# Phenotype-Environment Matching Predicts Both Positive and Negative Effects of Intraspecific Variation

**Christopher J. Dibble* and Volker H. W. Rudolf†**

BioSciences, Rice University, Houston, Texas 77005

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ABSTRACT: Natural populations can vary considerably in their genotypic and/or phenotypic diversity. Differences in this intraspecific diversity can have important consequences for contemporary ecological dynamics, but the direction and magnitude of these effects appear inconsistent across studies and systems. Here we proposed and tested the hypothesis that context-dependent ecological effects of altering phenotypic variance are predictable and arise from the relationship between a population's mean phenotype and the local environmental optimum. By factorially manipulating the mean and variance of a key host trait in environments with and without a lethal parasite, we demonstrate that increasing phenotypic variance can have beneficial effects for host populations (e.g., smaller disease epidemics) but only when the population's initial phenotype was poorly matched to the local environment. When phenotypes were initially well suited to environmental conditions, in contrast, greater phenotypic variance led to larger disease epidemics. Significant reductions in individual susceptibility occurred in both contexts over time, but the mechanisms leading to those reductions differed; strong selection was caused by either a suboptimal trait mean and insufficient trait variance or a near-optimal trait mean and too much trait variance. Increasing intraspecific variation is clearly not always beneficial for populations, instead producing predictable ecological and evolutionary effects that depend on environmental context and biological interactions.

*Keywords: Daphnia*, *Metschnikowia*, disease dynamics, eco-evolutionary feedbacks, selection, Jensen's inequality.

## Introduction

Natural populations are not homogenous groups of identical clones. Instead, individuals of a species differ genetically and phenotypically, and this variation can play an important role in contemporary ecological dynamics (Whitham et al. 2006; Hughes et al. 2008; Bailey et al. 2009). For example, increasing gene or trait diversity per se can enhance the establishment success of founder populations (Agashe 2009; Crawford and Whitney 2010; Forsman 2014; González-Suárez et al. 2015), increase host resistance to parasites (Altermatt and Ebert 2008; Ganz and Ebert 2010; King and Lively 2012), and alter both community assembly and ecosystem function (Crutsinger et al. 2006; Genung et al. 2010; Whitlock 2014). While these studies emphasize that intraspecific variation can have important ecological consequences, the effects are inconsistent across, and even within, systems. Several studies hypothesizing positive diversity effects have either failed to find strong evidence for such or found evidence in only a subset of treatments (table 1). Identifying the mechanisms that drive variable effects of intraspecific diversity is critical to developing a more comprehensive framework for understanding and predicting the ecological effects of intraspecific variation across systems (Bolnick et al. 2011). Here we suggest that this variability could be explained by differences in phenotype-environment matchup. Specifically, we propose and test the hypothesis that the ecological importance of intraspecific variation per se depends on the relationship between the current mean phenotype of a population and the optimal phenotype in their local environment.

Many positive diversity effects occur in situations where the average phenotype of a population was poorly matched to the local environment, for example, in terms of colonization success or pathogen susceptibility (table 1). While all populations are suboptimal in the local environment, those that are farther away from the local optimum should benefit more from additional phenotypic variance. Additional variance offers immediate access to peaks of the fitness landscape (Whitlock 1995). Augmenting phenotypic variance, often approximated by increasing genotypic richness, can therefore increase the probability that at least some high-fitness individuals are present in the population. If the trait is susceptibility to a pathogen, for instance, increasing variance in a highly susceptible host population could introduce more resistant individuals, which can reduce the size of disease epidemics (e.g., Dibble and Rudolf 2016).

* Present address: Battelle Memorial Institute, 505 King Avenue, Columbus, Ohio 43201.

† Corresponding author; email: volker.rudolf@rice.edu.

**ORCIDs:** Rudolf, https://orcid.org/0000-0002-9214-2000.

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**Table 1:** Context-dependent effects of intraspecific diversity

| Question | Effect of diversity | Strength of selection | Diversity measure | Summary | Reference |
|---|---|---|---|---|---|
| Does predator diet breadth alter prey communities? | None | ... | Trait variance | No effects of body size variance on predator diet breadth, prey community composition | Ingram et al. 2011 |
| Does intraspecific variation reduce competitive interactions? | None | ... | Genotypic richness | Polycultures showed performance identical to monocultures | Dimas-Flores et al. 2012 |
| | Positive | Strong | Genotypic richness | Positive effect of genotypic richness on seagrass shoot density, biomass | Hughes and Stachowicz 2004 |
| Does genetic diversity improve response to disturbance? | None | Weak | | No effects of genotypic richness | |
| | Positive | Strong | Strain richness | Strong positive effects of genotypic richness on founding population size, stability | Agashe 2009 |
| Does genetic diversity enhance success of founding populations? | Weakly positive | Weak | | Weaker effects of genotypic richness | |
| | Positive | Strong | Genotypic richness | Host diversity reduces overall infection prevalence | Ganz and Ebert 2010 |
| Do host polycultures better resist disease? | None | Weak | | No effect of host diversity on prevalence | |

Note: Strong selection was imposed by goose grazing (Hughes and Stachowicz 2004), colonization of a novel environment (Agashe 2009), and high-parasite-strain diversity (Ganz and Ebert 2010). This table is not a systematic review of all intraspecific-diversity studies.

When populations have trait means nearer the environmental optimum, in contrast, why would we expect a positive effect of increasing phenotypic variance? If the additional phenotypic variants are not much better than what is already in the population (e.g., Lenormand 2002; Marshall et al. 2010; Blanquart et al. 2012), the response to increasing variance—in terms of some aggregate measure such as population growth or abundance—may be correspondingly small. Alternatively, increasing phenotypic variance in populations well suited to their local environment could have negative effects. Negative effects could occur if the lower-fitness individuals, added via increasing variance around a constant mean, disproportionately influence the response of the population (Lacey et al. 1983). In models of population growth, for instance, near-optimal trait means can lead to negative population growth with high levels of trait variance (Lynch et al. 1991), depending on the environmental context. Using susceptibility again as an example, increasing the variance around a highly resistant mean phenotype increases the frequency of susceptible individuals in the population. If this increase in the susceptible fraction aids parasite establishment (Anderson and May 1979), increasing phenotypic variance could actually increase the size of disease epidemics.

Both positive and negative effects of increasing phenotypic variance require (or assume) a nonadditive response to an increase in phenotypic variance. This is because the null expectation of a symmetric increase in variance around a constant mean is no net effect, as the positive effects of higher-fitness individuals are offset by the negative effects of those on the other end of the trait distribution. Predictions of positive diversity effects thus rely on, to a degree, high-fitness individuals contributing more to the population than low-fitness individuals. Our hypothesized negative effect of increasing variance relies on the opposite pattern: individuals on the lower end of the augmented trait distribution influencing the population more. Jensen's inequality, driven by nonlinear relationships between trait value of individuals and an ecological function, is one mechanism that can produce nonadditive responses to a symmetric increase in phenotypic variance (Bolnick et al. 2011). Similarly, positive or negative feedback loops coupled with population growth can lead to nonlinear decline or increase of the frequency of phenotypes at the edge of the phenotypic distribution over time. For instance, if well-adapted phenotypes reproduce at twice the rate of all other individuals, their phenotype could exponentially (i.e., nonlinearly) increase over time relative to the rest of the population. Importantly, each of these mechanisms can produce positive or negative effects, depending on the concavity of the relationship between traits and fitness or on the nature of the predominant selective pressure in an environment (e.g., with or without parasites).

In this study, we asked whether the ecological consequences of phenotypic variance depend on the relationship between the trait mean and the local environmental optimum. In a zooplankton host (*Daphnia pulex*), we factorially manipulated the mean and variance of a key trait (the intrinsic rate of increase, *r*) in environments with and without a lethal parasite (*Metschnikowia bicuspidata*). Consistent with our predictions, we found that increasing phenotypic variance had positive ecological effects when populations expressed suboptimal trait means but negative effects when populations were already well suited to the local environment. Additionally, two populations showed significant reductions in individual susceptibility to the parasite, consistent with rapid evolutionary change. Overall, this study provides a more mechanistic framework for understanding and predicting the influence of intraspecific variation on eco-evolutionary dynamics.

## Material and Methods

### *Overview*

We first conducted a life table assay to quantify trait values from a large number of clones of our host species, *Daphnia pulex*. Using trait values from this life table (from 59 unique host clones), we established experimental populations that differed in both the mean and the variance of *r* while keeping host genotypic richness (i.e., number of genotypes) constant. By analyzing these host populations in the presence and absence of parasites, we determined the optimum trait value in each environment and thus tested whether the effects of increasing phenotypic variation depended on the relationship between the trait mean and the environmental optimum. After the ~13-week mean/variance experiment, we conducted an individual infection assay to assess whether parasite exposure significantly altered average susceptibility in each treatment.

### *Study System*

For our experimental study, we utilized a zooplankton-fungus host-parasite system (host *D. pulex*, parasite *Metschnikowia bicuspidata*), exploiting the cyclically parthenogenic life history of *D. pulex* to obtain and maintain unique clones with natural variation in key life-history traits (see also the appendix, available online). Disease transmission in the *Daphnia-Metschnikowia* system is linked with foraging ecology, as individual *Daphnia* consume both algal resources and infectious fungal spores via filter feeding. Previous work has shown that individuals and genotypes with faster foraging rates (i.e., that filter more milliliters of water per unit time) (1) encounter more fungal spores, (2) produce more spores when infected (because of more resources for parasite growth), and (3) show higher fecundity when uninfected (Hall et al. 2010, studying the closely related *Daphnia dentifera*). This represents a trade-off between fecundity and disease risk, as growth and reproduction are often associated with higher individual susceptibility (but see Auld et al. 2013).

### *Life Table Assay*

We started the life table assay with 177 individual females, 3 replicate individuals each of 59 clones, with each clone originally hatched from resting eggs (ephippia) collected from a natural pond in Huntsville, Texas. Individuals were housed singly in 50-mL centrifuge tubes and fed laboratory-reared *Scenedesmus obliquus*. Each day, we transferred individuals to new containers with fresh medium and algae and checked for the presence of offspring. When females reproduced, we counted their offspring and measured the adult body size to the nearest 0.01 mm (using a Leica rz95 microscope) before moving the adults to their new containers and discarding offspring. The life table assay began on February 13, 2013, and was terminated on March 28, 2013, by which point 111 of the 177 individuals had died of natural causes.

The intrinsic growth rate (*r*) of each clone was estimated by iteratively solving the characteristic Euler-Lotka equation:

$$1 = \sum e^{-rx} l_x m_x, \tag{1}$$

where $x$ is age (in days), $l_x$ is age-specific survival, and $m_x$ is age-specific reproduction. Summed over the life span of the population (i.e., $N = 3$ replicates) of each clone, $r$ represents the expected rate of population growth of a clone in a new environment. Additional methodological details for the life table assay can be found in the appendix.

### *Factorial Manipulation of Trait Mean and Variance*

We created populations that differed in the mean and variance of the intrinsic growth rate, *r*, which is a common metric of overall fitness and was hypothesized to play a role in natural disease epidemics (Dibble et al. 2014; Dibble and Rudolf 2016). Experimental populations each comprised a randomly selected combination of four clones of the host species *D. pulex*. We first arranged the clones in order of increasing *r*, obtained from the life table assay. We then split the ordered list of clones in half, artificially creating a pool of clones with low or high intrinsic growth rates ($N = 20$ clones for each mean treatment). Within each of the low- and high-mean subsets, we used a simple selection scheme to randomly assign clones to low-, medium-, or high-variance treatments (see the appendix for additional details). Importantly, this design kept the number of genotypes (genotypic richness) constant across treatments yet created independent phenotypic mean and

variance treatments (fig. 1), using unique combinations of clones. In this way, we avoided a degree of pseudoreplication, as 93% of our experimental populations contained unique four-clone host polycultures.

In general, our treatment combinations were successful in creating their intended trait values (fig. 1). Our random clone selection achieved distinct differences in mean trait values between our high- and low-mean treatments, and phenotypic variance was highest in the high-trait-variance treatments and lowest in the low-trait-variance treatments (fig. 1; the coefficient of variation shows a qualitatively similar pattern). Within the low-trait-mean treatments, however, there were larger differences in mean trait value among the three variance levels (fig. 1). Thus, we see a significant interaction of mean treatment × variance treatment in the initial mean trait values (ANOVA, $F_{2,114} = 3.72$, $P = .027$), albeit a smaller effect than the mean treatment itself (table A1; tables A1–A6 are available online). This largely reflects the natural distribution of traits among our clones, which was skewed slightly left (visible in fig. A1; figs. A1–A7 are available online), as well as the clone selection procedure, which made sampling of the most extreme low-mean phenotypes less likely in the low-variance treatment.

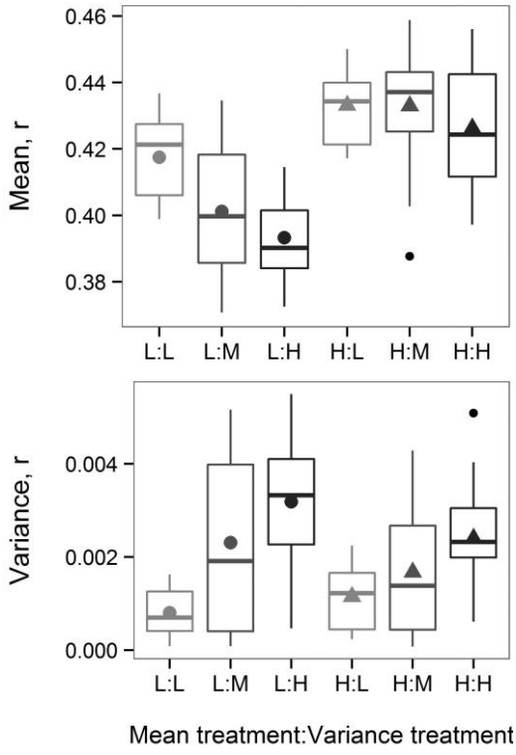


**Figure 1:** Initial estimates of mean and variance (coefficient of variation) for factorial manipulation of mean and variance combinations (mean ± 1 SE). Each experimental population consisted of four clones. Shades of gray represent levels of initial trait variance (light gray = low [L]; medium gray = medium [M]; dark gray = high [H]), and symbols represent mean treatments (squares = low; circles = high).

The experiment was conducted as a fully factorial, randomized block design, with two levels of trait mean treatment (high trait mean vs. low trait mean), three trait variance levels (high, medium, and low trait variance), and two parasite treatments (present/absent; i.e., 2 × 3 × 2 factorial design). As a result of previously documented trade-offs between disease risk and population growth (Hall et al. 2010, 2012; Dibble and Rudolf 2016), we expected that different trait ($r$) means would be favored in environments with versus without parasites; that is, higher trait values would lead to faster growth without parasites but increase disease risk when parasites were present. Each treatment was replicated 10 times, for 120 total experimental populations. Experimental containers were plastic tubs filled with 800 mL of medium (3-to-1 mixture of artificial medium [ADaM; Klüttgen et al. 1994] to filtered pond water [2-$\mu$m pore size]), kept at 20°C on a 16L : 8D cycle, and fed laboratory-reared algae daily ($1.5 \times 10^5$ cells/mL *S. obliquus*). We started populations with eight adult *Daphnia*, two each of the four randomly selected clones. Half of the populations received a dose of parasite spores (60 spores/mL) 14 days after *Daphnia* introduction. Because we did not detect any infected individuals in exposed populations after 2 weeks, we repeated the exposure. We sampled all tanks weekly by removing 25% of the container volume and enumerating and recording infection status for all sampled individuals. Individuals were returned to their experimental containers alive after sampling to minimize artificial fluctuations in population size. We maintained and monitored populations for 91 days (~13 generations), at which time populations began to decline rapidly (presumably because of deteriorating conditions in experimental containers). At the end of the experiment, we conducted an individual infection assay (see the appendix for details) to determine whether remaining individuals varied in their susceptibility to the parasite.

### *Statistical Analyses*

To understand how our factorial manipulation of trait mean and variance affected host population dynamics and disease epidemic severity, we used generalized additive mixed-effect models (GAMMs). We fitted GAMMs separately to host and parasite dynamics, testing for significant interactions of mean treatment, variance treatment, and the presence of parasites on mean *Daphnia* density and mean infection prevalence. GAMMs allow the testing of traditional fixed-effects parameters in a nonlinear system by fitting penalized regression splines to the nonlinear component of, in this case, time series data. The model coefficients from a fitted GAMM, which we use to report statistical significance for our host density and

infection prevalence models, are highly correlated with overall treatment means (e.g., linear slope between treatment mean and GAMM coefficient for host density = 1.1, $R^2 = 0.93$).

For host population density, we created a full model with fixed effects of mean treatment, variance treatment, parasite presence/absence, and all possible interactions between the factors. Because of a significant three-way interaction between mean, variance, and parasite presence (see "Results"), we fitted two subsequent GAMMs to treatments with and without parasites to determine whether the mean × variance interactions persisted in each. Models for host density used a negative binomial error distribution and included a smoothing term to account for nonlinear growth over time (Wood 2006) and a random effect of spatial block. The temporal smoothing term was allowed to vary by treatment, which improved the model fit as evidenced by Akaike information criterion (AIC; Burnham and Anderson 2002). Our model for epidemic severity (quantified by mean infection prevalence) included the same temporal smoothing trend but was fitted to data only after day 40 (before this point, there were no infections). The prevalence model used a binomial error distribution. Models were created in the R package mgcv (Wood 2006), and statistical significance was assessed by Wald $\chi^2$ tests.

### *Evolution of Susceptibility*

We hypothesized that exposure to parasites would significantly reduce individual susceptibility over time but that this change would depend on the initial trait mean and variance of each population and the strength of selection (i.e., size of disease epidemic). To test this hypothesis, we conducted a standardized infection assay after the 90-day mean/variance experiment. From each experimental container, we haphazardly removed three to five uninfected adult *Daphnia* (frequently all that were remaining) and allowed them to reproduce in a clean beaker. We randomly selected 10 of these offspring to include in the assay. Offspring were placed in a clean 50-mL vial and exposed to 250 spores/mL of *M. bicuspidata* spores for 2 days. Individuals were then removed and placed in 250-mL beakers with clean medium, and any offspring the test animals produced were removed. Infections were checked after 10 days by checking for the presence of spores under a microscope.

From this assay, we first used a generalized linear model to test how individual susceptibility differed across treatments, including spatial block and day of infection as random effects. Additionally, we calculated the mean reduction in individual susceptibility after a 90-day exposure to parasites and its 90% confidence interval. We matched parasite and parasite-free treatments within each spatial block (e.g., replicate 1: high trait mean, high trait variance, no parasites vs. high trait mean, high trait variance, with parasites) and calculated the difference in individual susceptibility, using the values for each replicate as unique samples with which to find the mean and confidence interval of any change. A significant reduction would indicate that trait change, consistent with an evolutionary response, was rapid enough to influence the host-parasite dynamics we observed in the mean/variance experiment. All data for this study are deposited in the Dryad Digital Repository (https://dx.doi.org/10.5061/dryad.99k4n5s; Dibble and Rudolf 2019).

## Results

### *Life Table Assay*

Our 59 unique clones of *Daphnia pulex* exhibited substantial variation in key traits, particularly their intrinsic rate of increase ($r$). Values of $r$ ranged from 0.286 to 0.515 (mean = 0.409), an 80% difference between the slowest- and fastest-growing clones. The component of phenotypic variation in $r$ attributable to genotypic differences (i.e., broad-sense heritability) was 0.296.

### *Factorial Manipulation of Trait Mean and Variance*

Host *Daphnia* density depended on a three-way interaction between the trait mean treatment, the trait variance treatment, and the presence or absence of parasites ($N = 1{,}320$, $\chi^2_2 = 11.22$, $P = .004$; table A2). When we separated treatments with and without parasites, we found that interactions between the mean and variance treatments drove *Daphnia* density (significant interaction with parasites: $N = 660$, $\chi^2_2 = 13.52$, $P = .001$; marginal interaction without parasites: $N = 660$, $\chi^2_2 = 5.33$, $P = .069$; table A3). Consistent with our initial hypotheses, however, the effects of the interaction were different in each environment (fig. 2).

First, we confirmed our expectations that high trait means are closer to the optimal trait value in environments without parasites ("near-optimal") but suboptimal in environments with parasites ("suboptimal"). With low trait variance, populations with high mean intrinsic growth rates were 20% larger, overall, than populations with low trait means (fig. 2), and this trend was maintained throughout the experiment (fig. A2). However, (i) populations with high mean $r$ had, on average, 25% lower population density after the start of the epidemic (after ~65–70 days; fig. 3); (ii) populations with high mean $r$ experienced much stronger decline in population density compared to parasite-free treatments (fig. A3); and (iii) populations that reached a higher maximum density initially also experienced the most dramatic population collapse once the epidemic started (figs. A2, A3). Furthermore, when parasites were present, populations with high trait means suffered twice the average disease burden (mean infection prevalence) as those with low trait means (fig. 4, low trait variance). Thus,

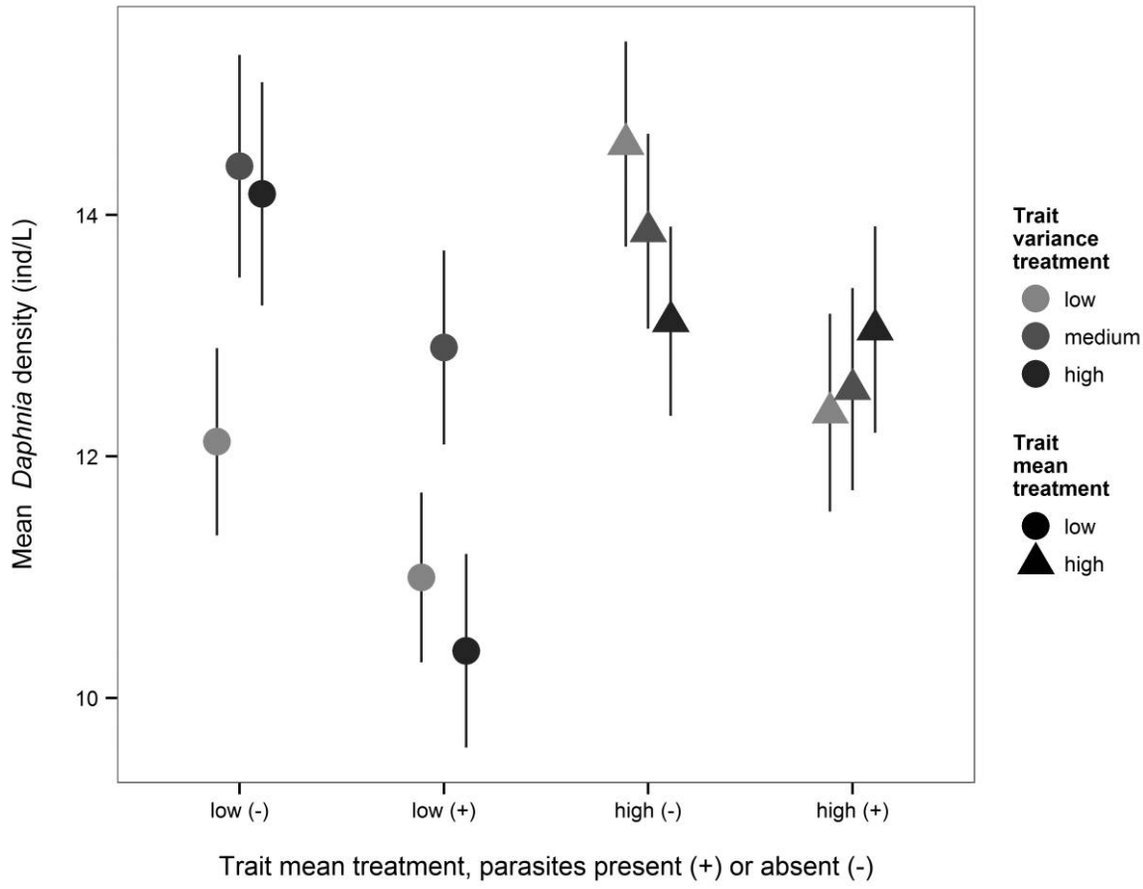


**Figure 2:** Mean *Daphnia* population density ± 1 SE for all treatments in the mean/variance experiment. Shades of gray indicate variance treatment (light gray = low; medium gray = medium; dark gray = high), and shape indicates mean treatment (circles = low; triangles = high). Parasite presence noted by a plus sign and absence by a minus sign.

high *r* values were not beneficial in the presence of parasites, and we see clear evidence consistent with a trade-off between fecundity and disease risk at the level of populations (Hall et al. 2010, 2012; Dibble et al. 2014).

Second, increasing trait variation benefitted populations with suboptimal trait means but tended to harm populations with near-optimal trait means. Importantly, the patterns were consistent in environments with and without parasites, despite the optimal mean trait value differing in each. Without parasites, for instance, increasing phenotypic variance in suboptimal (i.e., low *r*) populations increased overall host density (15.8% and 14.2% increase in abundance from low variance to medium and high variance, respectively; fig. 2). In contrast, increasing trait variance tended to reduce abundance in near-optimal, high-mean populations (6.3% and 10.4% mean reductions from low to medium and high variance, respectively; fig. 2). This pattern caused the significant three-way interaction between mean treatment, variance treatment, and parasite presence and the opposing responses to the mean × variance interaction in each of the parasite- or parasite-free submodels.

We saw the same pattern—that increasing phenotypic variance benefits suboptimal populations but harms near-optimal populations—in environments with parasites. At low levels of initial variance, populations with a low trait mean experienced small disease epidemics (i.e., were near-optimal), and those with a high trait mean suffered large disease epidemics (i.e., were suboptimal; fig. 4) and stronger parasite-mediated population decline (fig. 3). Increasing variation in the near-optimal population doubled the size of disease epidemics (fig. 4, low and medium variance compared to high) and dramatically reduced population density during the epidemic toward the end of the experiment (figs. A2, A3). On the other hand, increasing variation in the suboptimal population reduced epidemic severity by ~37% (fig. 4; high variance compared to medium and low) and slowed the decline in population size relative to disease-free populations (figs. 3, A3). This reflects a significant interaction between

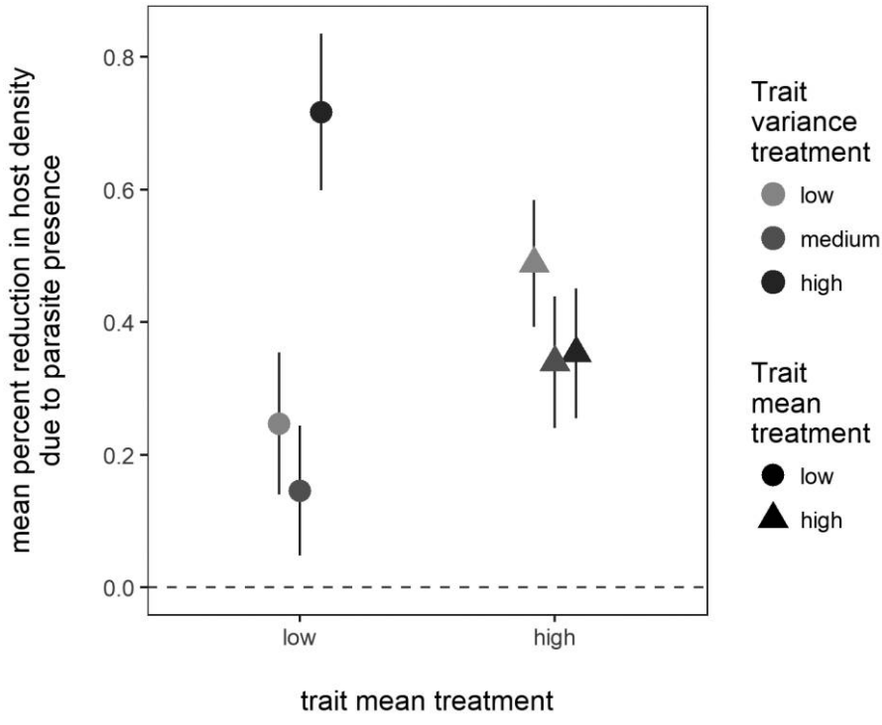


**Figure 3:** Parasite-mediated reduction in host density (mean ± 1 SE) after onset of epidemics (after day 60 to the end of the experiment). Differences are calculated as (density without parasites − density with parasites)/mean(density with + density without) for the same treatment within a block averaged across time steps (see fig. A3). Dashed horizontal line indicates no difference. Shades of gray indicate variance treatment (light gray = low; medium gray = medium; dark gray = high), and shape indicates mean treatment (circles = low; triangles = high).

mean and variance treatments on infection prevalence ($N = 420$, $\chi^2_2 = 15.81$, $P \leq .001$; table A4) and provides another line of evidence indicating that high $r$ values result in lower fitness in environments with parasites.

### *Evolution of Resistance*

The combined effects of increasing phenotypic variance on epidemic severity and parasite-mediated mortality influenced population-level change in individual host resistance to the parasite (fig. A5; table A6). Average susceptibility of individuals at the end of the mean/variance experiment ranged from 50% to 83% among treatments. Parasite presence during the experiment had a significant negative effect on susceptibility (table A6; fig. A5), indicating that populations exposed to parasites harbored more resistant individuals after parasite epidemics (mean susceptibility ± SE, without parasites: $0.78 \pm 0.04$; with parasites: $0.64 \pm 0.05$). Additionally, our mean and variance treatments interacted to affect individual disease risk after the experiment (table A6). This indicates that the effects of altering levels of initial trait variance on individual susceptibility were different, depending on the initial trait mean. For example, in exposed populations, reduction in susceptibility was strongest in high-trait-variance treatments, while the opposite was true in high-trait-mean treatments (fig. A5). Finally, the two treatments that experienced the largest epidemics and largest parasite-mediated reductions in host abundance (figs. 3, 4) also showed the strongest reduction in individual susceptibility over the course of the experiment (fig. 5).

## Discussion

Intraspecific variation plays a major role in ecological dynamics (Bolnick et al. 2011), but the effects of increasing genotypic or phenotypic variation often seem context dependent (table 1). Here we show that this context dependency could be explained by the relationship between a population's mean phenotype and the local environmental optimum. Consistent with our hypothesis, we found positive effects of increasing the variance of a key trait when populations expressed suboptimal trait means but negative effects with near-optimal means. Importantly, this pattern held across different environments (with and without a lethal parasite), despite different optimal trait values in each. Our work shows that increasing intraspecific variation does not always have positive ecological consequences for populations but instead has pre-

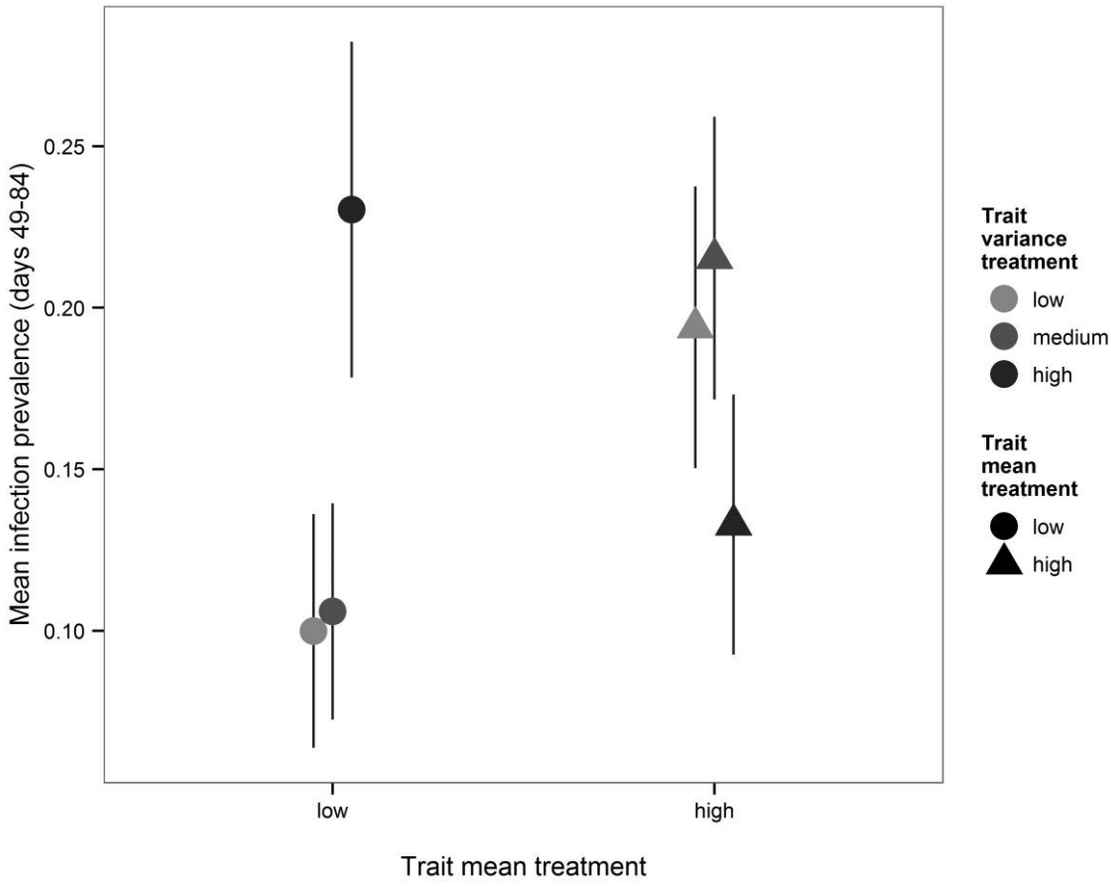


**Figure 4:** Disease epidemic severity (mean infection prevalence over the last 50 days of the experiment ± 1 SE) in the mean/variance experiment. Shades of gray indicate variance treatment (light gray = low; medium gray = medium; dark gray = high), and shape indicates mean treatment (circles = low mean; triangles = high mean).

dictable ecological and evolutionary effects that depend on environmental context and biological interactions.

### *Context Matters*

Depending on the initial ecological context, we found that increasing phenotypic variance increased population density by ~14%, reduced density by ~10%, reduced disease prevalence by ~37%, or increased disease prevalence by 100%. These context-dependent effects, however, were far from random. When populations expressed suboptimal trait means, increasing phenotypic variance allowed immediate access to a high-fitness portion of the phenotype space. This trait sampling (Bolnick et al. 2011) or sampling effect (Crutsinger et al. 2008; Hughes et al. 2008; Vellend et al. 2009) increased the frequency of high-fitness individuals, some of which survived and reproduced well enough to produce positive effects (on growth and disease prevalence) for the population overall. When populations were already near the environmental optimum, in contrast, the same variance manipulation resulted in negative effects for those same population responses.

These opposing effects make some intuitive sense. When populations are struggling, additional high-fitness individuals will have a large immediate advantage over what is already in the population, and selection will tend to increase that advantage over time. In this way, increasing variance can lead to adaptive peak shifts much faster than those produced by mutation and selection alone (Whitlock 1995). When populations are performing well, though, additional high-fitness individuals will have a much smaller advantage over what is already there. This increases the likelihood that the net result of a symmetric variance manipulation will be negligible. In our study, though, the result of a variance manipulation was not simply neutral, as has been seen previously in other systems failing to find positive diversity effects (table 1). Our results thus run counter to the common narrative that diversity is generally beneficial (Hughes et al. 2008; Bolnick et al. 2011; Forsman 2014). Specifically, we

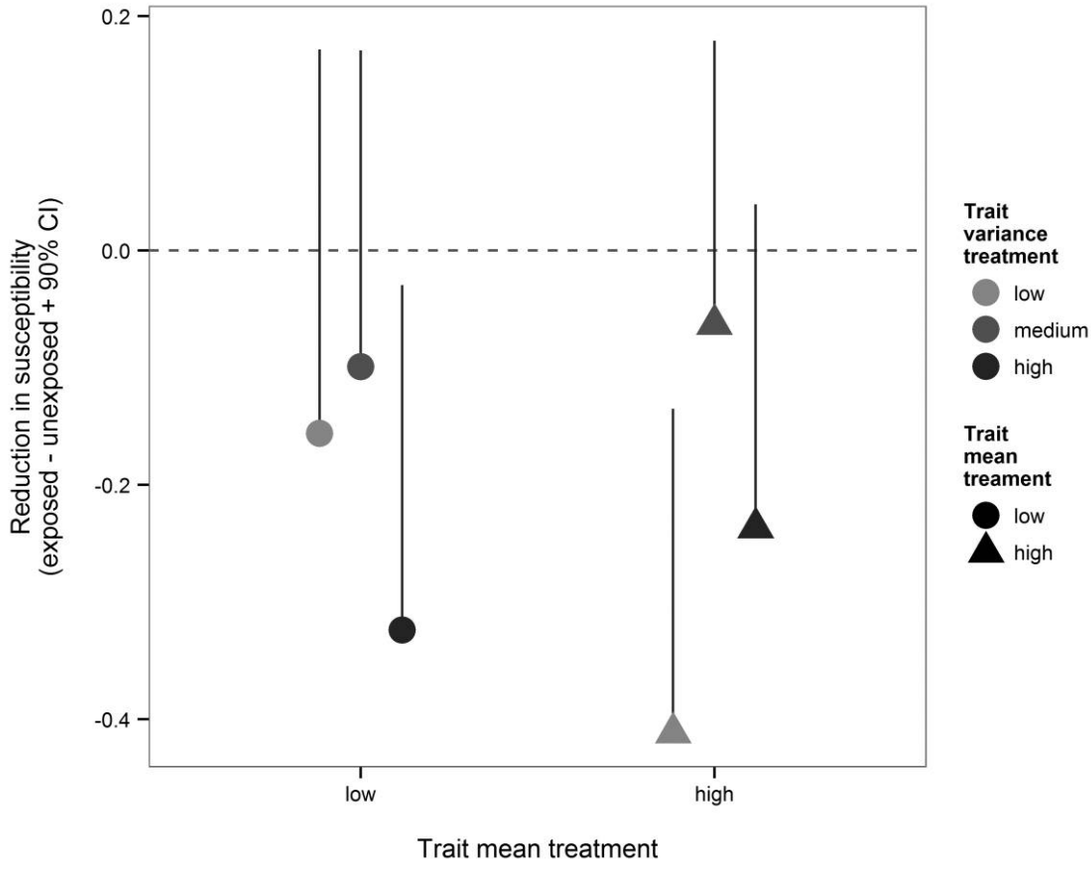


**Figure 5:** Reduction in individual susceptibility after experimental exposure to parasites. Negative values indicate that populations with parasites were less susceptible to parasites after the experiment than those without previous exposure. Error bars indicate a one-tailed test of whether the difference in susceptibility is significantly less than 0 (i.e., 90% confidence interval [CI] on the reduction in proportion infected).

saw negative effects of increasing phenotypic variance on population density and disease prevalence. Negative effects on density were likely due to resource competition (e.g., Dibble and Rudolf 2016), and negative effects on disease prevalence were likely due to an increase in the fraction of susceptible individuals in the population (Anderson and May 1979). Thus, the positive and negative effects of increasing phenotypic variance mirror the complex effects of gene flow into an environment, which can be dramatically positive (e.g., reducing inbreeding depression) or profoundly negative (e.g., outbreeding depression), depending on the environmental and ecological context (Tallmon et al. 2004).

### *Mechanisms of Nonadditivity*

The null expectation of any symmetric increase in phenotypic variance is no net effect, as any benefit conferred by higher-fitness individuals is offset by the detrimental effects of lower-fitness individuals (and vice versa). We observed nonadditivity in positive and negative directions and believe that the same underlying mechanisms contributed to both. Jensen's inequality comes about when the mean of a function evaluated over its range of inputs is different from the value of the function evaluated at the mean input value (Ruel and Ayres 1999; Bolnick et al. 2011). In our study, nonlinear relationships between individual traits (i.e., range of inputs) and the response of the population (i.e., the function) could generate Jensen's inequality. In this way, two populations with the same trait mean but different levels of trait variance could show different responses at the population level (Bolnick et al. 2011). Importantly, the shape of the nonlinear response determines whether the response of the population increases or decreases with increasing. If convex, increasing variance around a constant mean will increase the average response. If concave, increasing variance will reduce the response (Lacey et al. 1983).

Natural selection drives the shape of the relationship between individual traits and the response of the population.

This provides the biological underpinning of any nonadditivity due to Jensen's inequality. A curve in the trait response relationship comes about if the individual fitness consequences of a one-unit increase or decrease in a trait value differ (Lacey et al. 1983) and if these individual fitness values then disproportionately affect the overall response of the population. Such a curvature seems likely in our experimental suboptimal and near-optimal situations, simply because the fitness advantage or cost of a trait is unlikely to increase or decrease forever. Starting from either extreme (i.e., suboptimal vs. near-optimal trait means) might increase the likelihood of a decelerating relationship between individual traits and the population itself, creating a nonlinearity that can give rise to Jensen's inequality. Furthermore, selection itself can drive nonlinearities, if small initial advantages provided by certain phenotypes increase through time (e.g., exponential population growth and intraspecific competition). Given the recent evidence for rapid evolution affecting ecological dynamics (Schoener 2011; Pantel et al. 2015), this second effect may be pronounced, particularly as increasing phenotypic variance allows for large jumps in trait space (Whitlock 1995).

### *Eco-Evolutionary Dynamics and Community Assembly*

Context dependence in mechanism matters in both the short term and the long term. While we observed significant reductions in mean individual susceptibility after exposure to a pathogen, the interesting result is not the reductions themselves but rather the context that led to them. We saw the largest reductions in susceptibility in populations suffering from the strongest selection, in terms of both infection prevalence (fig. 3) and reductions in population size due to disease (figs. 2, 4). This makes sense, given that rapid evolution of resistance contributes to the termination of natural disease epidemics in the closely related *Daphnia dentifera* (Duffy and Sivars-Becker 2007; Duffy and Hall 2008). In one of our treatments, though, strong selection was caused by a suboptimal mean phenotype with insufficient phenotypic variance to escape the negative effects of parasites. In the other treatment, strong selection was caused by a near-optimal trait mean but too much variance, which likely altered the disease invasion threshold (fig. A4). In other words, increasing phenotypic variance in a resistant host population led to large disease outbreaks, but these outbreaks resulted in strong selection for increased resistance and led to significant trait change. Genotypic richness was fixed in all treatments, allowing us to isolate the effects of phenotypic variance. Our results show that different pathways can lead to the same eco-evolutionary dynamics, highlighting the importance of a better mechanistic understanding of how intraspecific variation contributes to biological patterns in nature.

### *Relevance and Extensions*

Our work reflects the relatively stochastic process of patch colonization in temporary habitats, where populations and communities are periodically restarted via immigration or from dormant egg banks. Founder effects during colonization can lead to mismatches between phenotypes and local environmental conditions (e.g., suboptimal trait means), and our positive effects of phenotypic variance help to explain why greater intraspecific variation often benefits colonizing populations (Agashe 2009; Forsman 2014). Once patches are colonized, dispersal can affect local trait distributions, and while it might not do so in the symmetric fashion of our experiment, our results provide a useful framework for predicting the ecological and evolutionary effects of immigration into an existing patch. For instance, an influx of maladapted phenotypes might reduce ecological performance in the short term (Urban 2006) but could also act as the spark that ignites a strong selective pressure and that results in future evolutionary change (e.g., figs. 2–5). Furthermore, populations with suboptimal trait means in a local environment and insufficient phenotypic variance might benefit from immigration of novel phenotypes, provided the additional variance is related to local fitness (e.g., a phenotypic rescue effect).

Interestingly, our results also suggest an important temporal component to diversity effects. As populations adapt to local conditions, our results show that the effects of increasing phenotypic variance (e.g., through immigration) should become increasingly negative. If immigration has more positive effects early in the process of colonization (when populations are more likely to exhibit phenotype-environment mismatches), gene flow should follow a similar pattern. There is thus potential for interesting temporal dependence in the interactions between founder effects, local monopolization (De Meester et al. 2002; Pantel et al. 2015), selection against migrant individuals (Nosil et al. 2005; Marshall et al. 2010), and the overall maintenance of gene and trait heterogeneity in natural environments.

### *Limitations and Caveats*

While our general framework should be applicable to a number of systems, our results have some caveats. First, our variance manipulation was not perfectly symmetrical. To reflect natural variation, we hatched unique *Daphnia* clones from resting eggs in dry soil. The trade-off was a constraint in the amount and distribution of phenotypic variation (fig. A1), which resulted in imperfect treatment allocations. Specifically, there were differences in mean intrinsic rate of increase among the low-mean treatments (fig. 1). If anything, however, these differences should have reduced our ability to see significant treatment effects by making the low-

mean, low-variance treatments closer to the high-mean treatments. Instead, we saw that low-mean, low-variance populations were consistently distinct, suggesting that our clone allocations created adequate distinctions among experimental treatments. Similarly, parasite introduction had to be delayed to allow populations to grow enough for a successful epidemic. This means that by the time the epidemics took off, population means and variances were likely not the same as at the setup, leading to overall increase in mean $r$ (and thereby susceptibility) and reduction in variance. This should have reduced our ability to detect effects of genotypic variance in parasite treatments. Yet we saw clear variance treatment effects with and without parasites, indicating that our treatments were still successful and, if anything, underestimate the effect of variance in parasite treatments. A second limitation of our study was that we did not track the fate of individual genotypes over time. In this way, we cannot definitively conclude that the significant trait change we documented was evolution. Phenotypic plasticity is a plausible alternative for this trait change, though we note that there is strong evidence from prior work in this system for rapid evolution in natural host populations (Duffy and Sivars-Becker 2007; Duffy and Hall 2008). Last, we prevented sexual reproduction in our experiment (by removing ephippia from populations), which limits the direct applicability to many systems. Nonetheless, we believe our main results are broadly applicable.

## Conclusions

Here we introduced and tested a conceptual framework to explain context-dependent effects of increasing intraspecific diversity in a population. Importantly, our results emphasize that there is no a priori reason to expect universally positive effects of intraspecific variation on an ecological timescale. Rather, our expectations regarding the result of manipulating intraspecific variation should be colored by phenotype-environment matching and likely eco-evolutionary responses. Studies that predict but do not find positive ecological effects of increasing genotypic or phenotypic variation, then, may have insufficient variation in important traits, be located in a flat section of the trait-fitness response curve, or lack the strong selection pressures necessary to generate Jensen's inequality. Furthermore, we show that increasing phenotypic variance around a fixed trait mean can alter the strength of local selective pressures, which is not commonly considered in ecological diversity studies (e.g., table 1). Our results also suggest that a negative effect of increasing intraspecific variation is a valid but undertested hypothesis—and one that might have more relevance to the natural world, given that immigrants are generally less fit than resident individuals in an environment (Kawecki and Ebert 2004). Intraspecific variation plays a complex role in ecological dynamics, but some of its context dependency comes from simple, predictable mechanistic processes.

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