5-2016

THE JOINT EFFECT OF PHENOTYPIC VARIATION AND TEMPERATURE ON PREDATOR-PREY INTERACTIONS

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by

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A DISSERTATION

Presented to the Faculty of

The Graduate College at the University of Nebraska

In Partial Fulfillment of the Requirements

For the Degree of Doctor of Philosophy

Major: Biological Sciences

(Ecology, Evolution and Behavior)

Under the Supervision of Professor John P. DeLong

Lincoln, Nebraska

May, 2016

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University of Nebraska, 2016

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Understanding the factors underpinning to food web structure and stability is a long-standing issue in ecology. This is particularly important in a context of global climate change, where rising environmental temperatures may impact the way species interact, potentially leading to changes in food web structure and to secondary extinctions resulting from cascading effects. In order to understand and predict these changes, we need to hone our comprehension on the way predators and their prey interact. Recent studies suggest that, in order to do so, we need to focus on the traits controlling those interactions, such as body size. Mean body size and its intraspecific variation can in turn be affected by temperature, a pattern known as the temperature-size rule. To understand how warming may affect predator-prey interactions and through them, food web structure and dynamics, we thus first need to understand how traits, their within species variation, and temperature, may jointly affect these interactions. Here, I address these unknowns using both empirical and theoretical tools. I have shown that variation in the traits controlling predator-prey interactions may determine the strengths of these interactions, and through them, their stability and overall dynamics. I have also shown this to be truth for species living as metapopulations, where variation in the traits controlling migration plays an important role in determining their chance of persisting. Moreover, I showed empirically that many of these findings hold in a freshwater predator-prey system, and based on empirical results on how temperature affects body size and its variation, I made predictions as to how warming may affect interaction strengths in this system. I thus found evidence of temperature determining the way predators and their prey interact, leading to important changes in the body size structure of entire food webs across aquatic ecosystems. My results highlight how intraspecific variation has important yet largely overlooked ecological effects, and how these effects can be mediated by environmental temperature.

To my parents, Gladys and Jean-François, for being the platinum-iridium alloy bar in my metric system.

To my wife, Marie-Claire, without whom none of this would have been possible.

Thank you for making me who I am.

I am grateful to my committee, Diana Pilson, Chad Brassil, Richard Rebarber and Stefano Allesina for holding my hand when needed and for making seemingly terrifying situations fun.

I am beyond indebted to my advisor, John DeLong, whom, despite telling me to my face that I'm Argentine, which I'm not, always went above and beyond the call of duty to ensure my success. I'm also thankful to John for always encouraging me to keep digging.

I want to thank my labmates from the Dezuka lab (DeLong-Shizuka, in case you're wondering) for ending my loneliness in the fourth floor and making things so much cooler (and full of nerf guns), as well as the good friends I have made in my time in Nebraska. I came for a PhD, I'm taking away so much more.

Last but not least, thanks to the Manter Hall staff, Mindy Peck, Carla Tisdale, Tammy Kortum, Kris Patrick, Deb Pinkelman, Gayle Schuler, Sarah Fredregill, Linda Trouba, and Barb Nordmeyer for making complicated issues have simple solutions.

I'm particularly grateful with Mindy Peck, for being an all around beautiful human being. I'm taking her to California with me, it's decided.

The University of Nebraska–Lincoln provided funding for my ~~survival~~ research through an Othmer Fellowship. My first years of research and travels to meeting were also partly covered by the School of Biological Sciences Special Funds. Last, a National Science Foundation Doctoral Dissertation Improvement Grant (DEB-1501668) made Chapters 3 and 5 possible.

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OVERVIEW

1. THE PROBLEM

One of ecology's most pressing goals is to predict how ecosystems will respond to global climate change. Understanding the structure and stability of complex networks of interacting species is crucial for the accuracy of these predictions, but refining this understanding is a challenging task. The structure of food webs – collections of species and their feeding interactions – ultimately depends on the interplay between multiple levels of biological complexity, from individuals to ecosystems. Recent studies argue that to successfully understand food web structure we need a mechanistic understanding of how prey and predator traits affect their feeding interactions, as these are ultimately determined by the traits involved in the processes of finding, capturing and consuming prey. To hone our capacity to predict how food webs will respond to future climates we thus need to address the following questions: *how do ecological processes scale up from predator and prey individuals and their traits to ecosystems? How do these effects cascade across levels of biological complexity to determine food web structure?* And, *how will global climate change alter the way in which this happens?* My research aims at addressing these questions by using tools and approaches at the interface between theory and data.

2. PHENOTYPIC VARIATION AND PREDATOR-PREY INTERACTIONS

While evolutionary biology has long recognized individual-level phenotypic variation as the key to understand evolution, ecology has historically dismissed individual variation as uninformative noise around mean values of interest. However, individual variation can have important effects on ecological processes in a number of

circumstances, especially when these processes have a non-linear dependence upon underlying traits. Whether and how this variation may affect predator-prey interactions was, however, poorly understood (Gibert et al 2015).

My theoretical work showed that individual variation in the traits controlling attack rate and handling time (e.g. body size) can decrease interaction strengths between consumers and resources, and, through that, increase stability and species persistence (Gibert and Brassil 2014, Gibert & DeLong 2015). This effect, however, is mediated by the difference between the mean trait value in the population and the optimal value (i.e. the phenotypic mismatch). This suggests that both current and past selection acting on traits, as well as ecological determinants of such traits (like temperature in the case of body size) may play a major role in modulating how phenotypic variation affects predator-prey interactions (Gibert et al 2015). I have also shown that phenotypic variation in traits involved in migration can also affect the persistence of species that live as metapopulations (Gibert 2016), that is, collections of populations that share migrants.

All in all, my theoretical work has shown that phenotypic variation in traits that have a functional effect on ecological processes can have, *per se*, important yet largely overlooked impacts on ecological dynamics.

3. THE JOINT EFFECT OF TEMPERATURE AND PHENOTYPIC VARIATION

Understanding the effect of temperature on predator-prey interactions is crucial to predict how global warming may affect the structure and dynamics of food webs. Through its effects on metabolic rates, temperature sets the pace at which myriad ecological processes occur, including how predators and prey interact. I have shown that temperature affects the speed at which animals move by incorporating

how metabolic rates scale with body size into biomechanical models of animal movement. By doing so, I have shown that temperature increases the speed at which animals move in predictable ways (Gibert et al 2016). The temperature-dependence of animal movement has important consequences for predator-prey interactions due to the dependence of predator attack rates on animal velocity. Based on this, I have shown that temperature determines how strongly predators interact with their prey, thus affecting the stability and persistence of predator-prey systems within food webs (Gibert et al 2016).

Temperature can directly determine both mean body size and its intraspecific variation in ectotherms (a process known as the temperature-size rule), and mediate predator-prey interactions through its effect on animal movement. Variation in body size, for example, can also alter predator-prey interactions, whenever attack rates and handling times depend nonlinearly upon body size. Thus, variation and temperature may jointly affect predator-prey interactions, but how or whether this occurs in nature is largely unknown. I was awarded a *Doctoral Dissertation Improvement Grant* from NSF to test whether the joint effect of temperature and variation in body size can be detected empirically in a freshwater predator-prey system. Thus far, my results suggest that both mean body size and its variation determine the parameters controlling foraging rates between the copepod predator *Eucyclops agilis* and the protist prey *Paramecium caudatum*, and that, when this variation is not taken into account, the overall effect of temperature on interaction strengths may be underestimated (Gibert & DeLong, *in prep*). Lastly, whether the effects of temperature on pairwise predator-prey interactions scale-up to entire food webs is by and large unknown, but my results suggest that temperature has important effects on

food web body-size structure, by altering predator-prey body size ratios in specific ways (Gibert & DeLong 2014).

4. FUTURE STEPS

So far, I have shown that phenotypic variation in traits controlling predator-prey interactions can determine their strength and dynamics (Gibert & Brassil 2014, Gibert & DeLong 2015, Fig 1). This variation can also affect the persistence of metapopulations (Gibert 2016), and can be detected empirically using simple experiments (Gibert & DeLong, *in prep*, Fig 1). Furthermore, the effect of phenotypic variation may be mediated by environmental temperature (Gibert & DeLong, *in prep,* Fig 1). Temperature can, in and of itself, directly affect predator-prey interactions through its effect on animal movement (Gibert et al. 2016) as well as on the body-size structure of complex networks of interacting species like food webs (Gibert & DeLong 2014, Fig 1). How phenotypic variation may directly or indirectly affect the structure and dynamics of entire food webs (and not just pairwise predator-prey interactions), as well as how temperature may mediate this effect, are, however, largely unknown (Fig 1).

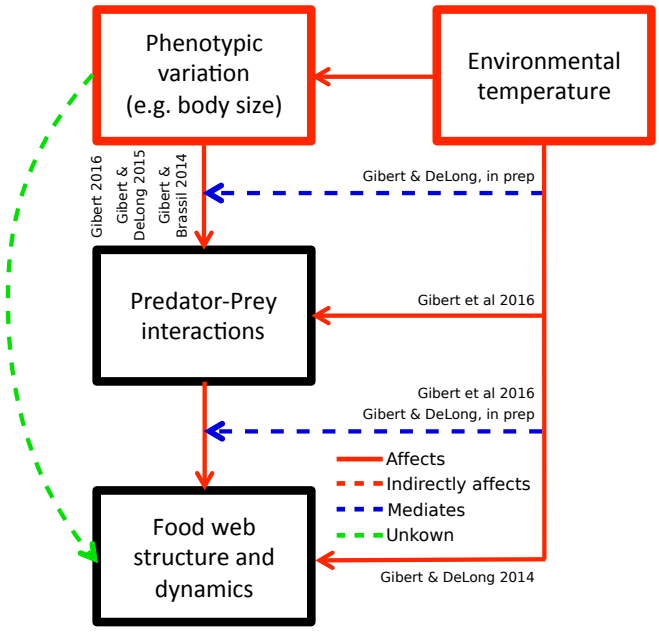

Fig. 1: Diagram showing how phenotypic variation and temperature can have effects across levels of biological organization, as well as how my work has contributed in our understanding of these processes.

I was awarded a *James S. MacDonnell Foundation Postdoctoral Fellowship in Complex Systems* to address this exact problem. For the next three years I will be assessing 1) how phenotypic variation influences predator connectivity (i.e. the number of feeding interactions of a predator within a food web), 2) how it determines predator trophic level, and, 3) how these ideas can be tested with empirical data. Next, I can make predictions as to how temperature may increase or decrease both species connectivity and trophic level based on its effect on variation in body size, and then test these predictions using food web data across latitudinal gradients. By doing so, I hope to contribute to our understanding of how traits and their variation may interact with temperature to determine food web structure, and thus help predict the response of complex food webs to rising global temperatures.

A major challenge to making any of these predictions, and thus, to our ability to take appropriate action in response to rising temperatures, is the fundamental impossibility to test any of them in natural conditions, this is, with actual large, complex food webs. My 5 to 10 year career plan is to develop an empirical system where these predictions can be tested: that is, an “ecosystem in a jar”. Ecosystems associated with moss along temperate forests may be exactly what I am looking for. Indeed, moss provides habitat to hundreds of species, from decomposers to top predators, from both aquatic and terrestrial communities. Species diversity can border on the hundreds, and organisms range from aquatic protists (a system with which I have experience), to small invertebrates such as mites and tardigrades. Both field and lab experiments can be performed, such as artificial warming experiments using small field greenhouses, or by transferring plaques of moss to the laboratory to put them in growing chambers at controlled temperatures. Also, individuals within species can be counted and their traits (e.g. body size) can be measured under the microscope. It is

therefore possible to assess not only how temperature may alter food web structure, but also how it may alter entire intraspecific trait distributions for entire communities. The composition of each community can also be manipulated in the lab by changing the number and identity of the species living in a given moss plaque, which opens-up multiple lines of inquiry in this system.

As a post-doctoral associate I plan on starting to develop such a system, a task that will probably take about 5 years to be completed. Moss ecosystems would allow me to answer some of the most pressing ecological issues of our time. These answers not only are greatly needed, but near impossible to obtain without an “ecosystem in a jar” like moss. During my career I thus hope to address fundamental questions in ecology at the interface between theory and data with important implications for a world where temperatures are on the rise.

CHAPTER 1

INDIVIDUAL PHENOTYPIC VARIATION REDUCES INTERACTION STRENGTHS IN A CONSUMER-RESOURCE SYSTEM

Jean P. Gibert & Chad E. Brassil

Key-words: Intraspecific variation, interaction strengths, species persistence, stability, invasion.

ABSTRACT

Natural populations often show variation in traits that can affect the strength of interspecific interactions. Interaction strengths in turn influence the fate of pairwise interacting populations and the stability of food webs. Understanding the mechanisms relating individual phenotypic variation to interaction strengths is thus central to assess how trait variation affects population and community dynamics. We incorporated non-heritable variation in attack rates and handling times into a classical consumer-resource model to investigate how variation may alter interaction strengths, population dynamics, species persistence and invasiveness. We found that individual variation influences species persistence through its effect on interaction strengths. In many scenarios, interaction strengths decrease with variation, which in turn affects species coexistence and stability. Because environmental change alters the direction and strength of selection acting upon phenotypic traits, our results have implications for species coexistence in a context of habitat fragmentation, climate change, and the arrival of exotic species to native ecosystems.

INTRODUCTION

Individuals of the same population often show extensive variation in morphology (Bolnick et al. 2003), phenology (Dupont, Trojelsgaard & Olesen 2011), behavior (e.g. Tinker, Bentall & Estes 2008), and resource utilization (e.g. Estes et al. 2003). This variation can arise from underlying genetic diversity (Lynch & Walsh 1998), or be plastic and result from environmental variability and genotype by environment interactions (Fordyce 2006). The importance of genetic and phenotypic variation within populations has long been recognized by evolutionary biology, as heritable individual variation constitutes the raw material upon which natural selection can act (Dobzhansky 1937). Despite a long tradition of considering variation in ontogenetic stages and size within populations, ecological theory has largely overlooked individual variation in its broader sense (Lomnicki 1988). Populations are generally treated as collections of homogeneous individuals and mean demographic parameters, such as mortality or attack rates, are generally used to study population and community dynamics (Sherratt & MacDougall 1995). However, mean demographic rates can be misleading (Inouye 2005), as individual variation may affect demographic parameters and ecological attributes in multiple ways (Bolnick et al. 2011; Pettorelli et al. 2012).

Extensive individual phenotypic and dietary variation has been described for several organisms such as carnivorous marine mammals (e.g. Harcourt 1993), pollinating insects (Dupont et al. 2011), marine and fresh water fishes (e.g. Vander Zanden et al. 2000), as well as several bird species (e.g. Golet et al. 2000). However, only a handful of these studies assessed the effect of individual variation upon demographic or ecological traits (e.g. Lloyd-Smith et al. 2005, Melbourne & Hastings 2008). For example, individual variation in resource utilization among southern sea

otters (*Enhydra lutris nereis*) structures population-level consumer-resource networks in predictable ways (Tinker et al. 2012). This dietary variation leads to differences in energy intake among individuals, as well as to differences in individual mortality rates through differential pathogen exposure (Tinker et al. 2008; Johnson et al 2009). Another study showed that the mean reproductive rate of sockeye salmons (*Oncorhyncus nerka*) increases over long time spans with increasing individual variation in life-history traits through a portfolio effect (Greene et al. 2010). Finally, coexistence could theoretically increase with increasing levels of individual variation in attack rates in apparent competition systems with heritable trait variation (Schreiber, Bürger & Bolnick 2011), and stability could be enhanced whenever behavioral variation is included in consumer-resource systems (Okuyama 2008). Together, these results suggest that the consequences of individual phenotypic variation for population and community dynamics can be important.

Populations embedded in large, complex networks of interacting species such as food webs, often show variation in anti-predator defense (Duffy 2010), competitive ability (Lankau & Strauss 2007), or resource utilization (e.g. Estest et al. 2003), all of which can affect interspecific interactions (Pettorelli et al. 2012). The strength of these interactions influences the fate of pairwise interacting populations (e.g. Wootton & Emmerson 2005) and food web stability (e.g. May 1972; Allesina & Tang 2012). Thus, any factor influencing interaction strengths could affect species persistence and stability in consumer-resource systems. To fully understand food web stability as well as population and community dynamics, we need to assess the effects of individual variation on ecological attributes that determine the strength of consumer-resource interactions.

Bolnick et al. (2011) identified several mechanisms through which individual variation could affect interaction strengths, including adaptive and stochastic eco-evolutionary feedbacks, increased food-web connectivity, portfolio effects, phenotypic subsidy and Jensen's inequality. The latter, a mathematical rule, implies that mean interaction strengths can differ from the interaction strength of the mean individual of the population whenever the variable trait or attribute has purely concave up or down effects on interaction strengths (Jensen 1906; Ruel & Ayres 1999), like attack rates or handling times do (Bolnick et al. 2011). Typically, interaction strengths have been assumed to be functions of mean attack rates and handling times, but, because of Jensen's inequality, this approach may miss crucial aspects of population and community dynamics. For example, individual variation in attack rates may decrease mean interaction strengths, while individual variation in handling times may increase mean interaction strengths (Fig. 1a, b, Bolnick et al. 2011). However, because attack rate and handling times are not independent from each other (DeLong & Vasseur 2012), it is important to understand what would happen when there is individual variation in both ecological attributes at the same time, as it may occur in a natural system.

In this study, we address how non-heritable individual variation in attack rates and handling times affect interaction strengths within consumer-resource interactions, and how this in turn can affect consumer-resource dynamics, species coexistence and overall stability. To do so, we included individual variation in traits controlling attack rate and handling time in classic consumer-resource models to assess how different levels of individual variation might affect ecological dynamics, species persistence and stability in simple consumer-resource models. By doing so, this study answers the following questions: what is the effect of individual variation on interaction strengths?

How does this effect alter ecological dynamics and stability? We found that individual variation in attack rate and handling time can increase species persistence and stability through its effect upon interaction strengths. This has in turn important implications for the conservation of endangered species and the management of exotic ones.

MATERIALS AND METHODS

Interaction strengths in classic consumer-resource models

In a consumer-resource interaction model, consumer populations grow through ingesting a resource, which affects the growth rate of that resource (e.g. Rosenzweig & MacArthur 1963). The rate of change of resource and consumers over time can be modeled as:

$$\begin{aligned} \frac{dR}{dt} &= r(R) - f(R,C) \\ \frac{dC}{dt} &= \varepsilon\, f(R,C) - g(C) \end{aligned}, \qquad (1)$$

where $f(R,C)$ and $g(C)$ are the mortality rates for resource and consumers respectively, and $r(R)$ and $\varepsilon f(R,C)$ are the reproductive rate of resource and consumers respectively. The functional form of $f(R,C)$ is typically assumed to be the same for both consumers and resources, but its magnitude is scaled in the consumer equation by an efficiency parameter, ε, that can take any non-negative real value. May defined interaction strengths (*IS*, from now on) in systems like (1) as the change in the rate of change of one of the species relative to a small change in the other species' density. Here we use May's definition on a per-capita basis, as advocated by

Wooton & Laska (1998), i.e. $IS_R = \frac{1}{R}\frac{\partial dR/dt}{\partial C}$ for resource and $IS_C = \frac{1}{C}\frac{\partial dC/dt}{\partial R}$ for consumers. Applying this definition to (1), we obtain:

$$IS_R = -\frac{1}{R}\frac{\partial f(R,C)}{\partial C} \tag{2}$$

$$IS_C = \frac{\varepsilon}{C}\frac{\partial f(R,C)}{\partial R} \; . \tag{3}$$

If we further assume a Holling type II functional response (Holling 1959), where $f(R,C) = \frac{\alpha RC}{1+\alpha\,\eta\,R}$, we can get expressions for these interaction strengths that depend on the main parameters controlling the consumer-resource interaction:

$$IS_R(\alpha,\eta) = -\frac{\alpha}{1+\alpha\,\eta\,R} \tag{4}$$

$$IS_C(\alpha,\eta) = \varepsilon\,\frac{\alpha}{\left(1+\alpha\,\eta\,R\right)^2}, \tag{5}$$

where α denotes the predator's attack rate and η its handling time. Because attack rates and handling times are ecological attributes that depend on phenotypic traits, it is possible to incorporate variation in those traits into (4) and (5).

Incorporating individual variation

In a previous theoretical study, attack rates were assumed to depend on the value, x, of a quantitative trait (Schreiber et al. 2011). Here, we assumed that both attack rate and handling time depend on the value of a normally distributed quantitative trait with mean $\bar{x}$ and variance σ^2. The probability density function of such a trait is:

$$p(x,\bar{x}) = \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left[-\frac{(x-\bar{x})^2}{2\sigma^2}\right]. \quad (6)$$

Following (Schreiber et al. 2011), we assumed the predator's attack rate, $\alpha(x)$, to be maximal at a given optimal trait value $x = \theta_\alpha$, and to then decrease away from that maximum in a Gaussian way:

$$\alpha(x) = \alpha_{\max} \exp\left[-\frac{(x-\theta_\alpha)^2}{2\tau^2}\right], \quad (7)$$

where $\alpha_{\max}$ is the maximal attack rate and τ determines how steeply the attack rate declines away from θ_α (Fig. 1c). We further assumed the handling time, $\eta(x)$, to be minimal at a given optimal value $x = \theta_\eta$, and to increase away from that minimum in a Gaussian way:

$$\eta(x) = \eta_{\max} - (\eta_{\max} - \eta_{\min}) \exp\left[-\frac{(x-\theta_\eta)^2}{2\nu^2}\right], \quad (8)$$

where $\eta_{\max}$ and $\eta_{\min}$ are the maximal and minimal handling times respectively, and ν determines how steeply the handling time increases away from θ_η (Fig. 1d).

The assumed functional forms for the attack rate and the handling time have been reported for a variety of organisms when body size is considered as the underlying trait of interest (Rall et al. 2012). Our model also assumes that the attack rate and the handling time have inverse functional forms: while attack rate goes down as the trait moves away from the optimum, handling time goes up. The latter is justified by recent empirical work in protists revealing that attack rate and handling time are negatively correlated (DeLong & Vasseur 2012).

We define $d_\alpha^2 = (\bar{x} - \theta_\alpha)^2$ and $d_\eta^2 = (\bar{x} - \theta_\eta)^2$, as the squared distance between the mean trait in the population and the optimal value. The optimal value is set by past

and existing selective pressures, and is the value at which attack rate is maximal and handling time is minimal (referred to as phenotypic mismatch). Phenotypic mismatch can be seen as a measure of how well adapted the predator species is at attacking and handling a particular resource. The larger the mismatch is, the smaller the attack rate and the larger the handling time. Phenotypic mismatch has been shown in other traits to affect ecologic interactions and speciation (Raimundo et al. 2014), as well as individual fitness (Anderson, Terblanche & Ellis 2010). However, it does not need to be the same for both attack rate and handling time, but was assumed to be so for simplicity throughout the main text (but see Appendix I and II for different assumptions).

To get mean interaction strengths, we thus integrated interaction strengths across the nonlinearity of the functional response and the underlying trait distribution as:

$$\overline{IS_R(\alpha,\eta)} = -\frac{1}{R}\frac{\partial}{\partial C}\left(\int_{-\infty}^{\infty}\frac{RC\,\alpha(x)}{1+\alpha(x)\,\eta(x)\,R}\,p(x,\bar{x})\,dx\right), \tag{9}$$

$$\overline{IS_C(\alpha,\eta)} = \frac{\varepsilon}{C}\frac{\partial}{\partial R}\left(\int_{-\infty}^{\infty}\frac{RC\,\alpha(x)}{1+\alpha(x)\,\eta(x)\,R}\,p(x,\bar{x})\,dx\right). \tag{10}$$

Using Leibniz integration rule, the derivatives can be passed under the integral sign and (9) and (10) can be simplified as:

$$\overline{IS_R(\alpha,\eta)} = -\int_{-\infty}^{\infty}\frac{\alpha(x)}{1+\alpha(x)\,\eta(x)\,R}\,p(x,\bar{x})\,dx, \tag{11}$$

$$\overline{IS_C(\alpha,\eta)} = \varepsilon\int_{-\infty}^{\infty}\frac{\alpha(x)}{\left(1+\alpha(x)\,\eta(x)\,R\right)^2}\,p(x,\bar{x})\,dx. \tag{12}$$

Equations (11) and (12) depend on individual variation (σ^2) as well as phenotypic mismatch (d^2) and can be estimated numerically either at equilibrium (when C and R are constant), or instantaneously (for any given time t).

General dynamics

To explore the effect of individual variation on consumer-resource interactions and species persistence through interaction strengths, we explored the dynamics of a Rosenzweig-MacArthur consumer-resource model (Rosenzweig & MacArthur 1963). We analyzed the behavior of the model under varying levels of individual variation using:

$$\begin{aligned} \frac{dR}{dt} &= rR\left(1-\frac{R}{K}\right)-\int_{-\infty}^{\infty}\frac{RC\,\alpha(x)}{1+\alpha(x)\,\eta(x)\,R}\,p(x,\bar{x})\,dx \\ \frac{dC}{dt} &= \varepsilon\int_{-\infty}^{\infty}\frac{RC\,\alpha(x)}{1+\alpha(x)\,\eta(x)\,R}\,p(x,\bar{x})\,dx - mC \end{aligned}, \qquad (13)$$

where K is the carrying capacity for the resource, m is the mortality rate of the consumer and all other parameters are as explained before. Our main objective is to tie the dynamic effect of phenotypic variation on attack rate and handling time through their effect on interaction strengths.

General questions

In this study we specifically addressed the following questions: first, does individual variation affect the magnitude of the interaction strength between consumers and resources? We addressed this question by evaluating equations (11) and (12) under increasing levels of individual variation. We also assessed how sensitive interaction strengths were to variation in attack rate and handling time by quantifying their elasticity for varying levels of individual variation (Appendix III). Second, if individual variation affects interaction strengths, it can potentially affect population dynamics through the latter. So, would individual variation affect species persistence in a consumer-resource interaction? And, would individual variation affect the stability of consumer-resource interactions? To address these, we derived

the conditions for consumer persistence. We also used model (13) to assess how individual variation affected the consumer resource-dynamics, and found approximate minimal levels of variation needed to achieve stable dynamics. Our approach mimics what is observed in the field (e.g. Matthews et al. 2010), where normally distributed quantitative traits affect the individual use of resources through attack rates and handling times (e.g. Robinson 2000). However, both trait distributions and ecological attributes may not be symmetric in nature; for example, trait distributions may be log-normal (e.g. Gows, Gaston & Chown 2011) and attack rates may be asymmetric (Vucic-Pestic et al. 2010). We therefore explored three other possible scenarios: (1) trait distributions are asymmetric (Appendix IV), (2) handling time and attack rate are asymmetric functions of the underlying trait x (Appendix V) and (3) both the trait distribution and the functions relating handling time and attack rate to the underlying phenotypic trait are asymmetric (Appendix VI).

RESULTS

Interaction strengths

When phenotypic mismatch is small ($d_\alpha \sim 0$ and $d_\eta \sim 0$), interaction strengths decay in both consumers and resources with increasing individual variation (Fig. 2a). This is also true under varying resource levels (Fig. 2b). In contrast, if phenotypic mismatch is sufficiently large ($|d_\alpha| >> 0$ or $|d_\eta| >> 0$), interaction strengths first increase with variation, and then decrease (Fig, .c), which is also true for varying resource levels (Fig, 2d). These effects seem to increase with resource levels in all cases (Fig. 2b, d). Increasing phenotypic mismatch leads to smaller interaction strengths across all levels of variation (Fig. 2a, c). Our results are robust to changes in the underlying assumptions such as incorporating asymmetric trait distributions

(Appendix IV), incorporating asymmetric attack rates and handling times (Appendix V), or both asymmetric distributions and asymmetric attack rates and handling times (Appendix VI). These results are robust to changes in parameter values (Appendix VII). Notice, however, that asymmetric distributions alone enlarge the range of possible scenarios where interaction strengths decrease with individual variation while the opposite is true for asymmetric attack rate and handling time, regardless of the underlying distribution (Apendices S4, S5 and S6).

Persistence and Stability

For a consumer to be able to persist, the following inequality must hold:

$$\frac{\varepsilon}{d} \underbrace{K \int_{-\infty}^{\infty} \frac{\alpha(x)}{1+\alpha(x)\,\eta(x)\,K}\, p(x,\bar{x})\, dx}_{I_R|_{R=K}} > 1. \qquad (14)$$

Notice that the absolute value of the interaction strength experienced by the resource at its carrying capacity (i.e. $I_R|_{R=K}$) from equation (11) is embedded in (14). We know that $I_R|_{R=K}$ depends on individual variation (σ^2) such that (14) is:

$$\frac{\varepsilon}{d} I_R(\sigma^2)\Big|_{R=K} > 1. \qquad (15)$$

Hence, if phenotypic mismatch is small ($d_\alpha \sim 0$ and $d_\eta \sim 0$), the consumer is less likely to persist since $I_R(\sigma^2)\Big|_{R=K}$ decreases monotonically with individual variation and (16) becomes less likely to hold (Fig. 3a). When phenotypic mismatch is large ($|d_\alpha| >> 0$ or $|d_\eta| >> 0$), the likelihood of consumer persistence gets larger at first and then decreases (Fig. 3b), following the effect of individual variation on interaction strengths (Fig. 2). The larger the phenotypic mismatch, however, the less likely the

persistence criteria will be met, as the interaction strength becomes consistently smaller with variation (Fig. 2).

Increasing phenotypic mismatch decreases consumer persistence regardless of individual variation (Fig. 4a). Increasing levels of variation can counter this effect by rescuing consumers from extinction under some conditions, and by stabilizing consumer-resource interactions (Fig. 4a). For a given level of phenotypic mismatch, an increase in individual variation can be accompanied by a change in persistence; from non-coexistence to coexistence, and a change in dynamics; from limit cycles to oscillatory dynamics to non-oscillatory dynamics (Figs. 4a, 4b, first and second rows). Increasing individual variation not only increases stability, but decreases interaction strengths concomitantly (Fig. 4b, third row). Both phenotypic mismatch and individual variation affect species coexistence through altering resource and consumer isoclines: the consumer isocline shifts to the right while the resource isocline moves up with increasing levels of individual variation (Fig. 4b first row). Nevertheless, extremely large values of individual variation can drive consumers to extinction, as they are no longer able to ingest resource at a high enough rate (Appendix VII, also equation (15)). Although Jensen's inequality predicts opposite effects of variation in attack rate and handling time when considered independently (Figs. 1a, 1b), the effects of individual variation upon the consumer-resource dynamics seem to be mainly driven by variation in the attack rate (Appendix III).

These numerical results are in accordance with our analytic predictions, where the condition for stability can be approximated as:

$$\sigma^2 > \frac{\alpha_{\max}\,\tau\,K\,\eta_{\max}\left(\varepsilon - d\,\eta_{\max}\right)}{\varepsilon + d\,\eta_{\max}} - \tau^2, \qquad (16)$$

whenever variation on attack rates has a larger effect on dynamics than that of handling time, phenotypic mismatch is small ($d_\alpha \sim 0$ and $d_\eta \sim 0$), and variation is small enough (Appendix VIII for the derivation). Here, d stands for the consumer death rate. In this case, the system is stable if individual variation is larger than a certain quantity that increases with the maximal attack rate ($\alpha_{\max}$), the carrying capacity (K) and the digestive efficiency (ε). Notice that (16) is similar to the CV rule of Hassel et al. (1991), where the coefficient of variation needs to be larger than 1 for a spatially variable consumer-resource parasitoid interaction to be stable.

Combined, these results suggest that the effect of variation in attack rates is dominant over that of handling times (Appendix III), which leads to a reduction in interaction strengths (Fig. 2), and an increase in coexistence and stability (Fig. 3), unless variation is too large (Equation (15), Fig. 3).

DISCUSSION

Individual variation in demographic parameters is pervasive in most systems (Bolnick et al. 2003), but only a handful studies have addressed the potential effects of this variation on population dynamics and species persistence (Okuyama 2008) or eco-evolutionary dynamics (Schreiber et al. 2011, Vasseur et al. 2011). Here, we show that non-heritable individual variation may drive ecological consumer-resource interactions through its effect on interaction strengths, as suggested by recent empirical studies (Agashe 2009, Jones & Post 2013). This effect may vary with the environment, and should be different for species with different levels of phenotypic mismatch, ultimately caused by past and existing levels of stabilizing selection. In what follows we propose testable predictions with respect to a possible trade-off between persistence and biological invasiveness mediated by phenotypic variation.

Finally, we show that the effect of individual variation through Jensen's inequality may strongly depend on assumptions regarding the functional form of ecological attributes, which underlines the need for more accurate estimates of trait and ecological attribute distributions using empirical and experimental approaches.

Interaction strengths, selection and whole community effects

Although individual variation can increase species persistence in the eco-evolutionary dynamics of an apparent competition system (Schreiber et al. 2011), the mechanisms through which this happens are unclear. Classical models of consumer-resource interactions suggest that larger interaction strengths destabilize equilibrium densities, and bring species closer to extinction thresholds, potentially leading to species extinction (Rosenzweig & MacArthur 1963). Our results are consistent with these classic studies, and by showing how individual variation can reduce interaction strengths, we provide a mechanistic explanation as to why interacting species with larger levels of variation seem to persist more than those with smaller levels of variation (Newman & Pilson 1997; Imura, Toquenaga & Fuji 2003).

However, our results also suggest that the effect of individual variation on interaction strengths depends on the levels of phenotypic mismatch between consumers and resources, and these are ultimately controlled by existing and past selective pressures (e.g. Fellowes, Kraajiveled & Godfray 1998; Nuismer, Gomulkiewicz & Ridenhour 2010). Small phenotypic mismatch can lead to large interaction strengths when variation is small, and can result from strong stabilizing selection. In contrast, large phenotypic mismatch reduces interaction strengths can result from weak stabilizing selection, a trade-off with another trait, or a recent environmental shift leading to maladaptation. Also, because constant environments can impose strong stabilizing selection and fluctuating environments can impose

weak stabilizing selection (Gavrilets & Hastings 1994, Zhang & Hill 2005), our results suggest that the effect of individual variation may be environment-dependent.

Our results could have important implications for food web theory. For example, interaction strengths have also long been known to drive the stability of large, complex networks of interacting species such as food webs (e.g. May 1972, Allesina & Tang 2012). Because individual variation affects interaction strengths, our results suggest that, to fully understand why complex food webs are stable in nature, we may need to take into account individual variation. For example, weak interaction strengths have been suggested to increase overall stability (McCann, Hastings & Huxel 1998), and we show here that weak interaction strengths occur with high individual variation or phenotypic mismatch. Hence, stable food webs may be characterized by species with high levels of individual variation and small phenotypic mismatch between consumers and resources, or by a mixture of species with low and high levels of individual variation, provided that phenotypic mismatch is large enough among species. Conversely, unstable food webs may be characterized by species with low levels of individual variation and small phenotypic mismatch. Testing some of these ideas in empirical food webs could strongly advance our understanding of how large complex food webs persist in nature despite their structural instability. Unfortunately, this may not be currently feasible.

Individual variation and biological invasions

We showed that variation can affect interaction strengths and species persistence. In what follows we argue that this could have important consequences for the establishment of biological invaders. For small phenotypic mismatch between consumers and resources, interaction strength decreases monotonically with variation (Fig. 2a), which results in an increase in resource persistence but an eventual decrease

in consumer persistence (Fig. 3a). The antagonistic effects of individual variation on persistence and stability suggest that invasive consumers able to invade and persist may have intermediate levels of variation whenever phenotypic mismatch is small (Fig. 3a). This prediction can be tested readily in the field, and is in line with previous empirical findings on invasive weeds (Genton, Shykoff & Giraud 2005). Whenever phenotypic mismatch is large, however, the hump-shaped relationship between variation and interaction strengths (Fig. 2c) may lead to successful invasive consumers with either low or high individual variation, both of which have been reported in the field (Estoup et al. 2001; Kolbe et al. 2004, respectively).

Invasive species can enter a new environment with a single or a few individuals, and could therefore have low individual variation during the establishment phase (Facon et al. 2006). If phenotypic mismatch is small, the interaction strength with native resource species may be high, and their effect upon native diversity may be devastating. Furthermore, failed attempts to eradicate the invasive species may just reduce the individual variation of the invasive species even more, resulting in stronger interaction strengths and deteriorated native species persistence. If phenotypic mismatch is large, however, even with moderately high levels of variation, interaction strengths could be quite low. In this case, eradication attempts could effectively reduce individual variation even more, resulting in weaker interaction strengths and improved species persistence provided that phenotypic mismatch does not change much over time. Finally, our results strengthen previous findings suggesting that the probability of a successful invasion depends on underlying variation (Jones & Gomulkiewicz 2012) and stress the need for taking individual variation into account in order to devise better management policies regarding invasive species.

Jensen's inequality and a plea for empirical estimation of trait variability

Because of Jensen's inequality it has been previously suggested that attack rates and handling times could have opposite effects on interaction strengths when individual trait variation was taken into account in each attribute independently (Figs. 1a, 1b this paper, Bolnick et al. 2011). Although variation in the traits controlling the attack rate seems to have more profound effects upon ecological dynamics than in those controlling the handling time, our findings also suggest that these predictions are contingent on the specific functional forms through which attack rate and handling time depend on underlying phenotypic trait variation. Hence, our results emphasize the need for gathering estimates about how ecologically relevant traits distribute in real populations, and assessing the functional form of their effect upon ecological attributes.

One possible way of doing so is to use controlled microcosm experiments of consumer and resource protists (e.g. DeLong & Vasseur 2013), where attack rates and handling times could be directly measured while underlying phenotypic variation is manipulated. These systems are particularly well suited for quantifying entire trait distributions (DeLong 2012) and are thus prime candidates to test some of our ideas. However, previous mesocosm studies assessed the effect of variation in defense traits in algal populations, showing that variation in defense mechanisms could alter biological dynamics (Yoshida, Hairston & Ellner 2004). Hence, while very difficult, it is not impossible to gather some of this information in fairly complex empirical systems.

Conclusions

Our results are in accordance with previous theoretical studies that have shown that increased behavioral variation (Okuyama 2008) and variation in the use of

space by parasitoids in heterogeneous landscapes (Hassell et al. 1991), are mostly stabilizing. Moreover, we derived conditions for stability that are qualitatively similar to those derived by Hassel and collaborators, which together suggest that there is a minimal threshold of variation below which ecological dynamics become highly unstable. We also note that spatial or environmental heterogeneity, as considered in the work by Hassel et al. (1991), can induce differences in space use among individuals. This variation in space use ought to be regarded as a type of individual phenotypic variation and we thus argue that these converging results may be due to variation decreasing interaction strengths through Jensen's inequality.

Other researchers have explored consumer-resource dynamics in the case where there is behavioral variation in foraging rates (Okuyama 2008), however, our approach differs from theirs in several important ways: first, we explicitly modeled variation in underlying quantitative phenotypic traits controlling attack rates and handling times, only making assumptions grounded on biological data; second, we accounted for the potential effects of phenotypic mismatch, or the difference between mean trait in the population and the adaptive peak; and last, we have drawn a mechanistic link between individual variation and population dynamics by exploring its effect on interaction strengths, which are the ultimate link to connect pairwise models to whole food web dynamics and stability (e.g. May 1972; Allesina & Tang 2012).

Overall our study shows that individual variation can affect species persistence and coexistence between consumer and resource through its effect on interaction strengths. Moreover, the effect of individual variation on interaction strengths depends on phenotypic mismatch and thus, on current and past selective pressures. This has important implications for species persistence embedded in food

webs or the arrival of invasive species to native ecosystems. Finally, this study underlines the need for accurately estimating the distribution of ecologically relevant phenotypic traits, as well as their functional relationship with ecological attributes, in order to test our predictions of how individual variation affects the ecology and persistence of interacting populations.

ACKNOWLEDGEMENTS

We are very grateful to John DeLong, Nelson Hairston Jr., Marie-Claire Chelini and two anonymous reviewers for providing insightful comments on early versions of this manuscript. JPG is specially indebt with John DeLong for his support and advise. JPG was supported by the School of Biological Sciences Special Funds and an Othmer Fellowship. CEB was supported by a National Science Foundation grant (DEB 0953766).

FIGURES

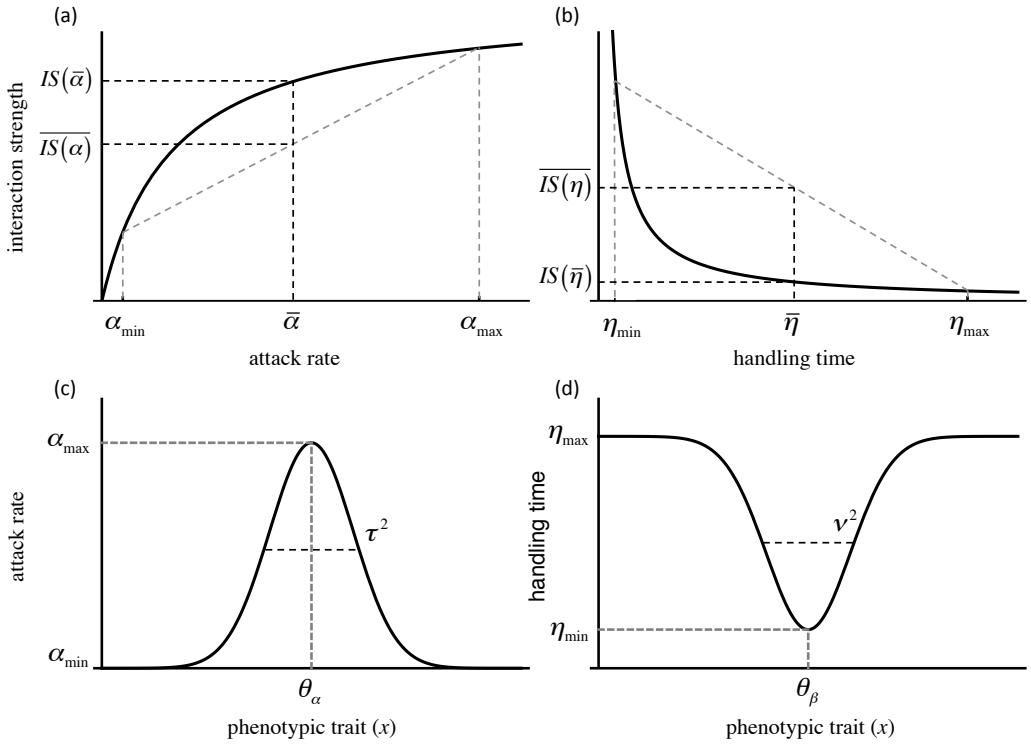

Fig 1. (a), (b); Plots of the magnitude of the interaction strength against attack rate and handling time. Grey dashed curves represent mean interaction strength, not considering individual variation in attack rates or handling times. Solid curves represent interaction strengths considering variation in mean attack rate and handling times. If variation in attack rate only is considered, mean interaction strengths (dashed) are expected to be smaller than actual interaction strengths. If variation in handling time only is considered, mean interaction strengths (dashed) are expected to be greater actual interaction strengths. (c), (d); Plots of attack and handling time against a given quantitative phenotypic trait, where θ_α and θ_η are the optimal trait values for attack rate and handling time respectively.

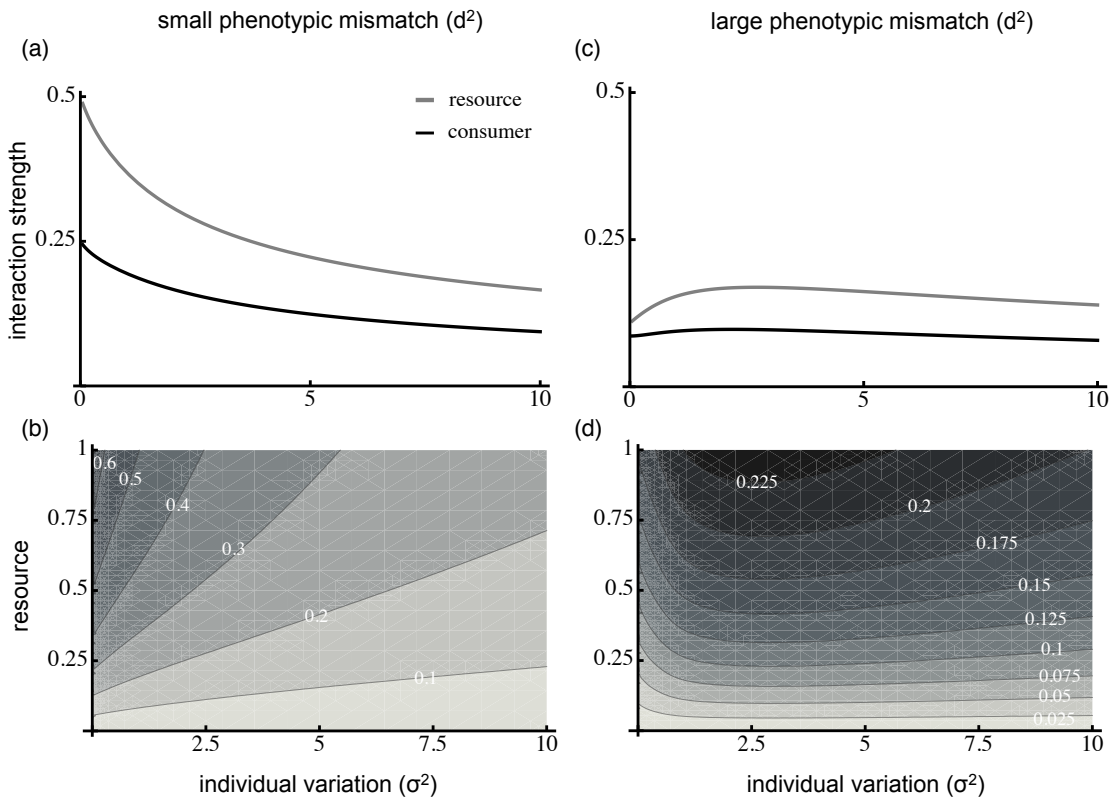

Fig 2. (a), (d); Plots of interaction strength against individual variation (σ^2) for consumers (black) and resources (grey). (b), (d); Contour plots of the interaction strength for varying resource levels against increasing individual variation (σ^2). Small phenotypic mismatch: left column. Large phenotypic mismatch: right column. Parameter values: (a) $\alpha_{\max} = 1$, $\eta_{\max} = 2$, $\eta_{\min} = 1$, $\varepsilon = 0.5$, $\tau = 1$, $\nu = 1$, $d_\alpha = 0$, $d_\eta = 0$, R=1; (b) same as in (a) but R varies from 0 to 1; (c) same as in (a) but for $d_\alpha = 2$, $d_\eta = 2$; (d) same as in (c) but R varies from 0 to 1.

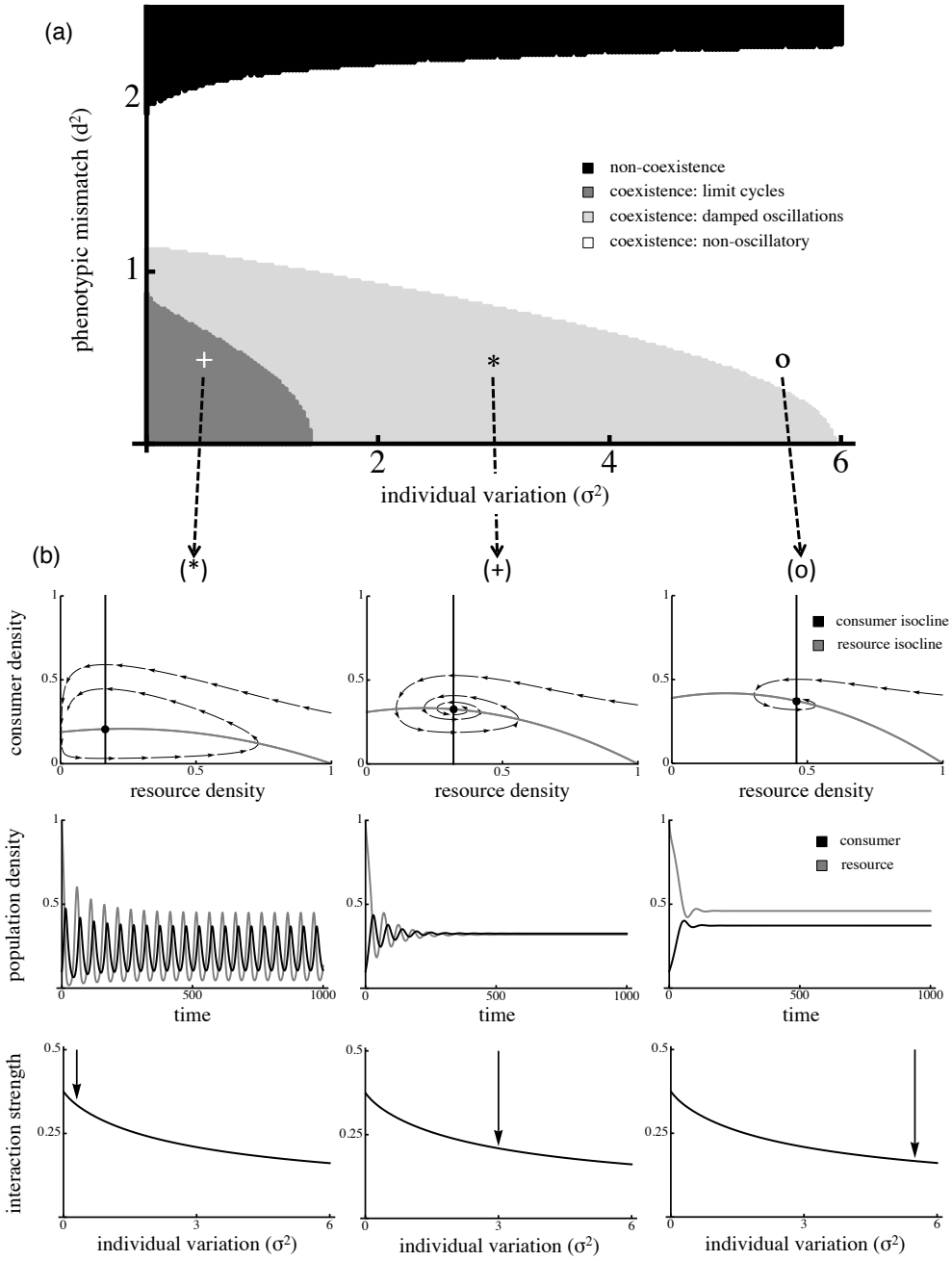

Fig 3. (a) Outcome of the interaction plotted against individual variation and phenotypic mismatch. Consumers can go extinct but the resource survives (black), or both species can coexist (limit cycles in dark grey, damped oscillations light grey, non-oscillatory behavior in white). The asterisk, the cross and the zero represent combinations of parameters we use as an example of how coexistence,

stability and interaction strengths change with variation. (b) First row: phase diagrams where the equilibrium occurs at the intersection the two isoclines (black: consumers, grey: resource, black dot: equilibrium). Arrows represent one possible trajectory of the system. Second row: dynamics for consumers (black) and resources (grey) through time. Third row: mean interaction strength in the system for both interacting species against individual variation. Parameter values: (a) r = 0.3, $\alpha_{\max}$ = 2, $\eta_{\max}$ = 2, $\eta_{\min}$ = 1, ε = 0.5, τ =1, ν = 1, K=1, β = 0.1, $d_\alpha = d_\eta$ = 0.5 and σ^2 =0.3 (asterisk); $d_\alpha = d_\eta$ = 0.5 and σ^2 = 3 (cross) and $d_\alpha = d_\eta$ = 0.5 and σ^2 = 5.5 (zero).

CHAPTER 2

INDIVIDUAL VARIATION DECREASES INTERFERENCE COMPETITION BUT INCREASES SPECIES PERSISTENCE

Jean P. Gibert & John P. DeLong

Key-words: Interference competition, Individual variation, Intraspecific variation, Species persistence, Stability, Competition.

ABSTRACT

Interference competition is thought to stabilize consumer-resource systems. The magnitude of interference is linked to that of attack efficiency: when both levels are intermediate, populations are maximally stable and have high competitive ability. Individual variation can affect ecological dynamics through its effect on attack efficiency and handling time. Because interference has a non-linear effect on consumer foraging rates, individual variation in mutual interference can strongly affect ecological dynamics. Here, we explicitly incorporate individual variation in attack efficiency, handling time and interference into a dynamic consumer-resource model and show that variation increases species coexistence by depressing attack efficiency to a greater extent than predator interference. We argue that this differential effect of variation affects the equilibrium densities of consumers and their prey, thus altering their competitive ability. Intermediate levels of variation can maximize both consumer persistence and competitive ability. Our results show the importance of

quantifying individual variation in natural populations for understanding the persistence and stability of species within communities.

INTRODUCTION

A major goal of ecology is to understand the factors underpinning species coexistence and stability in complex ecosystems (May 1972, 1973; McCann, Hastings & Huxel 1998; Allesina & Tang 2012). Seminal work by Tilman showed that when two competing species share a common resource, the one that can reduce resource density the most will outcompete the other (Tilman 1982, 1986). However, the ability to reduce resource density and persist may depend upon the factors controlling interaction strengths and consumer-resource interactions. A number of these factors have received a lot of attention, including foraging behavior (Schmitz, Beckerman & O'Brien 1997; Abrams & Matsuda 2004), consumer and resource body sizes (Vucic-Pestic *et al.* 2010) and relative velocities (Pawar, Dell & Savage 2012; DeLong 2014), prey defense mechanisms (Yoshida, Hairston Jr & Ellner 2004; Hammill, Petchey & Anholt 2010), and environmental temperature (O'Connor 2009; Gibert & DeLong 2014; Dell, Pawar & Savage 2014). And while all these factors are important, the underlying assumption in ecology has historically been that populations are homogeneous collections of individuals and that mean trait values are sufficient for understanding ecological processes (Lomnicki 1988). Unfortunately, whenever non-linear relationships between underlying traits and ecological processes of interest occur, using mean trait values can be misleading (Inouye 2005; Bolnick *et al.* 2011). Because non-linearities are common in consumer-resource interactions, overlooking individual phenotypic variation may impair our capacity to fully understand species persistence and competitive ability in natural communities.

Populations often show individual-level phenotypic variation in anti-predator defenses (Duffy 2010), competitive ability (Lankau & Strauss 2007), or resource utilization (e.g. (Bolnick *et al.* 2003; Estes *et al.* 2003)). Because interspecific interactions ultimately occur between individuals, individual phenotypic variation can affect interspecific interactions in multiple ways (Pettorelli *et al.* 2011). For instance, individual-level dietary specialization among southern sea otters (*Enhydra lutris nereis*) induces changes in the structure of the population-level resource utilization network, which in turn can alter the structure and dynamics of the food webs in which these organisms are embedded (Tinker *et al.* 2012). Individual variation also can affect the strength of consumer-resource interactions by changing the parameters of the functional response connecting species pairs ((Bolnick *et al.* 2011; Schreiber, Bürger & Bolnick 2011; Gibert & Brassil 2014), also see (Doebeli 1996) in an evolutionary context). In particular, increasing individual variation in attack efficiency (or attack rate) and handling time decreases interaction strengths, which in turn increases species persistence and stability (Gibert & Brassil 2014). Together, these results underscore the need to understand how individual level phenotypic variation affects ecological processes and, through that, the structure and dynamics of entire communities.

Interaction strengths can be influenced by 'mutual' interference competition among predators by dampening resource uptake at higher consumer densities (Arditi *et al.* 2004). Therefore, mutual interference is thought to stabilize the dynamics of consumer-resource interactions (Arditi *et al.* 2004; Forrester *et al.* 2006; DeLong & Vasseur 2011, 2013). Interference is often thought to occur through behavioral mechanisms associated with territoriality and aggressiveness (Connell 1961; Kennedy & White 1996; Forrester *et al.* 2006), but more generally, interference competition is

any form of interaction among consumers that inhibits foraging. Because interference is widespread among many different taxa, it may play an important role in stabilizing natural communities (Skalski & Gillooly 2001; DeLong & Vasseur 2011, 2013).

The parameters of the functional response, including mutual interference, are driven by organism traits, and these traits may influence more than one parameter at a time. For example, body size influences both attack efficiency and handling time in several taxa (DeLong & Vasseur 2012a). Also, variation in different parameters can have opposite effects on foraging rates (Bolnick *et al.* 2011), so it may be important to link variation in underlying controlling traits to multiple parameters simultaneously (Pettorelli *et al.* 2011; Gibert & Brassil 2014). Recently, a positive trait-based link between attack efficiency and mutual interference was discovered for predatory protists, where predator velocity was thought to increase the magnitude of attack efficiency and interference competition simultaneously (DeLong & Vasseur 2013). Thus, while increasing individual variation can increase stability by lowering interaction strengths through attack efficiency, individual variation might also lower interference competition, potentially undermining the overall stabilizing effect. Because of this, the challenge now is to understand how individual variation in both mutual interference and attack efficiency influences the fate of interacting populations among natural communities.

Our goal is to extend recent work about how individual variation alters consumer-resource dynamics by studying its impact upon linked ecological attributes such as attack efficiency, handling time and interference competition. Schreiber et al. (2011) explored the effect of individual-level heritable variation in attack efficiencies in eco-evolutionary dynamics, while Gibert and Brassil (2014) explored the simultaneous effect of non-heritable variation in the attack efficiency and the handling

time of a consumer-resource system. Here we incorporate non-heritable individual variation in mutual interference by taking into account its functional relationship with attack efficiency (DeLong & Vasseur 2013) and then we assess its effect upon the persistence and competitive ability of consumer-resource systems.

METHODS

The general model

To include mutual interference among consumers, we used a Rosenzweig-MacArthur consumer-resource model with a Hassell-Varley functional response (Rosenzweig & MacArthur 1963; Hassell & Varley 1969). The Hassell-Varley functional response introduces interference competition as a negative exponent, *m*, on the consumer density added to both numerator and denominator of the functional response (e.g. (Hassell & Varley 1969; Arditi & Akçakaya 1990; DeLong & Vasseur 2011):

$$\begin{aligned} \frac{dR}{dt} &= rR\left(1-\frac{R}{K}\right) - C\frac{\alpha RC^m}{1+\alpha\eta RC^m} \\ \frac{dC}{dt} &= \varepsilon\, C\frac{\alpha RC^m}{1+\alpha\eta RC^m} - \beta C \end{aligned}, \qquad (1)$$

where r is the maximal growth rate of the prey, K its carrying capacity, ε is the conversion efficiency, β is the mortality rate of the consumer, α its attack efficiency, η its handling time, and m is the parameter that represents interference competition.

If $m = 0$, the model reduces to the classic Rosenzweig-MacArthur formulation, and if $m = -1$, it reduces to the ratio-dependent formulation (e.g. (Arditi & Ginzburg 1989)). The level of interference, *m*, varies continuously in nature from 0 to -2.5, although it frequently takes intermediate values (Abrams & Ginzburg 2000; DeLong & Vasseur 2011, 2013). In the case of the predatory protist *Didinium*

nasutum preying upon *Paramecium aurelia*, the magnitude of *m* is linked to that of attack efficiency (α) by:

$$m = -0.26 \ln(\alpha) - 0.67, \quad (2)$$

which was determined by estimating the functional response of the consumer across replicate populations (DeLong & Vasseur 2013)(Fig 1-A). This relationship will later be used to introduce individual variation in interference.

Individual variation

Following previous theoretical studies we incorporated individual variation by assuming that both attack efficiency and handling time depend on the value of a normally distributed trait (Schreiber *et al.* 2011; Rall *et al.* 2012; Gibert & Brassil 2014), x, with mean $\bar{x}$, variance σ^2, and probability density:

$$p(x,\bar{x}) = \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left[-\frac{(x-\bar{x})^2}{2\sigma^2}\right]. \quad (3)$$

We assumed that the consumer's attack efficiency $\alpha(x)$ is maximal at a given optimal trait value $x = \theta_\alpha$, and decreases away from that maximum as:

$$\alpha(x) = \alpha_{\max} \exp\left[-\frac{(x-\theta_\alpha)^2}{2\tau^2}\right], \quad (4)$$

where $\alpha_{\max}$ is the maximal attack efficiency and τ^2 determines how steeply the attack efficiency declines away from θ_α (Fig. 1-B). The handling time, $\eta(x)$, was assumed to be minimal at a given optimal value $x = \theta_\eta$, and to increase away from that minimum as:

$$\eta(x) = \eta_{\max} - (\eta_{\max} - \eta_{\min}) \exp\left[-\frac{(x-\theta_\eta)^2}{2\nu^2}\right], \quad (5)$$

where $\eta_{\max}$ and $\eta_{\min}$ are the maximal and minimal handling time respectively, and ν^2 determines how steeply the handling time increases away from θ_η (Fig. 1-C).

We defined the quantities $d_\alpha^2 = \left(\bar{x} - \theta_\alpha\right)^2$ and $d_\eta^2 = \left(\bar{x} - \theta_\eta\right)^2$, as the distance between the mean trait in the population and the optimal value at which attack efficiency is maximal and handling time is minimal (referred to as phenotypic mismatch; see (Schreiber *et al.* 2011) and (Raimundo *et al.* 2014) for similar definitions). Because the optimal value is set by past and existing selective pressures (Anderson, Terblanche & Ellis 2010), the phenotypic mismatch can be seen as a measure of how well adapted the consumer species is at attacking and handling a particular resource (Gibert & Brassil 2014). The larger the mismatch is, the smaller the attack rate and the larger the handling time.

We explored three scenarios. We first recapitulated some of the results of Gibert and Brassil (2014) as a baseline for comparison, by including variation in attack efficiency and handling time only. Second, we included only variation in mutual interference, and, third, we included individual variation in all three parameters simultaneously. For the first scenario (variation in attack efficiency and handling time), the consumer-resource model is:

$$\begin{aligned} \frac{dR}{dt} &= rR\left(1 - \frac{R}{K}\right) - C\int_{-\infty}^{+\infty} \frac{\alpha(x) R C^m}{1 + \alpha(x)\,\eta(x) R C^m} p(x, \bar{x})\, dx \\ \frac{dC}{dt} &= \varepsilon C \int_{-\infty}^{+\infty} \frac{\alpha(x) R C^m}{1 + \alpha(x)\,\eta(x) R C^m} p(x, \bar{x})\, dx - \beta C \end{aligned}, \qquad (8)$$

where m is constant. For the second scenario (variation in interference only), the model is:

$$\begin{aligned}\frac{dR}{dt} &= rR\left(1-\frac{R}{K}\right)-C\int_{-\infty}^{+\infty}\frac{\alpha RC^{m(\alpha(x))}}{1+\alpha\eta RC^{m(\alpha(x))}}p(x,\bar{x})\,dx \\ \frac{dC}{dt} &= \varepsilon C\int_{-\infty}^{+\infty}\frac{\alpha RC^{m(\alpha(x))}}{1+\alpha\eta RC^{m(\alpha(x))}}p(x,\bar{x})\,dx-\beta C\end{aligned}, \quad (9)$$

where $m(\alpha(x)) = -0.26\ln(\alpha(x)) - 0.67$ and all other parameters are as in (1). Notice that in this model, α is only allowed to change with variation in the underlying trait x inside of function $m(\alpha(x))$, but not outside of it. Because this is not realistic, we only do it as a way of understanding variation in mutual interference alone while acknowledging that variation ought to be considered in multiple parameters at the same time. This leads to the third scenario, where variation is now being considered in all three parameters simultaneously (variation in attack efficiency, handling time and interference):

$$\begin{aligned}\frac{dR}{dt} &= rR\left(1-\frac{R}{K}\right)-C\int_{-\infty}^{+\infty}\frac{\alpha(x)RC^{m(\alpha(x))}}{1+\alpha(x)\,\eta(x)\,RC^{m(\alpha(x))}}p(x,\bar{x})\,dx \\ \frac{dC}{dt} &= \varepsilon C\int_{-\infty}^{+\infty}\frac{\alpha(x)RC^{m(\alpha(x))}}{1+\alpha(x)\,\eta(x)\,RC^{m(\alpha(x))}}p(x,\bar{x})\,dx-\beta C\end{aligned}. \quad (10)$$

We analyzed these three scenarios for varying levels of phenotypic mismatch using intermediate values for the maximal attack efficiency and mutual interference, as this combination of parameters is thought to be the most likely in nature (DeLong & Vasseur 2013). Considering different combination of parameters does not qualitatively affect our results.

The objective of our simulations was to assess the effect of individual variation on the equilibrium of the system (i.e. the intersection of the consumer and resource isoclines). Because of the way we incorporated individual variation in equations (8) to (10), solving for these isoclines (the conditions at which dR/dt=0 for the prey isocline, and the conditions at which dC/dt=0 for the predator isocline) and

their intersection is now impossible analytically, so it was done numerically. The farther away this equilibrium is from the axes, the less likely consumers and resources are to go extinct due to random fluctuations. Finally, to assess the effect of variation upon community structure, we investigated its effect upon the persistence of consumers through their equilibrium density, C^*, as well as their competitive ability, through the equilibrium density of the resource, R^*. Low equilibrium resource densities (R^*) are associated with strong competitive ability of the consumers (Tilman 1982, 1986). We therefore define the quantity $1/R^*$ as a measure of competitive ability: the larger the quantity, the larger the competitive ability of the consumer and vice-versa.

RESULTS

Overall, individual variation can have a strong effect on equilibrium densities and species persistence when interference competition is considered. The effect of individual variation on interference competition depends on the levels of phenotypic mismatch in the trait that controls the consumer-resource interaction (Fig. 2). This effect seems to be mediated mainly by the interplay between attack efficiency and interference competition and ultimately affects the equilibrium densities of the interacting pair, resulting in differential persistence and competitive ability for the consumer at different levels of individual variation (Fig. 3).

Low phenotypic mismatch

When phenotypic mismatch is low ($d_\alpha \sim 0$ and $d_\eta \sim 0$), individual variation in attack efficiency and handling time increases equilibrium densities of both the consumer and the resource, moving them away from extinction thresholds (Fig. 2-A).

By doing so, individual variation potentially increases species persistence, as extinction due to demographic stochasticity is less likely to occur.

When it occurs only in interference, individual variation reduces the equilibrium density of the consumer but increases that of the resource (Fig. 2-B). This makes consumers simultaneously less competitive due to a high R^* and more prone to extinction due to a low C^*. The change in equilibrium abundance for a given change in individual variation, however, is less pronounced than that observed when variation in both attack efficiency and handling time is considered (Fig. 2-A, B).

The net effect of individual variation in interference competition, attack efficiency, and handling time combined is intermediate to the effect produced when individual variation is included only in interference competition or in both the attack efficiency and the handling time. This is because the effects are opposite of each other. Individual variation increases the equilibrium density of consumers and resources, moving them away from the extinction threshold (Fig. 2-C), which is qualitatively different from what happened when variation only in interference was included (Fig. 2-B). However, this effect is also less pronounced than in a scenario with variation only in attack efficiency and handling time (notice the magnitude of the change in Fig. 2-A) and more similar in magnitude to a scenario with variation only in interference (notice the magnitude of the change in Fig. 2-B).

Large phenotypic mismatch

When phenotypic mismatch is large ($|d_\alpha| >> 0$) in a scenario with variation in both attack efficiency and handling time, low levels of individual variation decrease equilibrium densities, but high levels of variation increase equilibrium densities for both consumers and prey (Fig. 2-D). These changes in equilibrium densities occur

much farther away from extinction thresholds than in a scenario with small phenotypic mismatch (Fig. 1-A), but are of larger magnitude.

Individual variation in interference decreases the equilibrium density of the resource at first, but it then increases as variation gets larger (Fig. 2-E). This practically has no effect on consumer equilibrium densities and the overall effect of variation is comparatively small in magnitude.

The net effect of individual variation in interference competition, attack efficiency, and handling time is, again, intermediate to the effect produced in the previous scenarios. Indeed, the densities for both resource and consumers behave as if only variation in attack efficiency and handling time was considered (Fig. 2-F, D), but these fluctuations are of a much smaller magnitude, as in a scenario with only variation in interference (Fig. 2-F, B).

Interference, attack efficiency consumer persistence and competitive ability

Individual variation has the same overall effect on interference competition as it has on attack efficiency (Fig. 3). If phenotypic mismatch is low, both attack efficiency and interference competition decrease with individual variation, but the effect seems to be more pronounced on attack efficiency than on interference (Fig. 3-A). When phenotypic mismatch is large, however, both attack efficiency and interference increase with variation at first, and then decrease (Fig. 3-A). The magnitude of this effect is comparable for both parameters.

Because variation on attack efficiency and interference alters the equilibrium densities of both consumers and resources (Fig. 2), it will ultimately affect consumer persistence as well as its overall competitive ability. For the full model (equation 10), when phenotypic mismatch is low, the consumer equilibrium density, C^*, increases with variation but its competitive ability, measured as $1/R^*$, decreases. Because

variation maximizes C* and 1/R* simultaneously whenever the absolute value of that difference is small, our results suggest that intermediate levels of variation maximize the consumer's ability to persist (C^*) and to compete ($1/R^*$) (Fig. 3-B). When phenotypic mismatch is large, the consumer equilibrium density decreases with individual variation at first and then increases slowly. Consumer competitive ability, however, increases with variation and then decreases. Interestingly, intermediate levels of variation maximize the consumer's ability to persist and to compete (Fig 3-C), despite the larger phenotypic mismatch.

DISCUSSION

Variation and Interference

Individual variation in traits controlling ecological attributes such as attack efficiency and handling time can increase species persistence in consumer-resource interactions (Bolnick *et al.* 2011; Gibert & Brassil 2014). In classic consumer-resource models, an increase in the attack efficiency increases interaction strengths, resulting in a decrease of species persistence and overall stability (e.g. (Rosenzweig & MacArthur 1963)). Individual variation weakens interaction strengths by decreasing attack efficiencies, which in turn increases species persistence and stability (Gibert & Brassil 2014). Our results suggest that this effect also occurs when consumer interference is considered. Interference is generally stabilizing (Ginzburg & Jensen 2008), so it might be expected that individual variation in interference alone could have destabilizing effects, potentially leading to species extinctions. However, it seems to either decrease consumer equilibrium densities and increase resource equilibrium densities, or have negligible effects on both. When we consider variation in attack efficiency and link that to mutual interference through their empirically-

determined negative relationship (Fig 1-A), the net effect of variation is to increase species persistence. This may be due to a larger effect of individual variation in attack efficiency than in interference that would overcome the negative effect of variation in interference only. These results highlight the importance of considering possible functional relationships between dynamic parameters such as attack efficiency and interference as well as the importance of considering individual variation in the traits controlling these parameters in order to fully understand population dynamics and stability (Yodzis & Innes 1992; DeLong & Vasseur 2012b, 2013).

Variation and competitive ability

Our results also have important consequences for understanding community assembly. If phenotypic mismatch is low, the equilibrium resource density increases with individual variation, which decreases consumer competitive ability. If phenotypic mismatch is large, some variation can reduce resource density at first, momentarily increasing competitive ability. However, large phenotypic mismatch generally decrease competitive ability regardless of variation, meaning that poorly adapted species are in general poor competitors and populations that are already well adapted to their niche become less competitive when they become more variable. In the case of the *Didinium-Paramecium* system, after which our model is parameterized, if interference is too large, consumer uptake is heavily impaired, resulting in deterministic extinction (DeLong & Vasseur 2013). If interference is low, however, equilibrium densities increase up to a point where the competitive ability of the populations is reduced (Tilman 1982, 1986; DeLong & Vasseur 2013). A similar rule might apply to individual variation when it affects both attack efficiency and interference. If variation is too small, populations are close to their extinction threshold. If variation is too large, their equilibrium densities increase to a point

where it may impair their competitive abilities, threatening their persistence in the community. Variation in interference thus seem to counter the effect of variation in attack efficiency, naturally leading to the existence of an intermediate amount of variation that both minimizes the chance of extinction and maximizes the competitive ability of populations in a community.

Eco-Evolutionary feedbacks

Individual variation can be important for ecological dynamics, but it also is the raw material upon which natural selection acts (Dobzhansky 1937). In addition, evolutionary processes have been increasingly recognized to occur at ecological timescales, altering ecological processes and dynamics as they unfold (Thompson 1998; Grant & Grant 2002; Hairston Jr *et al.* 2005). The interplay between ecological and evolutionary processes, or eco-evolutionary feedbacks, thus needs to be considered in future work. In this sense, individual variation has been recognized to increase species coexistence in eco-evolutionary dynamics (Schreiber *et al.* 2011; Vasseur *et al.* 2011), but variation has been assumed to be constant through time. However, phenotypic variation generally scales with mean trait values, a pattern known as Taylor's power law (Taylor 1961) and prevalent across systems and taxa (DeLong 2012). Thus, individual variation in a given trait will track the evolution of the mean trait value, potentially leading to changes in community structure due to alterations in competitive ability that are a consequence of changes in individual variation that track the evolution of underlying traits. This makes it paramount to also track the evolution of variation over time to understand eco-evolutionary and the stability and persistence of ecological systems in nature.

The effect of individual variation may also depend on the strength of selection acting on the traits that control the consumer-resource interaction (Yoshida *et al.*

2003; Gibert & Brassil 2014). Strong stabilizing selection may reduce individual variation through time, with consequences for population stability and competitive ability. Unstable and uncompetitive populations will not fare well in communities, which implies that selection that reduces variation and increases mean fitness within populations may have negative effects for the population in a community. Populations may thus be the subject of antagonistic effects of natural selection (Raimundo *et al.* 2014). Together, this suggests that the interplay between ecological and evolutionary processes is central to understanding how communities are structured in nature (Thompson 2005; Bolnick *et al.* 2011; Guimarães, Jordano & Thompson 2011; Fontaine *et al.* 2011). Individual variation may be the key to bridging the gap between ecology and evolution.

Underlying controlling traits

Considering what traits operate as 'controlling' traits that influence parameters such as attack efficiency, handling time, and interference is also important. For instance, the amount of variation in the controlling trait is linked to mutation rates, the amount of phenotypic plasticity and the strength of selective forces operating on the trait. Thus, by identifying probable controlling traits, we may have a deeper grasp of the processes controlling the variation ultimately affecting consumer-resource dynamics. It is possible that some traits, such as body size, might act as ecological "magic traits". In an evolutionary context, "magic traits" traits are involved in both mating and ecological activities, and when they experience disruptive selection they can lead to adaptive speciation (Gavrilets 2004; Raimundo *et al.* 2014). Ecological "magic traits" would be traits influencing many dynamic parameters at once (e.g. (DeLong & Vasseur 2012b)), while other traits only influence a limited set of parameters, if any. Specific links between traits, their optima, and dynamic population

parameters are needed to fully understand how individual variation influences consumer-resource dynamics. Identifying such traits and quantifying their distribution and their effect upon ecological processes of interest should be a major goal of ecology in the upcoming future (Pettorelli *et al.* 2011; Violle *et al.* 2012a; b; Gibert & Brassil 2014).

Testable predictions from the theory of individual variation

To help moving toward that goal, we can make some simple testable predictions as to how individual variation can affect interaction strengths in a system with interference competition. If the per-capita foraging rate of a consumer preying upon a resource is:

$$f(R,C) = \frac{\alpha R C^m}{1 + \alpha \eta R C^m}, \tag{11}$$

then, we can find an expression for the average foraging rate that explicitly depends upon individual variation by integrating over the functional response and the underlying trait distribution. We thus get:

$$\overline{f(R,C)} = \int_{-\infty}^{+\infty} \frac{\alpha(x) R C^m}{1 + \alpha(x)\, \eta(x) R C^m} p(x,\bar{x})\, dx, \tag{12}$$

where *m* can be a function of the attack efficiency or a constant. In this case, we can see that while under some conditions increasing individual variation reduces foraging rates and thus, interaction strengths, this effect increases with resource density (Fig. 4-A) and decreases with consumer density (Fig 4-B, C). These predictions can be tested in foraging experiments where the resource and consumer densities are manipulated in the same way it would be done for quantifying the parameter of mutual interference, *m* (DeLong & Vasseur 2013). A measure of individual variation across treatments and one or several traits such as body size, would need to be quantified as well. The latter is particularly doable in microcosms with protists (DeLong 2012;

DeLong & Vasseur 2013)or mesocosm experiments with metazoan grazers and algae (Fussmann, Ellner & Hairston Jr 2003; Yoshida *et al.* 2003, 2004).

Conclusion

Because of their effect on population persistence and stability, understanding the interplay between individual variation and interference competition is central in ecology. Using dynamic models that explicitly take into account individual variation, we have shown that increasing individual variation simultaneously affecting attack efficiency, handling time and mutual interference can increase species persistence and stability as well as consumer competitive ability. Moreover, as variation is affected by selection, we argue that evolutionary processes may deeply affect the way communities are structured. Finally, our results underscore the need for comprehensive studies that quantify the level of individual variation in natural populations, making specific testable hypotheses as to how individual variation can interact with resource and consumer densities to alter foraging rates and through that, interaction strengths.

ACKNOWLEDGMENTS

JPG was supported by the School of Biological Sciences Special Funds and an Othmer Fellowship.

FIGURES

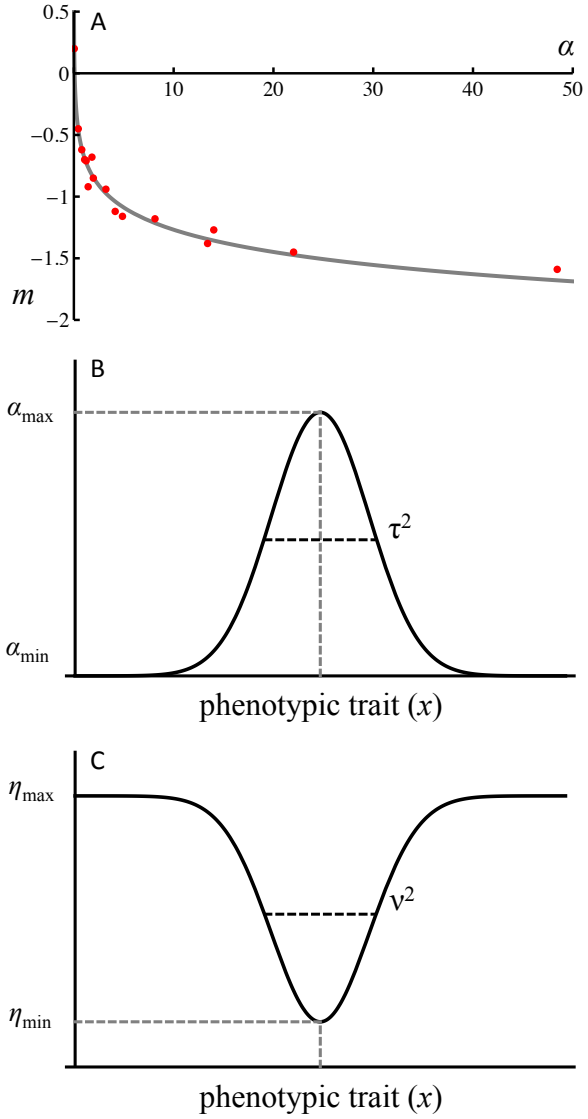

Figure 1: A. Plot of the empirically observed relation between the coefficient of mutual interference (*m*) and the attack efficiency (α) in replicate populations of *Didinium nasutum* preying upon *Paramecium aurelia* (modified from DeLong and Vasseur 2013). As attack efficiency increases, mutual interference becomes stronger. B. Plot of the assumed relation between the attack efficiency (α) and the underlying phenotypic trait (x). C. Plot of the assumed relation between the handling time (η) and the underlying phenotypic trait (A and B are modified from Gibert & Brassil 2014).

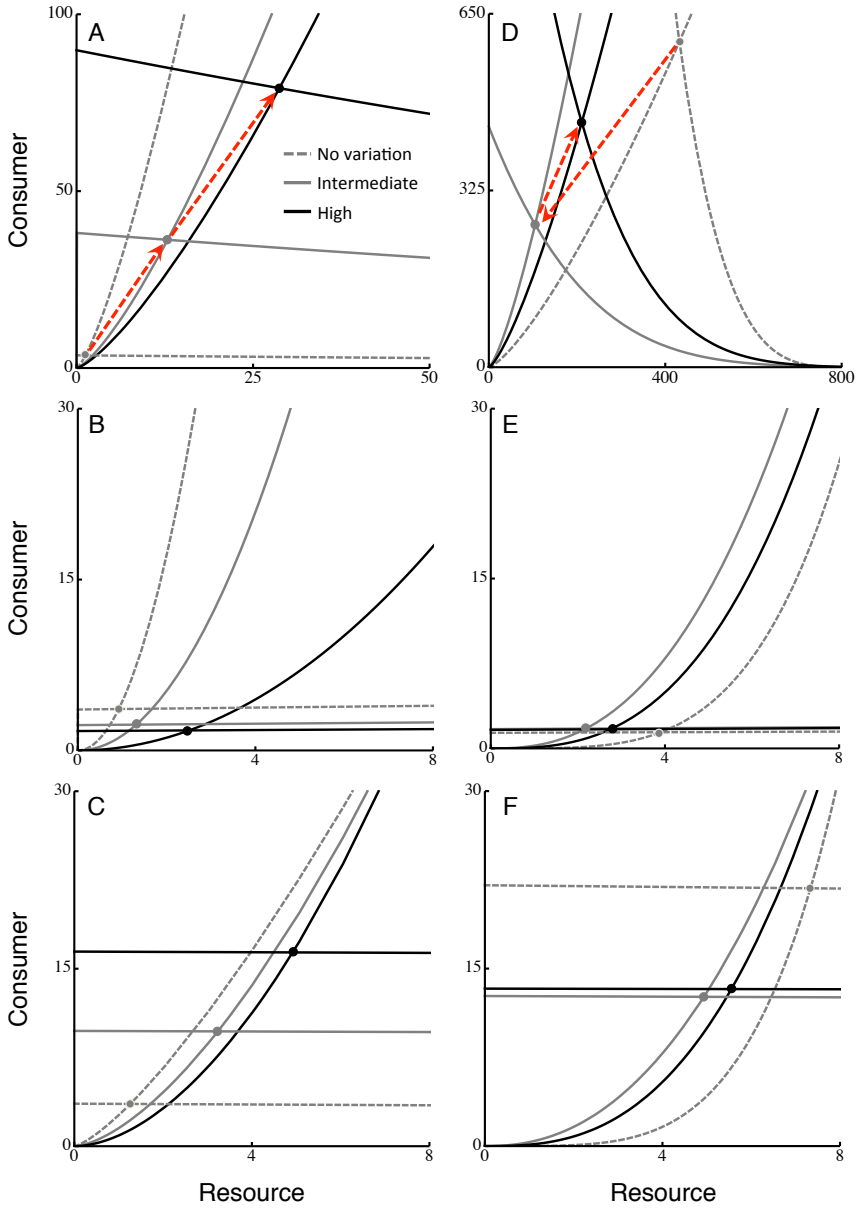

Figure 2: Phase-plane plots of consumer and resource isoclines for different levels of individual variation where the isoclines (values at which a species does not grow or decline) for consumers and resources are represented for different levels of individual variation. The intersection of the isoclines marks the equilibrium densities. Panels in the left column refer to cases with low phenotypic mismatch, and panels in the right column to cases with large phenotypic mismatch. For the panels in the top row individual variation was only considered in attack efficiency and handling time. In the

second row, individual variation in interference competition only was considered. In the third row, individual variation in attack efficiency, handling time, and interference is included. Variation in attack efficiency and handling time increases equilibrium densities (intersection moves away from axes) whenever mismatch is small, and decreases then increases densities whenever mismatch is large. Variation in mutual interference results in a small effect. The latter explains why variation in attack efficiency, handling time and interference results in a tempered version of the first case. Parameter values kept constant across all plots: $\alpha_{max} = 1.38$, $\eta_{max} = 0.08$, $\eta_{min} = 0$, $e = 0.15$, $r = 1.9$, $K = 841$, $\beta = 0.1$, $\tau = 1$, $\nu = 1$, $d_\eta = 0$. Parameters that changed: A. $d_\alpha = 0$, $\sigma^2 = 0$ (grey, dashed), $\sigma^2 = 2.26$ (grey), $\sigma^2 = 14.19$ (black); D. $d_\alpha = 2$, $\sigma^2 = 0$ (grey, dashed), $\sigma^2 = 1.31509$ (grey), $\sigma^2 = 16.7242$ (black); C., D., E., and F. as in A. but for $d_\alpha = 0$ for C. and E. and $d_\alpha = 2$ for E. and F.

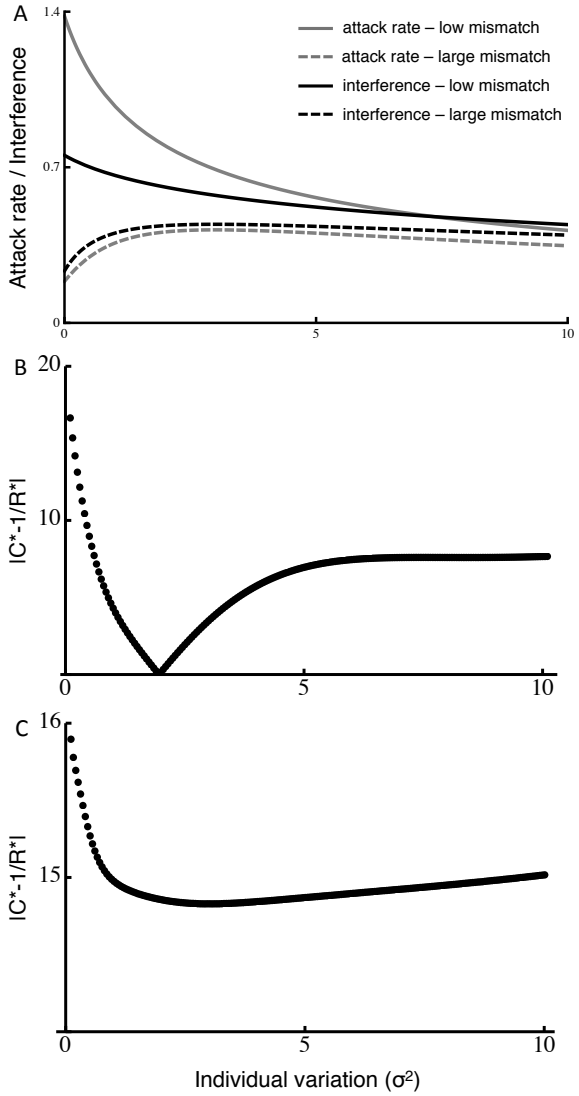

Figure 3: A. Plot of the attack efficiency (grey) and interference competition (black) against individual variation under low phenotypic mismatch (solid) and large phenotypic mismatch (dashed). Variation decreases attack rates to a larger extent than interference competition when mismatch is small, and the effect on both parameters is comparable when mismatch is large. Parameter values as in Figure 2. B. Plot of the absolute value of difference between consumer equilibrium density, C*, and consumer competitive ability 1/R*, as a function of individual variation under low phenotypic mismatch. Variation maximizes both simultaneously whenever the curve is at its lowest point. C. Same as in B but for large phenotypic mismatch. Parameter values as in Figure 2.

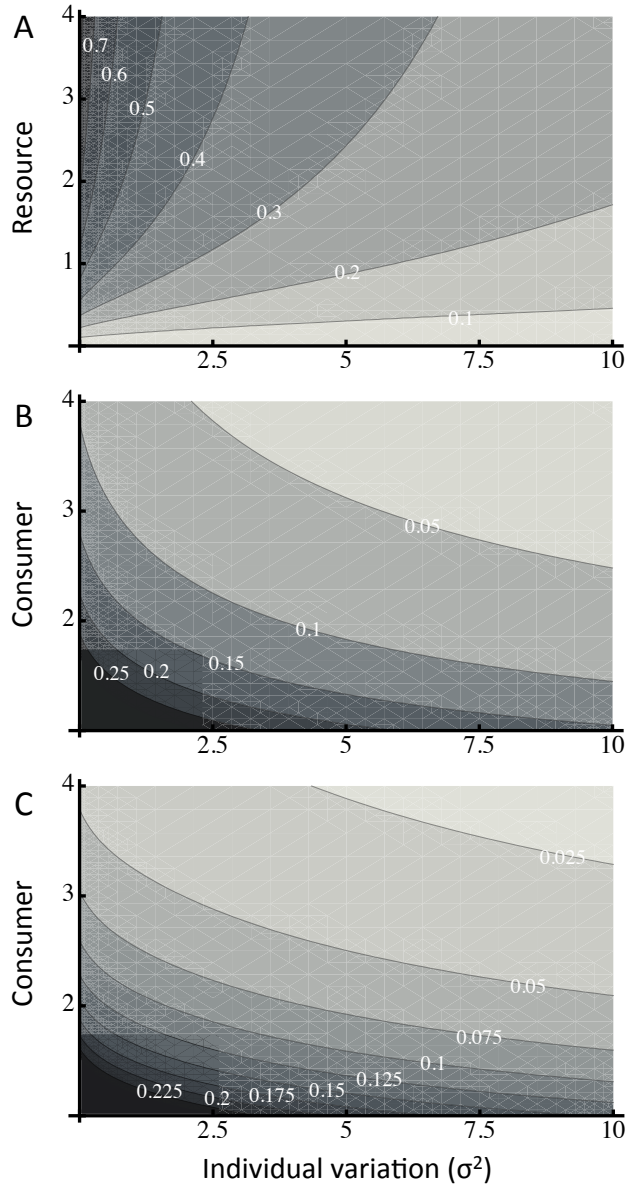

Figure 4: Plots of the effect of individual variation and either resource (A) or consumer density (A and B: foraging rate as in equation 8, C: foraging rate as in equation 10) upon foraging rates (gray scale). Individual variation and consumer and resource densities have a joint effect upon foraging rates, and should thus not be studied separately: foraging rates increase with resource density, decrease with consumer density, and decrease with individual variation. Parameter values as in Figure 2. R and C where kept constant and equal to 1 whenever the other variable was varied.

CHAPTER 3

THE EFFECT OF PHENOTYPIC VARIATION ON METAPOPULATION PERSISTENCE

Jean P. Gibert

Key-words: Extinction risk, Individual variation, Migration, Trait variation, Variability

ABSTRACT

Demographic stochasticity (due to the probabilistic nature of the birth-death process) and demographic heterogeneity (between-individual differences in demographic parameters) have long been seen as factors affecting extinction risk. While demographic stochasticity can be independent of underlying species traits, demographic heterogeneity may strongly depend on phenotypic variation. However, how phenotypic variation can affect extinction risk is largely unknown. Here, I develop a stochastic metapopulation model that takes into account the effects of demographic stochasticity and phenotypic variation in the traits controlling colonization rates to assess what the effect of phenotypic variation may be on the persistence of the metapopulation. Although phenotypic variation can lead to a decrease in metapopulation persistence under some conditions, it also may lead to an increase in persistence whenever phenotypic mismatch – or the distance between the optimal trait value and the population mean – is large. This mismatch can in turn arise from a variety of ecological and evolutionary reasons, including weak selection or a

recent history of invasion. Last, the effect of phenotypic variation has a deterministic component on colonization rates, and a stochastic component on persistence through colonization rates, but both are important to understand the overall effect. These results have important implications for the conservation of threatened species and management practices that may historically have overlooked phenotypic variation as unimportant noise.

INTRODUCTION

Understanding the factors leading to extinction is a central goal in ecology (e.g., (Gilpin & Hanski 1991; Lande 1993; Kendall & Fox 2003; Melbourne & Hastings 2008)). This understanding is crucial when it comes to making informed decisions about the management of endangered species or sets of species in threatened communities and ecosystems (Gilpin & Hanski 1991). Many factors influencing extinction risk have been identified, including abiotic factors (e.g., pollution), biotic factors (e.g., invasive species) as well as exogenous factors (i.e., that are external to the focal population) and endogenous factors (i.e., that are related to internal population level processes, (Roughgarden 1975; Melbourne & Hastings 2008).

Stochasticity in population growth and dynamics has long been seen as a major factor increasing extinction risk (May 1973; Chesson 1981; Fox & Kendall 2002). The sources of this stochasticity are many, and they can be broadly divided into two classes: demographic – or stochasticity in population growth due to the random nature of the birth-death process –, and environmental – or random fluctuations in environmental conditions that lead to fluctuations in the number of births and deaths – (e.g., (Caswell 2001, 2009; Lande, Engen & Sæther 2003; Engen

et al. 2005)). Although both types of stochasticity can increase extinction risk, demographic stochasticity is mainly a problem for small populations (Caswell 2001), while environmental stochasticity can affect much larger populations (Caswell 2001; Melbourne & Hastings 2008).

Another source of stochasticity in population growth is demographic heterogeneity (Conner & White 1999; Fox & Kendall 2002; Kendall & Fox 2003; Fox 2005; Vindenes, Engen & Saether 2008; Vindenes & Langangen 2015). This source of stochasticity occurs whenever there are actual differences among individuals (e.g., the traits they have) that lead to systematic differences in their chance of surviving and reproducing, as opposed to differences in the chance of surviving and reproducing due to the randomness of births and deaths, as it is the case in demographic stochasticity (Melbourne & Hastings 2008). How demographic heterogeneity affects extinction risk is an active area of research and has been shown to have opposite effects on extinction risk. Indeed, heterogeneity can reduce extinction risk (Conner & White 1999; Fox & Kendall 2002; Fox 2005), increase it (Robert, Sarrazin & Couvet 2003) or both increase and decrease extinction risk (Kendall & Fox 2003; Vindenes *et al.* 2008; Melbourne & Hastings 2008).

Demographic heterogeneity can arise from a number of factors including geographic or habitat heterogeneity (e.g., (Gates & Gysel 1978; Menge *et al.* 1994; Landis *et al.* 2005)), frailty effects and reproductive heterogeneity (Vaupel & Yashin 1985; Fox *et al.* 2006; Kendall *et al.* 2011), and both genetic and phenotypic variation (Chesson 1981). Ecologists have historically dismissed phenotypic variation as noise around mean trait values of interest (Lomnicki 1988). But even populations that are made of clones (i.e., individuals that share the same genetic makeup) will have slight differences in the way genes are expressed (e.g., (Price, Qvarnström & Irwin 2003)),

leading to potentially important amounts of phenotypic variation (Lomnicki 1988; Sherratt & MacDougal 1995). More importantly, there are strong reasons to expect ecological effects of variation *per se* whenever there exists a concave down or concave up relationship between a focal trait and the ecological process of interest through Jensen's inequality (Jensen 1906; Ruel & Ayres 1999; Gibert & Brassil 2014; Gibert & DeLong 2015). Phenotypic variation thus has the potential to alter demographic heterogeneity and stochasticity, and through that, have consequences for the persistence of populations with considerable phenotypic variation.

This paper assesses whether phenotypic variation in traits controlling ecological processes can have important effects on metapopulation persistence. Phenotypic variation has recently been shown to influence an enormous set of parameters and processes (Bolnick *et al.* 2011; Araújo, Bolnick & Layman 2011; Violle *et al.* 2012a; Gibert *et al.* 2015), including predator-prey interactions (through for example, attack rate, handling time and mutual interference,(Okuyama 2008, 2013; Pettorelli *et al.* 2011; Gibert & Brassil 2014; Gibert & DeLong 2015), dietary variation (Snowberg, Hendrix & Bolnick 2015), disease dynamics (Lloyd-Smith *et al.* 2005), food web structure (Svanbäck *et al.* 2015), tri-trophic interactions (Hughes *et al.* 2015), as well as trait evolution (Fisher 1930; Dobzhansky 1937; Frank 2012) and eco-evolutionary dynamics (Schreiber *et al.* 2011; Vasseur *et al.* 2011). In all cases, phenotypic variation was found to be largely stabilizing and to potentially increase population persistence.

Here I address how underlying phenotypic variation in traits that control dispersal, such as wing length or body size, might affect the persistence of a metapopulation model with demographic stochasticity. I argue that this effect may be mediated through deterministic consequences of the occurrence of phenotypic

variation in the parameters controlling the dynamics of the system, which in turn result in a stochastic effect on metapopulation persistence.

METHODS

Generalities

In metapopulations, persistence results from the balance between two main parameters: colonization and extinction rates (Levins 1969). Because the local extinction rate is largely dependent upon patch size (MacArthur & Wilson 1967; Laurance 2005; Arroyo-Rodríguez *et al.* 2009), I assume that phenotypic variation will mainly affect colonization rates and have no effect on extinction rates. Colonization rates depend in turn on the distance colonizers must travel (Levins 1969; Gibert *et al.* 2013) and on the production of colonizers (Alonso & McKane 2002), which is a function of demographic parameters and is thus ultimately determined by phenotypic variation, as has been shown empirically in three-spined sticklebacks (Laskowski *et al.* 2015). To address how phenotypic variation in the traits determining colonization rates affect persistence, I used two models: the first considers external migration from a mainland only whenever the metapopulation goes extinct; the second considers migration from a mainland as a process that can occur anytime.

The models

For the first model, I reformulated an already existing stochastic metapopulation model (Gurney & Nisbet 1978) to track the total number of occupied patches over time for a species living in a space consisting of N identical patches with no spatial correlation. I later modified this model (see section *"Incorporating phenotypic variation"*) to take phenotypic variation into account. The model assumes

that at each infinitesimal time step, there is a chance for an empty patch to be colonized and for an occupied patch to become unoccupied through local extinction. These one-step transition probabilities are independent of the state of the system at previous time steps and can be written as:

$$C(n+1 \mid n) = cn\left(1-\frac{n}{N}\right)dt\,, \tag{1}$$

$$E(n-1 \mid n) = en\,dt\,, \tag{2}$$

where $C(n+1 \mid n)$ is the probability that an unoccupied patch is colonized, $E(n-1 \mid n)$ is the probability that an occupied patch becomes unoccupied, N is the total number of patches in the metapopulation, n is the number of occupied patches, c is the colonization rate and e is the extinction rate (Levins 1969; Alonso & McKane 2002). We define $C(1 \mid 0) \equiv \phi\,dt$, which can be seen as a chance of receiving migrants from outside the metapopulation if the metapopulation was to go extinct. As ϕ decreases, the stationary probability of extinction tends to 1. Setting $\phi \neq 0$, however, does not preclude the metapopulation from going extinct because 1) ϕ can be arbitrarily small and 2) even if ϕ is large, its final effect will depend on the relative values of all other colonization and extinction transition probabilities. I nevertheless assessed the effect of ϕ in the dynamics of the model (see results) for thoroughness. This model would apply to scenarios where external migration from the continent is so negligible compared to $C(n+1 \mid n)$ (i.e., $C(n+1 \mid n) >> \phi$), that effectively the only time the external migration impacts dynamics is when the metapopulation as a whole goes extinct. It could also apply in situations where humans monitor the status of a metapopulation, and supply propagules when the metapopulation goes extinct either intentionally or unintentionally, which may be of relevance in management and conservation scenarios. Last, I imposed a boundary at n=0, by defining

$C(0\,|\,-1) = E(-1\,|\,0) \equiv 0$, since patch occupancy cannot be negative. To simplify notation, I will refer to $C(n+1\,|\,n)$ as C_n, to $C(n\,|\,n-1)$ as C_{n-1}, to $E(n-1\,|\,n)$ as E_n and to $E(n\,|\,n+1)$ as E_{n+1} from now on.

Under these conditions, the master equation controlling the change in the distribution of occupied sites ($P(n,t)$) over time can be written as:

$$\frac{dP(n,t)}{dt} = C_{n-1}P(n-1,t) + E_{n+1}P(n+1,t) - P(n,t)\left(C_n + E_n\right), \tag{3}$$

where the probability of finding *n* occupied patches at time *t* increases with the probability that a colonization event occurred multiplied by the probability of having *n-1* occupied sites ($C_{n-1}P(n-1,t)$), increases with the probability that an extinction event occurred multiplied by the probability of having *n+1* occupied sites ($E_{n+1}P(n+1,t)$), and decreases with the probability of having both an extinction or a colonization event multiplied by the probability of having exactly *n* occupied sites ($P(n,t)\left(C_n + E_n\right)$,(van Kampen 1981). The stationary distribution ($t \to \infty$) can be found by recurrence (Appendix IX), and is equal to:

$$P(n,\infty) = \frac{C_0 \ldots C_{n-1}}{E_1 \ldots E_n} P(0,\infty), \tag{4}$$

with

$$P(0,\infty) = \frac{1}{1 + \sum_{n=1}^{N} \frac{C_0 \ldots C_{n-1}}{E_1 \ldots E_n}}. \tag{5}$$

Using Eqs. (1) and (2) and assuming $\phi = 1$ for simplicity, the stationary distribution for the model becomes (Appendix X):

$$P(n,\infty) = \frac{\frac{1}{ne^n}\left(\frac{c}{N}\right)^{n-1}\frac{\Gamma(N)}{\Gamma(N-n+1)}}{1 + \sum_{n=1}^{N}\frac{1}{ne^n}\left(\frac{c}{N}\right)^{n-1}\frac{\Gamma(N)}{\Gamma(N-n+1)}}, \tag{6}$$

where Γ is the Gamma function. The mean number of occupied patches can then be found as:

$$\langle n \rangle = \sum_{n=1}^{N} \frac{\frac{1}{e^n}\left(\frac{c}{N}\right)^{n-1} \frac{\Gamma(N)}{\Gamma(N-n+1)}}{1+\sum_{j=1}^{N} \frac{1}{j e^j}\left(\frac{c}{N}\right)^{j-1} \frac{\Gamma(N)}{\Gamma(N-j+1)}} . \quad (7)$$

The mean number of occupied patches increases in this model with increasing colonization rate (Fig. 1a) and decreases with increasing extinction rate (Fig. 1b), as expected from classic Levin's model (Levins 1969). The model is the continuous-time Markov chain counterpart to Gurney and Nisbet's (1978) stochastic Langevin equation model, based on Levins' metapopulation model (Levins 1969). Even though my particular formulation has not been explored before, its behavior should in all respects be equivalent to that of Gurney and Nisbet (1978).

The second model I explored is a continuous-time Markov chain model developed by Alonso and McKane (2002), which, contrary to the previous one, assumes that the metapopulation can receive external migration from a mainland at any time. In that case, the probability that a colonization event occurs in a time lapse *dt* is

$$C(n+1 \mid n) = cn\left(1-\frac{n}{N}\right)dt + m(N-n)dt , \quad (8)$$

where *m* is the migration rate from the continent, and everything else is an in the first model. The number of occupied patches is approximately (Alonso & McKane 2002):

$$\langle n \rangle \approx \frac{N}{2}\left(1-\frac{m-e}{c}+\sqrt{\left(1+\frac{m-e}{c}\right)^2+4\frac{me}{c^2}}\right). \quad (9)$$

This approximation is particularly useful because once I incorporate phenotypic variation the mean number of occupied patches rapidly becomes difficult if not

impossible to compute numerically. In what follows, I modify these models to account for phenotypic variation in a trait that determines colonization rates.

Incorporating phenotypic variation

Building upon previous work (Schreiber *et al.* 2011; Gibert & Brassil 2014; Gibert & DeLong 2015), I incorporated phenotypic variation by assuming the existence of a normally distributed trait x with probability density function $p(x,\bar{x},\sigma^2)$, that determines colonization rates in a Gaussian way:

$$c(x) = c_{\max} \exp\left[-\frac{1}{2}\frac{(\theta - x)^2}{\tau^2}\right], \tag{10}$$

where $c_{\max}$ is the maximal colonization rate, θ represents a trait value at which colonization is optimal, and τ controls the rate at which colonization rates decrease away from the optimum value. This functional form is common in traits controlling dispersal such as body size (Manzaneda, Rey & Alcántara 2009) or wing length (Pulido & Widmer 2005), and can arise from either stabilizing or conflicting selection in the traits controlling dispersal (Manzaneda *et al.* 2009). Gaussian functional forms such as the one assumed here are also common in theoretical papers studying how traits and their evolution might affect metapopulation dynamics (Hanski & Mononen 2011; Hanski, Mononen & Ovaskainen 2011). Notice that colonization rates are defined at the metapopulation level, so the trait distribution considered is also defined across local populations. Using (10) it is possible to calculate the mean colonization rate as,

$$\langle c \rangle = \int_{-\infty}^{\infty} c(x) p(x,\bar{x},\sigma^2)\, dx, \tag{11}$$

which convolves nicely to:

$$\langle c \rangle = \frac{c_{\max}\tau}{\sqrt{\tau^2+\sigma^2}} \exp\left[-\frac{1}{2}\frac{d^2}{\tau^2+\sigma^2}\right], \quad (12)$$

where $d^2 = (\theta - \bar{x})^2$, hereafter referred to as phenotypic mismatch following previous work (Raimundo *et al.* 2014; Gibert & Brassil 2014; Gibert & DeLong 2015). Notice that while selection can reduce mismatch, it is not certain that it will. Indeed, whether mismatch will decrease ultimately depends on a number of other factors, including, but not limited to, whether selection is strong, whether there is antagonistic selection on the trait (e.g., imposed by other interacting species or environmental differences across patches that makes moving between patches deleterious) or whether the trait is plastic. Changes in τ affect how sensitive colonization rates are to changes in both phenotypic mismatch and phenotypic variation. Because $\langle c \rangle$ is an explicit function of phenotypic variation (σ^2), it is now possible to assess the effect of the latter in the mean number of occupied sites as well as in their variance for both models. Using (7) and (12), $\langle n \rangle$ becomes for the first model:

$$\langle n \rangle = \sum_{n=1}^{N} \frac{\frac{1}{e^n}\left(\frac{\langle c \rangle}{N}\right)^{n-1}\frac{\Gamma(N)}{\Gamma(N-n+1)}}{1+\sum_{j=1}^{N}\frac{1}{je^j}\left(\frac{\langle c \rangle}{N}\right)^{j-1}\frac{\Gamma(N)}{\Gamma(N-j+1)}}, \quad (13)$$

while for the second model it becomes:

$$\langle n \rangle \approx \frac{N}{2}\left(1-\frac{m-e}{\langle c \rangle}+\sqrt{\left(1+\frac{m-e}{\langle c \rangle}\right)^2+4\frac{me}{\langle c \rangle^2}}\right). \quad (14)$$

Alternatively, phenotypic variation can be incorporated in a different way. For the first model, by plugging (10) into (7), and convolving the whole with the trait distribution we get:

$$\langle n \rangle = \int_{-\infty}^{\infty} \sum_{n=1}^{N} \frac{\frac{1}{e^n}\left(\frac{c(x)}{N}\right)^{n-1} \frac{\Gamma(N)}{\Gamma(N-n+1)}}{1+\sum_{j=1}^{N} \frac{1}{j e^j}\left(\frac{c(x)}{N}\right)^{j-1} \frac{\Gamma(N)}{\Gamma(N-j+1)}} p(x,\bar{x},\sigma^2)\,dx\,. \qquad (15)$$

For the second model, the expression reads:

$$\langle n \rangle \approx \int_{-\infty}^{\infty} \frac{N}{2}\left(1-\frac{m-e}{c(x)}+\sqrt{\left(1+\frac{m-e}{c(x)}\right)^2+4\frac{me}{c(x)^2}}\right) p(x,\bar{x},\sigma^2)\,dx\,. \qquad (16)$$

Equations (15) and (16) are not always numerically computable, so in some cases I had to adjust the range of parameters studied. Comparing the two ways in which I incorporated phenotypic variation helped with assessing how robustly we can predict the effects of phenotypic variation on metapopulation persistence.

RESULTS

I explored two different scenarios, one where phenotypic mismatch is zero ($d^2 = 0$), and one where phenotypic mismatch is larger than zero ($d^2 > 0$). In the first model, it can be seen that, for $d^2 = 0$, the mean number of occupied patches goes down with phenotypic variation (Figs. 2a-d). This is true for both ways of incorporating phenotypic variation (Figs. 2a, b vs Figs. 2c, d), different metapopulation sizes (Figs. 2a, c) and values of the parameter τ (Figs. 2b, d). The effect of phenotypic variation on mean occupancy, however, is different when phenotypic mismatch is large ($d^2 > 0$): mean occupancy increases at first, and then decreases (Figs. 2e-h). These results also hold for the Alonso and McKane model (Fig. 3). When exploring the effect of variation using (16), however, it was not possible to analyze all scenarios explored for the first model, which is why I do not show results on how varying τ affects mean occupancy for that model, or why the

range of values analyzed for phenotypic variation is slightly smaller in this case as well (i.e., Figs. 3c, f).

Last, I explored how joint changes in parameters could affect metapopulation persistence. When phenotypic variation and phenotypic mismatch vary together in the first model, mean occupancy decreases as they jointly increase, and is maximal when there is no phenotypic variation or mismatch (Fig. 4a). This shows that phenotypic mismatch may be detrimental for metapopulation persistence in a case where underlying traits control colonization rates in a gaussian fashion, as is assumed here. This effect of variation and mismatch is not qualitatively affected by considering external migration (as in the second model, Figs. 4b, c), and is not qualitatively affected by the value of ϕ either (external migration when metapopulation goes extinct as explained for the first model, Fig. 4d). Even in the range of values where ϕ has a strong effect ($\phi \approx 0$), phenotypic variation generally decreases occupancy if phenotypic mismatch is small.

DISCUSSION

My results show that phenotypic variation can have both negative and positive impacts on extinction risk in metapopulations, which is consistent with what past studies have shown about the effect of demographic heterogeneity (Chesson 1981; Conner & White 1999; Fox & Kendall 2002; Kendall & Fox 2003; Fox 2005; Vindenes *et al.* 2008; Melbourne & Hastings 2008). Regarding the positive effect of phenotypic variation, my results are also in line with what others have shown in deterministic models (Schreiber *et al.* 2011; Vasseur *et al.* 2011; Gibert & Brassil 2014; Gibert & DeLong 2015). These results also suggest that the effect of phenotypic variation can strongly depend on other important factors, such as,

phenotypic mismatch, which is ultimately controlled by past and present selection acting on the traits controlling dispersal. Indeed, it is this parameter that ultimately determines whether the effect of phenotypic variation is positive or negative. Hence, to fully understand the effect of phenotypic variation on extinction risk, we may need to also understand how it interplays with past and current selection acting on the traits that control the process of interest, as recent studies argue (Hairston Jr *et al.* 2005; Hanski *et al.* 2011; Schreiber *et al.* 2011; Gibert *et al.* 2015).

Phenotypic mismatch, or the difference between the optimal and the mean trait value in the population can result from selection acting on a focal trait (Fellowes, Kraaijeveld & Godfray 1998; Nuismer, Gomulkiewicz & Ridenhour 2010). Indeed, low levels of phenotypic mismatch may result from strong stabilizing selection constantly pushing to maintain the trait on or near an optimal value (Nuismer *et al.* 2010). Large levels of mismatch may result from weak selection (Nuismer *et al.* 2010), or from a recent history of invasion of the species to its current habitat or location (Jones & Gomulkiewicz 2012). Here, we show that this phenotypic mismatch can have important consequences for the survival of a metapopulation, since at low levels of mismatch, phenotypic variation decreases mean occupancy and increases the chance that the metapopulation will go extinct, while at larger levels of mismatch, phenotypic variation can have the opposite effect. Because phenotypic mismatch may change over time through rapid evolutionary change of the traits controlling dispersal, these results suggest the possibility that eco-evolutionary feedbacks may have important consequences for metapopulation persistence, as other studies stressed (Hanski & Mononen 2011; Hanski *et al.* 2011).

It is possible that selection may reduce phenotypic mismatch over time, which eventually would lead to a scenario that could gradually erode phenotypic variance. It

is thus important to contemplate mechanisms that could maintain phenotypic variation over time for the results of this paper to hold for metapopulations under strong stabilizing selection. Such possible mechanisms include all classic evolutionary processes such as gene flow from outside the metapopulation, mutation and pleiotropy (e.g., (Mitchell-Olds, Willis & Goldstein 2007)). However, depending on how heritable the traits are, it is possible that some if not most of the variation in the traits controlling dispersal might arise through phenotypic plasticity, which will not be eroded by selection, even though it can fuel evolutionary change as well (Price *et al.* 2003). Thus, even in scenarios under strong stabilizing selection, it is possible to still find core levels of irreducible phenotypic variation and phenotypic mismatch, with potentially important ecological effects on the persistence of metapopulations.

It has also been empirically shown that phenotypic variation controls dispersal capacity in the three-spine stickleback *Gasterosteus aculeatus* (Laskowski *et al.* 2015). Together with my findings, these results suggest that individual phenotypic variation may be a key yet largely overlooked factor when it comes to devising conservation and management plans for threatened metapopulations. For example, by not taking phenotypic variation into account, the probability of extinction of a metapopulation might be largely underestimated if the phenotypic mismatch is small, or largely overestimated if the mismatch is large. Moreover, the increasing temperatures associated with global warming are likely to affect the mean body size of some if not most ectothermic species (Daufresne, Lengfellner & Sommer 2009; Sheridan & Bickford 2011), with important consequences for food web body size structure (Gibert & DeLong 2014). Yet, little is known as to how warming may affect variation in body size even though there are strong reasons to believe that both the mean and variance of body size can change with temperature, as it was empirically

shown in a protist system (DeLong 2012). Together, these results further emphasize the need for a deeper understanding of the effect of phenotypic variation upon ecological processes, and how these effects may be potentially mediated by environmental temperature.

Importantly, the effect described here occurs through two important but distinct components: first, there is a deterministic effect of phenotypic variation on colonization rates, then, this deterministic effect leads to a stochastic effect of phenotypic variation on metapopulation persistence through colonization rates (which determines demographic stochasticity in these models). Our results thus highlight the importance of considering the joint effect of both deterministic and stochastic effects in regulating the fate of natural populations. Notice, however, that we may not be able to separately quantify their effects in nature, as the deterministic effect of phenotypic variation on colonization rates is a prerequisite for the stochastic effect of phenotypic variation on metapopulation persistence. These two components of the overall effect of phenotypic variation occur sequentially and are fundamentally linked, which makes their separate quantification potentially challenging. Both ought to be considered together as a complex pathway through which phenotypic variation affects persistence.

It is important to notice as well that my model depends on a number of parameters whose values will affect the results shown here. For example, the value of τ, the parameter that controls the sensitivity of the colonization rates to changes in phenotypic variation, is arbitrary in my models, and larger values of the parameter will lead to scenarios where no effect of phenotypic variation may be observed. Also, the effects described here strongly depend on the total number of patches considered (Fig. 2, Fig. 3), with larger populations needing larger levels of phenotypic mismatch

to show a similar change in the total number of occupied sites. Last, it is interesting to notice that the two models explored showed qualitatively similar but quantitatively different responses to phenotypic variation (Fig. 2 vs Fig 3). In Levins' model (Eqs. (13) and (15)), the effect of phenotypic variation was much stronger than in Alonso and McKane's (Equations. (14) and (16)). This is because of the way both models depend on c: the Levins' model can be roughly seen as parabolic function of c of the form $\frac{\sum_{i=1}^{N} c^i}{1+\sum_{i=1}^{N} c^i}$, thus tending faster and faster to 1 with increasing N; Alonso and McKane's model, on the other hand, goes to 1 as $1-\frac{1}{c}$, which does so at a slower pace. This difference in the concavity of both functions with respect to c leads to a difference in how much they respond to variation in the parameter. The faster the function tends to 1, the stronger their concavity with respect to c and the stronger they respond to variation in that parameter, as a previous study also suggested (Inouye 2005).

Overall, this paper shows that the effect of phenotypic variation may be more complex than meets the eye. Indeed, phenotypic variation underlies demographic heterogeneity, but its effects on metapopulation persistence depends on other factors such as phenotypic mismatch as well as its deterministic effects on the parameters controlling the dynamics of interest. At low levels of mismatch, phenotypic variation decreases persistence, but at larger levels of mismatch, the pattern can be reversed. This paper emphasizes the fact that both phenotypic variation and phenotypic mismatch may need to be taken into account when devising conservation plans of endangered species living in patches connected by migration in a context of global change.

ACKNOWLEDGMENTS

I am indebted to John DeLong for his advice, constant support and insightful comments. I am grateful with Volker Grimm and two anonymous reviewers for helpful suggestions. I was supported by a National Science Foundation Doctoral Dissertation Improvement Grant (DEB-1501668).

FIGURES

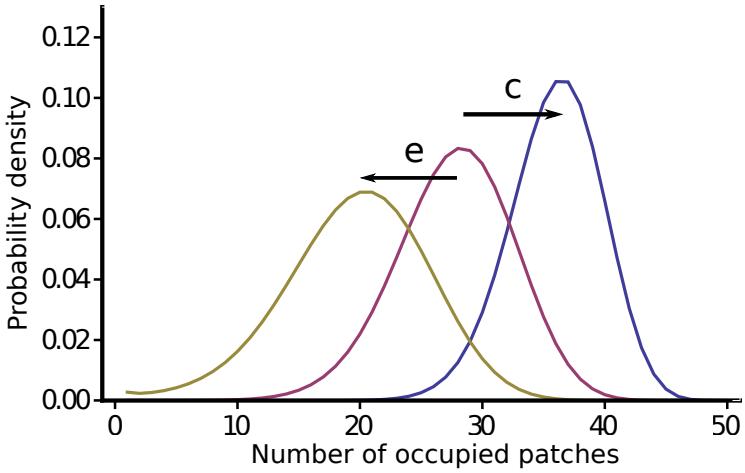

Fig 1: Plot of the probability density associated to the number of occupied patches from Eq. (6). We can see how an increase in colonization rate (c) leads to an increase in the mean number of occupied patches while an increase in extinction rate (e) leads to a decrease in the mean number of occupied patches. In red, our canonical parameter set for comparison: $c = 1.4$, $e = 0.6$, $\phi = 1$ and $N = 50$. In yellow, everything as in red but for $e = 0.6$. In blue, everything as in red but for $c = 2.2$.

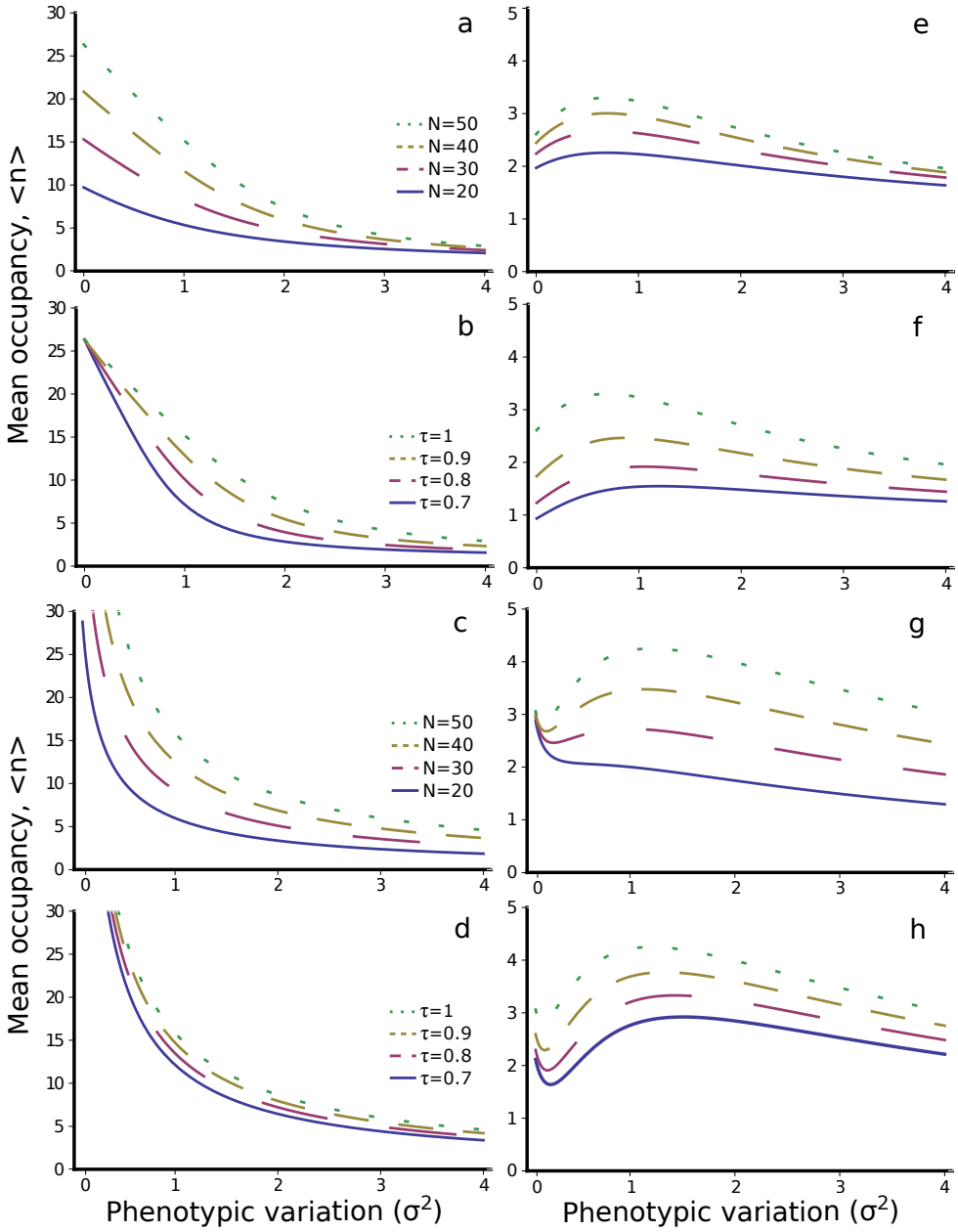

Fig 2: a-d. Plots of the mean number of occupied patches ($< n >$) in the first model against phenotypic variation (σ^2) for varying levels of the total number of patches (N) (a and c), varying levels of the parameter τ (b and d), and low phenotypic mismatch ($d^2 = 0$). Plots a and b were obtained using Eq. (13) and plots c and d were obtained using Eq. (15). e-h. Same as in a-d but for large phenotypic mismatch ($d^2 = 1.3$). Other parameters: $c_{\max} = 2.2$, $e = 0.6$, $\phi = 1$ and $\tau = 1.0$ (unless otherwise stated), $N = 50$ (unless otherwise stated).

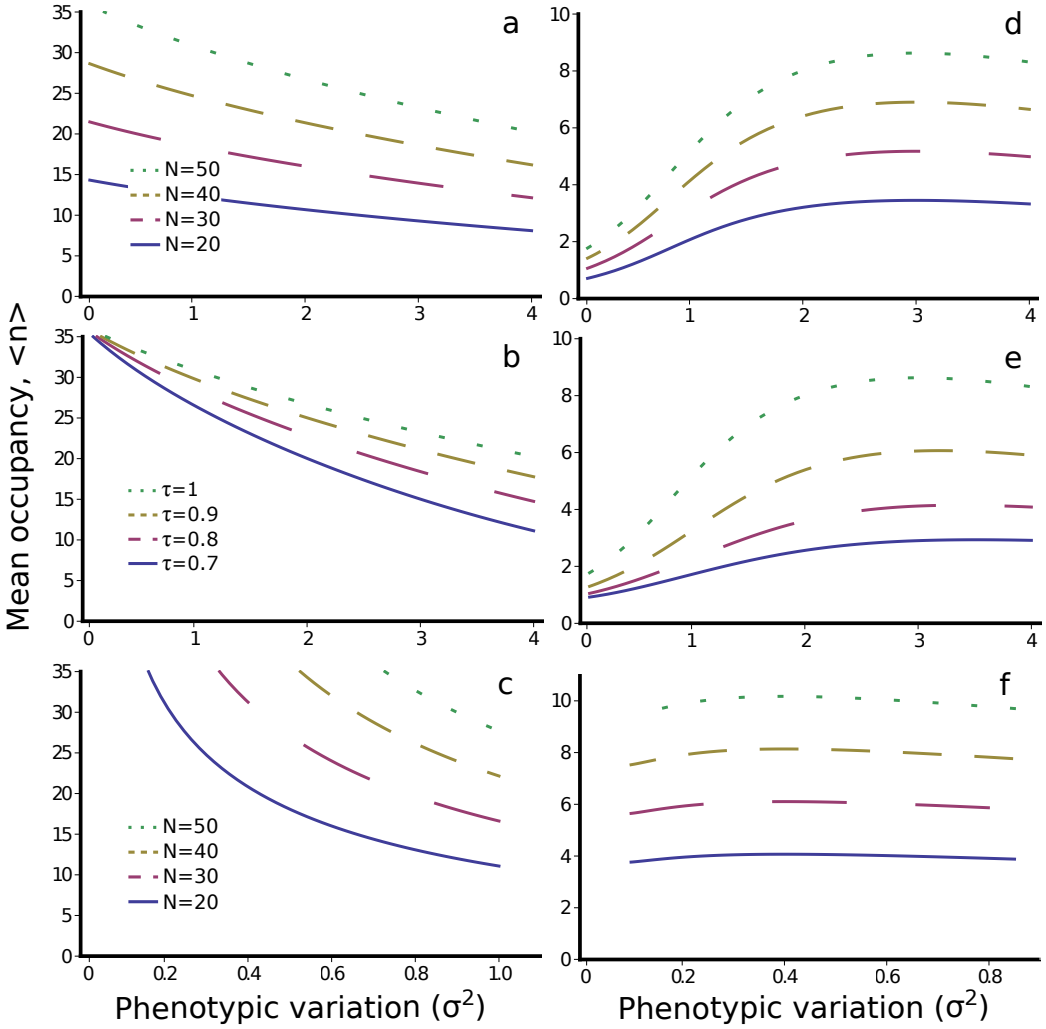

Fig 3: a-c. Plots of the mean number of occupied patches ($< n >$) in the second model against phenotypic variation (σ^2) for varying levels of the total number of patches (N) (a and c), varying levels of the parameter τ (b), and low phenotypic mismatch ($d^2 = 0$). Plots a and b were obtained using Eq. (14) and plot c was obtained using Eq. (16). e-f. Same as in a-c but for large phenotypic mismatch ($d^2 = 1.3$). All other parameters as in Fig. 1.

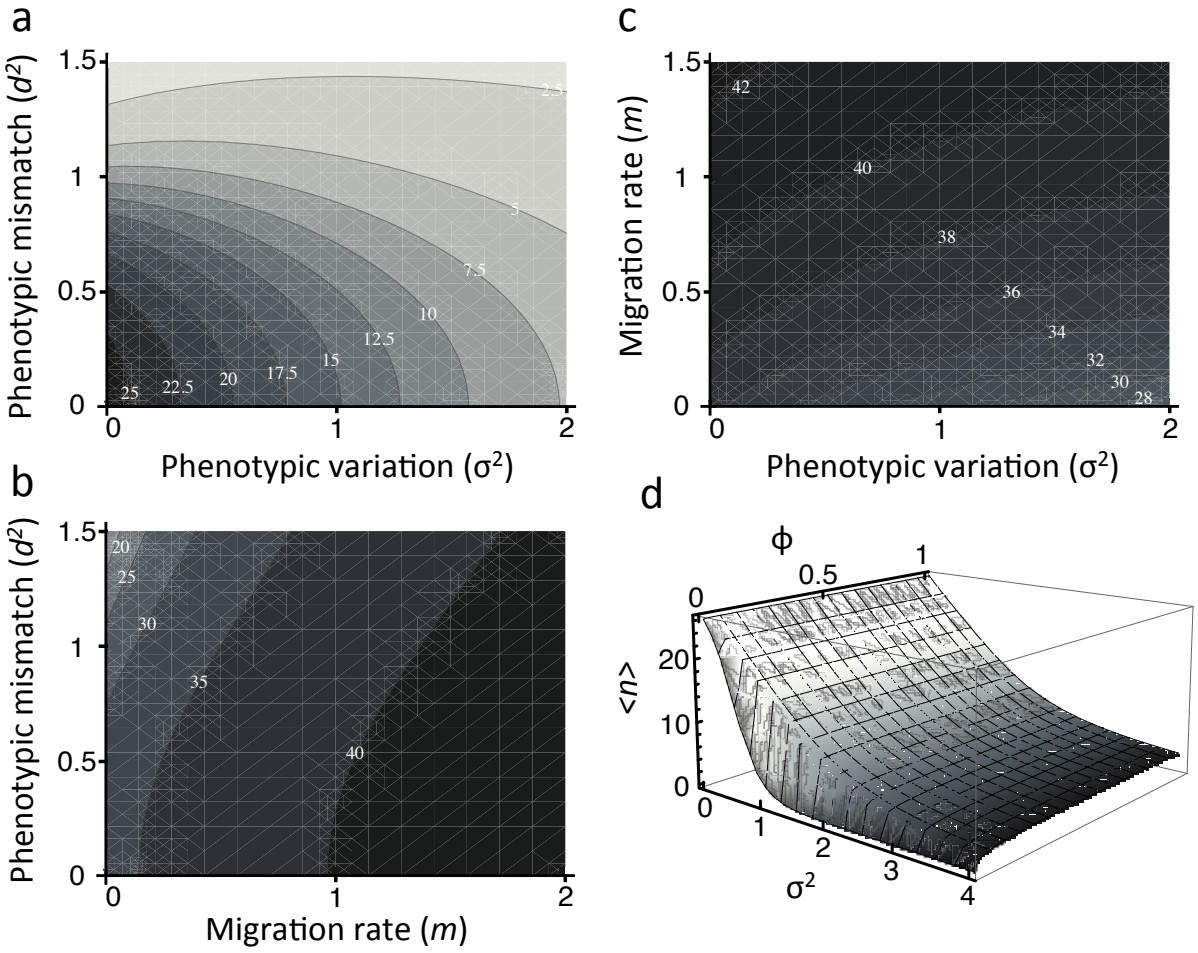

Fig 4: a. Contour plot of how the mean number of occupied patches (grey tones, white numbers), changes as a function of phenotypic mismatch (d^2) and phenotypic variation (σ^2) for the first model. b. Same as in a but for migration rate (m) and phenotypic variation for the second model. c. Same as in b but for phenotypic mismatch and migration rate. d. Surface showing how the effect of phenotypic variation on mean occupancy changes with ϕ, for low phenotypic mismatch ($d^2 = 0$). All other parameters as in previous figures

CHAPTER 4

THE JOINT EFFECT OF BODY SIZE, INTRASPECIFIC VARIATION AND THEIR CHANGE WITH TEMPERATURE ON A FRESHWATER PREDATOR-PREY SYSTEM

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Key-Words: Traits, Warming, Individual Variability, Consumer, Resource

ABSTRACT

Understanding the factors underpinning food web structure and stability is a long-standing issue in ecology. This understanding, however, ultimately hinges on honing our comprehension of the factors influencing predator-prey interactions. Mean body size is an important determinant of predator-prey interactions, and although intraspecific variation in body mass has long been dismissed as noise around mean values of interest, recent studies suggest that it may play a larger role than previously thought. Moreover, although temperature can influence both the mean and variance of body size, how body size, body size variation, and temperature jointly affect predator-prey interactions is not known. Here, we address this issue in a freshwater copepod-protist predator-prey system using an integrative approach that tests mathematical models with empirical data from foraging trials. We show that mean body size plays a major role in determining the parameters of the predator functional response, which leads to important and predictable effects of intraspecific variation, per se, on those parameters. Moreover, these effects are mediated by temperature, and we make

testable predictions as to how increasing average temperatures resulting from global climate may lead to an increase in interaction strengths between copepods and their prey. Because copepods are a major intermediate predator in aquatic food webs, our results suggest that the structure and dynamics of complex food webs may be determined by the joint effect of mean body size and variation in body size, mediated by environmental temperature. These results are central for our understanding of how natural systems will respond to increasing temperatures.

INTRODUCTION

Food webs are complex systems influenced by myriad factors such as species diversity (May 1972; Allesina & Tang 2012), the strength of feeding interactions (Paine 1992; McCann *et al.* 1998), and environmental conditions (Binzer *et al.* 2012; Gibert & DeLong 2014). Assessing how food webs persist in nature is thus a challenging task that requires a fine-grained understanding of the factors influencing food-web building blocks, that is, the predator-prey interactions themselves. Many behavioral, physiological and biomechanical processes play a central role in determining the strength of predator-prey interactions (e.g. (Kennedy & White 1996; Hammill *et al.* 2010; Riede *et al.* 2011; Schmitz & Price 2011; Jonsson 2014; DeLong, Hanley & Vasseur 2014a). Yet how population-level patterns are affected by processes occurring at the individual level, such as traits individuals have, and their variation within populations, has been largely overlooked in ecology (Lomnicki 1988; Bolnick *et al.* 2011).

One striking exception is long-term focus on how body size affects predator-prey interactions. Body size often determines the diet (e.g. (Schneider, Scheu & Brose 2012)) and trophic level of predators (e.g. (Riede *et al.* 2011)), as well as the

parameters of the functional response (e.g. (DeLong & Vasseur 2012b)). Body size thus plays a paramount role in determining how predator-prey interactions occur, especially in gape-limited organisms (Arim, Bozinovic & Marquet 2007). Body size can be extremely plastic in nature (David, Legout & Moreteau 2006), which can lead to large amounts of variation within populations (Giometto *et al.* 2013). Traits that directly affect ecological processes, such as body size, are also likely to affect those processes at the population scale via their intraspecific variation. Indeed, variation in traits like body size has been shown theoretically to affect predator-prey interaction strengths and dynamics (Gibert & Brassil 2014; Gibert & DeLong 2015), as well as eco-evolutionary dynamics (Schreiber *et al.* 2011; Vasseur *et al.* 2011). Empirical support for these findings are mixed, however, with some studies showing no effect of intraspecific variation (Ingram, Stutz & Bolnick 2011) and others showing important effects on predator-prey interactions (Pettorelli *et al.* 2011; Hughes *et al.* 2015; Cronin *et al.* 2016).

Both body size and its variation within populations can change dramatically over short periods of time (DeLong, Hanley & Vasseur 2014b), responding to changes in resource availability (e.g. (Anholt & Werner 1998)) or predation (e.g. (MacLeod *et al.* 2007)). Abiotic factors such as temperature can also influence body size. Indeed, organisms generally become smaller at warmer temperatures, a pattern known as the temperature-size rule (Sheridan & Bickford 2011; DeLong 2012; Forster, Hirst & Atkinson 2012). This is particularly important in the context of global climate change where future average temperatures are expected to rise by about 3ºC. Changes in body size ostensibly due to global climate change are already occurring in disparate taxa (Ozgul *et al.* 2009; Hoffmann & Sgrò 2011) and are expected to become more prevalent as temperature continues to increase. Because temperature

can affect both mean body size and its intraspecific variation (DeLong 2012), to properly understand how climate change may affect predator-prey interactions and, through such changes, the structure and dynamics of food webs, we need to assess the joint effects of changes in mean and variance in body size caused by warmer temperatures.

Here we assess how predator body size and its intraspecific variation affect the interaction between the predator copepod *Eucyclops agilis* and the protist prey *Paramecium caudatum*. To do so, we used foraging experiments to test whether body size affects any of the parameters of the predator functional response using foraging experiments. Second, we used mathematical models to predict the effect of variation in body size given assumed functional relationships between body size and the parameters of the functional response, and we tested those predictions against our data. Finally, we used empirical estimates of mean change in body size with temperature for copepods to predict how the interaction strength between *Eucyclops agilis* and *Paramecium caudatum* might change for different possible scenarios of future temperatures.

Copepods and protists are abundant and important consumers and prey at multiple trophic levels within aquatic food webs (Fryer 1957; Novich *et al.* 2014; Kalinoski & DeLong 2015). Copepods are generalist predators that play a central role as primary and secondary consumers in aquatic food webs and, as such, are thought to be one of the main carbon sinks in the oceans (Jónasdóttir *et al.* 2015). Copepods thus play a major role in turning over energy and matter across ecosystems, making our model system and results of relevance to understand the effect of trait-mediated temperature effects on food web structure and dynamics.

METHODS

Study system

We used the copepod *Eucyclops agilis* preying on the protist *Paramecium caudatum*. *P. caudatum* is a ~300μm long highly mobile bacterivorous protist. Protists such as *P. caudatum* are important components of aquatic food webs and are often eaten by copepods (Fryer 1957). Cyclopoid copepods like *E. agilis* are highly mobile generalist predators, capable of eating a wide range of prey items, from detritus to other cylops (Fryer 1957; Kalinoski & DeLong 2015). Both species were collected from a pond at the Spring Creek Prairie Audubon Center, ~30 km southwest of Lincoln, NE, USA (Novich *et al.* 2014). *P. caudatum* were maintained in laboratory cultures for months prior to the experiment, while *E. agilis* was collected 2 days before they were used in foraging trials and kept in pond water with food *ad libitum* at room temperature (~23°C) until experiments started.

Foraging trials

We set up foraging experiments using a factorial design with three predator densities (1, 2 and 3 ind/2ml) and five prey densities (6, 12, 18, 24 and 30 ind/2ml). Each density combination was replicated four times, for a total of 60 trials. Copepods were acclimated for 5 minutes in Petri dishes with 2 ml of filtered and autoclaved pond water (collected from the same Spring Creek Prairie site) at room temperature (~23°C) prior to the start of the trial, after which prey were introduced to the dish to begin the foraging trial. We only used adult cyclops to control for potential ontogenetic diet shifts. Foraging trials lasted for 10 minutes, after which remaining prey were counted. Trials were conducted in four consecutive days and all combinations of predator and prey levels were run once every day across four consecutive days. No copepods were used more than once.

After foraging trials, the cyclops were euthanized and photographed individually using a Leica stereomicroscope, and body length was measured, as a proxy for body size, from the beginning of the prosome to the tip of the caudal ramus. Cyclops grow in molts (Williamson & Reid 2009), so unless a molt was observed in the petri dish after a foraging trial (none were), there was no growth in body length during the 10 minute foraging trials. These individual measurements were used to calculate the standing mean and variance in body length within each trial.

The effect of mean body length on the parameters of the functional response

We used the Rogers predator equation, which is a type II functional response that takes into account prey depletion (Rogers 1972; Bolker 2011):

$$R_e = R_0 - \frac{W\left(\alpha_0 \eta R_0 e^{-\alpha_0 (t-\eta R_0)}\right)}{\alpha_0 \eta}, \quad (1)$$

where R_e is the number of prey individuals/2ml eaten in time t, R_0 is the initial prey density, α_0 is the attack rate of the predator, η is its handling time, and W is the Lambert W function, which for $f(x) = xe^x$ satisfies $W\left(f(x)\right) = x$. To incorporate interference competition, we set $\alpha_0 = \alpha C^m$ following previous studies (DeLong & Vasseur 2013), where α is the maximal attack rate in the absence of interference, and m is the coefficient of mutual interference, which typically ranges from 0 to -2, where m=0 represents the scenario where there is no interference (Arditi & Ginzburg 1989; DeLong & Vasseur 2013).

Using the estimated foraging rates across all combinations of predator and prey levels, we can estimate the parameters of the functional response in (1). Unfortunately, doing so would only yield one set of parameters (attack rate, handling time and interference), making it impossible to assess how body size and its variation affect them. To do so, we devised a procedure than makes use of the natural variation

in mean body length and its variation across foraging trials to our advantage, by bootstrapping the data from the individual foraging trials 5,000 times (i.e. sampling data points with replacement). By doing so, we generated 5,000 pseudo-replicated datasets that we used to fit equation (1), obtaining a set of parameters, (the attack rate (α), the handling time (η), and the coefficient of mutual interference (m)) from each, as well as an average body length (calculated among all data points within a bootstrap replicate), median variance in body length and day of trial. For each bootstrap replicate, we thus obtained the three parameters controlling the predator-prey interaction as well as the average and intraspecific variation in body length associated with that particular set of parameters. We disregarded any bootstrap replicate that yielded parameter estimates in the upper or lower 1% of the distribution in any one of the parameters. We used this bootstrapped data to assess how mean body size and its variation affected the parameters of the functional response.

Previous theoretical work suggested that mean body size can affect all three parameters of the functional response in specific ways (DeLong 2014). While the attack rate has been shown to scale with body mass across pairs of interacting species (Rall *et al.* 2012), whether that relationship is expected within any given pair of predator and prey species is largely unknown. Indeed, attack rates have been empirically shown to be a hump-shaped functions of predator-prey body size ratios (Vucic-Pestic *et al.* 2010; Rall *et al.* 2012). Since predator-prey body size ratios increase with predator body size, we would then expect a hump-shaped relation between attack rate and body size within species, as many theoretical studies often assume (Aljetlawi 2004; Schreiber *et al.* 2011; Gibert & Brassil 2014; Allhoff *et al.* 2015; Nonaka *et al.* 2015). We therefore model that relationship as follows:

$$\alpha(x) = \alpha_{amp} \exp\left[-\frac{\left(x-\theta_{\alpha}\right)^2}{2\tau^2}\right] + \alpha_{min}, \quad (2)$$

where α_{amp} is a shape parameter that controls the height of the function, α_{min} is the minimal attack rate, θ_{α} is the body size at which the attack rate is maximal (i.e. optimal body size), and τ controls how fast the attack rate declines with body size as we move away from the optimal value ((Gibert & Brassil 2014), Fig 2a). Interference is often linked to attack rates by a logarithmic function, $m(x) = \gamma \log(\alpha(x)) + \delta$, where γ and δ delta are system-specific (DeLong & Vasseur 2013). Using the bootstrapped data, we found that relationship to be $\gamma = -0.56 \pm 0.0057$ SE and $\delta = -0.38 \pm 0.0038$ SE (Appendix IX). The attack rate therefore specifies interference, which also ends-up being hump-shaped (Fig 2b). Finally, the handling time has also been shown to be a concave-up function of predator-prey body size ratios, and thus a concave-up function of predator body size (Rall *et al.* 2012), so we modeled it as:

$$\eta(x) = \eta_{max} - \left(\eta_{max} - \eta_{min}\right) \exp\left[-\frac{\left(x-\theta_{\eta}\right)^2}{2\nu^2}\right], \quad (3)$$

where η_{max} controls the height of the function, η_{min} is the minimal handling time, θ_{η} is the body size at which the handling time is minimal (i.e. optimal body size), and ν controls how fast the function declines with body size way from the optimal value ((Gibert & Brassil 2014), Fig 2c). To test how mean body size affects the parameters of the functional response, we fitted (2) and (3) to the bootstrapped data and then compared those fits to Generalized Additive Mixed Models (GAMM) with the parameter of interest as a response variable (e.g. attack rate), mean body length as the explanatory variable and day as a mixed effect. Additive models are useful in this way because they make no assumptions about the functional form of the relationship

between the parameters and either mean body size or variation in body size. A close match between our theoretical model and the GAMMs would suggest that our model captures in some way the effect of mean body size and its variation on the parameter of interest. Notice that the effect of interference is specified by (2). Thus, by fitting 2, we can simply parameterized the logarithmic relationship described above and compare that to the respective GAMM.

The effect of variation in body length

Theory predicts that if there is a non-linear functional relationship between a trait and an ecological process, we should expect an effect of variation in that trait *per se* on the process (Bolnick *et al.* 2011; Gibert & Brassil 2014). Thus, fitting the models described in the previous section would show whether such an underlying nonlinear functional relation exists between body length and the parameters of the functional response. In what follows we show how the effect of variation in body length can be assessed given the underlying relationship with mean body length. To do so, we assumed body length (x) to be normally distributed

$$p(x,\bar{x}) = \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left[-\frac{(x-\bar{x})^2}{2\sigma^2}\right], \tag{4}$$

where $\bar{x}$ is the mean body length, and σ^2 is its variance. Then, following previous theoretical work (Gibert & Brassil 2014; Gibert & DeLong 2015), the mean attack rate can be found as:

$$\bar{\alpha}(x) = \int_{-\infty}^{+\infty} \alpha(x) p(x,\bar{x}) dx . \tag{5}$$

Equation (5) convolves nicely into:

$$\bar{\alpha}(x) = \frac{\alpha_{amp}\tau}{\sqrt{\tau^2+\sigma^2}} \exp\left[-\frac{1}{2}\frac{d_\alpha^2}{\tau^2+\sigma^2}\right] + \alpha_{min} , \tag{6}$$

which is a function of variation in body length (σ^2), and where $d_\alpha^2 = (\theta_\alpha - \bar{x})^2$, hereafter referred to as phenotypic mismatch, increases as the predator becomes less effective at taking down that particular prey ((Gibert & Brassil 2014), Fig 2d). The effect of variation in mutual interference (m) can be found through:

$$\bar{m}(x) = \int_{-\infty}^{+\infty} \left[\gamma \log(\alpha(x)) + \delta\right] p(x, \bar{x})\, dx\,. \tag{7}$$

which can be solved numerically. Finally, the effect of variation in body length on the handling time can be found through:

$$\bar{\eta}(x) = \int_{-\infty}^{+\infty} \eta(x) p(x, \bar{x})\, dx\,, \tag{8}$$

which becomes

$$\bar{\eta}(x) = \eta_{max} - \frac{\nu\left(\eta_{max} - \eta_{min}\right)}{\sqrt{\nu^2 + \sigma^2}} \exp\left[-\frac{1}{2}\frac{d_\eta^2}{\nu^2 + \sigma^2}\right], \tag{9}$$

where $d_\eta^2 = (\theta_\eta - \bar{x})^2$ is the phenotypic mismatch with respect to handling time. The predator becomes less effective at handling a particular prey as the mismatch increases ((Gibert & Brassil 2014), Fig 2f). Using the fits between mean body size and the parameters of the functional response (i.e. equations (2), (3) and $m(x)$), we parameterized (6), (7) and (9) to predict what the effect of variation in body length should be on the parameters of the functional response. We then tested how well those predictions did against GAMMs where each parameter was the response variable, and standing variation in body length was used as an explanatory variable with day as a random effect.

The effect of temperature

Freshwater organisms show, on average, between 1% and 2.5% decrease in body size with temperature per ºC (Forster *et al.* 2012). Also, as mean body size

decreases, variation in body size decreases as well (DeLong 2012). Taking this into account, it is possible to assess how a change in mean body size and its variation may affect the mean interaction strength (*IS*) between *E. agilis* and *P. caudatum*. To do so, we use foraging rates as a proxy for interaction strengths (Laska & Wootton 1998; Novak & Wootton 2008), and write:

$$\overline{IS} = \int_{-\infty}^{+\infty} \frac{\alpha(x)\eta(x)RC^{1+m(x)}}{1+\alpha(x)\eta(x)RC^{m(x)}} p(x,\overline{x})dx\,, \qquad (10)$$

which can be parameterized using the fits from equations (2) and (3) as well as the logarithmic relationship between attack rate and interference (*m(x)*). Equation (10) simply is the average foraging rate of the copepod assuming a type-II functional response and interference competition, that takes into account the effect of mean body size and its variation (Gibert & DeLong 2015). Assuming no changes in predator or prey densities, we assessed two different scenarios for the effects of size change on interaction strength: a scenario where only mean body length changed with temperature for *E. agilis* and a scenario where both body length and its variation changed. The size of the prey remained constant, simulating a situation where there is an asymmetrical response to temperature for predators and prey as suggested by previous studies (Dell *et al.* 2014).

RESULTS

Both average body length and standing variance in body length varied widely across foraging trials (Fig 3a,b). The fitted functional response has parameters α =4.35 (CI: 1.31, 7.38), η =0.026 (CI: 0.013, 0.039) and *m*=-1.14 (CI: -1.62, -0.65, Fig 3c), which implies that *E. agilis* showed rather large levels of interference competition (literature mean for *m*=–0.8, (DeLong & Vasseur 2013)). Mean body size

nonlinearly affected all three parameters of the functional response (GAMM smooth components $p_{\text{attack rate}} = 1.48*10^{-8}$, $p_{\text{interference}} = 3.87*10^{-14}$, $p_{\text{handling time}} = 0.0107$), and there is good agreement between the model fits and GAMMs (Fig 4a-c). Both attack rate and interference mostly decline with body size in the range observed (Fig 4a,b), while handling time decreases at first and then increases (Fig 4a,b). The body size at which attack rate peaks (~1.34mm) is slightly different from that at which handling time is minimal (~1.38mm).

GAMM models showed a nonlinear effect of variation in body length upon attack rate (smooth term $p_{\text{attack rate}} = 0.0087$, Fig. 4d), a linear effect on interference (smooth term $p_{\text{interference}} = 0.133$, Fig. 4e) and no effect on handling time (smooth term $p_{\text{handling time}} = 0.85$, Fig. 4f). With respect to the predicted effects of variation from (6), (7) and (8), we predicted a shallow hump-shaped function of variation on attack rate, however, GAMM fits suggest a much stronger hump-shaped relationship than we predicted, despite some visible overfitting (Fig 4d). For interference, our model predictions and the GAMM are in agreement at first, but while the model predicted a slight decrease at larger levels of variation, GAMM suggests an increase in interference throughout (Fig 4e). Both our model and the GAMM show no change with variation for handling time, but the theoretical model predicts much lower handling times throughout (Fig 4f).

Last, our results show that a change in mean body size due to temperature can have important effects on interaction strengths, increasing them at first and then decreasing them (Fig 5, solid line). When a change in both mean body length and variation is taken into account, the increase and the decrease in interaction strengths are exaggerated (Fig 5, dashed line).

DISCUSSION

While the importance of body size for predator-prey interactions has long been recognized (Pimm, Lawton & Cohen 1991; Jonsson, Cohen & Carpenter 2005; Brose *et al.* 2006), its joint effect with variation in body size and how this might be affected by temperature is largely unknown. Here, we show that both mean body size (Fig 4a-c) and intraspecific variation in body size (Fig 4d-f) play a major role in setting the parameters of the functional response in this freshwater predator-prey system. We also show that, by taking this information into account, we can predict how increasing temperatures associated with global climate change may affect the interaction strength between this pair of species (Fig 5). We expect an increase in average temperature to increase interaction strengths at first, then to decrease them, and this effect is stronger if the change in body size induced by increasing temperatures is accompanied by a change in its intraspecific variation (Fig 5).

The ecological effects of phenotypic variation are increasingly recognized as important, yet they are still largely overlooked for predator-prey dynamics (Benedetti-Cecchi 2003; Bolnick *et al.* 2011; Gibert *et al.* 2015). Evidence showing important effects of phenotypic variation *per se* on ecological interactions is, however, rapidly increasing (Ingram *et al.* 2011; Hughes *et al.* 2015; Snowberg *et al.* 2015; Cronin *et al.* 2016), as is that of genetic variation (Agashe 2009; Steiner & Masse 2013). Our results add to this growing literature by showing that we can empirically detect the effect of phenotypic variation in predator body size on foraging rates with a simple experimental approach. Moreover, we show that predicting the effect of phenotypic variation is possible, and these predictions are qualitatively and quantitatively robust, despite some disagreement with predictions from our statistical models. We believe that the effect of phenotypic variation in the parameters of the functional response

may be weaker than that of mean body size. Our rather small sample size may thus partly explain the larger levels of disagreement observed between our model predictions and the data for the effect of variation on functional response parameters (Fig 4d-f), compared to the quite tight agreement between theory and data for mean body size effects (Fig 4a-c).

Body size has long been known to affect the parameters of the functional response (Vucic-Pestic *et al.* 2010; DeLong 2012; Pawar *et al.* 2012). While most studies suggest that attack rate scales positively with body size while handling time does the opposite (e.g. (DeLong & Vasseur 2012b)), whether this pattern holds within specific predator-prey systems, rather than across predator-prey pairs, is not known. Furthermore, theoretical studies often assume hump-shaped relationships between body size and some parameters of the functional response such as the attack rate (e.g. (Schreiber *et al.* 2011; Nonaka *et al.* 2015)), but empirical evidence for such a pattern is slim (but see (Rall *et al.* 2010)). Here, we show that contrary to across species expectations, the attack rate goes down with body size in the range observed, and peaks on the lower end of the body size distribution, suggesting that smaller bodies copepods (e.g. juveniles), may have lower attack rates than do adults. This pattern is consistent with previous findings in copepods as well (Novich *et al.* 2014), potentially suggesting that some of the observed patterns across species may not be valid assumptions within species.

The strength of predator-prey interactions plays an important role in determining the stability of food webs (e.g. (McCann *et al.* 1998). Temperature has been shown to affect food web body size structure (Gibert & DeLong 2014) and dynamics (Binzer *et al.* 2012), showing an interactive effect with body size (Binzer *et al.* 2015). The specific mechanisms through which this interactive effect happens,

however, are largely unknown. Here, we show that interaction strengths can depend on both mean body size and its intraspecific variation, and that this effect can be mediated by temperature. With temperature regulating body size and its variation through the temperature-size rule (Atkinson 1994; DeLong 2012), our results suggest a potential mechanism through which temperature may lead to important shifts in food web body size structure and dynamics. Furthermore, we show how it is possible to predict future interaction strengths between *E. agilis* and *P. caudatum* under different scenarios. We believe that approaches like ours, firmly rooted in the natural history of the study system and coupled with novel theoretical approaches, are the key to understanding and predicting how natural ecosystems may be affected by increasing temperatures worldwide.

In conclusion, we designed simple foraging experiments using a copepod-protist predator-prey system to show that body size and its intraspecific variation play an important role in setting the parameters of the functional response, consistent with what our theoretical expectations. Furthermore, we predicted how increasing temperature may affect the interaction strength between the pair of species through changes in both mean and variation in body size. These results have important implications for the structure and stability of food webs as climates change.

ACKNOWLEDGMENTS

We are indebted to Stefano Allesina, Diana Pilson and Richard Rebarber for insightful suggestions about experimental design and data analysis. We also are very grateful with Chad Brasil, whose suggestions were instrumental for the clarity of the text and figures. We thank Janet Reid for helping us identify the species of copepod we worked with. JPG was funded by an NSF Doctoral Dissertation Improvement

Grant (DEB-1501668), JPD was funded by a Binational Science Foundation grant (# 2014295).

FIGURES

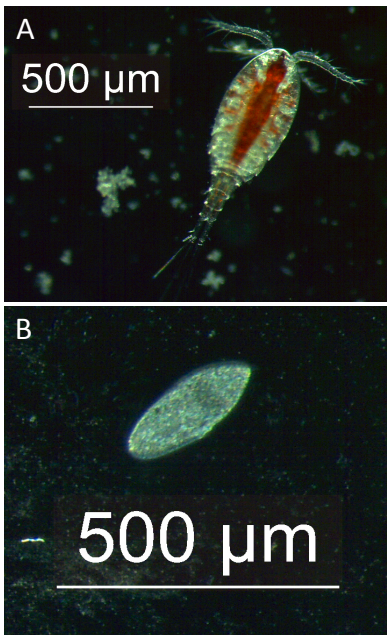

Figure 1: A. *Eucyclops agilis*. B. *Paramecium caudatum*

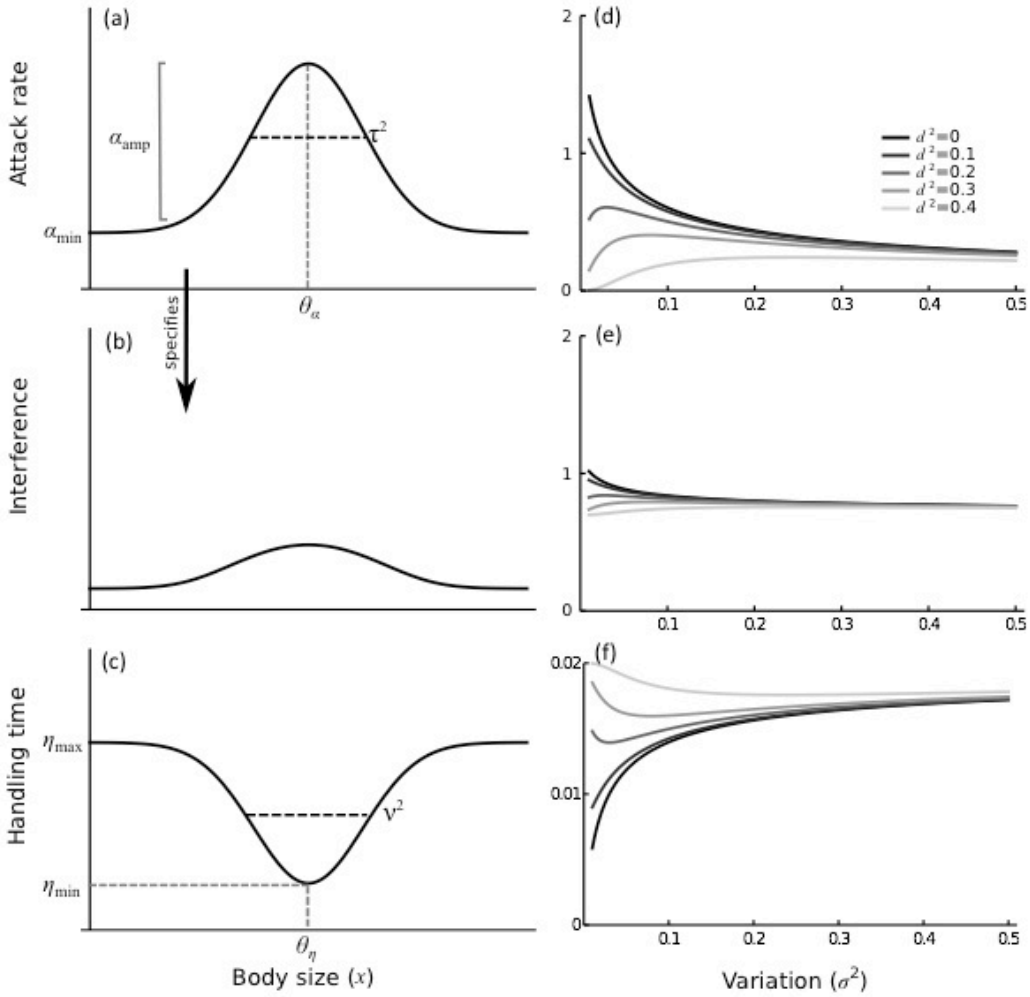

Figure 2: (a) The assumed relation between body size and attack rate. (b) How interference depends on body size given the logarithmic relation with attack rate. (c) How handling time depends on body size. (d) The effect of intraspecific variation in body size on attack rate for different values of phenotypic mismatch (d_α^2). (e) Same as in (d) but for interference competition. (f) The effect of variation in body size on handling time for varying phenotypic mismatch (d_η^2).

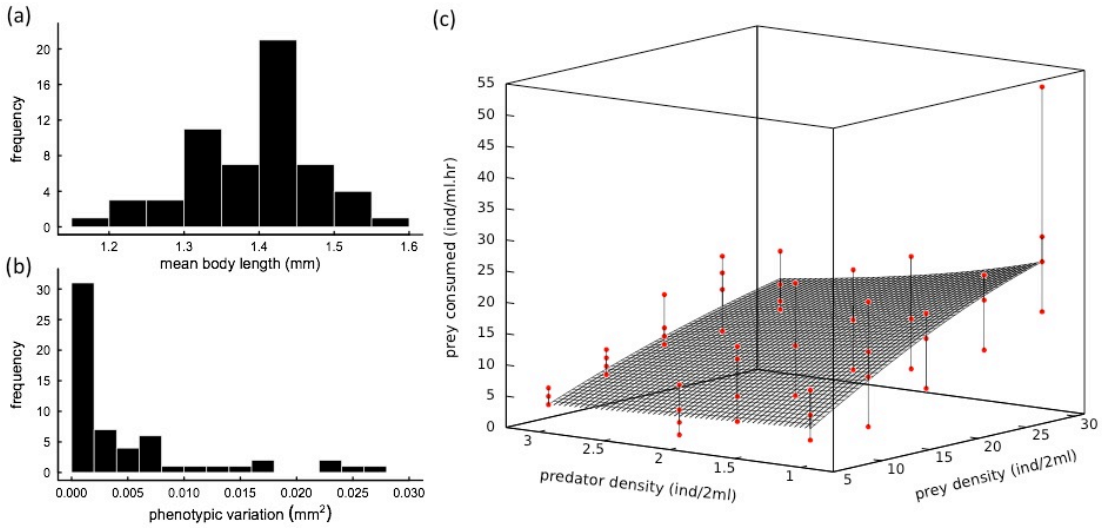

Figure 3: (a) Distribution of average body sizes across all 60 foraging trials. (b) Distribution of variation in body size across trials. (c) Observed foraging rates (prey consumed/ml.hr) as a function of predator (ind/2ml) and prey density (ind/2ml). Red dots represent the measured forging rate for each trial, and the grey surface is the fitted functional response (equation (1), parameters: α=4.35 (CI: 1.31, 7.38), η =0.026 (CI: 0.013, 0.039) and m=-1.14 (CI: -1.62, -0.65)).

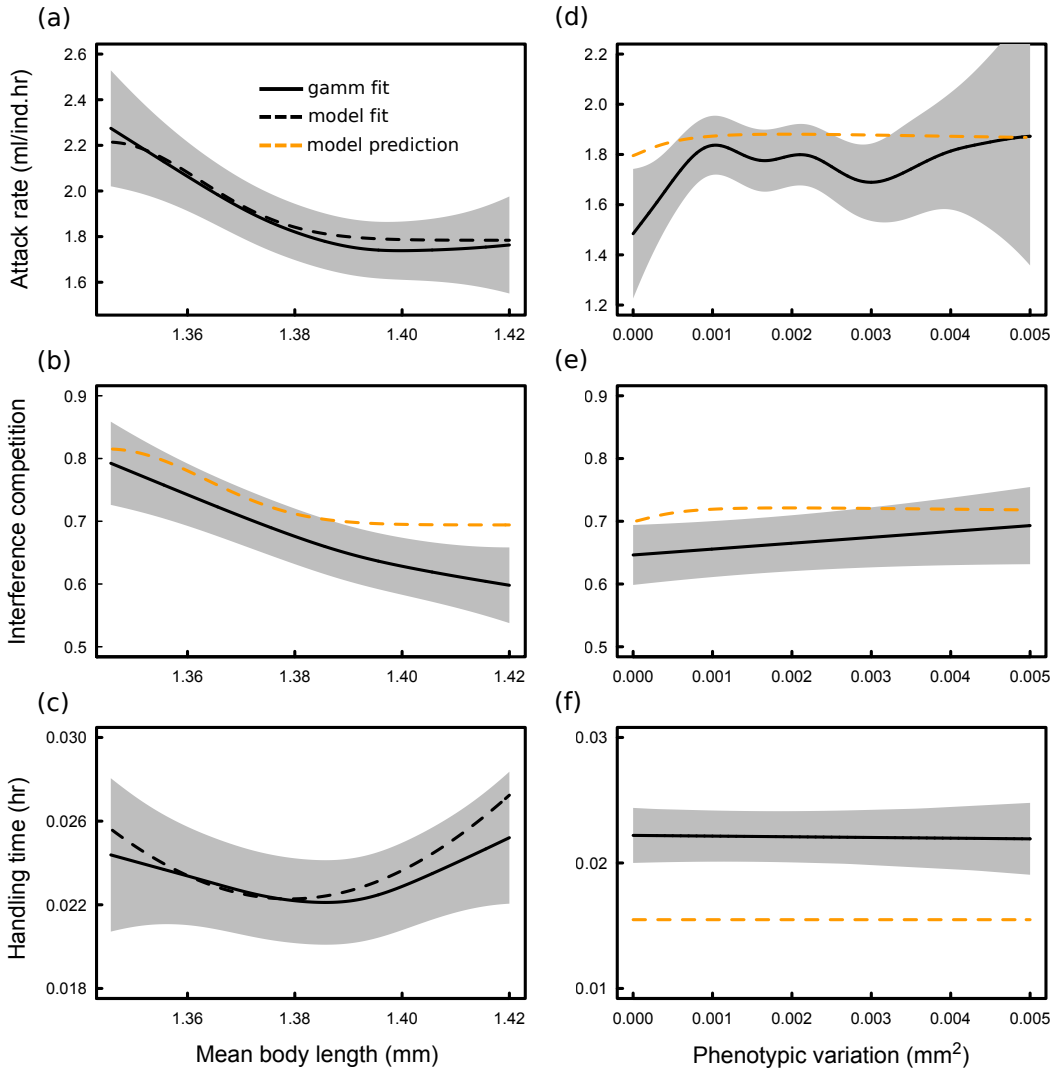

Figure 4: (a)-(c) Dashed black line represents the fit of the model, while the solid line represents the prediction of the GAMM with its 95% confidence interval in grey. In (b), the dashed line is orange to indicate that the relationship was no fitted, but predicted from the fit of equation (1) in (a). (d)-(f) Dashed orange lines represent the predicted effect of variation in body length given the relationships in (a)-(c), and the solid lines represent the prediction of the GAMM with confidence intervals in grey. Conceptually, GAMM predictions can be seen as a way of collapsing all 4.277 data points into one line. Also, the actual data points are not shown for clarity purposes.

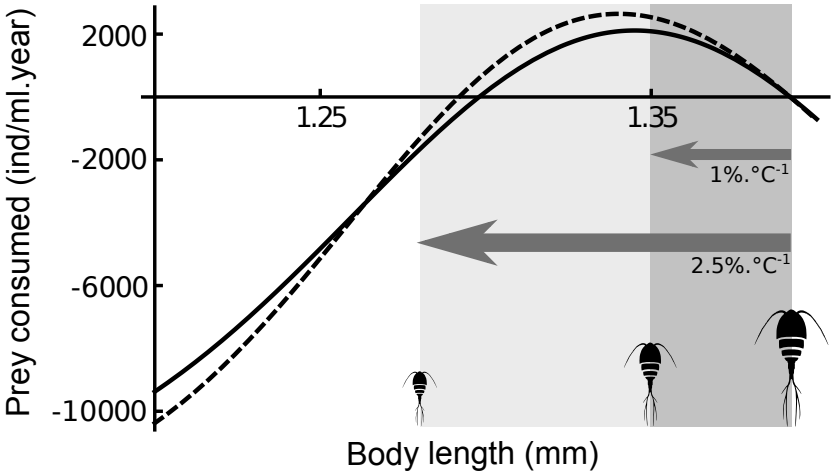

Figure 5: Predicted effect of temperature on interaction strengths (measured as prey consumed per milliliter per year) for two scenarios: change in mean body size only (solid), and a change in both mean body size and intraspecific variation (dashed). The rightmost point at which the curves intersect the x-axis represent the current average body size of *E. agilis*. Moving to the left from that point, the effect of temperature can be assessed assuming: a decrease of 1%.ºC^{-1}, or a decrease of 2.5%.ºC^{-1}, for copepods only. Black copepod silhouettes are not to scale and are there for visual guide only.

CHAPTER 5

TEMPERATURE ALTERS FOOD WEB BODY-SIZE STRUCTURE

Jean P. Gibert & John P. DeLong

Key-word: Global warming, Temperature, Food web structure, Body-size ratios, Temperature-size rule

ABSTRACT

The increased temperature associated with climate change may have important effects on body size and predator-prey interactions. The consequences of these effects for food web structure are unclear because the relationships between temperature and aspects of food web structure such as predator-prey body size relationships are unknown. Here we use the largest reported dataset for marine predator-prey interactions to assess how temperature affects predator-prey body size relationships among different habitats ranging from the tropics to the poles. We found that prey size selection depends on predator body size, temperature, and the interaction between the two. Our results indicate that 1) predator-prey body size ratios decrease with predator size at below-average temperatures and increase with predator size at above-average temperatures, and 2) that the effect of temperature on predator-prey body-size structure will be stronger at small and large body sizes and relatively weak at intermediate sizes. This systematic interaction may help to simplify forecasting the potentially complex consequences of warming on interaction strengths and food web stability.

INTRODUCTION

Body size is a fundamental trait influencing multiple aspects of species ecology, including landscape use and locomotion (Lurgi, López & Montoya 2012), energetic requirements (Brown *et al.* 2004), and prey selection (Brose *et al.* 2006). Larger organisms tend to eat larger prey, a pattern that holds across ecosystems and taxa (Memmott, Martinez & Cohen 2000; Jonsson *et al.* 2005; Brose *et al.* 2006). The ratio of predator body size to prey body size affects predator-prey dynamics (Yodzis & Innes 1992; Kalinkat *et al.* 2013), interaction strengths (Berlow *et al.* 2009; Vucic-Pestic *et al.* 2010), trophic position (Berlow *et al.* 2009; Riede *et al.* 2011), and the size structure and function of food webs (Cohen, Jonsson & Carpenter 2003). Because of this, body size is increasingly recognized as a factor influencing species persistence and the stability of complex food webs (Berlow *et al.* 2009; Yvon-Durocher *et al.* 2011; DeLong 2014).

In addition, body size often declines with rearing temperature, a pattern known as the temperature-size rule (TSR) (Atkinson 1994; DeLong 2012). The TSR is widespread (Atkinson 1994) and could potentially affect the way species interact because smaller organisms tend to eat smaller prey (Brose *et al.* 2006). It has recently been proposed that increasing temperature will decrease average body size in food webs, leading to a reduction in the number of trophic levels and overall food-web connectivity (Daufresne *et al.* 2009; Brose *et al.* 2012). Hence, temperature could have important consequences for food web stability and species persistence. Because of increased global average temperatures due to human related activities (Houghton *et al.* 1996), the challenge now is to fully uncover the relationship between body size, temperature and food web body-size structure in order to predict and respond to warming-induced changes in ecological systems. To this end, we ask whether

temperature alters the relationship between predator and prey body size using the largest known dataset compiled for aquatic food webs (Barnes *et al.* 2008).

METHODS

Data set

We used EcoData Retriever to download and prepare the dataset (Morris & White 2013). The data consists of 34 941 observations of predator-prey interactions from 27 locations, including shoreline to open ocean ecosystems from the poles to the tropics with different mean annual temperatures measured at sea level (Barnes *et al.* 2008, 2010). The data include 93 different types of vertebrate and invertebrate predators ranging from 0.1 g to 415 kg, and 174 different types of vertebrate and invertebrate prey from 10^{-15} kg to 5 kg. In some cases, the original dataset had mass estimates derived from body length measurements (Brose *et al.* 2006; Barnes *et al.* 2008). Temperatures were included as average temperature by location measured at sea level (Barnes *et al.* 2008).

Data analysis

Because a previous study analyzing this same dataset failed to find an effect of temperature, in order to assess the effect of temperature on the relationship between predator body mass and prey body mass, we compared three different linear mixed effects models aimed at controlling for the hierarchical structure of the data (package lme4 in R (Bates, Martin & Bolker 2011)). We log-transformed both predator and prey body sizes before analysis. The first model included prey body size as the response variable and predator body size as the predictor variable, with habitat type as a random intercept and predator identity (species) as a random slope. This also helped control for the error associated with the allometric estimates of predator body mass.

The second model also considered the additive effect of temperature, with random effects as in the first model. The third model considered the interactive effect of predator body mass and temperature, with random effects as before. We selected the most plausible model using Akaike's information theoretical criteria (Burnham & Anderson 2002). Finally, we compared the relationship between predator body mass and prey body mass with simple ordinary least squares (OLS) and reduced major axis (RMA) regression. RMA regression allows for error in the x-axis variable, so this comparison would allow us to determine whether accounting for error in predator mass estimates would qualitatively change our results. Since it did not, we report only the results from the linear mixed models.

RESULTS

The best model suggests that prey size increased with predator size, and that effect is temperature dependent (intercept = -10.66 ± 1.43SE, slope = 0.43 to 1.43 ± 0.16SE, table 1). In short, prey size increases with predator size and temperature increases the intercept of the relationship (+0.33 ± 0.03SE per degree ºC) but decreases its slope (-0.04 ± 0.01 per ºC). Hence, smaller predators tend to eat relatively larger prey at warmer temperatures than at lower temperatures, while the reverse was true for larger predators (Fig. 1). Note that a slope close to one implies that body-size ratios remain constant across the entire range of predator masses. In contrast, a slope < 1 indicates an increase in the ratios, and a slope > 1 indicates a decrease. Thus, our best model indicated that prey size depended on the interaction between temperature and predator body size (Table 1, Fig. 1). The cut-off at which the effect of temperature gets reversed is somewhere between a predator mass of 10g and 150g.

DISCUSSION

Consistent with previous studies, our results show that prey size increases with predator size (Memmott *et al.* 2000; Jonsson *et al.* 2005; Brose *et al.* 2006). Unlike previous studies (Barnes *et al.* 2010), however, we show that this relationship depends on the interaction between temperature and predator body size, as the slope of the curve becomes shallower and the intercept gets larger as temperature increases (Fig. 1). The difference between our results and previous analyses with this data (Barnes *et al.* 2010) may simply be due to the fact that the previous analysis only controlled for the effect of location and not for the hierarchical structure of the data in terms of temperature across sites. We do not believe our results contradict their main conclusions, but they rather add an extra layer of understanding as to how predator body size and temperature can interact to yield particular body size ratios in any given location. The magnitude of the temperature effect changes with habitat, but the direction of the effect does not, indicating some generality across sites (Fig. 1). Although there is error in the estimates of body size for both predator and prey, and we were only able to consider average temperatures, our broad scale analysis clearly reveals that body size and temperature can have strong interactive effects on food web body size structure.

There are three important consequences of this change in body-size structure. First, the range of prey body sizes is narrower in warm habitats than in cold habitats (Fig. 1). Second, because trophic level increases with body size (Cohen *et al.* 2003; Riede *et al.* 2011) and temperature affects body size through the TSR (Daufresne *et al.* 2009; Gardner *et al.* 2011), the trophic level of some species may vary across temperatures. In warmer habitats, larger species may have down-shifted trophic levels while smaller species may have raised trophic levels, potentially decreasing the total

number of trophic levels in warmed food webs (see also (Brose *et al.* 2012)). Finally, species at intermediate trophic levels, which are those of intermediate body size, would be the least affected by this body size-temperature interaction. Importantly, warming affects the size of predators and their prey. Thus, to actually change the body-size structure of food webs, warming must have a differential effect on predator and prey size, with predators becoming smaller at a faster pace than their prey. There is yet to be any experimental evidence suggesting that this can happen in nature, although this pattern can be obtained through a differential effect of warming in predator and prey mobility (Lurgi *et al.* 2012), which has been in turn shown to greatly affect food web network structure (Gravel *et al.* 2013; Albouy *et al.* 2014).

The effect of temperature on the predator-prey body-size scaling may also influence interaction strengths and food web stability. Interaction strengths are relatively large at higher trophic levels because they increase with body mass, which increases with trophic level (Cohen *et al.* 2003; Berlow *et al.* 2009; Riede *et al.* 2011). Our results suggest that, with warming, larger species at higher trophic levels may eat relatively smaller prey, so these prey could experience larger interaction strengths than they would at colder temperatures. The opposite may be true for smaller predators. It has also been shown that the effect of temperature on interaction strengths depend upon asymmetries in the underlying parameters of the predator-prey interaction (Gilbert *et al.* 2014), which are often controlled by body-size (DeLong 2012). Although there are many ways in which temperature may affect interaction strengths, and the temperature variation we report reflects spatial variation rather than warming, our results suggest that the potential effects of warming upon trophic interaction strengths may be trophic-level dependent.

The link between temperature and body-size structure might be related to species identity across habitats, to differences in the way prey-selection occurs between species of different habitats (Brose *et al.* 2006) or to range shifts with temperature (Lurgi *et al.* 2012; Albouy *et al.* 2014). Finally, it can also be due to body-size changes of species occurring in different habitats due to differences in environmental temperatures (Atkinson 1994; Daufresne *et al.* 2009; Gardner *et al.* 2011). If this is the case, smaller predators might be getting smaller with temperature, displaying the typical TSR pattern (Fig. 2). Large predators, however, might be getting larger with temperature (Fig. 2). Alternatively, smaller prey might be getting larger with temperature and larger prey might be getting smaller (Fig. 2). More focused analysis on body size and species identity across food webs at different temperatures are needed to tease this apart.

It is not clear why predator-prey body sizes scale the way they do in any system. In aquatic ecosystems, such as the ones analyzed here, gape-limitation may play an important role constraining food web body-size structure (Arim *et al.* 2007). If this is a driving mechanism, our results suggest that gape-limitation may be less important in warmer temperatures, as the slopes of the curves are shallower. Our results also suggest the possibility that there are limits to the slopes of these relationships, as the range of slopes observed across temperatures in this study matches the range observed across taxa, which varies from 0.5 for protists (DeLong & Vasseur 2012b) to 1.5 for mammalian terrestrial carnivores (DeLong & Vasseur 2012a), and habitats, where it varies from 0.7 in stream food webs to about 2 in terrestrial food webs (Riede *et al.* 2011).

Overall, our results suggest that temperature has an interactive effect upon predator-prey body-size relationships, where smaller predators tend to eat larger prey

at warmer temperatures and smaller prey at colder temperatures, while larger predators will do the opposite. This might lead to food webs with larger interaction strengths but fewer trophic levels in warm temperatures, while smaller interaction strengths and more trophic levels could be expected in colder food webs. Thus, we have shown that temperature has strong consequences for food web body-size structure, and very likely stability as well, which in turn has important implications for species persistence in the context of global warming.

ACKNOWLEDGEMNTS

We are indebted to Chad Brassil, Diana Pilson, Stefano Allesina, Dominique Gravel and an anonymous reviewer for valuable feedback on earlier versions of this manuscript. JPG was supported through an Othmer Fellowship and the School of Biological Sciences Special Funds.

FIGURES & TABLES

Table 1: Model selection for the mixed effects linear models

Model	K	AICc	Delta_AICc	AICcWt
log(prey mass) ~ log(predator mass) * temperature	8	153682.8	0.00	1
log(prey mass) ~ log(predator mass) + temperature	7	153839.2	156.40	0
log(prey mass) ~ log(predator mass)	6	153859.1	176.32	0

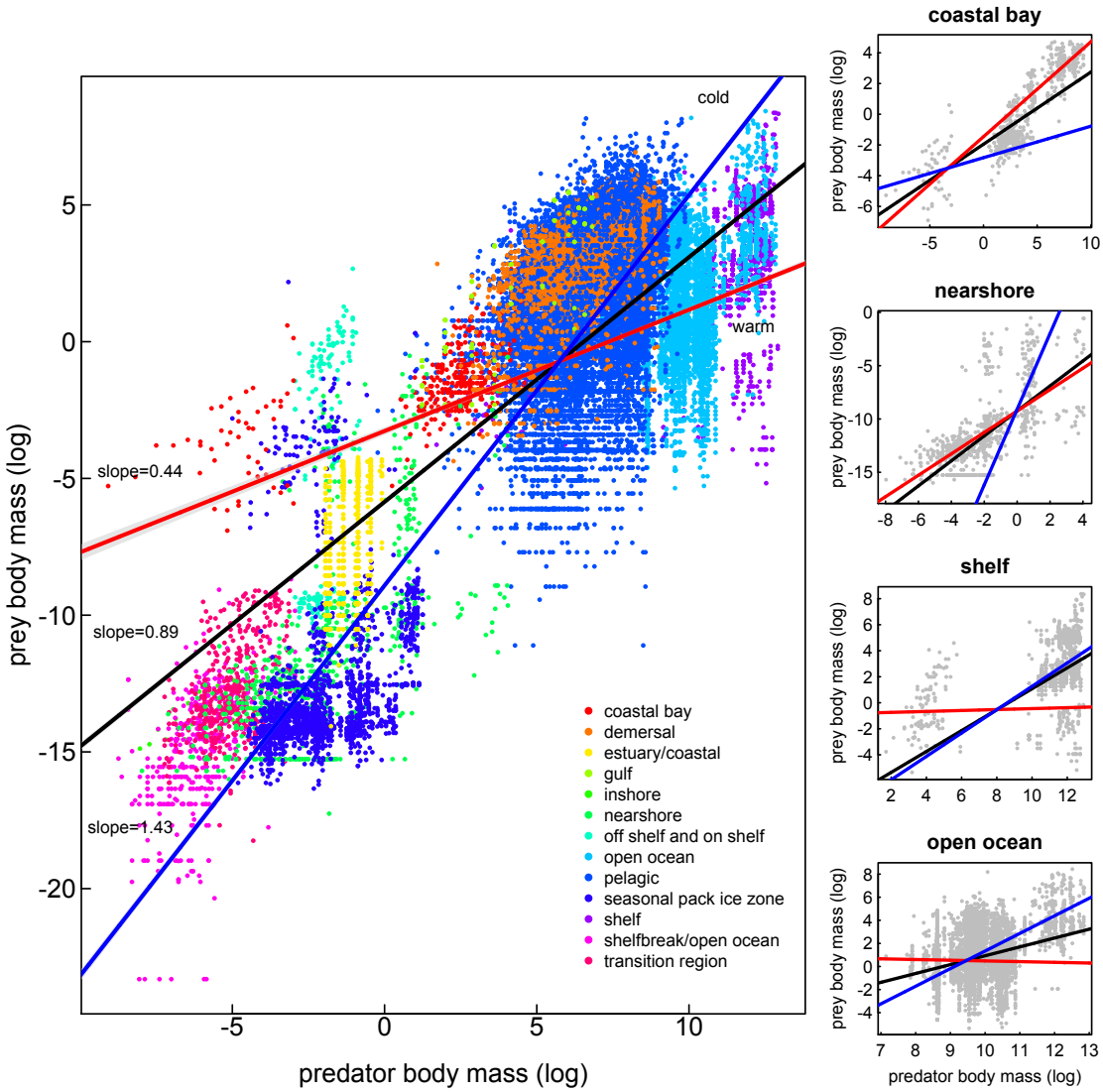

Figure 1: Left; prey body size (log) against predator body size (log) across marine habitats. Red (Tº=29ºC), black (Tº=15ºC) and blue lines (Tº=-1.3ºC) represent predicted curves from the best mixed effects linear model. 95% confidence intervals are displayed in grey. Right; same as in left for a subset of the habitats studied (coastal bay is not significant).

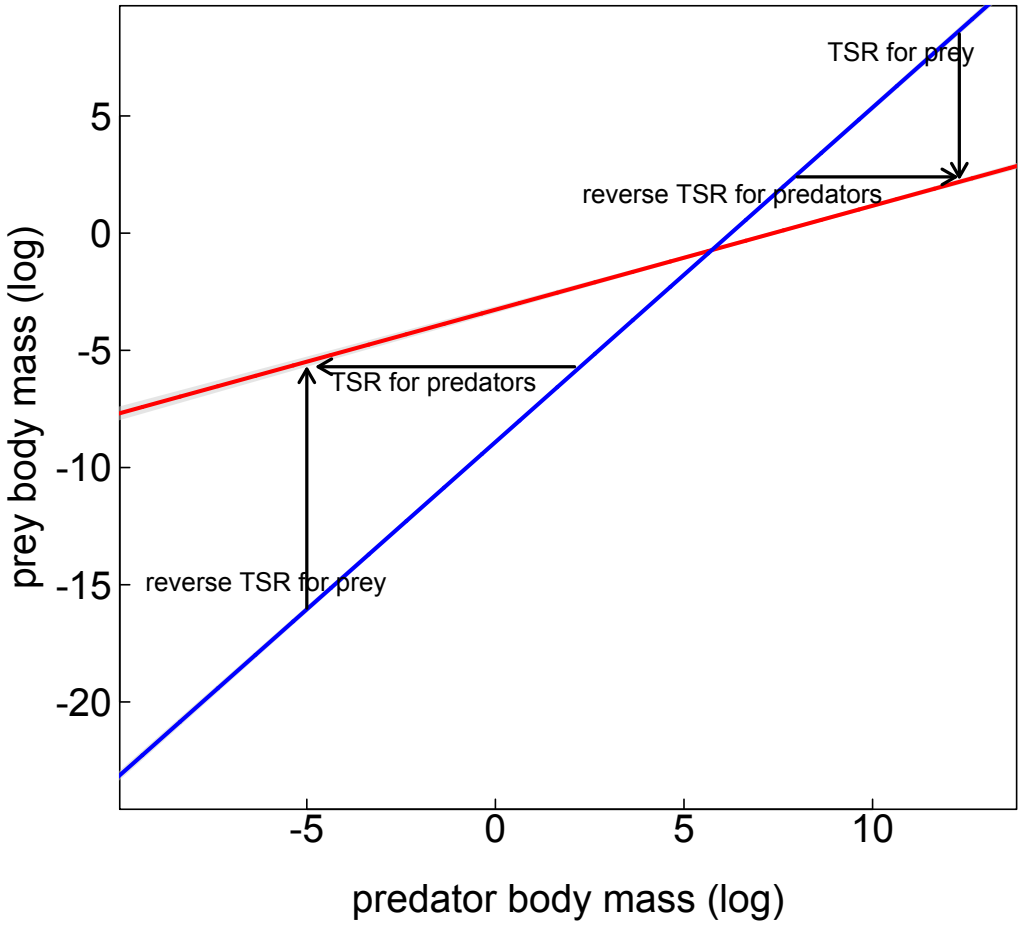

Figure 2: The effect of temperature on prey and predator body size. Red and blue lines represent the slope of the predator-prey body size relationship for warm (red) and cold (blue) temperatures. Black arrows represent body size changes with temperature.

APPENDIX I

EXPLORING PARAMETER SPACE

In this section we assess how robust our results are to a change in parameter values. We did so by exploring other possible values for d_α, d_η, τ and ν. For changes in d_α and d_η our qualitative results hold, but an increase in d_η seems to have a less pronounced effect than one in d_α (Fig. S1-1; also see Appendix 3). As τ and ν increase, the effect of individual variation decreases (Fig. S1-2). This occurs because the attack rate and the handling time become constant, and largely independent of the value of the controlling trait. Small τ or ν leads to a large dependency of the attack rate and the handling time upon the underlying trait value, and hence, to an increased effect of individual trait variation (Fig. S1-3).

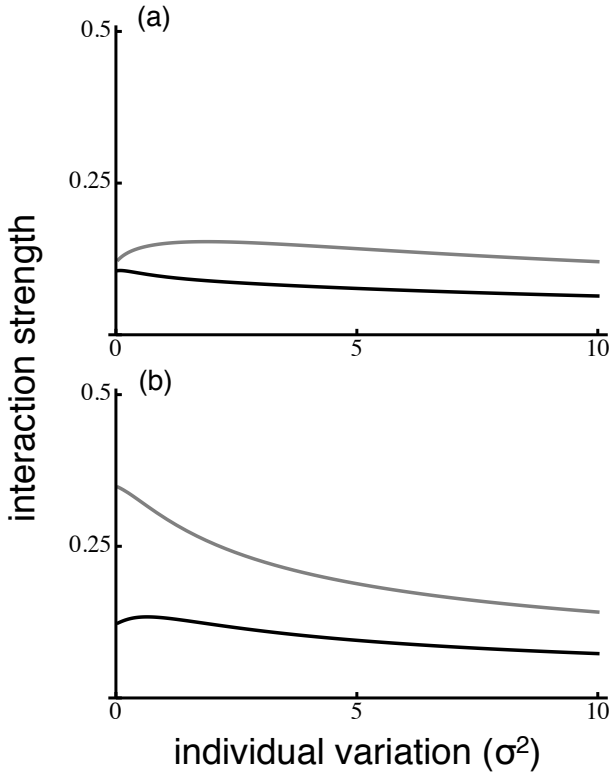

Fig S1-1: Plots of interaction strength against increasing individual variation (gray: resource, black: consumer). (a) $\alpha = 1,\ \eta_{\max} = 2,\ \eta_{\min} = 1,\ \tau = 3,\ \nu = 1,\ d_\alpha = 2,\ d_\eta = 0.$ (b) same as (a) but for $d_\alpha = 0,\ d_\eta = 2.$

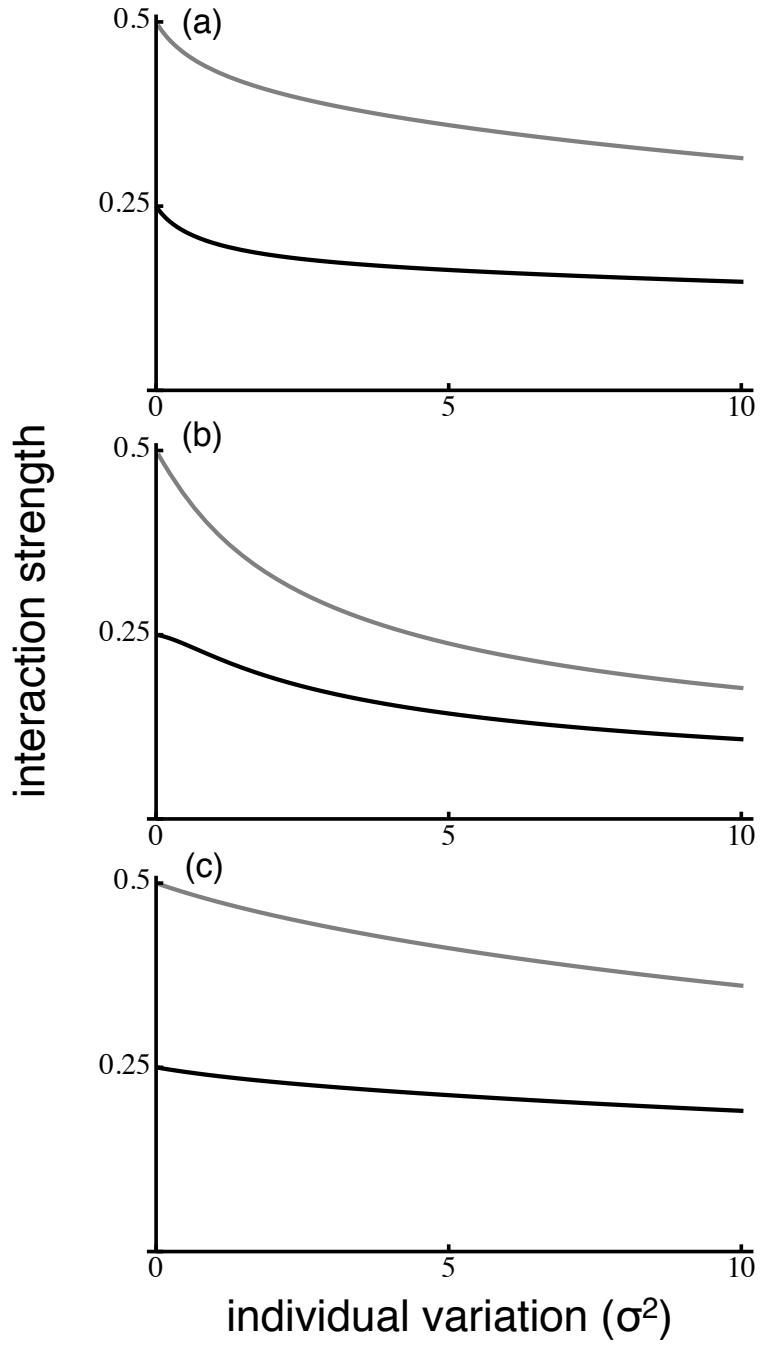

Figure S1-2: Plots of interaction strength against individual variation measured as σ^2 . Parameter values: (a) $\alpha = 1,\ \eta_{\max} = 2,\ \eta_{\min} = 1,\ \tau = 3,\ \nu = 1,\ d_\alpha = 0,\ d_\eta = 0$. (b) same as (a) but for $\tau = 1,\ \nu = 3$. (c) same as (a) but for $\tau = 3,\ \nu = 3$.

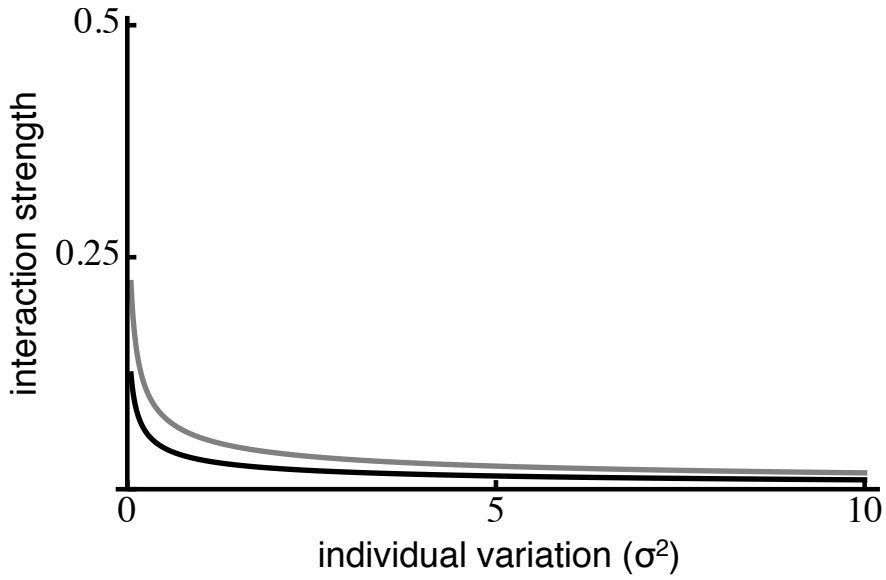

Figure S1-3: Plots of interaction strength against individual variation measured as σ^2 . Parameter values: $\alpha = 1,\ \eta_{\max} = 2,\ \eta_{\min} = 1,\ \tau = 0.1,\ \nu = 0.1,\ d_\alpha = 0,\ d_\eta = 0.$

APPENDIX II

MEAN ATTACK RATE AND MEAN HANDLING TIME

In what follows we show how the mean attack rate and the mean handling time change with increasing levels of individual variation. While attack rate decreases with individual variation whenever phenotypic mismatch is small, handling time increases (Fig. S2-1a). When phenotypic mismatch is large, however, attack rate increases at first with variation and then decreases, and the opposite is true for handling time (Fig. S2-1b).

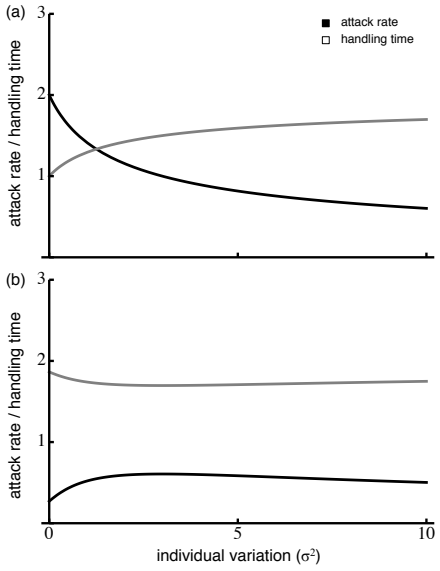

Figure S2-1: Plots of how mean attack rate (black) and mean handling time (grey) change with individual variation under small phenotypic mismatch (a) and larger phenotypic mismatch (b). Parameter values: (a) $\alpha_{\max} = 2,\ \eta_{\max} = 2,\ \eta_{\min} = 1,\ \varepsilon = 0.5,\ \tau = 1,\ \nu = 1,\ d_\alpha = d_\eta = 0$; (b) same as in (a) but for $d_\alpha = d_\eta = 2$.

APPENDIX III

ELASTICITY

The elasticity is a measure of model sensitivity defined as the absolute value of $\partial \log(f)/\partial \log(a)$, where f is the function of interest (interaction strength in this case), and a is the parameter of interest (attack rate or handling time in this case). The larger the elasticity, the more sensitive the function is to a change in the parameter.

The effects of individual variation upon consumer-resource dynamics seem to be mainly driven by variation in the attack rate, as its elasticity is generally larger than that of the of handling time regardless of phenotypic mismatch or individual variation (Fig. S3-1). Although Jensen's inequality predicts opposite effects of variation in attack rate and handling time when considered independently (Fig. 1a, 1b), interaction strengths incorporating individual variation in both attack rate and handling time simultaneously seem to mainly be affected by variation in attack rate.

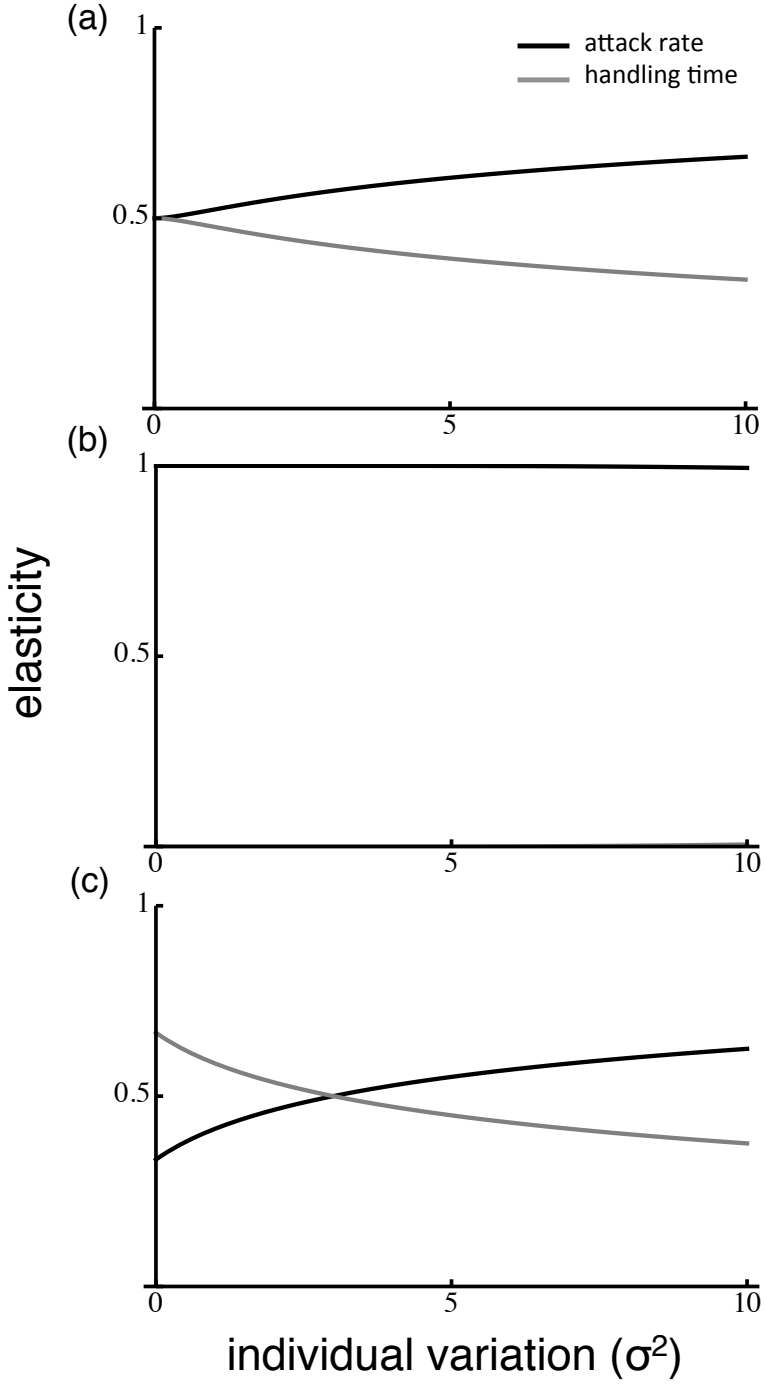

Fig S3-1: Plot of the elasticity of the interaction strengths for with respect to the attack rate (black) and the handling time (gray). (a) $\alpha = 1,\ \eta_{\max} = 2,\ \eta_{\min} = 1,\ \tau = 1,\ \nu = 1,\ d_\alpha = 0,\ d_\eta = 0$. (b) same as (a) but for $d_\alpha = 2$. (c) same as (a) but for $d_\eta = 2$.

APPENDIX IV

ASYMMETRIC TRAIT DISTRIBUTION

In the main text we assumed the trait that controls the ecological interaction through its effect on attack rate and handling time to be normally distributed. However, the distribution of some traits is highly asymmetric and skewed (Gouws *et al.* 2011). In this section, we break this assumption by incorporating an asymmetric distribution (log-normal distribution, Fig. S4-1). We show that the effect of individual variation is not largely affected by the choice of the underlying trait distribution but the range of scenarios at which interaction strength decreases with individual variation becomes larger when asymmetry is taken into account.

Here, we assumed both attack rate and handling time to depend on the value of a log-normally distributed trait with location parameter $\bar{x}$ and scale parameter σ^2. Then its density in the population is:

$$Lp(x,\bar{x}) = \frac{1}{x\sqrt{2\pi\sigma^2}} \exp\left[-\frac{\left(\log(x)-\bar{x}\right)^2}{2\sigma^2}\right]. \quad (1)$$

Note that as both the location and scale parameter control the shape of the distribution, the variance of the distribution, and hence, individual variation, now depends on both parameters. For simplicity, we focus on the case where only σ^2 varies. We have numerically integrated $\overline{I_{R,L}(\alpha,\eta)}$ and $\overline{I_{C,L}(\alpha,\eta)}$ to find the interaction strength with varying levels of individual variation σ^2 as:

$$\overline{I_{R,L}(\alpha,\eta)} = -R\int_{-\infty}^{\infty} \frac{\alpha(x)}{1+\alpha(x)\,\eta(x)\,R}\, Lp(x,\bar{x})\, dx \quad (2)$$

$$\overline{I_{C,L}(\alpha,\eta)} = \varepsilon\, C \int_{-\infty}^{\infty} \frac{\alpha(x)}{\left(1+\alpha(x)\,\eta(x)\,R\right)^2}\, Lp(x,\bar{x})\, dx \qquad (3)$$

We found that the interaction strength has a qualitatively similar behavior with respect to individual variation than in the case with a symmetric distribution. This is, there is a range of scenarios at which the interaction strength decreases monotonically with individual variation, and a range of scenarios at which the interaction strength is maximized by intermediate values of individual variation (see main text). Indeed, there is an optimal amount of individual variation that maximizes interaction strength when trait mismatch is large, if the average trait value in the population is smaller than the selective optimum ($d_\alpha << 0$ or $d_\eta << 0$, Fig S4-2a), and this behavior is also quantitatively comparable to the one obtained with a symmetric trait distribution. The interaction strength still decreases with individual variation whenever trait mismatch is small ($d_\alpha \sim 0$ and $d_\eta \sim 0$, Fig S4-2b), but this is also true for cases where the average trait value in the population is larger than the selective optimum ($d_\alpha >> 0$ or $d_\eta >> 0$, Fig S4-2c). Thus, asymmetric trait distributions can increase the range of scenarios in which interaction strengths decreases with individual variation.

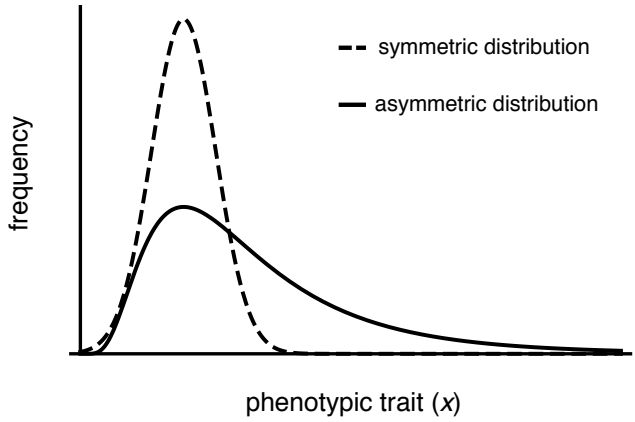

Figure S4-1: Plot of a symmetric distribution (e.g. normal) and an asymmetric distribution

(e.g. log-normal). The log-normal distribution used in the supplementary material mainly differs from the normal distribution used in the main text in that it the former is more skewed than the latter.

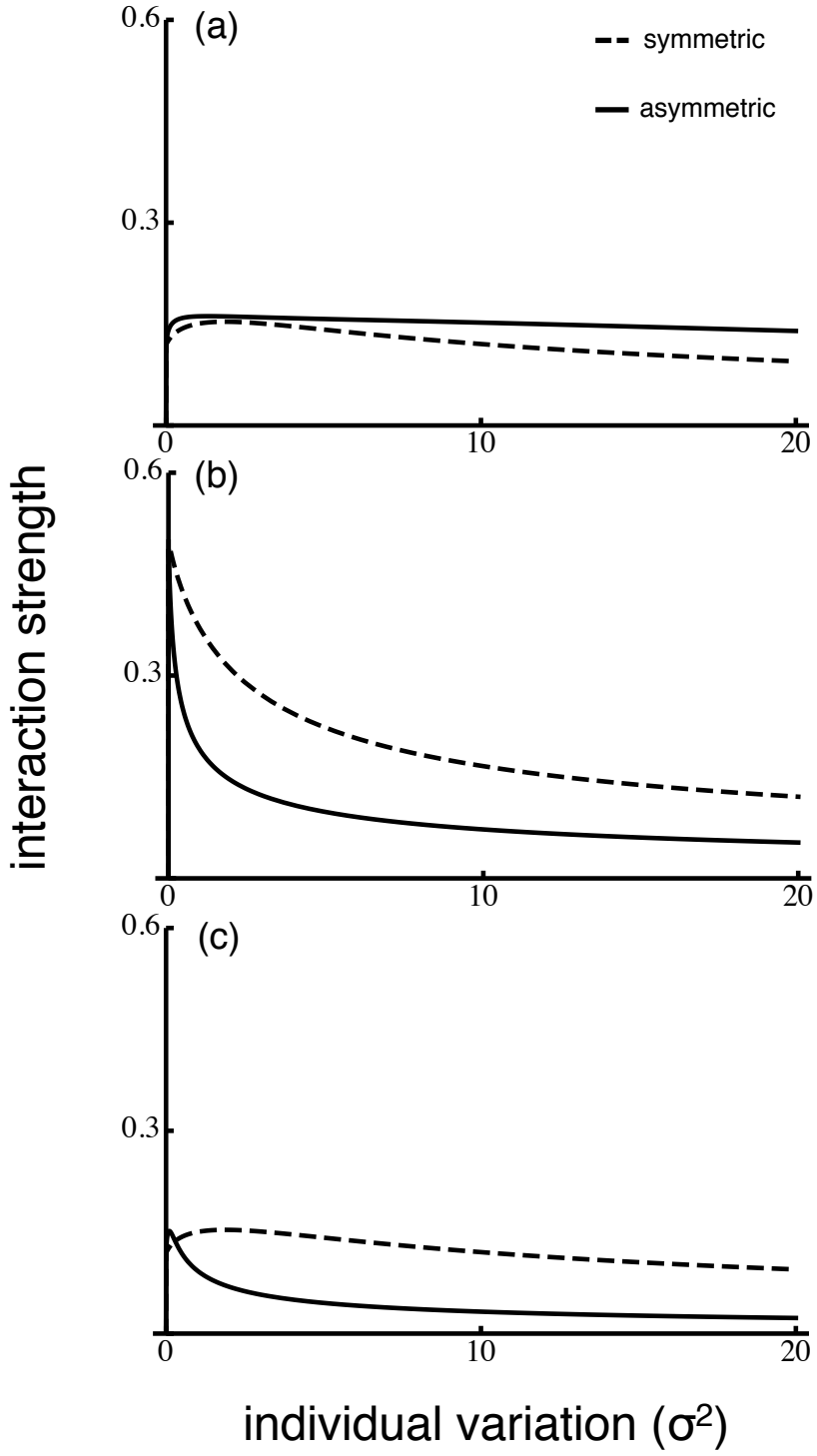

Figure S4-2: Plots of interaction strength against individual variation measured as σ^2. Phenotypic mismatch is large (a) and (c), and small in (b). Parameter values: (a) α = 1, $\eta_{\max} = 2$, $\eta_{\min} = 1$, $\tau = 1$, $\nu = 1$, $d_\alpha = -2$, $d_\eta = 0$. (b) same as (a) but for $d_\alpha = 0$. (c) same as (a) but for $d_\alpha = 2$.

APPENDIX V

ASYMMETRIC FUNCTIONAL FORMS FOR ATTACK RATE AND HANDLING TIME

In the main text, we assumed the attack rate and handling time to be non-linear, yet symmetric functional forms of the underlying controlling quantitative phenotypic trait. However, these ecological attributes could be asymmetric, as found in most thermal response curves (Vasseur et al. 2014). The asymmetry of these functional forms generally arise from important physiological or biomechanical constrains (Vucic-Pestic et al. 2010), which need to be taken into account to accurately describe the non-linear relationship between underlying phenotypic traits and the ecological attributes they influence. In this section, we break the assumption of symmetry for the attack rate and the handling time, by incorporating asymmetric functional forms (Fig. S5-1). We found that the asymmetry in attack and handling times can have a quantitative effect in the way individual variation affects interaction strengths, mostly by reducing the range of possible scenarios in which interaction strength decreases monotonically with increasing individual variation.

The now asymmetric predator's attack rate, $\alpha_{asymm}(x)$, can be assumed to be maximal at a given optimal trait value $x = \theta_\alpha$, and to decrease away from that maximum at a different rate depending on the direction. Such a scenario can be modeled by:

$$\alpha_{asymm}(x) = \alpha_{\max} - \alpha_{\max} \exp\left[-\frac{\left(\log(x) - \log(\theta_\alpha) \right)^2}{2\tau^2} \right], \qquad (4)$$

where $\alpha_{\max}$ is the maximal attack rate (Fig. S5-1a) and the rest of the parameters are as described in the main text. Similarly, the predator's handling time, $\eta_{asymm}(x)$, is minimal at the given optimal value $x = \theta_\eta$, and increases away from that minimum at a different rate depending on the direction like:

$$\eta_{asymm}(x) = \left(\eta_{\max} - \eta_{\min}\right)\exp\left[-\frac{\left(\log(x) - \log(\theta_\eta)\right)^2}{2\nu^2}\right], \quad (5)$$

where $\eta_{\max}$ and $\eta_{\min}$ are maximal and minimal handling times respectively (Fig. S5-1b) and the rest of the parameters are as described in the main text. Because of the asymmetry, it is now impossible to derive analytic expressions for the mean (asymmetric) attack rate and handling times, so we have numerically integrated $\overline{I_{R,asymm}(\alpha,\eta)}$ and $\overline{I_{C,asymm}(\alpha,\eta)}$ to find the interaction strength with varying individual variation σ^2 as:

$$\overline{I_{R,asymm}(\alpha,\eta)} = -R\int_{-\infty}^{\infty}\frac{\alpha_{asymm}(x)}{1+\alpha_{asymm}(x)\,\eta_{asymm}(x)\,R}\,p(x,\bar{x})\,dx \quad (6)$$

$$\overline{I_{C,asymm}(\alpha,\eta)} = \varepsilon\,C\int_{-\infty}^{\infty}\frac{\alpha_{asymm}(x)}{\left(1+\alpha_{asymm}(x)\,\eta_{asymm}(x)\,R\right)^2}\,p(x,\bar{x})\,dx \quad (7)$$

Overall, we found that the asymmetry in attack rate and handling time seems to preclude a monotonically decreasing relation of interaction strengths with individual variation. If phenotypic mismatch is large enough and the average trait value in the population is smaller than the selective optimum ($d_\alpha << 0$ or $d_\eta << 0$), both the symmetric and the asymmetric case predict a hump shaped relationship between interaction strengths and individual variation. If phenotypic mismatch is small ($d_\alpha \sim 0$ and $d_\eta \sim 0$), interaction seems to only increase with individual variation when asymmetric attack and handling rates are considered, rather than

showing a monotonic decrease as with symmetric attack rates and handling times (Fig. S5-2b). Finally, if the average trait value in the population is larger than the selective optimum ($d_\alpha >> 0$ or $d_\eta >> 0$), both the symmetric and the asymmetric case are congruent.

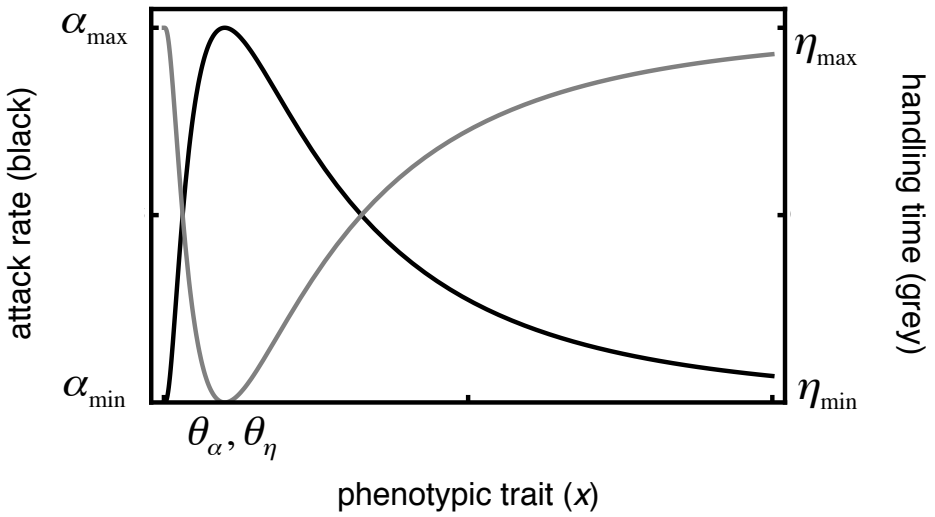

Figure S5-1: Plots of attack and handling time against a given quantitative phenotypic trait, where θ_α and θ_η are the optimal trait values for attack rate and handling time respectively. Note that the ecological attributes are now asymmetric with respect to the trait of interest in contrast to what was assumed in the main text (Fig. 2, main text).

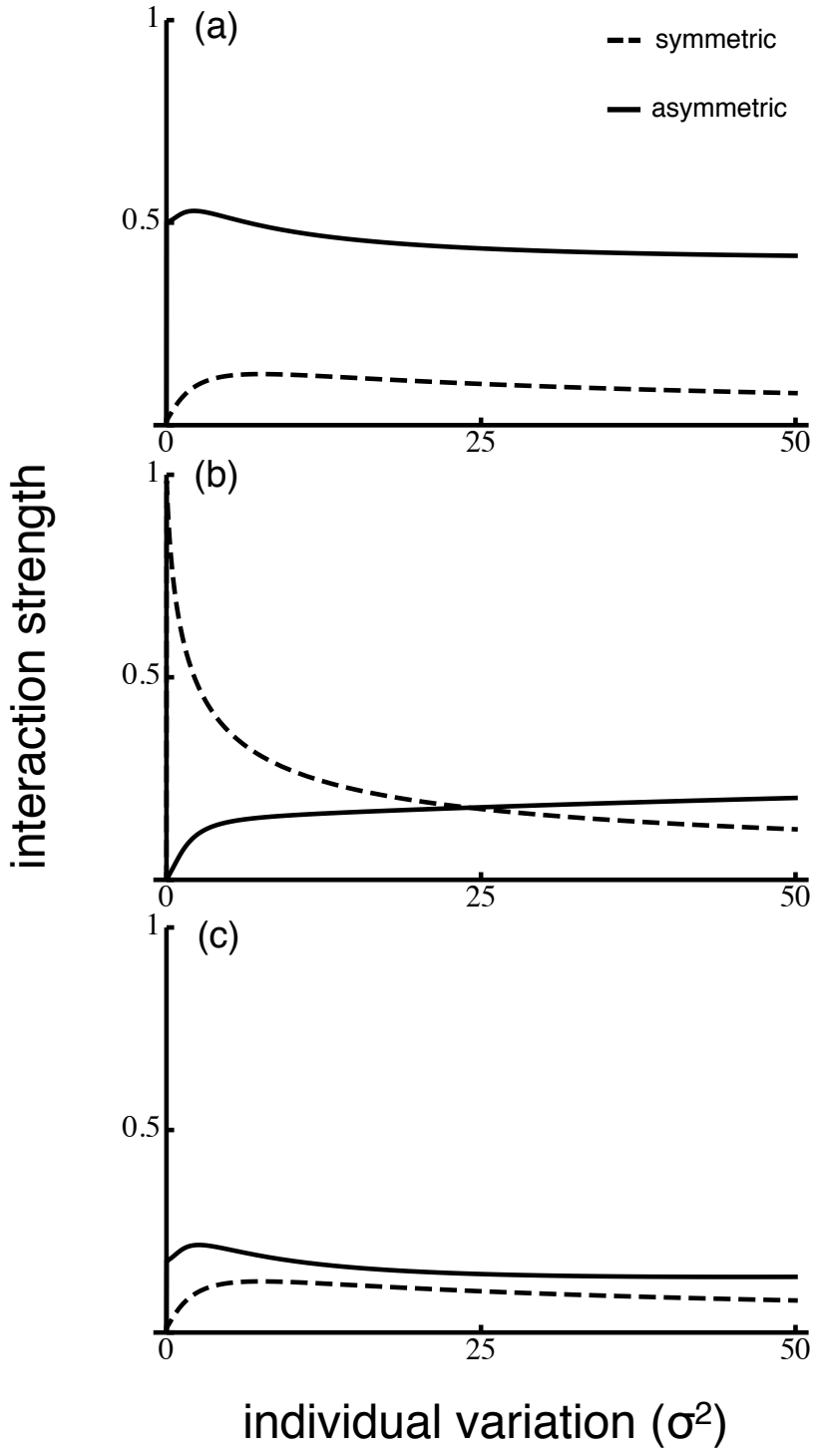

Figure S5-2: Plots of interaction strength against individual variation measured as σ^2. Phenotypic mismatch is large in (a) and (c), and small in (b). Parameter values: (a) $\alpha = 1,\ \eta_{\max} = 2,\ \eta_{\min} = 1,\ \tau = 1,\ \nu = 1,\ d_\alpha = -3,\ d_\eta = 0$. (b) same as (a) but for $d_\alpha = 0$. (c) same as (a) but for $d_\alpha = 3$.

APPENDIX VI

ASYMMETRIC TRAIT DISTRIBUTIONS, AND ASYMMETRIC ATTACK RATE AND HANDLING TIME

In this section, we incorporate asymmetric trait distributions as well as asymmetric attack rate and handling times by means of equations (1), (4) and (5) of the supporting information. Because of the asymmetry, it is now impossible to derive analytic expressions for the (asymmetric) attack rate and handling time, so we have numerically integrated $\overline{I_{R,Lasymm}(\alpha,\eta)}$ and $\overline{I_{C,Lasymm}(\alpha,\eta)}$ to find the interaction strength with varying individual variation σ^2 as:

$$\overline{I_{R,Lasymm}(\alpha,\eta)} = -R \int_{-\infty}^{\infty} \frac{\alpha_{asymm}(x)}{1+\alpha_{asymm}(x)\,\eta_{asymm}(x)\,R}\, Lp(x,\bar{x})\,dx \tag{8}$$

$$\overline{I_{C,Lasymm}(\alpha,\eta)} = \varepsilon\, C \int_{-\infty}^{\infty} \frac{\alpha_{asymm}(x)}{\left(1+\alpha_{asymm}(x)\,\eta_{asymm}(x)\,R\right)^2}\, Lp(x,\bar{x})\,dx \tag{9}$$

The results for asymmetric distribution and asymmetric attack rate and handling time are comparable to those found in Appendix S5. Specifically, whenever phenotypic mismatch is large enough and the average trait value in the population is smaller than the selective optimum ($d_\alpha << 0$ or $d_\eta << 0$), the symmetric and the asymmetric cases yield comparable predictions (Fig. S6-1a). Conversely, the interaction strength seems to be maximized by intermediate levels of individual variation whenever phenotypic mismatch is small ($d_\alpha \sim 0$ and $d_\eta \sim 0$), but this differs from what is predicted by the symmetric case (Fig. S6-1b). Finally, whenever the average trait value in the population is larger than the selective optimum ($d_\alpha >> 0$

or $d_\eta >> 0$, Fig. S6-1c), both symmetric and asymmetric cases are congruent. Overall, it seems that asymmetric relationships between the attack rate and the handling time with the underlying controlling quantitative trait precludes interaction strengths to decrease with individual variation, but the opposite is truth whenever only asymmetric distributions are considered.

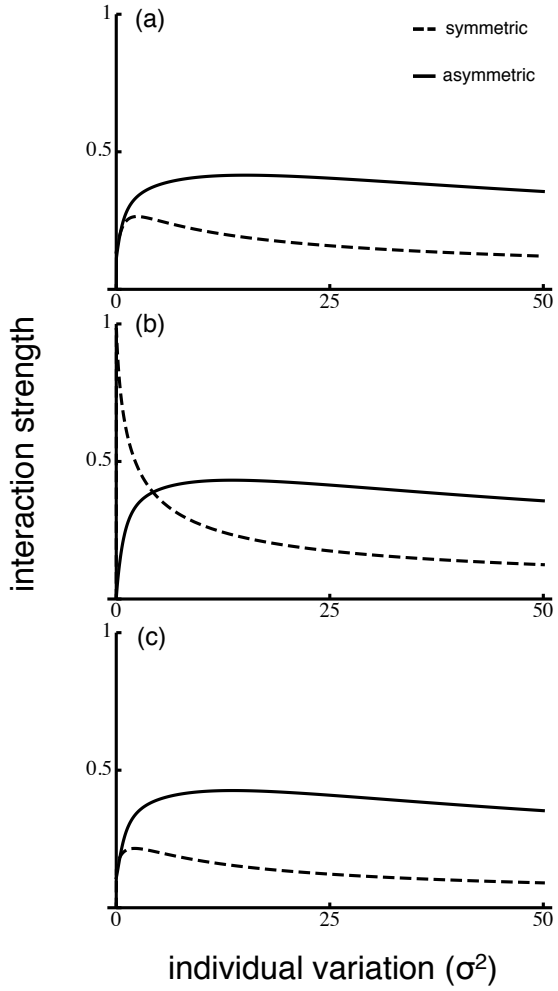

Figure S6-1: Plots of interaction strength against individual variation measured as σ^2. Phenotypic mismatch is large in (a) and (c), and small in (b). Parameter values: (a) $\alpha = 1,\ \eta_{\max} = 2,\ \eta_{\min} = 1,\ \tau = 1,\ \nu = 1,\ d_\alpha = -2,\ d_\eta = 0$. (b) same as (a) but for $d_\alpha = 0$. (c) same as (a) but for $d_\alpha = 5$.

APPENDIX VII

CONSUMER PERSISTENCE

Large values of individual variation can lead to consumer extinction (Fig S7-1), as suggested by eqn 14 and eqn 15 of the main text.

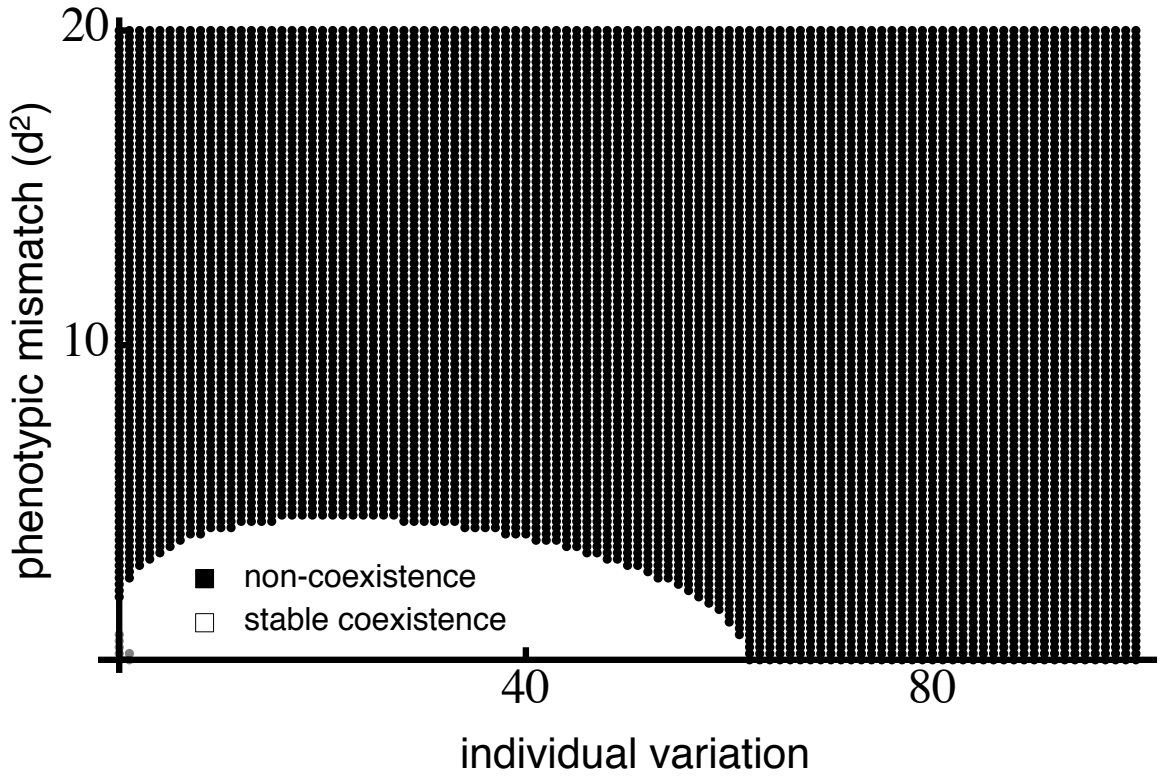

Figure S7-1: Outcome of the consumer-resource interaction as a function of individual variation (σ^2) and phenotypic mismatch between preys and predators (d^2). In the black region, consumers go extinct but the resource survives, while in white and grey regions both consumers and resources coexist. Parameter values: $\alpha_{\max} = 2$, $\eta_{\max} = 2$, $\eta_{\min} = 1$, $\varepsilon = 0.5$, $\tau = 1$, $\nu = 1$, $d_\alpha = d_\eta$, K=1, $\beta = 0.1$.

APPENDIX VIII

PERSISTENCE CONDITIONS

Here we show that for those values of σ^2 for which coexistence is ensured, the larger σ^2 is, the more stable the system becomes. To do so, we observe that, if σ^2 is very small, then the following equality holds,

$$\frac{\bar{\alpha}(\bar{x})\,R}{1+\bar{\alpha}(\bar{x})\,\bar{\eta}(\bar{x})\,R} = \int_{-\infty}^{\infty} \frac{RC\,\alpha(x)}{1+\alpha(x)\,\eta(x)\,R}\,p(x,\bar{x})\,dx\,, \tag{10}$$

where:

$$\begin{aligned}\bar{\alpha}(\bar{x}) &= \int_{-\infty}^{\infty} \alpha(x)p(x,\bar{x})\,dx \\ &= \frac{\alpha_{\max}\,\tau}{\sqrt{\sigma^2+\tau^2}}\exp\left[-\frac{d_\alpha^2}{2\,(\sigma^2+\tau^2)}\right], \end{aligned}\tag{11}$$

$$\begin{aligned}\bar{\eta}(\bar{x}) &= \int_{-\infty}^{\infty} \eta(x)p(x,\bar{x})\,dx \\ &= \eta_{\max} - \frac{\nu\left(\eta_{\max}-\eta_{\min}\right)}{\sqrt{\sigma^2+\nu^2}}\exp\left[-\frac{d_\eta^2}{2\,(\sigma^2+\nu^2)}\right], \end{aligned}\tag{12}$$

and $d_\alpha = \bar{x}-\theta_\alpha$ and $d_\eta = \bar{x}-\theta_\eta$, are the distance between the mean trait in the population and the adaptive optimum (phenotypic mismatch).

Hence, assuming that individual variation is small enough, we can assess local stability of the dynamic system by replacing the functional response defined in the main text (in eqn 13 of the main text, or right side of eq. 10 in appendix) by the functional response evaluated at $\bar{\alpha}(\bar{x})$ and $\bar{\eta}(\bar{x})$, and by then calculating the Jacobian of the system at its equilibrium:

$$J\big|_{R^*,C^*} = \begin{pmatrix} -\dfrac{r\,d\left[\varepsilon - K\varepsilon\,\bar{\alpha}(\bar{x})\bar{\eta}(\bar{x}) + d\,\bar{\eta}(\bar{x})\left(1 + K\,\bar{\alpha}(\bar{x})\bar{\eta}(\bar{x})\right)\right]}{K\;\bar{\alpha}(\bar{x})\bar{\eta}(\bar{x})\varepsilon\left(\varepsilon - d\,\bar{\eta}(\bar{x})\right)} & -\dfrac{d}{\varepsilon} \\ r\left(\dfrac{d}{K\,\bar{\alpha}(\bar{x})} + \varepsilon - d\,\bar{\eta}(\bar{x})\right) & 0 \end{pmatrix}. \qquad (13)$$

The system is stable, if and only if the determinant of $J\big|_{R^*,C^*}$ is positive but its trace is negative. The latter is true whenever:

$d < \dfrac{\varepsilon}{\bar{\eta}(\bar{x})}$ and $\bar{\alpha}(\bar{x}) < \dfrac{\varepsilon + d\,\bar{\eta}(\bar{x})}{K\,\bar{\eta}(\bar{x})\left(\varepsilon + d\,\bar{\eta}(\bar{x})\right)}$. We can now use (11) of the appendix to obtain:

$$\frac{\alpha_{\max}\,\tau}{\sqrt{\sigma^2 + \tau^2}} \exp\left[-\frac{d_\alpha^2}{2\,(\sigma^2 + \tau^2)}\right] < \frac{\varepsilon + d\,\bar{\eta}(\bar{x})}{K\,\bar{\eta}(\bar{x})\left(\varepsilon + d\,\bar{\eta}(\bar{x})\right)}. \qquad (14)$$

If phenotypic mismatch is small ($d_\alpha^2 \sim 0$), we can rearrange the eq. 14 to obtain:

$$\sigma^2 > \frac{\alpha_{\max}\,\tau\,K\,\bar{\eta}(\bar{x})\left(\varepsilon - d\,\bar{\eta}(\bar{x})\right)}{\varepsilon + d\,\bar{\eta}(\bar{x})} - \tau^2. \qquad (15)$$

Finally, if we further assume that variation in attack rate has a larger effect than that in handling time, as observed in appendix 3, we get eq. 3.3 of the main text:

$$\sigma^2 > \frac{\alpha_{\max}\,\tau\,K\,\eta_{\max}\left(\varepsilon - d\,\eta_{\max}\right)}{\varepsilon + d\,\eta_{\max}} - \tau^2. \qquad (16)$$

Eq. 16 implies that for the system to be stable, individual variation needs to be larger than a certain amount. This is supported by our simulations (Fig 3, main text), as increasing variation forces the system through a Hopf bifurcation, from an attractive limit cycle to an attractor node. Although the limit cycle is orbitally stable, the population fluctuations underwent by both interacting species makes the system more likely to lose species due to demographic or environmental variability.

APPENDIX IX

RELATION BETWEEN ATTACK RATE AND INTERFERENCE COMPETITION

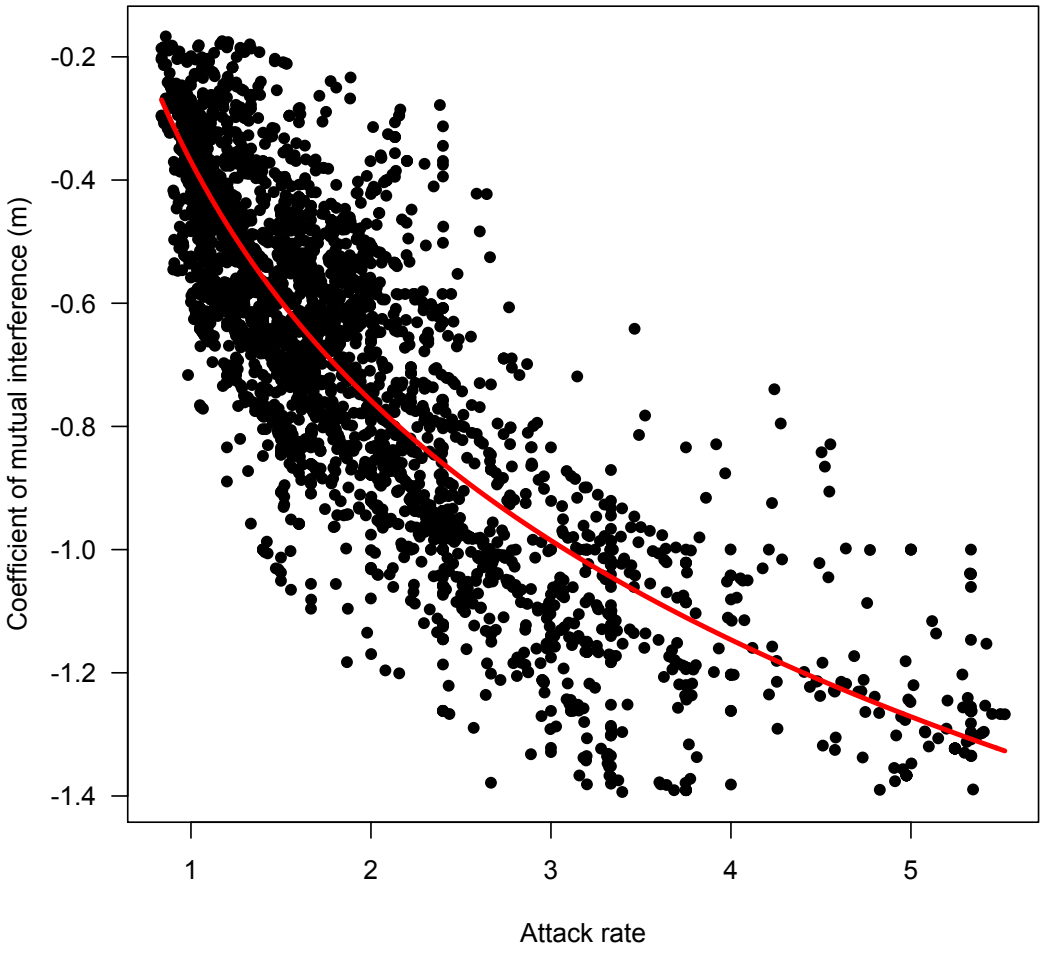

Figure 1: Plot of interferences against attack rate. The red line represents the fitted logarithmic relation between the two variables.

APPENDIX X

EQUILIBRIUM SOLUTION OF THE MASTER EQUATION

In this appendix I show how Eq. (4) of the main text can be derived from Eq. (3). Assuming that as $t \to \infty$, the system will go to a stationary distribution $P(n,\infty)$, we can set $\frac{dP(n,\infty)}{dt} = 0$ and solve:

$$C_{n-1}P(n-1,\infty) + E_{n+1}P(n+1,\infty) - P(n,\infty)\left(C_n + E_n\right) = 0 . \quad (1)$$

At n=0, $C_{-1}P(-1,\infty) + E_1P(1,\infty) - P(0,\infty)\left(C_0 + E_0\right) = 0$. Because $C_{-1} = 0$ and $E_0 = 0$ (see main text), we obtain:

$$P(1,\infty) = \frac{C_0}{E_1}P(0,\infty) . \quad (2)$$

At n = 1, we obtain:

$$P(2,\infty) = \frac{C_1C_0}{E_2E_1}P(0,\infty) . \quad (3)$$

So, by recurrence, we obtain:

$$P(n,\infty) = \frac{C_0 \ldots C_{n-1}}{E_1 \ldots E_n}P(0,\infty) . \quad (4)$$

Now, $P(0,\infty)$ can be determined from the normalization condition, $\sum_{n=0}^{N} P(n,\infty) = 1$:

$$P(0,\infty) + \sum_{n=1}^{N} P(n,\infty) = 1 . \quad (4)$$

Then, we replace with (4) to obtain:

$$P(0,\infty) + P(0,\infty)\sum_{n=1}^{N} \frac{C_0 \ldots C_{n-1}}{E_1 \ldots E_n} = 1 , \quad (5)$$

which reduces to,

$$P(0,\infty) = \frac{1}{1+\sum_{n=1}^{N} \frac{C_0 \ldots C_{n-1}}{E_1 \ldots E_n}} \quad . \tag{6}$$

APPENDIX XI

CLOSED FORM EXPRESSION FOR $P(n,\infty)$

In this appendix I show that by replacing Eqs. (1) and (2) from the main text in Eqs. (4) and (5) we can obtain the stationary distribution in Eq. (6) of the main text. Replacing (1) and (2) on (23), and assuming $\phi = 1$ we obtain:

$$P(n,\infty) = \frac{c\left(1-\frac{1}{N}\right)\times 2c\left(1-\frac{2}{N}\right)\times\ldots\times(n-1)c\left(1-\frac{n-1}{N}\right)}{e\times 2e\times\ldots\times ne}P(0,\infty). \qquad (1)$$

Which can be rearranged as follows:

$$P(n,\infty) = \frac{1}{e^n}\frac{\frac{c}{N}(N-1)\times 2\frac{c}{N}(N-2)\times\ldots\times\frac{(n-1)}{N}c(N-n+1)}{1\times 2\times\ldots\times n}P(0,\infty),$$

$$\Leftrightarrow P(n,\infty) = \frac{1}{e^n}\left(\frac{c}{N}\right)^{n-1}\frac{(N-1)\times(N-2)\times\ldots\times(N-n+1)}{n}P(0,\infty),$$

$$\Leftrightarrow P(n,\infty) = \frac{1}{ne^n}\left(\frac{c}{N}\right)^{n-1}\frac{(N-1)\times(N-2)\times\ldots\times 1}{(N-n+2)\times\ldots\times 1}P(0,\infty),$$

$$\Leftrightarrow P(n,\infty) = \frac{1}{ne^n}\left(\frac{c}{N}\right)^{n-1}\frac{\Gamma(N)}{\Gamma(N-n+1)}P(0,\infty). \qquad (2)$$

By replacing $P(0,\infty)$ by (6) we obtain Eq. (6) of the main text.

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