_A , but here we simplify the dynamics and assume the variance is constant and independent of predation. In the examples that follow, a constant variance re-

sults from assuming that offspring trait values are near the mean of their parents. This causes an equilibrium to be reached between the variance introduced by segregation and recombination and the variance lost through random mating (see supplementary Mathematica file, available online, for details).

We now consider a changing environment. Let there be some trait value, θ , that is optimal in the sense that it maximizes prey growth rate at a given density in the absence of predators (i.e., $z = \theta$ maximizes $b - m$ at a given N). We assume that the environmental change causes this optimum to increase linearly at rate δ . If the prey persists, the mean prey trait will evolve to track the optimum, eventually settling into a steady-state lag (sensu Lynch et al. 1991), with the mean trait value typically trailing the optimum by a constant amount. A steady state is achieved when prey density is constant in time (eq. [1] is zero) and the rate of evolution (eq. [2]) is equal to the rate of change in the environment, δ . Assuming a stable steady state exists, we can solve for equilibrium population density (\hat{N}) and the corresponding steady-state lag ($\hat{L} = \lim_{t \rightarrow \infty} L = \lim_{t \rightarrow \infty} (\theta - \bar{z})$). The minimum rate of change in the optimal trait value that causes extinction at this steady-state lag ($\hat{N} = 0$) gives the critical rate of environmental change, δ_c (sensu Lynch et al. 1991). This is the slowest rate of environmental change that deterministically causes the prey to go extinct (unless the steady state first loses stability; see discussion in “Generalist Example 2: Evolutionary Hydra Effect”).

What we ultimately want to know is how increased predation intensity affects prey persistence, $d\delta_c/dk$. This will depend on how predators affect prey adaptation, which we turn to first. We begin with a very general description and follow this with concrete examples, where we choose specific functional forms for the terms in equation (1).

Effect of Predators on Prey Adaptation

We begin with the reasonable assumption that increasing predation intensity decreases prey density at a given mean lag, $\partial N/\partial k < 0$ (see Abrams 2009 for exceptions). Without loss of generality, we specify the direction in which the trait is measured such that the mean prey trait value lags behind the optimum, $\theta > \bar{z}$. Then a small increase in the mean trait value would increase the mean prey growth rate in the absence of the predator, $\partial(\bar{b} - \bar{m})/\partial \bar{z} > 0$. We say that predation selects in the same direction as the environment when increasing mean prey trait value decreases predation as well as increases the mean prey growth rate in the absence of predators, that is, when $\partial \bar{f}/\partial \bar{z}$ and $\partial(\bar{b} - \bar{m})/\partial \bar{z}$ have opposite signs.

Despite its negative effect on prey density, predation could help the prey persist if it helps the prey adapt, that is, if increasing the predation intensity increases the rate

at which prey evolve. Mathematically, predators help prey adapt when

$$0 < \frac{d}{dk} \frac{d\bar{z}}{dt},$$

$$0 < \frac{\partial}{\partial N} \left(\frac{\partial \bar{b}}{\partial \bar{z}} \right) \frac{\partial N}{\partial k} - \frac{\partial}{\partial N} \left(\frac{\partial \bar{m}}{\partial \bar{z}} \right) \frac{\partial N}{\partial k} - k \frac{\partial}{\partial N} \left(\frac{\partial \bar{f}}{\partial \bar{z}} \right) \frac{\partial N}{\partial k} - \frac{\partial \bar{f}}{\partial \bar{z}}. \quad (3)$$

Thus, to know if increasing predation intensity will speed up the rate of prey evolution, we need to know the signs of the terms on the right-hand side of equation (3).

Most intuitively, the last term in equation (3) shows that when predators select in the same direction as the environment (what we call a selective push; here $\partial \bar{f}/\partial \bar{z} < 0$) increased predation intensity will help the prey adapt directly through selection regardless of whether the predation rate is density dependent (fig. 1). Of course, selection from the predator that opposes selection from the environment (a selective pull; here $\partial \bar{f}/\partial \bar{z} > 0$) will hinder prey adaptation.

Even without direct selection via predation ($\partial \bar{f}/\partial \bar{z} = 0$), predators can facilitate adaptation by altering prey demography. Negative density dependence causes birthrates to decline with population density, $\partial \bar{b}/\partial N < 0$. Thus we may expect that the mean per capita birthrate will increase less with mean trait value at higher densities, $\partial(\partial \bar{b}/\partial \bar{z})/\partial N < 0$. Therefore, with negative density-dependent population growth and our initial assumption that predation decreases prey density ($\partial N/\partial k < 0$), increasing predation intensity can help prey adapt when selection and density dependence affect birthrates (first term in eq. [3]). Essentially, the predator (or any other form of nonselective mortality) depresses prey density, which elevates prey birthrates, increasing the expected number of selective events in a given time and thus accelerating the rate of evolution in a changing environment (fig. 1). We call this phenomenon the evolutionary hydra effect (after the nonevolutionary hydra effect described by Abrams 2009), referring to the ability of the mythical monster to regrow two heads for every one severed by Heracles. In our case, due to selection, the heads that grow back tend to be better adapted than the one that was severed. The evolutionary hydra effect can occur whenever generation times can be decreased, allowing more opportunity for selection per unit time. This applies to univoltine species with overlapping generations (e.g., many temperate bird species), whenever the average age of parents is decreased by mortality. Generation times cannot, however, be decreased in univoltine species with nonoverlapping generations, where generation time is fixed (e.g., annual plants; see supplementary Mathematica file for discrete time models with overlapping and nonoverlapping generations).

The opposite is true for per capita death rates; with negative density dependence, increasing density increases death

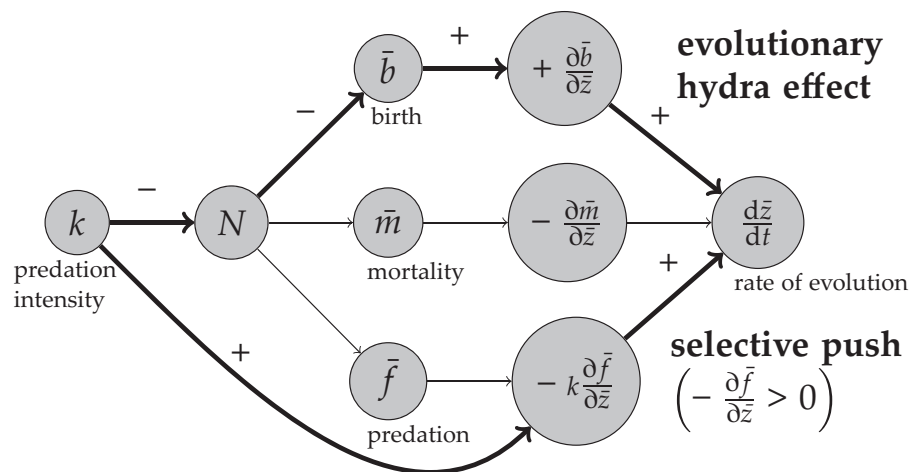


Figure 1: Pathways by which predation intensity, k , can affect the rate of prey evolution, $d\bar{z}/dt$. Boldface lines show our two examples: the evolutionary hydra effect (*top*) and the selective push (*bottom*). Positive and negative symbols give the sign of the partial derivative of the right variable with respect to the left variable in our examples (e.g., the negative symbol between k and N indicates that prey density, N , declines with increasing predation intensity, $\partial N/\partial k < 0$). Increasing predation intensity increases the rate of prey evolution (toward larger trait values) via a specific pathway when the product of the signs along that pathway is positive.

rates, $\partial \bar{m}/\partial N > 0$, making it likely that death rates decrease more with increases in trait value at high densities, $\partial(\partial \bar{m}/\partial \bar{z})/\partial N < 0$. That is, the opportunity for selection via natural mortality is likely to be stronger at larger population sizes (in the absence of predators) than at smaller ones (with predators). Thus, selection and negative density dependence acting on prey mortality (along with dN/dk) means that predation likely reduces the ability of prey to adapt (the second term in eq. [3] is likely negative). However, with positive density dependence (e.g., when the population size is very small and there are Allee effects), the situation is reversed; increased predation rates could then help the prey adapt when selection and density dependence both act on death rather than birth.

Assuming negative density dependence again, predation can also help the prey adapt if $-\partial(\partial \bar{f}/\partial \bar{z})/\partial N < 0$ (third term in eq. [3]). This is most likely when per capita predation rates weaken as the population size grows ($\partial \bar{f}/\partial N < 0$) so that predation is more effective—and more selective—when predation has depressed prey densities. Whether this occurs depends on the nature of the functional response. A type I functional response implies that density has no effect on per capita predation rates ($\partial \bar{f}/\partial N = 0$), a type II functional response implies that density has a negative effect on per capita predation rates ($\partial \bar{f}/\partial N < 0$), while a type III functional response implies that density has a positive effect on per capita predation rates at low densities ($\partial \bar{f}/\partial N > 0$) and a negative effect on per capita predation rates at high densities ($\partial \bar{f}/\partial N < 0$). It is therefore possible for increasing predation intensity to aid adaptation indirectly through changes in density (as in the evolutionary hydra effect de-

scribed above) even if prey birth and natural mortality are not selective (first two terms in eq. [3] are zero) but predation is ($\partial \bar{f}/\partial \bar{z} \neq 0$). This would occur, for example, with a type II functional response and predation that selects in the same direction as the environment. In this case, the decreased prey density resulting from increased predation intensity causes an increase in the per capita rate of predation, and since predation selects in the same direction as the environment, it accelerates the evolution of the mean prey trait value toward the optimum (i.e., the evolutionary hydra effect enhances the selective push).

Effect of Predators on Prey Persistence

While considering the form of equation (3) provides insight into when predation can help prey adapt (decreased phenotypic lag), satisfying this condition does not necessarily mean that predation will help prey persist (maintain positive population density) in a more rapidly changing environment. Because predation reduces prey density, it can lower the critical rate of environmental change, δ_c , even if the prey are more adapted.

From equation (2), we can write the critical rate of environmental change as

$$\delta_c = V_A \left. \frac{\partial g(\bar{z}, k)}{\partial \bar{z}} \right|_{\bar{z} = \bar{z}_c(k)}, \quad (4)$$

where $g(\bar{z}, k) = \bar{b}(0, \bar{z}) - \bar{m}(0, \bar{z}) - k\bar{f}(0, \bar{z})$ is the per capita growth rate when rare (invasion fitness) and $\bar{z}_c(k)$ is the mean trait value that causes equilibrium population

size to be zero (critical trait value). Letting $h(\bar{z}_c(k), k) = \partial g(\bar{z}, k) / \partial \bar{z} |_{\bar{z} = \bar{z}_c(k)}$ be the selection gradient at the critical rate, we can take the derivative with respect to predation intensity on both sides, showing that predation increases the critical rate when

$$0 < \frac{d\bar{z}_c}{dk} = V_A \left[\frac{\partial h}{\partial k} + \frac{\partial h}{\partial \bar{z}_c} \frac{\partial \bar{z}_c}{\partial k} \right], \quad (5)$$

$$0 < V_A \left[\frac{\partial^2 g}{\partial \bar{z} \partial k} + \frac{\partial^2 g}{\partial \bar{z}^2} \frac{\partial \bar{z}_c}{\partial k} \right]_{\bar{z} = \bar{z}_c(k)}.$$

Restricting ourselves to cases where extinction occurs because equilibrium population density goes to zero (as opposed to an unstable steady state), predation helps persistence when it helps adaptation by bringing the trait closer to the optimum (eq. [3] is satisfied) and increases the critical rate of environmental change (eq. [5] is satisfied).

The first term inside the brackets of equation (5) is a mixed derivative that describes the impact of selective predation on persistence. It is negative when there is a selective pull, positive when there is a selective push, and zero otherwise. The second term is comprised of two derivatives. One of these is a second derivative that describes how the shape of invasion fitness changes with mean trait value. It is zero when invasion fitness depends linearly on trait value, negative when invasion fitness increases slower with trait value closer to the optimum (concave down), and positive when invasion fitness increases faster with trait value closer to the optimum (concave up). The second part of the second term describes how the critical trait value depends on predation intensity. Since predation is assumed to lower per capita growth rate, the critical trait value will increase toward the optimum with increases in predation intensity, and thus this derivative is positive (i.e., a prey population on the brink of extinction needs to become better adapted to the abiotic environment if it is to persist with more predation). We can therefore reason that in the absence of selective predation, the evolutionary hydra effect will help persistence if and only if invasion fitness is concave up at the critical trait value. For example, if selection occurs only through births, the evolutionary hydra effect cannot help persistence if birthrate is a quadratic function of phenotype (concave down for all trait values), but it can if birthrate is a Gaussian function (concave up for critical trait values that are sufficiently far from the optimum). In other words, small decreases in lag at the persistence boundary must cause large enough increases in invasion fitness for the evolutionary hydra effect to help persistence. Recall too that equation (3) must also be satisfied, so that if selection is acting through births, so too must density dependence.

We now explore two specific examples, comparing prey populations with and without a generalist predator, to dem-

onstrate that, when the environment begins to change, predation can indeed increase prey persistence by facilitating prey evolution through (1) a selective push and (2) the evolutionary hydra effect. We do so with analytical expressions for steady-state phenotypic lag and equilibrium density as well as numerical and simulated temporal trajectories beginning from demographic equilibrium in an originally constant environment. These specific examples confirm the generic statements drawn from equations (3) and (5) (see supplementary Mathematica file).

Generalist Example 1: Selective Push

Here we explore an example where a selective push from the predator can increase prey persistence. To eliminate the possibility of an evolutionary hydra effect, we assume that per capita prey birthrates are negatively density dependent but independent of trait value $b(z, N) = b(N) = b_{\max}(R_{\text{tot}} - N)$. The per capita birthrate, equal for all individuals, reaches its maximum, $b_{\max} > 0$, at low prey density and declines linearly as prey density increases, due to decreasing resources. We assume that initial prey density is not greater than the total amount of resources in the system, $0 \leq N(0) \leq R_{\text{tot}}$, which ensures $0 \leq N(t) \leq R_{\text{tot}}$ and a nonnegative birthrate, $b(z, N) \geq 0$, for all time $t \geq 0$. We measure population density in units of resource content, such that if R_{tot}^* is the density of some limiting nutrient and each unit of prey density requires density q of this nutrient for life, then $R_{\text{tot}} = R_{\text{tot}}^*/q$. We assume the per capita prey death rate is density independent, with stabilizing selection on trait values around an optimum, $m(z, N) = m(z) = m_{\min} + \gamma(\theta - z)^2$. Individuals with the optimum trait value, $z = \theta$, have the minimum per capita death rate, $m_{\min} > 0$. Per capita death rate increases with the square of the deviation of the trait value from the optimum, with quadratic selection strength $\gamma > 0$. Thus, in the absence of predators, there is a single selective pressure, which drives the mean prey trait value, \bar{z} , toward the optimum, θ .

Finally, the selective push is modeled by assuming that the per capita predation rate on prey with trait value z is $kf(z, N) = kf(z) = k[1 + \gamma_k(\theta - z)^2]$, describing a system where maladapted prey also suffer more predation. Prey with the optimum trait value are predated on least, at per capita rate $k \geq 0$, and the rate of predation increases quadratically as prey traits vary from θ . Predation thus produces a second selective pressure, which magnifies the strength of selection and drives the mean prey phenotype toward the optimum. In the supplementary Mathematica file, we also analyze a model where the selective push originates from predators preferentially consuming prey with the smallest trait values (the results do not differ qualitatively).

With the above assumptions, trait and density dependence never affect the same demographic rate, so the first

three terms in equation (3) must be zero. Only the final term remains, describing the selective push of predation ($-\partial\bar{f}/\partial\bar{z} > 0$ when $\bar{z} < \theta$).

In the supplementary Mathematica file, we analytically derive the rate of change in prey density assuming prey trait values are normally distributed with phenotypic variance V_z . Following Débarre and Otto (2016), we derive approximations for the rate of evolution and the equilibrium additive genetic variance in a large population assuming that mating is random and that offspring inherit the average value of the two parental trait values plus a small normally distributed random effect with mean zero and variance α^2 (which mimics segregation and recombination). We then use these approximations to calculate the equilibrium density, steady-state lag, and critical rate of environmental change.

Figure 2A shows how the mean prey trait value with a predator (black) is pushed closer to the optimum than the mean prey trait value without a predator (gray). Even though predation decreases prey density for a given trait value (see fig. 2B at time 0), predation indirectly increases prey density in the long run through the above-mentioned selective push (fig. 2B). Throughout, phenotypic variance remains roughly constant near its predicted equilibrium value (fig. 2C). Figure 3A shows the various selective pressures experienced by the prey population with (black) and without (gray) predation at the steady state shown in figure 2A–2C.

Figure 2D–2F shows the steady-state lag and equilibrium density and phenotypic variance as functions of the rate of environmental change for a prey population with (black) and without (gray) predation. Here we can see that prey populations with a predator persist at higher rates of environmental change than those without predators, demonstrating that a selective push from predators can help prey persist ($N > 0$) in more extreme environments. Finally, it should be noted that higher rates of predation can depress prey density to the point that the demographic cost of predation outweighs the selective push and predation no longer helps prey persist (i.e., eq. [5] is not satisfied because the negative second term dominates; see supplementary Mathematica file).

Generalist Example 2: Evolutionary Hydra Effect

Here we show that, even in the absence of a direct selective effect, predation (or any other form of nonselective mortality) can increase prey persistence when selection and negative density dependence act on prey per capita birthrate. This occurs by increasing the rate of selective events via the evolutionary hydra effect.

Let the per capita birthrate have the same form of linear negative density dependence as in the previous example but now also depend on trait value. We assume that, at a given density, only individuals with trait value $z = \theta$ have the maxi-

mum per capita, per available resource birthrate, $b_{\max} > 0$, and that birthrate declines to zero as trait values deviate in either direction from θ . Using a Gaussian form of decline, the per capita birthrate is $b(z, N) = b_{\max} \exp[-(\gamma/2)(\theta - z)^2]$ ($R_{\text{tot}} - N$), where $\gamma > 0$ determines how quickly the birthrate declines with trait deviations from θ . We assume the per capita death and predation rates are constants that do not depend on trait values or density: $m(z, N) = m$ and $kf(z, N) = k$. Thus, with or without predators, the prey experience only one selection pressure (through births), although the strength of this selection pressure now varies with available resources, $R_{\text{tot}} - N$. Because predation affects prey density it also changes the amount of available resources in the system, altering the rate of selective events. Using the same assumptions as the previous example, we analyze the model in the supplementary Mathematica file and summarize the results below.

Figure 4A shows that nonselective predation can help prey adapt in a changing environment, and figure 4B shows that being better adapted can translate into higher population density at equilibrium. Figure 3B shows the selective pressures experienced by the prey population with (black) and without (gray) predation at the steady state shown in figure 4A–4C. Figure 4D then illustrates how the higher population densities with predation allow persistence at faster rates of environmental change. Thus even nonselective predators can help prey persist by increasing the rate of births and hence the rate of selective events (the evolutionary hydra effect). As in the selective push example, higher rates of predation can depress prey density to the point that they overwhelm the benefit provided by the evolutionary hydra effect, hindering prey persistence (the critical lag becomes smaller, causing the second term in eq. [5] to become negative; see supplementary Mathematica file).

In this example, not only does the rate of change in prey density directly depend on mean trait value, but the rate of change in the mean trait value directly depends on prey density (the latter was not true in the selective push example). There is thus potential for strong eco-evolutionary feedbacks. In particular, large additive genetic variances strengthen feedbacks that can cause eco-evolutionary cycles to emerge through a Hopf bifurcation. These cycles can grow and become unstable, causing prey extinction at rates of change smaller than the critical, δ_c . Such cycles require that evolutionary and ecological changes occur on similar time frames and do not emerge when genetic variation in the trait is small or predation intensity is strong (see supplementary Mathematica file).

Specialist Predation

Specialist predators—those that primarily consume a single prey species—require a sufficient amount of biomass production by their prey to persist. Thus, if a specialist

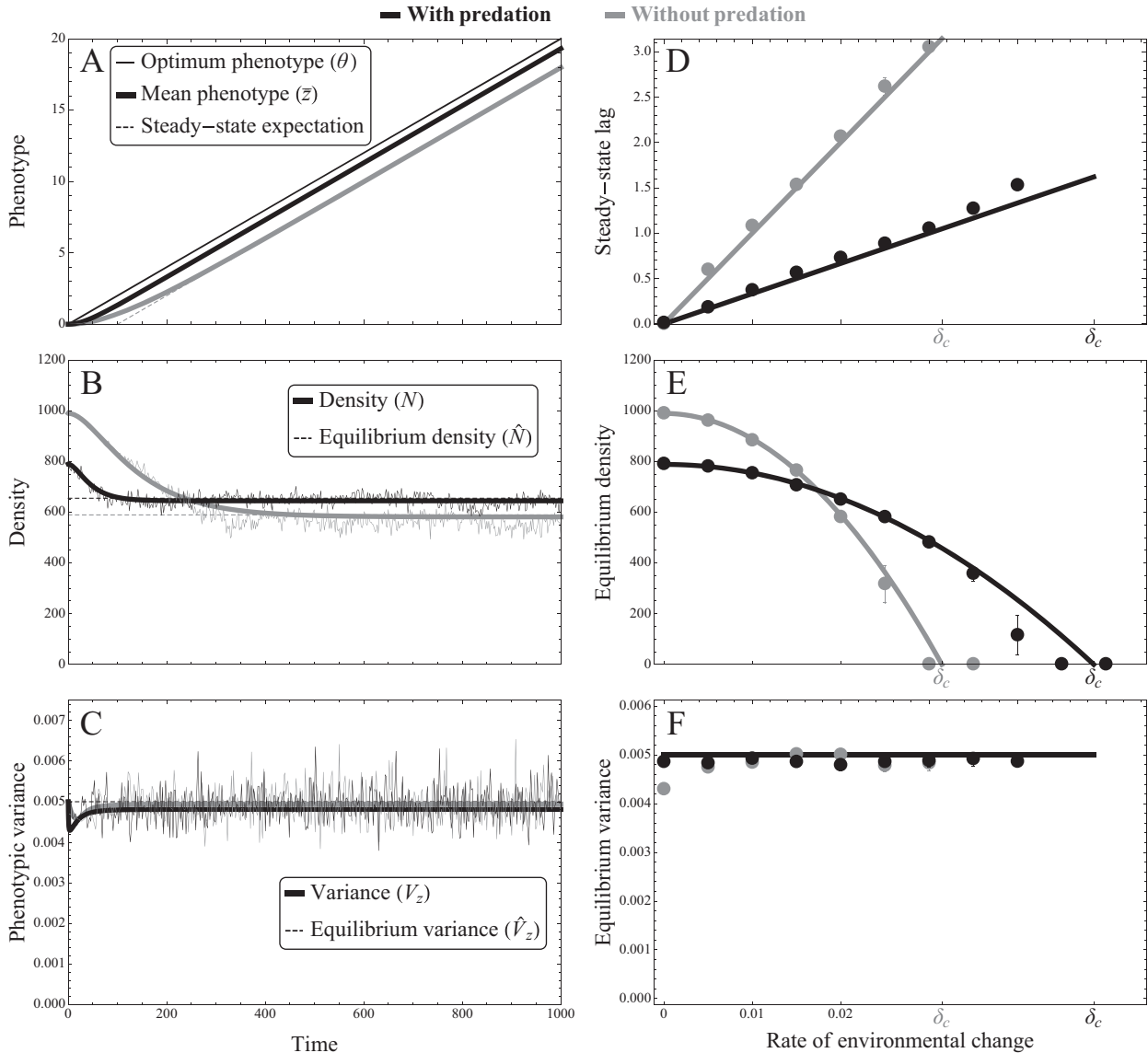


Figure 2: Selective push. A–C, Temporal dynamics of mean prey phenotype, population density, and phenotypic variance when the optimum phenotype changes at rate $\delta = 0.02$. Dashed curves are analytical solutions for equilibrium values (supplementary Mathematica file). Thick curves are numerical solutions (supplementary Mathematica file). Thin curves are results from one simulation (obscured by thick curves in A; appendix). D–F, Equilibrium dynamics of steady-state phenotypic lag, population density, and phenotypic variance as functions of the rate of environmental change. Curves give analytical results (supplementary Mathematica file). Dots give simulation results (mean ± 1.96 SE of 10 replicates; appendix). Parameters: $b_{\max} = 0.01$, $R_{\text{tot}} = 1,000$, $m_{\min} = 0.1$, $\gamma = 1$, $\gamma_k = 1$, $\alpha = 0.05$, $k = 0$, (gray), $k = 2$ (black).

did not affect prey lag, the predator would go extinct at a lower rate of environmental change than its prey (at the point where prey biomass production dropped below the threshold required for predator persistence), and therefore specialists would not affect prey persistence. However, specialist predators can affect prey lag in the same ways that generalists can. It can therefore be reasoned that, if a specialist predator reduced prey lag enough to maintain sufficient prey biomass production at rates of environ-

mental change greater than that which would allow the prey to persist on its own, the predator could sustain itself and, by necessity, help its prey persist.

We demonstrate the above logic by explicitly modeling specialist predator density, P , and trait values, z_p . In general, per capita rates of prey birth (b), death (m), and predation (kf) could now depend on the traits and densities of both species. However, our goal here is only to show that the selective push and evolutionary hydra effect can

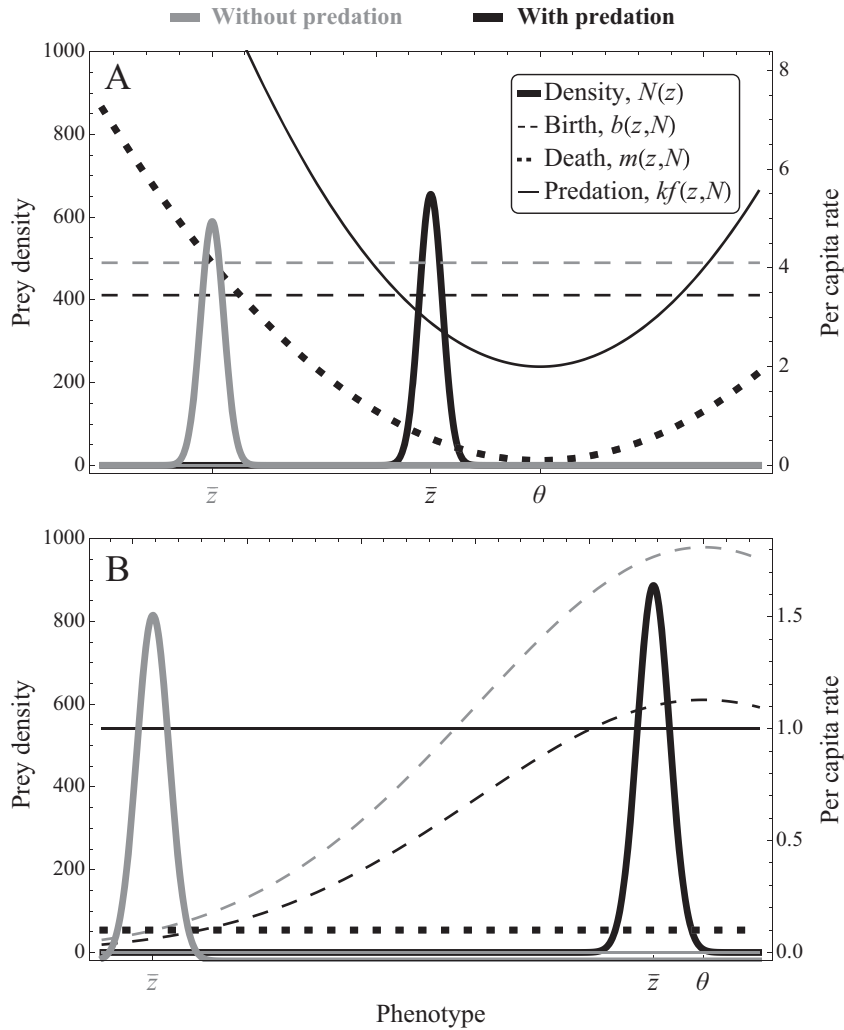


Figure 3: Graphical depiction of selective pressures in our two generalist predator examples with (black) and without (gray) predation. *A*, Selective push at the equilibrium shown in figure 2A–2C. *B*, Evolutionary hydra effect at the equilibrium shown in figure 4A–4C. *A*, Per capita birthrate (dashed line) is higher in the absence of predation (gray) because the population is further from the optimum and hence experiences more deaths (dotted line), freeing up resources that increase the birthrate.

also help prey persist when the predator is a specialist. In the main text, we present an example where a selective push arises from trait-matching coevolution. Coevolution is not necessary, however, and we show in the supplementary Mathematica file that even nonevolving specialists with a constant trait value, z_p , can also promote persistence in a manner similar to the generalist examples above (via a selective push and/or the evolutionary hydra effect). In all cases, the predator promotes prey persistence by increasing prey adaptedness.

To model coevolution with a specialist, we let the predation rate depend on both prey and predator trait values as well prey and predator densities. In the main text, we assume trait matching, such that a predator is best able

to catch prey with the same trait value as itself. This could be the case, for example, when z and z_p describe spatial locations or times of peak activity or, alternatively, predator trait values could be defined on this basis (i.e., the trait value of a predator is the trait value of prey they best consume). The mean predator trait value then evolves toward the mean prey trait value, while selection from predation drives the mean prey trait value away. Specifically, we assume the per prey rate of predation on prey with trait value z by predators with trait value z_p is $kf(z, z_p, P) = k \times \max[0, 1 - (\gamma_k/2)(z - z_p)^2]P$. At given densities, predation occurs at the maximum per prey per predator rate $k > 0$ when the trait values match. This rate falls off quadratically at rate $\gamma_k > 0$ as the trait values diverge from one an-

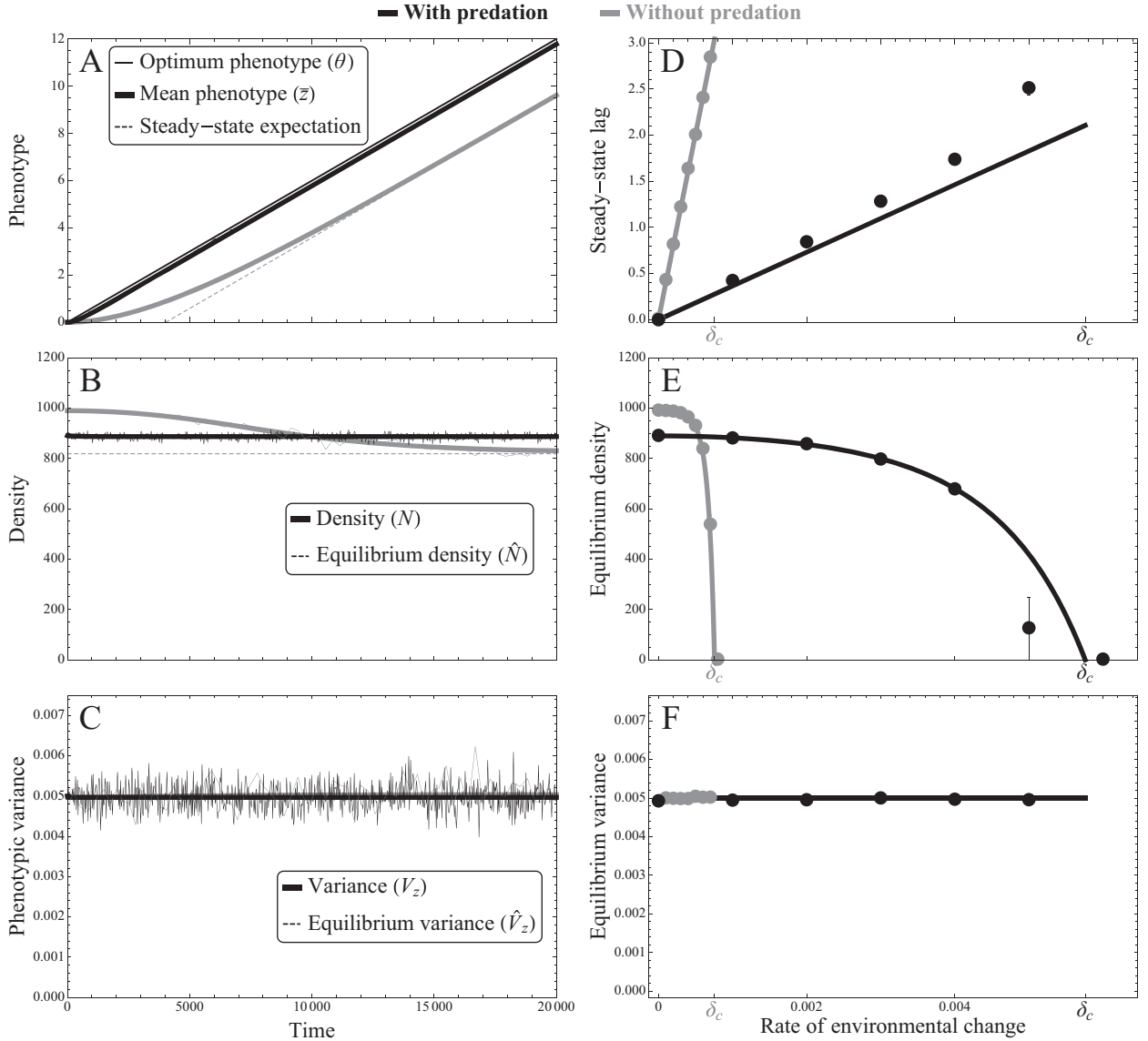


Figure 4: Evolutionary hydra effect. A–C, Temporal dynamics of mean prey phenotype, population density, and phenotypic variance when the optimum phenotype changes at rate $\delta = 0.0006$. Dashed curves are analytical solutions for equilibrium values (supplementary Mathematica file). Thick curves are numerical solutions (supplementary Mathematica file). Thin curves are results from one simulation (obscured by thick curves in A, B; appendix). D–F, Equilibrium dynamics of steady-state phenotypic lag, population density, and phenotypic variance as functions of the rate of environmental change. Curves give analytical results (supplementary Mathematica file). Dots give simulation results (mean ± 1.96 SE of 10 replicates; appendix). Parameters: $b_{\max} = 0.01$, $R_{\text{tot}} = 1,000$, $\gamma = 1$, $\alpha = 0.05$, $k = 0$, (gray), $k = 1$ (black).

other. In our analytic approximations, we assume that γ_k is small enough that $f(z, z_p, P)$ remains positive for the vast majority of prey and predators.

We assume predators are born at per capita rate $ekf(z, z_p, P)N$, where e describes how efficiently consumed prey are turned into new predators and die at constant per capita rate m_p . Thus the predator is not directly affected by the changing abiotic optimum, θ , but is indirectly influenced through its effect on the prey. Like the prey, predators are assumed to

randomly mate, with their offspring inheriting the mean trait value of the two parents plus a random normal segregation effect with mean 0 and variance α_p^2 . We assume predator trait values remain normally distributed with mean \bar{z}_p and variance $V_{z,p}$.

The prey's per capita birthrate is assumed to be $b(N, P) = b_{\max}(R_{\text{tot}} - N - P)$, where we subtract P from $R_{\text{tot}} - N$ to account for resources bound up in predators. The prey's per capita mortality rate is $m(z) = m_{\min} + (\gamma/2)(\theta - z)^2$, de-

scribing stabilizing selection around $z = \theta$. Prey growth rate in this model is therefore similar to what it was in the selective push example given above, except that the predation rate now depends on how close prey trait values are to predator trait values (instead of how close they are to θ) and on predator density. We provide analytical results for this model in the supplementary Mathematica file and here present a brief summary.

None of the prey's per capita rates depend on both trait value and prey density, and therefore there can be no evolutionary hydra effect (the first three terms in eq. [3] are zero). Instead we find, as expected, that direct selection from the coevolving specialist predator ($\partial \bar{f} / \partial \bar{z}$) can have dramatic effects on prey adaptation. At low to intermediate rates of environmental change, demographic and coevolutionary cycles occur (explaining the deviation from equilibrium in figure 5A and the large variance in the simulation results in figure 5D at low to intermediate δ ; evolutionary stability analysis in the supplementary Mathematica file). At fast rates of change, the cycles dissipate and the predator can push the prey mean trait value to evolve in advance of its changing optimum (fig. 5A–5C). Finally, at yet faster rates of change, the mean prey trait value falls back toward the optimum (fig. 5D). The decrease in the absolute value of prey lag at sufficiently fast rates of environmental change is caused by the selective push of predation (cf. gray to black in fig. 5D). This push maintains both prey and predator populations at rates of change that would cause prey extinction in the absence of the specialist predator (fig. 5E). Thus, when the specialist promotes prey persistence, both prey and predator must go extinct at the same rate of environmental change (since the prey cannot persist at sufficiently high rates of environmental change without the predator). Interestingly, in this example, extinction occurs not where the prey and predator densities simultaneously pass through zero but where they simultaneously become complex (a catastrophic saddle-node bifurcation; see supplementary Mathematica file for details). These results imply that small increases in the rate of environmental change can cause seemingly sustainable communities to suddenly go extinct.

As shown in the supplementary Mathematica file, even if specialist predators do not directly exert selection on the prey, they can also allow prey to persist at more extreme rates of environmental change via the evolutionary hydra effect (if they increase prey birthrates and if fitter prey individuals are more likely to give birth). Again, the prey population with predators persists at higher rates of environmental change than it could withstand without predators because of the increased rate of selective events (reduced generation time). When this occurs, both prey and predator go extinct at the exact same rate of environmental change.

Discussion

We have shown two ways that predators can help prey persist in a gradually changing environment. The first is termed the selective push and is the more intuitive phenomenon. Here predation acts to increase the strength of directional selection toward the optimum, and it thus speeds adaptation. Importantly, we have shown that this increase in the speed of adaptation can help prey persist in more extreme environments, despite the mortality imposed by predation. Similar ideas have been described to explain the potential effects of both predators (Jones 2008) and competitors (Osmond and de Mazancourt 2013) on a focal population's ability to adapt and persist in a changing environment. Surprisingly, the second way we find predators to help prey persist does not require the predator to exert a direct selective pressure on the prey. Instead, decreases in prey density feedback to increase rates of prey evolution. We term the phenomenon of increased prey mortality increasing rates of prey evolution the evolutionary hydra effect and demonstrate with specific examples that this can indeed lead to increased prey persistence. While our study has been motivated by recent empirical work on predation (Tseng and O'Connor 2015), the selective push and evolutionary hydra effect can equally arise from other sources of mortality, for example, from parasitism, competition, additional environmental stressors, or potentially even cannibalism. In particular, equations (3) and (5) continue to apply to other sources of mortality whose strength is also proportional to k .

Selective Push

A selective push acts much like an increase in the strength of selection (e.g., total strength of selection in the example in the main text equals $\gamma + k\gamma_k$). Previous models of sexually reproducing populations evolving alone in continuously changing environments have also shown that increasing the strength of selection can help persistence. For example, Lynch and Lande (1993) and Bürger and Lynch (1995, 1997) found the critical rate of environmental change to be maximized at intermediate strengths of selection, as we find here (at intermediate rates of predation, k). In contrast, more recent treatments (Jones 2008; Mellard et al. 2015) have found intermediate strengths of selection to minimize prey persistence. In the case of Mellard et al. (2015), the discrepancy is the result of differing model assumptions. They assume the prey is monomorphic (except for the occasional appearance and loss or fixation of mutations), and hence there is no decline in mean prey growth rate due to trait variance around the mean (genetic load; Lande and Shannon 1996), which decreases the cost of strong selection. They also assume that the rate of evolution is proportional to the population-scaled mutation rate, $N\mu$, instead of additive genetic variance.

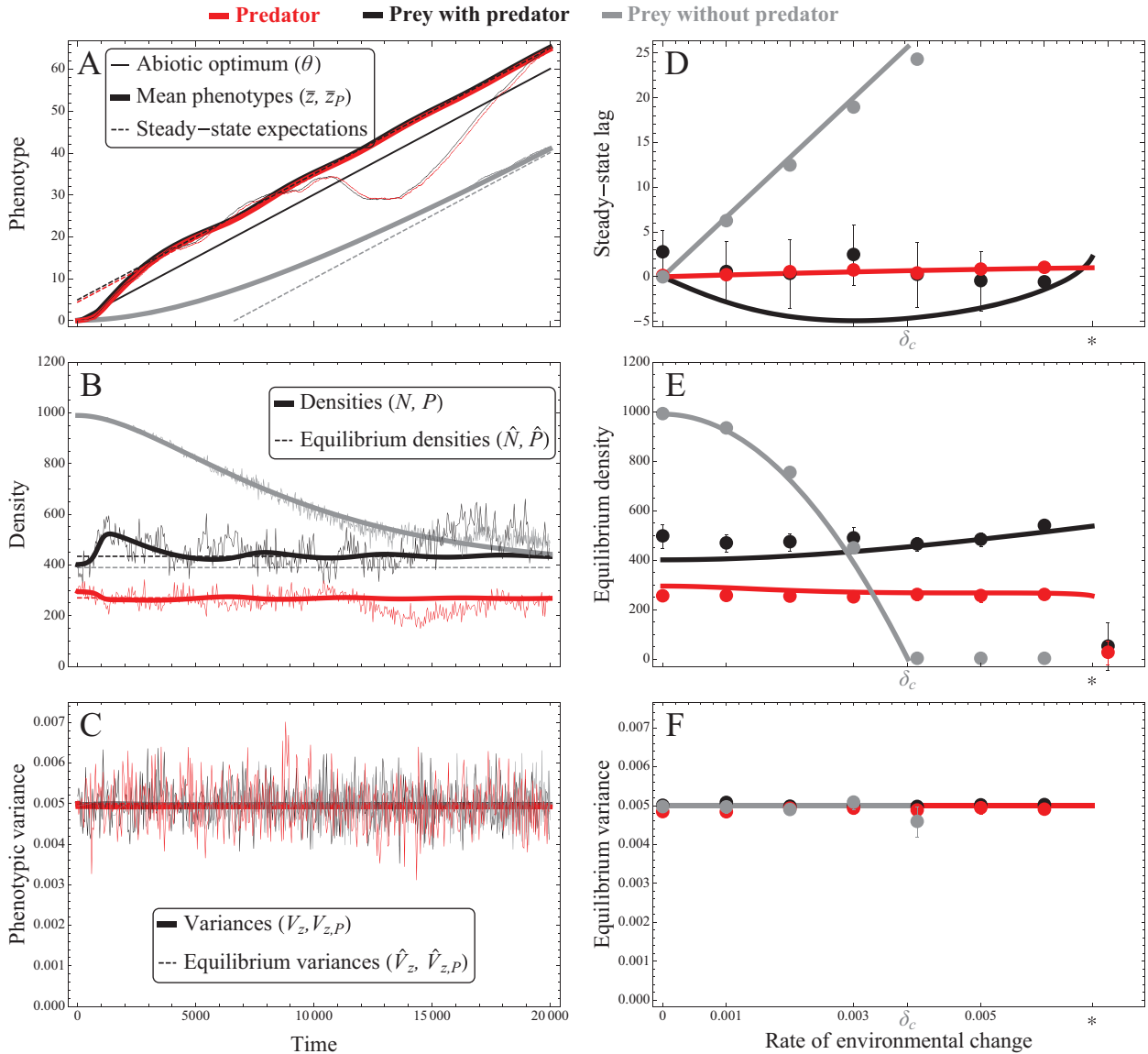


Figure 5: Coevolution between the prey (black) and a specialist predator (red). A–C, Temporal dynamics of mean prey and predator phenotypes, population densities, and phenotypic variances when the optimum phenotype in the absence of predators increases at rate $\delta = 0.003$. Dashed curves are analytical solutions for equilibrium values (supplementary Mathematica file). Thick curves are numerical solutions (supplementary Mathematica file). Thin curves are results from one simulation (gray obscured by thick curve in A; appendix). D–F, Equilibrium dynamics of steady-state phenotypic lags, population densities, and phenotypic variances as functions of the rate of environmental change. Curves give analytical results (supplementary Mathematica file). Dots give simulation results (mean \pm 1.96 SE of 10 replicates; appendix). Asterisks denote the rate of environmental change where coevolving prey and predator equilibrium densities simultaneously become complex values. Parameters: $b = 0.01$, $m_{\min} = 0.1$, $R_{\text{tot}} = 1,000$, $\gamma = 0.03$, $\gamma_k = 0.5$, $e = 1$, $m_p = 4$, $\alpha = 0.05$, $\alpha_p = 0.05$, $k = 0$ (gray), $k = 0.01$ (black and red).

Since weakening selection increases equilibrium population sizes, N , it also increases the response to selection (in contrast, in our model, the additive genetic variance reaches an equilibrium that is independent of selection or N). Together, these assumptions allow prey persistence to be maximized at the extremes of selection. The discrepancy with the simulations of Jones (2008) is due to the range of selection strengths he

examined. The parameter values in Jones (2008) were borrowed from Bürger and Lynch (1995). The latter went on to show, however, that persistence is often maximized at intermediate values when examining a wider range of selection strengths. Thus, our results are consistent with previous quantitative genetic models while demonstrating that predation can be the cause of the increased selection strength.

Evolutionary Hydra Effect

The evolutionary hydra effect—increasing mortality causing faster evolution—is related to previous ideas in the literature. For example, Zeineddine and Jansen (2009) have shown how increasing adult mortality can theoretically increase rates of evolution and therefore explain why an annual life history may be favored over a perennial one in changing environments. Similarly, Kuparinen et al. (2010, and references within) discuss how adaptation to longer growing seasons is hampered by low adult mortality in long-lived tree species and use simulations to show that increasing mortality enhances adaptation. Our study extends this previous work by including the possibility that predators could be the cause of increased mortality and, more importantly, by analyzing when the increase in adaptedness brought about by the evolutionary hydra effect outweighs the mortality cost and helps populations persist.

Increased mortality has been shown to increase persistence in models of evolutionary rescue following an abrupt environmental change. In these models, mortality facilitates persistence by decreasing competition (Uecker et al. 2014) and maladaptive epistasis (Uecker and Hermisson 2016). This increases the probability of adaptation by increasing the probability of establishment of beneficial genotypes. These population genetic models assume nonoverlapping generations with fixed generation times, where mortality affects adaptation by altering survival probabilities. The evolutionary hydra effect describes another way for mortality to increase rates of evolution and aid persistence: by decreasing generation times.

Generation times can be reduced in populations with overlapping generations (including, e.g., univoltine insects with resting stages and annual plants with seed banks) and in populations with nonoverlapping generations where the time between generations can diminish (e.g., biennial plants becoming annuals). However, the evolutionary hydra effect cannot act in populations with nonoverlapping generations where the time between breeding seasons is fixed (e.g., in univoltine insects and annual plants with no resting stages or seed banks). Nonselective death could then still help prey persist but not by reducing generation time and only under arguably more complex scenarios. For example, if decreased population density disproportionately increased the birthrate of well-adapted individuals, then increased random mortality could accelerate evolution and aid persistence.

Specialist Predators and Coevolution

We have found that specialist predators, like generalists, can help prey adapt and persist. The main difference between specialist and generalist predators is the presence

or absence of demographic feedbacks (as well as evolutionary feedbacks in the case of a coevolving specialist), where declining prey numbers sustain fewer specialist predators and thus reduce the rate of deaths from predation. These demographic feedbacks are a double-edged sword. At low predation intensities, the specialist predator becomes rare and loses its ability to help the prey adapt at high rates of environmental change. In this case, a generalist (i.e., with constant predator density) could nevertheless help the prey adapt and persist. On the other hand, at high predation intensities, prey can persist in the presence of a specialist predator at higher rates of environmental change than they can with a generalist, because the specialist becomes rare as the prey becomes less well adapted and does not induce too high a mortality cost. As a consequence, whether a specialist or a generalist predator better allows prey to persist in rapidly changing environments depends on the exact model conditions and particularly on the intensity of predation (fig. 6).

There are at least three other theoretical studies suggesting that coevolution with specialist predators can, at least temporarily, facilitate prey persistence in changing environments. The earliest of these is Jones (2008), who uses simulations to show that coevolution between a specialist predator and its prey can often increase the prey population's persistence time, given that all finite populations have some chance of going extinct each generation (Bürger and Lynch 1995). While the model in Jones (2008) differs from ours in a number of ways (e.g., nonoverlapping generations, density-independent predation), both studies show that predator-prey coevolution can help prey persist when they otherwise would not. Here we analytically clarify why coevolution can help: a selective push from coevolution can reduce prey lags enough to allow prey growth at rates of environmental change that would otherwise cause extinction.

In another study, Northfield and Ives (2013) show that coevolution between a specialist predator and its prey can reduce the effect of small environmental changes on equilibrium densities of both prey and predator through negative feedbacks. We also observe negative feedbacks in our coevolutionary specialist predator model. This is particularly apparent in a constant environment, where the ecoevolutionary system, as parameterized, is predicted to cycle. Here, the mean prey trait departs from the abiotic optimum, decreasing prey growth rate, which decreases predator density. As predator density declines, the predator's selective pressure on the prey dissipates, allowing the prey to evolve back toward the abiotic optimum, increasing prey growth rates and, hence, predator density. Despite the similar feedback, direct comparison with Northfield and Ives (2013) is difficult since they model one-step changes in the environment, allowing them to compare models with and without evolution. With a continuous environmental change, the absence of evolution at any nonzero rate of en-

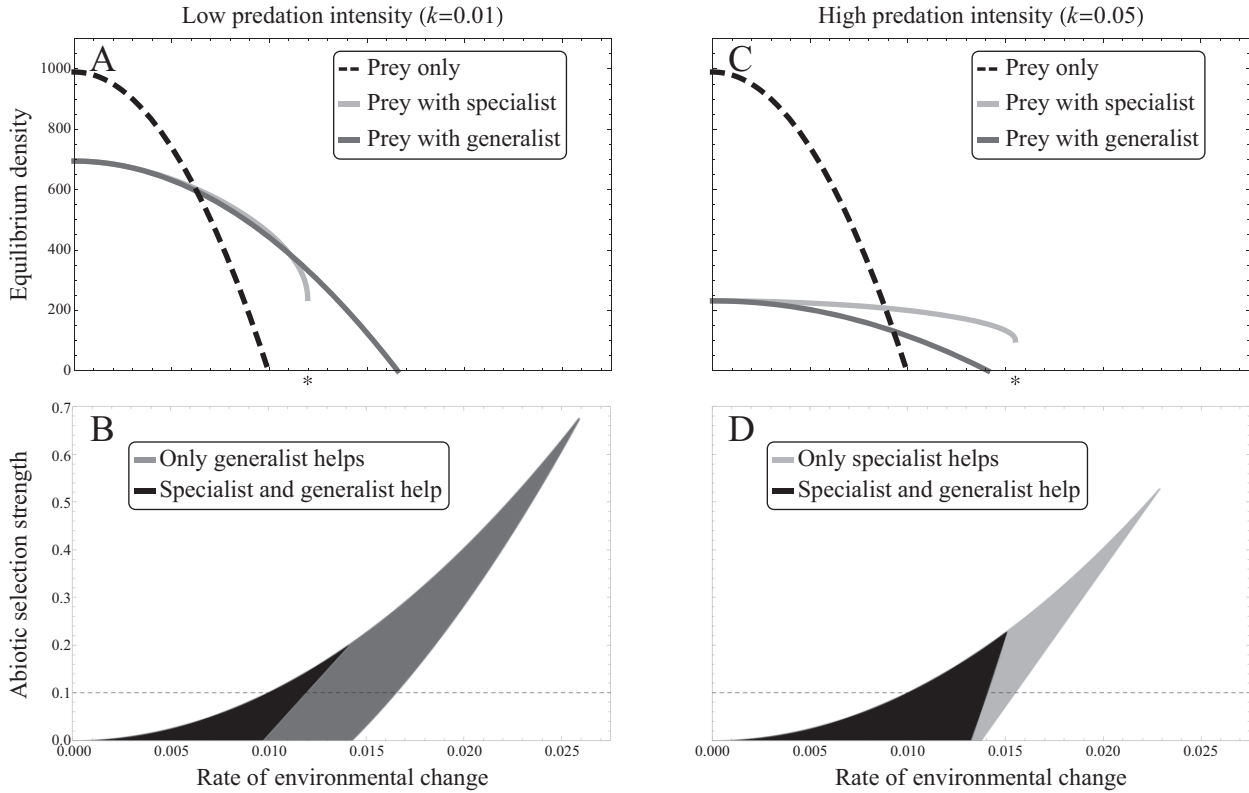


Figure 6: Prey persistence with a specialist versus a generalist predator. Shown here is the case of a noncoevolving specialist predator that causes a selective push by preferentially preying maladapted prey (replacing $f(z, z_p, P)$ in a coevolving case with $f(z, P) = k[1 + \gamma_k(\theta - z)^2]P$; see supplementary Mathematica file for details and other cases). *A, B*, With low predation intensity, the specialist predator becomes rare and does not sufficiently push the prey at higher rates of environmental change. *C, D*, With high predation intensity, the generalist predator exerts too high a demographic cost, while the specialist becomes rare when the prey are rare and can help the prey persist. *A, C*, Equilibrium prey densities across rates of environmental change with abiotic selection strength $\gamma = 0.1$. *B, D*, Parameter regions where predators help prey persist (dashed line corresponds to the γ used in *A, C*). Asterisks indicate the rate of environmental change where the specialist predator and prey equilibrium population sizes simultaneously become complex values. Parameter values: $b_{\max} = 0.01$, $m_{\min} = 0.01$, $R_{\text{tot}} = 1,000$, $e = 0.25$, $m_p = 1$, $\gamma_k = 0.1$, $\alpha = 0.05$.

environmental change would cause the mean prey trait lag to grow indefinitely, bringing both populations to extinction. We instead compare models with and without a specialist predator (fig. 5), a comparison Northfield and Ives (2013) do not make, allowing us to ask whether the presence of a predator (not evolution) helps the prey adapt and persist.

In a third theoretical study, Mellard et al. (2015) find that a specialist predator can increase prey persistence times under strict conditions. The strict condition is that coevolution causes the mean prey trait to evolve in the direction of the environmental change before the environment begins to change, giving the prey a head start. This transient effect, not observed in our steady-state results, does not affect the critical rate of environmental change and hence the ability of large populations to persist indefinitely in changing environments. That is, Mellard et al. (2015) show that a selective push from the predator can

increase prey persistence time but not whether it can increase long-term prey persistence. We extend this result by showing that the selective push from predator-prey coevolution can also affect long-term prey persistence.

Finally, there is one additional piece of theory that, despite assuming a constant environment and no evolution, aligns surprisingly closely with our results. Holt and Barfield (2009) discuss how predation affects the geographic range limits of prey when assuming that prey become increasingly maladapted away from the center of their range. This situation turns out to be roughly analogous to ours when one replaces time with space. Despite the fact that prey traits are fixed in their models (i.e., there is no evolution), Holt and Barfield (2009) reach many of the same conclusions. First, they find that generalist predators can expand prey ranges when mortality increases prey density (the nonevolutionary hydra effect per Abrams 2009, a sce-

nario we do not consider). Second, they find that specialist predators make demographic stability more likely nearer prey range edges (here nearer the critical rate of environmental change; stability analysis in supplementary Mathematics file) and maximize prey density away from its range center (here away from a constant environment; fig. 5E). Finally, Holt and Barfield (2009) find that specialist predators are less likely to determine prey range edges than generalist predators but may do so under special circumstances. One of these circumstances is sufficient predator dispersal, which, when replacing space with time, uncouples predator survival from local prey densities (acting more like our generalist model). Another special circumstance is when the specialist predator causes prey to flee and colonize new patches in a metapopulation, expanding the prey's range, which is roughly analogous to causing the prey to evolve faster in our model. Combining the two models to look at the effect of predators on the evolution of range edges could produce interesting predictions.

Empirical Work

Few empirical studies examine the effect of predators on adaptation to environmental change. A notable exception is Tseng and O'Connor (2015), who raised *Daphnia pulex* at a range of temperatures with or without predatory *Chaoborus* larvae. They found that *Daphnia* populations reared with predators evolved smaller body sizes than those populations without, potentially increasing the adaptedness of this ectotherm in warmer waters (Atkinson and Sibly 1997). Consistent with this adaptive hypothesis is the fact that, in warmer waters, *Daphnia* had higher intrinsic growth rates with predators than without. However, *Daphnia* population sizes tended to be smaller with predators than without, at all temperatures, and at the highest temperature assayed, *Daphnia* populations with predators went extinct before those without, suggesting that *Chaoborus* hinders *Daphnia* persistence. Interpreted in the light of our analysis, the benefits of greater adaptation do not outweigh the demographic costs (eq. [3] is satisfied, but eq. [5] is not).

More experiments in this area are needed to see if the potential selective benefits of predation ever outweigh its demographic costs. It would be especially interesting to examine the dynamics of prey near their extinction threshold (δ_c) and see how this threshold is affected by the presence of predators. This could be done by adding a predator treatment in experiments with gradual environmental change (e.g., Willi and Hoffmann 2009) or, perhaps more easily, by adding a predator treatment in evolutionary rescue experiments with a single abrupt environmental change (e.g., Bell and Gonzalez 2009). Alternatively, predation could be imposed by the experimenter themselves. For example, a selective push would result from the occasional re-

moval of individuals that are maladapted. Or, in a system where intraspecific competition is reducing prey birthrate and better-adapted prey produce more offspring, the evolutionary hydra effect could be induced by diluting the population at a higher rate.

Conclusion

We are in need of theory that explains and predicts how communities respond to environmental change (Low-Décarie et al. 2015). With communities comes species interactions, which affect how—and if—populations within communities adapt (Lavergne et al. 2010). Trophic interactions are ubiquitous, and it has been shown, both theoretically (Jones 2008; Northfield and Ives 2013; Mellard et al. 2015) and empirically (Tseng and O'Connor 2015), that predators can help prey adapt. However, whether predators can ever help prey persist has remained unclear. Here we have theoretically demonstrated that predators can indeed help prey persist, despite the mortality cost that predation imposes, and have highlighted two ways that this can occur. Predators can help prey adapt and persist through a selective push or through the newly defined evolutionary hydra effect whereby increasing prey death rates accelerate prey evolution by decreasing generation times. We have thus shown that predators can catalyze adaptation and allow prey to persist in more extreme environments, but it remains an open question how often this occurs in nature.

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APPENDIX

Simulation Methods

Individual-based simulations followed a Gillespie algorithm (Gillespie 1977). Populations were represented by a vector of trait values (one entry for each individual), which was initiated by choosing n entries (corresponding to demographic equilibrium in the absence of selection) from a random normal distribution with mean 0 and variance $2\alpha^2$ (the predicted equilibrium phenotypic variance).

For each iteration, (1) the propensities for each type of reaction (birth, death, predation) were calculated for each individual; (2) the propensities were summed to give the total propensity, a_0 ; (3) time was increased by $\tau = -\log(r_1)/a_0$, where r_1 is a random uniform number in $[0, 1)$; (4) a particular reaction was chosen by giving a reaction with propensity a_i a probability of occurrence a_i/a_0 and choosing a random uniform number in $[0, 1)$; and (5) the reaction occurred, and abundances were updated accordingly. To speed up the coevolutionary simulations, we made the propensity for a prey to be predated a function of the difference between its trait value and the mean predator trait value (instead of a separate value for each predator individual) and the propensity for a predator to capture a prey a function of the difference between its trait value and the mean prey trait value (instead of a separate value for each prey individual). When an individual was chosen to reproduce, a second individual of the same population was chosen randomly as the mate (possibly the same individual), and the offspring inherited the average of the two parental trait values plus a random normal effect with mean 0 and variance α^2 .

The abundances and the mean and variance in the trait distributions were recorded every 10^4 iterations (events). Simulations ended when the prey went extinct or the final time was reached, $t \geq 10^4$ (or $t \geq 2 \times 10^4$ in the evolutionary hydra example). Means of the final 10 recorded values were used to estimate steady-state lags, equilibrium population densities, and equilibrium genetic variances across replicates.

All simulations were written in Python (ver. 2.7.10; <http://www.python.org/>) and run on WestGrid's Orcinus cluster. Example scripts are available as supplementary material.

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