

A mathematical analysis of evolutionary rescue and niche construction

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Abstract

Laboratory experiments and field studies have shown that declining populations can avoid extinction by undergoing adaptation. This process, evolutionary rescue, entails a population approaching extinction until an adaptive mutation appears and subsequently establishes by escaping stochastic loss. While most models of evolutionary rescue emphasize mutations that allow organisms to persist in hostile environments, a less explored possibility—known as positive niche construction (hereafter niche construction)—involves mutants improving their fitness by modifying their environment. In Chapter 1 of this dissertation, I analyze a model of evolutionary rescue via a niche-constructing mutation. I show that the probability of rescue is highest under low-to-moderate rates of construction: some construction is needed to ensure that mutants proliferate quickly enough to avoid stochastic extinction; but because construction is costly, requiring time and energy to perform and develop, too much of it can lead to over-exploitation of the constructed habitats by the mutants' non-niche-constructing ancestors (hereafter residents). In Chapter 2, I then model a niche-constructing population that must undergo evolutionary rescue to withstand habitat exploitation by an invading species. I find that the same fecundity costs rendering constructors vulnerable to exploitation can help facilitate rescue from such exploitation by reducing the rate of construction and thus lowering the density of habitats available to invaders. The lower habitat density leads to slower invasion, which in turn buys constructors more time to mutate. Finally, in Chapter 3, I consider the possibility that invaders directly interact with the resident population instead of exploiting resident-constructed habitats. I show that a lower resident birth rate—whether it stems from a smaller resident density independent birth rate or stronger birth-limiting competition—can promote rescue by reducing variance in mutant fitness. Together, these findings suggest that lower reproductive success among members of a population can, under a range of conditions, improve the population's chances of evolutionary rescue.

Lay Summary

Understanding when a population is most likely to avoid extinction through adaptation, a process known as evolutionary rescue, can help address critical challenges such as eliminating antibiotic-resistant pathogens and preserving endangered species. Evolutionary rescue occurs when a population is on the verge of collapse but survives after a beneficial genetic change (hereafter mutation) spreads through the population. While studies of evolutionary rescue often focus on mutations allowing organisms to survive and reproduce in harmful habitats, an alternative possibility is that the mutations permit organisms to make their habitats more hospitable through what is known as niche construction. If a population adopts this strategy, however, it risks habitat exploitation by organisms that benefit from the constructed habitats but do not expend the time and energy needed to perform construction. In this dissertation, I first model evolutionary rescue via a niche-constructing mutation. Then I consider a niche-constructing population that must evolve to withstand habitat exploitation by an invading species. Finally, I consider the possibility that the invading species interacts directly with its victims instead of exploiting their habitats. Counterintuitively, my results suggest that lower reproductive success among members of a declining population can aid in the evolutionary rescue of that population.

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Chapter 1

Evolutionary rescue via niche construction

Abstract

A population experiencing habitat loss can avoid extinction by undergoing genetic adaptation—a process known as evolutionary rescue. Here we analytically approximate the probability of evolutionary rescue via a niche-constructing mutation that allows carriers to convert a novel, unfavorable reproductive habitat to a favorable state at a cost to their fecundity. We analyze competition between mutants and non-niche-constructing wild types, who ultimately require the constructed habitats to reproduce. We find that over-exploitation of the constructed habitats by wild types can generate damped oscillations in population size shortly after mutant invasion, thereby decreasing the probability of rescue. Such post-invasion extinction is less probable when construction is infrequent, habitat loss is common, the reproductive environment is large, or the population's carrying capacity is small. Under these conditions, wild types are less likely to encounter the constructed habitats and, consequently, mutants are more likely to fix. These results suggest that, without a mechanism that deters wild type inheritance of the constructed habitats, a population undergoing rescue via niche construction may remain prone to short-timescale extinction despite successful mutant invasion.

1 Introduction

Anthropogenic habitat destruction is among the greatest threats to global biodiversity (Segan et al. 2016). Habitat loss can prevent members of a population from reproducing, which can in turn cause an otherwise well-adapted population to decline toward extinction. Recent studies have attributed such a loss of breeding sites, and the subsequent decline in population size, to processes like competition with invasive species (Hernández-Brito et al. 2018), climate change (Mastrantonis et al. 2019), and deforestation (Walter et al. 2018). One way a population can avoid extinction due to habitat degradation is by undergoing genetic adaptation to the novel habitat—a process known as evolutionary rescue (Gomulkiewicz and Holt 1995). This process entails a population declining toward extinction until an adaptive mutation appears and establishes in the population, resulting in a U-shaped demographic time series (Orr and Unckless 2014). When habitat degradation occurs on ecological timescales, so too must the evolutionary change leading to rescue (Kirkpatrick and Peischl 2013). Evidence of such rapid evolution has been provided by laboratory experiments (Bell and Gonzalez 2009; Batarseh et al. 2020), as well as recent fieldwork (Clayton and Spicer 2020).

Adaptation is often the result of mutations that change an organism's traits to fit the environment. However, adaptation can also occur via mutations that permit positive niche construction (hereafter, niche construction), whereby the environment is modified to better match the organism (Laland et al. 1999; Odling-Smee et al. 2013). While niche-constructing behaviors are often the product of suites of traits that produce complex structures like nests (Sauce et al. 2012), webs (Correa-Garhwal et al. 2018), and fungus gardens (Grell et al. 2013), such behaviors can also have simple genetic underpinnings—like those responsible for the secretion of beneficial compounds by microbes. In budding yeast, for example, the secretion of the enzyme, invertase, is controlled by the regulation of a single locus (Carlson and Botstein 1982; Perlman et al. 1986). The invertase molecules metabolize sucrose from the environment, converting the sucrose into digestible glucose and fructose for the yeast. Likewise, a single mutation can convert a strain of the iron-dependent bacterium, *Pseudomonas aeruginosa*, which produces iron-scavenging siderophore molecules, into a non-producing cheater that exploits the siderophores (Ross-Gillespie et al. 2007).

Standard models of evolutionary rescue can capture some of the short-term fitness effects of niche construction, but they do not typically account for the possibility that constructed environments can persist long after the constructors of those environments have died or dispersed elsewhere (Uecker et al. 2014; Tomasini and Peischl 2020; Czuppon et al. 2021). Such persistence of constructed environments can lead to

ecological inheritance, in which individuals inherit environments that were constructed by other individuals in the past (Odling-Smee et al. 2013). Maturing hermit crabs, for instance, settle into vacant shells that were modified and outgrown by previous residents (Laidre 2012). Laboratory populations of *P. aeruginosa* have been found to reuse siderophores across multiple generations (Kümmerli and Brown 2010). Similarly, in laboratory populations of budding yeast, the glucose resulting from invertase production often accumulates in the growth media, permitting exploitation of the glucose by cheaters (Celiker and Gore 2012).

Here we study the effect of niche construction and ecological inheritance on evolutionary rescue. We model an environment whose reproductively suitable habitats are decaying to an unusable state, causing the wild type population to approach extinction. Similar to Gurney and Lawton's (1996) model of ecosystem engineering, niche construction is modeled as a habitat conversion process: mutants can convert an unfavorable novel habitat back to the favorable initial state. We incorporate ecological inheritance by allowing the constructed habitats to remain in the environment—available for use by anyone who randomly encounters them—until they decay to the unusable state. Following models of cooperation (Lehmann 2007, 2008; Henriques and Osmond 2020), we treat the constructed habitats as public goods that are costly to produce and can, therefore, be exploited by non-niche-constructing wild types. In contrast to quantitative genetic models of evolutionary rescue (Bürger and Lynch 1995; Ashander et al. 2016; Osmond et al. 2017), which are often used to identify conditions under which several small-effect loci can collectively track a gradually changing phenotypic optimum, we take a common population genetic approach (Uecker and Hermisson 2011; Wilson et al. 2017; Marrec and Bitbol 2020): we approximate the probability that a *de novo* niche-constructing mutation rescues the population.

Our analysis is structured as follows: we first introduce a stochastic, ecological model of niche construction and analyze the model's expected (i.e., average) dynamics. We find that the model is expected to exhibit two non-trivial equilibria: a population that is monomorphic for mutants and an oscillatory polymorphism. We then adapt the ecological model for evolutionary rescue and approximate quantities that characterize mutant and wild type dynamics during the rescue process. These quantities are subsequently used to approximate the probability of rescue, which includes the probability that the population survives the oscillations occurring shortly after mutant invasion. To understand how such post-invasion dynamics affect a single lineage's survival probability, we model a hard selective sweep (Hermisson and Pennings 2017; Wilson et al. 2017). Analytical results are prioritized to develop general intuition. We find that a phenotype that performs niche construction too often can facilitate wild type exploitation of the constructed habitats, thereby amplifying the oscillations and reducing the probability of mutant survival. This

reduction can be prevented by less frequent construction, more rapid habitat loss, a larger reproductive environment, or a smaller population carrying capacity. All else equal, such changes promote mutant fixation by lowering the frequency at which wild types inherit the constructed habitats.

2 Model

2.1 Ecological model

We model a continuous time birth-death process (Kendall 1948). The reproductive environment is composed of $H_{Max} > 0$ patches. Each patch consists of either habitat type 1 or habitat type 2. A single patch of habitat type 1 permits reproduction, while a patch of habitat type 2 does not. The abundance (i.e., number) of habitat type 1 patches is $H(t)$ and decays at a constant rate of $a > 0$, modeling degradation of the suitable habitat. We model haploid organisms whose adults reside in a single, well-mixed population with a global carrying capacity of $K > 0$. This population contains wild type and mutant organisms, which have abundances of $N_W(t)$ and $N_M(t)$, respectively. Both types have a per-capita death rate of $\mu(t) = \frac{N(t)}{K}$, where $N(t) = N_M(t) + N_W(t)$.

A birth event entails an adult dispersing to a randomly chosen patch and giving birth, followed by the adult and its offspring entering the population of adults. Because organisms do not reside in these reproductive patches for any length of time, there is no competition for patches. Mutants differ from wild types because, with a probability of $0 < g \leq 1$, they can perform niche construction—a process in which habitat type 2 is converted to habitat type 1—which allows mutants to reproduce despite dispersing to habitat type 2. Developing the ability to niche construct entails a fecundity cost of $0 < cg < 1$, defined herein as a reduction in the rate at which mutants disperse to a reproductive patch. This means that, similar to existing models (Krakauer et al. 2009; Chisholm et al. 2018), investing more energy into niche construction (i.e., higher g) increases one’s probability of successfully niche constructing but also decreases one’s rate of reproduction. Since the cost of niche construction is developmental, mutants pay the cost regardless of which habitat they disperse to. The per-capita rate at which mutants disperse to habitat type 1 and reproduce is then $\lambda_{M,1}(t) = (1 - cg) \frac{H(t)}{H_{Max}}$. And the per-capita rate at which mutants niche construct, and subsequently reproduce in what would otherwise be a patch of habitat type 2, is $\lambda_{M,2}(t) = (1 - cg) \left(1 - \frac{H(t)}{H_{Max}}\right) g$. This gives a total per-capita mutant birth rate of $\lambda_M(t) = \lambda_{M,1}(t) + \lambda_{M,2}(t)$. Since wild types can only reproduce if they disperse to habitat type 1, their per-capita birth rate is $\lambda_W(t) = \frac{H(t)}{H_{Max}}$. This model’s parameters are summarized in Table 1 and the model’s expected dynamics are described by

$$\begin{aligned}
\frac{d\bar{H}(t)}{dt} &= \bar{N}_M(t)\bar{\lambda}_{M,2}(t) - \bar{H}(t)a \\
\frac{d\bar{N}_M(t)}{dt} &= \bar{N}_M(t)\bar{\lambda}_M(t) - \bar{N}_M(t)\bar{\mu}(t) \\
\frac{d\bar{N}_W(t)}{dt} &= \bar{N}_W(t)\bar{\lambda}_W(t) - \bar{N}_W(t)\bar{\mu}(t),
\end{aligned} \tag{1a-c}$$

where $\bar{V}(t)$ is the expected value of variable $V(t)$ (Czuppon and Traulsen 2021), as opposed to the realized value. Setting Equations 1a-c equal to zero and solving for the three variables reveals that the equations exhibit two non-trivial, biologically relevant equilibria: a population that is monomorphic, containing only mutants, and a polymorphism. The monomorphism can be written as

$$\begin{aligned}
\bar{H}_{Mono} &= \frac{2g^2(1-cg)^2 H_{Max} K}{aH_{Max} + g(2g-1)(1-cg)^2 K + \zeta} \\
\bar{N}_{M,Mono} &= \frac{g(1-cg)^2 K - aH_{Max} + \zeta}{2g(1-cg)} \\
\zeta &= \sqrt{2ag(2g-1)(1-cg)^2 H_{Max} K + g^2(1-cg)^4 K^2 + (aH_{Max})^2} \\
\bar{N}_{W,Mono} &= 0.
\end{aligned} \tag{2a-c}$$

Equations 2a-b are positive throughout parameter space. For $(\bar{H}(t), \bar{N}_M(t), \bar{N}_W(t)) = (\bar{H}_{Mono}, \bar{N}_{M,Mono}, 0)$, the monomorphism is locally stable (i.e., satisfies the Routh-Hurwitz criterion; Otto and Day 2007) only if $K \leq K^* = \frac{aH_{Max}(1+c-cg)}{cg(1-cg)}$; whereas, if the system is reduced to two variables, $(\bar{H}(t), \bar{N}_M(t)) = (\bar{H}_{Mono}, \bar{N}_{M,Mono})$, the monomorphism is always locally stable. Thus, following a perturbation, the system tends toward the monomorphism if $K \leq K^* = \frac{aH_{Max}(1+c-cg)}{cg(1-cg)}$ or wild types are extinct. Otherwise, the system tends toward the polymorphism, which can be written as

$$\begin{aligned}
\bar{H}_{Poly} &= \frac{H_{Max}(1-cg)}{1+c-cg} \\
\bar{N}_{M,Poly} &= \frac{aH_{Max}}{cg} \\
\bar{N}_{W,Poly} &= \frac{K(1-cg)}{1+c-cg} - \frac{aH_{Max}}{cg} \\
\bar{N}_{Poly} &= \bar{N}_{M,Poly} + \bar{N}_{W,Poly}.
\end{aligned} \tag{3a-d}$$

The polymorphic abundance of mutants ($\bar{N}_{M,Poly}$) is always positive, whereas that of wild types ($\bar{N}_{W,Poly}$) is only positive when $K > K^*$. Moreover, when $K > K^*$, the polymorphism is locally stable and $\bar{N}_{M,Mono} > \bar{N}_{M,Poly}$. Analysis of the characteristic polynomial in its limit as K approaches infinity indicates that the polymorphism can exhibit oscillations (i.e., the polynomial has a negative discriminant

and therefore two complex conjugate eigenvalues) if and only if $g > f_1(a, c)$ and either $f_2(a) < c < \frac{1}{4}$ or $c \geq \frac{1}{4}$, where $f_1(a, c)$ and $f_2(a)$ are analytically unwieldy functions. These oscillations are damped since the polymorphism is stable when positive. Linearizing $f_1(a, c)$ and $f_2(a)$ around $a = 0$ reduces the oscillation conditions to $g \gtrsim \frac{a}{4c^2}$, meaning that, when K is large and a is small, oscillations can occur only if g is greater than

$$G \approx \frac{a}{4c^2}. \quad (4)$$

Table 1: Model parameters and variables.

Notation	Interpretation
a	Rate of habitat loss
c	Maximum fecundity cost of niche construction
g	Probability of niche construction
G	Value of g above which oscillations can occur
H_{Max}	Total number of habitats
K	Carrying capacity
u	Per-capita mutation rate
U	Expected cumulative mutation rate
$H(t)$	Abundance of habitat type 1
$N_M(t)$	Abundance of mutants
$N_W(t)$	Abundance of wild types
$N(t)$	Total population size
$\lambda_j(t)$	Per-capita birth rate of type $j \in \{M, W\}$ organisms
$\mu(t)$	Per-capita death rate
$\bar{V}_{Pre}(t)$	Expected pre-invasion abundance of variable $V(t)$
\bar{V}_{Mono}	Monomorphic equilibrium abundance of variable $V(t)$
\bar{V}_{Poly}	Polymorphic equilibrium abundance of variable $V(t)$
$\bar{N}_{j,Post}$	Expected post-invasion minimum abundance of type j
t_0	Mutant appearance time
t_j^+	Earliest time at which type j can have a positive expected growth rate
t_{Inv}	Time at which invasion step of rescue completes
t_{Est}	Time at which mutants establish in the population

2.2 Evolutionary rescue

In this section, we adapt our ecological model for evolutionary rescue and, while doing so, use Equations 1a-c to approximate quantities that will later be used to approximate the probability of rescue. We model rescue as a three-step process: the first step is the pre-invasion step (*Pre*), whereby the population declines toward extinction in the absence of mutants. The second step is the invasion step (*Inv*), which entails a mutant lineage appearing and subsequently invading the population by overcoming initial rarity. The third step is the post-invasion step (*Post*), whereby the population persists throughout a finite time interval following mutant invasion. Figure 1 illustrates the expected dynamics of our rescue model.

2.2.1 Pre-invasion step

For times $t < 0$, we assume that habitat type 1 and wild types are at carrying capacity due to a constant environment (i.e., $a = 0$), while mutants are absent from the population. At time $t = 0$, habitat loss begins (i.e., $a > 0$), causing the population to decline toward extinction from $H(0) = \bar{H}(0) = H_{Max}$, $N_M(0) = \bar{N}_M(0) = 0$, and $N_W(0) = \bar{N}_W(0) = K$. The expected dynamics of the population's decline are described by the solution to Equations 1a-c:

$$\begin{aligned}\bar{H}_{Pre}(t) &= H_{Max}e^{-at} \\ \bar{N}_{W,Pre}(t) &= \frac{aK \exp\left(\frac{1}{a} - \frac{e^{-at}}{a}\right)}{a + \exp\left(\frac{1}{a}\right) \left[\text{Ei}\left(-\frac{1}{a}\right) - \text{Ei}\left(-\frac{e^{-at}}{a}\right) \right]},\end{aligned}\tag{5a-b}$$

where Ei represents the exponential integral function. Unfortunately, Ei is not an elementary function and is consequently analytically intractable. However, solving the ansatz $\bar{N}_{W,Pre}(t) = xKe^{-at}$ for x and linearizing the result around $a = 0$ yields $x \approx 1$. So, when a is small, Equation 5b is well-approximated by

$$\bar{N}_{W,Pre}(t) \approx Ke^{-at}.\tag{6}$$

2.2.2 Invasion step

Wild types mutate to a niche-constructing mutant at a per-capita rate of u . Back mutations are neglected. We assume that mutations are rare enough to have a negligible effect on wild type dynamics. To model a hard selective sweep (e.g., Wilson et al. 2017; Czuppon et al. 2021), we also assume that mutations are rare enough to be ignored (i.e., $u = 0$) after the time, $t^* > 0$; then for all times $t \leq t^*$:

$$u := U \left(\int_{t=0}^{t^*} \bar{N}_{W,Pre}(t) dt \right)^{-1}, \quad (7)$$

where $0 < U < 1$. This definition of u ensures that the expected cumulative mutation rate over $[0, t^*]$ equals U irrespective of a and K . Note that the value of t^* is nearly arbitrary: as long as u remains inversely proportional to the expected cumulative abundance of wild types over $[0, t^*]$, any value of $t^* > 0$ can be chosen without altering the expected number of mutations. If $t^* \approx \infty$, then Equations 6 and 7 give

$$u \approx \frac{aU}{K}, \quad (8)$$

where hereafter $aU \ll K$. Implicit in our full definition of u is that, if $N_M(t) = 0$ and $t > t^*$, extinction is certain. If a mutant appears, it does so at time $t_0 \geq 0$. We define t_0 as any time at which a mutation causes $N_M(t_0) > 0$. Under our rarity assumption, the expected initial growth rate of a mutant is approximately $(1 - cg) \frac{\bar{H}_{Pre}(t_0)}{\bar{H}_{Max}} + (1 - cg) \left(1 - \frac{\bar{H}_{Pre}(t_0)}{\bar{H}_{Max}} \right) g - \frac{\bar{N}_{W,Pre}(t_0)}{K}$. We can use this growth rate to approximate the threshold time, t_M^+ , beyond which a mutant can appear with a positive expected growth rate: using Equation 6 for $\bar{N}_{W,Pre}(t_0)$, setting the growth rate equal to zero, and solving for t_0 gives

$$t_M^+ \approx \frac{\ln \left(\frac{1+cg}{1-cg} \right)}{a}. \quad (9)$$

Noteworthy is that Equation 9 is simply the time at which $\bar{H}_{Pre}(t) = \bar{H}_{Poly}$ and $\bar{N}_{W,Pre}(t) = \bar{N}_{Poly}$, meaning that, when $\bar{N}_{W,Poly} > 0$, growth rates change direction whenever habitat type 1's abundance and the total population size cross \bar{H}_{Poly} and \bar{N}_{Poly} , respectively. Moreover, t_M^+ monotonically increases with respect to the cost of niche construction, cg ; thus, as cg increases, so too does the earliest time, on average, at which a mutant can appear with a positive growth rate. Following the appearance of a mutant, and assuming secondary mutations occur at a negligible rate, the population's expected dynamics over $[t_0, \infty]$ are given by Equations 1a-c, with $\bar{H}(t_0) \rightarrow H(t_0)$, $\bar{N}_M(t_0) \rightarrow N_M(t_0) = 1$, and $\bar{N}_W(t_0) \rightarrow N_W(t_0)$ (i.e., the system's realized and expected paths over $[t_0, \infty]$ start from the same point). We assume that the invasion step completes at the first time, t_{Inv} , at which $N_M(t)$ is greater than or equal to 90% of the expected mutant equilibrium abundance, which is $\bar{N}_{M,Poly}$ when $\bar{N}_{W,Poly} > 0$ and $\bar{N}_{M,Mono}$ otherwise.

2.2.3 Post-invasion step

Once mutants have successfully invaded the population, they are expected to either fix or coexist with wild types. When $\bar{N}_{W,Pol y} > 0$ and $\bar{N}_{W,Pre}(t_0) \approx \bar{N}_{Pol y}$, the first time at which wild types are expected to acquire a positive growth rate, $t_0 + t_W^+$, is roughly the time at which $\bar{H}(t_0 + t_W^+) = \bar{H}_{Pol y}$ due to mutants performing niche construction. We can approximate t_W^+ by first noting that, if a mutant appears sufficiently late (i.e., $t_0 \gg 0$), the mutant's expected initial growth rate is approximately $(1 - cg)g$ and, therefore, the expected trajectory of mutants during invasion is roughly $e^{(1-cg)gt}$. Assuming the growth rate of habitat type 1 is proportional to that of mutants during invasion, we can set the mutants' invasion trajectory equal to $\bar{H}_{Pol y}$ and solve for t to acquire

$$t_W^+ \approx \frac{\ln\left(\frac{H_{Max}(1-cg)}{1+c-cg}\right)}{g(1-cg)}. \quad (10)$$

Equation 10 approximates how long, on average, wild types must wait to begin their rebound in size following the appearance of a mutant. If $H_{Max} > 1 + c$, t_W^+ approaches infinity in the limit as g approaches zero from the right: when few habitats are being produced, the environment is repaired slowly and, consequently, wild types must wait a prolonged period of time to attain a positive growth rate. Evaluating Equation 6 at time $t_0 + t_W^+$ reveals that a longer waiting time gives wild types a lower expected minimum abundance of

$$\bar{N}_{W,Post} \approx K e^{-a(t_0 + t_W^+)}. \quad (11)$$

If mutants successfully invade and wild types survive, the population is expected to achieve a size of $\bar{N}_{Pol y} = \bar{N}_{M,Pol y} + \bar{N}_{W,Pol y}$. Since mutants are not expected to acquire a positive growth rate until $\bar{N}_{W,Pre}(t) < \bar{N}_{Pol y}$, however, the population's total size on average drops below $\bar{N}_{Pol y}$. Consequently, assuming the population remains near $\bar{N}_{Pol y}$ over time and K is large enough for $\bar{N}_{Pol y} \approx \bar{N}_{W,Pol y} \approx K$, mutants are expected to reach a post-invasion maximum abundance that is proportional to $(\bar{N}_{Pol y} - \bar{N}_{W,Post}) > 0 \Rightarrow (\bar{N}_{W,Pol y} - \bar{N}_{W,Post}) > 0 \Rightarrow (\bar{N}_{Pol y} - \bar{N}_{W,Post}) > \bar{N}_{M,Pol y}$, thereby overshooting $\bar{N}_{M,Pol y}$ and initiating the system's oscillations (Fig. 1a). We approximate the expected post-invasion minimum abundance of mutants, $\bar{N}_{M,Post}$, by first noting that, under our large- K assumption, the wild types' first post-invasion amplitude of oscillation is $A^* \approx \bar{N}_{W,Pol y} - \bar{N}_{W,Pol y} e^{-a(t_0 + t_W^+)}$. This amplitude comprises a fraction $A \approx \frac{A^*}{\bar{N}_{Pol y}}$ of $\bar{N}_{Pol y}$. Thus, the total population's post-invasion minimum size can be written as $\bar{N}_{Pol y} - A^* \approx (1 - A)\bar{N}_{M,Pol y} + (1 - A)\bar{N}_{W,Pol y}$. Letting $\bar{N}_{M,Post} \approx (1 - A)\bar{N}_{M,Pol y}$, we have

$$\begin{aligned}\bar{N}_{M,Post} &\approx (1 - A) \left(\frac{aH_{Max}}{cg} \right) \\ A &\approx \left(1 - e^{-a(t_0+t_W^+)} \right) \left[1 - \frac{a(1+c-cg)H_{Max}}{cg(1-cg)K} \right].\end{aligned}\tag{12}$$

We can see that a number of factors can affect $\bar{N}_{M,Post}$, e.g., K and t_0 . Particularly noteworthy is that an increase in g can amplify the oscillations, potentially increasing the risk of mutant extinction. To account for the possibility of post-invasion extinction on a short timescale, we assume that mutants establish in the population—and therefore the rescue process completes—at the earliest time, t_{Est} , beyond which the total population is expected to stay above 90% of its expected equilibrium abundance. Specifically, we define $t_{Est} := t_0 + \max \left[\{t \mid \bar{N}_M(t_0 + t) = 0.9\bar{N}_{M,Polym} \text{ or } \bar{N}_W(t_0 + t) = 0.9\bar{N}_{W,Polym}\} \right]$ if $\bar{N}_{W,Polym} > 0$ and $t_{Est} := t_{Inv}$ otherwise, such that the post-invasion step is characterized by the oscillations. We define t_{Est} in this way to narrow focus on the oscillations and so our time interval of interest, $[t_0, t_{Est}]$, does not include all possible extinction times. When $N_M(t) > 0$ and $t \geq t_{Est}$, mutants are deemed fixed if $\bar{N}_{W,Polym} \leq 0$ or $N_W(t_{Est}) = 0$.

3 Methods

3.1 Mathematical approximations

We use branching process theory (Haldane 1927; Uecker and Hermisson 2011) to analytically and numerically predict the probability of evolutionary rescue, P_R . This framework entails two primary assumptions: that mutants (or any focal type) are rare enough to reproduce independently of one another and that such mutants have a positive growth rate on average (Uecker and Hermisson 2011). As shown in the appendix of Uecker and Hermisson (2011), the general equation for P_R is

$$P_R = 1 - \exp \left(- \int_{t_0=0}^{\infty} u\bar{N}_W(t_0)P_{Est}(t_0)dt_0 \right),\tag{13}$$

where $u\bar{N}_W(t_0)$ is the expected rate at which mutations enter the population at time t_0 and $P_{Est}(t_0)$ is the probability that such a mutation establishes in the population. Since establishment can result in mutant fixation or a polymorphism, we let P_{Est} equal

$$P_{Est} = P_{Fix} + P_{Polym},\tag{14}$$

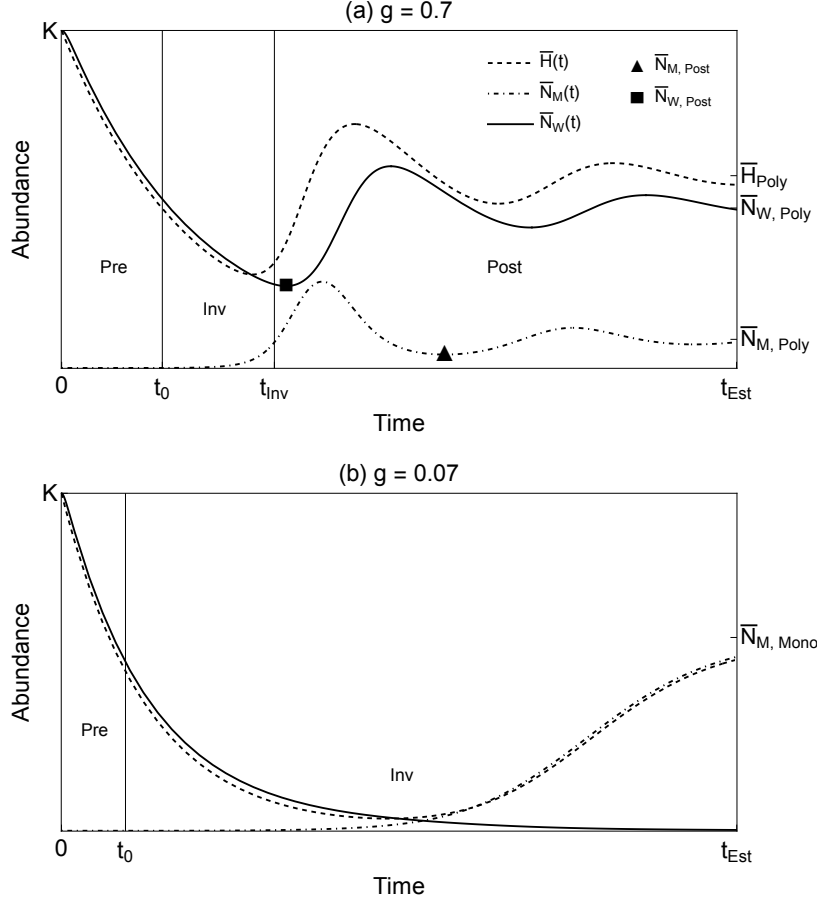


Figure 1: Expected dynamics of our evolutionary rescue model. (a) The pre-invasion step of rescue entails the population declining toward extinction in the absence of mutants. Once a mutant appears at time t_0 , the invasion step begins. The invasion step ends at the first time, t_{Inv} , at which mutants reach or surpass 90% of their expected equilibrium abundance. This abundance is $\bar{N}_{M, Poly}$ when $\bar{N}_{W, Poly} > 0$ and $\bar{N}_{M, Mono}$ otherwise. If g is sufficiently large, damped oscillations are expected to occur. These oscillations characterize the post-invasion step of rescue. During the oscillations, mutants and wild types are expected to reach minimum abundances of $\bar{N}_{M, Post}$ and $\bar{N}_{W, Post}$, respectively. The rescue process completes at the first time, t_{Est} , at which both types are expected to remain above 90% of their respective equilibrium abundances. (b) If g is sufficiently low, wild types are expected to go extinct and the oscillations disappear. In such a case, $t_{Est} := t_{Inv}$. Note that a reduction in g can extend the invasion step's duration by reducing the mutant invasion rate. The trajectories in this figure are acquired by numerically solving Equations 1a-c with $\bar{H}(0) = H_{Max}$, $\bar{N}_W(0) = K$, $\bar{N}_M(t) = 0$ for $t < t_0$, and $\bar{N}_M(t_0) = 1$. Parameter values are $a = 0.03$, $c = 0.5$, $H_{Max} = K = 10^3$, and $t_0 = 25$.

where P_{Fix} and P_{Poly} are the probabilities of mutant establishment via fixation and a polymorphism, respectively. We carry out our approximation of these two terms stepwise. We first approximate the probability that mutants invade the population, P_{Inv} . Then we approximate the probability of post-invasion wild type survival given mutant invasion, $P_{W, Post}$; and the probability that mutants survive the post-invasion step given that they invade and wild types survive, $P_{M, Post}$. We assume that $\bar{N}_{M, Mono}$ is large enough such that, if mutants invade and wild types go extinct, the probability of mutant survival is $P_{M, Mono} \approx 1$. Under

this assumption, Equation 14 can be written as

$$P_{Fix} + P_{Poly} \approx P_{Inv}(1 - P_{W,Post}) + P_{Inv}P_{M,Post}P_{W,Post}. \quad (15)$$

The first term in Equation 15 says that the probability of mutant fixation is equal to the probability that mutants invade the population and wild types go extinct. The second term says that the probability of establishment via a polymorphism is equal to the probability that mutants invade and both types subsequently survive the post-invasion step.

3.1.1 Analytical approximations

For our analytical approximation of P_R (Eq. 13), we calculate $\bar{N}_{W,Pre}(t)$ with Equation 6 and u with Equation 8. We make use of quantities derived in Section 2.2 to approximate each of Equation 15's constituent probabilities, P_i , where $i \in \{Inv; M, Post; W, Post\}$. Each of these calculations is carried out as follows. First, we initialize the focal type (e.g., M) at the minimum abundance, \bar{N}_i , that is expected to be reached during the focal step of rescue (e.g., $Post$). As required by branching process theory, we assume that \bar{N}_i has a minimum value of one. That is, we let $\bar{N}_{Inv} = 1$ and we modify Equations 11 and 12 with $\bar{N}_{W,Post} \rightarrow \max[1, \bar{N}_{W,Post}]$ and $\bar{N}_{M,Post} \rightarrow \max[1, \bar{N}_{M,Post}]$, respectively. Second, we approximate the focal type's expected growth rate, denoted as $\bar{\lambda}_i - \bar{\mu}_i$, which we assume has recently become positive. For simplicity, we assume that habitat loss is slow enough for this growth rate to remain effectively constant while the focal type is rare. Finally, we use the continuous time survival probability derived by Kendall (1948) and later applied to population genetics by Uecker and Hermisson (2011; Eq. 14-15 therein) to calculate P_i . Under the constant growth rate assumption, this equation is

$$P_i \approx 1 - \left(\frac{\bar{\mu}_i}{\bar{\lambda}_i} \right)^{\bar{N}_i}. \quad (16)$$

3.1.2 Numerical approximations

For our numerical approximation of P_R , we calculate $\bar{N}_{W,Pre}(t)$ with Equation 5b and u with Equation 7. We replace Equation 13's upper bound of ∞ with t^* , chosen to equal the time at which $\bar{N}_{W,Pre}(t) = 0.01K$. To numerically approximate P_i , we relax our constant growth rate assumption (Appendix A).

3.2 Simulations

We test the validity of our mathematical predictions with a tau-leaping variant of the Gillespie algorithm (Cao et al. 2006). Simulations testing Equation 15 are initialized with abundances of $H(t_0) = \lfloor \bar{H}_{Pre}(t_0) \rfloor$, $N_M(t_0) = 1$, and $N_W(t_0) = \lfloor \bar{N}_{W,Pre}(t_0) \rfloor$ (Figs. 2-3), while those testing Equation 13 are initialized with $H(0) = H_{Max}$, $N_M(0) = 0$, and $N_W(0) = K$ (Fig. 4). Mutation initially occurs at a rate of $N_W(t)u$, which is set equal to zero once $t > t^*$. Both u and t^* are calculated numerically (see Section 3.1.2). If a mutation causes $N_M(t) > 0$, we record $H(t_0)$ and $N_W(t_0)$, then numerically calculate the mutation's establishment time, t_{Est} , defined in Section 2.2.3. If mutants go extinct, we reset the values of t_0 and t_{Est} . The population changes over time according to the rates, state changes, and events listed in Table 2. The size of each tau-leap, and the number of times that each event occurs during the leap, is calculated using the procedure summarized in Cao et al. (2006)—specifically, sections IIC (excluding step 3) and IV therein. This procedure approximately bounds the change in each transition rate, relative to the sum of the transition rates, by a value of $\epsilon = 0.03$. Further, the algorithm ensures that, if a given event is within $n_c = 10$ occurrences of exhausting its corresponding variable, that variable is updated according to the direct Gillespie algorithm (Gillespie 1976). This switching behavior is intended to maximize speed while ensuring accuracy at critical moments. If Equation 32a in Cao et al. (2006) yields a value of zero, we add 10^{-9} to that value to prevent an undefined leap size. And if a given variable has a negative value, its abundance is set equal to zero. The simulation proceeds until $t \geq t_{Est}$ or $N_M(t) = 0$ and $t > t^*$. If $N_M(t) > 0$ and $t \geq t_{Est}$, mutants are considered fixed if either $\bar{N}_{W,Poly} \leq 0$ or $N_W(t_{Est}) = 0$. All numerical calculations and simulations are performed using Mathematica 13.2.0.0.

Table 2: Transition rates and their corresponding state changes and events.

Rate	State change(s)	Event(s)
$H(t)a$	$H(t) \rightarrow H(t) - 1$	A patch of habitat one is lost
$N_M(t) (1 - cg) \left(1 - \frac{H(t)}{H_{Max}}\right) g$	$H(t) \rightarrow H(t) + 1$ $N_M(t) \rightarrow N_M(t) + 1$	A mutant niche constructs and gives birth
$N_M(t) (1 - cg) \frac{H(t)}{H_{Max}}$	$N_M(t) \rightarrow N_M(t) + 1$	A mutant gives birth
$N_M(t) \frac{N(t)}{K}$	$N_M(t) \rightarrow N_M(t) - 1$	A mutant dies
$N_W(t) \frac{H(t)}{H_{Max}}$	$N_W(t) \rightarrow N_W(t) + 1$	A wild type gives birth
$N_W(t) \frac{N(t)}{K}$	$N_W(t) \rightarrow N_W(t) - 1$	A wild type dies
$N_W(t)u$	$N_M(t) \rightarrow N_M(t) + 1$	A mutation occurs

4 Results

We proceed stepwise, as outlined in Section 3, toward a calculation of P_R . Hereafter, any assumptions that are made pertain only to our analytical results.

4.1 The probability of mutant invasion

We begin by approximating the probability of mutant invasion, P_{Inv} . Since we are assuming constant growth rates, Equation 16 will only yield meaningful results if mutants have a positive growth rate. For this reason, we assume that the probability of mutant invasion is non-zero only when the first mutant appears at a time, $t_0 > t_M^+$. We assume that, during the period relevant for escaping genetic drift and determining initial invasion, such a mutant is rare enough to have a negligible effect on the system's growth rates. Under this assumption, the mutant has birth and death rates of $\bar{\lambda}_{Inv} \approx (1 - cg) \frac{\bar{H}_{Pre}(t_0)}{H_{Max}} + (1 - cg) \left(1 - \frac{\bar{H}_{Pre}(t_0)}{H_{Max}}\right) g$ and $\bar{\mu}_{Inv} \approx \frac{\bar{N}_{W,Pre}(t_0)}{K}$, respectively. Plugging these rates into Equation 16 gives

$$P_{Inv} \approx \begin{cases} \Phi, & t_0 > t_M^+ \\ 0, & t_0 \leq t_M^+ \end{cases} \quad (17)$$

$$\Phi = 1 - \frac{1}{(1 - cg)(1 - g(1 - e^{at_0}))}.$$

Setting the derivative of Φ equal to zero and solving for g reveals that P_{Inv} exhibits a local maximum with respect to g , where the optimal g value is $\frac{1}{2} \left(\frac{1}{c} + \frac{1}{1 - e^{at_0}} \right)$. This optimum is less than $\frac{1}{2}$ when $0 < at_0 \leq \ln \left(\frac{1}{1 - c} \right)$, meaning that, when at_0 is sufficiently low such that competition with wild types is strong due to a surplus of habitat type 1 patches, a less than even chance of construction is optimal for mutant invasion. Such a low value of g decreases cg and hence increases mutant fitness in habitat type 1. If g becomes too low, however, the mutant birth rate in habitat type 2 diminishes enough to decrease P_{Inv} .

4.2 The probability of post-invasion survival

We now want to approximate the probability that type $j \in \{M, W\}$ survives the post-invasion step of rescue given mutant invasion, $P_{j,Post}$. Since the oscillations characterize the post-invasion step, we assume $P_{j,Post}$ is less than unity only when $\bar{N}_{W,Post} > 0$ and $g > G$ (Eq. 4). For such regimes, we assume that the focal type j has an initial abundance of $\bar{N}_{j,Post}$ (Eq. 12, 13). Further assumed is that type j has recently acquired a positive growth rate such that the expected per-capita birth rate of wild types is $\bar{\lambda}_{W,Post} \approx \frac{\bar{H}_{Poly}}{H_{Max}}$, and

that of mutants is $\bar{\lambda}_{M,Post} \approx (1 - cg) \frac{\bar{H}_{Poly}}{H_{Max}} + (1 - cg) \left(1 - \frac{\bar{H}_{Poly}}{H_{Max}}\right) g$. We approximate type j 's expected per-capita death rate, $\bar{\mu}_{j,Post}$, by assuming that type j is rare enough to have a negligible direct effect on the system's growth rates, while the non-focal type's abundance is initially near a local maximum of $\bar{N}_{Poly} - \bar{N}_{j,Post}$. Supporting this approach is that, even when $\bar{N}_{j,Post} \gg 0$, the death rate scales with the total population's amplitude of oscillation and, therefore, $\bar{N}_{Poly} - \bar{N}_{j,Post}$. Assuming K is large enough for $K e^{-a(t_0+t_W^+)} \approx \bar{N}_{W,Poly} e^{-a(t_0+t_W^+)}$, we have $\bar{\mu}_{W,Post} \approx \frac{\bar{N}_{Poly} - \bar{N}_{W,Poly} e^{-a(t_0+t_W^+)}}{K}$.

To approximate $\bar{\mu}_{M,Post}$, we first consider the expected dynamics of mutants and wild types when mutants are rebounding from a size of $\bar{N}_{M,Post}$ to $\bar{N}_{M,Poly}$ during the time interval, Δt . When a is small, mutants are expected to maintain a low growth rate for an appreciable amount of time during Δt . Meanwhile, assuming K is large enough for $\bar{N}_{Poly} \gg \bar{N}_{M,Post}$, wild types are expected to decline from an abundance near $\bar{N}_{Poly} - \bar{N}_{M,Post} \approx \bar{N}_{Poly}$ toward $\bar{N}_{W,Poly}$. This change in wild type abundance is non-negligible (i.e., $\bar{N}_{Poly} - \bar{N}_{W,Poly} \not\approx 0$) when $aH_{Max} \gg 0$. To account for these dynamics, we assume that, when mutants acquire a non-negligible growth rate, wild type abundance is roughly $\frac{\bar{N}_{Poly} + \bar{N}_{W,Poly}}{2}$. Noting that $\frac{\bar{N}_{Poly} + \bar{N}_{W,Poly}}{2} = \frac{K(1-cg)}{(1+c-cg)} - \frac{aH_{Max}}{2cg} \approx K \left(\frac{(1-cg)}{(1+c-cg)} - \frac{a}{2cg} \right)$ under our large- H_{Max} and large- K assumptions, we have $\bar{\mu}_{M,Post} \approx \frac{\bar{N}_{Poly} + \bar{N}_{W,Poly}}{2K} \approx 1 - \frac{c}{1+c(1-g)} - \frac{a}{2cg}$. Plugging $\bar{\lambda}_{j,Post}$ and $\bar{\mu}_{j,Post}$ into Equation 16 then gives

$$P_{j,Post} \approx \begin{cases} 1, & \bar{N}_{W,Poly} > 0 \text{ and } g \leq G \\ \Psi_j, & \bar{N}_{W,Poly} > 0 \text{ and } g > G \\ 0, & \bar{N}_{W,Poly} \leq 0 \end{cases} \quad (18)$$

$$\Psi_M = 1 - \left(1 - \frac{a(1+c-cg)}{2cg(1-cg)} \right)^{\max[1, \bar{N}_{M,Post}]}$$

$$\Psi_W = 1 - \left[1 - e^{-a(t_0+t_W^+)} \left(1 - \frac{a(1+c-cg)H_{Max}}{cg(1-cg)K} \right) \right]^{\max[1, \bar{N}_{W,Post}]}$$

The parenthetic terms in $P_{M,Post}$ and $P_{W,Post}$ suggest that conditions limiting wild type inheritance of the constructed habitats—such as high a or low g values—can reduce the wild types' per-capita survival probability, and therefore their competitive effect on mutants, enough to increase the probability of a given mutant's survival. The predictions of Equations 17 and 18 are tested in the following sections.

4.3 The probability of mutant fixation

Now that we have approximated the terms comprising Equation 15, we can analyze the probability of mutant fixation, $P_{Fix} \approx P_{Inv} (1 - P_{W,Post})$. Figure 2a shows that a lower probability of niche construction,

g , can increase P_{Fix} , unless g is so low that mutants invade slowly enough for genetic drift to reduce the probability of mutant invasion, P_{Inv} (Eq. 17; Fig. 2b). Low values of g not only reduce the mutants' fecundity cost, cg , but also prolong the time required for mutants to restore the abundance of habitat type 1 through niche construction, thereby decreasing the expected minimum abundance of wild types, $\bar{N}_{W,Post}$ (Eq. 11). The smaller wild type abundance leads to a lower probability of wild type survival, $P_{W,Post}$ (Eq. 18).

Irrespective of g , there are multiple ways to increase P_{Fix} . One way is to increase mutant appearance time, t_0 (Fig. 2a). The lower wild type density corresponding to a higher value of t_0 not only decreases $P_{W,Post}$, but also gives mutants a larger initial growth rate, which increases P_{Inv} (Fig. 2b; see also Uecker and Hermisson 2014). An increase in the rate of habitat loss, a , has a similar effect (Fig. 2c-d), as the usable habitats are destroyed before wild types disperse to them. If a and t_0 are sufficiently small, P_{Inv} can exhibit a slight local maximum with respect to g (Fig. 2d), because the benefit of a reduction in cg can exceed the effect of density dependent competition on mutant fitness. All else equal, increasing the total number of habitats, H_{Max} , or reducing the population's carrying capacity, K , can also increase P_{Fix} (Fig. 2e), but only by decreasing $P_{W,Post}$. Both changes diminish the wild type growth rate enough to decrease $\bar{N}_{W,Post}$: a lower value of K directly reduces $\bar{N}_{W,Post}$, while a larger value of H_{Max} prolongs the time required for wild types to achieve a positive growth rate, t_W^+ (Eq. 10). The reason P_{Inv} is unaffected by K (see also Marrec and Bitbol 2020) and H_{Max} (Fig. 2f) is because the mutant fitness benefits of greater resource availability (i.e., higher K) and reproductive space (i.e., higher H_{Max}) are respectively negated by larger wild type and habitat type 1 abundances.

4.4 The probability of mutant establishment

Noteworthy is that P_{Fix} is analytically over-approximated for $t_0 \gg 0$ (Fig. 2a). This is largely because $\bar{N}_{W,Pre}(t_0) \ll \bar{N}_{Poly}$ when $t_0 \gg 0$, meaning that our assumption in Section 2.2.3, $\bar{N}_{W,Pre}(t_0) \approx \bar{N}_{Poly}$, is violated. This violation leads to an over-approximation of how much construction is needed for $\bar{\lambda}_W(t_0+t) - \bar{\mu}(t_0+t) > 0$, which in turn causes an under-approximation of $\bar{N}_{W,Post}$ and, hence, an over-approximation of $\bar{\mu}_{W,Post}$. For this reason, hereafter we assume that, when $\bar{N}_{W,Poly} > 0$, $\bar{\mu}_{W,Post}$ is small enough for $P_{W,Post} \approx 1$; and, as before, we let $P_{W,Post} \approx 0$ if $\bar{N}_{W,Poly} \leq 0$.

Figure 3 shows the probability of mutant establishment, $P_{Est} \approx P_{Inv}((1 - P_{W,Post}) + P_{M,Post}P_{W,Post})$. Comparing Figures 3a and 2b reveals that, even when mutant invasion is likely, an increase in g can reduce P_{Est} . In such a case, the higher rate of niche construction increases the birth rate of wild types, giving

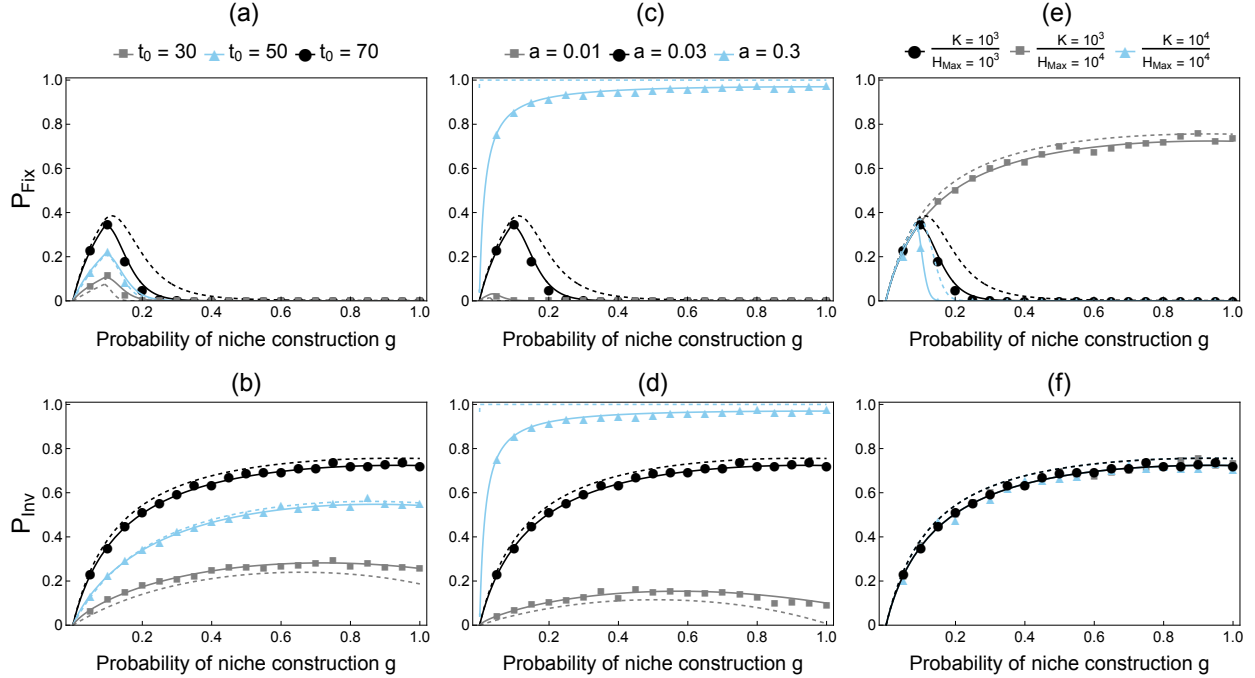


Figure 2: The probability of mutant fixation, P_{Fix} , as a function of the probability of niche construction, g . (a) A smaller value of g and larger mutant appearance time, t_0 , can increase P_{Fix} . Less frequent niche construction not only reduces the mutants' fecundity cost, but also—along with a larger value of t_0 —decreases the expected minimum abundance of wild types, $\bar{N}_{W,Post}$, which in turn increases the probability of wild type extinction and, thus, mutant fixation. (b) If g becomes too low, the probability of mutant invasion, P_{Inv} , can decrease. (c-d) Increasing the rate of habitat loss, a , promotes mutant fixation by raising the mutant growth rate during invasion and lowering the post-invasion growth rate and abundance of wild types. (e) A larger total number of habitats, H_{Max} , and a smaller population carrying capacity, K , can also increase P_{Fix} by decreasing the probability of wild type survival. (f) However, in contrast to an increase in a , a change in H_{Max} or K does not affect P_{Inv} . Markers: simulations; solid curves: numerical approximations; dashed curves: analytical approximations. Unless stated otherwise, parameter values are $a = 0.03$, $c = 0.5$, $H_{Max} = K = 10^3$, $t_0 = 70$, and $U = 0$. Simulation results are averages over 10^3 replicate runs.

them a larger expected polymorphic abundance, $\bar{N}_{W,Polym}$ (Eq. 3c). Due to negative density dependence, mutants in turn have a smaller polymorphic equilibrium abundance, $\bar{N}_{M,Polym}$ (Eq. 3b), and therefore a smaller expected post-invasion minimum abundance, $\bar{N}_{M,Post}$ (Eq. 12). A smaller value of $\bar{N}_{M,Post}$ leads to a lower probability of post-invasion mutant survival, $P_{M,Post}$ (Eq. 18).

Interestingly, the post-invasion decrease in P_{Est} can become more pronounced with an increase in t_0 (Fig. 3a), as the total population's amplitude of oscillation can increase with t_0 , thereby decreasing $\bar{N}_{M,Post}$ (Fig. 3b). Specifically, an increase in t_0 can result in a lower value of $\bar{N}_{W,Post}$, allowing mutants to reach a larger maximum abundance and construct more habitats; the increase in usable habitats leads to stronger selection against mutants, which in turn decreases $\bar{N}_{M,Post}$. Figure 3b illustrates this relationship between t_0 and $\bar{N}_{M,Post}$, with $t_0^* \approx t^*$ being an approximation of the latest time at which mutants can appear in our model (i.e., when Eq. 6 equals $\bar{N}_{W,Pre}(t^*)$). Note that the limit of our $\bar{N}_{M,Post}$ approximation as t_0

approaches infinity is $\frac{(aH_{Max})^2(1+c-cg)}{K(cg)^2(1-cg)}$, suggesting that there is a finite limit to the negative effect of t_0 on P_{Est} . Also note that, as indicated by the dotted curve in Figure 3a, our approximation can miss the post-invasion effect of $t_0 \gg 0$ on P_{Est} without the assumption that $P_{W,Post} \approx 1$.

The post-invasion decrease in P_{Est} can be prevented with a higher rate of habitat loss, a (Fig. 3c). Such an increase in a enhances the strength of selection for mutants, as the usable habitats remain at lower densities over time. Fewer usable habitats leads to weaker competition with wild types, causing an increase in $\bar{N}_{M,Poly}$ and a reduction in the amplitude of oscillation, which in turn increases $\bar{N}_{M,Post}$ (Fig. 3d). Mutants are expected to fix (i.e., $\bar{N}_{W,Poly} < 0$) when $a \geq a^* = \frac{K(1-cg)cg}{H_{Max}(1+c-cg)}$.

Similarly, increasing H_{Max} or shrinking K can prevent post-invasion extinction (Fig. 3e). When $\frac{K}{H_{Max}}$ is sufficiently small, wild types have a low probability of dispersing to a constructed habitat, which translates to a low value of $\bar{N}_{W,Poly}$. A reduction in $\bar{N}_{W,Poly}$ can decrease the amplitude of oscillation, since the amplitude is proportional to $\bar{N}_{W,Poly} - \bar{N}_{W,Post}$, which then increases $\bar{N}_{M,Post}$ (Fig. 3f). If $\frac{K}{H_{Max}} \leq \frac{K^*}{H_{Max}} = \frac{K}{H_{Max}^*} = \frac{a(1+c-cg)}{cg(1-cg)}$, then mutants are expected to fix. Thus, although $\bar{N}_{M,Poly}$ is not a function of K , the effect of a reduction in K on wild type abundance can transiently increase mutant fitness enough to increase P_{Est} (Fig. 3e). The limit of our $\bar{N}_{M,Post}$ approximation as K approaches infinity has a value of $\frac{aH_{Max}e^{-a(t_0+t_M^+)}}{cg}$, however, suggesting that the negative effect of K diminishes as H_{Max} increases. What this highlights is that, because $\bar{N}_{M,Poly}$ scales with H_{Max} , an increase in H_{Max} can boost P_{Est} even when $\frac{K}{H_{Max}}$ remains constant (Fig. 3e).

4.5 The probability of evolutionary rescue

We complete our analysis by approximating the probability of evolutionary rescue, P_R . To do this, we make two final assumptions: first, note that the e^{-at} term in Equations 5b and 6 quickly approaches zero when $a \gg 0$. Consequently, when a is sizeable (e.g., $a \geq 0.3$), mutant fitness can increase appreciably over a short time interval, rendering mutant fitness at time t_0 a poor proxy for that throughout invasion. For this reason, we shift time slightly forward for our approximation of P_{Inv} (and so t_M^+) such that $e^{-at_0} \rightarrow e^{-a(t_0+\delta)} = \max \left[e^{-a(t_0+\frac{1+c-cg}{1-cg}e^a)}, \frac{e^{-at_0}}{2+\exp\left[-\frac{(1+c-cg)}{(1+cg)}\right]} \right]$. The derivation of δ is given in Appendix B. Second, we let $\bar{N}_{M,Post} \rightarrow \max \left[1, \frac{(aH_{Max})^2(1+c-cg)}{K(cg)^2(1-cg)} \right]$, where the second term is the limit of Equation 12 as t_0 approaches infinity. This modification, along with our assumption that $P_{W,Post} \approx 1$, permits calculation of the integral in Equation 13 by making P_{Inv} the only term in P_{Est} that varies with t_0 . Replacing the lower bound of Equation 13's integral with $t_0 = t_M^+$ and integrating gives

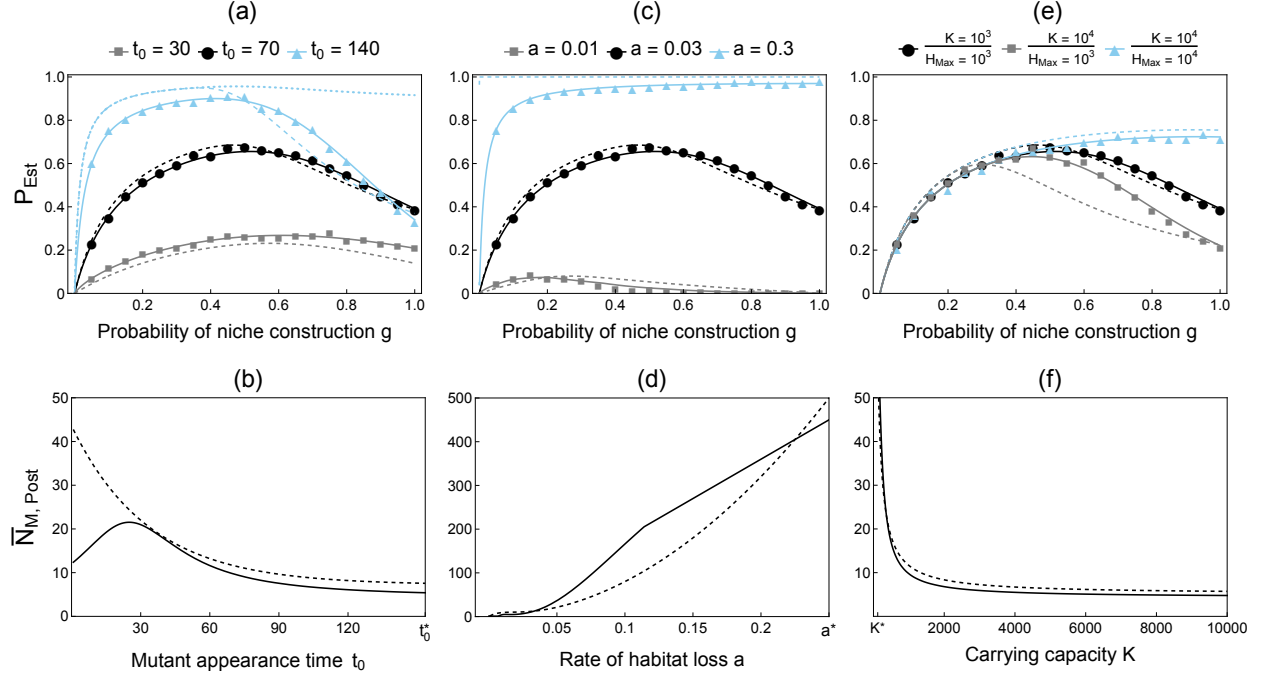


Figure 3: The probability of mutant establishment, P_{Est} , as a function of the probability of niche construction, g . (a) Post-invasion competition with wild types can reduce P_{Est} when g is sufficiently high. This reduction is less likely for low-to-moderate values of g , because such values of g increase the mutants' polymorphic equilibrium abundance, $\bar{N}_{M,Pol}$ —and therefore the mutants' expected post-invasion minimum abundance, $\bar{N}_{M,Post}$ —making mutants less vulnerable to post-invasion extinction. The dotted curve shows an example of our analytical approximation of P_{Est} without the assumption that wild type survival is certain. (b) An increase in mutant appearance time, t_0 , can decrease $\bar{N}_{M,Post}$, thereby increasing the risk of mutant extinction. This happens when a larger t_0 value augments the total population's amplitude of oscillation. The latest time at which mutants can appear in the population is t_0^* . (c-d) A higher rate of habitat loss, a , can prevent post-invasion extinction mainly by increasing $\bar{N}_{M,Post}$ through both an increase in $\bar{N}_{M,Pol}$ and a reduction in the amplitude of oscillation. Mutants are expected to fix when $a \geq a^*$. (e-f) Although $\bar{N}_{M,Pol}$ is not a function of K , a higher value of $\frac{K}{H_{Max}}$ can amplify the oscillations and, in doing so, reduce $\bar{N}_{M,Post}$ enough to decrease P_{Est} . Mutants are expected to fix only when $\frac{K}{H_{Max}} \leq \frac{K^*}{H_{Max}} = \frac{K}{H_{Max}^*}$. Even when $\frac{K}{H_{Max}}$ remains constant, however, P_{Est} can increase with H_{Max} due to $\bar{N}_{M,Pol}$ being proportional to H_{Max} . Markers: simulations; solid curves: numerical approximations; dashed curves: analytical approximations. Unless stated otherwise, parameter values are $a = 0.03$, $c = 0.5$, $g = 1$, $H_{Max} = K = 10^3$, $t_0 = 70$, and $U = 0$. Simulation results are averages over 10^3 replicate runs.

$$P_R \approx 1 - \exp \left[\frac{U e^{a\delta} g [\ln [g(1+c-cg)] + (1-g)(1-cg)] P_{Post}}{(1-g)^2(1-cg)} \right]$$

$$P_{Post} \approx \begin{cases} 1 - \left(1 - \frac{a(1+c-cg)}{2cg(1-cg)} \right)^{\max \left[1, \frac{(\alpha H_{Max})^2(1+c-cg)}{K(cg)^2(1-cg)} \right]}, & \bar{N}_{W,Pol} > 0 \text{ and } g > G \\ 1, & \bar{N}_{W,Pol} \leq 0 \text{ or } g \leq G, \end{cases} \quad (19)$$

where $e^{a\delta} = \min \left[e^{\left(a \frac{1+c-cg}{1-cg} e^a \right)}, 2 + e^{\left(1 - \frac{2+c}{1+cg} \right)} \right]$ and P_{Post} is the probability of post-invasion survival. Figure 4 shows that lower values of g can increase P_R . In such a case, less frequent niche construction increases

mutant fitness both directly—by lowering cg during the invasion and post-invasion steps—and indirectly, by repairing the environment slowly enough for wild types to reach and maintain a low post-invasion abundance. Similarly, larger values of a can augment P_R by reducing the density of usable habitats and, therefore, promoting mutant invasion (through $e^{a\delta}$ in Eq. 19) and post-invasion survival (Fig. 4a). The post-invasion benefits of larger H_{Max} values and smaller K values can also boost P_R (Fig. 4b), with aH_{Max} 's dual effect on P_{Est} —a larger $\bar{N}_{M, Poly}$ value and a smaller amplitude of oscillation—being captured by the squared H_{Max} term in Equation 19. Assuming $aH_{Max} \gg 0$ such that $P_{Post} \approx 1$ and $e^{a\delta} = 2 + e^{(1-\frac{2+c}{1+cg})}$, Equation 19's limit as g approaches one is $1 - \exp\left[-\frac{1}{2}U(1-c)\left(2 + e^{-\frac{1}{1+cg}}\right)\right]$. So, even if $aH_{Max} \gg 0$, P_R can further increase with a reduction in the maximum cost of construction, c , or greater mutational input, U (Fig. 4c).

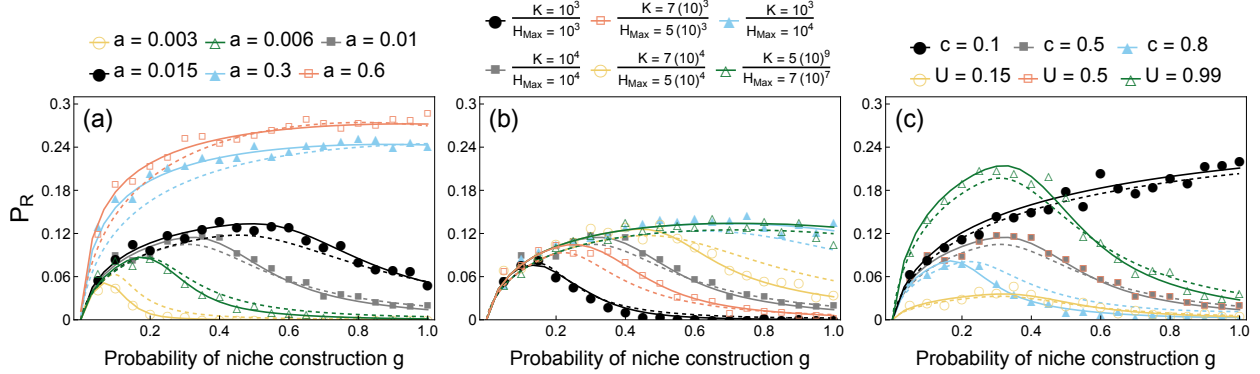


Figure 4: The probability of evolutionary rescue, P_R , as a function of the probability of niche construction, g . (a) A lower value of g can increase P_R . This is because infrequent niche construction not only reduces the fecundity cost of construction, cg , but also ensures that wild types remain rare enough for mutants to maintain a large post-invasion abundance. By reducing the density of usable habitats, an increase in a indirectly promotes mutant survival during the invasion and post-invasion steps of rescue, which can increase P_R . (b) Increasing H_{Max} or reducing K can boost P_R by increasing the post-invasion abundance of mutants. (c) P_R can also increase with a lower maximum cost of construction, c , or a higher expected cumulative mutation rate, U . Markers: simulations; solid curves: numerical approximations; dashed curves: analytical approximations. Unless stated otherwise, parameter values are $a = 0.01$, $c = 0.5$, $H_{Max} = K = 10^4$, and $U = 0.5$. Simulation results are averages over 10^3 replicate runs.

5 Discussion

We analyzed the probability of evolutionary rescue, P_R , via a niche-constructing mutation in competition with non-niche-constructing wild types who benefit from the constructed habitats. Our main result was that frequent niche construction promotes competition-driven damped oscillations that can induce population collapse shortly after mutant invasion. This problem can worsen if a mutant appears late in the population's decline, due to a larger amplitude of oscillation. Such post-invasion extinction can be avoided by decreasing

the probability of construction, increasing the rate of habitat loss, expanding the reproductive environment, or reducing the population's carrying capacity. All else equal, each of these changes promotes mutant fixation by impeding inheritance of the constructed habitats by wild types.

Density regulation and competitive release

Our results contribute to the growing list of known mechanisms by which rescue mutants can experience competitive release, whereby mutant fitness increases in response to a reduction in wild type density. Uecker et al. (2014) found that, in structured populations, a sufficiently high migration probability can reduce the number of wild types in a deme containing mutants, thereby increasing mutant fitness during invasion. In a rescue model with habitat choice, Czuppon et al. (2021) clarified that this benefit of frequent migration is typically highest when wild types tend to immigrate into a novel habitat—away from the mutants' habitat of origin. And, similar to our study, Uecker et al. (2014) revealed that competitive release can be achieved with an increase in the rate of habitat loss, granted that the probability of mutation remains sufficiently high. They found that this benefit of faster environmental change increases when, due to standing genetic variation, mutants are already present at the onset of rapid habitat loss. Here we showed that niche-constructing mutants can themselves promote competitive release by performing construction infrequently enough for the constructed habitats, and thus wild types, to remain rare.

The tragedy of the commons and niche monopolization

Population genetic models of rescue typically assume that, following mutant invasion, the population can hold a nearly infinite number of individuals. Here we showed that, when this assumption is relaxed, post-invasion competition with wild types can drive mutants to a small abundance shortly after invasion, thereby decreasing the probability of rescue. This result can be viewed as the tragedy of the commons (Rankin et al. 2007), by which a population is driven to extinction through cheater (wild type) exploitation of a shared resource (constructed habitats) produced by cooperators (mutants). In a deterministic model of rescue via cooperation, Henriques and Osmond (2020) also found that too much cooperation can lead to extinction, but, in their model, such extinction resulted from cooperation lowering the effective population size despite increasing the census size.

Multiple non-rescue models of niche construction and cooperation have identified solutions to the tragedy of the commons (Lehmann 2007, 2008; Krakauer et al. 2009; Chisholm et al. 2018). Perhaps most general among these solutions is that of Krakauer et al. (2009), who showed that mutants must exhibit some

degree of monopolization over the constructed resource to avoid extinction. Spatial monopolization, for instance, can occur under limited dispersal when the benefit of future descendants ecologically inheriting a resource outweighs the cost of producing the resource in the present (Lehmann 2007, 2008). Alternatively, monopolization can occur through pleiotropy, where the capacity to niche construct is controlled by the same gene or gene network as the benefit of the constructed resource (Chisholm et al. 2018). More solutions to the tragedy of the commons are reviewed in Rankin et al. (2007). Here we found that—when habitats are lost in the absence of niche construction—mutants can monopolize the habitats by either performing construction only occasionally or living where one’s constructed habitats remain rare due to rapid habitat loss or a large reproductive environment relative to population size.

Niche construction and experimental evolution

To our knowledge, the relationship between evolutionary rescue and niche construction has not been investigated empirically. Still, the results of multiple experiments corroborate some of our model’s qualitative predictions. For instance, Callahan et al.’s (2014) experiments with the bacterium, *Pseudomonas fluorescens*, revealed that niche construction can rapidly and repeatedly evolve in the face of consistent environmental changes. If given enough time (120 days), such environmental change led to wild type extinction and, thus, mutant fixation (Callahan et al. 2014). Further revealed was that, by the end of the experiment, mutants had evolved greater maladaptation to the ancestral environment (Callahan et al. 2014). The authors speculated that such maladaptation was the cost of evolving a greater ability to niche construct—and therefore only appeared once mutants were common—which would explain why the mutants so readily invaded (Callahan et al. 2014). Although herein we assumed that the capacity to niche construct is constant, our results support Callahan et al.’s (2014) hypothesis: during invasion, mutants likely exhibited a low-to-moderate capacity to niche construct, giving them a lower fecundity cost in the ancestral environment and so a higher probability of survival.

Furthermore, Kümmerli and Brown (2010) found that siderophore-producing strains of *P. aeruginosa* had the selective advantage over cheaters when production was facultative: by producing siderophores only when needed, producers were able to minimize the cost of production and, in doing so, reduce the strength of selection for cheaters (Kümmerli and Brown 2010). This result is similar to our finding that infrequent niche construction can promote mutant survival by reducing not only the fecundity cost of niche construction but also the density dependent effect of wild types. Moreover, Ross-Gillespie et al. (2009) found that the relative fitness of *P. aeruginosa* cheaters was greatest at high population densities. As with

large carrying capacities in our model, such densities made cheaters more likely to encounter and exploit the public good (Ross-Gillespie et al. 2009).

Finally, Celiker and Gore’s (2012) experiments with invertase-producing budding yeast populations showed that inter-specific competition with the bacterium, *Escherichia coli*, can facilitate producer persistence by depleting the public good and hence reducing cheater density (Celiker and Gore 2012). The lower public good density in turn generated selection for producers due to their slight preferential access to the resource upon its creation (Celiker and Gore 2012). In terms of our model, such a high degree of inter-specific competition for the public good is akin to a high rate of habitat loss, whereby the constructed habitats are destroyed before exploitation by wild types can take place. Our model’s predictions could be tested more directly with a rescue experiment similar to that of Bell and Gonzalez (2009), whose model organism was budding yeast (Carlson and Botstein 1982).

Analytical shortcomings and future model extensions

We incorporated the post-invasion oscillations into our approximation of P_R (Eq. 19) by first approximating the expected post-invasion minimum abundance of mutants, $\bar{N}_{M,Post}$, then calculating the probability that mutants rebound in size from their minimum. Our key assumptions were as follows: first, the rate of habitat loss, a , was assumed to be low enough for growth rates and abundances to be effectively constant while mutants are rare. To account for the rapid changes in mutant fitness corresponding to $a \gg 0$, we shifted mutant fitness slightly forward in time (Appendix B). The heuristic nature of this modification indicates that more work should be done to analytically account for our model’s time-inhomogeneous growth rates. Our numerical approximations showed that, if such time-varying growth rates are acquired, branching process theory yields accurate predictions. Second, to approximate the post-invasion minimum and maximum abundances of mutants and wild types, we assumed that the total population maintains a size near the polymorphic equilibrium, \bar{N}_{Poly} . Third, we assumed that the carrying capacity, K , is sizeable so that key quantities could be equated (e.g., $K \approx \bar{N}_{Poly}$). Fourth, we prevented a strong over-approximation of the fixation probability for late mutant appearance times, t_0 , by assuming that wild types are certain to survive due to having a negligible death rate when rare. Finally, we made the rate at which mutants appear and establish integrable with respect to t_0 by replacing $\bar{N}_{M,Post}$ with its limit as t_0 approaches infinity. Our approximation of $\bar{N}_{M,Post}$, and so P_R conditional on oscillations occurring, was most accurate when a was moderate (e.g., $a = 0.01$ in Fig. 4a); K and the total number of habitats, H_{Max} , were equal (Fig. 4b); and the maximum cost of construction, c , was near 0.5 (Fig. 4c).

To narrow focus on hard selective sweeps, we modeled rescue given a low expected cumulative mutation rate that is independent of a and K . Consequently, we did not recover the results of multiple studies (Bell and Gonzalez 2009; Lindsey et al. 2013; Martin et al. 2013; Marrec and Bitbol 2020): namely, a lower rate of wild type decline and a larger initial wild type abundance can boost P_R by increasing the number of mutational opportunities. Instead, we found that such changes can reduce the post-invasion abundance of mutants and, in turn, increase the probability of mutant extinction. Although beyond the scope of this study, extending our model to include high mutation rates could prove worthwhile by allowing one to pinpoint conditions under which post-invasion extinction risk outweighs the benefit of greater mutational input.

Furthermore, a recent simulation study by Scheiner et al. (2021) showed that, in structured populations, a phenotype performing niche construction after dispersal, as opposed to before dispersal, was always selectively favored because offspring were sure to benefit from the habitats constructed by their parents. Thus, dispersal patterns preventing inheritance of the constructed habitats by mutant offspring may decrease P_R in our model. Plasticity in the cost of niche construction would also likely affect our results: for instance, P_R would likely increase if construction was facultative such that mutants only pay the cost upon performing construction (e.g., Kümmerli and Brown 2010). All else equal, this would prevent the polymorphic oscillations by removing the wild types' competitive advantage in the constructed habitats. Note that, because we had to choose a finite time at which the rescue process completes, t_{Est} , our results pertain only to the time interval, $[0, t_{Est}]$. Greater generality could be achieved in future work by taking an approach like that of Gomulkiewicz et al. (2017), whereby the population's average time to extinction is calculated instead of the probability of post-invasion survival.

Conclusion

Analytical predictions are crucial for simplifying our understanding of the complex eco-evolutionary feedbacks that underlie evolutionary rescue. With great success in building intuition, existing analytical rescue theory has mostly focused on the initial mutant invasion process in an infinite population (e.g., Orr and Unckless 2014; Uecker et al. 2014; Czuppon et al. 2021). Here we considered both invasion and post-invasion dynamics in a finite population. We find that, without a means of limiting wild type inheritance of the constructed habitats, a population undergoing rescue via niche construction can experience damped oscillations that facilitate post-invasion extinction. More broadly, our results suggest that damped oscillatory populations lacking a mechanism to achieve a large equilibrium size or maintain a low amplitude

of oscillation can remain especially vulnerable to short-timescale extinction despite initially exhibiting the U-shaped curve characteristic of rescue.

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Appendix A. Equations for numerical analysis

(i) The probability of mutant invasion

Let $\bar{\lambda}_{Inv}(t) \approx (1 - cg) \frac{\bar{H}_{Pre}(t)}{\bar{H}_{Max}} + (1 - cg) \left(1 - \frac{\bar{H}_{Pre}(t)}{\bar{H}_{Max}}\right) g$ and $\bar{\mu}_{Inv}(t) \approx \frac{\bar{N}_{W,Pre}(t)}{K}$ respectively represent the expected per-capita birth and death rates of mutants. And let $t_1 = 10^4$ represent the length of time over which we observe the system's dynamics. Applying Uecker and Hermisson's Equations 14-15 (2011) gives

$$P_{Inv} \approx \frac{2}{1 + \int_{t=t_0}^{t_0+t_1} (\bar{\lambda}_{M,Inv}(t) + \bar{\mu}_{Inv}(t)) \exp \left[- \int_{T=t_0}^t (\bar{\lambda}_{M,Inv}(T) - \bar{\mu}_{Inv}(T)) dT \right] dt}. \quad (A1)$$

(ii) The probability of post-invasion survival

We numerically solve Equations 1a-c with initial conditions of $\bar{H}(t_0) = \bar{H}_{Pre}(t_0)$, $\bar{N}_M(t_0) = 1$, and $\bar{N}_W(t_0) = \bar{N}_{W,Pre}(t_0)$. Let $t_{Inv}^* := t_0 + \min \left[\{t \mid \bar{N}_M(t_0 + t) = 0.9\bar{N}_{M,Polym}\} \right]$. We define I_M as the set of time intervals in $(t_{Inv}^*, t_{Inv}^* + t_1]$ over which $\bar{N}_M(t_0 + t) \leq 0.9\bar{N}_{M,Polym}$. Similarly, we define I_W as the set of time intervals in $[t_0, t_0 + t_1]$ over which $\bar{N}_W(t_0 + t) \leq 0.9\bar{N}_{W,Polym}$. We assume type j organisms are vulnerable to stochastic extinction only over the time intervals in I_j . Also assumed is that type j 's extinction is certain if $0.9\bar{N}_{j,Polym} < 1$. Finally, letting $|I_j|$ represent the cardinality of I_j , we assume that mutant survival is certain if $|I_M| = 0$ and wild type extinction is inevitable if $|I_W| = 0$. Further applying Uecker and Hermisson's Equations 14-15 (2011), we approximate $P_{j,Post}$ with

$$P_{j,Post} \approx \begin{cases} 1, & 0.9\bar{N}_{j,Polym} \geq 1 \text{ and } (j = M \text{ and } |I_j| = 0) \\ \prod_{q=1}^{|I_j|} \left(1 - (1 - \rho)^{\bar{N}_j(I_{j,k,1})}\right), & 0.9\bar{N}_{j,Polym} \geq 1 \text{ and } |I_j| > 0 \\ 0, & 0.9\bar{N}_{j,Polym} < 1 \text{ or } (j = W \text{ and } |I_j| = 0) \end{cases} \quad (\text{A2})$$

$$\rho = \frac{2}{1 + \int_{t=I_{j,k,1}}^{I_{j,k,2}} (\bar{\lambda}_M(t_0 + t) + \bar{\mu}(t_0 + t)) \exp\left[-\int_{T=I_{j,k,1}}^t (\bar{\lambda}_M(t_0 + T) - \bar{\mu}(t_0 + T)) dT\right] dt},$$

where $I_{j,k,l}$ represents the l th element of the k th interval in I_j .

Appendix B. Derivation of δ

When $a \gg 0$, the expected density of wild types, $\frac{\bar{N}_{W,Pre}(t_0)}{K} \approx e^{-at_0}$, rapidly approaches zero. To capture this effect on mutant fitness during invasion, we shift time forward by δ units. We let $\delta = \min[\delta_1, \delta_2]$, where δ approaches δ_2 as a increases. Since wild type density only decreases over time intervals long enough for wild types to die, we let δ_1 equal the average time between consecutive wild type deaths, $\delta_1 = (e^{-at_0})^{-1}$. The expected growth rate of a rare mutant at time $t_0 + \delta_1$ is approximately $(1 - cg)e^{-a(t_0 + \delta_1)} + (1 - cg)(1 - e^{-a(t_0 + \delta_1)})g - e^{-a(t_0 + \delta_1)}$. To simplify this expression, we remove t_0 from δ_1 by assuming that the value of δ_1 most relevant to rescue is that corresponding to an early mutant appearance time, t_δ . Values of δ_1 corresponding to $t_\delta \gg 0$ are less important because mutants are less likely to appear at such times and, if they do appear, the death rate is low and hence less affected by the time shift. Since the focal mutant is expected to have a positive growth rate only if $t_\delta > t_M^+$, we use $t_\delta = t_M^+ + 1 \approx \frac{\ln\left(\frac{1+c-cg}{a}\right)}{a} + 1$. This gives $\delta_1 = \frac{e^a(1+c-cg)}{1-cg}$. We prevent $e^{-a(t_0 + \delta)}$ from converging to zero as a increases—and therefore retain the effect of small t_0 values on initial mutant fitness—by choosing a value of δ_2 that ensures $e^{-a(t_0 + \delta_2)}$ is of moderate distance from $e^{-a(t_0)}$. Specifically, we assume a is sizeable enough for $e^{-a(t_0 + \delta_2)}$ to reach what is roughly $e^{-a(t_0 + t)}$'s mean value, $\frac{e^{-at_0}}{2}$, between t_0 and the time of wild type extinction. We then heuristically assume, based on $e^{-a(t_0 + \delta_1)}$'s value, that $e^{-a(t_0 + \delta_2)}$ decreases as cg increases such that $\delta_2 = \left\{ \delta^* \mid e^{-a(t_0 + \delta^*)} = \frac{e^{-at_0}}{2 + \exp\left[-\frac{(1+c-cg)}{(1+cg)}\right]} \right\}$, where the $\frac{(1+c-cg)}{(1+cg)}$ term is inspired by δ_1 's value but uniquely contains a denominator of $(1 + cg)$ so that $e^{-a(t_0 + \delta_2)}$ decreases as cg increases. Simplifying δ yields

$$\delta = \min \left[\frac{e^a(1+c-cg)}{1-cg}, \frac{\ln \left[e^{at_0} \left(2 + e^{1 - \frac{2+c}{1+cg}} \right) \right] - at_0}{a} \right]. \quad (\text{B1})$$

Chapter 2

Evolutionary rescue of niche constructors from habitat exploitation

Abstract

Organisms can improve their fitness by modifying their environments—a process known as (positive) niche construction. Since niche construction is inherently costly, requiring time and energy to perform, niche constructors are vulnerable to displacement by non-niche-constructing invaders that exploit the constructed habitats. One way constructors could avoid such displacement is by adapting to withstand the invaders and thus undergoing evolutionary rescue. Here, we first analytically approximate the probability that a niche-constructing population—one constructing reproductive habitats (e.g., nests)—undergoes evolutionary rescue from habitat exploitation by an invading species. Then we evaluate the approximation under two different fitness costs of construction: a fecundity cost and a mortality cost. We find that fecundity costs are not only less harmful than mortality costs but can even promote rescue compared to no costs by reducing the rate at which constructors attempt reproduction and thus construction. The resulting lower habitat density decreases the invader growth rate, which then buys constructors more time to mutate. This benefit is stronger under a fecundity cost of niche destruction, where organisms destroy their own habitats. Our results suggest that the same fitness costs rendering constructors vulnerable to habitat exploitation can help rescue constructors from such exploitation.

1 Introduction

From naked mole-rats digging elaborate tunnel systems to microbes secreting nutrient-liberating enzymes, organisms throughout the tree of life improve their fitness by modifying their environments (Reeve, 1992; Sanchez & Gore, 2013). This process, positive niche construction (hereafter niche construction; Laland et al., 1999), is a double-edged sword. On one hand, niche construction facilitates persistence in hostile environments, such as when dung beetles protect their developing larvae from stressfully high temperatures by encasing and burying the larvae in a ball of dung (Dury et al., 2020). On the other hand, building a habitat diverts time and energy from reproduction and survival, and therefore imposes a fitness cost on the builder, rendering niche constructors vulnerable to displacement by non-niche-constructing invaders that exploit the constructed habitats (Krakauer et al., 2009). For example, in a growth medium requiring extracellular protein digestion, laboratory populations of the protease-producing pathogen, *Pseudomonas aeruginosa*, were found to go extinct in the face of non-protease-producing mutants (Lynn et al., 2024). Similarly, a field experiment showed that the cavity-excavating Eurasian nuthatch can suffer a significant population decline when limited nest site availability prompts the invasive, non-excavating rose-ringed parakeet to exploit the nuthatch's cavities (Strubbe & Matthysen, 2009). Understanding when a niche-constructing population can withstand such habitat-exploiting invaders, whether they arise through mutation or migration, can thus inform strategies in both pathogen elimination and species conservation.

While habitat-exploiting invaders can enjoy various competitive advantages over niche constructors, such as the rose-ringed parakeet's lack of natural predators (Shwartz et al., 2009) and its tendency to reduce constructor feeding rates by making constructors more vigilant (Peck et al., 2014), their fundamental advantage is that they evade niche construction's fitness cost (Wiebe et al., 2007). In some cases, this cost manifests as reduced reproductive success (i.e., fecundity and offspring viability; hereafter fecundity), lowering either the number of viable constructor offspring per reproductive attempt (e.g., fewer viable bird eggs per nest; Moreno et al., 2010) or the rate of such attempts (e.g., fewer nesting attempts per breeding season; Hauber, 2002). For instance, an experiment with *Ammophila pubescens*—a species of digger wasp in which individuals construct a separate environment for each of their offspring by first digging a burrow and then, within the burrow, provisioning the offspring with food—indicated that wasps investing the most effort into food provisioning exhibited the lowest rates of burrowing and so reproduction (Field et al., 2007). In other cases, the cost presents as a higher mortality rate, illustrated by queens of the leafcutter ant, *Atta sexdens rubropilosa*, dying more frequently with greater effort devoted to nest excavation during colony foundation

(Camargo et al., 2011). If constructors tend to destroy their habitats, causing niche destruction (Holt, 2009), they may incur an additional, destruction-induced cost (Laland et al., 1999). An experiment with Eastern phoebes, for example, showed that phoebes tasked with constructing their nests often built weak nests that collapsed during use, resulting in the loss of the builders' eggs; whereas phoebes exclusively reusing intact nests, despite reproducing more frequently, tended to avoid nest and egg loss (Hauber, 2002).

Given the inherent vulnerability of niche constructors to habitat exploitation, a key question is why niche construction is so common in nature. Although the environment alone can sometimes resolve the threat of habitat-exploiting invaders (e.g., frequent habitat loss periodically isolating niche-constructing microbes from exploitative mutants; Kümmerli & Brown, 2010), a potentially more robust solution is for a niche-constructing population to adapt to withstand the invaders (e.g., niche-constructing microbes selectively killing exploitative mutants; Huang et al., 2023). The process by which a population avoids extinction through adaptation is known as evolutionary rescue (ER; Gomulkiewicz & Holt, 1995). Characterized by a U-shaped demographic time series, ER entails a population declining toward extinction until an adaptive mutation appears and the lineage carrying the mutation establishes by overcoming drift (Orr & Unckless, 2014). A great deal of theory has been developed to understand how different ecological interactions like competition (Osmond & de Mazancourt, 2013), predation (Yamamichi & Miner, 2015; Osmond et al., 2017), and mutualism (Goldberg & Friedman, 2021) affect ER. However, such models, including those involving niche construction (Henriques & Osmond, 2020; Longcamp & Draghi, 2023), have only implicitly considered invasion as the cause of population decline. In contrast, work explicitly modeling ER from invasion has focused mostly on the ER of non-niche-constructing populations (Van Dyken, 2020). Consequently, still missing is an understanding of when niche constructors are most likely to undergo ER from habitat exploitation.

To bridge this gap, we analyze the probability, P_{ER} , that a niche-constructing population—one constructing reproductive habitats (e.g., nests)—undergoes ER from habitat exploitation by an invading species. We begin our analysis by introducing an eco-evolutionary model of niche construction and subsequently adapting the model for ER (Fig. 1). For simplicity, we assume that invaders are long-lived (e.g., due to few natural predators) enough to have a negligible death rate, ensuring that, unless ER occurs, niche constructors are certain to go extinct in the presence of invaders. Further, we assume ER happens, on average, through a single large-effect mutation (Uecker et al., 2014; Czuppon et al., 2021), as opposed to the several small-effect mutations characteristic of quantitative genetic and adaptive dynamic ER models (Bürger & Lynch, 1995; Henriques & Osmond, 2020). Given these assumptions, we analytically approximate P_{ER} ,

showing that P_{ER} increases with the initial ratio of the mutation rate to the per-capita invader growth rate (Fig. 2), with slower invasion, in particular, buying constructors more time to mutate. We then evaluate P_{ER} under two different costs of niche construction: a fecundity cost and a mortality cost. We find that ER is more likely under the fecundity cost than the mortality cost because the former not only generates less variance in mutant fitness but also—by decreasing the rate at which constructors attempt reproduction and therefore construct new habitats—slows invader growth enough to produce more mutational opportunities (Fig. 3). The latter benefit can even cause the fecundity cost to promote ER compared to no cost, given that habitat loss impedes invaders enough to compensate for the cost’s reduction in the density of potentially mutating constructors (Fig. 4). We also consider a fecundity cost of niche destruction and find that, by being tied to the elimination of both new and pre-existing habitats available to invaders, such a cost promotes ER more effectively than the fecundity cost of construction (Fig. 5). If constructors are too short-lived, however, fecundity costs hinder ER (Fig. 6), because the benefit of more mutational opportunities is outweighed by a slimmer chance of mutant establishment.

2 Model

In this section, we introduce an eco-evolutionary model of niche construction and then adapt the model for ER. For generality, we introduce the model without explicit fitness costs of niche construction and destruction and only later evaluate key parameters as functions of costs. The model is summarized in the main text for readability, with additional details given in Appendix A. Parameters and variables frequently mentioned in the main text are listed in Table 1.

2.1 Eco-evolutionary model

We model an ecosystem that changes according to a continuous-time birth-death process (Kendall, 1948). The ecosystem contains a reproductive environment frequented by a well-mixed haploid community. In the former, there are $K > 0$ patches that each contain either habitat 1 or habitat 2. Reproduction can only occur in patches of habitat 1. The abundance (i.e., number) of habitat 1 patches, $0 \leq H(t) \leq K$, decays at an organism-independent, per-patch rate of $a > 0$. The community is composed of three types of organisms: residents, invaders, and mutants, which have abundances of $N_R(t) \geq 0$, $N_I(t) \geq 0$, and $N_M(t) \geq 0$, respectively. Residents can perform niche construction, whereby an organism facilitates its own reproduction by converting a patch of habitat 2 into a patch of habitat 1. Invaders are non-niche-constructing organisms that

exploit the habitats constructed by residents, causing residents to decline toward extinction. Mutants are descendants of residents that can not only niche construct but are also unaffected by invaders.

2.1.1 Resident dynamics

Residents have a carrying capacity of K . We denote the density of each variable, $V(t) \in \{H(t), N_R(t), N_I(t), N_M(t)\}$, with respect to K as $v(t) = \frac{V(t)}{K}$. A resident birth event begins with a resident dispersing into the reproductive environment. The individual then settles in a randomly chosen patch of habitat 1 with a probability of $h(t)$ or habitat 2 with a probability of $1 - h(t)$. In the latter case, niche construction is performed. After performing construction if necessary, the resident can reproduce. If reproduction is successful, the newborn offspring starts developing into an adult. The activities of the parent and developing offspring can destroy the reproductive habitat through niche destruction. Following development, the parent and offspring enter the adult community, concluding the birth event. Thus, a birth attempt entails dispersal to a habitat \rightarrow construction if necessary \rightarrow possible reproduction \rightarrow possible destruction. The per-capita rate at which residents disperse to habitat 2 and successfully construct a new habitat (i.e., perform construction and avoid destruction) is $(1 - h(t))g^+$, where $g^+ > 0$ is the per-capita rate of successful construction (hereafter construction) conditional on settling in habitat 2. Conversely, the per-capita rate at which residents disperse to habitat 1 and induce destruction is $h(t)g^-$, where $g^- \geq 0$ is the per-capita rate of destruction conditional on settling in habitat 1. Residents give birth, on average, at a per-capita rate of $b > 0$. Meanwhile, residents die at a per-capita rate of $d + \delta(n_R(t) + n_I(t) + n_M(t))$, where $0 \leq d < b$ is the per-capita density independent death rate and $\delta \geq b - d$ is the per-interaction density dependent death rate. The constraint $\delta \geq b - d$ ensures that, on average, $N_R(t) \leq K$.

2.1.2 Invader dynamics

Invaders can only reproduce if they disperse to habitat 1. For simplicity, we assume a worst-case scenario for residents in which (i) invaders avoid niche destruction and thus avoid reducing their own fitness; and (ii) invaders are long-lived in the absence of competition, and have a sizeable carrying capacity, such that their death rate is negligible. Further assumed is that the rate of invader appearance is low enough to have a negligible effect on the invader growth rate. Given these assumptions, we let the per-capita invader growth rate equal $h(t)\Psi$, where $\Psi > 0$ is the maximum per-capita invader growth rate. The absence of invader mortality in this growth rate not only reduces parameter space but also prevents invader extinction, meaning that, similar to Van Dyken's (2020) model of ER from invasion, residents are certain to go extinct

in the presence of invaders.

2.1.3 Mutant dynamics

Residents mutate at a per-capita rate of $u > 0$. Back mutations are ignored. Mutations are assumed rare enough to have a negligible effect on resident dynamics. Mutants are identical to residents in terms of both fitness and effect on habitat density, except mutants are unaffected by the density of invaders and thus have a per-capita death rate of $d + \delta (n_R(t) + n_M(t))$. Despite being immune to invaders, however, mutants do not affect invader growth.

2.1.4 Expected ecosystem dynamics

In Appendix A (Table A1), we provide a detailed list of the different events that can occur during a given infinitesimal time interval, $(t, t + dt)$, in our model, as well as their probabilities of occurrence and the state change in each variable, $V(t)$, that they cause. Using this information, we calculate the expected change in $V(t)$ during $(t, t + dt)$ by first multiplying each of $V(t)$'s possible state changes by the probability of such a change occurring, then summing over those products, yielding

$$\begin{aligned}
\frac{d\bar{H}(t)}{dt} &= (\bar{N}_R(t) + \bar{N}_M(t)) [(1 - \bar{h}(t)) g^+ - \bar{h}(t)g^-] - \bar{H}(t)a \\
\frac{d\bar{N}_R(t)}{dt} &= \bar{N}_R(t)b - \bar{N}_R(t) [d + \delta (\bar{n}_R(t) + \bar{n}_I(t) + \bar{n}_M(t))] \\
\frac{d\bar{N}_I(t)}{dt} &= \bar{N}_I(t)\bar{h}(t)\Psi \\
\frac{d\bar{N}_M(t)}{dt} &= \bar{N}_R(t)u + \bar{N}_M(t)b - \bar{N}_M(t) [d + \delta (\bar{n}_R(t) + \bar{n}_M(t))],
\end{aligned} \tag{1a-d}$$

where $\bar{V}(t) = K\bar{v}(t)$ is the expected value, as opposed to the realized value, of $V(t) = Kv(t)$. The solution to Eq. 1 gives $V(t)$'s average value across an infinite number of replicate realizations and thus gives $V(t)$ when demographic variance vanishes (Czuppon & Traulsen, 2021). Letting $\bar{N}_I(t) = \bar{N}_M(t) = 0$, then solving $\frac{d\bar{H}(t)}{dt} = 0$ and $\frac{d\bar{N}_R(t)}{dt} = 0$ for $\bar{H}(t)$ and $\bar{N}_R(t)$, reveals that Eq. 1 exhibits an equilibrium at which habitat 1 and residents have respective abundances of

$$\begin{aligned}
\bar{H} &= K\bar{h} = \frac{K(b-d)g^+}{(b-d)(g^+ + g^-) + a\delta} \\
\bar{N}_R &= K\bar{n}_R = \frac{K(b-d)}{\delta},
\end{aligned} \tag{2a-b}$$

where $\bar{V} = K\bar{v}$ represents the value of $\bar{V}(t)$ at equilibrium. Eqs. 2a-b are positive, and the equilibrium is

locally stable, throughout parameter space (see Appendix B).

Table 1: Model parameters and variables.

Notation	Interpretation
a	Per-patch rate of organism-independent habitat loss
b	Expected per-capita resident birth rate
c^+	Fitness cost of niche construction
c^-	Fitness cost of niche destruction
d	Per-capita resident density independent death rate
δ	Per-interaction resident density dependent death rate
g^+	Per-capita rate of niche construction conditional on settling in habitat 2
g^-	Per-capita rate of niche destruction conditional on settling in habitat 1
K	Resident carrying capacity
Ψ	Maximum per-capita invader growth rate
u	Per-capita mutation rate
U	$U = Ku$: Maximum initial mutation rate
$H(t)$	Abundance of habitat 1 patches
$N_R(t)$	Abundance of residents
$N_I(t)$	Abundance of invaders
$N_M(t)$	Abundance of mutants
$v(t)$	Density of variable $V(t) \in \{H(t), N_R(t), N_I(t), N_M(t)\}$ with respect to K
$\bar{V}(t)$	$\bar{V}(t) = K\bar{v}(t)$: Expected value of variable $V(t)$
\bar{V}	$\bar{V} = K\bar{v}$: Equilibrium value of variable $\bar{V}(t)$

2.2 Evolutionary rescue

Fig. 1 illustrates the dynamics of our ER model. At times $t < 0$, invaders and mutants are, for simplicity, assumed absent from the community. In contrast, we assume $K \gg 1$ such that residents and habitat 1 have abundances of $\lfloor \bar{N}_R \rfloor \gg 1$ and $\lfloor \bar{H} \rfloor \gg 1$, respectively, where $\lfloor \bar{V}(t) \rfloor$ is the floor of $\bar{V}(t)$. At time $t = 0$, an invader appears (i.e., $N_I(0) = 1$), causing niche constructors and habitat 1 to decline from $N_R(0) = \lfloor \bar{N}_R \rfloor$, $N_M(0) = 0$, and $H(0) = \lfloor \bar{H} \rfloor$ toward extinction. Since ER is nearly certain under sufficiently high mutation rates in models like ours (e.g., Wilson et al., 2017; Marrec & Bitbol, 2020), we follow previous ER theory (e.g., Czuppon et al., 2021) by letting $u = \frac{U}{K} < 1$. This ensures that the mutation rate, $N_R(t)u = n_R(t)U$, has a maximum initial value of $0 < U < 1$ and is consequently, on average, less than one. If such a mutation appears, it does so at time $t_M \geq 0$, where t_M is defined as any time at which a mutation causes $N_M(t) > 0$. Mutants are considered established—and so ER occurs—only if $N_M(t) \geq \lfloor 0.95\bar{N}_R \rfloor$.

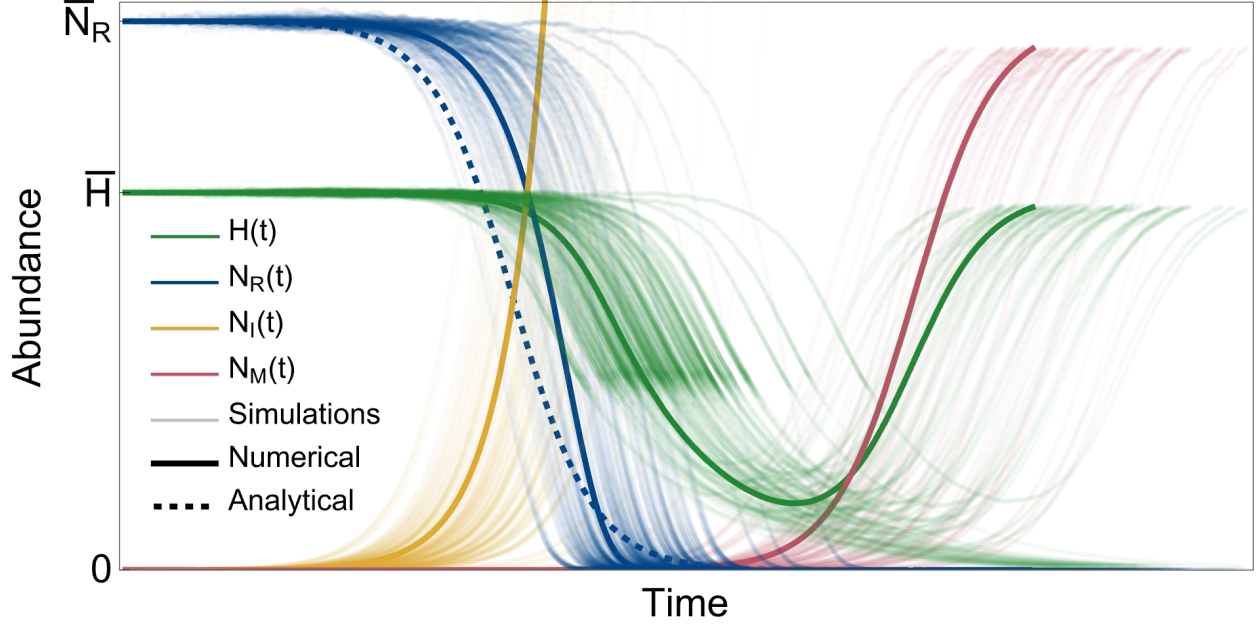


Figure 1: Stochastic and expected dynamics of our evolutionary rescue model. Residents, $N_R(t)$, and habitat 1, $H(t)$, are initially at their respective expected equilibria, $[\bar{N}_R]$ and $[\bar{H}]$. Invaders, $N_I(t)$, grow in abundance as they exploit the habitats constructed by residents, causing residents and habitat 1 to decline toward extinction. Prior to resident extinction, mutants, $N_M(t)$, that are immune to invaders can appear. Rescue occurs if $N_M(t) \geq [0.95\bar{N}_R]$. If rescue does not occur, all habitat 1 patches are lost. Thin transparent curves: 100 replicate simulations of the algorithm described in Section 3.1.2 (except with each simulation proceeding until $N_M(t) = 0$ and $H(t) = [0.01\bar{h}]$ or $N_M(t) \geq [0.95\bar{N}_R]$); thick solid curves: numerical solution to Eq. 1 (calculated as described in Section 3.1.2, except $u > 0$); thick dashed curve: Eq. 4. Parameter values are $\Psi = 1.45$, $a = 0.35$, $b = g^+ = \delta = 1$, $d = 0.04$, $g^- = 0.15$, $K = 3(10)^4$, and $U = 0.3$.

3 Methods

3.1 Mathematical predictions

We use time-inhomogeneous branching process theory (Uecker & Hermisson, 2011) to analytically and numerically predict the probability of ER, P_{ER} . Resident and invader carrying capacities are assumed large so that ecosystem dynamics are well-approximated by Eq. 1. Further assumed is that, during the period of time relevant to mutants escaping drift, mutant density is low enough to have a negligible effect on ecosystem dynamics such that the per-capita mutant birth and death rates are b and $d + \delta(\bar{n}_R(t) + \bar{n}_M(t)) \approx d + \delta\bar{n}_R(t)$, respectively. Following Uecker and Hermisson (2011; Eqs. A7 and 16 therein), P_{ER} can then be written as

$$\begin{aligned}
P_{ER} &\approx 1 - \exp\left(-\int_{t_M=0}^{\infty} \bar{n}_R(t_M)U P_{Est}(t_M) dt_M\right) \\
P_{Est}(t_M) &\approx \frac{2}{1 + \int_{t=t_M}^{\infty} \sigma^2(t) \exp\left(-\int_{T=t_M}^t (b-d-\delta\bar{n}_R(T)) dT\right) dt} \\
\sigma^2(t) &= b + d + \delta\bar{n}_R(t),
\end{aligned} \tag{3a-c}$$

where $\bar{n}_R(t_M)U$ is the rate at which mutations appear at time t_M ; $P_{Est}(t_M)$ is the probability of mutant establishment; and $\sigma^2(t)$ is the infinitesimal variance in mutant fitness at time $t \geq t_M$. A major difference between our analytical and numerical approximations of Eq. 3 is in how we calculate $\bar{n}_R(t)$ and therefore $\bar{N}_R(t)$.

3.1.1 Analytical predictions

For our forthcoming analytical approximation of Eq. 3, we approximate $\bar{N}_R(t)$ heuristically: we assume that the invader and resident growth rates are proportional with opposite signs (i. e., $\frac{d\bar{N}_R(t)}{dt} \propto -\frac{d\bar{N}_I(t)}{dt}$) such that, when invaders are rare (i. e., $\bar{N}_I(t) \approx \frac{d\bar{N}_I(t)}{dt} \approx 0$ but $\bar{N}_I(t) \neq 0$), residents and habitat 1 have effectively constant abundances of $\bar{N}_R(t) \approx \bar{N}_R$ and $\bar{H}(t) \approx \bar{H}$, respectively. Given these constant abundances, the invader trajectory can be approximated by solving Eq. 1c with $\bar{N}_I(0) = 1$, yielding $\bar{N}_I(t) \approx \exp(\bar{h}\Psi t)$. Since this trajectory is exponential—and therefore entails an initial phase of negligible growth—we assume residents decline approximately via a logistic function, remaining near their initial abundance until declining due to invader prevalence. In particular, we assume $\bar{N}_R(t) \approx \bar{N}_R [1 + \exp(-r(t_{1/2} - t))]^{-1}$, where $r > 0$ is the per-capita rate of resident decline and $t_{1/2} > 0$ is the resident half-life. To acquire r and $t_{1/2}$, we first assume that, as residents decline due to $\bar{N}_I(t) \gg 1$, they perform niche construction frequently (and niche destruction infrequently) enough for $\bar{h}(t) \approx \bar{h}$. Then we assume that the per-capita invader growth rate, $\bar{h}\Psi$, is sufficiently small (i. e., $\bar{h}\Psi \approx 0$ but $\bar{h}\Psi \neq 0$) such that, when multiplied by a given quantity, y , the product is effectively $y\bar{h}\Psi \approx \bar{h}\Psi$. Finally, we assume $\frac{d\bar{N}_R(t)}{dt} \approx -\frac{d\bar{N}_I(t)}{dt} = -\bar{N}_I(t)\bar{h}\Psi$ so that $\bar{h}\Psi$ dictates the rate of resident decline via $r \approx \frac{\bar{N}_I(t)}{\bar{N}_R(t)}\bar{h}\Psi \approx \bar{h}\Psi$; and, because $\frac{d\bar{N}_R(t)}{dt} + \frac{d\bar{N}_I(t)}{dt} \approx 0$, the community maintains a constant size of $\bar{N}_I(0) + \bar{N}_R(0) = \bar{N}_R + 1 \approx \bar{N}_R$, meaning $t_{1/2} \approx \{t \mid \exp(\bar{h}\Psi t) = \frac{\bar{N}_R}{2}\}$. The resulting logistic function, which is plotted in Figure 1, is

$$\begin{aligned}\bar{N}_R(t) &\approx \frac{\bar{N}_R}{1 + \exp(-\bar{h}\Psi(t_{1/2} - t))} \\ t_{1/2} &\approx \ln\left(\frac{\bar{N}_R}{2}\right) (\bar{h}\Psi)^{-1}.\end{aligned}\tag{4a-b}$$

3.1.2 Numerical predictions

For our numerical approximation of Eq. 3, we acquire $\bar{N}_R(t)$ by solving Eq. 1 numerically with $u = 0$ and initial conditions of $\bar{H}(0) = \lfloor \bar{H} \rfloor$, $\bar{N}_R(0) = \lfloor \bar{N}_R \rfloor$, $\bar{N}_I(0) = 1$, and $\bar{N}_M(0) = 0$. To prevent integer overflow, we let $\frac{d\bar{N}_I(t)}{dt} = 0$ when $\bar{N}_I(t) \geq 5K$. The upper bounds of ∞ in Eqs. 3a and 3b are replaced with the time at which $\bar{N}_R(t) = 1$ and the time, $t_M + 10^5$, respectively.

3.2 Simulations

We test our mathematical predictions with simulations of a tau-leaping algorithm (Cao et al., 2006). A simulation is initialized with $H(0) = \lfloor \bar{H} \rfloor$, $N_R(0) = \lfloor \bar{N}_R \rfloor$, $N_I(0) = 1$, and $N_M(0) = 0$. Each variable, $V(t)$, then changes according to its corresponding events, state changes, and transition rates (i.e., probabilities divided by dt) listed in Table A1 of Appendix A. The value of each tau-leap, and the number of times each event occurs during the leap, are calculated using the procedure summarized in Sections IIC (excluding Step 3) and IV of Cao et al. (2006). As implemented, this procedure ensures that, if $V(t) < n_c = 10$, then all transition rates corresponding to a non-zero state change in $V(t)$ are updated according to a nearly exact version of the Gillespie algorithm (Gillespie, 1976). Further, the algorithm helps prevent $V(t)$ from becoming negative during a leap by approximately bounding the per-leap change in each of $V(t)$'s transition rates, relative to the sum of all transition rates, by a value of $\epsilon = 0.03$. If $V(t)$ becomes negative despite this precaution, we set $V(t) = 0$. We also prevent a negative value of the term $1 - h(t)$ (which appears in certain transition rates; see Appendix A) by setting $H(t) = K$ if $H(t) > K$. We avoid an undefined leap size by adding 10^{-9} to the output of Cao et al.'s (2006) Eq. 32a if the output is zero; and the same adjustment is made to Eq. 32b if its output is zero. The simulation proceeds until either $N_R(t) = N_M(t) = 0$ or $N_M(t) \geq \lfloor 0.95\bar{N}_R \rfloor$. All numerical calculations and simulations are performed using Mathematica 13.2.0.0.

4 Results

In this section, we first analytically approximate P_{ER} . Then we study the effect of fitness costs on P_{ER} . Any assumptions made henceforth pertain only to our analytical results. All analytical approximations are made using Eq. 4 for resident density, $\bar{n}_R(t)$. For brevity we use the term ‘rate’ when referring to any conditional, expected, or per- x (e.g., $x = \text{capita}$) rate in Table 1.

4.1 The probability of evolutionary rescue

To begin our analytical approximation of P_{ER} , we approximate the probability that a mutant lineage appearing at time t_M establishes by overcoming drift, $P_{Est}(t_M)$. For simplicity, we assume that the invader growth rate, $\bar{h}(t)\Psi$ —and so the rate of resident decline—is small enough for mutants to maintain an effectively constant death rate of $d + \delta\bar{n}_R(t_M)$ throughout establishment. Given this constant death rate, the solution to the $P_{Est}(t_M)$ equation, Eq. 3b, is

$$P_{Est}(t_M) \approx 1 - \frac{d}{b} - \frac{\bar{n}_R \bar{N}_R \delta}{b [2 \exp(\bar{h}\Psi t_M) + \bar{N}_R]}. \quad (5)$$

Eq. 5 shows that, at least approximately speaking (assumed hereafter), $P_{Est}(t_M)$ is reduced by the stronger intraspecific, mutant-resident competition arising from a lower initial invader growth rate, $\bar{h}\Psi$, or a higher initial resident abundance, \bar{N}_R , and therefore density, \bar{n}_R . Even when the effect of density dependent competition on mutant fitness is negligible, however, mutants can still go extinct with a probability of $\frac{d}{b}$. So, all else equal (also assumed hereafter), raising the resident birth rate, b , or lowering the resident density independent death rate, d , always enhances $P_{Est}(t_M)$ by increasing the mutant density independent growth rate, $b - d$. To complete our approximation of P_{ER} , we first solve the P_{ER} equation, Eq. 3a, using Eq. 5 for $P_{Est}(t_M)$; then we simplify the resulting solution by taking its limit as $K \rightarrow \infty$ (see Appendix C), yielding

$$P_{ER} \approx 1 - \exp\left(-\frac{U\bar{n}_R}{\bar{h}\Psi} \left(1 - \frac{d}{b}\right)\right). \quad (6)$$

Eq. 6 reveals that P_{ER} increases with the initial ratio of the mutation rate to the invader growth rate, $U\bar{n}_R(\bar{h}\Psi)^{-1}$. To see why, we can approximate the total number of mutations appearing before resident extinction, $U_{Total} \approx \int_{t_M=0}^{\infty} \bar{n}_R(t_M) U dt_M$, which, if we let K remain finite, is

$$U_{Total} \approx \frac{U\bar{n}_R}{\bar{h}\Psi} \ln\left(1 + \frac{\bar{N}_R}{2}\right). \quad (7)$$

Eq. 7's right-hand side grows with $U\bar{n}_R (\bar{h}\Psi)^{-1}$ and an additional, logarithmic \bar{N}_R term. Thus, although reducing $\bar{h}\Psi$ or increasing \bar{N}_R can decrease $P_{Est}(t_M)$, such changes boost P_{ER} by increasing the total number of mutations, U_{Total} . Specifically, smaller $\bar{h}\Psi$ values raise U_{Total} by prolonging the time until invaders displace half of the resident population (i.e., the resident half-life; Eq. 4b), while larger \bar{N}_R values increase both the resident half-life and the initial mutation rate, $U\bar{n}_R$. A lower maximum invader growth rate, Ψ , can therefore promote ER (Fig. 2A) by reducing $\bar{h}\Psi$, as can the smaller initial habitat 1 density, \bar{h} , resulting from a lower rate of niche construction, g^+ (Fig. 2B), or higher rates of niche destruction, g^- (Fig. 2C), or organism-independent habitat loss, a (Fig. 2D). Raising b or lowering d further boosts P_{ER} (Figs. 2E-F) by not only increasing $U\bar{n}_R$ like a reduction in the resident density dependent death rate, δ (Fig. 2G), or an increase in the maximum initial mutation rate, U (Fig. 2H); but also augmenting the cumulative probability of mutant establishment across all possible mutant appearance times. As shown in Eq. 6, the latter is equal to the density independent probability of mutant establishment, $1 - \frac{d}{b}$, because, under our large- K assumption, \bar{N}_R 's increase in the resident half-life and reduction in $P_{Est}(t_M)$ counterbalance each other. Although Eq. 6 accurately predicts these different qualitative results, it loses quantitative accuracy under sufficiently high Ψ or g^- values (Fig. 2). Large Ψ values contradict our assumption of a small $\bar{h}\Psi$ value, while large g^- values invalidate our assumption in Section 3.1.1 that habitat 1's density remains constant throughout the resident population's decline.

4.2 The effect of fitness costs on evolutionary rescue

Table 2: Fecundity and mortality fitness cost regimes.

Regime	b	g^+	g^-	d	δ
Fecundity cost	$(1 - c^-)(1 - c^+)$	$(1 - c^-)(1 - c^+)$	$c^-(1 - c^+)$	d	1
Mortality cost	$1 - c^-$	$1 - c^-$	c^-	$(1 - c^-)c^+$	1

We now analyze the effect of fitness costs on ER. To do this, we first reduce parameter space by assuming that (i) the resident density dependent death rate is $\delta = 1$; and (ii) when fitness costs are absent, the rates of resident birth, b , niche construction, g^+ , and niche destruction, g^- , have values of $b = 1$, $g^+ = 1$, and $g^- = 0$. We then evaluate P_{ER} under the two fitness cost regimes given in Table 2. The fecundity cost of niche destruction, $0 \leq c^- < 1$, is consistent across both regimes: with a probability of c^- , a reproducing resident induces destruction, causing the resident's developing offspring to die. In contrast, the regimes differ with respect to the cost of niche construction, $0 \leq c^+ < 1$: the 'fecundity cost regime' includes a fecundity cost that decreases the rate of resident dispersal into the reproductive environment, but does not

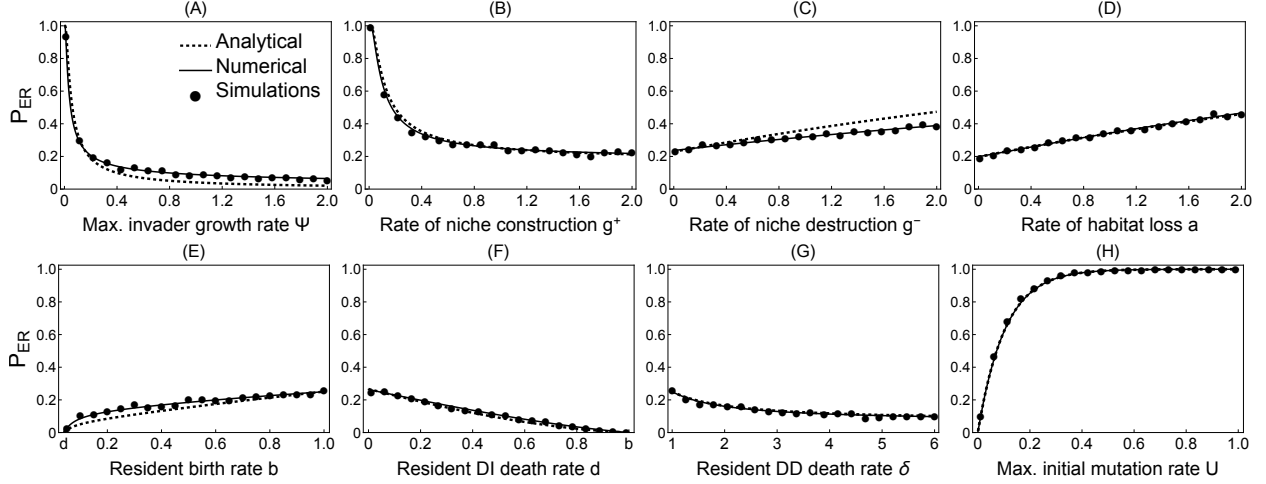


Figure 2: The probability of evolutionary rescue, P_{ER} , as a function of different parameters. P_{ER} grows with the initial ratio of the mutation rate to the invader growth rate due to greater mutational input. So, through a decrease in the initial invader growth rate, ER is promoted by (A) a smaller maximum invader growth rate, Ψ ; (B) a lower rate of niche construction, g^+ ; (C) a higher rate of niche destruction, g^- ; or (D) a greater rate of organism-independent habitat loss, a . Moreover, through a boost in the initial mutation rate, ER is promoted by (E) a larger resident birth rate, b ; (F) a smaller resident density independent death rate, d ; (G) a lower resident density dependent death rate, δ , or (H) a higher maximum mutation rate, U . Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: numerical approximations of Eq. 3; dashed curves: Eq. 6. Unless stated otherwise, parameter values are $\Psi = 0.145$, $a = 0.35$, $b = g^+ = \delta = 1$, $d = 0.04$, $g^- = 0.15$, $K = 3(10)^4$, and $U = 0.03$.

affect mortality, so that $b = g^+ = (1 - c^+)(1 - c^-)$, $g^- = (1 - c^+)c^-$, $\delta = 1$; and, for generality, the resident density independent death rate, $0 \leq d < b$, remains arbitrary. The ‘mortality cost regime,’ on the other hand, entails a mortality cost that increases the resident death rate via $d = bc^+ = (1 - c^-)c^+ < b$ (where multiplying c^+ by b ensures $d < b$), but does not affect reproduction, meaning $b = g^+ = 1 - c^-$, $g^- = c^-$, and $\delta = 1$. Plugging each regime into Eq. 6 gives the probability of ER under the fecundity cost regime, $P_{ER,f}$, and the mortality cost regime, $P_{ER,m}$:

$$\begin{aligned}
 P_{ER,f} &\approx 1 - \exp\left(-U \frac{[(1 - c^+)(1 - c^-) - d] \{a + (1 - c^+) [(1 - c^+)(1 - c^-) - d]\}}{\Psi (1 - c^+)^2 (1 - c^-)^2}\right) \\
 P_{ER,m} &\approx 1 - \exp\left(-U \frac{(1 - c^+) [a + (1 - c^+) (1 - c^-)]}{\Psi (1 - c^-)}\right).
 \end{aligned} \tag{9a-b}$$

To ensure a fair comparison of Eqs. 9a-b, we assume in Sections 4.2.1-4.2.2 that, under the fecundity cost regime, $d = 0$ such that both regimes yield the same initial resident density, $\bar{n}_R = b - d = (1 - c^+) (1 - c^-)$. We then let $d > 0$ in Section 4.2.3 to understand how mutant mortality affects $P_{ER,f}$.

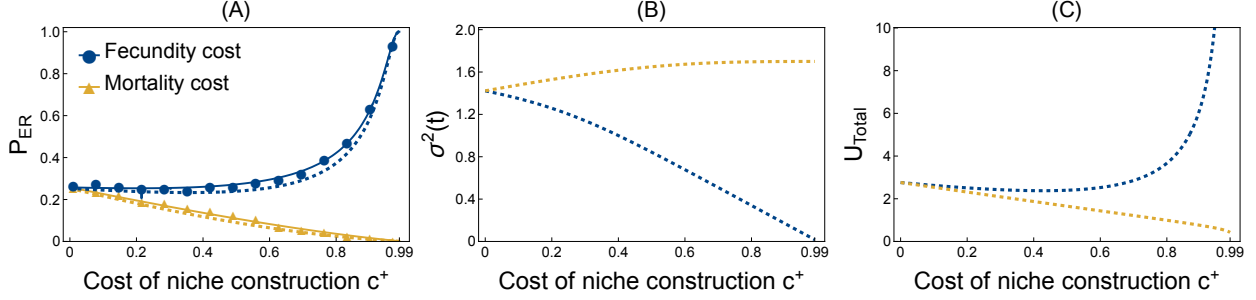


Figure 3: The probability of evolutionary rescue, P_{ER} ; the variance in mutant fitness, $\sigma^2(t)$; and the total number of mutations, U_{Total} , as functions of the cost of niche construction, c^+ . (A) Rescue is more likely under a fecundity cost than a mortality cost. This is because the fecundity cost generates (B) less mutant variance and (C) more mutations than the mortality cost. The lower mutant variance is a result of the fecundity cost reducing the total probability of a mutant event (birth or death). In contrast, the greater mutational input is due to the fecundity cost decreasing the rate of niche construction and therefore the invader growth rate. The latter benefit can be enough for P_{ER} to increase with c^+ . Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: numerical approximations of Eq. 3 evaluated under the two fitness cost regimes in Table 2; dashed curves: Eq. 9 and Eqs. 3c (evaluated with Eq. 4) and 7 evaluated under the two fitness cost regimes in Table 2. Unless stated otherwise, parameter values are $\Psi = 0.145$, $a = 0.35$, $c^- = 0.15$, $d = 0$, $K = 3(10)^4$, $t = 100$, and $U = 0.03$.

4.2.1 Evolutionary rescue is more likely under fecundity costs than mortality costs

Letting $d = 0$ in the fecundity cost regime and subsequently comparing Eqs. 9a-b reveals that ER is more likely if the cost of niche construction reduces fecundity instead of promoting mortality (Fig. 3A). That is, $P_{ER,f} \gtrsim P_{ER,m}$ (where the \sim symbol stems from Eq. 9's inexactness and is henceforth omitted from inequalities). The reasons for this are two-fold: first, by augmenting the mutant death rate and thus increasing the probability that a mutant undergoes an event (birth or death), the mortality cost generates greater variance in mutant fitness, $\sigma^2(t)$ (Eq. 3c), making mutants more susceptible to drift (Fig. 3B). For example, the initial mutant variance induced by the mortality cost is $\lim_{K \rightarrow \infty} \sigma^2(t_M) = \bar{n}_R + (1 - c^-)(1 + c^+)$, whereas that induced by the fecundity cost is $\bar{n}_R + (1 - c^-)(1 - c^+)$. Second, although the fecundity and mortality costs yield the same initial resident density, $\bar{n}_R = (1 - c^+)(1 - c^-)$, the fecundity cost generates the larger number of mutations, U_{Total} (Fig. 3C), by shrinking the rate of construction, g^+ . The lower g^+ value reduces the initial density of habitat 1, \bar{h} , and so the initial invader growth rate, $\bar{h}\Psi$.

4.2.2 Fecundity costs can promote evolutionary rescue compared to no costs

Interestingly, the benefit of a higher U_{Total} value stemming from the fecundity cost of niche construction can be enough for the fecundity cost to promote ER compared to no cost (Fig. 3A). Niche constructors can therefore indirectly facilitate ER by paying a fecundity cost that directly limits their rate of reproductive attempts: fewer attempts leads to less construction, which can in turn slow invader growth (and hence

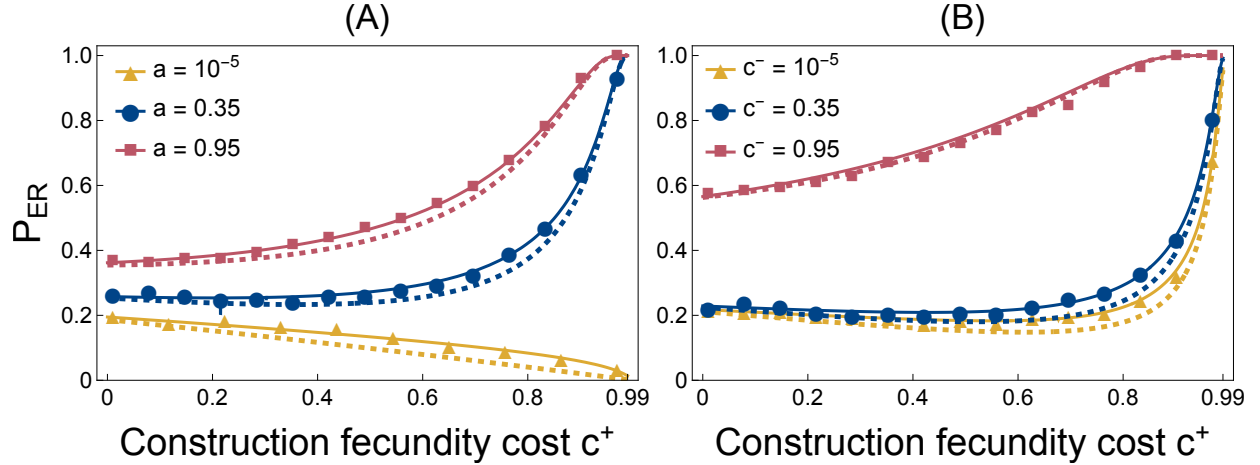


Figure 4: The probability of evolutionary rescue, P_{ER} , as a function of the fecundity cost of niche construction, c^+ . The range of c^+ values promoting rescue increases with (A) the rate of organism-independent habitat loss, a , and (B) the fecundity cost of niche destruction, c^- . Sufficiently large a and c^- values slow invader growth enough to offset the reduction in resident density caused by an increase in the fecundity cost of construction, which then allows the fecundity cost of construction to increase P_{ER} by lowering habitat 1's density and hence the invader growth rate. Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: numerical approximations of Eq. 3 evaluated under the fecundity cost regime in Table 2; dashed curves: Eq. 9a. Unless stated otherwise, parameter values are $\Psi = 0.145$, $a = c^- = 0.15$, $d = 0$, $K = 3(10)^4$, and $U = 0.03$.

slow resident decline) enough to increase mutational input. In such a case, P_{ER} increases despite the fecundity cost reducing resident density. If c^+ becomes too small, however, the reduction in resident density outweighs the benefit of slower invader growth, preventing P_{ER} from increasing (Fig. 3A). Specifically, simplifying $\frac{\partial P_{ER,f}}{\partial c^+} > 0$ reveals that ER is promoted only by c^+ values satisfying

$$c^+ > 1 - \sqrt{\frac{a}{1 - c^-}}. \quad (10)$$

Thus, the range of fecundity costs of niche construction promoting ER expands with an increase in the rate of organism-independent habitat loss, a , or the fecundity cost of niche destruction, c^- (Fig. 4). This is because rapid enough habitat loss—by lessening habitat 1's density and therefore the invader growth rate—compensates for the decrease in resident density caused by larger c^+ values, allowing such values to boost U_{Total} by further diminishing habitat 1's density. If habitat loss is too rare, P_{ER} declines with an increase in c^+ (Fig. 4A). Particularly, rearranging Eq. 10 shows that P_{ER} increases with c^+ only when

$$a > (1 - c^+)^2 (1 - c^-). \quad (11)$$

The benefit of niche destruction therefore also disappears with a , because, when $a \approx 0$, the decrease in

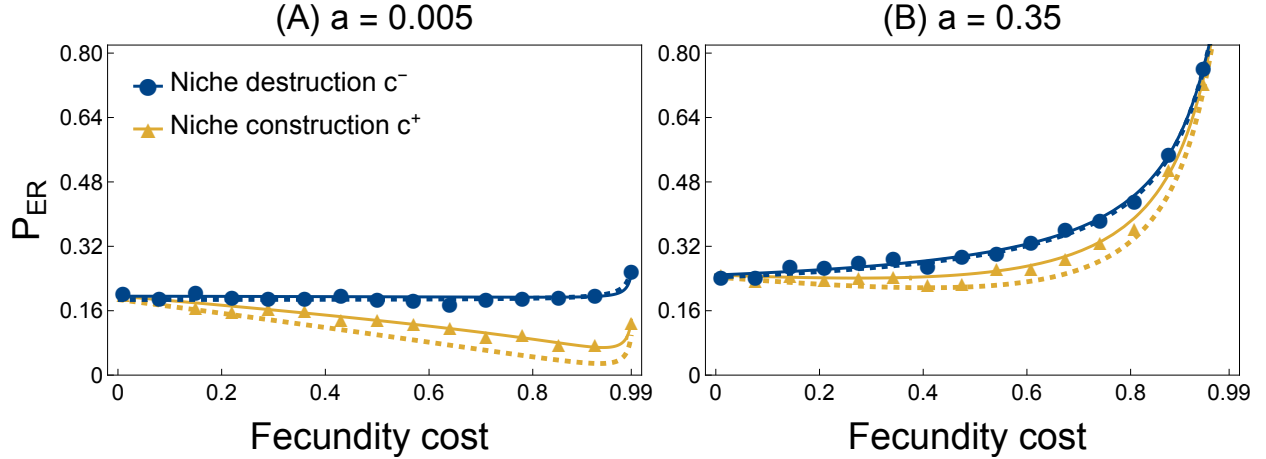


Figure 5: The probability of evolutionary rescue, P_{ER} , as a function of the fecundity costs of niche construction, c^+ , and niche destruction, c^- . (A) The fecundity cost of destruction yields a higher P_{ER} value than that of construction, because destruction can eliminate both pre-existing and freshly constructed habitats, while the cost of construction merely reduces the rate of construction. (B) The advantage of destruction becomes less pronounced with an increase in the rate of organism-independent habitat loss, a , due to pre-existing habitats being destroyed before they can undergo destruction. Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: numerical approximations of Eq. 3 evaluated under the fecundity cost regime in Table 2; dashed curves: Eq. 9a. Unless stated otherwise, parameter values are $\Psi = 0.145$, $c^+ = c^- = 0$, $d = 0$, $K = 3(10)^4$, and $U = 0.03$.

resident density caused by destruction is not compensated for by the lower invader growth rate resulting from organism-independent habitat loss. So when a is small, destruction must occur relatively often to generate the habitat loss needed to appreciably increase P_{ER} (Fig. 4B). If a and c^- sum to an adequately high rate of habitat loss, however, destruction promotes ER more than organism-independent habitat loss (Fig. 4). With the latter compensating for destruction's decrease in resident density, destruction prevails because it not only increases g^- —thereby destroying pre-existing habitats like organism-independent habitat loss—but also reduces g^+ by destroying freshly constructed habitats. We find that this dual benefit is enough for the fecundity cost of destruction to always promote ER more effectively than that of niche construction (Fig. 5A) because the latter only reduces g^+ (i. e., $\left. \frac{\partial P_{ER,f}}{\partial c^-} \right|_{c^+=0} > \left. \frac{\partial P_{ER,f}}{\partial c^+} \right|_{c^-=0}$). However, destruction's advantage over the fecundity cost of construction decreases with an increase in a (Fig. 5B), as large a values leave few pre-existing habitats available for destruction.

4.2.3 Long-lived niche-constructing species most readily benefit from fecundity costs

We now let $d > 0$ and, unless stated otherwise, narrow focus on the fecundity cost of niche destruction by letting $c^+ = 0$. We then simplify $\frac{\partial P_{ER,f}}{\partial c^-} > 0$, which reveals that P_{ER} grows with c^- only when

$$d < \frac{1 + a - c^- - \sqrt{a^2 + (1 - c^-)^2}}{2}. \quad (12)$$

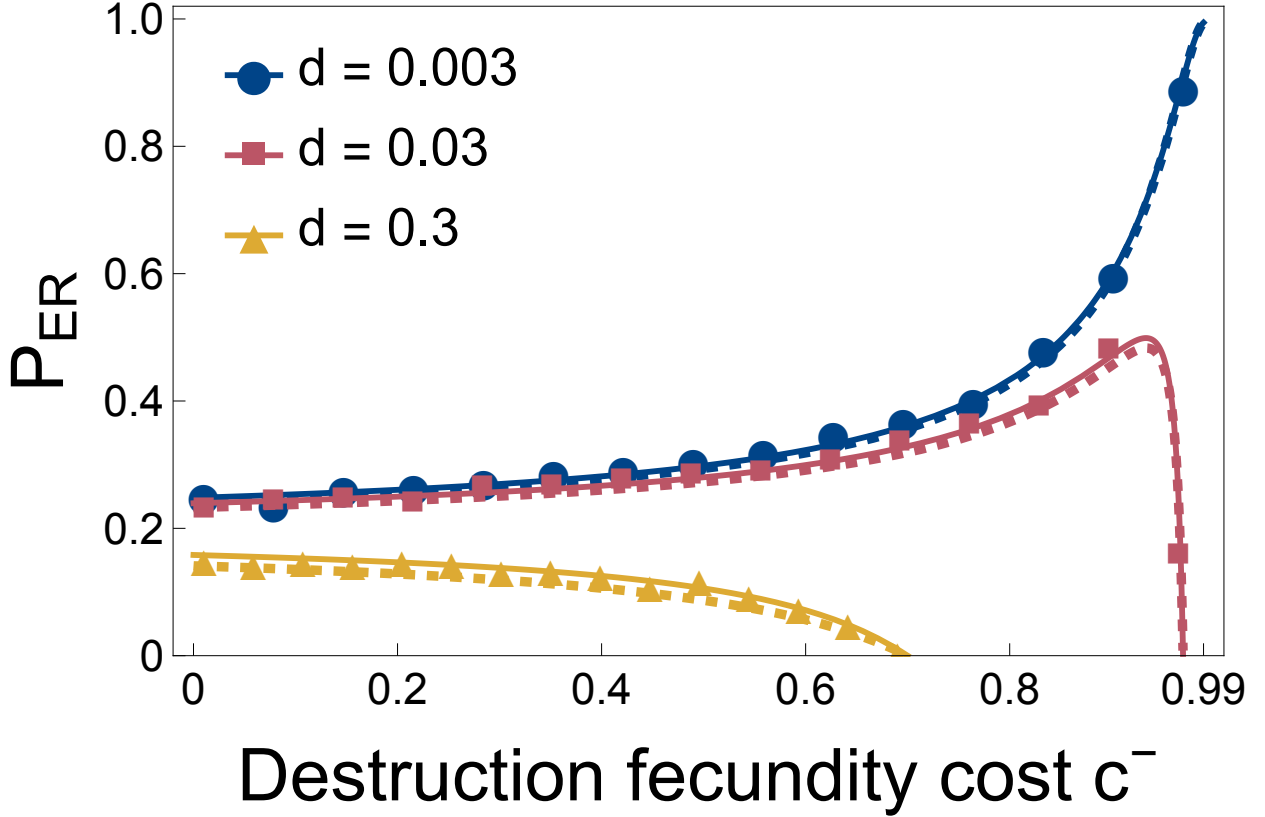


Figure 6: The probability of evolutionary rescue, P_{ER} , as a function of the fecundity cost of niche destruction, c^- . When the resident density independent death rate, d , is sufficiently large, P_{ER} is prevented from increasing with the fecundity cost of destruction, as the cost's reduction in the density independent probability of mutant establishment outweighs the cost's increase in mutational input. Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: numerical approximations of Eq. 3 evaluated under the fecundity cost regime in Table 2; dashed curves: Eq. 9a. Unless stated otherwise, parameter values are $\Psi = 0.145$, $a = 0.35$, $c^+ = 0$, $K = 3(10)^4$, and $U = 0.03$.

Thus, the range of fecundity costs of niche destruction promoting ER narrows as d increases (Fig. 6), meaning that only sufficiently long-lived niche constructors can benefit from fecundity costs. This is because, once d is adequately large, the increase in mutational input caused by the fecundity cost of destruction is outweighed by the cost's decrease in the density independent probability of mutant establishment, $1 - \frac{d}{b} = 1 - \frac{d}{1-c^-}$. Put analytically, if we condition ER on mutant establishment by letting $1 - \frac{d}{b} \rightarrow 1$ in Eq. 6 such that $P_{ER,f} \approx 1 - \exp\left(-\frac{U\bar{n}_R}{h\Psi}\right) = 1 - \exp\left(-\frac{U(1-c^- - d + a)}{\Psi(1-c^-)}\right)$ (which is the probability of a mutation

appearing prior to resident extinction), then $\frac{\partial P_{ER,f}}{\partial c^-} > 0$ for all $0 \leq d < b = 1 - c^-$; but if we do not condition on establishment, then $\frac{\partial P_{ER,f}}{\partial c^-} < 0$ for large enough d values. The same is true under the fecundity cost of niche construction (i.e., when $c^+ > 0$ and $c^- = 0$), assuming Eq. 11 is satisfied.

5 Discussion

We analytically approximated the probability, P_{ER} , that a niche-constructing population undergoes ER from habitat exploitation by an invading species. We first showed that P_{ER} increases with the initial ratio of the mutation rate to the invader growth rate (Eq. 6; Fig. 2), with slower invasion, in particular, ensuring a slower resident decline that provides more time for mutation. We then compared P_{ER} under two different costs of niche construction: a fecundity cost and a mortality cost. We found that the fecundity cost not only generates less variance in mutant fitness than the mortality cost but also—by lowering the rate at which niche constructors attempt reproduction and thus build new habitats—hinders invader growth enough to augment the total number of mutations appearing prior to resident extinction (Fig. 3). Our main result was that this latter benefit can cause the fecundity cost to promote ER compared to no cost, conditional on habitat loss deterring invaders enough to compensate for the cost’s reduction in resident density (Fig. 4). We also considered a fecundity cost of niche destruction, where constructors destroy their own habitats and consequently fail to reproduce. We showed that, by being linked to the removal of both new and pre-existing habitats available to invaders, the fecundity cost of destruction boosts P_{ER} more than that of construction (Fig. 5). If constructors have too short of lifespans, however, fecundity costs fail to promote ER (Fig. 6), as the benefit of greater mutational input is overshadowed by a lower chance of mutant establishment.

Previous theory

To our knowledge, the only previous study involving an explicit model of ER from invasion is that of Van Dyken (2020). In that study, Van Dyken (2020) showed that the chance of ER from invaders displacing residents through space and time can grow as the invader advantage decreases. This is because, as highlighted here and in studies without invasion as the cause of resident extinction, slower resident decline can mean greater *de novo* mutational opportunity (Uecker et al., 2014; Anciaux et al., 2018; Marrec & Bitbol, 2020). In addition, Van Dyken (2020) found ER to be more probable if invaders compete with residents for a spatially ubiquitous resource instead of directly harming residents, as the former scenario does not restrict the com-

petitive advantage of invader-immune mutants to locations where invaders are present (Van Dyken, 2020). Our work complements Van Dyken's (2020) study by showing that, when residents construct the exploited resource, they can increase mutational input—and in doing so promote ER—by slowing invader growth through either less frequent construction or more frequent destruction of the resource.

The prediction that infrequent niche construction can promote ER is consistent with previous work modeling ER of a non-niche-constructing population via a niche-constructing mutation (Longcamp & Draghi, 2023). Therein, the low rate of construction was found to promote mutant establishment by not only diminishing the fecundity cost of construction, which was assumed to grow with the rate of construction, but also by preventing over-exploitation of the mutants' constructed habitats by residents (Longcamp & Draghi, 2023). Here we extended this result by showing that infrequent construction can hinder habitat exploitation so effectively that fecundity costs themselves, whether they stem from niche constructors attempting reproduction less often or inducing niche destruction, can facilitate ER from habitat exploitation. For this to happen, habitat loss must compensate for the cost's reduction in resident density; and the constructor death rate must be low enough for the cost's increase in mutational input to outweigh the cost's reduction in the mutant establishment probability. Even when these requirements are unmet, however, fecundity costs are less harmful than mortality costs because mortality generates greater variance in mutant fitness. Henriques and Osmond (2020) derived a similar result regarding mutant variance in an adaptive dynamic model of ER via the construction of a public good, suggesting that the optimality of fecundity costs is robust to changes in mutation effect size.

Given that the constructed habitats in our model can benefit anyone that encounters them, our model, as with that of Henriques and Osmond's (2020), is one of cooperation. Instead of modeling ER via cooperation (Henriques & Osmond, 2020), however, here we have modeled ER from the tragedy of the commons (Hardin, 1968), whereby a population of cooperators producing a shared resource is driven to extinction by non-producing cheaters that exploit the resource. While a plethora of models have been developed to address the tragedy (for reviews see Sachs et al., 2004; Lehmann & Keller, 2006; West et al., 2007), relatively few have included our assumption that a resource constructed in the present can be used by individuals living in the future—a process known as ecological inheritance (Laland et al., 1999). Among the few models of cooperation with ecological inheritance (hereafter niche construction; e.g., Lehmann, 2007, 2008; Mullon & Lehmann, 2018), particularly relevant to our work is Krakauer et al.'s (2009) deterministic model of competition between niche constructors and cheaters. In that model, Krakauer et al. (2009) found that fecundity-promoting niche construction—the form of construction modeled herein—can only evolve in the

face of cheaters if constructors exhibit a sufficient degree of monopolization over the constructed resource. For instance, monopolization can be achieved when, under limited dispersal in a spatially structured population, the benefit of future constructor kin inheriting a resource outweighs the cost of producing the resource in the present (Lehmann, 2007, 2008). Furthermore, Krakauer et al. (2009) showed that, even with such preferential access, if organisms are sufficiently short-lived, they will tend to die before inheriting the resource and, consequently, the population will evolve to invest little energy in construction. Our work complements that of Krakauer et al. (2009) by showing that fecundity costs can help constructors monopolize their resource by limiting resource production, making the resource scarce enough to be rarely encountered by cheaters and thus used primarily by constructors upon its creation. The contingency of this result on a low constructor death rate—one allowing mutants to overcome drift—highlights another reason why constructors can be expected to have long lifespans.

Although other models of cooperation have revealed that fitness costs can promote cooperator persistence, the mechanisms making such costs beneficial differ from the resource-limiting mechanism in our model (Smaldino et al., 2013; Connelly et al., 2016). For example, with spatially explicit simulations, Smaldino et al. (2013) found that higher costs of cooperation facilitated cooperator persistence by allowing mutant cheaters to more rapidly invade large clusters of cooperators, leaving small, isolated clusters that thrived due to a lack of cheaters. Moreover, in another simulation study, Connelly et al. (2016) showed that cooperators promoted their own persistence with the help of both cooperation and niche destruction. Cooperation ensured that initially small, monomorphic subpopulations of cooperators quickly reached large abundances at which adaptive mutations appeared regularly (Connelly et al., 2016). Destruction then generated harmful, but novel, environments to which the frequently mutating cooperators could quickly adapt, allowing cooperators to remain better adapted than rare mutant cheaters (Connelly et al., 2016). Although we too found that destruction can promote cooperator persistence by generating new mutational opportunities, in our model such opportunities are acquired by slowing cheater growth, thereby reducing the rate at which potentially mutating cooperators are displaced.

Future theory

Several studies have provided evidence of ER from invasion, with examples including a native cricket population adapting to overcome an invasive, parasitic fly (Zuk et al., 2006); the Australian black snake evolving to more safely consume the toxic, invasive cane toad (Phillips & Shine, 2006); and cladocerans evolving to withstand toxin-producing, cyanobacterial competitors (Jiang et al., 2016). Despite such evi-

dence, however, most ER models have only implicitly included invasion as the cause of resident decline and, consequently, have not accounted for the diverse array of invader-resident interactions that could affect a population's chances of adaptation. Such interactions could, for instance, entail toxin-constructing invaders attacking non-toxin-constructing residents; or perhaps parasitic invaders killing resident predators involved in an otherwise stable coexistence with their prey. Addressing this knowledge gap using the branching process approach herein, and in Van Dyken (2020), could reveal analytical expressions that, if compared, would provide rules of thumb regarding which invader-resident interactions a population or community is most likely to withstand through natural selection.

Here we focused on an interaction between resident niche constructors and habitat-exploiting invaders. Throughout the analysis, we made several simplifying assumptions that could be worth relaxing in future work. For instance, we ignored the possibility of ER via standing genetic variation (SGV). Given that faster resident decline has been shown to sometimes facilitate ER by relieving SGV mutants of intraspecific competition (Uecker et al., 2014), permitting SGV may reveal that rapid invader growth can sometimes promote ER. If one were to extend our model with SGV, however, one should also relax our assumption of a negligible invader death rate. In addition to permitting invader extinction—which, if it occurred, would eliminate the need for ER—invader mortality would slow invader growth, likely hindering the establishment of SGV mutants while simultaneously promoting the appearance of *de novo* mutations.

We also assumed that competition promotes niche constructor mortality instead of limiting constructor births. To see how relaxing this assumption would affect our results, we modify Eq. 1 with resident birth and death rates of $b \rightarrow b[1 - \delta(\bar{n}_R(t) + \bar{n}_I(t) + \bar{n}_M(t))]$ and $d + \delta(\bar{n}_R(t) + \bar{n}_I(t) + \bar{n}_M(t)) \rightarrow d$, respectively; then re-derive P_{ER} , obtaining $1 - \exp\left(-\frac{U\bar{n}_R}{h\Psi} \ln\left(\frac{b}{d}\right)\right)$. Upon comparing this expression to Eq. 6 (i. e., $1 - \exp\left(-\frac{U\bar{n}_R}{h\Psi} \left(1 - \frac{d}{b}\right)\right)$), we see that, since $1 - \frac{d}{b} < \ln\left(\frac{b}{d}\right)$ for all positive mutant growth rates (i.e., $b - d > 0$), P_{ER} is lower under density dependent deaths than births due to the former generating greater variance in mutant fitness. Czuppon et al. (2023) validated a similar prediction with simulations of an antibiotic resistance model. More work should be done to understand how P_{ER} changes under the special case of brood parasitism, where, in addition to residents experiencing a density dependent birth rate, the invader growth rate is proportional to the density of both the constructed habitats and residents (e.g., resident birds incubating invader eggs in resident-constructed nests).

Finally, the probability of niche destruction was assumed to be the same irrespective of whether a niche constructor is using a new or pre-existing habitat. This means we did not account for the tendency of constructed habitats to decay in durability due to entropy. To understand how asymmetry in the resilience of

new and pre-existing habitats would effect our results, we compare Eq. 6's derivative with respect to the rate of niche construction, $\frac{\partial P_{ER}}{\partial g^+}$, and that with respect to the rate at which pre-existing habitats undergo destruction, $\frac{\partial P_{ER}}{\partial g^-}$. Doing so reveals that $\left| \frac{\partial P_{ER}}{\partial g^+} \right| > \left| \frac{\partial P_{ER}}{\partial g^-} \right|$ for all $g^+ = g^- > 0$, meaning that the entropic decay of pre-existing habitats would not promote ER as effectively as a reduction in the strength of new habitats following lower-effort construction. A remaining question worth probing in future work is whether these destruction dynamics would hold if construction promoted adult survival instead of fecundity. Plugging a destruction mortality cost regime (i.e., $b = 1, d = c^-, g^- = 1 - g^+ = c^-, \delta = 1$) into Eq. 6 shows that, in our current model, such a mortality cost never promotes ER (i.e., $\frac{\partial P_{ER}}{\partial c^-} < 0$ for all $c^- > 0$); but still unclear is whether this would change if, due to construction facilitating adult survival, destruction caused invader mortality.

Empirical work

A potential example of niche constructors undergoing ER from habitat exploitation involves the nest-building cliff swallow competing with the invasive, nest-usurping house sparrow (Peterjohn, 2001). Field data and observations suggest that, in the late 1800s—following the introduction of the house sparrow—the Ohio population of cliff swallows was declining until, in the 1970s, the swallows began nesting on bridges and dams avoided by the sparrows (Peterjohn, 2001; Rodewald et al., 2016). This shift to nesting at sites unfrequented by house sparrows, which is analogous to mutants avoiding competition with invaders in our model, was followed by a rapid rebound in cliff swallow abundance (Peterjohn, 2001; Rodewald et al., 2016). While the effect of fitness costs on this rebound is unclear, the cliff swallow incurs a type of fecundity cost that our model predicts could promote ER: specifically, cliff swallows require an estimated 7.8 days of their roughly ten-week breeding season to build a single nest (Withers, 1977; Brown et al., 2000), thereby constraining the number of nests that could be built during the breeding season. Moreover, the cliff swallow has coevolved with a nest-dwelling ectoparasite, the swallow bug, that often becomes highly abundant within a nest and can remain in the nest for over a year without feeding (Brown et al., 2021). Likely due to the cliff swallow's coevolutionary history with the swallow bug, cliff swallows tend to be less harmed by the bug than house sparrows (O'Brien et al., 2011; Fassbinder-Orth et al., 2013). This asymmetry in the bug's harmful effects on the two birds makes the bug akin to habitat loss in our model, which the model predicts can allow a fecundity cost to promote ER by reducing the invader growth rate without harming resident constructors.

For evidence that fecundity costs can limit invader growth, one can look to studies of digger wasps

that construct a separate developmental environment—composed of a burrow (or a chamber within a multi-chambered burrow) and provisioned food—for each of their offspring (Rosenheim, 1989; Polidori & Andrietti, 2006; Field et al., 2007). In some species, constructors risk exploitation by conspecifics that usurp the burrows, often without stealing any provisioned food (Field & Foster, 1995; Ghazoul, 2001); or by heterospecifics, such as kleptoparasitic flies or wasps, that deposit offspring into the burrows to feed on provisions (Rosenheim, 1989; Spofford & Kurczewski, 1992; Polidori et al., 2010). The results of several studies suggest that a reduction in burrow density helps digger wasps and other burrowing hymenopterans avoid exploitation by lowering the chance that exploiters locate burrows (Rosenheim, 1987, 1989; Enquist & Leimar, 1993; Polidori et al., 2005, 2006, 2010; Polidori, 2017). Experiments have shown that such a reduction in burrow density can stem from fecundity costs in the form of time spent provisioning food (Field et al., 2007) or niche destruction (Polidori & Andrietti, 2006). In the latter case, a given wasp actively destroyed its burrow by enlarging the burrow's entrance upon the arrival of a large conspecific capable of usurpation (Polidori & Andrietti, 2006). A similar behavior has been observed in hair-crested drongos (Lei et al., 2018). Evidence suggests that the drongos intentionally destroy their nests after use to limit strong future competition for nest sites (Lei et al., 2018).

Conclusion

Since constructing habitats is an inherently costly endeavor, niche constructors are vulnerable to displacement by invaders that exploit the constructed habitats. Here we modeled a niche-constructing population that must adapt to withstand habitat-exploiting invaders, thereby undergoing evolutionary rescue. Our main result was that fecundity costs of niche construction can promote rescue compared to no costs. In such a case, the fecundity cost diminishes the density of usable habitats, and therefore the invader growth rate, enough to generate additional mutational opportunities. Our results suggest that the same fitness costs making constructors vulnerable to habitat exploitation can help rescue constructors from such exploitation.

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Appendix A. Additional model details

Table A1 shows our full, detailed model. In the model, residents disperse into the reproductive environment at a per-capita rate of $m > 0$. After settling in a randomly chosen habitat and performing niche

Table A1: Model events and their corresponding probabilities and state changes.

Event	Probability	State change(s)
A patch of habitat 1 is lost	$H(t)adt$	$H(t) \rightarrow H(t) - 1$
A type $j \in \{M, R\}$ organism niche constructs and gives birth without niche destructing	$N_j(t) (1 - h(t)) \beta g^+ dt$	$H(t) \rightarrow H(t) + 1$ $N_j(t) \rightarrow N_j(t) + 1$
A type j organism niche constructs without giving birth or niche destructing	$N_j(t) (1 - h(t)) (1 - \beta)g^+ dt$	$H(t) \rightarrow H(t) + 1$ $N_j(t) \rightarrow N_j(t) + 0$
A type j organism niche constructs, gives birth, and niche destructs	$N_j(t)m (1 - h(t)) \beta (1 - \gamma^+) dt$	$N_j(t) \rightarrow N_j(t) + \Omega$
A type j organism gives birth and niche destructs	$N_j(t)h(t)\beta g^- dt$	$H(t) \rightarrow H(t) - 1$ $N_j(t) \rightarrow N_j(t) + \Omega$
A type j organism niche destructs without giving birth	$N_j(t)h(t)(1 - \beta)g^- dt$	$H(t) \rightarrow H(t) - 1$ $N_j(t) \rightarrow N_j(t) + 0$
A type j organism gives birth without niche destructing	$N_j(t)mh(t)\beta (1 - \gamma^-) dt$	$N_j(t) \rightarrow N_j(t) + 1$
A resident dies	$N_R(t) [d + \delta (n_R(t) + n_I(t) + n_M(t))] dt$	$N_R(t) \rightarrow N_R(t) - 1$
A mutant dies	$N_M(t) [d + \delta (n_R(t) + n_M(t))] dt$	$N_M(t) \rightarrow N_M(t) - 1$
A mutation occurs	$N_R(t)udt$	$N_M(t) \rightarrow N_M(t) + 1$
An invader gives birth	$N_I(t)h(t)\Psi dt$	$N_I(t) \rightarrow N_I(t) + 1$

construction if necessary, a resident reproduces with a probability of $0 < \beta \leq 1$. During the newborn offspring's development, niche destruction can occur. The per-capita rates at which a new habitat is constructed or a pre-existing habitat is destroyed are, conditional on the reproducing resident's choice of habitat, $g^+ = m\gamma^+ \leq m$ and $g^- = m\gamma^- \leq m$, respectively. In these expressions, $0 < \gamma^+ \leq 1$ represents the probability of a birth attempt resulting in the construction of a new habitat, meaning that $1 - \gamma^+$ is the probability of a new habitat undergoing destruction; and γ^- represents the probability that a pre-existing habitat undergoes destruction. We define γ^- as

$$\gamma^- := \begin{cases} \Gamma \in [0, 1], & \Omega = 1 \\ 1 - \gamma^+ \in [0, 1), & \Omega = 0, \end{cases} \quad (\text{A1})$$

where $\Omega \in \{0, 1\}$ is the number of offspring surviving destruction. Given this definition, the expected per-capita resident birth rate, $b = (1 - h(t))\beta g^+ + m(1 - h(t))\beta (1 - \gamma^+) \Omega + h(t)\beta g^- \Omega + mh(t)\beta (1 - \gamma^-)$, has a time-independent value of

$$b = \begin{cases} m\beta \in (0, m], & \Omega = 1 \\ m\beta(1 - \gamma^-) \in (0, m], & \Omega = 0. \end{cases} \quad (\text{A2})$$

The parameters β , γ^+ , and γ^- therefore permit $b = m\beta$, $g^+ = m\gamma^+$, and $g^- = m\gamma^-$ to vary independently of one another when $\Omega = 1$, which allows us to study the individual effects of each parameter on ER in Figure 2. To make Figures 1-2, we use $\Omega = 1$, $m = 2$, and, unless stated otherwise, $b = m\beta = 1$, $g^+ = m\gamma^+ = 1$, and $g^- = m\gamma^- = 0.15$. Meanwhile, our definition of γ^- links b , g^+ , and g^- when $\Omega = 0$, allowing us to study the two fitness cost regimes in Table 2. The b , g^+ , and g^- values in Table 2 are calculated by letting $\Omega = 0$, $\beta = 1$, and $c^- = \gamma^- = 1 - \gamma^+$ in both regimes; $m = 1 - c^+$ in the fecundity cost regime; and $m = 1$ in the mortality cost regime.

Appendix B. Stability analysis

Let $\bar{N}_I(t) = \bar{N}_M(t) = 0$ such that Eq. 1 becomes

$$\begin{aligned} f_1(t) &= \frac{d\bar{H}(t)}{dt} = \bar{N}_R(t) [(1 - \bar{h}(t))g^+ - \bar{h}(t)g^-] - \bar{H}(t)a \\ f_2(t) &= \frac{d\bar{N}_R(t)}{dt} = \bar{N}_R(t)b - \bar{N}_R(t)(d + \delta\bar{n}_R(t)). \end{aligned} \quad (\text{B1a-b})$$

When $f_1(t) = f_2(t) = 0$, the solution to Eq. B1 is $(\bar{H}(t), \bar{N}_R(t)) = (\bar{H}, \bar{N}_R)$ (Eq. 2). For this equilibrium to be locally stable, both eigenvalues of Eq. B1's corresponding Jacobian matrix, evaluated at $(\bar{H}(t), \bar{N}_R(t)) = (\bar{H}, \bar{N}_R)$, must have negative real parts; that is, the Jacobian must have a positive determinant and negative trace. The general Jacobian corresponding to Eq. B1 is

$$J(\bar{H}(t), \bar{N}_R(t)) = \begin{bmatrix} \frac{\partial f_1(t)}{\partial \bar{H}(t)} & \frac{\partial f_1(t)}{\partial \bar{N}_R(t)} \\ \frac{\partial f_2(t)}{\partial \bar{H}(t)} & \frac{\partial f_2(t)}{\partial \bar{N}_R(t)} \end{bmatrix} = \begin{bmatrix} -a - \frac{(g^+ + g^-)\bar{N}_R(t)}{K} & g^+ - \frac{(g^+ + g^-)\bar{H}(t)}{K} \\ 0 & b - d - \frac{2\delta\bar{N}_R(t)}{K} \end{bmatrix}. \quad (\text{B2})$$

This matrix has a determinant and trace of, respectively

$$\begin{aligned} \det(J(\bar{H}(t), \bar{N}_R(t))) &= \frac{\partial f_1(t)}{\partial \bar{H}(t)} \left(\frac{\partial f_2(t)}{\partial \bar{N}_R(t)} \right) - \frac{\partial f_1(t)}{\partial \bar{N}_R(t)} \left(\frac{\partial f_2(t)}{\partial \bar{H}(t)} \right) = \frac{[2\delta\bar{N}_R(t) - (b - d)K] [aK + (g^+ + g^-)\bar{N}_R(t)]}{K^2} \\ \text{tr}(J(\bar{H}(t), \bar{N}_R(t))) &= \frac{\partial f_1(t)}{\partial \bar{H}(t)} + \frac{\partial f_2(t)}{\partial \bar{N}_R(t)} = b - d - a - \frac{(2\delta + g^+ + g^-)\bar{N}_R(t)}{K}. \end{aligned} \quad (\text{B3a-b})$$

When $(\bar{H}(t), \bar{N}_R(t)) = (\bar{H}, \bar{N}_R)$, Eq. B3 becomes

$$\begin{aligned}\det(J(\bar{H}, \bar{N}_R)) &= \frac{(b-d)[a\delta + (b-d)(g^+ + g^-)]}{\delta} \\ \text{tr}(J(\bar{H}, \bar{N}_R)) &= -a - \frac{(b-d)(\delta + g^+ + g^-)}{\delta}.\end{aligned}\tag{B4a-b}$$

Since $\det(J(\bar{H}, \bar{N}_R)) > 0$ and $\text{tr}(J(\bar{H}, \bar{N}_R)) < 0$ when $b-d > 0$ and all other parameters are positive, $(\bar{H}(t), \bar{N}_R(t)) = (\bar{H}, \bar{N}_R)$ is locally stable throughout the parameter space defined in Section 2.

Appendix C. Approximation of P_{ER}

Assuming mutations are sufficiently rare, the number of mutant lineages appearing and establishing before resident extinction is an approximately Poisson-distributed random variable with an expected value of

$$\begin{aligned}\Lambda &\approx \int_{t_M=0}^{\infty} U\bar{n}_R(t_M)P_{Est}(t_M)dt_M \\ &= \int_{t_M=0}^{\infty} \frac{U\bar{n}_R\bar{N}_R[(b-d)(2\exp(\bar{h}\Psi t_M) + \bar{N}_R) - \bar{N}_R\bar{n}_R\delta]}{b(2\exp(\bar{h}\Psi t_M) + \bar{N}_R)^2} dt_M \\ &= \int_{t_M=0}^{\infty} \frac{U\bar{n}_R\bar{N}_R(b-d)}{b(2\exp(\bar{h}\Psi t_M) + \bar{N}_R)} dt_M + \int_{t_M=0}^{\infty} -\frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b(2\exp(\bar{h}\Psi t_M) + \bar{N}_R)^2} dt_M \\ &= \underbrace{\frac{U\bar{n}_R\bar{N}_R(b-d)}{b} \int_{t_M=0}^{\infty} \frac{1}{2\exp(\bar{h}\Psi t_M) + \bar{N}_R} dt_M}_{\Lambda_1} + \underbrace{\frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b} \int_{t_M=0}^{\infty} -\frac{1}{(2\exp(\bar{h}\Psi t_M) + \bar{N}_R)^2} dt_M}_{\Lambda_2},\end{aligned}\tag{C1}$$

where $\bar{N}_R(t_M)$ is given by Eq. 4 and $P_{Est}(t_M)$ is given by Eq. 5. To compute $\Lambda_1 = \frac{U\bar{n}_R\bar{N}_R(b-d)}{b} \int_{t_M=0}^{\infty} (2\exp(\bar{h}\Psi t_M) + \bar{N}_R)^{-1} dt_M$, we substitute $v = -\bar{h}\Psi t_M$ and $dv = -\bar{h}\Psi dt_M$ such that

$$\begin{aligned}\Lambda_1 &= \frac{U\bar{n}_R\bar{N}_R(b-d)}{b} \int_{t_M=0}^{\infty} \frac{1}{2\exp(\bar{h}\Psi t_M) + \bar{N}_R} dt_M \\ &= -\frac{1}{\bar{h}\Psi} \frac{U\bar{n}_R\bar{N}_R(b-d)}{b} \int_{v=0}^{-\infty} \frac{1}{2\exp(-v) + \bar{N}_R} dv \\ &= -\frac{U\bar{n}_R\bar{N}_R(b-d)}{b\bar{h}\Psi} \int_{v=0}^{-\infty} \frac{\exp(v)}{2 + \bar{N}_R \exp(v)} dv.\end{aligned}\tag{C2}$$

Then we substitute $w = 2 + \bar{N}_R \exp(v)$ and $dw = \bar{N}_R \exp(v) dv$, yielding

$$\begin{aligned}
\Lambda_1 &= -\frac{U\bar{n}_R\bar{N}_R(b-d)}{b\bar{h}\Psi} \int_{v=0}^{-\infty} \frac{\exp(v)}{2 + \bar{N}_R \exp(v)} dv \\
&= -\frac{1}{\bar{N}_R} \frac{U\bar{n}_R\bar{N}_R(b-d)}{b\bar{h}\Psi} \int_{w=2+\bar{N}_R}^2 \frac{1}{w} dw \\
&= -\frac{U\bar{n}_R(b-d)}{b\bar{h}\Psi} (\ln(2) - \ln(2 + \bar{N}_R)) \\
&= \frac{U\bar{n}_R(b-d)}{b\bar{h}\Psi} \ln\left(1 + \frac{\bar{N}_R}{2}\right).
\end{aligned} \tag{C3}$$

Similarly, we compute $\Lambda_2 = -\frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b} \int_{t_M=0}^{\infty} (2\exp(\bar{h}\Psi t_M) + \bar{N}_R)^{-2} dt_M$ by first substituting $v = -\bar{h}\Psi t_M$ and $dv = -\bar{h}\Psi dt_M$:

$$\begin{aligned}
\Lambda_2 &= -\frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b} \int_{t_M=0}^{\infty} \frac{1}{(2\exp(\bar{h}\Psi t_M) + \bar{N}_R)^2} dt_M \\
&= \frac{1}{\bar{h}\Psi} \frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b} \int_{v=0}^{-\infty} \frac{1}{(2\exp(-v) + \bar{N}_R)^2} dv \\
&= \frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b\bar{h}\Psi} \int_{v=0}^{-\infty} \frac{\exp(2v)}{(2 + \bar{N}_R \exp(v))^2} dv.
\end{aligned} \tag{C4}$$

Then we substitute $w = 2 + \bar{N}_R \exp(v)$ and $dw = \bar{N}_R \exp(v) dv$:

$$\begin{aligned}
\Lambda_2 &= \frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b\bar{h}\Psi} \int_{v=0}^{-\infty} \frac{\exp(2v)}{(2 + \bar{N}_R \exp(v))^2} dv \\
&= \frac{1}{\bar{N}_R^2} \frac{U\bar{n}_R^2\bar{N}_R^2\delta}{b\bar{h}\Psi} \int_{w=2+\bar{N}_R}^2 \frac{w-2}{w^2} dw \\
&= \frac{U\bar{n}_R^2\delta}{b\bar{h}\Psi} \left(\int_{w=2+\bar{N}_R}^2 \frac{1}{w} dw - 2 \int_{w=2+\bar{N}_R}^2 \frac{1}{w^2} dw \right) \\
&= \frac{U\bar{n}_R^2\delta}{b\bar{h}\Psi} \left(\ln(2) + 1 - \ln(2 + \bar{N}_R) - \frac{2}{2 + \bar{N}_R} \right) \\
&= -\frac{U\bar{n}_R^2\delta}{b\bar{h}\Psi} \frac{(2 + \bar{N}_R) \ln\left(1 + \frac{\bar{N}_R}{2}\right) - \bar{N}_R}{2 + \bar{N}_R}.
\end{aligned} \tag{C5}$$

The probability of evolutionary rescue is therefore

$$\begin{aligned}
P_{ER} &\approx 1 - \frac{(\Lambda)^0 \exp(-\Lambda)}{0!} \\
&= 1 - \exp(-\Lambda) \\
&= 1 - \exp(-(\Lambda_1 + \Lambda_2)) \\
&= 1 - \exp\left(-\frac{U \left[\bar{n}_R^2 \bar{N}_R \delta + \bar{n}_R (2 + \bar{N}_R) \ln\left(1 + \frac{\bar{N}_R}{2}\right) (b - d - \bar{n}_R \delta) \right]}{b \bar{h} \Psi (2 + \bar{N}_R)}\right) \\
&= 1 - \exp\left(-\frac{U \bar{n}_R^2 \bar{N}_R \delta}{b \bar{h} \Psi (2 + \bar{N}_R)}\right),
\end{aligned} \tag{C6}$$

where the final simplification results from $b - d - \bar{n}_R \delta = 0$ given $\bar{n}_R = \frac{b-d}{\delta}$. Assuming K is large enough for $P_{ER} \approx \lim_{K \rightarrow \infty} P_{ER}$ (or equivalently for $\bar{N}_R \approx 2 + \bar{N}_R$), Eq. C6 becomes

$$\begin{aligned}
P_{ER} &\approx 1 - \exp\left(-\frac{U \bar{n}_R^2 \delta}{b \bar{h} \Psi}\right) \\
&= 1 - \exp\left(-\frac{U \bar{n}_R}{\bar{h} \Psi} \left(1 - \frac{d}{b}\right)\right).
\end{aligned} \tag{C7}$$

Chapter 3

The effect of demography on evolutionary rescue from invasion

Abstract

Understanding when a population is most likely to undergo evolutionary rescue from invasion can help guide decisions in both species conservation and pathogen elimination. While models of evolutionary rescue from invasion exist, a critical gap remains in understanding how different types of invaders—varying in both their proliferation strategies and their effects on residents—impact the chance of rescue. Here we analyze a model of rescue from a directly antagonistic invading species that can range from generalist to specialist and from a species that only hinders resident births to one that exclusively causes resident mortality. We first show that the probability of evolutionary rescue varies nonlinearly with respect to the per-capita invader growth rate, with slow invasion facilitating the appearance of *de novo* mutations and fast invasion relieving standing genetic variants of competition with their resident ancestors. For this reason, rescue from slowly proliferating specialists is more likely to occur via *de novo* mutation, whereas rescue from quickly proliferating generalists is more likely to occur through standing genetic variation. We then show that, counterintuitively, a reduction in the resident birth rate can increase the chance of rescue by lessening the variance in mutant fitness, thereby improving the chance of mutant establishment. This benefit can be achieved whether the reduction in the resident birth rate is density independent or dependent. In contrast, a higher resident death rate always hinders rescue in our model because more frequent resident mortality means greater mutant variance.

1 Introduction

The invasion of a space by one species at the expense of another is a fundamental biological process that can stabilize or disrupt communities depending on the context. Invasive species, for example, are a leading driver of species extinction, with invasive predators alone contributing to roughly 58 percent of all known bird, mammal, and reptile extinctions (Doherty et al., 2016). Conversely, humans sometimes introduce biocontrol agents that can protect native communities by invading and eliminating pests and disease vectors (e.g., Marshall & Fenner, 1958). This dual impact of invasion also appears at the microorganismal level: pathogens are constantly attempting to invade healthy cell populations, while medical practitioners sometimes deploy bacteriophages that can be viewed as invaders targeting and displacing the pathogens (e.g., Teklemariam et al., 2023). Understanding the conditions that allow populations to resist invasion can therefore guide strategies in both conservation and medicine.

Invaders can vary in both the strategy they use to proliferate and the effect they have on resident populations. Some invaders are generalists that can proliferate independently of a given resident species: for instance, the invasive Burmese python preys on a wide range of mammals, such as raccoons, opossums, bobcats, and white-tailed deer, allowing it to rapidly establish itself in the Everglades at the expense of such species (Dorcas et al., 2012). In contrast, other invaders are specialists that rely on residents to proliferate, as exemplified by the emerald ash borer—an invasive beetle that, in its introduced range, almost exclusively feeds on ash trees (Sun et al., 2024). Furthermore, some invaders primarily reduce the resident birth rate (defined herein as fecundity and offspring viability), which is typically the case with brood parasites that exploit the parental care of residents (Payne, 1977); whereas other invaders mainly induce resident mortality, as with invasive predators (Doherty et al., 2016). Most invaders, however, likely affect both resident birth and death rates to some extent; for instance, the invasive Argentine ant has been found to not only kill adults but also to target brood during raids of native colonies (Zee & Holway, 2006).

Although invasion typically occurs on short, ecological timescales, there is evidence that populations undergoing invasion can avoid extinction by evolving to withstand the invaders (Marshall & Fenner, 1958; Zuk et al., 2006). For example, native field crickets on two different Hawaiian islands were approaching extinction due to the invasion of a deadly parasitic fly until a mutation prevented the male crickets from chirping, which in turn prevented the males from attracting the parasite (Zuk et al., 2006). Similarly, the Australian population of the invasive European rabbit was declining due to the controlled introduction of the Myxoma virus until the rabbits evolved greater innate immunity to the virus (Marshall & Fenner, 1958).

Evidence also suggests that the Australian black snake evolved to withstand the invasive cane toad's toxins (Phillips and Shine, 2006).

The process by which a population avoids extinction through adaptation is known as evolutionary rescue (Gomulkiewicz & Holt, 1995). Characterized by a U-shaped curve, rescue entails a population declining toward extinction until an adaptive mutant lineage appears—either via standing genetic variation or *de novo* mutation—and subsequently establishes by overcoming drift (Orr & Unckless, 2014). A great deal of theory has been developed to understand how different ecological interactions like competition (Osmond & de Mazancourt, 2013), predation (Yamamichi & Miner, 2015; Osmond et al., 2017), and cooperation (Henriques & Osmond, 2020) affect rescue. However, such models have only implicitly considered invasion as the cause of population decline. In contrast, although work explicitly modeling rescue from invasion has accounted for resident-invader interactions in the form of interference and exploitation competition (Van Dyken, 2020) and the exploitation of resident-constructed habitats (Longcamp & Draghi, see Chapter 2), still missing is an understanding of how rescue is differentially affected by generalist and specialist invaders, as well as birth-limiting and death-promoting invaders.

For this reason, we analyze the probability of evolutionary rescue, P_R , from a directly antagonistic invading species that can range from generalist to specialist and from a species that exclusively limits resident births to one that only induces resident mortality. We begin our study by introducing an eco-evolutionary model of resident-invader competition and subsequently adapting the model for rescue. Following previous population genetic models of rescue, we assume rescue occurs, on average, through a single large-effect mutation (Uecker et al., 2014), instead of the several small-effect mutations characteristic of adaptive dynamic and quantitative genetic models of rescue (Bürger & Lynch, 1995; Henriques & Osmond, 2020). We first find that P_R varies nonlinearly with respect to the invader growth rate: slow invasion promotes rescue by facilitating the appearance of *de novo* mutations, whereas fast invasion aids rescue by relieving standing genetic variants of competition with residents and variance in reproductive success (Fig. 1). The latter benefit is less prominent in the case of specialist invaders, because such invaders sometimes fail to encounter residents and thus fail to reproduce, thereby reducing the rate at which standing genetic variants are relieved of competition with residents. Our main result is that a reduction in the resident birth rate—but not a boost in the resident death rate—can appreciably increase P_R by further diminishing mutant variance and the effect of residents on mutant fitness (Figs. 2-3). This is true whether the lower resident birth rate stems from a decrease in the resident density independent birth rate (Fig. 2) or an increase in the sensitivity of residents to birth-limiting competition (Fig. 3). Unlike the benefit of a smaller resident density independent

birth rate, however, the benefit of stronger birth-limiting competition disappears in the absence of death-promoting competition (Fig. 4), because competition's effect on mutant fitness decays with resident density and is consequently weaker than density independent factors.

2 Model

In this section, we introduce a stochastic, eco-evolutionary model and subsequently adapt the model for evolutionary rescue. The model's constituent parameters and variables are listed in Table 1; and the different events that can occur in the model are listed in Table 2.

Table 1: Model parameters and variables.

Notation	Interpretation
b	Per-capita resident density independent birth rate
d	Per-capita resident density independent death rate
δ_b	Per-interaction resident sensitivity to birth competition
δ_d	Per-interaction resident sensitivity to death competition
K	Resident carrying capacity
Φ^-	Per-capita resident-independent invader growth rate
Φ^+	Expected maximum per-capita resident-dependent invader growth rate
u	Per-capita mutation rate
U	$U = Ku$: Maximum initial mutation rate
t_M	Time at which the mutant lineage responsible for rescue first appears
$t_{1/2}$	Time at which residents reach half of their initial abundance
$N_R(t)$	Abundance of residents
$N_I(t)$	Abundance of invaders
$N_M(t)$	Abundance of mutants
$v(t)$	Density of variable $V(t) \in \{N_R(t), N_I(t), N_M(t)\}$ with respect to K
$\bar{V}(t)$	$\bar{V}(t) = K\bar{v}(t)$: Expected value of variable $V(t)$
\bar{V}	$\bar{V} = K\bar{v}$: Equilibrium value of variable $\bar{V}(t)$

2.1 Eco-evolutionary model

We model a well-mixed community that changes according to a continuous time, birth-death process (Kendall, 1948). The community is composed of three types of organisms: residents, invaders, and mutants, which have abundances of $N_R(t) \geq 0$, $N_I(t) \geq 0$, and $N_M(t) \geq 0$, respectively. Resident decline

toward extinction in the presence of invaders, which can range from generalists that reproduce independently of residents to specialists that rely on residents to proliferate. Mutants are descendants of residents that are immune to invaders.

2.1.1 Resident dynamics

Residents have a carrying capacity of $K > 0$ such that, on average, $N_R(t) \leq K$. The density of each variable, $V(t) \in \{N_R(t), N_I(t), N_M(t)\}$, with respect to K is denoted as $v(t) = \frac{V(t)}{K}$. Residents give birth at a per-capita rate of $\max[0, b - \delta_b (n_R(t) + n_I(t) + n_M(t))]$, where $b > 0$ is the per-capita resident density independent birth rate and $\delta_b > 0$ is the per-capita sensitivity of residents to birth-limiting competition (hereafter birth competition; Czuppon et al., 2023). Similarly, residents die at a per-capita rate of $d + \delta_d (n_R(t) + n_I(t) + n_M(t))$, where $0 < d < b$ is the per-capita resident density independent death rate and $\delta_d > 0$ is the per-capita sensitivity of residents to death-promoting competition (hereafter death competition).

2.1.2 Invader dynamics

For simplicity, we assume that the rate at which invaders migrate into the community is low enough to have a negligible effect on invader dynamics. Moreover, we condition the invader growth rate on invaders avoiding extinction such that, similar to Van Dyken's (2020) model of evolutionary rescue from invasion, residents are certain to go extinct in the presence of invaders. Under this assumption, invader mortality merely reduces the invader growth rate without additionally augmenting invader extinction risk. This allows us to model invader growth as a pure birth process, with invaders growing in abundance at a per-capita rate of $\Phi^- + \bar{n}_R(t)\Phi^+ > 0$, where $\Phi^- \geq 0$ is the per-capita resident-independent invader growth rate and $\Phi^+ \geq 0$ is, on average, the maximum per-capita resident-dependent invader growth rate. Throughout our study, we refer to invaders with $\Phi^- > 0$ and $\Phi^+ \approx 0$ as generalists; and those with $\Phi^- \approx 0$ and $\Phi^+ > 0$ as specialists.

2.1.3 Mutant dynamics

Residents mutate at a per-capita rate of $u > 0$. Back mutations are ignored. Mutations are assumed infrequent enough to have a negligible effect on resident dynamics. Mutant fitness is equal to that of residents except mutants are unaffected by invader density and therefore have per-capita birth and death rates of $\max[0, b - \delta_b (n_R(t) + n_M(t))]$ and $d + \delta_d (n_R(t) + n_M(t))$, respectively.

2.1.4 Expected community dynamics

Table 2: Model events and their corresponding probabilities and state changes.

Event	Probability	State change
A resident gives birth	$N_R(t) \max [0, b - \delta_b (n_R(t) + n_I(t) + n_M(t))] dt$	$N_R(t) \rightarrow N_R(t) + 1$
A resident dies	$N_R(t) [d + \delta_d (n_R(t) + n_I(t) + n_M(t))] dt$	$N_R(t) \rightarrow N_R(t) - 1$
An invader gives birth	$N_I(t) (\phi^- + n_R(t)\Phi^+) dt$	$N_I(t) \rightarrow N_I(t) + 1$
A mutant gives birth	$N_M(t) \max [0, b - \delta_b (n_R(t) + n_M(t))] dt$	$N_M(t) \rightarrow N_M(t) + 1$
A mutant dies	$N_M(t) [d + \delta_d (n_R(t) + n_M(t))] dt$	$N_M(t) \rightarrow N_M(t) - 1$
A mutation occurs	$N_R(t) u dt$	$N_M(t) \rightarrow N_M(t) + 1$

Table 2 summarizes the different events that can occur during a given time interval, $(t, t + dt)$, in our model. Using this information, we calculate the expected change in each variable, $V(t)$, during (t, dt) by first multiplying each of $V(t)$'s possible state changes by the probability of the change occurring (and assuming $\max [0, b - \delta_b (n_R(t) + n_I(t) + n_M(t))] > 0$), then summing over those products, yielding

$$\begin{aligned}
 \frac{d\bar{N}_R(t)}{dt} &= \bar{N}_R(t) (b - d) - \bar{N}_R(t) (\delta_b + \delta_d) (\bar{n}_R(t) + \bar{n}_I(t) + \bar{n}_M(t)) \\
 \frac{d\bar{N}_I(t)}{dt} &= \bar{N}_I(t) (\Phi^- + \bar{n}_R(t)\Phi^+) \\
 \frac{d\bar{N}_M(t)}{dt} &= \bar{N}_R(t)u + \bar{N}_M(t) (b - d) - \bar{N}_M(t) (\delta_b + \delta_d) (\bar{n}_R(t) + \bar{n}_M(t)),
 \end{aligned} \tag{1a-c}$$

where $\bar{V}(t) = K\bar{v}(t)$ is the expected value, as opposed to the realized value, of $V(t) = Kv(t)$. The solution to Eq. 1 gives $V(t)$'s average value across an infinite number of replicate realizations and so gives $V(t)$ in the absence of demographic variance. Letting $\frac{d\bar{N}_I(t)}{dt} = \frac{d\bar{N}_M(t)}{dt} = 0$, and then solving $\frac{d\bar{N}_R(t)}{dt} = 0$ for $\bar{N}_R(t)$, reveals that Eq. 1 exhibits an equilibrium at which residents have an abundance of

$$\bar{N}_R = \frac{K(b-d)}{\delta_b + \delta_d}, \tag{2}$$

where hereafter $\delta_b + \delta_d \geq b - d$ such that $\bar{N}_R \leq K$. Since the derivative of $\frac{d\bar{N}_R(t)}{dt}$ with respect to $\bar{N}_R(t)$ has a negative value of $-(b-d)$ when evaluated at $\bar{N}_R(t) = \bar{N}_R$, we know that the equilibrium is locally stable throughout parameter space. This means that, when invaders and mutants are vanishingly rare, the resident population is expected to tend toward \bar{N}_R following a fluctuation in size.

2.2 Evolutionary rescue model

We assume evolutionary rescue can occur via standing genetic variation or *de novo* mutation. At times $t < 0$, invaders are assumed absent from the community, while, for simplicity, a single standing genetic variant is present. In contrast, we assume $K \gg 1$ such that residents have an abundance of $\lfloor \bar{N}_R \rfloor \gg 1$, where $\lfloor \bar{V}(t) \rfloor$ is the floor of variable $\bar{V}(t)$. At time $t = 0$, an invader appears, giving the community an initial composition of $N_R(0) = \lfloor \bar{N}_R \rfloor$ and $N_I(0) = N_M(0) = 1$. As the invader population grows in size, the resident population declines toward extinction. We follow previous rescue theory (e.g., Wilson et al., 2017; Czuppon et al., 2021) by letting $u = \frac{U}{K} < 1$ so that the mutation rate, $\bar{N}_R(t)u = \bar{n}_R(t)U$, has a maximum initial value of $0 < U < 1$ and is consequently, on average, less than one. Mutants are considered established, and so rescue occurs, only if $N_M(t) \geq \lfloor 0.95\bar{N}_R \rfloor$.

3 Methods

We now introduce the general theory that will be used in the Results section to analytically predict the probability of evolutionary rescue, P_R . Since rescue can occur either via standing genetic variation or *de novo* mutation in our model, P_R can be written as

$$P_R = P_{R,SGV} + (1 - P_{R,SGV}) P_{R,DN}, \quad (3)$$

where $P_{R,SGV}$ is the probability of rescue via standing genetic variation and $P_{R,DN}$ is the probability of rescue via *de novo* mutation. We approximate these quantities by assuming that residents and invaders have large enough carrying capacities for community dynamics to be well-described by Eq. 1. Further assumed is that, during the period of time relevant to mutants escaping drift, mutant density is low enough to have a negligible effect on community dynamics such that the per-capita mutant birth and death rates are $b - \delta_b (\bar{n}_R(t) + \bar{n}_M(t)) \approx b - \delta_b \bar{n}_R(t)$ and $d + \delta_d (\bar{n}_R(t) + \bar{n}_M(t)) \approx d + \delta_d \bar{n}_R(t)$, respectively. Under these assumptions, mutants grow independently of one another and have a positive growth rate at all times $t > 0$, meaning that we can approximate $P_{R,SGV}$ using time-inhomogeneous branching process theory (Uecker and Hermisson, 2011). Upon plugging the approximated per-capita mutant birth and death rates into Uecker and Hermisson's Eq. 16, we have

$$\begin{aligned}
P_{R,SGV} &= P_{Est}(0) \\
P_{Est}(t_M) &\approx \frac{2}{1 + \int_{t=t_M}^{\infty} \sigma^2(t) \exp\left(-\int_{T=t_M}^t r(T)dT\right) dt} \\
r(t) &= b - d - (\delta_b + \delta_d) \bar{n}_R(t) \\
\sigma^2(t) &= b + d + (\delta_d - \delta_b) \bar{n}_R(t),
\end{aligned} \tag{4a-d}$$

where $P_{Est}(t_M)$ is the probability that a mutant lineage appearing at time $t_M \geq 0$ establishes by overcoming drift; $r(t)$ is the per-capita growth rate of mutants at time $t \geq t_M$; and $\sigma^2(t)$ is the variance in the per-capita mutant growth rate at time t . Assuming mutations are also rare enough to appear independently one another, the number of times a *de novo* mutant lineage appears and establishes prior to resident extinction is a Poisson-distributed random variable with an expected value of $\int_{t_M=0}^{\infty} \bar{n}_R(t_M) U P_{Est}(t_M) dt_M$. Consequently, $P_{R,DN}$ is

$$P_{R,DN} \approx 1 - \exp\left(-\int_{t_M=0}^{\infty} \bar{n}_R(t_M) U P_{Est}(t_M) dt_M\right). \tag{5}$$

To prepare for our forthcoming approximation of Eqs. 4-5, we calculate resident abundance, $\bar{N}_R(t)$, and therefore density, $\bar{n}_R(t)$, heuristically: we first assume that the invader and resident growth rates are proportional with opposite signs (i. e., $\frac{d\bar{N}_R(t)}{dt} \propto -\frac{d\bar{N}_I(t)}{dt}$) such that, when invaders are rare, residents maintain an effectively constant abundance of $\bar{N}_R(t) \approx \bar{N}_R$. Given this abundance, the invader trajectory can be approximated by solving Eq. 1c with $\bar{N}_I(0) = 1$, yielding $\bar{N}_I(t) \approx \exp((\Phi^- + \bar{n}_R \Phi^+) t)$. Since this trajectory is exponential—and thus entails an initial lag phase of negligible growth—we assume residents decline approximately via a logistic function, $\bar{N}_R(t) \approx \bar{N}_R [1 + \exp(-k(t_{1/2} - t))]^{-1}$, where $k > 0$ is the per-capita rate of resident decline and $t_{1/2} > 0$ is the resident half-life. To acquire k and $t_{1/2}$, we assume $\frac{d\bar{N}_R(t)}{dt} \approx -\frac{d\bar{N}_I(t)}{dt}$ so that (i) the community maintains a constant size of $\bar{N}_I(0) + \bar{N}_R(0) = \bar{N}_R + 1 \approx \bar{N}_R$, meaning $t_{1/2} \approx \{t \mid \exp((\Phi^- + \bar{n}_R \Phi^+) t) = \frac{\bar{N}_R}{2}\}$; and (ii) assuming the initial per-capita invader growth rate is small enough to overshadow the effect of other factors on k , we have $k \approx \frac{\bar{N}_I(t)}{\bar{N}_R(t)} (\Phi^- + \bar{n}_R \Phi^+) \approx \Phi^- + \bar{n}_R \Phi^+$. The resulting logistic function is

$$\begin{aligned}
\bar{N}_R(t) &\approx \frac{\bar{N}_R}{1 + \exp(-(\Phi^- + \bar{n}_R \Phi^+) (t_{1/2} - t))} \\
t_{1/2} &\approx \ln\left(\frac{\bar{N}_R}{2}\right) (\Phi^- + \bar{n}_R \Phi^+)^{-1}.
\end{aligned} \tag{6a-b}$$

Since residents remain near their initial density until invaders become sufficiently common at some time $J > 0$, mutants maintain an effectively constant growth rate of zero during the time interval $[0, J]$. For this reason, we will make use of the probability that a neutral mutation persists throughout $[0, J]$, $P_{Persist}(J)$. Upon plugging the approximated mutant birth rate into the neutral mutation extinction probability derived in Kendall (1948; Eq. 17 therein) and then subtracting the resulting expression from one, we have

$$P_{Persist}(J) \approx (1 + (b - \delta_b \bar{n}_R) J)^{-1}. \quad (7)$$

3.1 Simulations

We test our analytical predictions with simulations of a tau-leaping algorithm (Cao et al., 2006). A simulation is initialized with $N_R(0) = \lfloor \bar{N}_R \rfloor$ and $N_I(0) = N_M(0) = 1$. Each variable, $V(t)$, then changes according to its corresponding events, state changes, and transition rates (i.e., probabilities divided by dt) listed in Table 1. The value of each tau-leap, and the number of times each event occurs during the leap, are calculated using the procedure summarized in Sections IIC (excluding Step 3) and IV of Cao et al. (2006). As implemented, this procedure ensures that, if $V(t) < n_c = 10$, then all transition rates corresponding to a non-zero state change in $V(t)$ are updated according to a nearly exact version of the Gillespie algorithm (Gillespie, 1976). Further, the algorithm helps prevent $V(t)$ from becoming negative during a leap by approximately bounding the per-leap change in each of $V(t)$'s transition rates, relative to the sum of all transition rates, by a value of $\epsilon = 0.03$. If $V(t)$ becomes negative despite this precaution, we set $V(t) = 0$. We avoid an undefined leap size by adding 10^{-9} to the output of Cao et al.'s (2006) Eq. 32a if the output is zero; and the same adjustment is made to Eq. 32b if its output is zero. The simulation proceeds until either $N_R(t) = N_M(t) = 0$ or $N_M(t) \geq \lfloor 0.95 \bar{N}_R \rfloor$. All simulations are performed using Mathematica 13.2.0.0.

4 Results

In this section, we first analytically approximate the probability of evolutionary rescue via standing genetic variation, $P_{R,SGV}$, and that via *de novo* mutation, $P_{R,DN}$. Then we analyze the total probability of evolutionary rescue, P_R . Any assumptions made hereafter pertain only to our analytical approximations. All approximations are made using Eq. 6 for resident abundance, $\bar{N}_R(t)$; and all results are interpreted assuming $N_R(t) \approx \bar{N}_R(t)$. For brevity we use the term 'rate' when referring to any per-capita, per-interaction, or expected rate in Table 1. A reader uninterested in our analytical derivations can safely skip to Section 4.3.

4.1 The probability of evolutionary rescue via standing genetic variation

We begin our analysis by approximating the probability of rescue via standing genetic variation, $P_{R,SGV}$. Because mutants have an effectively neutral growth rate until some time $J > 0$ at which resident abundance starts to appreciably decline, we write $P_{R,SGV} \approx P_{Persist}(J)P_{Est}(J)$, where $P_{Persist}(J)$ is the probability that an effectively neutral mutation persists to time J (Eq. 7) and $P_{Est}(J)$ is the probability that the mutant's lineage establishes after acquiring a positive growth rate (Eq. 4b). When $J \gg 0$, the chance of a neutral mutation persisting to time J is negligible and so the parameter regimes relevant to rescue via standing genetic variation are those in which the invader growth rate, $\Phi^- + \bar{n}_R(t)\Phi^+$ —and hence the rate of resident decline—is large. With this in mind, we first assume that $\Phi^- + \bar{n}_R(t)\Phi^+$ is large enough for J to roughly equal the resident half-life, $J \approx t_{1/2}$. Under this assumption, Eq. 7 becomes $P_{Persist}(J) \approx (1 + (b - \delta_b \bar{n}_R) t_{1/2})^{-1}$. Then we take Eq. 4b's limit as $\Phi^- + \bar{n}_R(t)\Phi^+ \rightarrow \infty$, which gives $P_{Est}(J) \approx 1 - \frac{d}{b}$. With these approximations, $P_{R,SGV} \approx P_{Persist}(J)P_{Est}(J)$ is

$$P_{R,SGV} \approx \left(1 + \frac{(b - \delta_b \bar{n}_R) \ln\left(\frac{\bar{N}_R}{2}\right)}{\Phi^- + \bar{n}_R \Phi^+}\right)^{-1} \left(1 - \frac{d}{b}\right). \quad (8)$$

4.2 The probability of evolutionary rescue via *de novo* mutation

We now want to approximate the probability of rescue via *de novo* mutation, $P_{R,DN}$. Since new mutations require time to appear, the parameter regimes relevant to rescue via *de novo* mutation are those in which the invader growth rate, $\Phi^- + \bar{n}_R(t)\Phi^+$, is small. For this reason, we make the integral in the mutant establishment probability, $P_{Est}(t_M)$ (Eq. 4b), solvable by assuming that $\Phi^- + \bar{n}_R(t)\Phi^+$ is small enough for a mutant lineage appearing at time t_M to maintain effectively constant birth and death rates of $b - \delta_b \bar{n}_R(t_M)$ and $d + \delta_d \bar{n}_R(t_M)$, respectively. Upon plugging these vital rates into Eq. 4b; solving Eq. 5; and then simplifying the resulting expression by taking its limit as $K \rightarrow \infty$, we have

$$P_{R,DN} \approx 1 - \exp\left(-\frac{U \bar{n}_R}{\Phi^- + \bar{n}_R \Phi^+} \left(1 + \frac{\delta_d}{\delta_b}\right) \ln\left(\frac{b(\delta_b + \delta_d)}{d\delta_b + b\delta_d}\right)\right). \quad (9)$$

4.3 The probability of evolutionary rescue

Now that we have approximated the probability of rescue via standing genetic variation, $P_{R,SGV}$, and that via *de novo* mutation, $P_{R,DN}$, we can analyze the total rescue probability, $P_R = P_{R,SGV} + (1 - P_{R,SGV})P_{R,DN}$. As shown in Fig. 1A, a key factor determining P_R is the invader growth rate, $\Phi^- + \bar{n}_R(t)\Phi^+$, which has

two components: the resident-independent invader growth rate, Φ^- , and the resident-dependent invader growth rate, $\bar{n}_R(t)\Phi^+$, where Φ^+ is the latter's maximum value. When $\Phi^- + \bar{n}_R(t)\Phi^+$ is small, rescue is more likely to occur through *de novo* mutation than standing genetic variation (Fig. 1A). The lower rate of invasion means a lower rate of rate of resident decline, providing more time for a *de novo* mutation to occur prior to resident extinction. In contrast, when $\Phi^- + \bar{n}_R(t)\Phi^+$ is large, rescue happens more readily through standing genetic variation than *de novo* mutation (Fig. 1A), as residents are displaced faster, causing standing genetic variants to achieve a non-negligible selective advantage over residents at an earlier time, which reduces the chance of such variants succumbing to drift. This benefit of rapid invader growth becomes less pronounced when invaders are specialists that rely on residents to reproduce (i.e., when $\Phi^- \approx 0$ and $\Phi^+ > 0$; Fig. 1B). In such a case, as long as residents are below carrying capacity, invaders sometimes fail to encounter residents and thus fail to reproduce. In turn, standing genetic variants suffer from slower relief of competition with residents while *de novo* mutations become more likely to appear.

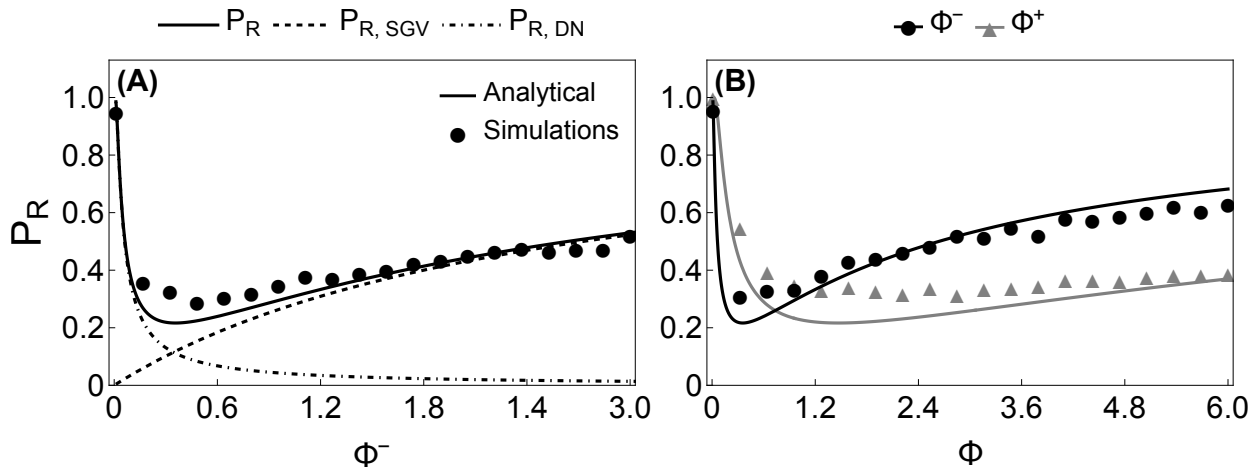


Figure 1: The probability of evolutionary rescue, P_R ; the probability of rescue via standing genetic variation, $P_{R,SGV}$; and the probability of rescue via *de novo* mutation, $P_{R,DN}$, as functions of the resident-independent invader growth rate, Φ^- , and the maximum resident-dependent invader growth rate, Φ^+ . (A) When invaders are generalists (i.e., when $\Phi^- > 0$ and $\Phi^+ = 0$), P_R varies nonlinearly with respect to the invader growth rate, as slow invasion promotes the appearance of *de novo* mutations while fast invasion facilitates the establishment of standing genetic variants by relieving mutants of competition with residents. (B) The same is true when invaders are specialists (i.e., when $\Phi^- > 0$ and $\Phi^+ = 0$), except standing genetic variants are less likely to establish because invaders can fail to encounter residents and thus fail to reproduce, causing residents to be displaced at a slower pace. Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: Eqs. 8-9 & Eq. 3 evaluated with Eqs. 8-9. Unless stated otherwise, parameter values are $b = 1$, $d = 0.03$, $\delta_b = 3$, $\delta_d = 1$, $K = 10^5$, $\Phi^- = \Phi^+ = 0$, and $U = 0.1$.

4.3.1 A lower resident birth rate can promote rescue from generalist invaders

Another factor that has a strong effect on P_R is the resident density independent birth rate, b (Fig. 2A). As b rises from near the resident density independent death rate, d , P_R initially increases due to b augmenting mutant fitness (Figs. 2A-B). However, once b is sufficiently greater than d , the effect of b on P_R depends on the invader growth rate, $\Phi^- + \bar{n}_R(t)\Phi^+$: when $\Phi^- + \bar{n}_R(t)\Phi^+$ is low enough to favor rescue via *de novo* mutation, P_R continues to increase with b due to the greater density of potentially mutating residents, $\bar{n}_R(t)$ (Fig. 2A). In contrast, when $\Phi^- + \bar{n}_R(t)\Phi^+$ is high enough to favor rescue via standing genetic variation, a lower b value can promote rescue by shrinking the total probability of a mutant event (i.e., birth or death) and hence reducing the variance in mutant fitness caused by such events (Fig. 2A). For this benefit of lower b values to appreciably increase P_R , invaders need to be generalists with an adequately large resident-independent growth rate, Φ^- (Figs. 2A-B). Otherwise, if invaders are highly specialized (i.e., $\Phi^- \approx 0$ and $\Phi^+ > 0$), then a larger b value is favorable (Fig. 2B). This is because the lower b value reduces the initial resident-dependent invader growth rate, $\bar{n}_R\Phi^+$, which—in the case of highly specialized invaders—can greatly prolong the time until mutants acquire a non-negligible advantage over residents, negating the benefit of less mutant variance. Importantly, even though a boost in d has the same effect on mutant fitness and mutational input as a decrease in b , larger d values do not enhance P_R (Figs. 2C-D), because they increase mutant variance.

4.3.2 Birth competition can facilitate rescue from both generalist and specialist invaders

Similar to a reduction in the resident density independent birth rate, b , an increase in the sensitivity of residents to birth competition, δ_b , can hinder or facilitate rescue depending on the invader growth rate, $\Phi^- + \bar{n}_R(t)\Phi^+$ (Figs. 3A-B). In the case of generalist invaders (i.e., invaders with $\Phi^- > 0$ and $\Phi^+ \approx 0$; Fig. 3A), the effect of δ_b on P_R resembles that of a smaller b value: when generalists invade slowly, δ_b hinders rescue by diminishing mutational input; whereas when generalists invade quickly, δ_b facilitates rescue by decreasing mutant variance (Fig. 3A). Moreover, the effect of δ_b on P_R also resembles that of a smaller b value in the case of rapidly invading specialists (i.e., invaders with $\Phi^- \approx 0$ and $\Phi^+ \gg 0$; Fig. 3B). In such a case, δ_b reduces P_R by slowing invasion and thus extending the time until standing genetic variants acquire an appreciable advantage over residents. In the case of specialists invading at a low enough rate for *de novo* mutation to be the likely path to rescue, however, larger δ_b values can promote rescue under regimes where smaller b values hinder it (compare square-marked curves in Figs. 2B & 3B). This happens because, unlike density independent factors, the effect of competition on mutant fitness decays with resident density, $\bar{n}_R(t)$,

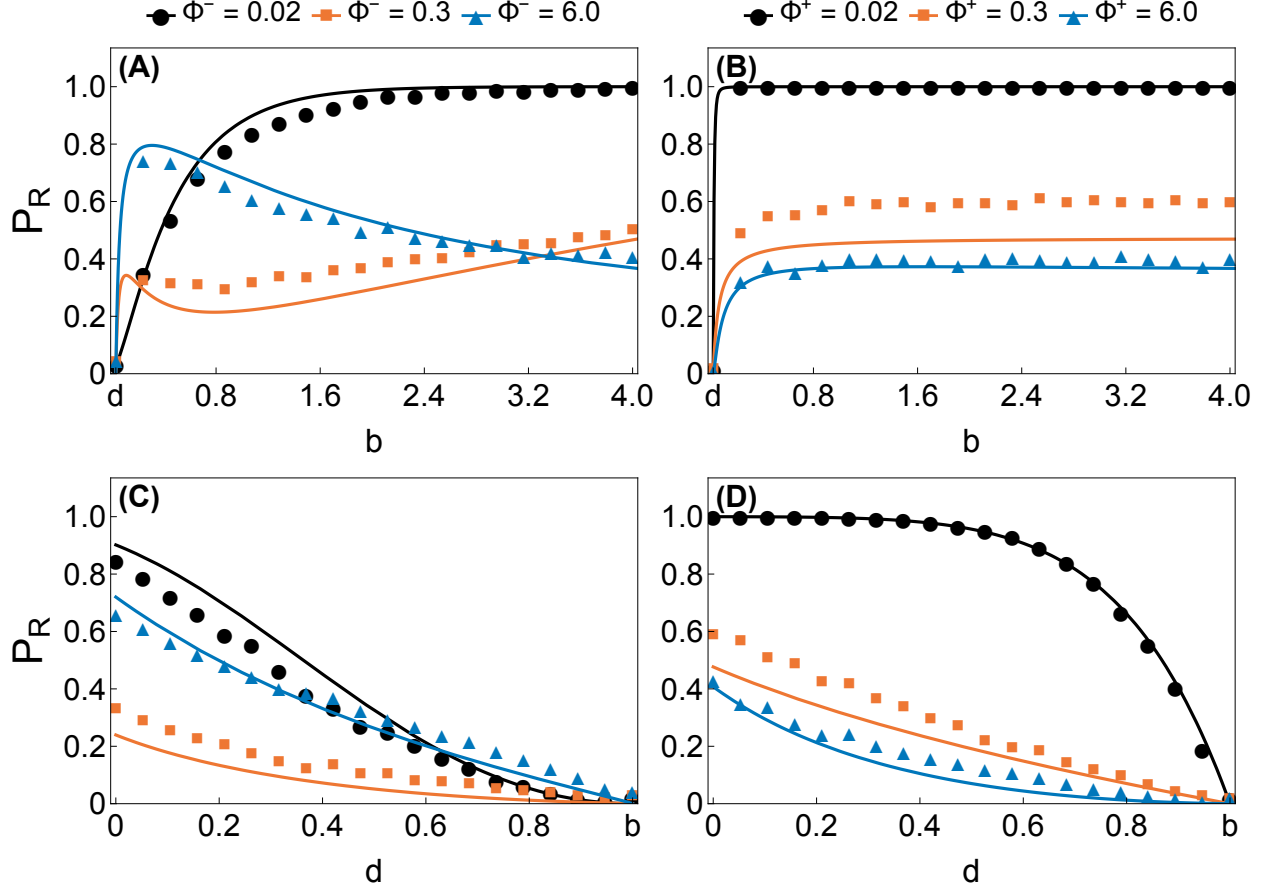


Figure 2: The probability of evolutionary rescue, P_R , as a function of the resident density independent birth rate, b , and death rate, d . (A) In the case of rapidly proliferating generalist invaders (triangle-marked curve), a reduction in b can facilitate rescue by reducing variance in mutant fitness; whereas in the case of slowly proliferating generalists (circle-marked curve), such a reduction hinders rescue by decreasing the density of potentially mutating residents. (B) The benefit of low b values nearly disappears when invaders are specialists. (C-D) Larger d values never promote rescue because they increase mutant variance. Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: Eq. 3 evaluated with Eqs. 8-9. Unless stated otherwise, parameter values are $b = 1$, $d = 0.03$, $\delta_b = 3$, $\delta_d = 1$, $K = 10^5$, $\Phi^- = \Phi^+ = 0$, and $U = 0.1$.

over time, which can allow the benefit of less mutant variance to outweigh the cost of lower mutant fitness. In turn, as long as invaders are specialized (i.e., when $\Phi^- \approx 0$) enough for δ_b 's reduction in both $\bar{n}_R(t)$ and the invader growth rate, $\Phi^- + \bar{n}_R(t)\Phi^+$, to effectively counterbalance one another, the decrease in mutant variance caused by δ_b overshadows the corresponding reduction in *de novo* mutational input. Notably, because an increase in the sensitivity of residents to death competition, δ_d , augments mutant variance, P_R tends to decrease with a boost in δ_d irrespective of $\Phi^- + \bar{n}_R(t)\Phi^+$ (Figs. 3C-D).

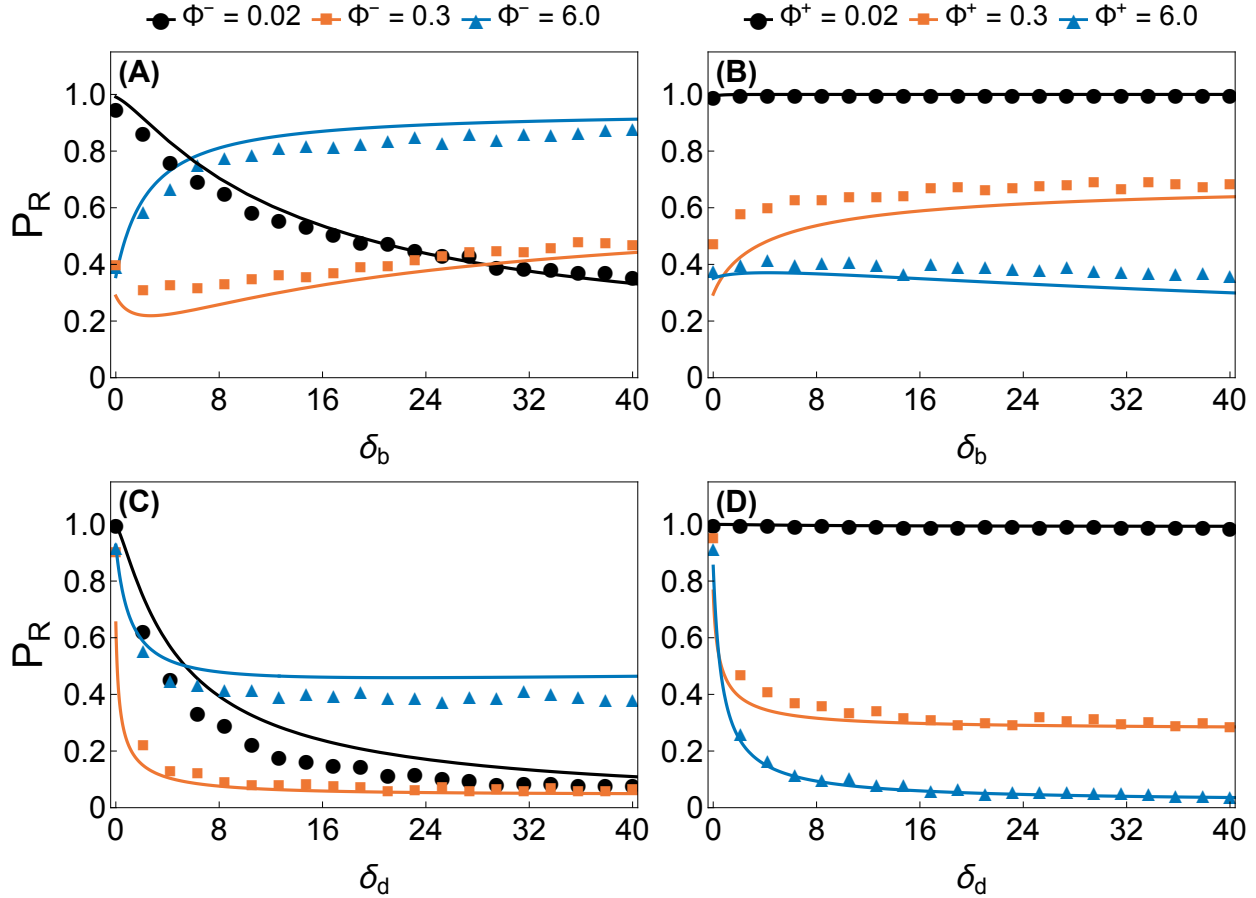


Figure 3: The probability of evolutionary rescue, P_R , as a function of the sensitivity of residents to birth competition, δ_b , and death competition, δ_d . (A) In the case of rapidly proliferating generalist invaders (triangle-marked curve), a larger δ_b value can facilitate rescue by reducing the density of residents competing with mutants; whereas in the case of slow-invading generalists (circle-marked curve), such a change hinders rescue by lowering the density of potentially mutating residents. (B) If invaders are specialists, then a boost in δ_b can hinder the establishment of standing genetic variants by slowing invader growth (triangle-marked curve). In contrast, such a reduction in the invader growth rate can be enough to offset the negative effect of δ_b on mutational input, allowing an increase in δ_b to promote rescue by lowering the demographic variance experienced by *de novo* mutants (square-marked curve). (C-D) Larger δ_d do not appreciably promote rescue because they augment mutant variance. Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: Eq. 3 evaluated with Eqs. 8-9. Unless stated otherwise, parameter values are $b = 1$, $d = 0.03$, $\delta_b = 3$, $\delta_d = 1$, $K = 10^5$, $\Phi^- = \Phi^+ = 0$, and $U = 0.1$.

4.3.3 Birth competition is only beneficial in the presence of death competition

Given that a reduction in the resident birth rate and a boost in the resident death rate have opposite effects on mutant variance, an interesting remaining question is how the latter affects our finding that a smaller resident birth rate can promote rescue. As shown in Fig. 4A, we find that, as the resident density independent death rate, d , approaches zero, the optimal resident density independent birth rate, b , declines (Fig. 4A). This decline occurs because b 's constant, linear influence on mutant variance allows smaller b values to

offset the effect of both d and competition on mutant variance, making a reduction in b beneficial even when $d = 0$. In contrast, as the sensitivity of residents to death competition, δ_d , decreases, the range of resident sensitivities to birth competition, δ_b , promoting rescue also narrows (Fig. 4B). Thus, unlike a reduction in b , an increase in δ_b only offsets the effect of δ_d on mutant variance. The reason for this difference between b and δ_b is that competition's effect on mutant fitness and variance decay with resident density over time, causing it to have a weaker effect on P_R than density independent factors.

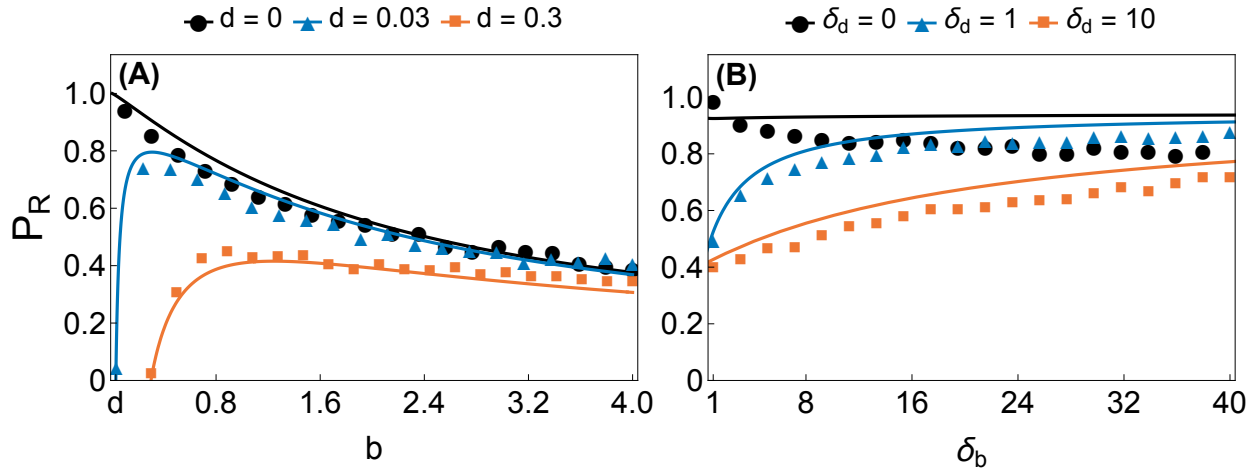


Figure 4: The probability of evolutionary rescue, P_R , as a function of the resident density independent birth rate, b , and the sensitivity of residents to birth competition, δ_b . (A) As the resident density independent death rate, d , decreases, smaller b values become optimal, meaning that a reduction in b promotes rescue by offsetting the variance generated by both density independent and competition-induced mortality. (B) In contrast, as the sensitivity of residents to death competition, δ_d , increases, the range of δ_b values promoting rescue diminishes. This means that, because the effect of competition on mutant variance declines over time with resident density, larger δ_b values are only strong enough to offset the variance generated by greater δ_d values. Markers: proportion of $2(10)^3$ replicate simulations that ended in mutant survival; solid curves: Eq. 3 evaluated with Eqs. 8-9. Unless stated otherwise, parameter values are $b = 1$, $d = 0.03$, $\delta_b = 3$, $\delta_d = 1$, $K = 10^5$, $\Phi^- = 6$, $\Phi^+ = 0$, and $U = 0.1$.

5 Discussion

We analyzed the probability of evolutionary rescue, P_R , from an invading species. We showed that P_R varies nonlinearly with the invader growth rate (Fig. 1A), as slow invasion promotes rescue by slowing resident decline and thus providing more time for *de novo* mutations to appear, while fast invasion facilitates rescue by relieving standing genetic variants of competition with their resident ancestors. The latter benefit is less pronounced when invaders are specialists that rely on residents to reproduce because, in such a case, invaders sometimes fail to locate residents and hence fail to reproduce, reducing the rate at which invaders displace residents (Fig. 1B). Our main result was that a lower resident birth rate—whether it stems from a

lower resident density independent birth rate or stronger birth-limiting competition—can facilitate rescue by reducing variance in mutant fitness (Figs. 2A-B & 3A-B), whereas a greater resident death rate tends to hinder rescue by enhancing mutant variance (Figs. 2C-D & 3C-D). When the lower resident birth rate is density independent, it facilitates rescue by offsetting variance generated by both density independent mortality and competition (Fig. 4A); whereas when the lower birth rate stems from stronger competition, it promotes rescue only by offsetting the variance generated by death-promoting competition (Fig. 4B). This difference is due to competition's effect on mutant variance decaying with resident density over time.

At least two other studies have explicitly modeled evolutionary rescue from invasion (Van Dyken, 2020; Longcamp & Draghi, see Chapter 2). One such study is that of Van Dyken (2020), who modeled rescue from invaders spreading through both space and time. In that study, Van Dyken (2020) found that rescue via *de novo* mutation becomes more likely with a reduction in the selective advantage of invaders, as slower invasion translates to more time for mutations to occur. Van Dyken (2020) also found that rescue is more likely when invaders compete with residents for a spatially ubiquitous resource (exploitation competition) instead of directly harming residents (interference competition), because, in the former scenario, the competitive advantage of mutants is not restricted to spatial locations where invaders are present (Van Dyken, 2020). Our work complements that of Van Dyken (2020) by highlighting conditions that can improve the chance of rescue from direct interactions with invaders. In particular, we showed that rescue via *de novo* mutation becomes more likely when invaders are specialists relying on residents to reproduce because, in such a case, invaders sometimes fail to encounter residents and thus fail to reproduce, causing residents to decline more slowly. In contrast, rescue via standing genetic variation is promoted by quickly-proliferating invaders that rapidly displace residents, relieving mutants of intraspecific competition.

The other study of evolutionary rescue from invasion is that of Longcamp and Draghi (see Chapter 2), who modeled the invasion of a resident niche-constructing population by invaders that exploit the resident-constructed habitats. In that study, rescue was found to be more likely under a fecundity cost of construction than a mortality cost because the mortality cost generated greater variance in mutant fitness (Longcamp & Draghi, see Chapter 2). For the same reason, birth-limiting competition was found to be less harmful than death-promoting competition in a model of antibiotic resistance (Czuppon et al., 2023). Here we showed that a lower resident birth rate is not only less harmful than a higher resident death rate but can even facilitate rescue relative to a larger birth rate. When invasion occurs quickly enough to favor standing genetic variation, the lower resident birth rate always promotes rescue, assuming it remains sufficiently greater than the resident death rate. In contrast, if invasion is slow enough for *de novo* mutation to be the

likely path to rescue, then a lower birth rate promotes rescue only when (i) it stems from stronger competition so that its effect on mutant fitness decays over time with resident density; (ii) invaders rely almost entirely on residents to proliferate, ensuring that the cost of fewer potentially mutating residents is offset by a reduction in the invader growth rate; and (iii) competition affects both births and deaths, with the former diminishing the variance generated by the latter.

To simplify our analysis, we made several assumptions that could be worth relaxing in future work. For instance, we assumed that carrying capacity is large so that variance in resident population size can be ignored. Given that deaths increase the variance in a focal type's fitness, relaxing this assumption may reveal that a higher resident death rate—despite increasing mutant variance—can promote the establishment of standing genetic variants in small populations, and therefore hinder the appearance of *de novo* mutations, by causing residents to go extinct at an earlier time than expected. Moreover, we assumed that residents randomly encounter invaders and other residents. Relaxing this assumption could also yield counterintuitive results, as it may show that a tendency for residents to seek out competitive interactions—perhaps due to an ecological trap where such interactions appear as though they will be beneficial—can promote rescue by reducing mutant variance. Finally, we assumed that the resident population declines solely due to its interaction with the invading species, meaning that the resident population follows a logistic-like trajectory, where it remains near equilibrium until invaders become sufficiently common. Consequently, still unclear is whether our results hold under different resident trajectories, such as the exponential curve that would be formed if carrying capacity were to decay toward a value of zero. Similar to the case of invasion, the decay in carrying capacity could occur independently of residents (e.g., habitat loss) or could depend on residents (e.g., resident-induced habitat loss). Also unclear is whether any of our results would hold in a scenario where a population suffering from inbreeding depression is saved via the immigration of new alleles (Whiteley et al., 2015). What may be the case is that, similar to standing genetic variants in our model, immigrants with low birth and death rates have the highest chance of rescuing an inbred population because they experience little variance in fitness.

In summary, we modeled evolutionary rescue from an invading species. Our main result was that a lower resident birth rate, whether it stems from a lower density independent birth rate or stronger birth-limiting competition, can facilitate rescue by reducing variance in mutant fitness. This most readily occurs when invaders proliferate rapidly enough for standing genetic variation to be the likely means of rescue. Our findings suggest that long-lived species, despite evolving at a slower pace, may be more likely to withstand rapid invasion than short-lived species.

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