Title

How density dependence, genetic erosion, and the extinction vortex impact evolutionary rescue

Running head: Density dependence & evolutionary rescue

Authors

Scott W. Nordstrom[1,2], Ruth A. Hufbauer[3], Laure Olazcuaga[4], Lily F. Durkee[3] and Brett A. Melbourne[1, *]

[1]Department of Ecology & Evolutionary Biology, University of Colorado, Boulder, Boulder, CO, 80309

[2]BioFrontiers Institute, University of Colorado, Boulder, Boulder, CO, 80303

[3]Department of Agricultural Biology and Graduate Degree Program in Ecology, Colorado State University, Fort Collins, CO 80523

[4]Department of Agricultural Biology, Colorado State University, Fort Collins, CO 80523

[*] For correspondence: brett.melbourne@colorado.edu

Abstract

Following severe environmental change that reduces mean population fitness below replacement, populations must adapt to avoid extinction, a process called evolutionary rescue. Models of evolutionary rescue demonstrate that initial size, genetic variation, and degree of

maladaptation influence population fates. However, many models feature populations that grow without negative density dependence or with constant genetic diversity despite precipitous population decline, assumptions likely to be violated in conservation settings. We examined the influences of density-dependent growth and erosion of genetic diversity simultaneously on populations adapting to novel environmental change using stochastic, individual-based simulations. Density dependence decreased the probability of rescue and increased the probability of extinction, especially in large and initially well-adapted populations that previously have been predicted to be at low risk. Increased extinction occurred shortly following environmental change, as populations under density dependence experienced more rapid decline and reached smaller sizes. Populations that experienced evolutionary rescue lost genetic diversity through drift and adaptation, particularly under density dependence. Populations that declined to extinction entered an extinction vortex, where small size increased drift, loss of genetic diversity, and the fixation of maladaptive alleles, hindered adaptation, and kept populations at small densities where they were vulnerable to extinction *via* demographic stochasticity.

Key words:

Genetic variation; adaptation; eco-evolutionary dynamics; population dynamics

Introduction

Adaptation to novel environments is often necessary for populations to persist in this current era of large-scale anthropogenic change and habitat alteration. Novel or sudden

environmental change can abruptly render a population poorly adapted to its habitat. When the environmental change is so severe that mean population fitness falls below the replacement rate, the population will go extinct if it does not adapt. This process of populations adapting to severe change in environmental conditions sufficiently to avoid extinction is called evolutionary rescue (Gomulkiewicz and Holt 1995, Carlson et al. 2014).

Gomulkiewicz and Holt (1995) formalized the idea of evolutionary rescue, highlighting the "U"-shaped demographic trajectory where populations initially decline due to maladaptation to novel environmental conditions, followed by adaptation that allows populations to avoid extinction and return to their original size. They found that evolutionary rescue was most likely to occur in large populations with high genetic diversity and a relatively low degree of maladaptation to their environment (i.e., population mean phenotype close to that favored by the novel environment) at the time of the environmental change. Subsequent work in controlled experiments has confirmed the importance of these processes to evolutionary rescue (e.g., Bell and Gonzalez 2009, Agashe et al. 2011, Hufbauer et al. 2015).

Models examining rescue often rely on assumptions of density-independent growth and constant additive genetic variance (Kopp and Matuszewski 2014), potentially making the findings less applicable to many conservation targets. For example, Gomulkiewicz and Holt's (1995) model assumes that population growth is density independent, and that populations can grow unconstrained after rescue. This assumption will often be violated in conservation settings where habitat loss, degradation, or other environmental change limits resource availability and increases intraspecific competition. Additionally, Gomulkiewicz and Holt's (1995) model (and others, e.g., Chevin and Lande 2010) rely on quantitative genetics results from Lande (1976),

67 which assume that population size is infinite and therefore that additive genetic variance
68 remains constant over time. However, threatened populations experiencing rapid decline are
69 likely to experience erosion of genetic diversity (Frankham et al. 1999, Bijlsma and Loeschke
70 2011, Pauls et al. 2013) that may decrease their ability to adapt to novel conditions (Houle
71 1992), possibly hindering rescue.

72 Incorporating demographic and genetic processes such as negative density dependence
73 and genetic erosion will improve the accuracy of predictions from models of evolutionary
74 rescue for populations and species of conservation concern. Negative density dependence such
75 as that caused by intraspecific competition should constrain evolutionary rescue by accelerating
76 population declines and slowing population recovery from reduced size (Chevin and Lande
77 2010). Some models of rescue and environmental tracking have included constrained
78 population size by including a ceiling-type carrying capacity (e.g., Lande 1993, Orr and Unckless
79 2014, Lyberger et al. 2021), but growth below carrying capacity in these models was density
80 independent, failing to capture how intraspecific competition or other negative density-
81 dependent processes depress population growth in declining and recovery phases of rescue.
82 Orr and Unckless (2008) analyzed a density-dependent model of adaptation with a single locus
83 trait, finding that density dependence impedes rescue by reducing the survival of novel
84 beneficial mutants. However, they did not investigate effects of density dependence on
85 adaptation from standing genetic variation. Relaxing the assumption of density independence,
86 Chevin and Lande (2010) found that density dependence impedes rescue, but their model held
87 genetic diversity constant, possibly ignoring effects of drift for populations reaching low
88 density. A population's additive genetic variance for a trait under selection in a population

influences its rate of adaptation to novel conditions (Lande 1976, Houle 1992); loss of this genetic diversity as populations decline in response to environmental change should slow rates of adaptation, increasing the time until rescue or making eventual rescue less likely. Populations that are small at the onset of environmental change are especially likely to experience genetic erosion through drift, inbreeding, and the fixation of maladaptive alleles (Hedrick and García-Dorado 2016). Boulding and Hay (2001) and Barfield and Holt (2016) modeled populations allowing genetic diversity to change over time, finding that drift could occur potentially slowing adaptation, but they did not focus on interactions of these factors with competition that constrains population growth rates.

Modeling these demographic and genetic processes simultaneously is important, as they can interact and reinforce each other, creating a positive feedback loop of demographic and genetic decline that populations in the process of evolutionary rescue may be drawn into (Fig. 1). In the conservation biology literature this positive feedback loop is called an "extinction vortex" (Gilpin and Soulé 1986). In an extinction vortex (Fig. 1) small populations experience drift and inbreeding, reducing fitness directly *via* increased genetic load and indirectly by erosion of genetic variation and reducing the rate of future adaptation (Frankham et al. 1999), keeping populations small or causing them to decline further to a smaller size. This in turn leads to further genetic erosion or increased genetic load, reduced fitness, and further declines in population size until inevitably the population is highly vulnerable to extinction *via* stochasticity in births and deaths, demographic heterogeneity, and sampling variation in sex ratios (Melbourne and Hastings 2008). This feedback loop potentially opposes or resists the evolutionary rescue process, where adaptation increases fitness, increasing population growth

rates to the point where population size stabilizes and eventually returns to original size. Negative density dependence has the potential to disrupt evolutionary rescue and draw populations back towards the vortex by accelerating reductions in, or slowing increases in, population growth rate, fitness, and genetic diversity (Fig. 1). The original conceptualization of an extinction vortex does not necessarily assume a degraded or altered habitat where adaptation is necessary for persistence (Gilpin and Soulé 1986). Perhaps for this reason, to our knowledge the extinction vortex has not been explicitly studied in the context of evolutionary rescue and adaptation to novel environments. Yet, these interactive, combined effects of small population size could be of particular importance in evolutionary rescue, as by definition populations undergoing rescue initially decline to smaller sizes, which could initiate entry into an extinction vortex. The extinction vortex also necessitates understanding how density dependence and genetic erosion interact with other important determinants of the success of rescue, e.g., the initial conditions with respect to population size, genetic diversity, and degree of maladaptation (Gomulkiewicz and Holt 1995, Bell and Gonzalez 2009, Agashe et al. 2011). For example, large populations adapting to sudden environmental change might never approach the extinction vortex under density-independent population growth but might more often enter the vortex under density dependence. Ultimately, under realistic conditions in natural settings, evolutionary rescue might often involve a population escaping from an extinction vortex.

Here, we relax the assumptions of density independence and constant genetic diversity to examine their simultaneous effects on evolutionary rescue and the extinction vortex, establishing a connection between these two major concepts in conservation biology. We do

133 this by combining stochastic Ricker population growth and a genetic framework with a finite-
134 locus quantitative trait in the same individual-based model. We simulate both rapid (15
135 generations) and longer (50 generations) timescales. We address two main objectives. First, we
136 assess how negative density dependence interacts with initial conditions with respect to
137 population size, additive genetic variance, and degree of genetic maladaptation in the novel
138 environment to influence trajectories of population size, probabilities of extinction and rescue,
139 additive genetic variance and fitness over time. Second, we test for the presence of an
140 extinction vortex and evaluate how negative density dependence influences this vortex.
141

142 **Methods**

143 ***Model description***

144 We explored evolutionary rescue and extinction using stochastic simulations. This
145 approach allowed us to incorporate several stochastic demographic and genetic processes that
146 are important for rescue or extinction (Melbourne and Hastings 2008, Carlson et al. 2014).
147 While extinction is straightforward to identify, the classical criterion of evolutionary rescue
148 (population growth rate above replacement) is more difficult to operationalize for individual
149 populations because rescued populations may decline in size or growth rate due to
150 demographic or genetic stochasticity, inbreeding, and genetic load and revert to an unrescued
151 state. We operationalize rescue using two separate criteria: (1) expected number of offspring
152 per individual ($\bar{R}_t$, as defined below) greater than 1 for three consecutive generations
153 (hereafter "fitness-based rescue") and (2) populations exceeding their initial size for three
154 consecutive generations (hereafter "size-based rescue").

155 We derive a discrete-time individual-based model with diploid genetic inheritance that
156 has general features of the semelparous life history strategy common to many sexually
157 reproducing organisms. Our genetic submodel is a finite quantitative locus model with random
158 and independent segregation of alleles, similar to that of Boulding and Hay (2001). Our
159 demographic submodel is based on an expanded stochastic Ricker model (Ricker 1954,
160 Melbourne and Hastings 2008). We use this model because it is a common form of density
161 dependence while also accurate for the population dynamics of the model organism *Tribolium*
162 *castaneum* in a discrete-time laboratory culture (Melbourne and Hastings 2008) thus
163 complementing current and future experiments of rescue using this system (e.g., Agashe et al.
164 2011, Hufbauer et al. 2015, Stewart et al. 2017), effectively grounding our simulations in
165 concrete details of biology.

166 Each individual has an intrinsic fitness determined by genetic and environmental
167 components. The genetic component is determined by each of *m* biallelic diploid loci. Each
168 allele-copy contributes a positive ($1/(2\sqrt{m})$) or negative (-$1/(2\sqrt{m})$) amount to the genotype
169 of individual *i*, g_i, which is the sum of allele-copies across all loci. Parental alleles are segregated
170 independently to offspring during reproduction. The additive genetic variance in the population
171 is thus

$$\sigma_a^2 = 2\sum_{j=1}^{m}\frac{p_j(1-p_j)}{m} = 2\left(p'(1-p') - \sigma_p^2\right) \quad (1)$$

172 where p_j is the frequency of the positive allele at locus *j* across the population and p' and σ_p^2 are
173 respectively the mean and variance of allele frequencies across all loci (see Lynch and Walsh

174 1998 and Supporting Information A). An individual's phenotype, z_i, is the genotype, g_i, plus a
175 deviation due to environmental variation drawn from a normal distribution with mean zero and
176 variance σ_e^2. Intrinsic fitness, W_i, the expected fitness of an individual in the absence of density
177 effects, is determined by Gaussian selection (Lande 1976, Gomulkiewicz and Holt 1995) such
178 that $W_i = W_{max}\, exp(-(z_i - \theta)^2 \,/\, 2w^2)$, where θ is the optimal phenotype, w^2 is the width of
179 the fitness landscape and is inversely proportional to selection strength , and W_{max} is the
180 maximum intrinsic fitness (i.e., intrinsic fitness of an individual with the optimal phenotype).
181 The expected number of offspring of an individual, R_i, is determined according to a Ricker
182 function that reduces fitness due to density dependence such that $R_i = W_i e^{-\alpha N_t}$, where α is
183 the strength of density dependence and N_t is the population size at time t (Ricker 1954). The
184 realized fitness of an individual is a stochastic process as described next.
185 Each individual is born with a randomly determined sex assigned with equal probability.
186 Each generation, each female mates with a male chosen uniformly at random from the
187 population. In practice, mating with multiple males during a single generation occurs in female
188 *T. castaneum* (Pai and Yan 2020); however, our model makes the simplifying assumption that
189 each female mates with exactly one male while males can mate with any number of females,
190 including zero. This assumption corresponds to well-documented sperm competition and the
191 fertilization precedence of the last male mate *in T. castaneum* (e.g., Arnaud et al. 2001). Each
192 female produces a Poisson-distributed number of offspring with mean $2R_i$. Generations are not
193 overlapping, i.e., only one breeding generation is present at a time. As such, if all females have
194 zero offspring, extinction is guaranteed in the next time step. We use non-overlapping
195 generations both for comparison with other models of adaptation and evolutionary rescue

(e.g., Gomulkiewicz and Holt 1995, Boulding and Hay 2001, Chevin and Lande 2010) and because it provides a tractable setup for experimental validation using the *Tribolium* model system (e.g., Hufbauer et al. 2015, Stewart et al. 2017).

Experimental Design, Simulations, and Analysis

We simulated populations with m = 25 loci and an environmental change that moved the phenotypic optimum from its prior state of 0 to θ = 2.8. Because θ > 0, the environment in our simulations favors a shift toward positive alleles. Populations were founded with positive and negative alleles assigned to each individual's loci at random with equal probability such that across simulation trials the expected population mean genotype was zero, although due to sampling there was variation in initial population mean genotype across trials. Thus, to reach the new phenotypic optimum θ = 2.8, a typical population needed to change from 25 positive allele copies (out of 50) per individual to 39 allele copies per individual (see Supporting Information B). All simulations were run with maximum intrinsic fitness W_{max} = 2, non-genetic phenotypic variance component σ_e^2 = 0.5, and w^2 = 3.5 as the width of the fitness landscape Parameter choices are justified in full in Supporting Information C; briefly, parameters were chosen to resemble observations from the *T. castaneum* model system (e.g., Wade et al. 1996, Hufbauer et al. 2015) while also being realistic for a variety of natural populations (e.g., Hone et al. 2010, Bonnet et al. 2022). We chose our fitness landscape width (w^2) based on analysis of the model of Gomulkiewicz and Holt (1995) demonstrating that rescue is only likely to occur when the width of the fitness landscape is 1-10 times the amount of phenotypic variance (Supporting Information C). With these parameter values, mean intrinsic fitness in the typical

population immediately following the environmental change was approximately 0.65 such that populations were sufficiently challenged to the point that both rescue and extinction were possible on rapid timescales and resembled observed population dynamics in previous studies with *T. castaneum* (e.g., Hufbauer et al. 2015, Stewart et al. 2017). All parameter values are summarized in Supporting Information, Table D1.

To evaluate the effects of negative density dependence on evolutionary rescue and its potential interactions with initial population size and amount of genetic diversity, we conducted a 2 x 2 x 2 factorial simulation experiment (yielding eight treatments, Table 1). Populations were simulated with the following parameter values: Negative density dependence absent (α = 0; hereafter "density-independent populations") or present (α = 0.0035, hereafter "density-dependent populations", value from Melbourne and Hastings 2008); initial size N_0 = 100 or N_0 = 20, hereafter referred to as "large" and "small" populations in reference to their initial state rather than their eventual size; initial genetic diversity high ($\sigma_a^2 \approx 0.5$, heritability approx. 0.5) or low ($\sigma_a^2 \approx 0.25$, heritability approx. 0.33, Lynch and Walsh 1998), hereafter "high diversity" and "low diversity" populations, again in reference to their initial state rather than their eventual level of diversity. These initial heritabilities are similar to those found in *T. castaneum* by Wade et al. (1996), who found heritability of fitness of approximately 0.44 in outbred lines and approximately 0.25 in lines subject to two generations of inbreeding. To simulate populations with initial low diversity, at the beginning of each simulation, six loci were set to fixation for the positive (adaptive) allele and six were set to fixation for the negative (maladaptive) allele, leaving only 13 loci (approximately half of the genome) available for selection to act upon while leaving the expected mean population genotype unchanged

compared to the high-diversity treatments. Stochasticity in the founding of populations produced sampling variation in the initial mean population genotype; we took advantage of this sampling variation to also analyze a third important determinant of a population's fate, initial degree of maladaptation (Gomulkiewicz and Holt 1995, Barfield and Holt 2016), and its interactions with density dependence as described in paragraphs below. A subtle point is that expected initial additive genetic variance, $(\sigma_a^2)_0$, across replicate populations is slightly less than the target values because of the sampling variation in allele frequencies associated with founding the populations (Eq. 1). Likewise, due to sampling variation in initial allele frequencies, there is more variation in initial mean population genotypes and phenotypes among small populations than among large populations.

To assess adaptation and extinction on short time scales, we ran 4000 simulations lasting fifteen generations for each treatment. All simulations were performed in R, version 4.1.2 (R Core Team, 2020). In each simulation, for each generation we tracked population size (N_t), mean genotype ($\bar{g}_t$), mean phenotype ($\bar{z}_t$), mean intrinsic fitness ($\overline{W}_t$), where all means are across individuals within a population, as well as additive genetic variance ($(\sigma_a^2)_t$), and the proportion of loci at fixation within the population for both alleles. Throughout our analysis, we will use an overbar to represent a mean across individuals within one population in one timestep. We define adaptation as an increase in $\overline{W}_t$. Because fitness is optimized at a positive genotypic value (i.e., $\theta > 0$) and fitness is a monotonic function of maladaptive genetic load, adaptation can be equivalently defined as an increase in $\bar{g}_t$ or a reduction in genetic load, $(\theta - \bar{g}_t)$.

As a first evaluation of how density dependence, population size, and genetic diversity influence rescue and extinction over 15 generations, we visualized mean population size over time for each treatment group. This visualization shows whether the U-shaped population trajectory characteristic of rescue (Gomulkiewicz and Holt 1995) occurs, and if so, how it differs by treatment. All mean population sizes were estimated including extinct populations as size zero, thus incorporating the possibility of extinction into expected population size in each time step. To quantify the average dynamics of the model we also estimated and visualized the following response variables, averaged over the ensemble of all populations within a treatment: mean population genotype ($\langle \bar{g}_t \rangle$), mean intrinsic fitness ($\langle \overline{W}_t \rangle$), mean additive genetic variance ($\langle \sigma_a^2 \rangle_t$), and number of loci at fixation for positive and negative alleles across all populations in each generation. To differentiate ensemble means from population means for a single trial, we use angle brackets ($\langle\rangle$) to denote means of population-level means across an ensemble of simulations. Because these variables are undefined for extinct populations (which have no genotype, fitness, or additive genetic variance), estimating unconditional expectations for each generation was impossible, and thus we estimated these means conditioned on survival or extinction at the end of the simulation. Differences in ensemble means for these variables between density-independent and density-dependent populations may be due directly to density dependence, but they may also be due indirectly to non-random extinctions. For example, a population with low mean intrinsic fitness may persist under density independence but go extinct under density dependence, lowering the ensemble mean population fitness for extant populations in a density-independent treatment but not affecting the ensemble mean of extant populations in a density-dependent treatment, confounding

comparisons between the treatments. As such, we only use plots of variables conditioned on survival/extinction for qualitative assessment, noting general patterns and interpreting them with caution.

To assess the probabilities of extinction and rescue on longer timescales, we ran 1000 simulations lasting 50 generations for each treatment. Due to the computational intensity of simulating large populations, we stopped a trial upon population size exceeding 10000. Because of this censoring, we did not analyze trajectories of demographic or genotypic state variables in these longer simulations, instead focusing only on the probabilities of extinction and rescue. We evaluated extinction in each generation in two ways: (1) instantaneous probability of extinction (the probability of extinction in generation *t* conditioned on surviving to *t*) and (2) cumulative probability of extinction over time (the probability of extinction in or prior to *t*). We also used two separate criteria of rescue (defined above): (1) fitness-based rescue and (2) size-based rescue. We also estimated time to rescue across treatments, defining time to rescue as the first of the three generations where the criterion was met (e.g., a population exceeding original size in generations 10-12 would be considered rescued in generation 10).

We estimated treatment effect sizes on the probabilities of extinction and rescue in the 50-generation trials using Bayesian generalized linear models (GLMs) in the R package *rstanarm,* 2.21.1 (Goodrich et al. 2020). We modeled both probabilities of extinction and rescue for each simulated population as a Bernoulli response and each of our three treatments (density dependence, initial size, genetic diversity) as categorical variables. Additionally, we estimated effects of initial degree of maladaptation, an important determinant of extinction risk (Gomulkiewicz and Holt 1995, Barfield and Holt 2016) by including the mean population

genetic load in the initial generation ($\theta - \bar{g}_0$, henceforth, "maladaptation") as a continuous explanatory variable. This was possible for our stochastic model because sampling variation in the founding of populations produced random among-trial variation in mean initial genotype. We estimated the effects of negative density dependence and its interactions with other variables by fitting a model with a four-way interaction and examining effect sizes as described in Supporting Information E. The uncertainty around these estimates was sufficiently low and could be arbitrarily improved by running more simulations. An effect size for a categorical variable (for example, initial population size) gives the effect of changing that variable's parameter on the log odds of extinction (or rescue) while holding all other parameters constant, while an effect size for maladaptation gives the change in the log odds of extinction (rescue) associated with switching five alleles from the positive allele to the negative allele in all individuals.

To test for the presence of an extinction vortex we first reversed and re-centered time for each extinct population to give τ, the time until extinction, such that populations had a common origin ($\tau = 0$) with consistent meaning across populations (Fagan and Holmes 2006). We define τ such that $\tau = 0$ is the first time step where the population is extinct (i.e., population size of 0 or 1), $\tau = 1$ is the last time step in which the population size is above 0 or 1, etc. The extinction vortex is characterized by accelerating declines in population size, genetic variation, and/or rate of adaptation as τ approaches zero (Gilpin and Soulé 1986, Fagan and Holmes 2006). Specifically, we calculated the rate of population growth ($r_\tau = ln(N_\tau/N_{\tau+1})$), the proportional loss of genetic diversity ($\nu_\tau = 1 - ((\sigma_a^2)_\tau/(\sigma_a^2)_{\tau+1})$), and the rate of adaptation ($1 - k_\tau$). k_τ is defined as in Gomulkiewicz and Holt (1995) such that $k_\tau = (\theta - \bar{g}_\tau) / (\theta -$

327 $\bar{g}_{\tau+1}$); as ($\bar{g}_{\tau} - \bar{g}_{\tau+1}$) approaches zero (i.e., as the rate of genotypic change approaches zero),
328 k_{τ} approaches 1 and $1 - k_{\tau}$ approaches 0. If adaptation proceeds at a constant rate, then k_{τ}
329 will be constant over time. We use these normalized rates of change instead of non-normalized
330 state variables (e.g., r_{τ} instead of N_{τ}) to facilitate comparisons across treatments and to remove
331 potential biases due to time until extinction. We estimated the ensemble means $\langle r_{\tau} \rangle$, $\langle v_{\tau} \rangle$, and
332 $1 - \langle k_{\tau} \rangle$ for all extinct populations in each treatment group using the 15-generation
333 simulations, where all state variables were tracked for each population. Following Fagan and
334 Holmes (2006), we considered decreasing $\langle r_{\tau} \rangle$ (accelerating decline in population size),
335 increasing $\langle v_{\tau} \rangle$ (accelerating loss of genetic diversity), or decreasing ($1 - \langle k_{\tau} \rangle$) (slowing rate of
336 adaptation) in the lead up to extinction (i.e., as τ approaches zero) to be evidence of an
337 extinction vortex. Likewise, we assessed how the shape of the extinction vortex varies between
338 density-dependent and density-independent populations qualitatively by noting differences in
339 the mean trajectories of the respective experimental treatments. We did not estimate rates for
340 $\tau = 0$ because $r = -\infty$ for a population at size zero and because additive genetic variance and
341 mean genotype are undefined for extinct populations.
342
343 **Results**
344
345 ***Population size***
346
347 For all combinations of genetic diversity and initial population size, in each generation
348 density-dependent populations were smaller on average than corresponding density-

349 independent populations (compare black and purple lines, Fig. 2). As populations declined, the
350 minimum average size (bottom of the "U") reached by density-dependent populations was
351 smaller than that of density-independent populations across treatments, with larger effects in
352 large populations (two-thirds reductions in minimum size) than small populations (one-third
353 reduction). Density-dependent populations were also smaller on average at the end of 15
354 generations; populations with low diversity had slightly larger reductions in final size (92-95%
355 reduction) than populations with high diversity (85 - 90% reduction). Regardless of starting
356 population size, density-dependent populations declined for longer and took longer to recover
357 their original size (dotted line, Fig. 2), if at all.
358
359 ***Extinction and rescue***
360 These differences in population size and growth rates over 15 generations translated
361 into more frequent extinctions for populations with density-dependent growth over the course
362 of the 50-generation simulations (Fig. 3). For both density-dependent and density-independent
363 populations, most extinctions occurred in the first 20 generations (Fig. 3*A*,*B*). Density
364 dependence increased extinction probabilities primarily in large populations (Table 2): among
365 large populations, density dependence increased the cumulative odds of extinction 5.0-fold for
366 high-diversity populations and 3.6-fold for low-diversity populations (Fig. 3*Ci*,*ii*). Among small
367 populations, density dependence increased the cumulative odds of extinction 1.5-fold and 2.0-
368 fold for high- and low-diversity populations, respectively (exponentiated effect sizes, Table 2,
369 Fig. 3*Ciii*,*iv*). Greater initial maladaptation (i.e., a mean initial genotype further from θ) was
370 associated with increased extinction risk across all treatments (Table 2, Fig. 3*D*). The effect of

371 maladaptation on extinction was stronger for large populations than small populations (Table
372 2); in large populations with high diversity in particular, only the most strongly maladapted
373 populations had considerable extinction risk (Fig. 3*Di*). Rather than strongly affecting the
374 relationship between maladaptation and extinction risk (i.e., the slope of the regression line,
375 Fig. 3*D*), density dependence increased extinction risk for all genotypes and only marginally
376 affected the relationship between genotype and maladaptation (i.e., raised the intercept while
377 slightly lowering the slope, Table 2, Fig. 3*D*). The combined result was that in large populations
378 with density-independent growth, only populations that were highly maladapted were at risk of
379 going extinct, but under density-dependent growth all populations had substantial extinction
380 risk (Fig. 3*D*).

381 The results for probability of rescue were qualitatively identical to those for probability
382 of extinction but with directions switched (Table 3 and Table F1, Figure F1). Likewise, the results
383 were qualitatively similar regardless of the type of rescue (i.e., fitness-based rescue or size-
384 based rescue). For both types, the probability of rescue declined with density dependence and
385 density dependence had stronger effects in large populations than small ones. Density
386 dependence increased mean rescue time only slightly (less than 1.5 generations) for all
387 population treatments except for large populations, for which density dependence increased
388 the time to return to initial size by 9-10 generations (Fig. F2). Notably, many small populations
389 were rescued instantaneously, i.e., they achieved three generations either exceeding initial size
390 or $\bar{R} > 1$ (or both) either instantly or after one time step (Table 3, Fig. 3*A*). These
391 "instantaneous rescues" happened primarily in small populations, comprising up to 16% of
392 rescue events observed in small population treatments. Both the overall frequency and the

393 proportion of total rescues comprising these instantaneous rescues decreased with low genetic
394 diversity and with density dependence. Pooling across all large treatment populations, only four
395 populations achieved instantaneous rescue of either type, comprising less than 1% of rescue
396 episodes for all large treatments. Extinctions occurred after both fitness- and size-based rescue
397 (Table 3). Extinction after rescue was more common for small populations than large ones, low-
398 diversity populations than high-diversity ones, as well as more common under density
399 dependence than density independence for both size- and fitness-based rescue.
400
401 ***Genetic variance, fixation, and fitness***
402 Over the first 15 generations, mean additive genetic variance, $\langle(\sigma_a^2)_t\rangle$, declined for all
403 treatments (Fig. 4*A*). Surviving density-dependent populations lost more genetic variation than
404 density-independent populations (Fig. 4*A*, solid black *vs.* solid purple lines); this effect was
405 greater in large populations. Across treatment and throughout time, populations that went
406 extinct had lower mean genetic variance prior to extinction than populations that survived (Fig.
407 4*A*, solid *vs.* dashed lines). This loss of genetic variation was associated with fixation for both
408 positive (adaptive) and negative (maladaptive) alleles (Fig. 4*B*,*C*). Populations that went extinct
409 typically had on average more loci at fixation for either allele than surviving populations (Fig.
410 4*B*,*C*, solid *vs.* dashed lines), although in large populations this difference did not appear until
411 the fifth generation, when populations approached minimum size. Of populations that survived,
412 density-dependent populations had higher rates of fixation for the positive allele than density-
413 independent populations (Fig. 4*B*, solid black *vs.* solid purple lines). Conversely, surviving
414 density-dependent and density-independent populations both typically had few loci at fixation

415 for the negative allele (Fig. 4*C*). Differences in the trajectories over time of genotype and
416 intrinsic fitness between density-dependent and density-independent populations conditioned
417 on survival or extinction were slight (Figs. G2, G3).
418
419 ***The extinction vortex***
420 Extinct populations in all treatments within the 15-generation simulations had negative
421 mean population growth rate ($\langle r_\tau \rangle$, Fig. 5*A*) and a non-zero mean loss of genetic diversity ($\langle \nu_\tau \rangle$,
422 Fig. 5*B*) for each time step. Declines in population growth rates and rate of loss of genetic
423 diversity accelerated, as expected for an extinction vortex (Gilpin and Soulé 1986, Fagan and
424 Holmes 2006) in the three generations prior to extinction ($\tau \leq 3$), with populations losing an
425 average of 17% of their standing additive genetic variance in the final generation preceding
426 extinction. In contrast, the rate of adaptation ($1 - \langle k_\tau \rangle$) decreased steadily over time in all
427 treatments rather than accelerating immediately prior to extinction (Fig. 5*C*). In the three
428 generations prior to extinction trends in density-dependent and density-independent
429 populations were similar. Four or more generations prior to extinction ($\tau > 3$), large density-
430 dependent populations had faster population decline (i.e., more negative $\langle r_\tau \rangle$) than large
431 density-independent populations, for which the rate of population decline was constant over
432 time (Fig. 5*A*). In contrast, density-dependent and density-independent populations had similar
433 rates of loss of genetic diversity (all differences in $\langle \nu_\tau \rangle$ between density-dependent and density-
434 independent treatments within 0.019, Fig. 5*B*). In the generation prior to extinction,
435 populations in all treatments had a mean adaptation rate of approximately zero (i.e., $\langle k_{\tau=1} \rangle \approx$

436 1 and $\langle \bar{g}_{\tau=1} \rangle \approx \langle \bar{g}_{\tau=2} \rangle$). Figures of non-normalized state variables conditioned on generation of
437 extinction, revealing qualitatively similar results, are available in Supporting Information H.
438
439 **Discussion**
440
441 We found that evolutionary rescue is constrained by the combined effects of negative
442 density dependence and loss of genetic variation. For all treatments, negative density
443 dependence decreased population growth rates, causing populations to decline more quickly,
444 reach lower densities, and stay at low densities for longer. This accelerated the loss of additive
445 genetic variance and slowed rates of adaptation. Ultimately, these demographic and genetic
446 processes often reinforced each other to produce an extinction vortex, precluding evolutionary
447 rescue. Here we discuss the effects of negative density dependence, loss of genetic variation,
448 and the extinction vortex on evolutionary rescue.
449
450 ***How negative density dependence constrains rescue***
451 Although negative density dependence increased extinction risk for all treatments (Fig. 3,
452 Table 2) and correspondingly reduced incidence of evolutionary rescue (Table 3), it had
453 disproportionate effects on large populations with relatively low maladaptation to the novel
454 environment. In the density-independent model of Gomulkiewicz and Holt (1995) and in
455 laboratory experiments in density-independent settings (Bell and Gonzalez 2009, Hufbauer et
456 al. 2015) large and better-adapted populations had minimal extinction risk. Our model agrees
457 with these previous findings: extinction was rare in large, density-independent populations that

458 were not strongly maladapted (Fig. 3, Table 1). Likewise, our results agree with Chevin and
459 Lande (2010) that density dependence hastens initial population declines and slows subsequent
460 population recovery during rescue (Figs. 2, 3*A*). We extend and refine these previous findings
461 by highlighting that density dependence increased extinction risk and reduced evolutionary
462 rescue more for large populations than small ones, narrowing the considerable advantage in
463 survival odds that large initial size confers in density-independent settings (Fig. 3*C*, Table 2).
464 Furthermore, under density dependence even large populations somewhat adapted to the
465 novel environment might go extinct (Fig. 3*D*). Thus, populations considered at low risk by
466 classic, density-independent models face a non-trivial threat of extinction from environmental
467 change under more realistic density-dependent population growth.

468 The effects of negative density dependence on population size, population growth rates,
469 and extinction primarily occurred during the initial generations after environmental change.
470 Interestingly, negative density dependence had only minimal effects on extinction and rescue
471 once populations were sufficiently large. Populations with mean intrinsic fitness greater than
472 one that were large enough to avoid extinction *via* demographic stochasticity approached a
473 carrying capacity (Fig. 3*A*). In this event, when populations again declined in size through
474 stochasticity, they regained positive growth rates as the effects of density dependence
475 weakened, allowing them to return to carrying capacity. On the timescales we modeled, no
476 extinctions occurred during the period after rescued populations reached carrying capacity
477 (generations 20 – 50 in our simulations). However, over longer timescales, populations that
478 fluctuate around a carrying capacity might be susceptible to drift (Frankham et al. 1999) or have
479 a low supply of beneficial mutations (Orr and Unckless 2008, Osmond and de Mazancourt

480 2013), reducing future evolutionary potential (Forester et al. 2022). Furthermore, if there is
481 future environmental change that necessitates further adaptation, populations at a constrained
482 size (e.g., a carrying capacity) will begin adaptation from a smaller size than populations with
483 unconstrained growth.

484 While our model shows that density dependence hinders adaptation through increased
485 genetic erosion, this result is sensitive to how competition interacts with selection. In our
486 model, density dependence acts on all individuals equally; that is, the reduction in fitness an
487 individual faces under competition does not depend on its phenotype. However, if the outcome
488 of competition depends on fitness, for example by more-fit individuals excluding less-fit
489 individuals from resources, then competition could aid, rather than hinder, rescue by
490 accelerating the rate of adaptation (Schluter et al. 1985, Osmond and de Mazancourt 2013).
491 This explains an apparent contradiction of our results with those of Vinton and Vasseur (2020),
492 who found that density dependence assisted the ability of populations to track a moving
493 environment; their model also featured density dependence that led to stronger demographic
494 penalties for more maladapted individuals. Furthermore, adaptation may even strengthen
495 competition by increasing per capita resource use and lowering carrying capacities (Bisschop et
496 al. 2022). Intraspecific competition can also increase the severity of inbreeding depression (Yun
497 and Agrawal 2014); in a case where purging removes few deleterious alleles, this severe
498 inbreeding depression can reduce population growth rates (Bozzuto et al. 2019), further
499 endangering populations facing genetic erosion as they adapt to novel conditions.
500

501 ***How genetic erosion constrains rescue***

502 Adaptation to environmental change was accompanied by a loss of genetic diversity and
503 potential for adaptation not captured in most models (but see Boulding and Hay 2001, Barfield
504 and Holt 2016). Quantitative genetic models often assume populations are large for analytical
505 tractability (Lande 1976); under this assumption fixation and loss of alleles are rare as there is
506 only marginal sampling variation and thus minimal drift. However, population decline is by
507 definition part of the rescue process, and our model demonstrates that additive genetic
508 variance is lost even in populations that survive environmental change (Fig 4*A*). This loss of
509 genetic diversity was associated with slowing rates of adaptation, contributing to the prolonged
510 time until rescue. This loss was partly driven by fixation of both adaptive and maladaptive
511 alleles, typically occurring when populations were at their smallest size (Fig. 4*B*,*C*). This matches
512 theoretical results of Boulding and Hay (2001) as well as empirical results of Rêgo et al. (2019)
513 in a seed beetle adapting to a novel host plant. Likewise, laboratory studies with *T. castaneum*
514 adapting to novel conditions found that accumulating genetic load in populations likely slows
515 the rate of adaptation (Stewart et al. 2017) and that more genetic diversity likely assists
516 population recovery through higher rates of recombination (Agashe et al. 2011). Models of
517 evolutionary rescue should thus include genetic erosion, as environmental stress (Nowak et al.
518 2009) and inbreeding due to bottlenecking (Frankham et al. 1999) are known to erode standing
519 genetic variation (reviewed in Biljsma and Loeschke 2011). Barfield and Holt (2016) were
520 among the first to model heritability as a function of population size. They similarly found a
521 long-term decline in heritability (analogous to additive genetic variance in our model) in extinct
522 populations, which they attributed to favored alleles approaching fixation. However, we did not
523 find evidence of the initial transient increase in heritability they reported; in our simulations

524 genetic variance declined most in early generations as populations were shrinking, driven by
525 fixation of not just adaptive but also of maladaptive alleles (Fig. 4). It is possible this difference
526 is due to our simulations being initialized with both alleles present at roughly equal frequencies,
527 which maximizes additive genetic variance (Equation 1). We ran additional simulations with the
528 negative (maladaptive) alleles at higher initial frequencies and found that genetic variance
529 remained approximately constant for 2-3 generations before declining, a result more similar to,
530 yet still distinct from, Barfield and Holt's (2016) results (Supporting Information I).
531 Our simulations were on short timescales compared to other studies (e.g., 50,000 time
532 steps in Osmond and de Mazancourt 2013) but reductions in population size and genetic
533 diversity could be important in the long term even after populations have been rescued. An
534 effective population size on the order of hundreds is needed to reduce longer-term extinction
535 risk due to accumulation of deleterious alleles (Lynch et al. 1995, Whitlock 2000, Frankham et
536 al. 2014). Specifically, populations need to spend long durations at large size to offset episodes
537 of drift and genetic erosion experienced even over short periods (Whitlock 2000), e.g., during
538 the "trough" or low point in population size following environmental change. In our
539 simulations, not only were density-dependent populations held at carrying capacity, potentially
540 precluding this re-establishment of variation, but they also spent more time at smaller densities
541 before being rescued (Figs. 2, 3*A*), leading to a greater erosion of variation (Fig. 4). Indeed, in
542 our simulations we saw as many as 40% of populations in a treatment group that were
543 "rescued" under our criteria go extinct within 50 generations, with stronger effects in
544 populations with initially low genetic diversity (Table 3). Furthermore, population size and
545 remaining standing genetic variation are important for persisting through future environmental

546 changes. The erosion of genetic variation, particularly of populations that experience severe
547 decline, is akin to a genetic bottleneck that would hinder the population's ability to adapt to
548 future environmental changes (Frankham et al. 1999, Whitlock 2000), particularly for changes
549 that are unidirectional or predictable (Lande and Shannon 1996). In our simulations, we
550 observed fixation of the positive allele in surviving populations (Fig. 4*B*), suggesting that
551 adaptive fixations did not guarantee extinction. However, given a future environmental change
552 favoring the other allele, its loss following the first environmental change event would hinder
553 subsequent adaptation (Lyberger et al. 2021).
554
555 ***How an extinction vortex impedes evolutionary rescue***
556 Our model shows that challenging environments can draw populations into an extinction
557 vortex (Gilpin and Soulé 1986). This vortex was characterized by multiple generations of
558 accelerating decreases in population growth rate (Fig. 5*A*) and loss of genetic diversity (Fig. 5*B*),
559 the latter associated with a slowing rate of adaptation (Fig. 5*C*). Deterministic models often
560 feature a critical density below which extinction due to demographic stochasticity is very likely
561 (e.g., Gomulkiewicz and Holt 1995, Chevin and Lande 2010); our stochastic model allowed us to
562 observe this process more mechanistically as we directly modeled the small-population
563 processes. Rather than change the rate of population decline in the generations immediately
564 preceding extinction, density dependence widens the opening to the vortex, as populations are
565 more likely to quickly decline to small size several generations before extinction (Fig. 5*A*). Gilpin
566 and Soulé's (1986) concept of the extinction vortex did not assume a population is initially
567 severely maladapted to its environment, only that small population size increases susceptibility

568 to drift and inbreeding. In contrast, evolutionary rescue is defined by populations facing severe
569 maladaptation that pushes mean fitness below the rate of replacement. Thus, rather than an
570 extinction vortex arising by a population slowly becoming less fit due to drift, a population
571 experiencing environmental change immediately becomes less fit and also faces an extinction
572 vortex *via* drift and stochasticity that further reduces its ability to sufficiently adapt to the new
573 environment (Fig. 5*B*,*C*). For managers of small, at-risk populations that are susceptible to
574 environmental change and extinction, alternative strategies such as assisted migration may be
575 needed to pull populations out of the self-reinforcing demographic and genetic processes of the
576 vortex (Hufbauer et al. 2015, Bell et al. 2019).
577
578 ***Rapid rescue in small populations due to founder sampling variation***
579 One surprising result was that upon environmental change, some small populations already
580 had a large proportion of phenotypes adapted to the environmental change due to sampling
581 variation, allowing them to be rescued very quickly (Fig. 3*D*, Table 3). In contrast, large
582 populations were rarely rescued immediately (Table 3) because the average initial phenotype
583 was less variable across populations (Fig. 3*D*). This appears to run counter to theoretical
584 arguments that larger populations are more likely to have novel variation compared to small
585 populations due to increased likelihood of individuals from the tail of the phenotypic
586 distribution being present in the population (Bell 2013). However, distinguishing between
587 variation in the population mean and variation among individuals within a population shows
588 that both phenomena are possible. In small populations, mean population phenotypes may be
589 initially closer to the new optimum due to sampling variation and drift (Fig. 3*D*) and this relative

590 abundance of adapted phenotypes can rapidly move the population to this new optimum.
591 Large populations are more likely to have individuals close to the new environmental optimum,
592 but it may take several generations for their alleles to dominate the mean population
593 phenotype due to the large number of maladapted individuals also initially present. Although
594 small populations were more likely to experience these very rapid rescue episodes, in the long
595 run larger populations were more likely to be rescued (Table 3). In part, this is because
596 sampling variation in small populations was equally likely to lead to an over-representation of
597 maladaptive alleles leading to a large disadvantage following environmental change (Fig. 3*D*).
598 Furthermore, considering that on the whole rescue is less likely to occur in small populations
599 compared to larger ones, the fact that some small populations were rescued due to a lucky
600 initial state underscores the degree to which small populations with unfavorable initial
601 phenotypes are at a disadvantage when the environment changes.
602
603 ***Implications for conservation biology***
604 We introduce a more biologically realistic model for a more complete and applicable
605 understanding of population rescue after environmental change, providing better prediction of
606 potential outcomes and guidance for conservation and management. In our model, we
607 demonstrate that negative density dependence impedes the process of persisting in a novel
608 environment, and that large and well-adapted populations previously predicted to be at
609 minimal risk (Gomulkiewicz and Holt 1995, Bell and Gonzalez 2009, Barfield and Holt 2016) do,
610 after all, face non-trivial risk of extinction under density dependence. For managers, one
611 prescription is to increase carrying capacities, which will alleviate reductions in growth rates as

populations recover from environmental change. This can be done by increasing resource and habitat availability. Although our simulated experimental design only featured density dependence as a binary treatment, in natural settings it is a continuous variable that managers can sometimes control. Because expected time to extinction increases exponentially with carrying capacity (Lande 1993), decreasing the strength or degree of competition could drastically improve the long-term fates, particularly of large populations previously perceived as having low extinction risk. A corollary is that, in addition to other predictors of extinction risk under rescue (Bell and Gonzalez 2009, Kopp and Matuszewski 2014), the carrying capacity or degree of intraspecific competition should be considered when assessing risks to populations subject to novel habitat alteration, degradation, or reduction. Furthermore, managers implementing strategies to maximize evolutionary potential (Forester et al. 2022) should consider how competition and population size may influence adaptation under environmental or climate change. These predictions highlight the effects of density dependence and loss of genetic diversity on populations exposed to environmental change, which is crucial information in an age of global anthropogenic changes that put populations at unprecedented risk of extinction.

Acknowledgements

The authors would like to thank Robert Holt for helpful suggestions following an early presentation on work from this project and Ruth Shaw for feedback on Figure 1. This work was supported by NSF award 1930222 to BAM and 1930650 to RAH. SWN was also supported by

NSF IGERT award 114807 while working on this project and LFD was also supported by NSF Graduate Research Fellowship number 006784.

Data and code availability

Data and simulation code to produce data and analysis will be archived on Dryad upon acceptance of the manuscript. Code and output files are also stored on GitHub at the address https://github.com/melbourne-lab/evo_rescue_ndd_erosion.

642 **References**

643 Agashe, D., Falk, J.J., and Bolnick, D.I. 2011. Effects of founding genetic variation on adaptation
644 to a novel resource. Evolution, 65(9): 2481 – 2491.
645
646 Arnaud, L., Gage, M.J.G., and Haubruge, E. 2001. The dynamics of second- and third-male
647 fertilization precedence in *Tribolium castaneum*. Entomologia Experimentalis et Applicata,
648 99(1): 55 - 64.
649
650 Barfield, M., and Holt, R.D. 2016. Evolutionary rescue in novel environments: towards
651 improving predictability. Evolutionary Ecology Research, 17: 771 – 786.
652
653 Bell, G. 2013. Evolutionary rescue and the limits of adaptation. Philosophical Transactions of the
654 Royal Society of Biology, 368: 20120080.
655
656 Bell, G., and Gonzalez, A. 2009. Evolutionary rescue can prevent extinction following
657 environmental change. Ecology Letters, 12: 942 – 948.
658
659 Bell, D.A., Robinson, Z.L., Funk. W.C., Fitzpatrick, S.W., Allendorf, F.W., Tallmon, D.A., and
660 Whiteley, A.R. 2019. The exciting potential and remaining uncertainties of genetic rescue.
661 Trends in Ecology and Evolution, 34(12): doi:10.1016/j.tree.2019.06.006.
662

663 Bijlsma, R., and Loeschcke, V. 2012. Genetic erosion impedes adaptive responses to stressful
664 environments. Evolutionary applications, 5: 117 – 129.
665
666 Bisschop, K., Alzate, A., Bonte, D., and Etienne, R.S. 2022. The demographic consequences of
667 adaptation: evidence from experimental evolution. The American Naturalist, 199(6): 729 – 742.
668
669 Bonnet, T., et al. 2022. Genetic variance in fitness indicates rapid contemporary adaptive
670 evolution in wild animals. Science, 376(6596): 1012 - 1016.
671
672 Boulding, E.G., and Hay, T. 2001. Genetic and demographic parameters determining population
673 persistence after a discrete change in the environment. Heredity, 86: 313 – 324.
674
675 Bozzuto, C., Biebach, I., Muff, S., Ives, A.R., and Keller, L.F. 2019. Inbreeding reduces long-term
676 growth of Alpine ibex populations. Nature Ecology and Evolution, 3: 1359 – 1364.
677
678 Carlson, S.M., Cunningham, C.J., and Westley, P.A.H. 2014. Evolutionary rescue in a changing
679 world. Trends in Ecology and Evolution, 29(9) 521 – 530.
680
681 Chevin, L-M., and Lande, R. 2010. When do plasticity and genetic evolution prevent extinction
682 of a density-regulated population? Evolution, 64(4): 1143 – 1150.
683

Fagan, W.F., and Holmes, E.E. 2006. Quantifying the extinction vortex. Ecology Letters, 9: 51 – 60.

Forester, B.R., Beever, E.A., Darst, C., Szymanski, J., and Funk. W.C. 2022. Linking evolutionary potential to extinction risk: Applications and future directions. Frontiers in Ecology and Evolution, 1 - 9. http://doi.org/10.1002/fee.2552

Frankham, R., Lees, K., Montgomery, M.E., England, P.R., Lowe, E.H., and Briscoe, D.A. 1999. Do population size bottlenecks reduce evolutionary potential? Animal Conservation, 2(4): 255 – 260.

Frankham, R., Bradshaw, C.J.A., and Brook, B.W. 2014. Genetics in conservation management: revised recommendations for the 50/500 rules, Red List criteria, and population viability analysis. Biological Conservation, 170: 56 – 623.

Gilpin, M.E., and Soulé, M.E. 1986. Minimum viable populations: processes of extinction. In: *Conservation Biology: The Science of Scarcity and Diversity,* ed. Soulé, M.E. Sinauer Associates, Sunderland, MA.

Gomulkiewicz, R., and Holt, R.D. 1995. When does evolution by natural selection prevent extinction? Evolution, 49(1): 201 – 207.

Goodrich, B., Gabry, J., Ali, I., and Brilleman, S. 2020. Rstanarm: Bayesian applied regression modeling via Stan. R package, version 2.21.1. https://mc-stan.org/rstanarm.

Hedrick, P.W., and García-Dorado, A. 2016. Understanding inbreeding depression, purging, and genetic rescue. Trends in Ecology and Evolution, 31(12): 940 – 952.

Hone, J., Duncan, R.P. and Forsyth, D.M. 2010. Estimations of maximum annual growth rates (r_m) of mammals and their application in wildlife management. Journal of Applied Ecology, 47(3): 507 - 514.

Houle, D. 1992. Comparing evolvability and variability of quantitative traits. Genetics, 130: 195 – 204.

Hufbauer, R.A., Szűcs, M., Kayson, E., Youngberg, C., Koontz, M.J., Richards, C., Tuff, T., and Melbourne, B.A. 2015. Three types of rescue can avert extinction in a changing environment. PNAS, 112(33): 10557 – 10562.

Kopp, M., and Matuszewski, S. 2014. Rapid evolution of quantitative traits: theoretical perspectives. Evolutionary Applications, 7(1): 169 – 191.

Lande, R. 1976. Natural selection and random genetic drift in phenotypic evolution. Evolution, 30: 314 – 334.

Lande, R. 1993. Risks of population extinction from demographic and environmental stochasticity and random catastrophes. The American Naturalist, 142(6): 911 – 927.

Lande, R., and Shannon, S. 1996. The role of genetic variation in adaptation and population persistence in a changing environment. Evolution, 50(1): 434 – 437.

Lyberger, K.P., Osmond, M.M., and Schreiber, S.J. 2021. Is evolution in response to extreme events good for population persistence? The American Naturalist, 198(1): 44 – 52.

Lynch, M., and Walsh, B. 1998. *Genetics and the analysis of quantitative traits.* Sinauer Associates, Sunderland, MA.

Lynch, M., Conery, J., and Bürger, R. 1995. Mutational meltdowns in sexual populations. Evolution, 49(6): 1067 – 1080.

Melbourne, B.A., and Hastings, A. 2008. Extinction risk depends strongly on factors contributing to stochasticity. Nature, 454: 100 – 103.

Nowak, C., Vogt, C., Pfenninger, M., Schwenk, K., Oehlmann, J., Streit, B., and Oetken, M. 2009. Rapid genetic erosion in pollutant-exposed experimental chironomid populations. Environmental Pollution, 157: 881 – 886.

750

751 Orr, H.A., and Unckless, R.L. 2008. Population extinction and the genetics of adaptation. The
752 American Naturalist, 172(2): 160 – 169.

753

754 Orr, H.A., and Unckless, R.L. 2014. The population genetics of evolutionary rescue. PLoS
755 Genetics, 10(8): e1004551.

756

757 Osmond, M.M., and de Mazancourt, C. 2013. How competition affects evolutionary rescue. Phil
758 Trans R Soc B., 368: 20120085.

759

760 Pai, A., and Yan, G. 2020. Long-term study of female multiple mating indicates direct benefits in
761 Tribolium castaneum. Entomologia Experimentalis et Applicata, 168(5): 398 – 406.

762

763 Pauls, S.U., Nowak, C., Bálint, M., and Pfenninger, M. 2013. The impact of global climate change
764 on genetic diversity within populations and species. Molecular Ecology, 22: 925 – 946.

765

766 R Core Team. 2020. R: A language and environment for statistical computing. R Foundation for
767 Statistical Computing, Vienna, Austria. https://www.R-project.org/.

768

769 Rêgo, A., Messina, F.J., and Gompert, Z. 2019. Dynamics of genomic change during evolutionary
770 rescue in the seed beetle Callosobruchus maculatus. Molecular Ecology, 28: 2136 – 2154.

771

Ricker, W.E. 1954. Stock and recruitment. Journal of Fisheries of the Board of Canada, 11: 559 – 623.

Schluter, D., Price, T.D., and Grant, P.R. 1985. Ecological character displacement in Darwin's finches. Science, 227(4690): 1056 – 1059.

Stewart, G.S., Morris, M.R., Genis, A.B., Szűcs, M. Melbourne, B.A., Tavener, S.J., and Hufbauer, R.A. 2017. The power of evolutionary rescue is constrained by genetic load. Evolutionary Applications, 10: 731 – 741.

Vinton, A.C., and Vasseur, D.A. 2020. Evolutionary tracking is determined by differential selection on demographic rates and density dependence. Ecology and Evolution, 10: 5725 – 5736.

Wade, M.J., Shuster, S.M., and Stevens, L. 1996. Inbreeding: its effect on response to selection for pupal weight and the heritable variance in fitness in the flour beetle, *Tribolium castaneum*. Evolution, 50(2): 723 – 733.

Whitlock, M.C. 2000. Fixation of new alleles and the extinction of small populations: drift load, beneficial alleles, and sexual selection. Evolution, 54(6): 1855 – 1861.

793 Yun, L. and Agrawal, A.F. 2014. Variation in the strength of inbreeding depression across

794 environments: effects of stress and density dependence. Evolution, 68(2): 3599 – 3606.

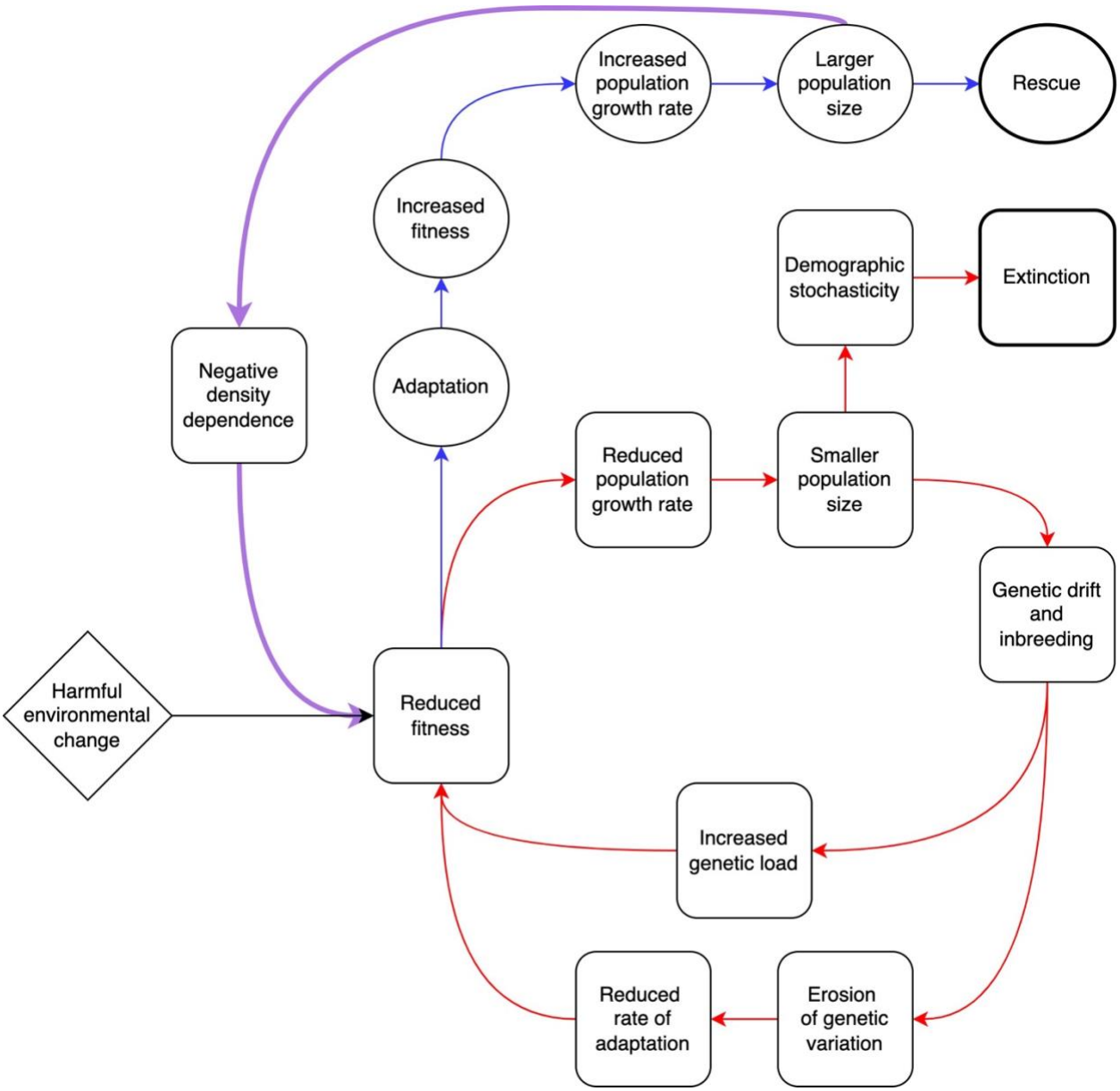

Figure 1: Event sequence diagram of evolutionary rescue (blue, ovals) and two connected extinction vortices (red, squares) that populations may face following harmful environmental change. The effects of negative density dependence, potentially impeding rescue and drawing populations into a vortex, are indicated in purple.

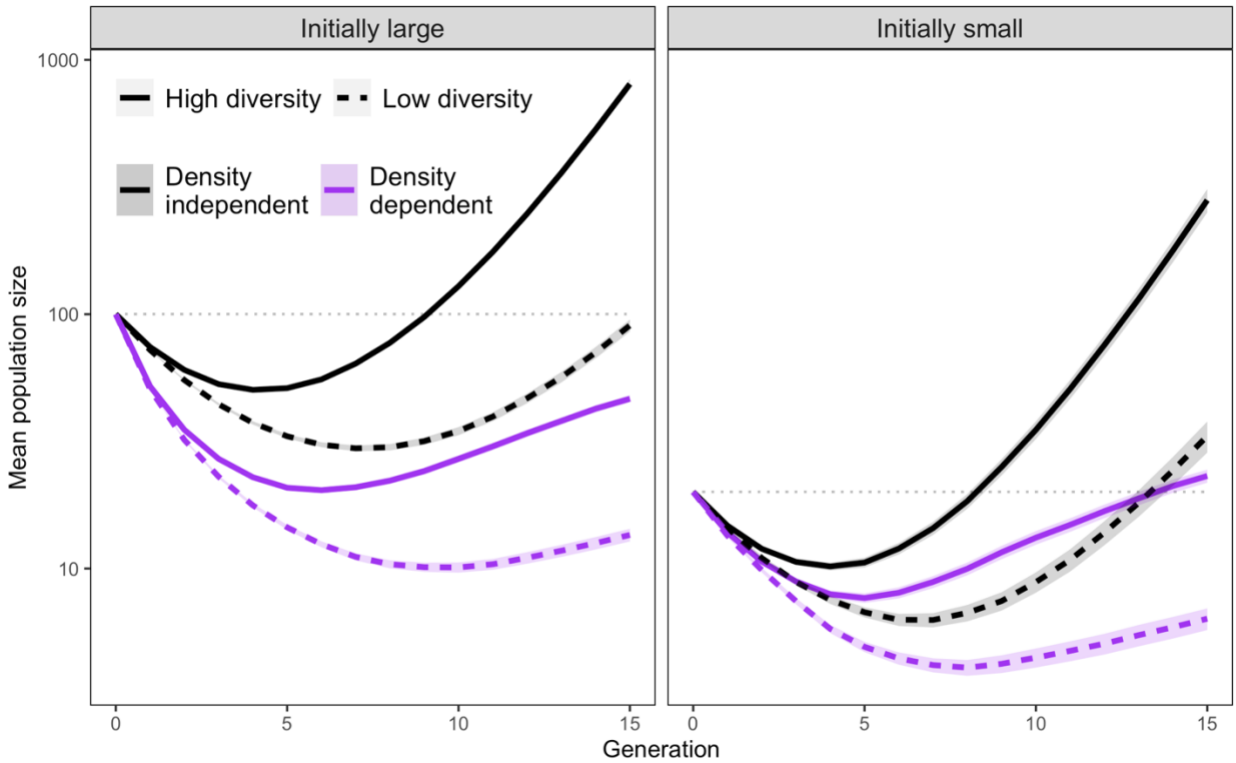

Figure 2: Mean population size in each generation for all treatments (n = 4000 for each treatment). Means in each generation include extinct populations as zeros. Shaded areas are twice the standard error on each side of the mean.

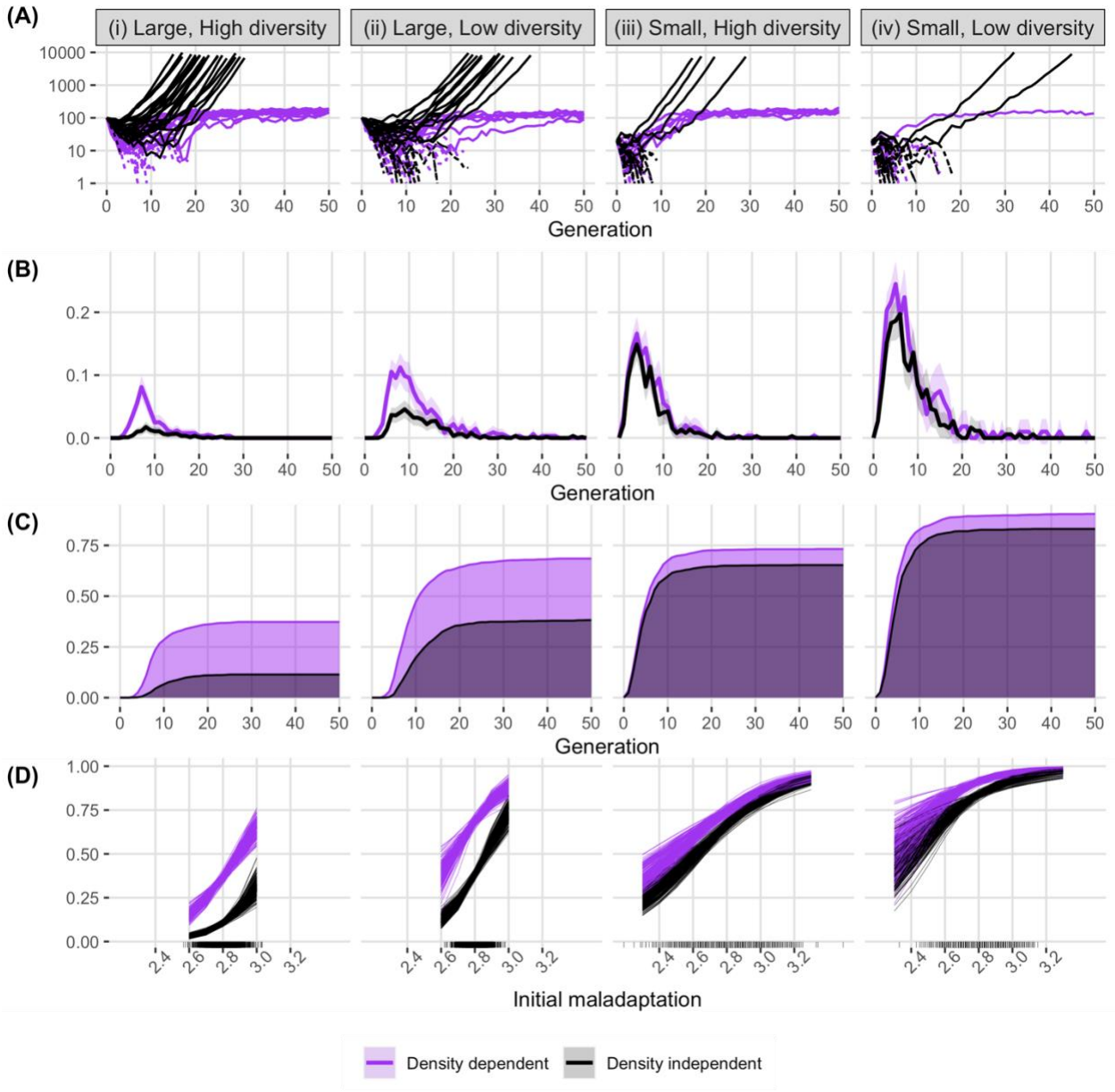

Figure 3: Population size and extinction characteristics of 1000 simulations per treatment group run for up to 50 generations. (A) mean population size of 25 randomly selected trials for each treatment. Dashed lines are extinct populations. Simulations were truncated for populations surpassing size 10000. (B) Instantaneous probability of extinction in each generation, conditioned on survival until that generation. Ribbons represent twice the standard error on both sides of the estimate. (C) Cumulative probability of extinction over time. Areas under the curve are shaded to highlight the difference between density-dependent and density-independent populations. (D) 200 posterior estimates per treatment of extinction probability as a function of initial degree of maladaptation, as predicted by a generalized linear model (Table 2). Marks on x-axis indicate the distribution of initial mean population genotypes for those treatments.

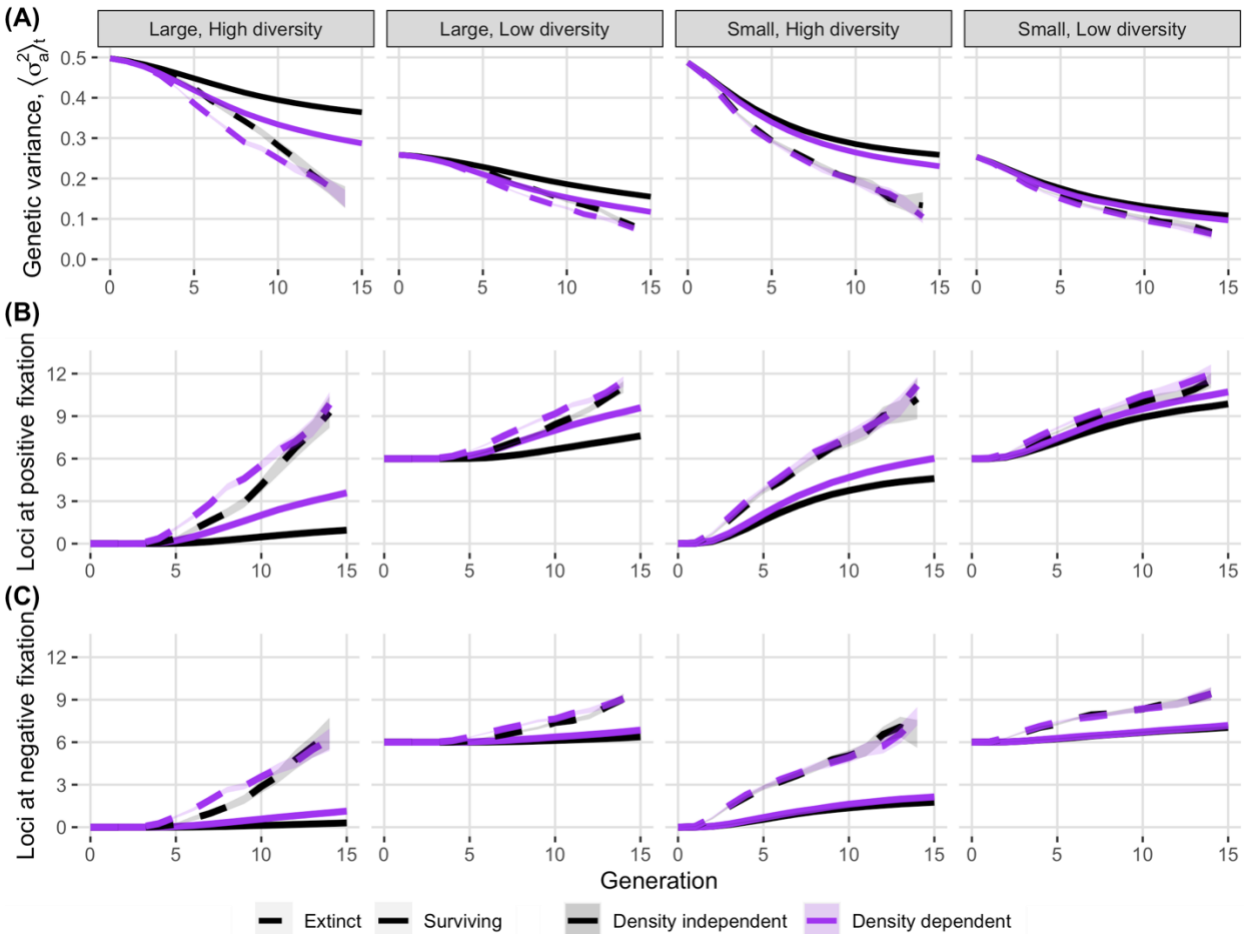

Figure 4: (A) Mean additive genetic variance and the mean number of loci at fixation (out of 25) for (B) positive and (C) negative alleles over time in surviving and extinct populations. Shaded areas represent twice the standard error on each side of the mean (the standard error is often too small to be visible on this plot).

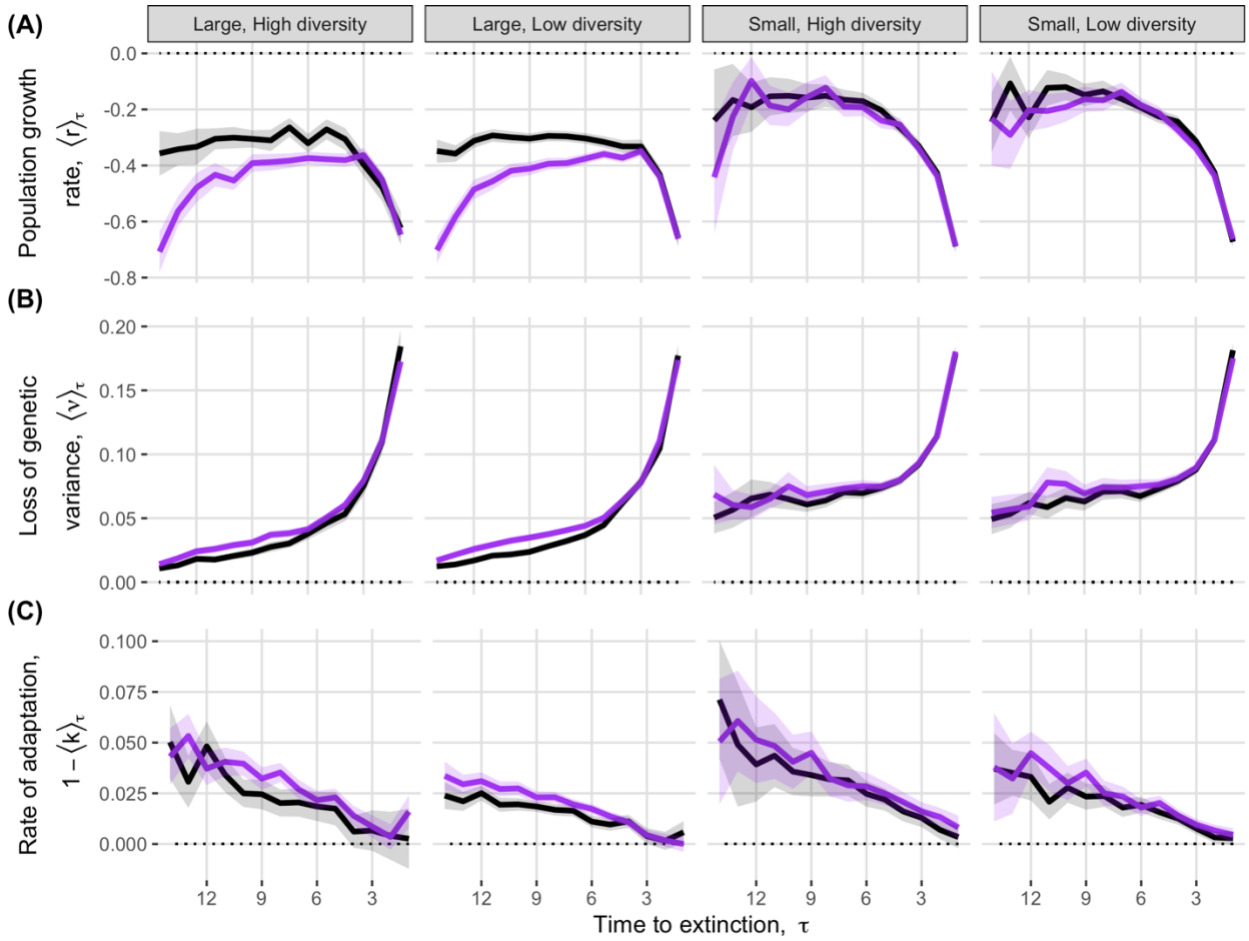

Figure 5: Rates of change as a function of time to extinction (τ), demonstrating the presence of an extinction vortex, in (A) population growth, (B) loss of genetic variation, and (C) rate of adaptation. Small τ represents time steps closer to the time of a population's extinction. Shaded areas represent twice the standard error on both sides of the mean.

Tables

Table 1 Parameter values simulated for the three-way factorial simulation experiment

Factor of interest	Treatment	Value	Name in main text
Density dependence	Absent	$\alpha = 0$	density-independent populations
	Present	$\alpha = 0.0035$	density-dependent populations
Genetic diversity	High	$\sigma_a^2 \approx 0.5$	high-diversity populations
	Low	$\sigma_a^2 \approx 0.25$	low-diversity populations
Initial population size	Large initial size	$N_0 = 100$	large populations
	Small initial size	$N_0 = 20$	small populations

Table 2 Effect sizes of treatment variables changed while others are held constant on log odds of extinction in 50 generations and accompanying 95% credible intervals.

Variable changed	Held constant	Effect size (95% CI)
Density independent to dependent	Large, high diversity	1.60 (1.36, 1.85)
	Large, low diversity	1.27 (1.08, 1.46)
	Small, high diversity	0.41 (0.21, 0.61)
	Small, low diversity	0.71 (0.44, 1.00)
Large to small	High diversity, DI	2.82 (2.58, 3.08)
	High diversity, NDD	1.63 (1.44, 1.83)
	Low diversity, DI	2.13 (1.92, 2.35)
	Low diversity, NDD	1.58 (1.31, 1.85)
High to low diversity	Large, DI	1.67 (1.43, 1.91)
	Large, NDD	1.34 (1.15, 1.53)
	Small, DI	0.99 (0.77, 1.21)
	Small, NDD	1.29 (1.03, 1.56)
Increased maladaptation	Large, high variance, DI	6.65 (4.38, 8.99)
	Large, high variance, NDD	6.04 (4.16, 7.99)
	Large, low variance, DI	7.24 (4.77, 9.79)
	Large, low variance, NDD	6.19 (3.82, 8.55)
	Small, high variance, DI	3.76 (2.90, 4.63)
	Small, high variance, NDD	3.39 (2.40, 4.40)
	Small, low variance, DI	3.99 (2.46, 5.50)
	Small, low variance, NDD	4.48 (2.67, 6.34)

Table 3 Summary of rescue probabilities, proportion of rescue events occurring instantly, times to rescue, and extinction probabilities after rescue 50 generation simulations for both types of rescue.

Size and diversity	**Prob. rescue**		**Prop. (number) instant rescue†**		**Mean time to rescue**		**Prob. extinct after rescue**	
	DI	NDD	DI	NDD	DI	NDD	DI	NDD
	Fitness-based rescue							
Large, high	0.92	0.71	0.00 (3)	0.00 (0)	5.1	6.7	0.04	0.12
Large, low	0.69	0.41	0.00 (0)	0.00 (0)	8.3	9.2	0.10	0.24
Small, high	0.44	0.36	0.16 (68)	0.06 (23)	4.0	4.7	0.21	0.27
Small, low	0.24	0.16	0.04 (11)	0.01(2)	5.7	7.0	0.31	0.39
	Size-based rescue							
Large, high	0.89	0.62	0.00 (1)	0.00 (0)	11.2	20.3	0.00	0.00
Large, low	0.62	0.26	0.00 (0)	0.00 (0)	17.4	27.7	0.00	0.00
Small, high	0.37	0.29	0.16 (61)	0.05 (15)	6.3	7.9	0.06	0.07
Small, low	0.19	0.12	0.13 (24)	0.08 (9)	7.9	8.6	0.12	0.17

† proportion instant rescue is the proportion of rescued populations in the treatment where rescue occurred in 0 or 1 generations; the number is the total number of observed populations (out of 1000) in the treatment experiencing instantaneous rescue